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# TRANSACTIONS

OF THE

## Thirtieth Annual Meeting

OF THE

# AMERICAN LARYNGOLOGICAL ASSOCIATION

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MONTREAL, CANADA

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TRANSACTIONS OF THE THIRTIETH ANNUAL MEETING  
OF THE AMERICAN LARYNGOLOGICAL  
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HELD AT MONTREAL, CANADA.

MAY 11, 12, AND 13, 1908.

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THE PRESIDENT'S ADDRESS.

BY HERBERT S. BIRKETT, M. D.

A BRIEF ACCOUNT OF THE HISTORY OF MEDICINE IN THE PROVINCE OF QUEBEC, FROM 1535 TO 1838.

*Fellows of the American Laryngological Association :*

Permit me to express my deep sense of appreciation of the honor you conferred upon me in electing me your President. It has been my endeavor to discharge the responsibilities of this high office to the best of my abilities, and I trust to your satisfaction and with credit to the Association.

It is with very great pleasure that the medical profession of this city and of this Dominion, welcome you to the first convention of this Association to be held on Canadian soil. Your loyal support in the preparation for this year's meeting, and especially the co-operation of our able Secretary, have enabled us to present an excellent programme of papers for your consideration. I can only add that we shall endeavor to make your brief stay in Montreal as pleasant as I trust it will be profitable.

At the recent Tuerck-Czermak Jubilee, this Association was unfortunately not represented, but our good wishes were conveyed to the President through a letter from the Secretary.

It is fitting that I should refer on such an occasion to the loss

which this Association has sustained by the death, on the 9th of December last, of one of our most eminent corresponding members, Professor Moritz Schmidt.\* Perhaps I may recall to you the chief points in his brilliant career. He was born in Frankfurt-on-Main, on the 15th of March, 1838. He studied in Göttingen, Vienna, Berlin, Utrecht, England and France, and graduated in 1860. After the practice of general medicine for twenty-six years, he began in 1886 to devote his time exclusively to diseases of the ear, nose, throat and lungs. He retired from practice in 1903, after receiving signal honors from his colleagues and his Sovereign. He made many valuable contributions to laryngology, most notably the work with which you are no doubt familiar, on diseases of the upper respiratory passages.

We have to chronicle a sad event in connection with the Tuerck-Czermak Jubilee, in the sudden death from cardiac syncope of our recently elected Honorary Fellow, Professor Leopold von Schroetter, which occurred on Wednesday morning, April 25th, he having on the previous day delivered, apparently in the best of health, the oration on the opening of the First International Congress of Rhino-laryngology.

Hofrat Professor Dr. Leopold von Schroetter,\* Ritter von Kristelli, was born in Graz on the 27th of February, 1837. He studied medicine at Vienna, and graduated in 1861, receiving immediately an appointment as Assistant to Skoda, after whose death he acted as substitute in the Professorship of clinical medicine, and in 1875, became extra-ordinary Professor of throat and chest affections. In 1881, he was appointed Director of the clinic for laryngology in the Allgemeines Krankenhaus, which position he relinquished in 1890 to accept the Professorship of clinical medicine, which position he was about to resign, having reached the age-limit of seventy.

His principal writings of laryngological interests are: "Laryngologische, Mittheilungen," (1875), "Beitrag zur Behandlung der Larynxstenosen" (1876), "Vorlesungen ueber Krankheiten des Kehlkopfs" etc., (1867), "Vorlesungen ueber Krankheiten

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\*I desire to express my indebtedness to my friend, Sir Felix Semon, K. C. V. O., for the notes kindly supplied regarding the deceased members.

der Trachea" (1901), besides many important papers on clinical medicine. He took great interest in tuberculosis, and was the founder of the celebrated Austrian Sanatorium "Alland." He together with Stoerk and Schnitzler were the great laryngological authorities of Vienna. Finally, his taking part in the celebrated November consultation of 1887 in the case of the then Crown-Prince of Germany, has become historical.

Dr. Newcomb, our worthy Secretary, has but expressed, I am sure, our concordant wish in conveying to the families of our deceased members, the sincere condolence of this Association on the loss of such distinguished men.

The unique significance of this time and place have suggested to me the topic upon which I am to address you. I feel that the honor which you have conferred upon me in electing me your President is in a sense international, and may perhaps justify the choice of a subject which is in a measure national in its scope. It occurred to me that a brief account of the early history of medicine in the Province of Quebec might not be inappropriate in a meeting which is held for the first time in the history of the Association in Canada, in this, the oldest Province of the Dominion, and on this, the ter-centenary of the founding of Quebec by Samuel de Champlain.

On the 3rd of July, 1908, Canada will celebrate the three hundredth anniversary of the foundation of Quebec, the real birthday of the Canadian Nation. The history of medicine in this Province, however, began seventy-three years before, when Jacques Cartier built his rude fort on the banks of the Saint Charles, facing the Indian village of Stadaconé.

It was on the 14th of September, 1535, that Cartier chose this place as a temporary shelter, and, leaving his men to construct an abode against the approaching winter, sailed up the river St. Lawrence as far as the village of Hochelaga, the site of which includes a part of the McGill University.

Upon his return to Stadaconé, he found the little fort in a state of alarm. Scurvy had broken out in the Indian villages and, according to the account of the leader of the tribe, fifty had already succumbed. Every precaution possible was taken to prevent intercourse with the natives; but, nevertheless, the

disease began to manifest itself amongst Cartier's crew, "In-somuch as some did lose all their strength, and could not stand on their feete, then did their legges swel, their sinnowes shrinke as black as any cole. Others also had their skins spotted with spots of blood of a purple colour; then did it ascend up to their ankels, knees, thighes, shoulders, armes and necks: their mouth became stincking, their gummes so rotten that all the flesh did fall off, even to the roots of the teeth, which did also almost all fall out. With such infection did this sickness spread itselke in our three ships, that about the middle of February, of a hundred and tenne persons that we were, there were not ten whole, so that one could not help the other, a most horrible and pitifull case, considering the place we were in, forasmuch as the people of the country would dayly come before our fort and saw but few of us. There were already eight dead, and more than fifty sicke, and as we thought, past all hope of recovery."

The winter wore on; disease and death fell like a pall over the little fort and Cartier was threatened with a danger more to be feared even than scurvy. Upon the heights of Cape Diamond stood the villages of the Ajoaste Starnatam, and Tailla, branches of the ferocious Iroquois, the first passion of whose souls was war. Hitherto they had been overawed by the presence of the wonderful ships manned by sturdy crews, but gradually, as signs of activity disappeared amongst the white men, they grew bolder, and at any moment the sound of the war-whoop might be the signal for an invasion of the ships.

Among the crew, there was evidently a man possessing a knowledge of surgery, who determined to hold a post mortem on the body of one Philip Rougemont, 22 years of age, to ascertain the cause of death and, if possible, save the remnant of the ship's company. Here, in the quaint language of the time, is the result of the first autopsy held in Canada: "He was found to have his heart white, but rotten, and more than a quart of water about it; his liver was indifferent faire, but his lungs blacke and mortified, his blood was altogether shrunke about the heart so that when he was opened, great quantities of rotten blood issued out from about his heart; his milt towards the backe was somewhat perished, rough as if it had been rubbed against

a stone. Moreover, because one of his thighs was very blacke without, it was opened, but within, it was whole and sound; that done, as well as we could he was buried."

The captain was a man of observation. Walking one day on the ice, he perceived a band of the Indians from Stadaconé; amongst them was one Domagia, who, less than two weeks before, was in the grip of the disease, "with his knees swolen as bigge as a child of two years old, all the sinews shrunke together, his teeth spoyled, his gummes rotten and stincking. Our captain seeing him whole and sound, was thereat marvellous glad, hoping to understand and know of him how he had healed himselfe, to the end that he might ease and help his men." From Domagia he learned that the specific was the bark and sap of a certain tree called in their language "Ameda." This tree is known to-day as "l'épinette" or the spruce. The method of preparation was as follows:

"To take the bark and leaves of the sayd tree, and boile them together, then to drink of the sayd decoction every other day, and to put the dregs of it upon his legges that is sicke: moreover they told us that the vertue of that tree was to heale any other disease."

The sailors in those days, as in these, were troubled with another complaint, "French Pockes," but after liberal doses of the bark, they "were cleane healed." From this time forth, the decoction was at a premium and men were ready to kill each other in their eagerness to test its efficiency, "so that a tree as big as any oake in France, was spoiled and lopped bare, and occupied all in five or sixe daies, and it wrought so wel, that if all the physicians of Montpelier and Lovaine had bene there with all the drugs of Alexandria, they would not have done so much in one yere, as that tree did in sixe daies, for it did so prevaile, that as many as used of it, by the grace of God, recovered their health."

With the opening of navigation, however, Cartier returned to France, prepared for fresh deeds of adventure. Brave, indeed, were the hearts of those men who lived their lives under the perpetual menace of destruction, facing the perils of the deep to meet the still greater dangers of pestilence on land. Steadfast, heroic,

subdued only by the grim reaper, Death, such were the discoverers of old.

Cartier visited our shore again, and spent another winter under scarcely more favorable conditions, notwithstanding that he had found a sovereign remedy for scurvy. With his departure from our shores in 1542, a curtain falls over the history of the country between Stadaconé and Hochelaga, for a period of sixty years, and we find no reliable account of a settlement on the banks of the St. Lawrence. Nevertheless, in the year 1578 there were one hundred and fifty vessels engaged in trade with France in the St. Lawrence, and over two hundred ships belonging to the English, the Spanish and the Portuguese. We may presume therefore that there were temporary settlements, and it is probable that medical supervision in some form was not overlooked.

In the month of April, 1604, Des Monts undertook to colonize New France, and among the men who accompanied him was Louis Hébert, an apothecary, born in Paris. Hébert remained at Port Royal until the month of August, 1607, when the settlement was abandoned. Ten years later, he was invited by Champlain to take up his residence at Quebec, and he was amongst the first real settlers who cultivated the soil for a livelihood.

His residence was in the Upper Town, near the Basilica. He soon became a prominent figure in the little community, and in 1621 was named "Royal Procurator." The first entry of a marriage in Quebec is that of Hébert's daughter. In 1826, he was granted the fief St. Joseph on the river St. Charles, with the title of *Sieur d'Espigny*. He died in January, 1627, as the result of an accident, and was buried in the cemetery of the Recollets. He appears to have been held in high esteem by the Governor, the Recollets and the Citizens.

When Champlain arrived in Canada in 1608, he left his ships at Tadousac, near the Saguenay, and sailed up the St. Lawrence in a small craft to found the future city of Quebec. In his company were a number of mechanics, and a surgeon named Bonnerme. The axemen felled the trees on the narrow belt which skirts the promontory of Cape Diamond, and soon a modest building arose rudely fashioned into a fort, styled the "Abitation de Quebec." While this building was in the course of construction



a plot was developing to murder Champlain and deliver Quebec into the hands of a band of Spaniards at Tadousac. Bonnerme was suspected of complicity in the plot, but his innocence was established and he was released. He died, however, during the winter from the effects of either scurvy or dysentery. It is difficult to determine which disease decimated the colony.

Amongst the earliest settlers in Quebec was Adrien Duchesne, a surgeon of Dieppe, who probably arrived in 1618. When Champlain surrendered the fort to Sir David Kirke in 1629, Duchesne remained and practised his profession under the English regime. The colony was restored to France in 1632, under the treaty of St. Germain-en-Laye, and a year later, Champlain returned as Governor. A few months before the death of the Governor in 1635, Duchesne received a grant of land from the Crown and later transferred it to Abraham Martin, after whom the Plains of Abraham were named. In 1645-6 we find him interested in the young Charles Lemoine, Seigneur of Longueuil, and father of the famous Iberville. His practice extended from Quebec to the post at Three Rivers, midway between Quebec and Montreal.

Another surgeon, a contemporary of Champlain, was Robert Giffard, Sieur de Beauport, a native of Montagne au Perche, in France. He was born in 1587 and came to Quebec in 1628, in the capacity of surgeon to the company of "The Hundred Associates." He resided on the Beauport shore and appears to have spent his leisure in hunting and fishing. A year later, however, Quebec fell into the hands of the British, and he returned to France. After the restoration, he obtained a grant of the Seigneurie de Beauport, in 1634. He was evidently a man of considerable influence and enterprise, for within the space of two years he induced fifty excellent families to settle on the banks of the St. Lawrence.

The task of colonization in those days was exceedingly difficult. Canada was under the monopoly of the great fur trading companies, whose policy was opposed to any extensive plan of settlement. A sufficient number of residents to facilitate trade was all that was thought desirable. His efforts, therefore, under the circumstances, were remarkable.

In 1647 Giffard obtained a larger grant of land, known as the fief St. Gabriel. A year later he was appointed a member of the Council, a body empowered to enact local laws and to arbitrate the differences of private individuals. He practised his profession in Quebec and in the surrounding country, and was visiting physician to the Hôtel Dieu, founded in 1639. Giffard was recognized as a man of considerable importance in his time, and when he died in 1648, the community of the Hôtel Dieu felt that they had lost not only an eminent physician, but a true friend.

The colony founded by Champlain at the foot of Cape Diamond had been in existence for half a century, but its institutions were still very primitive. In 1658, Jean Madry, a surgeon of Quebec, while on a visit to France, obtained from the Sieur Barinois, Premier Surgeon in Ordinary to the King, and provost of the Royal College of St. Come, in the University of Paris, "Letters of Surgeon" for himself, with power to establish in all parts of Canada the authority of surgery, in order that the sick might receive more efficient medical care. These letters were registered in the Sovereign Council at Quebec, but they do not appear to have been of much service to the profession. Little appears to have been preserved regarding Madry. He was elected the first Mayor of Quebec, an office which was abolished after a trial of five weeks. The machinery of municipal government was too complicated and expensive for the needs of a community of less than five hundred souls.

There was at this time evidently a need of more physicians to attend the wants of the settlers, for we find that Jean Martinet, Sieur de Fonblanche, who was born in Moustiers, Bourgogne, undertook to teach his brother-in-law, Paul Prudhomme, the art of medicine and surgery by the system of apprenticeship. This is the first recorded instance of a method of teaching, which subsequently became much in vogue. This system led to such an increase in the number of physicians that keen competition and rivalry resulted in the establishment of a form of "Combine," or partnership. Those entering into such a partnership "had all their possessions, furniture, provisions, roots and peltry; all the products which they gathered from the ground, their instruments of surgery, their medicines and, lastly, all the revenue which they



would obtain from their work and industry" pooled. It was further agreed that each of them would devote himself to the profit of the partnership as far as lay in his power, and that he would not run into debt (without the authority of his associates) exceeding the sum of five sous. At the end of four years, the profits were equally divided. It was further stipulated that in the case of the death of a member before the expiration of this term, all the goods of the community would belong to the survivors.

An interesting physician of this period was Dr. Gauthier, who, as a corresponding member of the Royal Academy of Sciences, had made many botanical observations in Canada. It was he who discovered the Wintergreen plant, which to-day carries his name, and is known as the "*Gaultheria Procumbens*." Gauthier devoted himself also to the study of law under the tutorship of the *Sieur Verrier*, Procurator-General.

With the considerable increase of population, not only at Quebec, but also at *Ville Marie* (Montreal), disease in various forms had become much more prevalent. Smallpox especially had spread to such a degree that it was necessary to provide some suitable place to care for and attend the unfortunate sick.

The *Duchesse d'Aiguillon*, who came to Canada from *Dieppe*, with the intention of relieving and converting the Indians, finding such an unfortunate condition of affairs, decided to found a hospital. She received a grant of land in 1637, and established in 1639 the *Hôtel Dieu*, at Quebec. Of the attending staff of this hospital, there is one who deserves especial notice, *Michel Sarrazin*, who came to Canada in the capacity of Surgeon-Major of the French troops. After practising his profession in Quebec for some years, he returned to France for the purpose of pursuing his medical studies at the University of *Rennes*. Three years later he received the title of physician to the King, and was delegated by the Academy of Science of France to make a special study of animals and plants in Canada. His work in this direction was remarkable. In the "*Transactions of the Academy of Science*" for 1704 there appears a paper by *Sarrazin* on the "*Anatomy of the Beaver*," which even to-day is regarded as the best work on the subject. His further anatomical researches dealt

with the musk-rat, the seal and ground hog. He also investigated the mineral waters at Cap de la Madeleine. In his botanical researches he discovered the pitcher plant, which is known to-day as the "*Sarracenia Purpurea*." In recognition of these extensive researches, he received a pension of 550 pounds. To Sarrazin's skill as a physician the records of the Hôtel Dieu bear ample testimony.

Expansion and colonization in this new country frequently followed in the wake of missionary activity. "Not a cape was turned," writes Parkman, "but a Jesuit led the way." Few more striking examples of religious enterprise are to be found than the establishment of the little colony at Ville Marie (Montreal). The founders were Jerome Royer de la Dauversière, receiver of taxes at La Flèche, in Anjou, and Jean Jacques Olier, a young priest of Paris. To both of these men there came at nearly the same time (1636) the idea of founding a religious colony at Montreal. They formed an Association for this purpose and called it the "Society of Montreal." At first, the Society consisted of six members, but the number was soon increased to forty. Amongst these was Madame de Bullion, who gave 42,000 livres for the purpose of endowing a hospital in the new settlement. In 1641, Paul de Chomedey, Sieur de Maisonneuve, set out for New France with a company of 100 soldiers under his command. Amongst those who joined the expedition was Mademoiselle Jeanne Mance, the descendant of an honorable and influential family in France, who became a member of the Society of Montreal. To Mademoiselle Mance was entrusted the control of the funds given by Madame de Bullion for the establishment of a hospital at Montreal. On the arrival of the expedition in the St. Lawrence, the summer was found too far advanced to proceed to Montreal, so the winter was spent in Quebec. Many efforts were put forth to dissuade the company from pushing on to Montreal, but in vain. The following spring they sailed up the St. Lawrence and took possession of the island on the 17th of May, 1642. The settlement was named Ville Marie, in honour of the Virgin Mary, to whom it was especially dedicated.

Of a hospital, however, there seemed for a time to be little need. The colony was exceptionally free from sickness, and

there were as yet no wounded to be attended, for the Iroquois knew nothing of the little settlement and left the colonists to build in peace. Mlle. Mance even suggested that the endowment for the hospital be given to the Jesuit Missions. To this, however, Madame de Buillion refused to consent. Work on the hospital was continued and the building was opened on the 8th of October, 1644. There were two rooms for the sick, a room for Mlle. Mance, a kitchen, apartments for the servants and a chapel ten feet square, built of stone. During the following year the furniture, medicine and surgical instruments arrived, and some live stock was brought for the use of the inmates. The usefulness of such an institution was soon conceded, for scarcely had the walls been completed when news of the settlement reached the Iroquois and encounters with the savages began. The wounded were among the first to reap the benefit of the shelter and careful attention which the hospital afforded.

A few years later it was rumored that the "Society of Montreal" had lost interest in the undertaking and was on the point of dissolution. This prompted Mlle. Mance to return to France and approach Madame de Bullion for further assistance for the hospital. In this she was entirely successful. It now became evident, as the work of fortification went on, that the hospital was likely to become permanently established. The Iroquois, however, decided to renew their attempts to destroy it. In July, 1651, the hospital was besieged and had to be abandoned. The defenders took refuge in the fort. The colony was in danger of destruction and it was necessary to have reinforcements. To procure aid Maisonneuve undertook a journey to France, for which the hospital endowment furnished the money. Upon his return with more than 100 men, a new hospital was built and opened in the spring of 1654. The reinforcement of men brought by Maisonneuve put new life into the colony and enabled them successfully to resist the Iroquois. Thus, a second time, Mlle. Mance, by her judicious use of the endowment funds of the hospital, saved the colony of Montreal.

In 1657, Mlle. Mance, who had been Directress of the Hospital since its foundation, fell on the ice, fractured her right arm and dislocated her wrist. Etienne Bouchard, a surgeon, set the

arm, but failed to discover the dislocation of the wrist. Jean Madry, also examined her injuries, but with no better success. The arm remained entirely useless until Mlle. Mance's temporary return to France, occasioned by the financial needs of the hospital. Here the use of her arm, pronounced by the most eminent surgeons in Paris to be beyond recovery, was miraculously restored by touching the casket containing the heart of M. Olier. Mlle. Mance issued the following statement, in writing, regarding the miraculous cure:

AN NOM DE LA TRES SAINTE TRINITE, AMEN.

"Je, Jeanne Mance, reconnais et confessé en la presence de mon Dieu avoir receu la grace de l'usage de ma main droite par les merites de feu Monsieur Olier, ce qui s'est passe en cette maniere, . . . . . Je declare que tout ce que j'ay escrit cy-dessus en ces deux petites feuilles est veritable et sincere en foi dequoy je l'ay escrit et signe de la mesme main dont j'ay receu l'usage a Paris, ce 13 Fevrier, 1659.

"JEANNE MANCE."

The efforts to secure additional funds for the hospital from Madame de Bullion were successful. Of the 22,000 francs obtained for the purpose, Mlle. Mance entrusted 20,000 to the care of Le Royer de la Dauversière for the purpose of investment; but upon his death, it was found that the funds had been misappropriated, and the hospital was left without necessary means properly to carry on the work. On account of this loss, the inmates were reduced to extreme poverty and suffered intensely from the cold and lack of nourishing food. Under such circumstances Mlle. Mance continued to administer the affairs of the hospital until 1673. After her death, in that year, the financial affairs of the hospital caused increasing anxiety. The income had been reduced by heavy losses to 12,000 livres a year, four hundred of which were derived from the cultivation of one hundred arpents of land. Owing to the French Revolution their revenue for a time was lost altogether, but the funds were recovered in 1821.

Hôtel Dieu was destroyed by fire in 1695, in 1721 and in 1734.

During the siege of Quebec, in 1759, Surgeon Robert Adair

established hospitals for the British on the Island of Orleans, while the surgeons of Quebec appear to have attended to the needs of the French. André Arnoux, a native of Rochefort, was Surgeon-Major of the French troops. He and his son established themselves on St. Louis street, near the Ursuline Convent. When Montcalm received his mortal wound, on the morning of the 13th of September, he was taken to the home of the surgeon, where his wounds were dressed by the younger Arnoux. It was here that Montcalm died, at four o'clock on the following morning.

After the year 1773 the attention of the Government of Lower Canada was drawn to the increasing ravages of a peculiar disease which, originating at Mal Baie, had spread to other parts of the Province. A tradition exists that it was brought to Mal Baie by a detachment of Scottish troops, who were either sent there on service or had been thrown there by some accident of navigation a few years before. The great resemblance of this disorder to syphilis and its disgusting character, led the unhappy sufferers who were aware of that resemblance, to conceal their condition as long as possible, while those who were not aware of it looked upon the symptoms as the result of scurvy, and trusted to ordinary remedies or to time, for relief. From both these causes, in addition to the remoteness of the parish where the disease first made its appearance, and the want of medical assistance on the spot, the disease was not commonly known until it reached such a height as to require the attention of the Government.

The symptoms of the disease, as stated in a printed description circulated by the Government in 1785, are as follows:

"Ulcerations appear on the soft palate, tonsils, uvula and mucous membrane of the mouth. As time goes on, these extend and cause destruction of these parts. At a later period, nodes appear on the head, arms, legs and trunk, which, breaking down, produce extensive stinking ulceration. When the disease has attacked the nose for a length of time, it results often in the bridge falling in. Loss of the hair of the head, eyesight and hearing and a horrible stench are generally the precursors of death. No one is exempt from the disease, the child at the breast as well as the aged or infirm being equally attacked."

Shortly after the disease first attracted the attention of the



Government in 1775, General Carleton, who was Governor at the time, sent a surgeon's mate of the 7th Regiment to St. Paul's Bay with instructions to administer medical relief. Upon investigation, the remedy which he distributed was found to consist of calomel and mercurial ointments. This officer was recalled to Quebec, and upon his death, Philippe Louis Francois Badelard, who came from France as Assistant-Major of the French troops, was appointed by General Haldimand, after the departure of General Carleton, to investigate the disease and report thereon. He therefore wrote a pamphlet on the subject, which was printed by the Government. It is stated that the use of the foregoing remedies resulted in many of the afflicted ones being cured.

Upon the exact nature of this disease the opinions of medical men were found to differ. Among those most keenly interested in the subject was Dr. John Bowman, who was employed by the Government in investigating the affected districts and in distributing the remedies. One Robert Jones, of Montreal, wrote an elaborate pamphlet, published in Montreal in 1786, to prove that the disease was not syphilis, while a letter of Dr Charles Blake, Surgeon of H. M. 39th Regiment, states that "This disease is nothing more than a confirmed syphilis showing itself in different ways in different parts of the body and making anomalous symptoms and appearances accordingly. The habits of the Canadians facilitate its communication in various ways. They use the same cup, often borrow one another's pipe to smoke, chew their infant's food and spit it into their mouths. They constantly spit on their floors, and never clean them, and the lower classes are generally regardless of cleanliness. All these circumstances help to communicate the disease, which may take effect anywhere that the skin happens to be broken; the disease being of the nature already stated, it gives way to no remedy but mercury."

The Province of Quebec had, as yet, no facilities for teaching medicine, other than the form of apprenticeship, which did not appeal to the ambitious student. It was necessary, therefore, to go elsewhere for a medical education. The earliest student to go abroad was Francois Blanchet, who was born in 1776, and received his preliminary education at the Seminary at Quebec. At

the completion of his course at the Seminary, he studied medicine in New York, and published a work on "The Application of Chemistry to Medicine." After graduation, Blanchet returned to his native home, Quebec, and entered upon the practise of his profession. His ambition, however, was not satisfied in medicine. He launched into journalism, founded a newspaper called "*Le Canadien*," published several seditious articles, and was imprisoned for a short time by the haughty Governor. His active interest in politics and education, however, led to his election as a member of the Legislative Assembly, and it was he who proposed during his tenure of office the first law on education. During the war of 1812 he occupied the post of Chief Medical Officer in this Province.

Following the example of his preceptor, Blanchet, we find Jacques Labrie, after serving his apprenticeship, going to Edinburgh to complete his course of studies. He was the first to visit and study at the Royal University there. Before leaving for Edinburgh, Labrie founded a newspaper called "*Le Courrier*," which announced his return to his native city in August, 1808. Like his preceptor, Labrie entered politics and became a member of the Chamber of Assembly; and it is to him and his medical colleagues that we are indebted for the institution of those tribunals which now guard the honor of the medical profession.

The condition of the medical profession and the state of education were like everything else, in a state of transition. The medical men of good professional status who were practising in the country at this time were either Canadians who had served their apprenticeship with a physician of standing and then gone to the Mother Country for graduation and further study, or they were, as often happened, Englishmen and Scotchmen who had received their entire training at home. Many of these were former Army Surgeons who had found the demand for their medical services in civil life so great that they had retired from active duty, and were engaged only in their profession as private practitioners. It is said that the type of Englishmen who thus first practised medicine in Canada was as good as British education could make it.

On the other hand, as was natural in a country where there was

no provision for a medical education, and where the demand for medical assistance far exceeded the supply, quackery seems to have flourished abundantly. It was to correct such a condition of affairs that in 1750, the Intendant Bigot introduced an ordinance which may be considered the code of the medical profession in Canada. This ruling provided that no one should practice medicine without passing an examination. Those wishing to practise in a city were to be examined before the physician of the King in the presence of the Lieutenant-Governor of the Jurisdiction. Those wishing to practise in the outlying districts were to pass an examination before the physician of the King and a sub-delegate.

This ruling continued until 1788, when the British Parliament passed an Act which provided that no one should practise physic and surgery within the Province of Quebec or midwifery in the towns of Quebec and Montreal without a license. A license was to be obtained by passing an examination conducted by capable persons appointed by the Governor or Commander-in-Chief of the Province. This regulation did not subject those to an examination who had already taken a degree in any University or had been commissioned as surgeons in the army or navy. "But the ordinance of 1788 had a radical fault. It placed the study and the practice of medicine absolutely in the hands of the Executive Power. It opened the door to partiality, to injustice and to administrative tyranny. The licenses were to be given by the Governor, and the Examiners were to be nominated by the Governor also, which made them simply officers of the Government Executive. At a period when prejudice and narrow ideas in official spheres reigned, this domestication of the profession might cause serious rejection of justice.

"If one can believe the memoirs of a contemporary, this deplorable side of the ordinance soon became evident. The celebrated Pierre de Sales Laterriere, who had studied medicine for some time before leaving France, and who practised medicine from time to time since his arrival in Canada in 1766, found himself subject to the new legislation. He had practised without a diploma, often with success, according to what he says himself. But henceforth, he must, under the penalty of a fine, abandon the



practise of medicine or submit to an examination before the members of a bureau appointed by the Governor. He decided on the latter alternative, and was the first candidate for the study of medicine who presented himself before the Board of Medical Examiners in Canada. Let him relate his unhappy experience:—

“‘I had lost my certificate of St. Come, and the documents of my apprenticeship in Paris, but I knew I had talent enough to pass an examination. I was the first to present myself before the medical bureau of examination, at 8 o'clock in the morning, in the presence of four practitioners and four councillors, and a very large assembly attracted by curiosity because there was a new institution in the country. The examination lasted until four in the afternoon. One question did not wait for another. However correct my answers were, my examiners, because of my failure to present my letters to them, did not wish to permit me to continue practising. It was necessary for me to go to another college to obtain other certificates. Their partiality, their hardness, their malice, were so apparent that many of my friends, mere spectators, said to me, “Go to Cambridge, near Boston, where in a short time, with the knowledge that you have obtained here, you would obtain what you now lack.” My examiners having seen that, and the reproaches that the public were making, rather through shame than friendship said, “Yes, if the candidate should go to Cambridge or elsewhere, we will give him a certificate favourable to the talents and knowledge which he has displayed.”’—(Memoirs of Pierre de Sales Laterriere.)

“During the winter and spring of 1788, M. Laterriere studied at Cambridge. After passing a satisfactory examination, in June, 1789, he obtained the diploma of Bachelor of Medicine and returned to Canada. He appeared again before the Medical Board, consisting of Drs. John Foote, James Fisher, James Davidson, Francois Lajus and Fred Oliva.

“The last clause of the Act provided that anyone holding a university degree should not be subject to an examination. Nevertheless, the examiners wished to examine him, saying that as his diploma was from a foreign university they could only give him a simple certificate of study, and could not receive him

unless he passed an examination. Laterriere submitted to the examination, which he passed successfully.

"The ordinance of 1788 relative to the Medical Profession opened the door to disadvantages of which the most serious was the arbitrariness of the executive. In proportion as the country developed, as the population increased and as the number of candidates for medicine became greater, the acts of injustice also became more frequent. The influence of the national and political question was felt in this sphere as well as in most others. The French-Canadian physicians complained that, owing to official power, the English physicians made them wait and retained the exclusive authority over the administration of the profession and of the conditions of admission to the learned fraternity. It is to be noted that for about forty years there was not a single French-Canadian examiner, either at Quebec or Montreal."—(J. E. Roy, "Histoire du Notariat au Canada.")

This act was repealed in 1831, when another Act was passed regulating the practice of medicine (1 William 4 caput 27). This Act provided that no one should act as Physician without a license from the Governor, and that all persons intending to study physic with a view to obtaining a license should undergo an examination before a *Board of Examiners*, for the election of which provision was made in this Act. The Board was to consist of twelve members, seven forming a quorum.

It was also enacted that no license should be granted unless the candidate were of full age and had served an apprenticeship of five years.

In 1841 an Act was passed to enable persons authorized to practice physic or surgery in Upper or Lower Canada, to practise in the Province of Canada.

By the Act of 1847, the Medical Profession in Lower Canada was incorporated under the name of "*The College of Physicians and Surgeons of Lower Canada*," and was empowered, under certain restrictions, to frame its own statutes for the regulation of the study of medicine in all its departments, and by-laws for its own Government.

In 1849, an amendment to this Act was passed which authorized the Provincial Medical Board to issue licenses.

Let us pause to consider for a few moments what were the general conditions, at this time, of the people of Nouvelle France.

During the course of time, increased intercourse with France and England had led to greater numbers of immigrants reaching the colony. The marked increase of population had brought about the prevalence of disease, "that everlasting traveller which follows humanity wherever it goes," and one of its earliest manifestations was in epidemic form.

The earliest disease to be mentioned was scurvy, (Mal de Terre et Mal de Mer). A description of the symptoms and the treatment, as noted by Jacques Cartier, has already been given. It is interesting to note here that new and more drastic remedies were evidently resorted to. Captain Knox, in his Journal, writes:

"This morning I was an eye-witness to the ceremony of burying a man alive, *mirabile dictu*, for the sea scurvy. To explain this matter, it must be observed that a pit was made in the ground, and the patient stood in it, with his head only above the level earth; then the mould was thrown in loose about him, and there he remained for some hours; this, I am told, is to be repeated every day until his recovery is perfected."

Scurvy, which was known in the early days of New France under the name of "Mal de Terre," and found later to be the same disease as "Mal de Mer," was certainly one of the chief causes which retarded the colonization of Canada. The disastrous results were seen at Tadousac (1600), Island of St. Croix (1604-5), Port Royal (1606-9), Quebec (1609), and on the Island of Miscou (1635).

After scurvy came smallpox, with its attendant train of fatalities. There were during the French regime four characteristic epidemics: 1703, 1732, 1733 and 1755.

The epidemic of 1703 was particularly severe. The registers of the Province of Quebec show that there were over two thousand deaths, Indians included. "Never had such a misery been seen," remarks the historian of the Hôtel Dieu of Quebec. "Every one was deploring the loss of a relative, one of his wife, another of her husband, one his brother, another of his children.

Orphans wept for their parents; all were in tears, and there were no gatherings except for funerals."

In a letter written on the 14th of October, 1733, by Beauharnois to Hocquart, it is stated that "The smallpox, which has afflicted this country since the end of the autumn of 1732, up till to-day, and which has carried off nearly two thousand persons from this colony, has exhausted the resources of everyone, and the suspension of all work has necessarily followed. The working classes at Quebec are becoming from day to day more of a burden upon men of means, and more upon us than upon others by their too frequent importunities. We cannot find any other expedient than to find work for them to take them out of misery.

"It spread on all sides throughout this Government (Province or Country), and in that of Three Rivers, where it is still prevalent. By the account which has been brought to me, about 900 persons have died from this disease in the environs of Montreal, as many men as women, girls and infants. At Quebec, 278. I have not yet had a list of the number in the environments of Quebec or Three Rivers, but the number may well be five or six hundred which this disease has carried off from the colony. It is true that the greater number are children, but there have been a great number of sick at Quebec, there being at one time more than 2,000, between those in the Hôtel Dieu and in private houses.

"This disease was commonly accompanied by a circumstance which was as annoying for the sick as for those who cared for them. This was the insupportable odour which accompanied the disease. I think it will not be necessary for me to go into details. I have seen several persons attacked with this disease in France, but it was far from being as virulent as it is in Canada."

A disease as dreadful in results as smallpox naturally incited the pioneers in medicine to try various methods of treatment. We find that Michel Sarrazin thought that in his discovery of the "*Sarracinea purpurea*" he had found a remedy; but this proved useless and it was not until 1765 that we find any attempt being made to introduce vaccination as a preventive. This was first employed in September 1768 by a Mr. Latham, surgeon to the King's or 8th Regiment of Foot, who announced to the inhabi-

tants of Quebec that he was prepared to carry on this method of prevention.

The people of New France were indeed sorely afflicted, for we find that plague made its appearance amongst them on no less than three different occasions (1711, 1718 and 1740). The historian of the Hôtel Dieu at Quebec relates that it was brought to this country by a ship hailing from Siam, where the plague was prevalent about that time. The disease was therefore known here as the "Disease of Siam." There are no records affording any description of the symptoms of those afflicted with the disease, hence any opinion as to the exact nature of the epidemic must be mere conjecture. The same authority, however, makes the following comments: "I have never seen so many sick in the hospital; the wards, garrets and outer buildings—all were filled, and we can hardly pass between the beds. All became as black as coal as soon as they were dead." This last remark may possibly justify the opinion that the disease was what is known to-day as the "Pestis Bubonica." History also relates the recurrence of other epidemics of a similar nature, extending over several periods (1710, 1746, 1750, 1756, 1757 and 1759). The death rate was evidently very high.

Owing undoubtedly to unhygienic conditions at this time existing, diseases other than those just mentioned, of an epidemic nature, were prevalent amongst the colonists. Chest affections were frequent, and the methods adopted to effect a cure were perhaps unique in the history of medicine in this country. In a letter from Vaudreuil and Bégon, dated at Quebec on the 6th of October, 1720, we learn that "there are a great number of persons in these parts who are attacked by chest diseases, and that the most efficacious remedy is asses' milk. As there are no animals of this kind in the colonies, we beg the Council to see that a male and a female are sent out next year by the ships of His Majesty."

The use of ice for the purpose of reducing the temperature in typhoid fever is thought by many to be of comparatively recent date; but about 1780, we learn from the memoirs of Philip de Gaspé, that when a child, he had been cured of a very severe attack of typhus, or typhoid fever, by the use of ice. "I showed



scarcely any signs of life for three days," he writes, "my death was expected each minute." So far, the patient had been treated by some one who possessed only a pretentious knowledge of medicine: as the condition of the patient became one of extreme gravity, Dr. Fred Oliva was sent for. He suggested that the child be dipped in a bath of iced water and then wrapped in hot flannels. "At the end of half an hour," writes de Gaspé, "there followed an abundant perspiration, which saved my life."

From 1800 to 1832 the country appears, as far as one is able to glean from available sources, to have been singularly free from diseases of an epidemic nature. Nevertheless, Asiatic Cholera, which had originated in India during the early part of the nineteenth century, gradually spread over extensive areas, until in 1832, it reached France and England. The English Government, as Christie says, "with characteristic providence and anxiety for the welfare of the province, had advised the Governor to make preparation against it in Canada, as in all probability it would, with the influx of immigrants hither, attend them. "Legislative provision accordingly was made to meet the impending evil, and sanitary precautions were adopted. Grosse Isle, thirty miles below the port of Quebec, was made a quarantine station, to which medical men were appointed, and temporary wooden buildings were erected upon it for hospitals, and the establishment put under military authority."

The spring of 1832 was rainy, cold and backward, and remarkable for the prevalence of heavy north easterly gales. "With the arrival," writes Christie, "of every vessel bringing immigrants, the pestilence was expected to make its appearance in Quebec. At length, on the 8th of June, the appalling news reached the city of its actual appearance at Grosse Isle, by the Garricks, from Dublin, with 133 passengers on board, fifty-nine having died of cholera during the passage. From Quebec, it bounded onward as it were by leaps in its direful progress up the St. Lawrence, breaking out on the 10th, at Montreal, with great violence." The severity of the epidemic may be gathered from the fact that from the 9th of June to the 2d of September no less than 2,215 people died from this alone in the city of Quebec. Since that time there

have occurred five outbreaks (1834, 1849, 1852 and 1854) of Asiatic Cholera in the Province of Quebec.

Let us now leave Quebec, with its illustrious past, and turn our attention to the city where we are now meeting, which was destined to become the centre of medical education. Montreal was, in 1816, a thriving town of 20,000 inhabitants, a distribution centre for immigration and the headquarters for the thriving trade of the North West Company.

The various charitable institutions of this city were for several years found inadequate to the increasing wants of the population, due to the increase of immigration. The termination of the war in 1815, brought to our shores vast numbers of persons who, through sickness on their passage or poverty on their arrival, were incapable of reaching their destination. Unable to procure either support or medical attendance from any funds of their own, they made an urgent appeal which prompted the benevolent inhabitants of our cities to devise means for the relief of these miserable exiles. An association of women in Montreal designated as the "Ladies' Benevolent Society" was formed expressly for the relief of the indigent immigrants; but more than this was needed. The sick required medical aid; and to attain this object, some medical men of the city agreed to give their assistance, provided a house could be obtained where the most necessitous cases of sick poor could be relieved. A small building of four apartments was then hired, and called the "House of Recovery." Small as it was, and inadequate to meet the numerous cases that needed relief, it became the germ of that noble institution which redounds so greatly to the benefit and honour of the city of Montreal—the Montreal General Hospital. This building was soon found totally inadequate to relieve all those who made request for assistance; and means were taken of raising a sum of money which when sufficient enabled them to rent a larger house in Craig street. The medical department was placed under the direction of four physicians, who attended monthly in rotation, one of them as house surgeon attending daily in cases of accident. On the 1st of May, 1819, such patients as were in the House of Recovery, together with the little property belonging to that establishment, were

removed into the new institution, which now assumed the title of the "Montreal General Hospital."

Considerable interest had been aroused in favour of this newly established charity, and to such a degree that in August, 1820, sufficient funds had been raised to enable them to buy a new site on which to erect a modern building. The site which was purchased was that which is now occupied by the hospital. On the 6th of June, 1821, the foundation stone was duly laid, and in May, 1822, it was opened for the reception of patients. The medical staff consisted of Doctors Caldwell, Robertson, Holmes, Leodel and Stephenson. Within ten years from the first foundation of the hospital the demands for admission exceeded its capacity, and an important addition was made to it, which was opened for the reception of patients in December, 1832. The hospital at this time contained nineteen wards and had a capacity of 160 beds. (*Hochelaga Depicta.*)

An institution attended by active and energetic physicians and supplied with abundant clinical material being now a fully established fact, the staff was naturally led to approach the vexed problem of medical education. The means by which it was eventually solved have been more thoroughly described by Dr. Maude Abbott in her history of the medical faculty of McGill University, and from this I shall venture to quote at some length:

"The formation of a medical school was the natural outcome of a general impulse in a rapidly growing city where a real need for medical teaching exists. As a result, therefore, we find that in 1822 lectures were publicly announced and given by some of the members of the staff of the Montreal General Hospital, notably Drs. Stephenson and Holmes. This continued for a year, when a meeting of the hospital medical staff was held on the 20th of October, 1823, to consider the expediency of establishing a medical school. Steps were immediately taken to organize a teaching staff and to utilize the hospital for clinical purposes, to give a course of lectures, and these were begun on the 15th of November, 1824, and delivered at the home of the institution, the first announcement of teaching reading as follows:

" 'Principles and Practice of Medicine,' Dr. Caldwell.

" 'Surgery, Anatomy and Physiology,' Dr. Stephenson.



“ ‘Midwifery and Diseases of Children,’ Dr. Robertson.

“ ‘Chemistry, Pharmacy and Materia Medica,’ Dr. Holmes.”

This organization was then known as the Montreal Medical Institution, and the first session of this body was held in the academic years 1824-5. Once established, the Montreal Medical Institution seems to have worked its way on quickly, winning good repute until 1829, which marks an epoch in its history, when it became “engrafted upon” McGill University as its medical faculty. This university, founded by the will of the Hon. James McGill, did not come into actual operation for many years, in spite of the need that was felt for such an educational institution among the English-speaking population. The bequest, which consisted of £10,000 and the estate of Burnside, was left in trust for a college to The Royal Institution for the Advancement of Learning, a body contemplated by a Provincial Act of 1801, but which was not incorporated and therefore not able to hold property until the year 1818. This circumstance, combined with an almost total lack of funds in the institution, delayed action. And delay was further occasioned by protracted litigation over the bequest. Indeed, had it not been for the far-seeing condition of the bequest, that the legacy should revert to certain persons unless a university were established within ten years of the testator's decease, it is more than probable that the project would have been indefinitely delayed.

In the year 1829 that portion of the lawsuit which referred to the bequest of the lauded estate of Burnside was decided in favour of the university, but the question of the £10,000 continued unsettled for many years longer, and no funds were at the disposal of the college. Yet it was felt that to fulfil the conditions of the will actual teaching must now be done in the university, and the purely nominal appointments of 1823 be refilled or supplemented.

At this time the eyes of The Royal Institution and of the friends of education were turned upon the Montreal Medical Institution, now an active teaching body of established reputation, and it was decided to bridge the difficulty by making this body the medical faculty of the university, its officers becoming professors or lecturers in their respective branches.

On June 29th, 1829, the first meeting of the Governors of

"Burnside University of McGill College" was called at Burnside House, which had just come into the hands of The Royal Institution under the will. This meeting had as its object the promulgation of the Charter and the Inauguration of the University. It was largely attended by representative citizens of all classes, and at this meeting the members of the Montreal Medical Institution who had been invited to be present were formally engrafted upon the University of McGill College as its Medical Faculty.

The Montreal Medical Institution, as it still continued to be called for some years, was now a faculty of an established university, capable of granting degrees. For some years the only active work done in the University was that in this faculty, and by its simply fulfilling the time limit of the will it may justly be said to have saved the University at least from much involved and tedious litigation, if indeed it did not preserve for it the bequest itself."

The improvement of medical education was even as early as this period (1837) a much discussed subject, and relevant to it is an extremely interesting report and suggestion made by Sir John Doratt, M. D., then Inspector-General of Hospitals, who was appointed by the British Government to investigate, report upon and advise regarding this question. In his report is suggested the establishment of a medical school in the city of Quebec in connection with Hotel Dieu and the Marine Hospitals. These hospitals, it was considered, would conjointly provide clinical beds to the number of three hundred. The selection of Quebec for the proposed medical school was due to various reasons. The population of the city was now between 30,000 and 40,000. As the principal port also for ocean shipping, it was at Quebec that the diseases continually brought by incoming vessels and the accidents from the process of loading and unloading in port were most in evidence. It was here, therefore, that the aid of medicine and surgery was most urgently required. It was felt also that the school would attract from the colonies of New Brunswick, Nova Scotia and Prince Edward Island the young men who had hitherto been compelled to go abroad for an opportunity of studying medical science.

Among the suggestions regarding the course of medicine to be

pursued was the proposed regulation that the whole period allotted for education at the medical school should be five years. The first three years were to be passed in strict attendance at the school of medicine, and the last two years in close attendance to duties at the hospital. It was further suggested that the clinics at the hospital should be given from 8 to 10 A. M. These suggestions are worthy of note, since it is only within the last year that both McGill and its sister university, Laval, have adopted five years as the period necessary for the prescribed course of study. The suggestion of early clinics is now also under consideration.

The year 1826, it is to be noted in closing, marked an era in the history of medicine of the Province of Quebec. It gave birth to the first attempt which had been made with a view to opening a free intercourse with the literary world, and to promoting the interest and cultivation of medical science within the limits of our country by the formation of the first medical society in Quebec. The president was Joseph Morrin. It was at the same time that the first medical journal in the Province appeared, with the following words on the title page: "*Journal de Medecine de Quebec, publié et redigé par Xavier Tessier.*"

The period of time allotted to the consideration of the history of medicine in our province has been covered, and it remains to me now to express with the greatest of pleasure my sincerest thanks to Dr. A. G. Doughty, C. M. G., Dominion Archivist, for the privilege accorded in having access to the valuable manuscripts and works in the Archives; also for his invaluable assistance and suggestions in the preparation of this paper, and his arrangement of the bibliography; to Dr. N. E. Dionne, Librarian of the Provincial Legislature of Quebec, for his kind assistance; to Mr. C. B. Gould, Librarian of the Peter Redpath Library, McGill University, for the privilege of consulting the valuable works of reference; to his able assistant, Miss Hepburn, for her untiring energy in carrying out and preparing the research work; and to Miss Charlton, Librarian of the Medical Faculty of McGill University, for her very kind assistance.

## CASE REPORTS.

BY THOMAS J. HARRIS, M.D.

## I.

A CASE OF SUB-GLOTTIC PAPILLOMA CURED BY  
LARYNGO-TRACHEAL FISSURE.

Sub-glottic tumors are among the most difficult to treat and demand at times all the patience and skill that the surgeon has at his command. The following case is reported, not because it is in any way unique, but because of the unusual complications which it presented in diagnosis and treatment and with the hope that the experience gained may prove of service to some one in a similar case.

Ralph D., age eleven, came under the writer's care in the Manhattan Eye, Ear and Throat Hospital last October through the courtesy of Dr. Charles A. Manson. The following history was obtained. He had suffered from measles and pneumonia, but otherwise had always been well. The family history was negative. The present trouble with his throat began in January, 1907, when his voice became hoarse and he noticed that he easily got out of breath. There was no pain in phonation and no expectoration of sputum or blood. The boy had been for several weeks attending the Tuberculosis Clinic of the Department of Health, where it was suspected that the condition might be tubercular because of a temperature which he was running. His health was, however, good and the examination of his chest negative.

Inspection by us showed a rather anaemic lad who spoke with considerable hoarseness and effort. A view of the throat was rendered difficult by an over-hanging epiglottis, as well as by the pronounced irritability, leading on the slightest manipulation to repeated vomiting. The cords were seen to be healthy. On phonation, a tumor was visible, filling the anterior commissure and at-

tached to the under surface of the right cord. This growth appeared to be solitary, red in color and not vascular. A number of attempts were made to remove it endo-laryngeally. On account of his extreme timidity, crying at the least examination, this was found impossible. On October 15 he was admitted to the hospital and an attempt was made to do this under ether through the Jackson's tube spatula. Although it was easy to expose the tumor to view and to engage it in the seizing forceps, it was found that they were too light to remove more than a small portion of it. This procedure had a favorable effect on the breathing and voice. Without, however waiting for suitable forceps, it was determined to perform a laryngo-fissure and remove the growth from without. Preliminary to this, tracheotomy was deemed advisable. This was performed under general anaesthesia on November 18. Before we were ready to open the trachea, the boy stopped breathing and it was necessary to stab him through the crico-thyroid membrane. On account of the excessive oozing, the removal of growth was delayed for a few days. A tracheotomy tube was introduced and the boy sent back to the ward. Marked reactionary swelling in the larynx took place and all laryngeal breathing was cut off. A week later Dr. Chevalier Jackson was kind enough to examine the larynx and trachea by the direct method. The growth was to be seen, but so surrounded by inflammatory swelling, involving both the vocal cords and the anterior wall of larynx and trachea, that he deemed it unwise to make any attempt either by the inner or outer route to extirpate it till the reaction had subsided. This course was followed and the lad kept under observation. The laryngeal swelling gradually went down and he was again able to breath per vias naturales. Much apprehension was felt lest a permanent cicatricial deposit should be left, seriously interfering with normal breathing. At the end of two months however the oedema had virtually disappeared and, what was more surprising, careful examination failed to show any trace of the tumor remaining. The landmarks in the larynx were still distorted and the cords showed much swelling. It was therefore felt advisable, after consultation with Dr. W. Kelly Simpson, to introduce an intubation tube and remove the tracheotomy tube.

This was done February 10, 1908, and excellent breathing secured. This continued for about ten hours, when suddenly he



began to find great difficulty in getting his breath. He was again readmitted to the hospital and the tracheotomy tube reintroduced. The intubation tube could not be seen in the larynx, and was discovered in the stool the next morning. He was again intubated, this time with a smaller tube. This tube gave equal comfort and remained in situ for four days. When I again saw him at the end of that time he was breathing perfectly, but the thread had been bitten off and there was no trace of the tube to be seen in the larynx, nor was it ever found. Inasmuch as the respiration was so good, we decided not to reintroduce the tube but merely to keep him in the hospital. This was done for two weeks, when he was allowed to go home. The voice, which was at first very hoarse, has continued to improve and when last seen, April 18, was clear and quite strong, considering the injury which the larynx had sustained. Laryngeal examination showed the cords assuming their normal color and outline. The boy had gained in weight and appeared to all intents perfectly well.

A review of the case suggests the following interesting points:

1. The misleading effect on the diagnosis of the two symptoms complained of, viz., hoarseness and dyspnoea, which taken in connection with fever caused a suspicion of tuberculosis. This was readily cleared up as soon as a laryngeal examination was made, which had apparently been omitted up to the time he came to us.

2. The second point that impressed itself on us was what is being so generally recognized at present, viz., the practicability and usefulness of the direct method for removal of laryngeal growths, especially those situated sub-glottically or in children. We are confident that the tumor in this case could easily have been removed at the time of the original examination with a proper Mosher forceps, and thus all the danger and annoyance of the subsequent external operations avoided. The experience in this case and in other similar cases which we have seen and read of has served to demonstrate to us that the brilliant work of Killian abroad and Jackson among others in this country in the direct inspection of the upper air passages is applicable not only to foreign bodies in the trachea and bronchi, but also to the removal of benign neoplasms in the larynx itself.

3. The third point that was brought to our attention was the danger in cases of laryngeal obstruction due to new growths, of interference with the respiration during the tracheotomy and the profound reaction which may follow the hasty opening of the wind-pipe thereby necessitated. What was the cause of the respiratory failure here, it is hard to say. It did not appear to be due to the anaesthesia. It may have been possibly due to the mass falling into the chink of the glottis. There did not seem to be any wounding of the cords at the time of the operation, but it is evident that this had occurred.

4. Still more interesting was the way that this oedema and what looked like adhesions entirely cleared up. To all that saw the case it seemed inevitable that a web would be the permanent result of the inflammation. What existed and was taken to be a perichondritis was chiefly oedema or otherwise it would not have disappeared as it did.

5. But most remarkable of all was the total disappearance of the tumor. Without any other treatment, solely under rest secured by means of the tracheotomy tube, every vestige vanished. This is of course no new observation. It however serves to confirm the value of this classical treatment for laryngeal papilloma, which this growth in all probability was.

6. Reference is to be made, too, to the loss of the intubation tube, an accident which has doubtless occurred many times, but which is none the less suggestive of dire consequences.

7. Finally, mention should be made of the surprisingly good voice which resulted. When seen at the height of the reaction, it did not seem possible that he could ever recover at the best more than a hoarse voice.

## II.

### A CASE OF BRANCHIO-GENETIC CYST.

The second case is reported merely for its extreme rarity and not because it possesses any particular clinical interest. Up to 1904, when Dr. C. G. Coakley reported before this association an extremely interesting case of branchial cyst of the throat, there was no record in the literature of such a condition having been



discovered in that locality. His case presented many symptoms of unusual interest which for a time obscured the diagnosis, and which are described at length in his paper. The present case can in no way be compared with this in any of its clinical aspects.

The history is as follows: I. N., aged 30, married, born in Russia, presented herself at the clinic of the Post-Graduate Hospital last January. She was the mother of three children. Family history negative. She denied any previous illness or accidents. She stated that she had no symptoms of nasal difficulty up to five months ago, when she began to be troubled with a feeling of discomfort in the naso-pharynx and dropping of mucus especially at night, when she felt, as she expressed it, "all stopped up."

Examination of the throat showed what appeared to be a mass of adenoids in the naso-pharynx. The nose was clear. A more careful examination by means of the finger gave the sensation of entering a cyst with thickened walls and filled with a tissue resembling an adenoid. The posterior wall was covered by a membrane measuring at least a quarter of an inch in diameter. No secretion of any kind was seen. The growth was removed by my colleague, Dr. Forbes, with the forceps and a specimen submitted to the pathologist for examination.

Dr. Erdwurm made the following report: "Sections show tissue to be made up mostly of lymphadenoid structure; one surface shows the normal stratified squamous epithelium of the pharynx. There are a few mucous glands, and scattered through the section, in several places, are tubules lined with ciliated columnar epithelium. Beneath this epithelium the tissue has the appearance of embryonal connective tissue, and from these characteristics the tissue removed is evidently the remains of a branchio-genetic cleft."

The specimen was also kindly examined by Dr. Jonathan Wright, who confirmed the microscopic diagnosis. Dr. Wright made the following report: 1. The surface epithelium here is everywhere metaplastic, *i. e.*, the columnar walls on the surface have everywhere become of the pavement type. Except where the surface is turned into a protected depression, where these depressions or sulci are cut across the epithelial cells are seen to be not only columnar but ciliated. This is the more marked because some of the metaplasia has gone to the point of the flat

epithelium assuming intercellular bridges or presenting the appearance of so-called stickle cells.

2. There are not the usually well marked germ centers here that are characteristic of lymphoid hypertrophy.

3. There are patches of spindle-celled fibrous tissue not predominant enough to warrant suspicion of sarcoma, but more prominent than usual.

4. In a few areas of chronic inflammation there are atypical giant cells, apparently arising from the fusion of the cytoplasm of some of the large mononuclear lymphocytes, of which in these regions there are many.

The clinical history of the case subsequent to the operation is uninteresting. All symptoms of obstruction disappeared and the patient was discharged.

## A RARE HYPERTROPHIC FORM OF SYPHILIS INVOLVING THE FAUCES AND EPIGLOTTIS.

BY WALTER F. CHAPPELL, M.D.

Carl Jacobson, age fourteen years, visited the Manhattan Eye, Ear and Throat Hospital in 1903 and had his tonsils and adenoids removed. He was kept in the hospital one day and nothing unusual was observed in his upper air passages. Nearly a year later he had a severe fall, which lacerated his scalp in the occipital region to the extent of about eight inches. He sustained some other injuries, the nature and extent of which I have not been able to discover, but he remained in the Harlem Hospital nearly eighteen months. Six months after he left the hospital he developed a leucoma on the left eye, which lasted about five months. Eight months after the left eye was restored the right eye became involved in the same way and he is still under treatment for it.

In February, 1907, he again came under my observation at the Manhattan Eye, Ear and Throat Hospital. His mother then stated that for about three months he had snored very loudly while asleep and that his voice had assumed a guttural tone, and that he was unable to raise it to any high pitch, and could not sing at all. During the three or four weeks preceding their visit to the hospital she noticed that when he walked about the house his breathing was labored. The boy also stated that at school his teachers complained of not being able to understand his reading or, in fact, his ordinary conversation. He also stated that he could not play with the other boys, as something seemed to choke him. These choking or strangling spells had recently become very frequent at night, so much so that some one slept in the room with him, fearing he would suffocate. The boy looked well nourished and seemed to be in good physical condition.

The anterior and posterior pillars of the pharynx were much hypertrophied and a great deal of hypertrophic tissue occupied the position of the tonsils. This condition seemed uniform on both sides. The uvula was about one and one-fourth inches long and nearly an inch wide, and seemed to almost completely fill the

centre of the pharyngeal cavity, and no doubt accounted in part for the respiratory symptoms and also the night choking spells.

The epiglottis was also enormously hypertrophied and seemed to have lost all its flexibility. Often while eating he complained



of food or liquid entering the larynx. The arytenoid cartilages also shared this general hypertrophy and seemed exactly similar in appearance. I could not discover that any other part of the

larynx was implicated. The mucous membrane covering these hypertrophied parts was smooth and had a grayish, shining appearance. The water color I present is a good representation of the hypertrophy and color of the mucous membrane. The family and personal histories were negative, and no information was obtainable from the family which could account for the condition described. A strong solution of adrenalin was sprayed on the surface of the hypertrophied membrane, but seemed to have no effect beyond blanching the color of the membrane to a slight degree. The obstruction to respiration was so great at times that it was thought best to remove a portion of the uvula. This was done under cocaine and, although the hemorrhage following the operation was considerable, it ceased after a few hours and did not recur. The wound healed in about ten days. The removal of the uvula immediately stopped the night attacks of difficult respiration and also greatly relieved his breathing during the day. His voice was also much improved.

The piece of uvula removed was given to Dr. Jonathan Wright, who sent me the following report:

"Horizontal sections through the amputated uvula show the investing epithelium to be somewhat, though not markedly, thickened. This covers an extreme fibrous hyperplasia of the stroma. Newly formed blood vessels channel it vertically in large numbers. The lumina of many of these vessels are occluded by the marked hyperplasia of the endothelial cells of the inner coat. These in many places form a plug which the hardening and fixing process (formalin and alcohol) have caused to shrink from the rigid walls. In many places fibrous connective tissue has invaded these endothelial plugs and the lumina of the blood vessels are in this process being converted into the connective tissue of the stroma. In some of the arteriole walls there is a great increase of the fibrous coat. Around some there is a wide fringe of round celled infiltration. In places throughout this low grade fibrous tissue and especially around the blood vessels there are droplets of degenerated, structureless material. Iodin, iodine and sulphuric acid, methyl violet and the Giemsa stain fail to elicit any evidence of amyloid degeneration. With the Van Gieson stain the hyaloid areas in the fibrosis stand out prominently, owing to their affinity for fuchsin. The sections include some of the muscle tissue and some few fat cells. There are no pseudo-eosinophil or eosinophil cells. In view of the vascular changes and of the general fibrosis, there is little doubt in my mind that

this pathological condition is dependent upon syphilitic infection, probably hereditary."

The boy was put on the mixed treatment and certainly improved for a time. As his digestion was poor, we were not able to continue it, and I substituted Donovan's solution of arsenic and mercury, which benefited him considerably, but there is still a good deal of hypertrophy in the pharynx and larynx and the parts already described.

This case is quite unique in my experience. An article entitled, "*Lokales Amyloid im gesammten Respirationstrakt*," von Dr. Felix von Werdt, and also some cases in "*Beiträge zur pathologischen Anatomie* (Ziegler), Band 43 Heft 2," and a few by Sir Felix Semon, describe cases of hypertrophy in the pharynx and larynx, but they were all amyloid degeneration. After reading these cases, I thought mine might be of a similar nature, although the mucous membrane and hypertrophied parts lacked the distinct yellowish appearance. I think Dr. Wright's report and the partial response to anti-syphilitic treatment is fair proof that the case I have described is of a syphilitic nature and probably hereditary.



*Candidate's Thesis.*SOME NEGLECTED POINTS IN THE ANATOMY OF  
THE PHARYNX.

BY B. ALEX. RANDALL, M.D.

The clinical, as well as the systematic, anatomy of the pharynx has been well studied and is doubtless receiving continued consideration; yet certain points have seemed imperfectly recognized or even often misstated, and thus to merit reiterated emphasis. With little expectation, therefore, of bringing new observations to notice, I beg leave to emphasize some which seem neglected, although having practical bearing. And while conceding the great importance of the nose in respect to its anatomy and physiology, we must seek in the pharynx behind it many etiological elements of moment to the organism and to the surgeon who has to treat the lesions both localized here and secondarily produced elsewhere. Not only is this true of the ear, with its eustachian extension to the pharynx, but all other parts of the upper air passages depend in marked degree upon healthy conditions of structure and functions in the pharynx.

The systematic anatomy of the pharynx is rather intricate and its difficult dissection is rarely worked out by the student, who is content to accept its explicit and sometimes *too* diagrammatic presentation in the treatises. Like all of the anatomical fields which have been minutely worked by a few enthusiasts, it has presented numerous variations, and each investigator has been apt to set forth his ideal chosen as the normal from among these, rather than to give a composite picture that shall more truly average the findings. Polemic clashes as to these ideals have served rather to obscure and narrow the statements of the typical relations than to broaden the views entertained; and Luschka or some other authority has been rather blindly followed in disputed matters, especially by the clinicians, who have rarely made personal investigations.



Yet it is the clinical anatomy and physiology that are of most use to us in practice, and we are constantly seeing in our daily work proof that this or that contested point is or is not true for the case in hand. Let the observation be close and accurate and it may be more important than that of the anatomist, however careful and exhaustive, who depends wholly or largely upon the study of dead tissues, and describes the action of muscles more on the basis of the supposed mechanics of their attachments and insertions than upon observation of their activities.

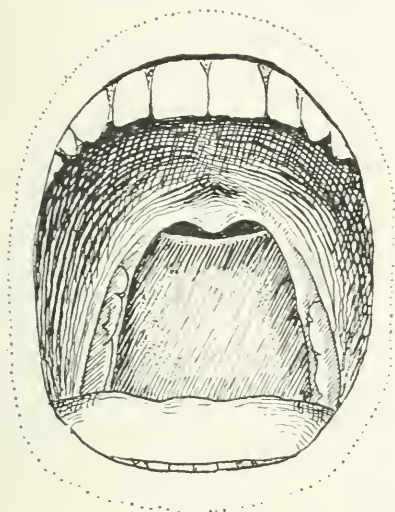


Fig. 1.

The excessively raised palate reveals the projecting Passavant's ridge of the back wall of the pharynx.

Some twenty-five years ago I was struck by the appearance of a pharynx in which the soft palate was excessively lifted in gagging and failed to shut off the naso-pharynx, thus revealing a sharp-edged prominence of the back wall which it had failed to meet in completing the closure. Above this prominence the back of the pharynx looked unduly deep in the shadow with the shelf-like ridge below it. It was some years later, after repeated similar observations and some study of cleft palate cases, that I learned that this was "Passavant's cushion or prominence," and not the anomalous or unobserved structure that most of the text-

books implied by their silence about it. Even to-day few text-books mention it, and none lay upon it the stress which its clinical importance at times merits. Yet, really one cannot attempt to paint the pharynx-vault without having to consider this prominence; and I believe its habitual recognition will result in better work in the treatment of this region. Many an attempt to paint or probe the pharyngeal tonsil fails or gives a wrong impression because of the projection of the Passavant ledge below it.

The usual description of the uppermost fibres of the superior constrictor as passing *up* for insertion upon the pharyngeal tubercle of the occipital bone, implies motion of the pharynx up to this point; but I believe it will usually be found that this insertion is aponeurotic and long, so that above the level of the palate there is no mobility of the pharynx tissues; hence the collection of secretions at this still point, detained also by the shelf-like fold which comes forward at each swallowing motion as well as in speech. I strongly contend that "Thornwaldt's Disease" is generally secondary to such collections and their irritation of the underlying wall, instead of being a more primary infection of the hypothetical "bursa" or diverticulum of Luschka. At least we must recognize the part played by the upper edge of the superior constrictor in arresting secretions here so as to form crusts, which can hardly be detached until large enough to spread upon the surface of the constrictor and to be loosened by its movements.

Study of Passavant's paper in Virchow's Archiv, Vol. XLVI., 1869, which is an amplification of his earlier "Festschrift," shows that he was led to his investigation by noting cases of cleft-palate in which the ridge served a very useful purpose in partly closing the gap and prevented constant regurgitation of food into the nose. He therefore dissected carefully and well presented the structures of this region, answering the criticisms which Luschka had made on his earlier study. But his paper has probably obtained far less notice than Luschka's beautiful monograph, in which his views are unfavorably judged; and later anatomists, like Prennant, in Poirier's work, have seemed to follow Luschka in copying his illustrations, although close reading shows that they support many of Passavant's contentions, as, indeed, do some of Luschka's pictures. Without entering much

into the controversy, I submit some of the illustrations of each author as among the best in existence, and add one or two sketches and photographs from preparations of my own. From these it would clearly appear that there is a sharp-cut upper edge of the superior constrictor of the pharynx (the pterygo-pharyngeus) which must tend to form such a shelf-like fold both at the back and sides of the pharynx as Passavant has demonstrated, contributing notably to the closure of the pharynx in speech and in deglutition. This band must be below the level of the eustachian tubes, the levator palati muscles and the Rosenmueller fossae, and in the middle line posteriorly it is probably little, if at all, above the level of the hamulus, although one of Merkel's cuts shows it passing high into the vault, as his next cut does not. Luschka's side view of these structures shows little looping up of the superior constrictor to a high attachment; but the posterior view indicates such insertion, which is probably only aponeurotic as a rule.

One can hardly ever observe the movements of the throat in swallowing without noting a decided narrowing of the fauces; and over-action can bring the half-arches or tonsils into contact. Much less than this is commonly seen in speech, and Passavant is probably right in claiming that the quick, frequent and easy movements of palate-closure in speech enlist only the superior constrictor and the levator palati; the other muscles aid them in swallowing, but soon tire if forced by any but the physiological stimulus of food. He says: "There are, to my thinking, two different forms of palate-closure—the first serves speech, and is mainly achieved by action of the levator and superior constrictor, or, if you will, of that portion of the muscle which as pterygo-pharyngeus springs from the hamulus; a second form is carried out in swallowing, &c. In this the previous closure is reinforced by the contraction of the palatino-pharyngeus, which can advance the lateral walls of the pharynx to contact in the middle line."

The photographs, which I have made from my sections of the frozen head show marked asymmetry through accidental distortion of the parts previous to freezing, and the backward gravitation of the tongue has narrowed the pharynx at the palate-level until it is hardly more than the closed slit we assume it to become when the velum is lifted. I therefore submit a semi-schematic

sketch from a tracing on glass after moulding of the tissues to more normal configuration, which will be found fully to conform to Passavant's section and confirms his diagram as to the course

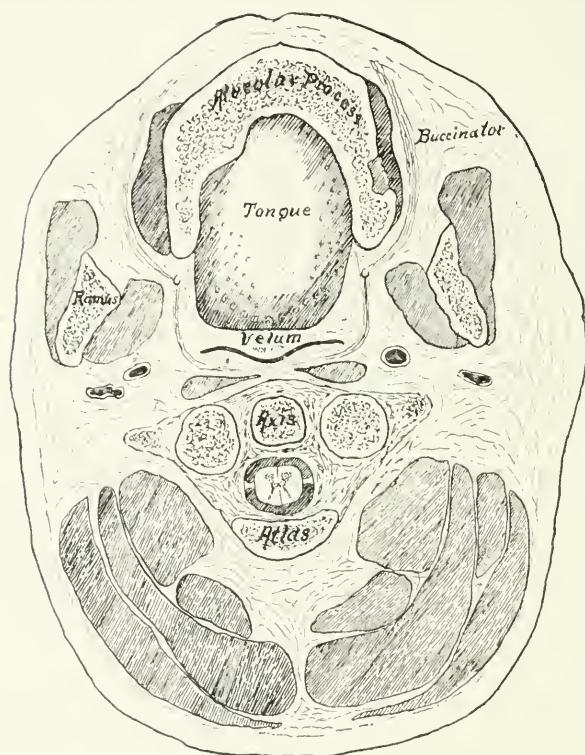


Fig. 2.

Semi-schematic sketch of a horizontal section through the Atlas, showing the horseshoe course of the superior constrictor.

of the constrictor fibres and shows that they must usually act to narrow the pharynx and advance its back wall as he has claimed.

## THE TREATMENT OF INOPERABLE TONSILLAR HYPERTROPHY BY INTERSTITIAL INJECTIONS OF SILVER NITRATE.

BY JOSEPH L. GOODALE, M.D.

During the past six months, three cases of haemophilia, associated with infectious arthritis, have been referred to me for examination, of the nose and throat. Considerable tonsillar hypertrophy was present in all of them, in association with dilatation of the crypts and retention of cheesy detritus. Our past experience in infectious arthritis has shown that improvement of the joint lesions frequently follows excision of such foci of infection, and the question here arose in what manner we could reach the desired result without involving the patient in risk from hemorrhage. Cauterization of the tonsils by the galvano-cautery or by chromic acid will, it is true, cause a diminution in the size of the organs, but the employment of these agents is followed by irregular contraction, with narrowing of the orifices of the crypts out of proportion to the diminution in the size of the lumen near its base. Such a contraction would manifestly be insufficient to prevent the retention of detritus in the crypts, and might be supposed even to increase the tendency to the absorption of septic material. Silver nitrate, in promoting sclerosis and contraction of hypertrophied tissue in other parts of the nose and throat, has been employed for many years, and it seemed to me worth while to make a test of its effect in the conditions in question. In the three cases, silver nitrate was employed, both by introduction into the crypts of the tonsils, and by injection into the substance of the organs themselves, by means of a hypodermic needle. The clinical history of the cases is, in brief, as follows:

E. H. M., a young man, twenty-eight years of age, was referred to me November 27, 1907, by Dr. Goldthwait, with a history of recurrent arthritis since three years of age. On several occasions he had had severe hemorrhages from accidental cuts, which had been arrested with difficulty. A cut of the face from the razor



would bleed for a number of hours, without evidence of spontaneous coagulation of blood. Silver nitrate was injected in 4% solution in January, 1908, into the tonsils, at intervals of four days, with the result of causing moderate reaction. A rapid symmetrical diminution in the size of the tonsils followed, and on April 8, after ten injections of silver, the tonsils were about one-fourth their original size, and showed no retention of detritus in the crypts. The arthritis improved simultaneously and his condition at the time of my last examination was considered satisfactory.

R. F. Y., nineteen, was referred to me in February by Dr. Goldthwait for infectious arthritis of six years' duration. The history of haemophilia was here marked, as slight incisions of the skin were followed by prolonged and obstinate bleeding. A few injections of silver nitrate in 4% solution were made into the tonsils, and the treatment has since then been carried out by his physician, Dr. D. A. Campbell, of Halifax, N. S., who has written me that the tonsils have been materially reduced in size, while his rheumatic symptoms are less.

A. J. R. was referred to me in March by Dr. E. G. Brackett with a history of recurrent arthritis. The tonsils were markedly enlarged on both sides, with considerable fetid detritus in the crypts. About ten years previously an attempt had been made to remove the tonsils. Severe bleeding followed, which was only controlled by firm pressure applied to the wound and maintained in place for several hours. Bleeding from the skin lasts for several hours after slight wounds. Eight injections of silver nitrate have been made at intervals of five to eight days into the tonsils in solutions ranging from 4% to 8%. The amount of reaction has varied from slight to severe pain, lasting for several days, depending upon the strength of the solution. A progressive diminution in the size of the tonsils has taken place.

For making the injections I have found most serviceable a hypodermic syringe, to which a handle is attached, enabling it to be held readily and, at the same time, giving sufficient length for reaching the tonsil. A bent hypodermic needle of the kind employed by dentists for injections into the gums is better than a straight one, as it passes more easily around the anterior pillar.

This method of treatment is offered for your consideration at this time, chiefly for the reason that its effect is theoretically in harmony with the normal process of retrograde metamorphosis of the organ, namely, a sclerosis proceeding from the base of the organ to the periphery and leading to a symmetrical diminution

in the size of the crypts. The clinical material is so far naturally too meagre to justify me in attempting to draw conclusions as to its efficiency in relieving the conditions of arthritis, but I am encouraged to give it a further trial in chronic inflammations of the tonsils where excision is contraindicated.



THE QUESTION OF RHEUMATISM ORIGINATING  
THROUGH TONSILLAR INFECTION.

BY GEORGE B. HOPE, M.D.

It is certainly not because the subject is rare, as, in fact, it has developed into inordinate proportions, with conclusions so opposed and with outcomes so little, that it is fascinating to travel again over paths so varied and divergent as to obscure any useful unanimity of opinion in a matter that yet remains pure speculative theory.

The term, "the tonsil, the portal of entry of rheumatic infection," is a form of speech that has become almost a platitude in the course of medical literature to the degree that it is accepted and dispensed with the regularity of an acquired habit and, no doubt, frequently without stopping to inquire how far it agrees with personal experiences and recurring clinical material. This remark is illustrated in a conversation with a physician whose wide practice entitled him to have among his patients many cases of the rheumatic character. To the request for his opinion as to the active part played by the tonsil in its production, the answer came quickly that "of course it was so considered." To the further question concerning subjects often suffering from rheumatic seizures—reasoning backwards—if there were equal examples of preceding tonsillitis, there was a hesitating and doubtful reply. The habit of the tonsil and its function has always been a fertile debating ground, and the wildest theories have not been without popular credence. It is yet within the memory that the supposititious function dominating over procreation was so strongly fixed within the minds of the multitude and, sadly, not alone among the laity, as to have been a barrier against the removal of these parts when diseased. Truly the wisdom of the ancients—"omne ignotum pro magnifico." It is no wonder, then, if in more modern days there is the lingering disposition to attribute occult causative principles to these organs when coincident dis-

ability is manifest. While the pathologist still lingers with his report and the clinician doubts and doubles with the rheumatic principle, the time has not arrived when the scientist may boldly announce undisputed cause and effect and settle beyond a peradventure this much-discussed subject. However unfortunate this may be, there are nevertheless many clinical details that should go far to disabuse what appear to be traditional views and traditional fallacies.

Although it is impossible to disregard the testimony of so many who find the rheumatic sequence of tonsillitis almost inseparable, there remain a sufficient number whose equal experiences have led them to question the undemonstrated fact, and still others who are ready to deny in unmistakable terms any logical relation beyond that of the occasional concurrence of two distinct diseases that are somewhat possessed of a common underlying principle, but without interdependence upon a mutual infectious causation. Whatever histological role the normal tonsil is designed to fill, in disease it certainly is not possessed of the active function to disseminate bacterial infection to the degree commonly conceived. Many careful investigations from independent sources lead frankly to this conclusion. Notwithstanding bacteria are found in abundance in its crypts, and seem to possess a field unusually fitted for culture, they are rapidly lost in number as the deeper structures are reached and by so much more rarely are they allowed to pass beyond the capsule into the surrounding lymph-nodes to develop into the processes of active inflammation. For the most part, clinically, a local limitation is the even course of an expected regularity. In the acute stage resolving into abscess formation it is especially to the individual who is marked for recurrent attacks according to the common rule of the diminished resistance and faulty tissue involved. By the same experience myositis of proximate parts running parallel with a septic history is so exceptional as to have nearly escaped a bare mention in the text-books, and small retention abscesses may exist for months without attracting attention either of patient or physician. It is well known to surgeons that malignant disease of the tonsils is especially slow in progressing to neighboring lymphatics and affords a more hopeful field for operation. Among the exan-

themata, as scarlet fever and measles, that show a predilection for an early manifestation in the throat, the tonsil does not ordinarily present concurring signs of their presence. La grippe, the most abrupt and active among the accepted infectious diseases that dominate over the mucous membranes, has but little influence upon the tonsils, either as a starting point or in subsequent evolution of any of the forms of angina. It is true that diphtheria is most commonly found in this situation, but if limited to this area it follows a course usually so uneventful as to require but few measures for its treatment, and no great apprehension for the results, while in contrast, nasal diphtheria, in the days past, was regarded as among the most fatal within the range of the infections.

"Rheumatism," up to the present time, receives an interpretation of so vague a character as to be broadly generic, and includes a range of symptoms in childhood and adult life that spreads from skin lesions to rapid and fatal chorea, in the one, and muscular, joint and cardiac degeneration, in the other. To those employing the term "diasthesis" it is an inheritance that may be revived after generations of remote ancestry. Surely no other distinctive disease having accredited acute infectious methods can claim so wide an exhibition and so deep a foundation. It is due to such empiric classification that there must be innumerable errors that creep into the published statistical report of cases that are made to tally in such large numbers with acute tonsillar attacks, and serves to strengthen the belief in a theory that still depends upon this sole evidence as its most conclusive demonstration. No other sufficient working basis has appeared to strengthen the hypothesis of an exciting infection, and every experimental test along these lines has met with negative results. After all has been said, the sum of the subject has been given as follows:

"1. It is undoubted that a certain number of cases of acute rheumatism are preceded by angina in a proportion varying from 30 to 80 per cent.

"2. Both rheumatism and angina have many etiological points in common: season of the year, cold, wet, fatigue, depression, vitiated air, etc.

"3. The connection between angina and rheumatism, although undoubted in a number of cases, is not clearly established.

"4. The tonsil may be the port of entry of the rheumatic virus, and this even though the naked-eye appearance of the throat gives no indication of its being affected.

"5. The particular affection of the throat which is associated with rheumatism is not yet established; apparently it is not peritonsillar abscess (quinsy).

"6. Peritonsillar infection does not appear to be arrested by the administration of anti-rheumatic remedies. Many cases of lacunar tonsillitis, on the contrary, are considerably benefitted by the administration of salicin or sodium salicylate. That this action proves the rheumatic nature of the disease cannot yet be accepted.

"7. The question requires further research in two directions: one in differentiating the various forms of angina and settling the one which is associated with rheumatism; the other in further research to discover the true nature of rheumatism."

Assuming that the foregoing is a fair statement, it must appear that the theory of a tonsillar infection rests entirely upon pure inference, rather than upon approximate facts, and that it must lack a convincing force until it has gathered a higher order of etiological and clinical demonstration. It is a familiar feature with published statistical notes to find them so rearranged as to comply with a convenient conclusion of identity of causation. The author, in one such selected review, takes "275 cases of articular rheumatism in which there was absolute record of preceding tonsillitis, and draws the least period of separation between the two diseases to six days and the greatest sixty days." In order to satisfy a more proximate term of the infection, "the longer limits are assumed to be coincidences and are eliminated, and so brings the extremes within three (?) and thirty days, and an average of twenty-four days." In the same category is the wide variation, according to the personal standard and experience, of accredited rheumatism following tonsillitis, but which must be relatively sufficiently large to establish a guide for causation. Furthermore, in the light of a natural reasoning, is the questionable statement from high authority: "Finally it is worthy of note that anti-

rheumatic remedies are our best agents with which to combat tonsillitis."

For many years back it has been the custom to examine into the history of cases of tonsillitis, antedating and as far as possible subsequent to their presence as patients at the Metropolitan Throat Hospital. With a curious unanimity the responses have shown a practical lack of coincidence, proximate or remote, of a rheumatic sequence. All forms of tonsillitis, follicular, simple interstitial, peritonsillitis so often recurring with the expected regularity and extending over considerable periods, have forced the same conclusion. A clinic of this sort embraces every age, sex, occupation, and should furnish the necessary opportunity to fix upon the evidence that could hardly be misleading in this matter, and it is certainly surprising to find it stands so much alone against the general experience. For instance, there have been the examples of successive anginas without intermediate rheumatism, but when followed by tonsillar excision and several years of immunity comes a succeeding rheumatism. In others, the presence of rheumatism of the articular type, but never the slightest evidence of tonsillitis. Again, as in a recent subject (German, male, married, twenty-seven years), who when fourteen and for three years suffered periodically from acute rheumatic fever that disappeared on reaching England and, more recently, this country. Following a clear interval of four years there began frequent attacks of interstitial tonsillitis that at last became peritonsillar, but without reversion to his earlier rheumatism. These varying conditions are mentioned with no other reason than to demonstrate the range of instances inclusive with all the suggested vagaries of infection through the portals of the tonsils and running a typical course without fulfilling such a morbid destiny.

If the treatment carries any weight under the implied identity of the disease in aborting or mitigating a severity of tonsillitis, it is certainly not the practice of making use of any of the so-called anti-rheumatic remedies, and in a personal experience never is the smallest grain of a salicylate administered, and yet the course and consequence appear to adjust themselves to an equal if not diminished time period that agrees with any selective treatment.

These facts that are positive, unmistakable and are abundantly familiar to our working staff are confidently asserted, notwithstanding all evidence from other sources.

It is well worth the while to quote from a recent paper by Jos. E. Winters on "The Etiology of Rheumatism" words that are unusual for their earnestness as fixed in a belief: "Logical analysis quashes the preposterous and monstrous assumption that the tonsils are the portal of entry for rheumatic infection. Every observant physician is constrained to concede that faults of diet lie at the root of tonsillitis. Tonsillectomy for this condition is as illogical and unscientific as rubbing the joints for the cure and prevention of rheumatic arthritis. The incidence of rheumatic throat is neither lessened nor mitigated by complete extirpation of the tonsils. Hypertrophied and indurated tonsils must be removed. This, however, is wholly independent of their having causative relation to rheumatism. It is unbelievable, unthinkable, that an hypothesis so superficial, so pregnant with harm should find lodgment with a profession of such grave responsibilities."



CASE REPORT: LARYNGEAL STENOSIS IN THE  
ADULT, SUCCESSFULLY TREATED BY INTUBA-  
TION; CONTINUOUS WEARING OF TUBE  
FOR FOUR YEARS.

BY WILLIAM K. SIMPSON, M.D., NEW YORK.

The patient, a female, married, aged thirty-two, presented herself to me in the early part of March, 1903, to be relieved from wearing a tracheotomy tube which had been inserted some five months previously on account of urgent laryngeal dyspnoea. She gave the following history as bearing on the case, dating from the above time:

*Family History*—Negative, habits good.

*Previous*—Measles, mumps and scarletina in early childhood. Diphtheria when eight years old; it was rather a severe case and there is said to have been some trouble with throat at the time; exact nature not known. Typhoid fever five years ago, which was complicated by erysipelas (facial). No rheumatism, no renal, cardiac or pulmonary symptoms. Patient is said to have always had more or less trouble with her throat during childhood. At eight years of age (not associated with her diphtheria, she thinks) she had a great deal of difficulty in breathing, and some applications were made to her throat which gave relief. Otherwise well till present; has had one miscarriage.

*Present Illness*—Began about October 1st last, five months ago, as result of a severe cold, when she had spasmodic attacks of dyspnoea and cough with hoarseness, usually coming on at night and lasting variable periods, but usually some hours. These increased in severity until finally she went to Roosevelt Hospital, where tracheotomy was performed. On October 15, 1902 (two weeks after onset of symptoms) two attempts were made to remove tube, but it had to be reinserted after three hours and one-half hour respectively. After five weeks she went home with the tracheotomy tube in place and has worn it constantly till the present time, March 1, 1903, from which date the following history begins:

She was very desirous of dispensing with the tracheotomy tube, as its constant irritation, causing incessant coughing and excessive secretion, had caused rapid loss of flesh and had



reduced her general physical condition to a very low ebb. Examination of her pharynx at this time revealed quite a decided loss of tissue from old ulceration on the right free border of the soft palate extending up toward the uvula. This was a very important point, bearing on the cause of her laryngeal stenosis, as the ulceration was strongly suspicious of being of a specific origin, but careful questioning could not elicit any direct specific history.

She was placed, however, on anti-specific treatment at once, hoping that it might have an effect on the laryngeal condition, but she did not bear the iodides well and the treatment was discontinued for a while, but was resumed at various times during the subsequent progress of the case, to be discontinued for the same reason. It is difficult to say just how much the iodide of potassium aided in any improvement of her laryngeal condition.

Examination of her larynx showed it to be practically entirely closed, all the normal landmarks were obliterated; the superior portion, including the arytenoids, being incorporated in a more or less uniform bilateral swelling of a somewhat dense character, leaving a mere slit in the center, through which a fine probe could be passed so as to touch the metal tracheotomy tube below; there was no apparent loss of tissue and practically no motion of the parts.

There was a small goitre present, but not sufficient to produce any external pressure.

On March 28, 1903, under chloroform anaesthesia, with the assistance of Dr. John Rogers, of New York, I passed a medium-sized adult hard rubber intubation tube, using considerable force, until the tracheotomy tube was reached, then the tracheotomy tube was withdrawn and the intubation tube continued below the tracheotomy opening. Before intubating, dilatation from below upward was made into the larynx with steel sounds through the tracheal opening.

The intubation tube was retained with comparative comfort for four weeks, when it was expelled and had to be replaced at the end of seven hours owing to rapidly increasing suffocation. The tube was retained this time till May 17, 1903, when it was coughed out, to be replaced May 24 (one week) on account of severe dyspnoea.

June 21, 1903. Tube was expelled and patient breathed well until June 24, when it was necessary to replace it again owing to severe attack of difficult breathing. During this interval an opportunity was afforded for examination of the larynx.

There was some motion of arytenoids, especially of the left. The inter-cordal space very narrow, the vocal cords, or what appeared to be vocal cords, were irregular in shape with a flabby, fleshy appearance, there was no motion whatever either in phonation or respiration; the right cord was more irregular in outline than the left, as if more pressure had been exerted on it; the color of larynx was generally very red. At the anterior commissure or just below it on the tracheal wall there was a small whitish slough which was forced up in the larynx on expiration, giving a sensation to the patient as if something were loose or flapping and causing her considerable annoyance in coughing and breathing. Various examinations showed but little change in the appearance of the larynx, excepting that the inter-cordal space seemed to become a little wider.

It was very evident that the intubation tube had exerted considerable pressure absorbtion.

The patient was very apprehensive and nervous about being without the tube, which I think might have been a factor in bringing on the suffocative attacks at this time.

The tracheal fistula had been kept open since the beginning, but it was now decided to close it, which was done under chloroform on July 1, 1903, by Dr. Forbes Hawkes at the Presbyterian Hospital, the edges being excised and brought together by sutures; this is the first instance of which I know of a patient going under a general anaesthetic, breathing entirely through an intubation tube; the operation was performed apparently as well as if she had been breathing through a normal larynx.

The operation resulted in a permanent closure of the tracheal wound.

The intubation tube was worn from June 24, 1903 till May 23, 1905, when it was coughed out, but was replaced in a few hours, as the patient did not wish to take any chances in being without it.

During this long period the patient was perfectly comfortable

as far as her breathing was concerned, and did her ordinary work, and traveled considerable distances throughout the country. Occasionally during attacks of coughing the tube would become partially expelled, but she had learned the art of pushing the tube back in place with her finger and exerting pressure, until the attack would cease. She could eat and drink with comparative comfort and her general condition became much improved, it being in marked contrast to the period in which she was wearing the tracheotomy tube. She often expressed herself that it would have been impossible to have lived under the severe physical strain of the tracheal cannula. She was often asked to have the intubation tube removed, but refused, stating that she was perfectly satisfied with her condition. However, on April 23, 1907, as the tube was becoming somewhat foul from secretion, and becoming convinced of the necessity of trying to do without it, she consented to its removal, which was done, and up to the present time, May, 1908, there has been no occasion for its reintroduction.

The condition of the tube was as follows:

Some odor, owing to adherent secretion on its superior exposed portion, some small areas of very superficial erosions on external surface, principally on the posterior surface just above the swell, indicating the points of greatest pressure. Internally the tube was coated with fine calcareous deposits evenly disseminated over its entire surface.

Immediate examination of the larynx showed a pretty fair glottic space, larger than at the time of last removal. The mucous membrane was very red in color, but no ulcerations; the motion of the larynx was good, excepting the left side which did not move as well as the right, the immobility seeming to be limited to dense tissue about the left arytenoid; both cordal areas seemed to move well, there was considerable general superficial swelling of the mucous membrane, breathing good, voice aphonic. The patient was somewhat apprehensive as to her ability to breathe without the tube; this wore away as soon as she regained her confidence. The breathing has continued to improve and is normal in character. She has also gained decidedly in flesh and strength, and considers herself as well as ever.

Her voice, though not normal, is variable as to strength and quality: at times being excellent, but becoming hoarse when taking cold, and in damp weather. Examination of larynx at the present time, May, 1908, one year and over, since the final removal of the tube, shows the following:

*Color.*—Normal, with tendency to paleness.

*Motion.*—Somewhat restricted, due mostly to induration about the left crico-arytenoid region, the right side moving with much less restriction.

*Right Vocal Cord.*—Is fairly normal in contour, color and motion.

*Left Vocal Cord.*—Seems to have been more or less absorbed.

*Right Arytenoid.*—Slightly enlarged, movable.

*Left Arytenoid.*—Considerably indurated; the glottic space, though irregular in shape, is most ample in size for breathing purposes and bids fair to remain so unless encroached upon by future inflammations.

The anterior wall of the trachea is quite red in color and shows signs of irregularity in contour at the seat of the original tracheotomy wound.

The goitre seems to have diminished in size during the year.

The dimensions of the intubation tube used were:

|  |                   |
|--|-------------------|
| Length, 3 inches from the highest point of the head. |                   |
| Ant. Post. diameter of head.....                     | $\frac{3}{4}$ in. |
| Transverse diameter of head.....                     | $\frac{7}{8}$ "   |
| Ant. Post. diameter of lumen.....                    | $\frac{1}{2}$ "   |
| Transverse diameter of lumen.....                    | 5-16 "            |
| Outer circumference at neck.....                     | 1 $\frac{5}{8}$ " |
| Outer circumference at median swell.....             | 1 6-8 "           |
| Outer circumference at lower extremity.....          | 1 5-16 "          |

The exact time of the intubating period from the date of the first introduction was four years and twenty-five days, and with the exception of a few days the tube remained in continuously.

The object in presenting this history, other than to record a successful outcome in a case which at first seemed to indicate the wearing of a tracheal cannula or an intubation tube for the remainder of the patient's life, is to emphasize the following points:

1. The tolerance of the larynx to long continued pressure.
2. The superiority of continuous pressure in causing absorption over the older methods of the temporary introduction of dilating instruments.
3. The comparative comfort with which an intubation tube may be indefinitely worn.
4. The improved general condition of the patient while wearing the intubation tube in contrast to the debilitating influence and local annoyance of the tracheotomy cannula.
5. The superiority of the hard rubber over the metal tube in forming less amount of calcareous deposit, thus lessening the danger of ulceration and formation of exuberant granulation tissue.

MEMBRANOUS TERTIARY SYPHILITIC LESIONS  
OF THE PHARYNX, TONSILS, MOUTH AND  
SOFT PALATE (RESEMBLING VINCENT'S  
ANGINA), REPORT OF THREE CASES.

BY D. BRADEN KYLE, M.D.

Inflammatory conditions in which there is associated membrane formation involving any part of the upper respiratory tract are always suggestive of grave import. As to whether this membranous inflammation is an acute process and associated with the systemic inflammatory phenomena, or whether it is a chronic process lacking these systemic phenomena, will determine the immediate gravity of the case.

It is not my purpose in this paper to take up the various varieties of the membranous inflammations specifically, but rather generally, and to report three rather unique cases that have come under my observation. Of the many membranous lesions involving the mucous membrane of the throat, these three were unlike any I have ever seen. Whether the condition is uncommon, or whether it has been my misfortune not to see such cases, I do not know, although in looking up literature I find that there is very little on this subject. In speaking of "membranous lesions" I do not wish it confused with the crust formation which occurs in tertiary syphilitic ulceration; this was in nowise similar to such a condition, but was a distinct membrane.

The cases are so nearly alike that a general description of the three will suffice, with a few details of each particular case. The three cases, all males over thirty years of age, when they presented themselves at my office, showed a membranous condition involving the soft palate, lateral pharyngeal walls, the tonsillar tissue and the structures in the vestibule of the larynx. This membrane could be stripped off, and left a ragged, worm-eaten, bleeding surface. There were no associated systemic symptoms in two of the cases, although in the early history given



there evidently were systemic phenomena. The third case presented at intervals a slightly different phase, of which I will speak later. There was the local annoyance, such as irritation produced when taking cold or hot liquids; also, on eating, a local irritation sufficient to produce coughing, and at times the burning sensation which is always present in any local ulceration or irritation, although it lacked the phenomena of an acute inflammatory process. The tissue itself had that chronic appearance so easily recognized by the eye, but so difficult to describe in words. The line of demarkation was fairly well marked, showing a slightly inflammatory but very irregular border. The patient in each instance denied any specific history, but in spite of this, after using all known local and systemic remedies, the anti-syphilitic treatment was resorted to. The iodides were pushed to the physiological limit. The mixed treatment was then resorted to without result. The membrane continued to form, the ulceration growing deeper, and the discomfort of the patient increasing. In one case the tissue showed considerable thickening, and this, with the ragged, ulcerative surface, aroused a suspicion of malignant disease.

The bacteriological examination in one of the cases failed to reveal any specific micro-organism, various bacteria being present, but no specific germ. The other two cases showed Vincent's spirilla, besides many other bacteria. As the cases were under my care for several weeks, I noticed the tendency to rather an exacerbation of the condition, which seemed to light up in a rather sluggish acute process for a few days, and then to fade away to the chronic appearance. From the general appearance of the patient, the anemic condition and general debility, I determined that it certainly must be a specific lesion, and persuaded the patient to go into the hospital, and the anti-syphilitic treatment was given by inunction. In the course of ten days the membranous condition began to disappear, the ulceration gradually lessened, and the patient went on to fairly rapid recovery. Two of the patients, after leaving the hospital, went to Hot Springs, Arkansas, where they remained for two months, and at the present time have had no return whatever of the ulceration or membranous formation in the throat. They do, however, have



some slight scar tissue, but not sufficient to cause contraction. The third case did not receive treatment elsewhere, and made a complete recovery under the mercurial inunctions.

The first case I observed some ten years ago; the second one eight years ago; and the last one within the present year. The third case, which was one of the two in which the spirillum of Vincent was found present, presented more acute symptoms than the two preceding cases. His general condition was bad and he had acute attacks in which the temperature would run up to 101 and 102½. The cervical glands were involved, and the process was decidedly more acute than in the other cases. He was treated by every known remedy, and failed to obtain any relief. There was something about the appearance of the individual that made me suspect that it was a case similar to the two I had seen before, and I therefore persuaded him to go into the hospital. He had taken potassium iodide, also the mixed treatment, without result; I then ordered daily mercurial inunctions in connection with the iodide of potash. In exactly one week the patient began to show marked improvement, and to-day is entirely free from the membranous condition. When the membranous formation began to respond to the inunctions, I then assured him that he had had a specific infection, which he then admitted. The same was true in the other cases, although for months they stoutly denied any such infection. In two of the cases my suspicion of a specific infection was confirmed by finding on examination, when the patient was placed in bed in the hospital, that he had slight ulceration involving the skin over the shin and about the ankle. This history had never been given by the patient. Needless to say, the ulceration disappeared simultaneously with the membranous and ulcerative condition of the throat under the mercurial inunctions.

My object in reporting these cases is, principally, to call attention to three points: First, this unusual membranous formation in a tertiary syphilitic lesion; second, its close resemblance to the membrane as observed in Vincent's Angina, and also the presence of Vincent's micro-organism in two of the cases; and, third, the importance of the therapeutic test. I do not mean merely the pushing of the iodides or the mixed treatment, but in

these obscure cases, in which no results are obtained by the administration of the iodides or the mixed treatment, I certainly would urge the administration of mercury by inunction, and pushing this method of medication to the physiological limit.

Another interesting feature to me was the fact that the membrane was so well formed, could be stripped off and left a bleeding surface. The re-formation of this membrane was also interesting. In the beginning of the formation it was apparently part of the tissue, and would gradually necrose and form a distinct semi-fibrinous membrane.

The resemblance to the membranous inflammation of Vincent's Angina was certainly sufficiently marked to be misleading, and to lead to error in diagnosis, and in the two cases the isolation of this micro-organism would lead one to suspect a mixed infection. This I do not believe to be true. Otherwise, it is not reasonable to suppose that the mercurial inunctions would have any material effect on a local Vincent's Angina infection. I am inclined to think that the presence of this micro-organism was in nowise an etiological factor, but was merely accidental. As to the etiological factor in such membranous formation, it seems to me, from my observations in these three cases of tertiary lesions, that we cannot trace it to any bacteria. It seems to me to be structural, the underlying cause coming from within and not microbic in origin.

Upon reviewing the literature, I find no mention of this membranous condition associated with the specific lesion in the tertiary form. In the ulcerative membranous angina involving the mucous membrane of the mouth, tonsils and pharynx, and where there is formation of the false membrane described under various names—Vincent's Angina, etc.—it has occurred to me that probably some of these cases had, after all, an underlying specific infection. There has been a good deal of literature in the last few years on this subject of ulcero-membranous lesions, and various fusiform bacilli and spirilla have been described. There is no doubt that we do have a number of separate and distinct lesions, but I merely raise the question as to whether or not some of these forms might be a latent specific lesion merely observed and described in different stages. Certainly, the report

of some cases in which there is destruction of tissue, even extending to the point of necrosis, would suggest some underlying systemic causes, and there is no one disease with which the physician has to contend that presents more varied and curious phenomena than are observed in syphilis, especially in the latent forms and in the tertiary variety. In the streptococcal forms we always have an acute inflammatory process with the rapid, systemic phenomena, and it may be observed in either the robust individual or those below par; while the various forms of pseudo-membranous anginas are more frequently observed in the individual who is decidedly below par.

In the cases reported above, the sloughing was marked, and the necrosis was in the ulcerative form, but irregular. The odor in each case was distinct and offensive, being that observed in gangrenous or sloughing surfaces. The ulcero-membranous inflammations observed in pneumonia and typhoid fever, in which various specific micro-organisms are found, do not present the marked ulcerative or gangrenous tendency that is shown in these specific cases. In a general way they could be divided into the ones due to a specific micro-organism, and the ones not associated with any specific micro-organism; or, in other words, the acute and chronic.

Under the variety in which no specific micro-organism is found could be classed the chemical ulcer, in which frequently a membrane forms over the ulcerative surface. In many cases it is difficult to determine whether the micro-organism present is an etiological or merely an associate factor, as we all know that many forms of bacteria are constantly present on the mucous membrane surfaces of the upper respiratory tract. One thing is certain, this class of chronic inflammation as described in the three cases above, shows no signs of being contagious or infectious, as I made careful inquiry and observation in these cases, and although no precautions had been taken until the patient came under my observation, yet no other member of the family showed any signs of infection or any other evidence of having contracted the disease, which, of course, would be true in the tertiary form of syphilis. But, if it was due to any specific etiological factor in the form of bacteria, such escape would hardly be possible.

The Follicular Angina, the Lacunar Angina, Friedländer's Angina, Vincent's Angina, the Angina Ulcerosa Benigna of Heryng, the aphthous sore throat, the herpetic sore throat, all come under the variety Ulcerous Membranous Angina.

*Candidate's Thesis.*

## MULTIPLE PAPILLOMA OF THE LARYNX IN CHILDREN.

BY HARMON SMITH, M.D.

The presence at the same time in my clinic at the Manhattan Eye, Ear and Throat Hospital of three cases of multiple papilloma of the larynx in children varying in age from five to ten years, affords such unusual opportunities for observation of the vagaries of this trouble that I have ventured to submit their histories, with comments, as material worthy of your consideration.

Last year Coakley presented a paper upon the same subject which elicited considerable discussion; but there seemed to exist as many divergent opinions upon treatment as existed some years ago when Dr. Hunter Mackenzie advocated so strongly tracheotomy alone as a curative measure in all cases of papilloma of the larynx in children.

Case I. F. L., male, aged seven, was first admitted to the Manhattan Eye, Ear and Throat Hospital in 1905. At that time the patient complained of hoarseness only. There was no family history bearing upon the case. I am indebted to Dr. Duncan McPherson for the history of the case when first observed, and under whose skillful treatment the patient still remains.

The growth at first appeared single, sessile, and of mulberry shape. It was removed with the Mackenzie forceps at varying intervals, and at times was completely eradicated. About four to eight weeks generally intervened between operations, and this continued until the child came under my observation in October, 1907. There had been difficult breathing occasionally, which would be immediately overcome by removing the growth. Dr. McPherson has noted the diminished rapidity of recurrence in the last year, and frequently a period of one to two months will now intervene between recurrence. The general health of the

patient has improved, and from a poorly-nourished, anaemic child, he has developed into a sturdy boy.

A fibrosis has occurred in the anterior commissure, which may occasion some loss of voice, even after the growth has disappeared. However, it is the opinion of Dr. Jonathan Wright, under whose surgical care the patient has been for a part of the three years' observation, that the fibrosis will also disappear when the operative interference ceases. Dr. Wright considers



Case I.

F. L., male, aged seven years. Drawing made after beginning operations for removal of the growth. The white area surrounding the base of the neoplasm is the fibrosis resulting, probably, from the frequent removal.

the method pursued in this case to be the ideal one when conditions favor it. The instrumentation is easy of accomplishment; the growth is single and does not interfere particularly with respiration; and there is nothing prohibitive in keeping the larynx clear until that physiological change occurs which will prevent recurrence.



Case II. A. Le R., Italian girl, aged ten, was admitted to the clinic July 11, 1907. She was brought to the hospital particularly for the removal of tonsils and adenoids, although she had been hoarse for five months previously. Owing to her struggles, it was absolutely impossible to examine her larynx in the clinic. No family history bearing upon the case was obtainable. There were five other children in the family, all of whom were healthy and without hoarseness. No warts were found upon the body. The patient had had none of the diseases of childhood, but had lost about fifteen pounds in the previous five months.



Case II.

A. Le R., female, aged ten years. The drawing presents the condition following the first operations for removal of the neoplasm.

At the time of examination there was slight cyanosis, and that anxious "facies" denoting difficult respiration. The voice was a hoarse whisper. I advised immediate admission to the hospital, and tried adrenalin sprays and steam inhalations, by which she was temporarily relieved of her distress. On July 18th she was anaesthetized for the removal of the tonsils and adenoids, at which time I obtained a view of the larynx, and

removed a small papilloma for examination. The larynx had apparently three rather large growths in it, two upon the cords anteriorly, and one in the sub-glottic region.

Dr. Wright, in reporting upon the specimen, said that the growth was a papilloma springing from a base of granulation tissue, probably tuberculous. Examination of the lungs, however, revealed no evidence of tuberculosis, nor the sputa any bacilli.

The case was immediately put on cod liver oil and arsenic, and seemed to respond to the treatment by gaining four pounds in two weeks. On August 1 I performed tracheotomy, believing it to be the best thing for the patient, for three reasons: First, her breathing was difficult and she became quite cyanotic at times; second, she was absolutely intractable for operative measures "*per vias naturales*"; third, the presence of a tracheal tube would permit the better use of the Jackson tubes for frequent removals of the growths, if necessary.

The trachea was opened high up under chloroform anaesthesia, and the immediate results were good; but the next day, and for many subsequent days, severe hemorrhages would occur from the trachea and from the wound itself. The child would work at the tube until it was out of place, and owing to the many attempts to replace it a false passage was made along the side of the trachea. This condition continued for about ten days, until finally the inflammation subsided, and the tube became less irritating. Exuberent granulations sprang up around the tracheal wound, which were removed with scissors, and the bases were then cauterized with fused silver nitrate.

On October 15, under chloroform anaesthesia, a small number of papillomata were removed from the larynx through the Killian tubes. The light was unsatisfactory, and the speculum was not adaptable for use in a child's larynx. On November 2, another attempt was made by my assistant, Dr. McPherson, to remove more of the growths with the Killian tubes. He was not successful with the tube, but quite a few were removed by Mackenzie's forceps with the aid of the laryngoscope.

Following this operation, the patient was quite sick and could retain no food for several days. There was no laryngeal oedema, however. On February 13 she was again operated upon, and

this time I used the Jackson self-illuminating speculum, and also his forceps. I removed a number of papillomata and found them in many places where I had not seen them previously when using the laryngoscopic mirror. I found the Jackson tube much more satisfactory, and no untoward effects followed. The patient had continued to gain in weight, and was breathing so well that she had been permitted to make frequent visits home of one or two days duration.

On March 27 Dr. McPherson operated upon the patient with the Jackson speculum and removed as many growths as had been previously taken out altogether. Since this time there has been more space in the larynx and the child can speak in much clearer tones than before. There are, however, quite a number of the growths still present, and they do not yield to arsenic and potassium iodine internally, nor to alcohol, zinc chloride, adrenalin and silver nitrate locally. I am now injecting castor oil into her larynx daily.

Case III. J. M., of German parentage, male, aged five years; admitted to the Throat Clinic of the Manhattan Eye, Ear and Throat Hospital, October 26, 1907. The mother gave a history for the child of snoring and mouth breathing, and at the time of admission my attention was directed only to the presence of adenoids and tonsils. Nothing was said at this time to make me suspect laryngeal growths. Upon subsequent investigation I obtained the following facts from the father: When the child was two years old he had sore throat, with fever, and after three days' sickness had a sudden attack of dyspnoea and became slightly cyanotic. A physician was called in, who administered some medicine which relieved the condition for a time, but the hoarseness continued. After several months of hoarseness the father took the child to the Post-Graduate Hospital, where some growths were removed from the throat, resulting in no apparent relief, although the child remained in the hospital for one week. The father took the child home, where for about six months he remained with no discomfort other than hoarseness. However, about one year ago the child began to breathe badly and would choke at night. The father said that frequently the boy would rise in bed, stretch out his hands, and apparently plead for air. He then took the child to Bellevue Hospital, where the doctor removed some growths from the larynx, and after two weeks a second removal occurred. These two operations overcame the choking condition, and he was brought to the Manhattan Eye, Ear

and Throat Hospital seven months ago for the snoring and sore throat only. No family history bears upon the case. There are three other children in the family with normal voices and in good health.

Upon admission the child was fairly well nourished and the breathing was not bad except upon excitement. I removed his tonsils and adenoids upon October 29, after admission to the hospital, and not until he protested rather vigorously against the



Case III.

J. M., male, aged five years. The drawing shows the appearance of the larynx before any radical interference had taken place. Only a few papillomata had been removed when this drawing was made.

anaesthetic did I notice anything unusual in the voice. He took his anaesthetic badly, but before removing his tonsils and adenoids I examined his larynx and found it almost completely blocked with papilloma. I removed some of these growths at this time, to give him better breathing space, and after the opera-

tion he was placed under a tent. With the exception of bad breathing and delayed recovery from the anaesthetic, nothing eventful resulted from the operation. The breathing and hoarseness became worse after the operation, and continued to increase until November 16, when I performed a tracheotomy. This overcame the difficult respiration. The patient made a good recovery from the operation, and on January 25 I removed a large number of papillomata from the larynx with the Jackson speculum and forceps. Chloroform anaesthesia was employed. Temporary



Case III.

As it appears May 1, 1908, after frequent removals of all visible papillomata.

relief was obtained only, for new growths had sprung up in different areas, completely filling the larynx and even encroaching upon the epiglottis. On February 18, pursuing the same methods as before, I again removed at least a half dram of these warts. On March 3 I again performed the same operation and removed more growths than ever previously. The larynx is now filled up again with a greater number and larger growths than ever—one



is about the size of a raspberry. I have applied locally alcohol, zinc chloride and nitrate of silver, and now I am injecting pure castor oil into the larynx once a day in hopes that the same results may obtain in the larynx as upon the skin.

The microscopical reports upon all these growths have been simply papilloma. The patient has steadily gained in weight. In this case tracheotomy was imperative, as no amount of operative interference would have kept down the growths.

#### ETIOLOGY.

Apparently nothing is determinable as to the cause of these growths in children, for they may occur at birth or at any time during life. Morell Mackenzie performed a tracheotomy upon a child aged two years and four months, in which the larynx was found, upon autopsy, to be filled with papillomata. The history of a hoarse, suppressed cry at birth, and a continued loss of voice led him to conclude that it was unquestionably a case of congenital papilloma. All congenital tumors of the larynx have been found to be papillomatous (Morell Mackenzie, *Laryngeal Growths*, 1871).

The appearance of a papilloma at birth precludes in a measure the probability of voice strain as a cause. The history of a number of these cases shows that hereditary tendencies, lowered vitality and unsanitary conditions seem to play no important part in their formation. In some instances, however, hereditary syphilis is a possible cause, and frequently the growths will disappear under the administration of potassium iodide.

The same contagiousness that characterizes the skin wart may properly apply to the laryngeal variety, if we accept as evidence the presence of new formations in different parts of the larynx following operative measures, which areas were previously free from the growths. Papilloma of the larynx may be either pedunculated or sessile, single or multiple, and may present any size from that of a millet seed to that of a raspberry. Some authors claim that upon minute observation all of these tumors are pedunculated (Knight, *Diseases of the Nose and Throat*).

Another point of resemblance between the skin wart and the laryngeal variety is the uncertainty or vagary of disappearance.



Frequently a skin wart will disappear without any recognized cause, and the same may be said of the laryngeal variety. It is this inexplicable change which has led some observers to conclude that, irrespective of treatment, the growths will not disappear until that physiological change has occurred in the subject which will retard the activity of the cell organism (J. Payson Clarke, *Papilloma of the Larynx in Children*, Boston Medical and Surgical Journal, September 28, 1905).

Morell Mackenzie calls attention to the presence of skin warts as originating probably from the irritation of dirt, as he had observed these growths in cooks, coachmen and artisans. As giving additional weight to this argument, I would add the observation of Jonathan Wright in regard to the greater frequency of warts upon country children's hands, particularly those who are permitted to keep them dirty—to which observation I can readily accede. Another point is that of location of the skin wart, which is usually at the knuckle or between the fingers, where the papillae of the skin would be most irritated by the dirt being rubbed in. Irritation of the mucous surfaces, however, would have to result more from friction against another mucous surface or from the rubbing in of the mucus itself. Local predisposition seems to be the only etiological factor, however, that can be definitely cited. Of course there are those cases of papilloma of the larynx in adults due to the inspiration of irritating vapors and particles of dust, but this condition is unlikely to occur with children.

#### LOCATION.

These growths appear more frequently upon the cords than upon the ventricular bands, and more frequently in the anterior than in the posterior part of the larynx. They rarely occur in the posterior commissure, except the so-called tubercular papillomata. However, in Case No. III., they spring from all parts of the larynx and even upon the under surfaces of the epiglottis and the ary-epiglottic folds.

#### SYMPTOMS.

The symptoms are somewhat characteristic, though they might easily be simulated in retro-pharyngeal abscess—abscess at the

base of the tongue—laryngeal diphtheria, or in laryngeal stenosis from any other cause. Croup simulates papilloma of the larynx very strongly, and the converse of this is also true, and no doubt many cases of papilloma have been diagnosed as croup.

Hoarseness, or possible loss of voice, dyspnoea, cyanosis and the peculiar facial expression of anxiety accompanying any disturbance of respiration are the significant symptoms present. In Case III. no suspicion of papillomata existed when the child was examined for adenoids, and it was only when he objected to the anaesthetic that my attention was called to the peculiarity of his voice, which led me to examine his larynx under anaesthesia before removing the tonsils and adenoids. Papillomata may be present in a great many cases where the hoarseness has been attributed to adenoids, and the possibility of this error should induce us to make laryngeal examination in every case where hoarseness is present. The parent of Case III., who is a German, offered no suggestions as to the previous history of the case, and the many experiences related were all obtained after tracheotomy was performed.

#### FREQUENCY.

Although this neoplasm is regarded as existing more frequently than any other laryngeal growth, yet the statistics of large clinics show that it is of relatively rare occurrence in children. In the clinic of Dr. Chappell, with which I was associated for ten years, at the Manhattan Eye, Ear and Throat Hospital, there was but one case of papilloma of the larynx in a child. In another clinic in the same institution there were only two cases during the same period. Clarke reports only twelve cases in the Massachusetts General Hospital in the examination of twelve thousand six hundred and twenty-three children under fourteen years of age (*Boston Med. & Surg. Jour.*, Sept. 28, 1905). In eleven hundred tumors of the larynx reported by V. Bruns, six hundred and two were papillomata, but of this number the author does not state how many existed in children.

In 300 tumors of the larynx reported by Fauvel, 206 were papillomata; of this number, only nine were in children under fifteen years old (*Traité Pratique des Maladies du Larynx*).

In 200 tumors of the larynx reported by Massei, ninety-four were papillomata (*Sui Neoplasma Laryngei, Studii e Casuistica, Napoli, 1885*).

In 100 tumors of the larynx reported by Morell Mackenzie, sixty-seven were papillomata, and of this number only eight were in children.



The larynx of an infant who died of asphyxia at the age of seven months. The case was probably one of congenital papilloma, and the history and appearance of the growth bear a striking similarity to Case III. The drawing is a reproduction from the *Archives of Pediatrics*, Nov., 1904, in which journal the case was reported by Theodore J. Etterich.

In 310 cases of laryngeal tumors reported by Schwartze, 163 were papillomata, but of these the author does not state the number occurring in children (Des Tumeurs du Larynx).

#### VARIETY.

To the eye of the examiner, many different kinds of papillomata are noted. The most frequent is pale, granular, apparently friable, and with but feeble attachment to the cord or ventricular band. This variety more nearly resembles the skin wart than any of the others.

A second kind has about the same shape and size as the first, but appears more vascular, and varies from a *polypoid* condition to a *pinkish mucus-soaked* appearance, which would indicate a more rapidly growing and a better nourished neoplasm.

A third is where the growth appears sessile, or where there is a large base from which spring other projections of the same neoplasm. These assume more of the cauliflower appearance. Such a growth in an adult larynx would occasion doubt in making a diagnosis.

All these macroscopical varieties, however, appear to be of the same structure when examined microscopically. The only variation to this statement in my three cases is in the report of Case II., upon the examination of some of the papillomata removed at the first operation. Dr. Wright adds that the growth sprang from a basis of granulation tissue, probably tuberculous, and in the last report says: "This seems to be a simple papilloma, but near the periphery of the epithelial papillæ there are numerous cells containing degenerated hyaline matter, and at one place a distinct and typical whorl or epithelial pearl may be seen." The consistency is variable, being that of a polyp in some instances, and in others being horny and friable, even necrotic when of a tuberculous or of a syphilitic nature.

#### PATHOLOGY.

"The growth may be occasioned either by an hypertrophy of the normal papillae or may be entirely new formations, in the part. The basic substance is formed of connective tissue. The

free surface is covered by epithelium, which may vary in its thickness and in the number of its layers, according to the seat of the tumor. Blood vessels and even nerves enter into the interior of the papillae." (Paget, Lectures on Surgical Pathology, 3d Edition.)

#### TREATMENT.

Each case has to be treated according to the peculiarity of the symptoms present, and from the literature upon the subject one is forced to conclude that there are many cases apparently similar which will not yield to the same line of treatment.

• I believe that treatment can well be considered under three heads: First, that of *Necessity*; second, that of *Expediency*; third, that of *Election*. Under the first head will come those cases brought under observation—slightly cyanotic and with marked dyspnoea; where the child is emaciated, narrow chested and with the characteristic "facies," denoting insufficient respiration. This child will show little disposition to cry, because the exertion lessens still more the inspired air. In such a case there is nothing justifiable but immediate tracheotomy. To attempt anaesthetization with a view to using the laryngoscopic tubes would be unwarrantable, and without an anæsthetic no child would stand the use of the Killian or the Jackson tubes. Likewise, under these circumstances, the time necessary to accustom the patient to laryngeal examination and manipulation is not permitted the operator, and the only expedient is tracheotomy. I do not believe that any further operative measure is justifiable at this time, for the patient's resistance is already lowered to the minimum, and prolonged anæsthesia or laryngeal manipulation but adds another factor in diminishing this resistance.

Since the advent of the Killian and the Jackson tubes, particularly the self-illuminating laryngeal speculum of Jackson, which enables the operator to remove all the laryngeal neoplasms directly and without endangering the laryngeal structure, thyrotomy with its subsequent possibilities is absolutely unjustifiable in children. Even though we consider as favorable the results in a number of cases in which thyrotomy has been performed—there having been no recurrence during the period of observation—yet there are so many instances where it has failed utterly or

where it has resulted in a complete loss of phonation that one is compelled to look upon it with disfavor in the case of children, especially now since entirely safe measure are possible through the use of the Jackson laryngoscopic tube for the continued removal of these growths until nature has brought about the change that will result in their disappearance.

Taken as a whole, *successful* thyrotomies are materially in excess of *unsuccessful* ones, according to the reports in literature, but we have a number of particularly *unsuccessful* cases reported in which thyrotomy has failed.

Abbe reports a case in which thyrotomy was performed four times, with continued recurrence of the growth, although the bases of the structures were cauterized. Tracheotomy had to be done finally, and the tube remained in for six years, when the growths disappeared; but the voice was lost—which condition was most assuredly due to the frequent thyrotomies. (N. Y. Med. Rec., April 28, 1900.)

Walker Downie reports a case in which thyrotomy was performed six times in one year in a child two years old. Lindon reports another remarkable case in which thyrotomy was performed seventeen times in two years, resulting finally in laryngeal stenosis and necessitating the permanent use of a tracheal tube. (The Treatment of Laryngeal Growths in Children, by G. Hunter Mackenzie, Brit. Med. Jour., Sept. 28, 1901.)

Following are a number of recorded cases of unsuccessful thyrotomies, performed by various operators in different countries:

## Cases.

- |           |  |
|-----------|--|
| Massei    | 1 Internat. Centralblatt für Laryng. Jahrgang, x, s. 362.  |
| Garel     | 2 Annales des Malad. de l'Oreille et du Larynx, Juni 1891. |
| Oertel    | 2 Sammlung Klinischer Vorträge, No. 315, s. 2807.          |
| Railton   | 2 Brit. Med. Jour., Feb. 19, 1898.                         |
| Eliasburg | 1 Jour. of Laryngol., vol. v., p. 245.                     |
| White     | 1 Jour. of Laryngol., vol. vi., p. 486.                    |
| Cowgill   | 1 Phil. Med. News, Oct. 4, 1890.                           |
| Chappell  | 1 Jour. of Laryngol., vol. ix., p. 555.                    |

Summers reports a case of a child two years old with dyspnoea and hoarseness, in whom low tracheotomy was performed and then thyrotomy. Upon attempting to remove the tracheal tube the child would choke. O'Dwyer's tube was tried unsucces-



fully. Then a second thyrotomy was performed to no purpose, and finally the tracheal tube was left in place for two years, with satisfactory results. He also mentions two other cases in which the tracheal tube was successfully used.

Prof. B. V. Bruns, of Tübingen (*Handbuch der Praktischen Chirurgie*, 1902), collected statistics of 127 children with laryngeal papilloma; 48 were not operated upon, and of these 32 died—28 by suffocation; three were cured spontaneously; 26 were tracheotomized—7 dying after the operation; 21 were subjected to laryngotomy—8 of these were permanently cured; 40 were treated by intra-laryngeal methods, of whom 13 were permanently cured.

Rosenberg's Statistics:—109 children were subjected to laryngotomy, the operation being performed 144 times; 11 children were operated upon twice; 3 children were operated upon 3 times; 1 child was operated upon 4 times. In all 52 children were not over four years old; 20 died mostly from suffocation due to recurrence of the papillomata; recurrences took place in 43 after repeated operations; 40 (or 36 per cent.) of these were cured; 10 of these had more or less loss of voice; 88 children were treated by intra-laryngeal methods; 13 were not over four years old; 3 died, one from suffocation due to recurrence. Forty (45 per cent.) were cured, but 16 of these were of too recent date to determine the final outcome.

Thyrotomy may occasion loss of voice and stenosis of the larynx. It may be unsuccessful in eradicating the growth and require frequent repetitions of the operation without ultimate success, and finally necessitate employment of other measures to effect a cure. It may also be classed under the head of dangerous to life, as shock, producing death, frequently follows operative interference in the larynx. In view of the fact that it only enables the operator to remove successfully all growths from the larynx at the time, with no definite assurance of their non-recurrence, and as this may be almost as successfully accomplished with a Jackson tube, without endangering the laryngeal structures, I believe that thyrotomy is unjustifiable.

Second. Under the head of Expedient treatment would come that of tracheotomy. When is it justifiable? Should it be performed in every case of laryngeal papilloma? If all observers and operators had had the pleasing experience of Hunter Mackenzie there would be no argument against tracheotomy in every instance, but for some reason there have been those who have

not met with the same favorable experience, though these cases have occurred in the hands of men equally skilled and equally desirous of favorable results. A marked example of the ineffectiveness of tracheotomy is the case reported by Dundas Grant, in which a tube had been worn for twenty-two years with no disappearance of the growth. (*Internat. Med. Annual*, 1904.) Sympton reports a case of recurring papilloma in a child aged seven, in which a tracheotomy tube was kept in place for one year and three months, with no improvement, when thyrotomy was performed, with a recovery. (*Brit. Med. Jour.*, Nov. 9, 1901.)

The consensus of opinion justifies tracheotomy in those cases of multiple papillomata of the larynx in which there is difficult respiration or impaired health apparently due to bad breathing; also in cases in which there is no evidence that the patient will become tractable in the use of intra-laryngeal methods; likewise in cases of rapid recurrence after removal. The presence of the tracheal tubes in cases of multiple papillomata is a source of great assurance to the operator, whether the removal be by the old laryngeal method or with the Jackson tube, for even slight operative measures in the larynx may result in unexpected oedema. The tube is a safeguard against suffocation, but its long-continued use may also result in either stenosis of the trachea or a collapse of the rings due to long-continued disuse. The stenosis may be overcome by dilatation, but the collapse is of a more serious nature. This unfortunate occurrence may be obviated in a measure by frequent removal of the tracheotomy tube or by substituting a soft rubber tube for the metal one. I have endeavored to use a very short tube—one that would just engage the tracheal wall—but every fit of coughing disengages the tube, which renders it impracticable. The wearing of the tube probably hastens that physiological change in the mucosa necessary to the disappearance of the warts by reducing the vocal activity and thereby reducing the blood supply to the area. But in view of the instances cited, in which tracheal tubes have been worn for a long period without eradicating the growth it is unreasonable to conclude that the mere presence of the tube without some collateral aid would result in the disappearance of the growth.

When the papilloma is single and in no way endangers the respiration, I would deem it inadvisable to do a tracheotomy.

Third, Election. Treatment under this head would involve no surgical procedure other than that which might be accomplished through the "*vias naturales*." In a limited number of cases papilloma may be removed from the larynx of children after the use of cocaine only, with Mackenzie's forceps, the snare or the curettes of Krause, Heryng, Landgraf, and Schrötter. In case I. the child has been trained to hold a perfectly immovable larynx and no difficulty is experienced in removing the growth with any of the instruments mentioned, though the position of the tumor renders the Mackenzie cutting forceps the most effective. I have attempted snaring the growth, but have always failed, owing to the loop slipping off. In order to use the Jackson self-illuminating speculum, it is necessary to anæsthetize the patient, which in itself adds an element of danger; but if the patient will not permit of intra-laryngeal examination it becomes imperative to anæsthetize, in order to facilitate the employment of the Jackson tubes.

Chloroform anæsthesia, rather than ether, is preferable in these cases, because it lessens the laryngeal secretions by being less irritating to the mucosa. After partial anæsthesia by chloroform, it is well to apply a 10 per cent. solution of cocaine to the interior of the larynx, and also a one-to-one thousand solution of adrenalin, which renders the larynx less sensitive to manipulation and obviates the necessity of deeper general anæsthesia.

By the time the patient is under the anæsthetic the cocaine has overcome the laryngeal reflexes, which has to be accomplished before the introduction of the speculum. The patient is then placed in the Rose position, with the head well over the table and supported with the left hand of the assistant, who sits to the right of the patient with his knee under the head and his foot on a stool. With his right arm extended under the neck of the patient, he introduces his index finger, protected with a large iron thimble, between the jaws and opens them sufficiently to admit the speculum. The larynx, trachea, and mouth should be brought into as direct a line as possible. The operator kneels upon a low stool at the head of the patient and introduces the speculum between the tongue and hard palate and presses upward and

forward until the epiglottis is seen, when he engages this upon the tip of the speculum, elevating the tip and depressing the handle, until the laryngeal structures come into view. Gentle manipulation is necessary to present the structures in anything like their normal appearance, as too much pressure will pull the ventricular bands together and occlude the view of the structures beneath.

In my two cases it has not been difficult, after a little experience, to see the growth plainly in the larynx. I have used the Jackson forceps with complete satisfaction in removing the warts. It is by no means as easy of accomplishment as it appears to the onlooker, and the instrument frequently interferes with the illumination of the growth. It is quite possible to mistake the tips of the arytenoids for a papilloma when the parts are covered with blood, and I deem it one of the essentials of the operation to keep the parts well sponged and as bleached as possible. Adrenalin acts fairly well as an astringent at first, but I have found a weak solution of zinc chloride, or a solution of tannic and gallic acid controls the hemorrhage for a longer period. I do not consider it wise to keep the patient anæsthetized too long, and would advise frequent removals rather than prolong the anæsthesia. There never has been any oedema following these operations in my cases.

After the operation the patient is kept under a tent for twenty-four hours and the air is moistened by the steam from a croup kettle. Ordinarily I use a mixture of *oi. Pini pumilionis*, one dram; menthol, 10 grains; and milk of magnesia, 2 ounces. Of this mixture, one teaspoonful to a pint of water is sufficient. This mixture gives off a steam laden with a non-irritating medication which is very acceptable to the patient.

#### LOCAL TREATMENT.

Nearly every conceivable astringent has been applied locally to these growths. Quite a few observers have seen beneficial results from alcohol applied in full strength. Delavan has particularly recommended this in the papilloma of adults. I have applied it consistently in two of my cases but with no apparent improvement. *Thuja Occidentalis* has been used by quite a number, most frequently in adults. Brown, of Chicago, used *Thuja* in an adult with apparently good results. (Jour. Amer. Med. Ass., August

3, 1901.) Zinc chloride, nitrate of silver, adrenalin, lactic acid, and many other astringents have been applied locally for the removal of these growths, with but little apparent result.

#### INTERNALLY.

Arsenic has had more advocates than any other internal drug, and no doubt arsenic hastens that physiological change necessary to the disappearance of the warts. I have had cases II. and III. on Fowler's solution since their admission to the hospital, and they have both gained in weight; but I am skeptical as to the effect upon the larynx. I have also had these cases on potassium iodide for a limited time, but with no apparent effect. Bostock is a very strong advocate of potassium iodide and arsenic internally, alternating between the two (*Monatsschrift für Ohrenheilkunde*, 1905, Part II.)

I believe that any internal medication which will build up the general system will aid in hastening the departure of these growths, and likewise the local application of any astringent will retard the growth of the wart by diminishing the blood supply.

As castor oil applied frequently to a skin wart will often result in its disappearance, I thought it might act as well in the larynx. To this end I have injected into the larynx daily a few drops of castor oil. Time for positive conclusions in regard to its efficiency has not yet elapsed. Castor oil is certainly not irritating and can result in nothing unfavorable.

SARCOMA OF THE TONSIL—REPORT OF A CASE—  
OPERATION—PATHOLOGICAL FINDINGS.

BY JOHN EDWIN RHODES, M.D.

S. W. F., a traveling salesman, was referred to me December 6, 1907, for operation upon a tumor of the left tonsil. He was a man thirty-five years of age, married, the father of a healthy child, and apparently a man of robust health except for the condition of the throat. He gave me the following history:

In July he was first conscious of any trouble in the throat. He noticed at that time some fulness, and at first a considerable amount of pain, which persisted for a short time only. There was occasional pain at that time, but not of a severe character. There was none from external pressure at the angle of the jaw. The pain was of a dull, aching character and not constant. He had been losing in weight for some time, and was about fifteen pounds below his normal weight of one hundred and eighty-eight pounds. His appetite was good and his digestion normal, but the present size of the growth somewhat interfered with deglutition. His strength was as good as usual, and he had followed his avocation up to that time. He had had local treatment by his physician since August, but the growth had continued to increase slowly in size, although the discomfort had been somewhat ameliorated by the applications. Recently, however, he had complained of an increasing difficulty in breathing, interfering with his rest at night, which was broken by many choking sensations, and he had been obliged very recently to sleep in a half reclining position to get any rest at all. His greatest fear was that he would choke to death in one of the spasms of dyspnoea. He had been a little hoarse at times, but the functions of the larynx seemed not to be affected. There had never been any external swelling. He had never had any severe illness, and there was no history of lues. He had had an occasional sore throat.

He recalled a local application made to his tonsil six years ago by a physician, who used a piece of wire and injured the tonsil in swabbing it, and it did not heal for some time afterward. He never used alcoholics to excess, but had been a confirmed tobacco user. He had a small mass of fine cut tobacco in his left cheek almost continuously from the time he arose in the morning until he retired at night, often swallowing the saliva. He chewed five



cents' worth of fine cut tobacco every two days, and smoked a pipe or half a dozen cigars daily in addition. This had been his habit for many years. His father died at sixty-two, of diabetes; his mother is living at sixty-two and is well. He has four brothers living, aged respectively thirty-three, thirty-one, twenty-seven and sixteen. Two sisters are living, aged twenty-three and twenty-one. Two sisters died, one in infancy and the other aged thirty-three, of typho-pneumonia and spinal meningitis. One of his sisters was treated for goitre a year ago, and cured, and one brother is now being treated for the same affection. The patient is of a very nervous temperament. He had had some bleeding from the throat every morning, but of small amount. His temperature was 99° F., and his pulse 96 and regular. The tongue was clean and its motility normal. There was no headache or dizziness.

On examination I found no enlargement of the glands externally about the angle of the jaw or neck. The nasal cavities were free. On depressing the tongue a tumor of the left tonsil was shown situated within and extending from the tonsillar space outwards two-thirds across the fauces, attached only within the tonsillar triangle. The lower part of the growth hung over the base of the tongue as far as, and impinging slightly on, the epiglottis. This tumor was irregular in outline, smooth on its surface, but somewhat lobulated, pinkish in color, rather easily penetrated by a smooth, flat, blunt probe and bleeding quite easily. Blood vessels indistinctly showed over the surface of the tumor. The patient gave a history of an obstinate hemorrhage in childhood from a wound in the foot, several days after the injury, but there was no reason to think he had a hemorrhagic diathesis. I decided it would be much safer to provide a bloodless field for operation, if possible, having in mind the tendency of similar growths in the throat to bleed, sometimes alarmingly, at the time of operation, and recalling the statement of Chevalier Jackson last year that external carotid ligation may be of use not only to forestall hemorrhage, but to inhibit growth. So I determined upon the preliminary ligation. Inasmuch, however, as collateral circulation is quickly established, I cannot see that there is much to be hoped for in the inhibition of growth in these tumors, although from the fact he emphasized that sarcoma spreads by the vessels, such ligation must have a temporary effect upon growth.

An operation was desired by the patient at once to relieve the difficulty of breathing. On the 8th of December I sent him to hospital for operation. Because of the great difficulty in breathing I decided to operate entirely under local anaesthesia. The patient was tractable and brave. The first step in the operation was to ligate the external carotid, which was done by Dr. Freer.

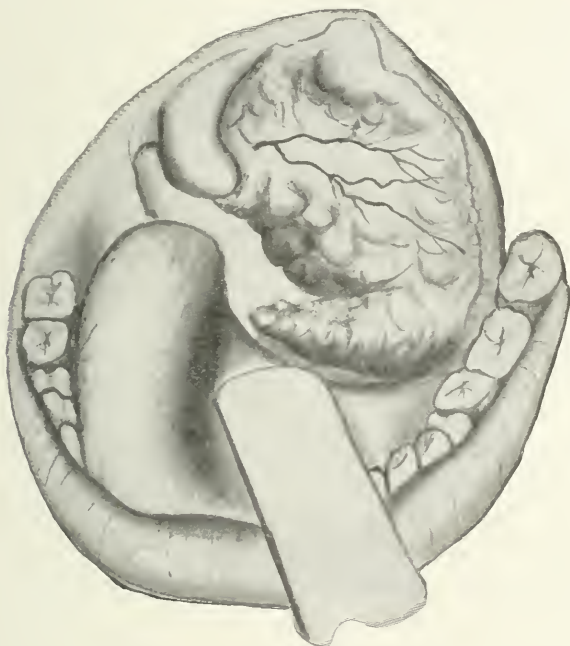


Fig. 1.

My friend Dr. J. Gordon Wilson, at the University of Chicago, has made some painstaking investigations regarding the blood supply of the tonsil, and he tells me that the blood supply in every case he has ever examined has been so definite that there can be no doubt, in his opinion, as to what it normally is. The main blood vessels, the tonsillar, come from the facial, either directly or from the ascending pharyngeal ramus. This artery, or the tonsillar, arises from the facial soon after its origin from the external carotid. The tonsillar artery passes through the pterygo-pharyngeal space towards the deep surface of the tonsil. In the

fibrous tissue which surrounds the gland it divides into two parts, one branch ascending to its upper pole, the other downwards to the lower one. As these arteries pass outward from the middle point of the tonsil—that is into the tonsillar tissue—they divide into a great number of small branches, each lying in a considerable amount of fibrous tissue, derived from the fibrous investment of the tonsil. A number of English and American text books state that the blood supply also comes from the lingual and the palatine. This is the case in some of the lower animals, but is not so in man. The lingual sends a branch to the anterior pillar, the ascending palatine to the posterior pillar, the descending palatine branches go to both. Obviously, therefore, the ligation of the external carotid should afford a practically bloodless field for operation, unless there should be an anomalous distribution of the blood vessels. After the ligation of the external carotid artery, bearing in mind that the ligature must be nearer the trunk of the common carotid than two centimetres, to include the facial, which sometimes arises by a common trunk with the lingual, there are two possibilities of hemorrhage, notwithstanding the ligation—first, from one of the arteries in the pillars, and second, an abnormal tonsillar branch, as cases have been recorded of the origin of the tonsillar artery from the external carotid directly. A hemorrhage might take place from the latter if the external carotid were ligated above this origin of the tonsillar. Care should therefore be taken to ligate close to the origin of the external carotid.

In order to facilitate this procedure it is customary to have the patient's head thrown strongly backward while he occupies a position on his back with his face turned slightly away from the operator. This lifts the artery towards the surface, and raises the jaw and submaxillary gland up out of the way, thus giving a wide operative field. In this patient the ligature of the artery was made extremely difficult because he had to keep his head flexed and chin lowered in order to breathe. This brought down the submaxillary gland in front of the trunk of the external carotid artery and made it necessary to hunt for the vessel in the lowest part of a deep pocket created by the larynx, sterno-mastoid muscle and the lower jaw.

The incision was made along the anterior border of the sternomastoid muscle, beginning above the angle of the jaw and extending to the cricoid ring, after the injection of 2% cocaine freely. After division of the platysma fibers the first object seen was the submaxillary gland, which covered the arterial trunk and had to be dissected out of its bed and retracted upward in order to render the vessel accessible. The next important structure to appear was the facial vein, which was liberated and pulled downward out of the way. Some small glands were then dissected out, and by working upward the digastric tendon was at last brought to view with the hypoglossal nerve following its lower border. Even then the artery was not visible, and its pulsations had to be felt for in order to expose it.

In ligating the external carotid artery it is important to avoid including the ramus descendens of the hypoglossus nerve in the knot.

The retractors used in holding open the wound should draw the facial and superior thyroid veins downward, the internal carotid artery and internal jugular vein outward, while the digastric muscle, hypoglossal nerve and submaxillary gland are pulled upward out of the way.

A common impression prevails that ligation of the external carotid artery is an easy matter. This is not always the case, although the operation is sufficiently easy so that any laryngologist can acquire the technic by a number of operations on the cadaver. The operative field is circumscribed by the submaxillary gland, facial vein, digastric muscle, hypoglossal nerve and larynx, so that there is little room, and no matter how long the skin incision be made, the work must still be done in the deep, small area confined by the structures mentioned and filled with important nerves and vessels.

Palpation is a great aid in finding the vessel, if the pulse be strong enough to make its pulsations evident. The operation, except in children, may always be done under cocaine anaesthesia.

The technic proposed by Dr. Jackson is to expose the common carotid first and then follow up to the bifurcation, and ligate between the superior thyroid and the lingual, bearing in mind the anomalous distribution that sometimes obtains, the giving off

of these arteries from the common carotid, and the danger of ligating the common carotid. In this case the difficulty of breathing from the presence of the large tumor made it necessary to follow the plan I have indicated in my report.

I am of Dr. Jackson's opinion that external carotid ligation is something to which laryngologists should resort more frequently in the removal of growths such as this, and hereafter I shall not feel quite safe in doing these operations without it.

After the ligation had been completed, injections of adrenalin (1 to 1,000) and a 2% solution of cocaine, equal parts, were freely made into the base of the tumor through the outward part of the anterior pillar. I first freed the growth as thoroughly as possible from the anterior pillar, with a right angle knife, bent on the flat, with sharp cutting edges and point. As there was no bleeding, I did this boldly, both anteriorly and posteriorly, and then loosened it above as thoroughly as possible, so that I had the growth very well enucleated above before beginning to excise the tumor. As the size of the growth interfered a good deal with the patient's breathing, and he retched so constantly, it was not possible to remove it in one piece, so I dissected it out in fragments. In the upper zone there was no hemorrhage, and I succeeded in almost completely removing the growth from the lower without much bleeding. In clearing up this, however, I was anxious to remove every suspicious looking mass, and this was done by seizing it with notched forceps, drawing it outward and excising only the part thus seized. A small fragment was thus removed at the central and posterior part of the lower zone of the space, and a very severe hemorrhage took place immediately. Compression was resorted to at once, and I saturated a piece of gauze with the adrenalin, and this was held firmly into the bleeding point. The patient fainted, very speedily, becoming very pale and almost pulseless, when, to our relief, the bleeding ceased. He was immediately put to bed in a sitting posture and strict quiet enjoined. In about two hours he wanted to lie down, and in doing so another hemorrhage took place, but it did not recur afterward. He rallied well, and was soon able to sit up. A ligature abscess was the only complication of the external wound, and the site of the tumor soon cicatrized over nicely. I

found, however, that the interrupted operation had left a mass of the growth in the upper angle. I removed this at the office on December 26, cutting deep down to muscular tissue and leaving no suspicious looking part.

I then felt that the operation was satisfactorily radical. It created very little pain, except once, when I found it necessary to cut through and remove a part of the posterior pillar, and then the pain was very severe, but only as the excision was being made. He left the hospital on December 29 for his home, and in excellent condition, with the wound well and firmly cicatrized, and pulse and temperature normal. I placed the excised tissue in the hands of Professor H. Gideon Wells, at the University of Chicago laboratories, and he furnished me an excellent microscopical study. He reports:

No traces of normal tonsillar tissue can be detected in any of the sections cut from various parts of the growth, which is of uniform structure in all parts examined. It consists essentially of a solid mass of cells, which are everywhere much alike, forming a solid cellular growth in which there is a slight but very imperfect tendency to alveolar arrangement. The stroma is not abundant. It consists of narrow bands of mature fibrous tissue about the larger vessels, from which slender anastomosing processes pass out at irregular intervals, tending to subdivide the tumor into imperfect alveoli. By special connective tissue stains (Mallory's Anilin Blue and Van Gieson's Stain) fine fibrils may be seen occasionally within these atypical alveoli, among the tumor cells, but they do not form a distinct reticular structure as in a typical lymphosarcoma. In the larger connective tissue bands are numerous small deeply staining nuclei, "lymphoid cells;" also, very rarely, an occasional eosinophile cell. The blood vessels are not conspicuously abundant, but all the connective tissue bands exist as the supportive structure of small vessels, which always have a distinct endothelial lining and a connective tissue adventitia; no vessels can be found within the alveoli, and there is no free blood and no blood pigment. The larger vessels have a distinct media. It is apparent that this vascular arrangement is adequate for the nourishment of the tumor, since nowhere can there be found the slightest evidence of necrosis, or even of less severe



retrogressive changes. There is also a total lack of evidence of hemorrhage, either old or recent, and there is no inflammatory reaction whatever, no leucocytes at all being found either free or in clumps.

The free surface of the tumor is in part covered by the stratified epithelium with occasional short processes running down into the deeper tissues, characteristic of the surface of the normal

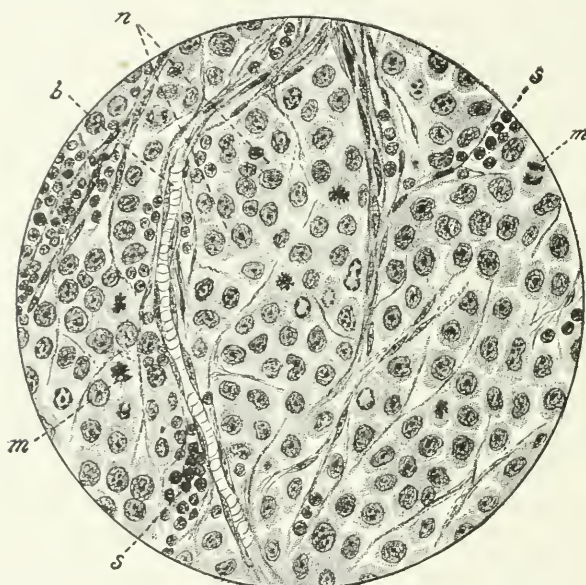


Fig. 2.

Alveolar Sarcoma of the Tonsil. The drawing shows a typical field, illustrating the alveolar structure, with strands of stroma running between the individual cells. Composite drawing from sections stained with hematoxylin and eosin, Mallory's and Van Gieson's connective tissue stains. (The artist has somewhat exaggerated the amount of cytoplasm about the nuclei.) (m) Mitotic figures; (s) cellular stroma; (b) capillary; (n) typical nest of tumor cells.

tonsil. This epithelial layer rests upon a layer of loose connective tissue in which occasional isolated tumor cells are seen, but they do not penetrate the epithelium, except in a few places where an island of tumor cells occurs free in the epithelial coat. Much of the free surface is, however, devoid of epithelium, which seems to have been destroyed by a gradual pressure atrophy rather than

by necrosis and ulceration, which is shown by the gradual thinning out of the epithelial cells and the absence of inflammatory reaction. The denuded surface of the tumor is remarkable in the absence of evidences of infection and inflammation; it is covered by a very thin layer of hyalin fibrin mingled with a small amount of necrotic cells and stroma, and enclosing a small number of leucocytes; the total thickness of this surface layer of degeneration is, however, not much greater than that of the normal epithelial covering. Just beneath the surface the tumor tissue shows a relatively large proportion of stroma, and some of the small vessels are thrombosed.

The cells of which the tumor growth itself is composed are of very uniform nature throughout the tumor. They are cells of medium size, having a nucleus about twice the diameter of the small lymphoid nucleus, and a considerable amount of cytoplasm, which in this poorly hardened tissue is quite granular. The shape of the cells is entirely dependent upon the pressure of the surrounding cells, therefore they are polyhedral. The nuclei are of circular outline when not modified by pressure, and of a rather vesicular type in the mature cells, having a distinct nuclear wall, one to three deeply staining spherules, and a very small amount of chromatin as granules or threads. Younger cells are more rich in chromatin. Rarely a cell is found with two nuclei, but there is no formation of multinuclear giant cells, or of cells with a single giant nucleus. Karyokinetic figures are very abundant, frequently half a dozen being seen in a single field (1-12th immersion lens); most of the figures are regular, but occasionally heterotype mitoses (characteristic of malignant tumors) may be seen. There are a very few cells showing disintegrated and fragmented nuclei, often associated with a hyalin transformation of the cytoplasm. Eosiniphile and plasma cells are not present among the tumor cells, although they are very occasionally found in the stroma.

The diagnosis is round cell sarcoma, of alveolar type. The vesicular nuclei, the considerable cytoplasm and the alveolar arrangement suggest carcinoma. However, against this diagnosis are (1) the intimate relation of the cells to the stroma, which almost forms a reticular network within the imperfect alveoli;

(2) the homogeneity of the structure; (3) the small proportion of mature stroma; (4) the absence of any attempt at formation of tubules or ducts; (5) the total absence of necrosis and inflammatory reaction, which indicates a close relation between the tumor cells and the blood vessels. On account of the abundance of mitotic figures it is evident that this tumor is exceptionally malignant.

After I had received this report of the pathologist, on January 29, thirty-four days after the last operation, the patient presented himself again for examination, and I found a confirmation of the exceptionally malignant character of the growth, clinically, in a tumor in a new site upon the lateral wall of the pharynx, behind the left posterior pillar, about 5 centimeters in length,  $2\frac{1}{2}$  centimeters in width and a centimeter in thickness, extending upward behind the soft palate and downward to the vestibule of the larynx. As there were no symptoms of obstruction to breathing at this time or to be apprehended in the immediate future, I refused to operate, promising the patient to do so if there should be danger of his choking to death hereafter. I then advised him to return to his home city and submit himself to treatment by the X-Ray, and, if possible, to take the Coley Toxins. I have not seen the patient since that time, but his physician informs me that he has been having X-Ray treatment, with apparent benefit, and is in good health at the present time. In view of the laboratory findings and the clinical history of the case, however, I am not optimistic as to the eventual cure of the disease.

### *Discussion.*

Dr. D. Bryson Delavan: Dr. Dawbarn has suggested a method of treatment for this class of cases, namely, ligation of the carotid. While it does not succeed in carcinoma, it has met with considerable success in sarcoma. It is quite possible that before proceeding to the extirpation of such a tumor ligation of the carotid would be the better procedure. As is known, starvation of such a tumor has resulted in cure, and even if it should not in such a case as the one at hand, we still have the alternative of doing the more extensive operation. The main points in reference to ligation of the vessels in tonsillar hemorrhage will be found in an

article on the tonsil published by me in Buck's Reference Handbook of the Medical Sciences about fifteen years ago.

Dr. J. Edwin Rhodes: It seems to me that in connection with these growths the collateral circulation would be so quickly re-established that we could not hope for very much from this procedure in the inhibition of growth. This infection travels by the blood vessels, and we could hardly expect the starvation to be complete for any length of time. The call for immediate operation in this case was on account of the size of the tumor, and it proved to be all that could be desired in the relief afforded.

Dr. B. Alexander Randall: I would like to say that after such a ligation as just mentioned I found entire absence of pulsation in the temporal for at least sixteen days. This was certainly a surprise to me, and will give some idea as to how long it may be before collateral circulation would be re-established in such cases.

## THE INFLUENCE OF ADRENALIN IN THE CAUSATION OF ARTERIO-SCLEROSIS.

BY FREDERICK E. HOPKINS, M.D.

Since the publication of Josué's (1) experiments in 1903 a considerable literature has appeared upon the subject of adrenalin as a cause of arterio-sclerosis. The marked uniformity in the results of the experiments reported during the four or five succeeding years apparently established the case against adrenalin. In fact, judged by the influence upon the minds of many who read these reports or who saw references to them, the case was proven, since they hesitated to employ the agent or countenance its use. Later investigations have cast much doubt if they have not entirely disproven the conclusions of these early observers; and that these later and more accurate reports may have as wide a dissemination as possible I am led to ask your discussion of the subject, since your experience also with the use of adrenalin will be reassuring. Adrenalin has proven so useful and valuable an agent in our hands, both in allaying acute congestions and in affording a bloodless field for operation—not to mention other merits—that any suggestions of its responsibility in causing grave remote effects like arterio-sclerosis arouse in one a feeling of resentment akin to that experienced when reflections are cast upon the integrity of an old and tried friend.

Having no original work to offer, this presentation shall be brief, yet that it may have somewhat of symmetry and historical sequence, reference will be made to some of the published observations, in the order of their appearance.

The attempt to produce arterio-sclerosis experimentally is not new, and a variety of methods have been employed to that end; such as the direct mechanical injury of the vessel walls, the intravenous injections of micro-organisms or their toxins (2) the subcutaneous injections of nicotine, barium chloride, digitalin; (3) the administration of lead salts and tobacco, etc., as well as the use of adrenalin. Since 1903 experiments have practically been con-

fined to the latter, owing to the interest aroused by the report of Jores, of the Bonn Pathological Institute, who fed animals on adrenalin extract; and the reports already referred to, published by Josué the same year, who made use of adrenalin hypodermically, the injections being repeated daily for varying periods.

Josué's method was adopted by the many experimenters in this country and Europe. Rabbits were used for the experiments, since the vessels of other animals were found resistant to degenerative action.

The reports of the findings of the various workers, including Miller (2), Pearce and Stanton (4) in this country, Oscar Klotz (3) in Canada, Erb in Germany, Pic and Bonnamour in France, and many others, show a remarkable unanimity, and a quotation from one will serve for all. All agree that the smaller vessels are not involved, and that the media is the layer exhibiting the maximum degree of degeneration. Miles (5) says of the gross appearance of the lesions: "They were, in general, grayish white in color, more or less opaque, circular to angular in outline, or sometimes appearing as short lines running lengthwise with the vessel, usually raised above the surface as the vessel was laid open, and varying in size from a pinpoint to several millimetres in diameter. The larger areas were often somewhat uneven both in elevation and outline. Those larger areas of calcification often showed tendencies to aneurismal dilations. Calcification could usually be recognized grossly in the larger lesions, and often in the smaller ones, by the more opaque white appearance of the lesion and by the gritty character when touched with a needle or scalpel. The lesions were more frequently found in the thoracic aorta, especially about the mouths of the branching vessels, often as an aggregation of pinpoint areas, sometimes as larger plaques. They were frequently observed in the abdominal aorta and less frequently in the iliac arteries. Here also there was a greater tendency for them to appear about the mouths of the branching vessels, as in the thoracic region."

But he says in the same connection that the gross appearance of the lesions and their location are the same in the injected and in the non-injected animals. Also that the result of no individual experiment can be predicted with certainty, no matter whether the dose administered be large or small, the duration of the treat-



ment long or short, and he mentions the fact that the vascular system of normal rabbits has not been sufficiently investigated. In the same paper he publishes the statistics gathered by himself and Johnstone. Sixty-one rabbits received injections three times daily in amounts from  $\frac{1}{2}$  m. to 15 m. at a dose, and for varying periods, the total quantity administered to each varying from 3 m. to 1350 m. of a 1-1000 solution. The most extensive lesion was found in the rabbit receiving 5 m. doses, 150 m. in all, while many others receiving four to eight times that total showed absolutely no lesion. Of the sixty-one thus treated, seventeen, or 27.86%, showed lesions of the larger vessels on post mortem examination.

They then examined fifty-eight "normal" rabbits, and twenty of these, or 34.48%, were found to have arterial lesions of exactly the same character as the animals treated. These animals were from the same sources, and so far as possible kept under the same conditions as the rabbits experimented upon.

The foregoing report of Miles and Johnstone, together with the admitted fact that it is only in rabbits that vascular lesions have been found, and that it is questionable if the lesions in these animals are identical with arterio-sclerosis in man, together with the statements of all experimenters that they find it difficult if not impossible to produce lesions in young rabbits; that lesions are reported after only two injections (Braun reported by Miller) (2) and calcification as early as the fifth day after beginning treatment, that Klotz (3) found exactly the same results produced by barium chloride, and he states that the similarity is so striking that neither macroscopically nor microscopically can the lesions be distinguished from one another—all this variable and uncertain testimony, I say, throws doubt upon the assumption that adrenalin is the cause of the vascular lesions found after experimentation, and leads to the conclusion that most if not all the changes in the large vessels existed before the experiments were begun.

Practically this same conclusion is reached by Adler, who read an exhaustive paper upon the subject before the New York Academy of Medicine on March 19, 1908—a paper soon to appear in the "American Journal of the Medical Sciences." On the same occasion Harlow Brooks read a paper to be published duly in

the "Archives of Medicine," and he reported a case which goes farther toward proving the innocence of adrenalin under reasonable use than can any amount of experimentation upon animals or any theorizing to prove the contrary.

Doctor Brooks' case was that of a woman thirty-five years old, who was under observation in a hospital for three years. She suffered from asthma to such a degree as to be totally disabled. No ordinary remedy afforded her relief. It was found, long after exhausting the usual list, that hypodermics of adrenalin overcame the spasm and brought relief for a longer or shorter interval. In view of the experimental evidence against adrenalin, its employment was sanctioned only as a last and desperate resort and because it was the only treatment which gave any relief. This patient received at times as much as 120 m. at a dose, and during the three years of its use forty-four one-ounce bottles of the usual 1-1000 solution were given hypodermically. She died after three years, but not from a cause attributable to the adrenalin, and the examination of her arteries revealed only the changes in vessel walls to be expected at her age. Nothing unusual was found macroscopically, but microscopically in the median coat of the aorta were a few scattered points of degeneration, not sufficient to materially impair the strength of the vessel walls, and the patient might apparently have endured the hypodermics of adrenalin many years longer, had not her life been taken by other disease than that of her arteries.

In a word, then, the unreliability of animal experimentation to date, and the proven innocence of the large doses continuously administered in Doctor Brooks' case, give ample ground for reassurance in the reasonable use of adrenalin.

### *References.*

1. Quoted by Miller, American Journal Medical Sciences, April, 1907.
2. Miller, American Journal Medical Sciences, April, 1907.
3. Oskar Klotz, British Medical Journal, Dec. 22, 1906.
4. Pearce and Stanton, Albany Medical Annals, February, 1906.
5. Miles and Johnstone, Journal American Medical Association, Oct. 5, 1907.

*Discussion.*

Dr. C. F. Theisen: Recent experiments show that atheroma of the vessels is only usual when the adrenalin is injected intravenously.

Dr. Richard M. Pearce, of the Albany Bender Laboratory, has demonstrated this in his investigations upon animals.

Dr. H. P. Mosher: About two years ago I became interested in this subject and attempted in a rather amateurish way some experiments on animals. For these six guinea pigs were used. Solutions of adrenalin were put in the nose, and the dosage and the length of the time during which the drug was given were varied. The longest time was six weeks. The object was to see if the adrenalin, when absorbed by unbroken nasal mucous membrane, would give changes in the aorta. In none of the cases did the aorta show any changes.

Dr. B. A. Randall: Some six or eight years ago I employed the adrenal in a case of thyroid swelling with the general symptoms of exophthalmic goitre in the commencing stages. It gave immediate and undoubted relief to the symptoms. This was a case of a somewhat neurotic trained nurse, and she has continued it for these good many years, and rather frequently without any untoward results. I have also found a great deal of value from the adrenal in cases of otitic vertigo, not of the fulminating Menière type, but of the still severe kind which seems to be aural in origin. I have used it in these cases quite freely and for long periods of time, although trying to use it in as small an amount as possible and to taper off as soon as the patient has had a reasonable degree of relief. I have had quite a number taking this without the slightest suggestion of trouble. I always prefer the desiccated adrenal in 2 or 3 grain doses, using the adrenalin chloride only as an emergency preparation which could be taken instantly at the first threat of trouble, and the other as a slower and more persistent measure to forestall, if possible. Thus far I have certainly had no reason to regret its employment, however free and long-continued it has been.

REPORT OF A CASE OF REMOVAL OF A FOREIGN  
BODY, A GRAIN OF CORN, FROM THE TRACHEA  
OF A CHILD SIX YEARS OLD.

BY JOSEPH H. BRYAN, M.D.

The following is a short account of a successful case of the removal of a foreign body from the trachea of a child by tracheotomy, after an unsuccessful attempt at tracheoscopy had been made.

Paul B., aged 6 years, was admitted to the Episcopal Eye, Ear and Throat Hospital, January 22, 1908, giving the following history:

Several days before his admission to the hospital he was playing horse, and while in the act of munching corn from the cob, a large grain was drawn through the larynx into the trachea. He was seized with violent coughing and dyspnoea, but he soon quieted down to such an extent that when his family physician examined him he could not find any difficulty with the breathing, and concluded that the grain of corn had not passed into the trachea. During the night, however, he was seized with several very severe attacks of dyspnoea and coughing which continued through the following day, when the breathing became quiet again.

He was then brought to Washington and admitted to my service in the Eye, Ear and Throat Hospital, January 22.

On admission I made a careful examination of the child and found him suffering from a large hypertrophy of the post-nasal lymphoid tissue and hypertrophy of the faucial tonsils. His color was natural, his voice thick and somewhat husky, which could be accounted for by the enlarged tonsils and the adenoids. The respirations were somewhat quickened, but not more so than one would expect in a timid child when in the presence of strangers. Auscultation showed large and small mucous rales over both sides of the chest anteriorly and posteriorly. The air apparently entered the lungs freely.

An X-ray picture was made, but it did not reveal the presence of a foreign body in the air passages. He was kept in the hospital under close observation for three days, but as no dyspnoea

was observed in this time he was allowed to return to his home in Maryland.

On the morning of January 30, he returned to the hospital suffering from intense dyspnoea and marked cyanosis. An examination of the lungs at this time showed a complete absence of the respiratory sounds on the right side, while mucous rales were heard on the left side both anteriorly and posteriorly. When I saw him about two and a half hours after his admission, these physical signs had completely changed. The air was found to be passing freely into all parts of the lungs. He was still breathing with great difficulty and complaining of intense pain in the subglottic region. Evidently the foreign body which had been engaged in the large bronchus on the right side had been dislodged and had been carried into the upper part of the trachea, where it was caught by the swollen mucus membrane.

I had him sent to the operating room, and placed under chloroform anaesthesia with a view to trying to remove the body through a Killian tracheal tube. I made several ineffectual efforts to pass the small tube through the larynx, but owing to the swollen condition of the mucus membrane lining the larynx, I found this was impossible. Just at that moment the child ceased to breathe and became very cyanotic. I then did a rapid tracheotomy, expecting to pass the tube through the tracheal opening and remove the foreign body in this way. As soon as the trachea was opened the child commenced to breathe freely again, and a grain of corn was seen to pass the tracheal opening on its way to the lower trachea. It made several such excursions before I was able to grasp it with my forceps and remove it. (Specimen submitted.) A tracheotomy tube was then introduced into the tracheal opening. He rallied quickly from the operation, and as all obstruction to the respiration ceased after the removal of the foreign body, he was sent to his bed.

When the grain of corn was removed it was found to be an unusually large one and very much swollen from the moisture absorbed since its introduction into the trachea.

The tracheotomy tube was removed on the fourth day and the wound allowed to close. Some laryngeal stridor remained for at least twenty-four hours longer, when it ceased. The patient

made an uninterrupted recovery and was discharged from the hospital on the ninth day.

This case is interesting and shows it is not always best to persist in passing the Killian tubes through a swollen larynx after several ineffectual attempts have been made. Had I continued to reach the trachea in this way, I am sure I would have done irreparable injury to the soft parts, and had I succeeded in introducing the tube through the larynx it would have been impossible to remove a grain of corn of this size through the tube, but with a large tube introduced through a tracheal opening I might have been able to reach the foreign body and remove it. Fortunately the introduction of the tube through the tracheal opening in this case was not necessary, as the above history shows.



PERSONAL EXPERIENCE IN THE USE OF THE BRONCHOSCOPE, ŒSOPHAGOSCOPE AND GASTROSCOPE, WITH REPORTS OF FOURTEEN CASES.

BY THOMAS H. HALSTED, M. D.

So recent and so valuable is the art of bronchoscopy, œsophagoscopy and gastroscopy that it seemed to the writer that a report, necessarily brief, of the work which he has done along these lines should be of value, because at this stage of development the cumulative experience of various workers will be helpful in suggesting, at least, to what extent this method can be applied with safety and benefit. There will be no reference in this paper to the history of the development of this art, though it may be pertinent to say that a number of men in this country, especially Coolidge, Ingals, Mosher, Mayer, Einhorn, Jackson and Boyce, have done most valuable work, much of it original, in bringing this method to its present state of practical utility.

The work described and cases reported in this paper cover a period of but seven months, and include eighteen operations on fourteen patients, fourteen times under general anaesthesia, three times under cocaine, once without any anaesthetic. These eighteen operations were all done either as means of treatment or of diagnosis and do not include four esophagoscopies on four young and willing physicians under cocaine, made for experimental purposes, nor a number of direct laryngoscopic examinations made in the office for the purpose of diagnosis as well as to acquire skill in technique. Other experimental and exceedingly valuable work has been on the dog, usually in association with Professor Knowlton, of the Physiological Department of Syracuse Medical College, and once, the most valuable experience of all, under the direction and instruction of the master of this art in America, Dr. Chevalier Jackson and his assistant, Dr. Boyce, to whom I cannot sufficiently express my deep sense of gratitude for and appreciation of their generous instruction. I have been

assisted in my operations mainly by Drs. J. C. Palmer and Adalaide Dutcher, and by Dr. J. J. Buettner as anaesthetist. Assistants should be specially trained, as the proper performance of their work is essential to a good result, and I have been fortunate in having good assistants.

The instruments used are those known as Jackson's, having the light at the distal end of the tube, the current supplied by a double dry cell battery, though in the two last cases a transformer recently perfected by the Electro-Surgical Instrument Co., using the city alternating current, was employed. Jackson's instruments, which I have, consist of the separable speculum or spatula for direct laryngoscopy, two bronchoscopic tubes of 5 and 7 mm. in diameter for upper bronchoscopy, two tracheoscopes for tracheoscopy and lower bronchoscopy, two œsophagoscopes of 7 and 10 mm. in diameter, and one gastroscope, 10 mm. by 80 in length for the examination of the stomach. The small lamp, no larger than a grain of wheat, has given me surprisingly little trouble, and the blurring of the light by mucus and secretions covering it is easily cleared by the use of the suction pump, swabbing or the removal of the light carrier and wiping the lamp; this latter procedure requiring but a fraction of a minute. Among the many advantages of Jackson's instruments over Killian's, in which the light is thrown from the outside down the tube, is the fact that physicians other than the operator can look down the tube and note the findings as well as the operator himself can.

Accessory instruments consist of probes, hooks, cannula forceps of various lengths, Mosher's alligator forceps, Coolidge's gauze sponge carriers, suction pump and suction canulas for removal of fluids and secretions. For special cases, I had made for myself a spud or hoe-like curette, used in my first case, a very long galvano-cautery electrode, intended to be used in a case of specific web stricture of the larynx. There is a demand rapidly being met for a new and complete armamentarium of straight instruments to meet the various conditions that are being encountered.

The general anaesthetic used in most cases was ether, usually preceded by somnoform, with atropine to lessen bronchial secretions. While in certain cases cocaine might be sufficient, yet

in œsophagoscopy and gastroscopy the retching against the rigid tube might be disastrous locally by producing perforation or even the rupture of a cerebral blood vessel. In the case of a physician on whom I did œsophagoscopy under cocaine there were produced sub-conjunctival hemorrhages through the violent retching. Cocaine is sufficient for direct laryngoscopy in most cases, but in some cases, especially operative, ether is preferable.

Ether was used in all cases where upper bronchoscopy was done, excepting once, when I did it quite satisfactorily under cocaine, and have no doubt it will often be sufficient, as indeed Killian and others have repeatedly shown. In the one case in which I examined the bronchus under cocaine I had previously examined the same patient under ether and consequently knew what to look for. In her case the ether examination was much the most satisfactory. Rectal anaesthesia should here be the ideal method, but I have had no experience with it.

The patient, excepting for direct laryngoscopy under cocaine, is placed on the back on the table with shoulders projecting and head hanging over and supported in the Boyce position. The correct position of the head and its proper holding are absolutely essential to the proper introduction of the instruments, and an assistant must be trained to do this. In some cases the mouth is best gagged with a tailor's thimble on the assistant's index finger and introduced between the teeth on the left side. In many cases too wide gagging of the mouth interferes seriously with breathing, and in some cases the gag is best dispensed with, but to protect the thin tubular instrument from being bitten and dented the thimble, at least, should be constantly held between the teeth. Ferguson's mouth gag was the one used when a gag was employed.

In one case of bronchoscopy, during the act of freeing the bronchus from a plug of mucus the small gauze sponge became detached from the carrier and was left in the bronchus until a forceps, quickly introduced, removed it. Since that accident, all sponges have had long white threads attached, the thread being loosely wrapped around the carrier. So delicate and so long are the forceps and the cotton carriers that the utmost care must be exercised to see that the forceps jaws are properly tempered. Several of mine have been broken while being handled, for-

unately not while inside the patient. In working on the larynx or bronchi one should have instruments ready for an immediate tracheotomy.

While all the cases were of great interest to the writer, and all of them warrant an extended report, yet to give a minute report of all would occupy too much time and space. The majority will be given simply in brief outline, some in greater detail.

Case I.—Œsophagoscopy (ether). Penny lodged for four and one half years at the cardiac end of the œsophagus in a five-year-old boy. Obstruction always supposed to be due to congenital stricture. X-ray examination revealed foreign body. Operation under fluroscope unsuccessful. Successful removal of penny by œsophagoscopy under ether. Recovery.

Donald C., aged five years, residing in Cortland, N. Y., was brought to me by his parents on October 11, 1907, to have bougies passed to dilate a stricture of the œsophagus which he was supposed to have had from birth. He had considerable difficulty from birth in keeping down milk. Between six months and two years of age had two or three attacks of bronchitis and wheezed a great deal of the time, the doctor regarding it as asthma. At two years he had a severe attack of dysentery. The following winter coughed and wheezed considerably, chest filling up when he ate, and relieved on regurgitating his food. At about two and a half years he began having severe pain in the stomach, at times lasting several hours. Months would elapse without these attacks, and again they would continue for several weeks. Had been treated for stomach trouble up to this time.

One consultant said there was a narrowing of the pyloric end of the stomach and suggested gastro-jejunoscopy. When four years of age he could tell about his swallowing, knowing as soon as he swallowed whether or not the food would be kept down or regurgitated. He could retain liquids provided he drank them before eating any semi-solid food. Sometimes he would go two or three days without retaining even a drink of water, perhaps he would throw up a piece of food he had eaten several days previous, when he would get relief and could drink nearly a quart of milk, or other liquid. Was constantly hungry, asking at times every five minutes for something to eat.

A few weeks before bringing the boy to me the parents had taken him to a well-known physician in Buffalo, who made a very careful examination of him, including a chemical exami-

ination of the regurgitated food, which he said had not entered the stomach, but had evidently been retained in a pouch. His diagnosis was a congenital stricture of the œsophagus, and advised that he be placed in the care of a laryngologist for the passage of œsophageal bougies, which was the reason for his being brought to me. A congenital stricture or narrowing of the œsophagus, near the cardiac end of the stomach, had been the almost unanimous diagnosis of the greater number of physicians who attended him or who saw him in consultation at home or in neighboring cities.

He was undersized, rather listless and peevish, but in fairly

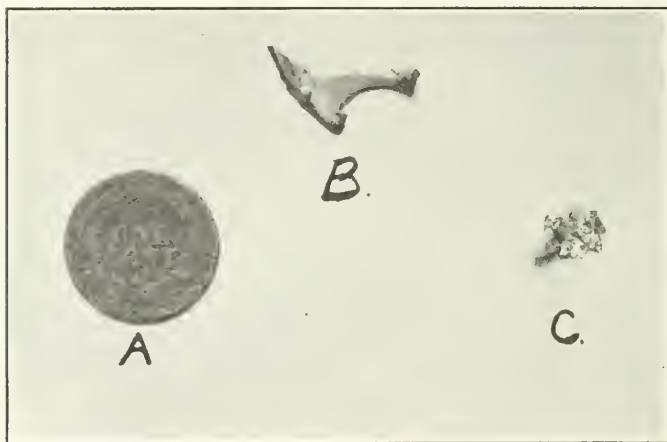


Fig. 1.

A. Penny removed from œsophagus of five-year-old boy, imbedded four and a half years; Œsophagoscopy. B. Duck bone, removed from right bronchus; Upper Bronchoscopy. C. Fish bone (fin), removed from trachea of eight-months-old infant; Laryngoscopy, Tracheotomy and Tracheoscopy.

good flesh, teeth badly decayed, nothing abnormal about the throat. I gave him a graham cracker which he readily ate with half a glass of water, and at once began to wheeze. Within two or three minutes he asked for a basin, when the food was regurgitated, not vomited, and the wheezing ceased.

My diagnosis was a stricture at the lower end of the esophagus, with a dilatation or pouch above it, and advised that a radiograph



be taken after bismuth and milk had been swallowed, in order to localize, if possible, the stricture and dilatation. That afternoon Dr. E. C. Coon made an examination with the X-ray. We first gave him four ounces of milk containing one ounce each of sub-nitrate of bismuth and gum acacia. He sipped this, taking twenty minutes to do it, but said it was all going down, and it was retained.

On developing the plate, a round body the size of a ten-cent piece or penny was revealed opposite the fourth or fifth dorsal vertebrae. The parents were apprised of this and they returned with the child on October 16 (five days later), when a lateral exposure was made to make sure the foreign body was a round flat body and not a spherical one, such as a marble. This skiagraph showed it to be a flat body, apparently lying in or against the posterior wall of the gullet.

The child was put under ether, and under the guidance of the fluoroscope I introduced into the œsophagus a long Mosher's alligator forceps encased in a soft rubber tubing, the child being in the recumbent position, with down hanging head. The fluoroscope showed plainly the progress of the forceps, and when it reached the foreign body the tubing was pulled off, leaving the jaws of the forceps free. The distance from the teeth to the coin was eight and one-half inches, or about one inch from the cardiac end of the stomach. The forceps was opened when down to the coin, but notwithstanding repeated attempts carefully made, the foreign body could neither be grasped nor felt. We concluded, as we had at first feared might be the case, that the coin was probably encysted in the posterior wall of the œsophagus. The child remained one night at the hospital, and the next morning returned home in good condition. I now ordered a set of Jackson's œsophagoscopes and bronchoscopes and during the next three weeks familiarized myself with their use as well as I could.

### ŒSOPHAGOSCOPY.

On November 6 the child entered the Women's and Children's Hospital. No food or liquids were allowed for eighteen hours prior to the operation. Atropine 1-150 was given and ether administered by Dr. J. J. Buettner.



I was assisted by Drs. Dutcher, Bogart and Coon. The child was placed in the dorsal position, with head hanging down over the table, mouth held open with Ferguson's mouth gag. The œsophagoscope, 40 cm. long by 7 mm., was then introduced and a careful inspection of the gullet made, gradually extending the tube downward toward the cardia. The wall of the œsophagus, pinkish in color, was beautifully illuminated, and at the lower end, about seven inches from the teeth, entered a distinctly enlarged cavity.

After more than an hour's searching and using the probe to feel any metallic substance, but without result, I introduced through the tube a right angled curette or spud like instrument, which I had had made for the purpose, and gently curetted or scraped the posterior wall of the œsophagus at a distance of about eight and one-half inches from the teeth, corresponding with the location we had made on the previous fluroscopic examination. This was done very gently and carefully. On removing the spud and drying the surface of the few drops of blood, the edge of a coin was clearly seen. The long tube forceps was now introduced and on the second introduction the forceps caught the coin firmly and penny, tube and forceps were all removed together.

He made an uneventful recovery, not regurgitating any food after the second or third day, though ten days after the operation he had an attack of acute indigestion. There began at once an improvement in his swallowing, which has steadily continued, although now six months since the removal of the foreign body the food swallowed is sometimes returned without apparently entering the stomach. He has gained greatly in his general condition, he eats regularly and seldom complains of being hungry, or wanting food between meals. Whether a permanent stricture at the cardiac end of the stomach will remain, time alone will tell, but it is quite possible that as time goes on the thickening may gradually lessen and disappear, and the pouch become obliterated.

The penny had become absorbed to such an extent that it lost 25 per cent in weight, is thinner than a ten-cent piece, not more than two-thirds of its normal thickness, and in diameter is midway between a ten-cent piece and a penny. It had lodged in the posterior wall of the cardiac end of the œsophagus, probably when the child was six months old, possibly earlier, and had become im-

bedded or encysted, and had been there for at least four and a half years. At the rate of absorption that had been going on, had the child lived, the penny might have been absorbed in twenty years. The X-Ray was of the greatest value, in fact, without it it is probable the cause of the obstruction would not have been revealed, because the œsophagoscope failed to discover it until the wall covering it was removed.



Fig. 2.

Penny from cardiac end of œsophagus in posterior wall; patient four and one-half years old; the X-ray bulb having been too low, has thrown the shadow of the penny higher than it was actually found.

Case II.—Direct Laryngoscopy and Tracheoscopy (chloroform). Foreign body, fin of a fish, lodged for two weeks in the

trachea of an eight-months-old infant. Direct laryngoscopy, tracheotomy and tracheoscopy under chloroform. Removal of foreign body.

I was called in consultation with Dr. Hotaling on December 10 to see this baby, with a view to possible intubation, though Dr. Hotaling felt confident that diphtheria was not the cause of the dyspnoea from which the baby had been suffering for nearly a week. Epigastric and episternal recessions were marked, hoarseness on crying, no fever, no pseudo-membrane in throat. The dyspnoea was not urgent. After a careful examination, a diagnosis of probable foreign body in larynx or trachea was made. The parents were urged to take the child to the Women's and Children's Hospital, which was done. An X-ray examination by Dr. Coon was negative. A culture was negative. Steam inhalations were employed, and gave some relief. On December 12, 1907, two days later, the parents consented to an examination and operation if necessary under general anaesthesia.

Operation: Preparations for an immediate tracheotomy were made. The child was given chloroform, and after much difficulty, because of its age (eight months), its undersize (weighing but fifteen pounds), my own inexperience (this being my first attempt to do a direct laryngoscopy), a beautiful view of the larynx through the separable speculum was obtained. The cords were seen perfectly, and beyond the chink of the glottis in the trachea was clearly seen a foreign body, located about an inch below the cords and seemingly attached to the right side of the trachea, looking much like a loose piece of cartilage. Several times during the examination the dyspnoea became so threatening, the heart so weak, pulse so rapid, and at times obliterated, that I was on the point of opening the trachea, when the breathing would clear up, pulse improve, and the examination and attempt to remove the foreign body through the mouth could be continued. I was unable to introduce between the cords the smallest bronchoscope, the glottic opening being so small, but introduced both the cannula forceps and Mosher's alligator forceps several times, and attempted to grasp the foreign body, but always unsuccessfully. Finally the breathing suddenly became so bad that I did an immediate tracheotomy, with relief to the dyspnoea, but I could not

find the foreign body until the small tracheoscopic tube was carried through the wound into the windpipe, when it was discovered and removed, proving to be the fin of a fish. A tracheal cannula was inserted and worn for five days, at the end of which time it was removed, and the wound promptly healed, the child making an uneventful recovery.

Case III.—Direct Laryngoscopy (ether) for removal of papilloma of vocal cords in boy nineteen years old. Operation successful.

I had previously made a number of unsatisfactory attempts at removal in the old way, under reflected light and cocaine, and later with direct laryngoscopy under cocaine, but the boy had such complete lack of control of himself that not much could be done. By the direct method, under ether, the operation was entirely successful.

Case IV.—Œsophagoscopy (ether). In a neurotic girl of fourteen, for diagnosis of esophageal obstruction. Spasmodic stricture and all symptoms entirely relieved by the examination.

The girl, who was rather anaemic and of a marked neurotic temperament, was referred to me because of inability to swallow solid food for the previous two months. There was no pain, no history of traumatism. The tonsils were enlarged and there was a large mass of adenoids present. These were removed with relief to the nasal obstruction, but not to obstructed deglutition. Two months later, diagnosing the condition as probably a hysterical neurosis, but still uncertain, I advised an œsophageal examination. This was done on December 24, 1907, at St. Joseph's Hospital under ether. The œsophagus, pale pink in color, with no ulceration, no stenosis, was explored throughout, and nothing that seemed abnormal discovered, beyond a very decided fluttering or thumping, at a point half-way down, of the anterior wall of the œsophagus against the œsophagoscope. This was synchronous with the heart beat, and was visible to the eye and felt against the instrument, and even heard outside the body. On passing this particular point the fluttering stopped. Whether this was an abnormality or of pathological significance, I do not know.

It is nearly five months since the examination, and the girl has

had absolutely no difficulty in swallowing solids, as well as liquids, since the time of the operation. I concluded it was a case of hysterical dysphagia in a highly neurotic young girl, though I dislike to use the term hysterical.

Case V.—Direct Laryngoscopy and upper Œsophagoscopy (cocaine) on a girl, for suspected foreign body, which was not present, but had been expelled.

An examination under cocaine was made with the separable speculum of the larynx, lower pharynx, under and upper end of the œsophagus of a girl of twenty, giving a history of having swallowed a pin, which she thought was lodged in the throat. An X-ray by Dr. Burch made prior to my examination was negative. My examination revealed a swelling of the arytenoids, caused possibly by the previous efforts of a physician groping in the dark with forceps to find the pin. The patient remained in the Hospital three days, at the end of which time all swelling and symptoms had disappeared.

Case VI.—Upper Bronchoscopy (ether) for diagnostic purposes. Syphilitic stricture of both bronchi, with ulceration and crusts revealed; Second bronchoscopy two months later under cocaine; Third bronchoscopy one month later under ether, when arygol was applied directly to ulcers in both bronchi.

I was called on January 10th, 1908, in consultation with Dr. Mulherin, to see Mrs. E., aged fifty-five years, because of a dyspnoea of obscure origin. The appointment was for three o'clock, but at one an urgent telephone message came from the family to come as quickly as I could, as the patient was dying. When I reached there I found Dr. Mulherin had preceded me by a few minutes. She was just recovering from an acute attack of dyspnoea. Her forehead was clammy, covered with cold perspiration, hands cold, lips bluish, and breathing labored but not alarmingly severe, as she was now recovering from what had been a very severe attack of suffocation. The voice was clear. Dr. Mulherin had seen her in several similar attacks during the past year, and said they were relieved on the expulsion by coughing of a dark grayish, thick and tenacious mucopurulent discharge, the expulsion of which was made easier by the inhalation of steam, applications of heat externally, and the



swallowing of vaseline. She had been having these attacks every few days for the past two months, and at longer intervals, with less severity, for the past eighteen months.

A laryngoscopic examination showed no laryngeal obstruction, no abductor paralysis, and the trachea, as far as could be seen, showed no obstruction. The difficulty was apparently at the bifurcation of the trachea, or in the mediastinum. There was a fulness of the right lobe of the thyroid with distinct pulsation, some dulness over the upper part of the chest on both sides in front. No bruit could be detected. The diagnosis of mediastinal pressure upon the lower end of the trachea or bronchi was made, and she was sent to St. Joseph's Hospital for further observation and care. Here she came under the care of Dr. Elsner on the medical side and myself. Dr. Coon made several skiographs, some of which were unsatisfactory, but he thought he was able to detect a dilatation of the innominate. Dr. Elsner's opinion was that a mediastinal tumor was causing the dyspnoea. After entering the hospital she had no severe attacks of dyspnoea, though the breathing was labored at all times, more on exertion, but when quiet was not very troublesome. Inhalations of steam were prescribed every two hours.

A bronchoscopic examination was made under ether on January 18th. The separable spatula was introduced down to the larynx, which was free from obstruction, the bronchoscopic tube was then inserted between the cords, discovering no obstruction here until the bifurcation was reached. At the bifurcation of the trachea, as detected both by the eye and the probe, was a mass of cicatricial tissue which extended into the right bronchus on the right side, and seemingly narrowed the left bronchial opening to a narrow slit, through which the large bronchoscopic tube could not pass. An inch below the bifurcation in the right bronchus there was a web of cicatricial tissue encircling the circumference of the bronchus and narrowing the calibre of the tube at least a half. The large bronchoscope was removed and a smaller one inserted and passed through the narrow opening into the left bronchus.

I was able to demonstrate the bronchial strictures and the



cicatricial tissue at the bifurcation to the eight or ten physicians who were present, and who were able to recognize the conditions as clearly as I could. It is possible the left bronchus was narrowed by external pressure as well as by the scar tissue. The diagnosis was syphilitic stricture of both bronchi, with probably some glandular thickening in the mediastinum which might account for the shadows as seen in the radiograph. I was not aware that I had seen this patient before, but learned at the time of the examination that I had treated her at the Dispensary some years previous for some throat affection, the nature of which I had no recollection.

After the examination I looked up the Dispensary records and found I had treated her five years ago for syphilitic (tertiary) ulcer on the posterior wall of the naso-pharynx, and which had promptly healed under the iodides.

Under mercurial inunctions, large doses of the iodides and inhalations of steam, the patient immediately began to improve, and during the three months she was in the hospital had but one serious and threatening attack of dyspnoea. This was relieved on expelling a large mucilaginous semi-solid crust. Two months later, under cocaine at my office, with the patient sitting up, bronchoscopy was again done and the strictures again seen. One month ago, under ether, at the hospital the patient submitted to a third bronchoscopy, this time with a view of local treatment. The strictures were located as before, the ulcerations covered with a hard scab or crust, exactly like the crusts that form on the anterior part of the nasal septum, were removed by forceps and moist gauze sponge and a ten per cent solution of argyrol applied. There was no reaction; she left the hospital in two days, and is very much improved. She is taking one hundred and eighty grains of iodide of potassium daily.

My present plan is to repeat the applications of argyrol under cocaine bronchoscopy, hoping to get rid of the crusts, and when that occurs she will be relieved of her dyspnoea almost entirely, and if not, dilatation of the stricture will follow.

Case VII.—Direct Laryngoscopy under ether in an eight-months-old baby revealed sharp piece of glass imbedded in

larynx. Attempt at removal dislodged it, causing its disappearance but not removal. A second operation refused. Baby in former condition.

The clinical history of this baby, who was a patient of Dr. Werfelman's, was almost exactly that of case 2—and after consultation a diagnosis of foreign body in the larynx was made. An X-Ray by Dr. Coon was negative. On Feb. 5, 1908, the child was received at St. Joseph's Hospital and was etherized. With the separable spatula the larynx was brought into view, and both Dr. Werfelman and I recognized at once a foreign body, looking like a piece of glass about an eighth of an inch long and shaped like the end of a wooden toothpick lodged in or projecting from the surface of the swollen left arytenoid. There was no mistaking it, and its removal seemed simple, but on attempting to grasp it with the forceps it simply changed position and, notwithstanding several attempts at removal, it finally disappeared from sight, either being dislodged and swallowed, or dislodged and entering the lower respiratory tract, or, what seemed most probable, disappeared into the soft tissues and became buried. It seemed wisest to desist and await developments. The child was better for a few days, when the same symptoms recurred, and continue. Unfortunately the parents have changed physicians, and will not allow another operation, hoping that with the advent of warm weather its so-called asthma will clear up. I hope, however, to be able to make another attempt soon.

Case VIII.—This was the removal from a man aged fifty of a papilloma from the lower end of the pharynx, at the entrance to the œsophagus, using the separable speculum, and a long Jarvis snare.

Case IX.—Direct Laryngoscopy (cocaine) for galvano-cautery of syphilitic stricture of the larynx in a woman. Showed impracticability of galvano-cautery, in this case. Intubation.

This patient, a woman of fifty, who has been under my care for two years, required repeated intubations. I had hoped that I might be able to use the galvano-cautery electrode in destroying what had appeared in the laryngeal mirror to be a thin cicatricial web, but under direct laryngoscopy, what seemed a

web was quite a thick, deep mass of cicatricial tissue, too thick to think of using the galvano-cautery. Intubation was continued.

Case X.—Upper Bronchoscopy (ether) removal of a sharp duck bone from the second subdivision of the middle right bronchus. Operation successful.

Mrs. E., of Parish, N. Y., while eating duck at noon dinner in March, 1908, on taking a deep inspiration while laughing, felt a piece of bone drawn into the throat, choking and greatly alarming her. Attempts at removal were unsuccessful, and the bone seemed every minute to be drawn more deeply into the wind-pipe, and on into the chest. The dyspnoea, which was great at first, gradually subsided. Her physician, Dr. Foote, was sent for and on his arrival advised her going to Syracuse, and referred her to Dr. S. B. Craton, who saw her five hours later. He sent her to St. Joseph's Hospital and asked me to take care of her. I saw her at 8 P. M. with Dr. Craton, seven hours after the accident. She was sitting up, breathing quietly, no dyspnoea, voice clear, pain on coughing and a sense of oppression over the middle of the right lung. Since the accident she had coughed up blood, first clear, later mixed with mucus.

On examination with laryngeal mirror, a distinct scratch was seen on the posterior wall of the pharynx, one vocal cord was reddened and swollen and showed signs of an abrasion, blood in trachea. Auscultation and percussion were negative. She was sent to the operating room and etherized. The separable spatula was introduced and through it the longest bronchoscope was passed down between the vocal cords to bifurcation of the trachea, and, finding nothing, the right main bronchus was entered and explored, and nothing being discovered, the upper branch was entered, then the middle branch, and at the second subdivision of this branch a white ivory-like foreign body, seemingly the size of the head of a match, was seen. On touching it with the bronchoscope or probe, cough became very severe. A pledget of cotton, saturated with a 5 per cent cocaine solution, was carried down and the parts were soon anaesthetized. On the third introduction of the tubular forceps the body was firmly grasped and bronchoscope, forceps and foreign body were withdrawn together. It proved to be one of the

small bones of a duck's leg, somewhat L shaped, not unlike part of a wishbone, six or eight times larger than it had appeared, because one of the prongs or processes had projected down into one bronchiole, while the other had entered another, and only a small part at the angle of the bone had presented to the bronchoscope. One of the bony processes was facettèd and smooth, the other as sharp as a needle, and this had evidently cut as it went down. A localized bronchitis developed but cleared up, so that the patient returned home on the fifth day.

The next day being Sunday, friends in great numbers visited her and heard her repeated recitals of her experience, until she became almost hysterical. At supper she felt certain that a piece of cake was inhaled and drawn down to the same place in the lung. Dr. Foote was called at midnight, and in the morning brought her back to the city to my office. There was no evidence of anything, beyond her being exceedingly nervous and almost hysterical. She was sent to the hospital, where she remained quietly, and with the aid of bromides soon regained her normal condition, and went home in a few days. There has been no further difficulty.

Case XI.—Gastroscopy (under ether) for diagnosis in recurrent gastric ulcer after a gastro-jejunostomy in a woman of fifty years. Marked stricture at the cardia with spasm and cyanosis. Second gastroscopy under ether three weeks later. Hemorrhage caused cessation of operation. Third gastroscopy (Ether) two weeks later. Gray scar observed, Argyrol applied to reddened inflamed area. Patient greatly relieved of all symptoms.

Mrs. L., housewife, patient of Dr. J. J. Buettner, aged fifty, has been an invalid and entirely incapacitated for work for more than two years. In 1905 she began to vomit blood and had upwards of one hundred hemorrhages from the stomach till her entrance to the Women's and Children's Hospital on April 16th, 1907. Severe attacks of cyanosis and syncope, accompanied by great pain in the gastric region were pronounced features of her clinical history. These attacks were so severe that death was frequently thought imminent. They occurred weekly, sometimes daily, for months and several years prior to her

entering the hospital. Gastric ulcer was diagnosed, and gastro-jejunostomy performed by Dr. McLennan, in August, 1907. Patient left the hospital greatly improved five weeks later. Two months after the operation bloody vomit and pain referred to the site of operation recurred, and she was gradually getting into her former condition when she re-entered the Hospital January 27th, 1908. Dr. Buettner was anxious to have me use the gastroscope. So bad was her general condition that I refused for several weeks, but finally, on February 27th, 1908, she was etherized by Dr. Buettner, and the gastroscope passed. There was no difficulty until the cardia was reached and the tube entered the stomach. There was decided stricture or tightness encountered on going through the cardia. Breathing immediately ceased, but resumed as soon as the tube was withdrawn into the œsophagus. The gastroscope was passed into the stomach and soon withdrawn, say in fifteen to twenty seconds (because of breathing ceasing) eight or ten times. The folds and rugae were seen; no ulcer discovered. She left the operating table in good condition. No vomiting or hemorrhage followed, and she was so much relieved of all her symptoms that she begged me to again make another examination.

Second gastroscopy under ether on March 12th, 1908. There was considerable hemorrhage at this examination, and the complete absence of cardia spasm and of stricture on entering the stomach was noteworthy. Her improvement continued, and again, at her own urgent request, I made a third gastroscopy under ether, on April 14th, 1908, again noting absence of cardia spasm and stricture. No hemorrhage, and a splendid view of the stomach wall obtained. A gray scar was observed, and to a small, reddened, inflamed area a 50 per cent solution of argyrol was applied with a cotton carrier. The patient left the hospital three weeks ago (a few days after last operation) and during this time she has been visited but twice by Dr. Buettner, the longest period in three years without medical attendance. She has walked several blocks. There has been no cyanosis since first gastroscopy. A slight hemorrhage of blood at menstrual period.

Case XII.—Gastroscopy in a girl of twenty-six years (under



ether) with history of gastric ulcer, revealed a scar and an ulcer and pyloric opening seen. Argyrol applied to the ulcer. Marked improvement followed.

Miss B., aged twenty-six, servant, patient of Dr. Buettner.

In Spring of 1907 first symptoms referable to stomach. In June, 1907, she came under the care of Dr. Buettner, who diagnosed ulcer of the stomach and has treated her for it since that time. In October, 1907, she entered the Women's and Children's Hospital, remaining under treatment one month, when she left improved. In January, 1908, she became much worse and gave up work in February. Vomited blood, and blood in stools almost daily. On March 6th, 1908, she entered the Women's and Children's Hospital for the purpose of having an examination of the stomach with the gastroscope. On March 7th, under deep ether anaesthesia, the gastroscope was introduced, and gradually passed down the esophagus, which was pale in color, until the cardia was reached and entered. The stomach wall was thoroughly and systematically explored, the rugae folding and unfolding as the instrument passed over the wall, giving many and varied pictures, the mucous membrane deep red in color. The pyloric opening was seen and the duodenum itself viewed for perhaps an inch in depth. A grayish patch, oblong and half an inch in length, was seen. It was apparently a scar. A reddened and inflamed area of about the same size was localized and thought to be an ulcer. A 50 per cent solution of argyrol was applied by means of cotton carriers to this. She recovered from the ether with no unpleasant or untoward symptoms following and left the hospital two days later. Since then, two months, there has been no return of symptoms and she feels better than she has at any time during the past two years.

Case XIII.—Asphyxia in the new born infant, caused by pressure of enlarged thyroid and thymus glands. Direct laryngoscopy, and passage of long cannula into the trachea and bronchus giving temporary relief and prolonging life sixteen hours. Intubation. Death. Autopsy.

On April 13, 1908, at 2 P. M., I was called by Dr. Geo. B. Broad to St. Joseph's Hospital to do a tracheotomy on a child



just delivered by him. When I reached the hospital the child was two hours old, almost asphyxiated, blue, and kept alive only as the father breathed deeply into the child's mouth. When he stopped the child's breathing ceased. The usual methods employed in resuscitating new born babies had been employed before I arrived.

Dr. Broad was about to do a tracheotomy as I entered the room. The child's pulse was only perceptible. The heart was apparently normal. The child had never cried or taken a deep inspiration. On examination it was seen to be a large, plump baby, weighing about ten pounds, face and neck a deep blue, puffed and swollen, veins distended and a large mass, apparently an enormously enlarged thyroid gland covered by a large plexus of veins, was easily felt covering the anterior part of the neck, extending from the thyroid cartilage to the sternum. The mass was longest in the transverse diameter. A tracheotomy would necessitate going through this mass and would surely be immediately fatal in the child's condition. On passing the finger into the mouth to palpate the entrance to the larynx, the sensation to the finger was that there was no orifice, suggesting a partial atresia of the larynx. The father kept the child alive by continuing to breathe into it, while I prepared for a direct examination of the larynx. On introducing the spatula beyond the epiglottis and elevating it for inspection of the larynx, the child took its first unaided inspiration. The larynx was normal, the miniature vocal cords and chink of the glottis with trachea beyond showed up perfectly. I have never seen the interior of the larynx more clearly in adult or child. Respiration now became regular, and the color began to improve. As soon as the spatula was removed asphyxia returned. An attempt to introduce the smallest bronchoscope was made but failed, because it was too large. A one year O'Dwyer intubation tube was introduced through the speculum and gave such relief for half an hour that the child's lips became red, and it breathed regularly, but at the end of half an hour it suddenly became worse, and the tube was withdrawn, and the long straight suction cannula was passed into the trachea and bronchus, an enlarged thymus pressing on the trachea and

bronchus being now suspected, in addition to the large thyroid gland. This relieved the cyanosis for a time. The suction pump to withdraw any secretion in the tube was used, but without result. A soft rubber catheter was passed through the mouth into the trachea and gave some relief. At the end of four hours of this work the breathing was regular, though very superficial, and its lips and face were red and had entirely lost the blue color, so long as the head was hyper-extended on the neck. We left the hospital at 7 o'clock with instructions to keep the head thrown back. The child's condition precluded any hope of its surviving a tracheotomy, considering the large veins and glands that covered the trachea.

At 10 o'clock the father resumed his inflation of the lungs for ten or fifteen minutes, when it improved, only to have another failure at 4 A. M., when it died, sixteen hours after birth. Autopsy made by Dr. Broad.

"The baby, a girl, was very well developed and nourished, weighing about ten pounds. A mass consisting of thyroid tissue was palpable in the middle of the neck and extending from the thyroid cartilage to the upper border of the sternum. There were many very large veins in and about the mass. The chest was then opened and the relationship of the trachea to the surrounding structures observed. So far as could be determined there was no great pressure on the trachea or compression of it. The thymus gland covered the whole front of the trachea just below the upper border of the sternum, and extended downward for about two inches. Each lobe was from two to two and a quarter inches long. They were about one-half inch in thickness at the upper part, much thinner below, apparently large, but normal in contour. The trachea, with the larynx, was removed, but no abnormality or defect was observed in either structure. Both lungs contained air. The foramen ovale was open, otherwise the heart was normal. The structure of every organ excepting the thymus, both in the thorax and abdomen, seemed normal. The cranium was not opened."

• Case XIV.—Œsophagoscopy and Bronchoscopy, woman seventy years of age. Stricture of œsophagus due to new growth. Pouch or dilatation, with well marked rugae in the walls. Bougie passed through instrument and on through the stricture.

Mrs. K., age seventy years, large woman, weighing 190 pounds, was seen by me in consultation with her physician, Dr. Dwinelle, in January, 1908. She had been unable to swallow and retain any solid food for two years, with a history of regurgitating food beginning sixteen years ago, and for two years constantly present and getting worse. Has wheezed after eating, more or less all of this time. No pain, no history of hemorrhage. A diagnosis of stricture of œsophagus, with dilatation or pouch above it was made, and a radiograph and œsophagoscopic examination advised. A radiograph, made by Dr. Coon, showed distinctly a pouch of the œsophagus. On May 1, 1908, at the Women's and Children's Hospital, I made an examination of the œsophagus and bronchus. The large sized œsophagoscope was passed down the gullet to a point nine inches from the teeth, when it entered a pouch or dilatation two inches in length, and containing some remnants of food, which were removed with forceps. The wall of this pouch was exactly like that seen in the stomach, showing the same rugae and folds, excepting that it was paler. At the bottom of the pouch the tube came up against the obstruction. This was caused by an infiltration of the posterior and left lateral walls of the œsophagus, hard and firm to the probe; to the eye it seemed distinctly papillated. A No. 12 hard rubber olive tipped bougie was, with difficulty, passed down through the stricture, which was exceedingly tight and narrow and apparently two inches in length. Two small pieces of the wall were removed by forceps and reported by Dr. Steensland, the pathologist, to be simply squamous epithelium. The obstruction was evidently due to a tumor, probably carcinoma, in the mediastinum, and involving the wall of the œsophagus. The trachea and bronchus were examined with the bronchoscope, but nothing adventitious discovered.

#### *Recapitulation.*

Eighteen operations or examinations were performed on fourteen patients, fourteen times under general anaesthesia (ether in all but one), three under cocaine, and one without any anaesthetic. The age of the patients varied from a new born babe of two hours, to an elderly woman of seventy years. In

four cases the operations were for the removal of foreign bodies; penny in the œsophagus of a five-old-year boy, a bone in the bronchus of a woman, a fish fin in the trachea of an eight months' infant, a spicule of glass in the larynx of an eight months' infant, all being removed but the last.

Of the remaining cases, fourteen operations and examinations were for papilloma of the larynx, stricture of larynx, papilloma of lower pharynx, stricture (spasmodic) and stricture (organic) of the œsophagus, suspected foreign body in œsophagus, three times on one patient for stricture and ulceration of the bronchus, once for relief of asphyxia in a new born infant caused by pressure of enlarged thyroid and thymus glands, and four times for examination of the stomach for gastric ulcer or other lesions.

Experimental work on the dog to determine the cause of the inhibition of respiration on the passage of the gastroscope from the œsophagus to the stomach, and of the introduction of the bronchoscope into the bronchus, was made in conjunction with Dr. Frank P. Knowlton, Professor of Physiology. This was suggested because of the marked effect on respiration observed in the passage of the gastroscope into the stomach, noted in Case XI., the same effect being also observed while working on a dog.

Professor Knowlton reports the result of the experiment as follows:

| Light Anaesthesia.                      | Respirations,<br>rate per<br>minute. | Remarks.  |
|---|--------------------------------------|---|
| Before introduction.                    | 93                                   |   |
| Gastroscope in œsophagus.               | 43                                   |   |
| Gastroscope in stomach.                 | 10                                   | Respiration inhibited in phase of complete inspiration. Expiration inhibited. |
| Later: Normal.                          | 84                                   | Longest inhibition of respiration was ten seconds.                            |
| Gastroscope in œsophagus.               | 45                                   |   |
| Gastroscope in stomach.                 | 10                                   |   |
| Normal before introducing bronchoscope. | 52                                   |   |
| Instrument in trachea.                  | 20                                   | Coughing expirations. Inspirations inhibited.                                 |
| Vagi cut.                               | 33                                   |   |
| Gastroscope in stomach.                 | 30                                   |   |

The inhibition of respiration is apparently due to reflex effect on the respiratory center, arising from irritation of the gastric mucous membrane, the vagus nerves being afferent pathways. A similar effect has been noted in the rabbit, and further experiments are being carried out.

It would seem from this experiment that no danger need be feared from this reflex irritation, which could be overcome in any given case by the application of cocaine.

In conclusion. Direct visual examination of the larynx, the trachea and the bronchus, down almost to the last subdivision of the bronchiole, is a practical thing and of inestimable value, not alone for the removal of foreign bodies in the lower air passages, but for the diagnosis and the local treatment, both by topical application and surgical interference, of various lesions that affect the larynx, trachea, bronchus and the lung, such as laryngeal tumors, tracheal and bronchial ulcers, tuberculous or other lesions and lung abscesses, discovering the cause and location of pressure on the trachea or bronchus.

By means of œsophagoscopy, the accurate diagnosis and treatment of many hitherto inaccessible diseases of the gullet becomes possible. The causes of painful and difficult deglutition, the etiology, diagnosis and localization of œsophageal obstructions and strictures, will depend not so much upon the findings of the bougie as upon the observations of the eye, aided by the probe.

Thanks to the brilliant work of our fellow member, Chevalier Jackson, the stomach has been added to the list of internal organs capable of being examined directly by the eye. Through the gastroscope, he has demonstrated the possibility of making a systematic and complete examination of from two-thirds to three-fourths of the stomach wall, that in many cases the pylorus can be seen and probed, that gastric ulcers may be treated locally, that early diagnosis of carcinoma may be made, that sections of tumors may be removed for microscopic examination, in a word that the stomach, like the larynx, is subject to direct and local examination and treatment.



*Discussion on the Papers of Drs. Bryan and Halsted.*

Dr. Emil Mayer:—In connection with this subject I have a radiograph and two foreign bodies which I would like to show. These cases occurred during the last month and were removed by bronchoscopy.

The first, a grain of corn, was removed from the right bronchus of a boy aged seven years on March 15th by superior bronchoscopy, at the Newark City Hospital. The other specimen is a reed from a whistle removed from the trachea of a boy aged eight at the Presbyterian Hospital, also by superior bronchoscopy. The radiograph shows it distinctly.

In presenting these cases not hitherto recorded, I feel that it is necessary that those of us who have had some experience in this comparatively new field should bring such instances forward, so that when an individual case comes to any of us we may be somewhat prepared. And here I would reiterate what I said some time ago, and which I think is the experience of everyone who has had occasion to treat such cases, namely, that foreign bodies of the nature of kernels of corn, or any other vegetable matter, should be removed as promptly as possible. It does not do to wait in those cases, as such a body is sure to absorb moisture and swell. And I would insist (or prefer to have nothing to do with the case) that immediate removal be effected of such bodies lodging in a bronchus. I think that everyone present who has had any experience of this kind will have noted that foreign bodies of this kind left in for even a comparatively short period will, as in Dr. Bryan's case, cause a great deal of trouble, the delay being sufficient to produce an œdema of the larynx of such a nature that it was impossible for him to remove it by superior bronchoscopy, which he would have been able to do had the case come earlier under his observation. My own method in these cases follows closely that of Killian with this exception, that is, in spite of the general anæsthesia to still use cocaine in addition and use it quite freely even in very young children. Dr. Halsted's case is of added interest in that it is probably the youngest on record, eight months being younger than any heretofore recorded, I am quite sure. I thought I had seen the youngest case in a child of ten months, but this of eight months is still younger.

Another thing is the absolute need of well trained assistants. I feel confident that my own success could not have been as great as it was if it had not been for the valued assistance I received from Dr. Yankauer, to whom much credit is due. There is no doubt that when we all have mastered the difficulties that the removal of these bodies by means of the tubes without

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incision will be possible to us all. In both cases cited the patients were discharged cured within a week after removal of the foreign bodies.

Dr. H. L. Swain:—As Dr. Halsted has well said, this theme is so new that the experience of each man as he tries to accustom himself to procedures and qualify himself to the urgency of any given case, is of exceeding great value. I have had two cases this winter which have been very interesting to me owing to the size of the foreign body which I was able to remove by the Killian tubes.

The first was that of two false teeth from a portion of a dental plate swallowed by an epileptic in a convulsion. It had been lodged in the upper part of trachea for 12 hours. Great œdema resulted and the urgency was so grave that tracheotomy had to be performed before I operated on the case. When I arrived it was extremely easy to see the body by the Killian tube, but owing to the great swelling it took me some time to effect removal.

The other case was that of a young boy who had swallowed a doubled-up, old-fashioned miniature tin-type which he had been using as a whistle. It had gone down into the larynx and lodged in the trachea below, and knowing nothing of the nature of the body I was greatly surprised to discover what it was. It was readily removed by Jackson's forceps.

I suppose you have all seen this Morton's foreign body forceps which has proved to be a very effective little thing in dealing with such things as pieces of cotton wool, which may have slipped into the larynx during operations.

Another instrument is a more powerful forceps, which I have used successfully in dealing with laryngeal papillomata by the direct method.

Dr. E. Fletcher Ingals:—There is one point that I would like specially to emphasize, and that is that if the patient is suffering from severe dyspnœa due to a foreign body, we ought not to try upper bronchoscopy, as there is great danger from the anæsthetic and in most of the cases where it has been tried under these conditions a hasty tracheotomy has been necessary. Tracheotomy in these cases should be done first to relieve the dyspnœa, and in many the foreign body will be coughed out at once, but if it should not be the bronchoscopy will be much easier than through the glottis. As to the necessity of trained assistants, I hardly agree with the speaker, but an assistant who will hold the head firmly is very important. In the first bronchoscopy I did I have sometimes said that the good results were due more to the assistant than to the operator, for he held the head as in a vice.

I have not used ether as an anæsthetic in more than one or

two cases. I have used chloroform, which seemed to me best. I realize the dangers of chloroform, but the danger from the after-results seem to me greater in these cases than from chloroform. The chloroform can be administered easily during a long operation through a small tube held over the bronchoscope. The use of atropine with ether to prevent secretion is theoretically correct, but as you all know I have two unaccountable deaths following bronchoscopy where I could not conceive of a possible cause unless it was either the atropine, cocaine or adrenalin. I wrote to Killian about these and he was inclined to attribute the fatal result to some of these drugs.

The first case that Dr. Halsted reported was of extreme interest because of the great time the penny had remained in the œsophagus, and because of his ingenuity in exposing it to view. I have heard of a case where a penny had been swallowed and, as it was not detected by the œsophagoscope, it was thought to have ulcerated through the œsophagus. In that case an œsophagotomy proved fatal.

It would seem to me that the use of the galvano-cautery in the bronchi is too dangerous. I should not care to try it. Dr. Halsted spoke of one case in which he said œsophagoscopy and bronchoscopy had been done, but it was not clear to me whether or not he did them both at the same time. I wish to call attention to the necessity for sterilizing the instruments after passing them into the œsophagus before introducing them into the air passages.

I do not look for the brilliant results which Dr. Halsted's paper anticipates in the treatment of disease of the bronchi through the bronchoscope, or of the œsophagus through the œsophagoscope. It seems to me that too much is expected from it by him and also by Dr. Jackson. It has been their good fortune to have no mortality, but there have doubtless been several deaths that have not been reported. I recall one fatality evidently due to the operation in a series of cases reported from Europe, when it was stated that the operation was without danger, and there have undoubtedly been others. Bronchoscopy is a brilliant operation and it enables us to save many lives; but it should not be undertaken without full realization of its dangers. Excepting for the removal of foreign bodies I doubt whether we will often be justified in carrying out bronchoscopy for the treatment of diseases that have not first been treated by less radical measures. In cases that cannot be reached by safer methods and which do not yield within a reasonable time bronchoscopy should be tried, provided it appears to afford the patient the best chance, but we must remember that it is not entirely safe. It cannot be done satisfactorily without either local or general anæsthesia, either of which may cause a fatal

issue, and the operation itself, excepting in the most skilled hands, is very liable to cause some injury.

Dr. W. E. Casselberry:—In respect to direct laryngoscopy in the removal of multiple papillomata of the larynx, I would suggest the desirability of simplifying the technique. Every one has laid stress upon the necessity for trained assistants, general anæsthesia and hospital accommodation. That is all very well for bronchoscopy in general, and even for direct laryngeal operating when the operation can be completed in one or two periods, but in diffuse papillomata of the larynx repeated manipulations are usually necessary, and to keep the patient in the hospital and cause multiple administrations of general anæsthesia becomes impracticable. Taking this into consideration, I tried and succeeded with two patients in making, by direct laryngoscopy, a means of operative office treatment, without general anaesthesia and without skilled assistance. The patient would come to the office, lie on the table, let his head hang downward over a pillow pendant from the edge of the table, both pillow and head being supported by a stool of adjustable height. Papillomata filling a drachm vial were removed from the larynx of one of these patients and a small fibroma from the larynx of the other. The latter I had previously failed to remove by the mirror method. The patients required some preliminary training, but, having the alternative put to them of going to the hospital to stay for an indefinite period of time, they were willing to try, and soon got the idea of the hanging head and muscular relaxation. Each of them had the tube passed from ten to fifteen times but were rewarded by success, and I am convinced this simpler procedure is practicable in many cases.

In respect to general anæsthesia, we know that under ether, unless the patient is profoundly anæsthetized, the laryngeal reflex is not suspended and in one case of œsophagoscopy I experienced great difficulty in this respect, for though the general anæsthesia was as profound as the expert anæsthetist deemed safe, the patient retched while the tube was in, interfering considerably with the examination, and as it was followed for twenty-four hours by a bloody discharge, it showed that there had resulted at least an abrasion of the œsophagus. A preliminary hypodermic injection of morphine would probably prevent this reflex, which is dangerous under the circumstances, otherwise I should, with Dr. Ingals, prefer chloroform instead of ether for bronchoscopy and œsophagoscopy on account of the greater ease in maintaining a uniform anæsthesia. In all throat operations made with etherization I use in addition a 5% cocaine solution applied locally by spraying for a few seconds. I have done so in all adenoid and tonsil operations for years past and have never had a death from the cocaine, so that I can scarcely credit Dr.

Ingals's explanation of the fatal case he mentioned as due to the cocaine.

Dr. A. Coolidge, Jr.:—In most cases a foreign body in the bronchus can be reached by upper bronchoscopy, provided the necessary instruments and ability to use them are available. If not, as is often the case with the general surgeon at a distance from large centers, tracheotomy and lower bronchoscopy are indicated. After tracheotomy an ordinary head mirror, a hollow tube, such as is used as a cystoscope for the female bladder, and the proper forceps will be sufficient to reach the primary bronchi. Lower bronchoscopy may be advisable also if the symptoms are urgent, or if the nature and position of the foreign body offer particular difficulties for its removal. Cases have been reported in infants in which upper bronchoscopy has been followed by obstruction, due to swelling of the larynx.

The value of upper bronchoscopy for diagnosis should not be overlooked. I have within a week seen a child with a history of having inhaled a half of a peanut kernel, but the child positively stated that on its way to the hospital he had coughed it up. Physical signs were uncertain, and there was a temptation to temporize. Under ether anæsthesia, however, the bronchoscope was passed without the slightest difficulty, and the peanut seemed impacted in the right bronchus. Failing to dislodge it, and fearing that further manipulation would result in breaking it into small fragments, I did tracheotomy and easily removed it intact through the shorter and larger tube. I do not like to use transformers in connection with the small distal lamps. It is safer not to run the least risk of short circuiting powerful currents. A small battery is generally obtainable. A few cells, such as are used for ringing door bells, can be used if nothing better is at hand.

A sponge holder must hold the sponge absolutely securely. I have one which, by a screw, locks the sponge in place. The device shown by the writer of tying a string to the sponge is excellent.

We have seen the tongue depressor modified and elongated into the autoscope of Kirstein, then into the tube spatula of Killian which reaches into the larynx. Dr. Mosher's device enlongates it still farther, so that it reaches below the arythenoids and pulls the cricoid forward, opening widely the upper part of the œsophagus to direct inspection.

In connection with the discussion Dr. H. P. Mosher showed the following instruments:—

1. An Œsophageal Speculum for the examination of the upper end of the œsophagus. With this form of speculum the œsophagus can be examined by direct vision almost to the clavicle. One great advantage of the instrument is that it has no hood.



2. An Oval Œsophageal Tube. This tube is three-quarters of an inch wide, which is considerably larger than the œsophagoscope is usually made. This size of tube, however, can be introduced readily in the lateral position by using the open œsophageal speculum which has just been described. The tube is fitted with a plug which has a glass face over one end. By means of this plug the tube can be made air tight, so that if air is forced into the œsophagus through the drainage tube on the side of the main tube the œsophagus can be dilated. This procedure should be a help in distinguishing between the mouth of a diverticulum and the lumen of the œsophagus.

3. A Mechanical Dilator for Strictures of the Œsophagus. This dilator is a modification of the Kollman dilator used in the urethra. It is twenty-four inches long and is fitted with two tips. The first tip is perforated so that a thread can be used to guide the dilator; the second tip ends in a small gum elastic bougie about an inch and a half long. By changing the inner rod the dilator can be made flexible or stiff, as the operator may wish.

The object of the instrument is to make possible rapid dilatation of a stricture. It was used recently for this purpose in a case of stricture in a child two years old who had swallowed potash. In this instance the stricture was dilated at one time from 20F to 30F. After this bougies were passed easily without ether.

4. A Triangular Laryngeal Tube. This triangular laryngeal tube is a modification of the fenestrated oval tube. It was designed for cases of papillomata of the vocal cords which are placed near the anterior commissure. Such growths occasionally are very hard to secure even under ether. By making the tube triangular instead of oval it can be pushed well into the anterior commissure. The tube is fenestrated not only on the sides and in front, but has a window behind for the posterior commissure.

Dr. J. Payson Clark:—In this connection I should like to mention a device which I used in a case which I reported before the Eastern Section of the American Laryngological Rhinological and Otological Association last winter, of stricture of the œsophagus. The case was that of a boy about eight or nine years of age who had swallowed a quantity of caustic potash; he had almost complete stricture and could swallow nothing but liquids, and those only very slowly. The œsophagoscope went down into a large pouch, but I could not find an opening. I again examined him a day or two afterward, but before passing the tube gave him a glass of water to drink in which I placed a silk thread, one end being attached to his face with adhesive plaster. On passing the œsophagoscope this thread could be followed down the œsophagus until it was seen to disappear into a small opening at one side of the pouch, giving me the exact location of the stricture. It has been found difficult at time to find such small

strictures, but with this silk thread it was very easy. This device is not a new one, but I have not heard of its use in connection with the œsophagoscope.

Dr. J. H. Bryan:—I have had no experience in removing these multiple papillomata and I should like to know if they do not tend to recur.

Dr. J. M. Ingersoll:—I had one case, a boy who had had a small tin whistle in the œsophagus about three weeks. A skiagram showed the whistle just below the inferior border of the larynx. A tube was passed into the œsophagus and an attempt made to remove the whistle. It was held so firmly in position that I was afraid the œsophagus would be ruptured if more force was used. The following day a second skiagram was taken and it showed that the whistle had been dislodged by the attempt to remove it and had passed down into the stomach and a few days later was passed in the natural way.

Dr. W. E. Casselberry:—In one of the cases I referred to of papilloma of the larynx I may say that they did recur; in the course of six weeks I was at it again. But this is the advantage of my method, it is so simple and there is not the distress incident to general anæsthesia. The treatment can be given as many times as required, the patient getting up, able to continue his affairs without trouble. Thus followed up in adults they ultimately cease to recur. This patient now writes to me that he is talking as well as he ever did.

Dr. J. H. Bryan:—I do not know that cases of this nature have been reported as cured by this method but they have promptly recurred. One case I saw reported as cured and now the child is worse off than it was before.

Dr. E. Fletcher Ingals:—I think that in young children these growths are more liable to recur than in adults; but that the application to the base, after removal, of carbolic acid or iodine, which are sometimes used by dermatologists for the destruction of warts, may sometimes prevent the tendency to new growth.

Dr. J. M. Ingersoll:—I have had two cases of papilloma of the larynx in children which have been under observation for more than a year. After removal of the papilloma, the children were treated with the X-rays for a month every day or two and then at longer intervals. There has been no recurrence in these cases.

Dr. T. H. Halsted:—With reference to the use of the galvanocautery in the bronchi I am entirely in accord in thinking that that might be a very dangerous proceeding, and one that could be employed only in an exceptional case, and then only in a main bronchus. I had my cautery electrode made for a laryngeal case, but found I could not use it in that case. The application of argyrol to the ulceration in the bronchus is certainly showing good results and there has been no disagreeable reaction.



With reference to the matter of trained assistants: Trained assistants are very desirable; the work will be done much more easily and much better if the assistant, for instance, who holds the head, understands his work and does it properly. The anæsthetist should be experienced in this class of work. I have done upper bronchoscopy in my office under cocaine anæsthesia, my assistant being the trained nurse in my office. Dr. Coolidge has called attention to the great assistance in having a good equipment. This is certainly important, as the number of emergencies and unexpected conditions that arise, calling for immediate action, is remarkable and one should be prepared by as full an equipment as possible. As to the anæsthetic, I almost always employ ether when a general anæsthetic is used, believing it to be safer than chloroform as a general thing, and like to have a trained anæsthetist in any kind of operation, and especially in this class of work. The anæsthetic used and the manner of its administration are exceedingly important matters. Cocaine is often sufficient.

As to the future of direct examination of the larynx, bronchus, œsophagus and stomach, I am very optimistic and think we are at the threshold of what will soon be recognized as an exceedingly wide field. The purpose of my paper was to bring out the fact that the field of operation was by no means limited to foreign bodies; in fact this is its least important field, but for the detection and removal of foreign bodies it is the ideal method. For diagnosis and operation in pathological conditions in the larynx, the trachea and bronchus and the œsophagus, it is to-day the very best method we have, because we can see directly with the eye, can remove with cutting forceps new growths for microscopical examination or their surgical removal may be performed.

I am enthusiastic over the possibilities in the early diagnosis and often of treatment of pathological conditions within the stomach. As Jackson has demonstrated, in two-thirds to three-fourths of the cases, the pylorus can be examined and a section of suspected growth can be removed for microscopic examination, determining the nature of the tumor many months before it is by present methods demonstrable. It means that the diagnosis in suspected malignancy may often be made with certainty and the patient receive all the benefit of early diagnosis. In gastric ulcer it may offer one of the best, if not the best, means of treating this affection. In one of my gastroscopies I detected a scar, located the pyloric opening and saw into the gut for perhaps an inch, and think I could have entered the intestine itself. In another case, both a scar and a localized inflammatory area were observed, and the latter treated locally. The gastrologists are going to find gastroscopy of great assistance both from a diagnostic and therapeutic point of view, and the surgeon operating on the stomach will find the knowledge given him by the gastrologist either before or during the operation of the utmost value.

*Candidates' Thesis.*SOME SURGICAL PRINCIPLES ESSENTIAL TO THE  
CURE OF FRONTAL SINUS EMPYEMA.

BY JOHN F. BARNHILL, M.D.

A review of the recent literature of the surgical treatment of the pyogenic affections of the frontal sinus shows clearly that much difference of opinion exists among operators of experience concerning the proper plan of procedure. The varying degrees of severity of the disease that may be present in the sinus, and the several accessory conditions that may occur in a given case, often make the choice of plan debatable; but in those cases of suppuration of the sinus where the symptoms are quite similar in every essential it would seem that too great a difference of opinion exists among operators of apparently equal prominence and experience. Such a difference of opinion is unfortunate for those operators of less experience, who, as a result, too often feel a sense of doubt and uncertainty when contemplating the performance of frontal sinus surgery. The diversity of opinion emphasizes the fact that this class of surgery is often difficult, and that ideal surgical measures have not yet been established. To some extent, no doubt, the present views concerning frontal sinus surgery are the result of the rather rapid evolution of the rhinologist from a condition of practice in which his chief duty, only a few years ago, seems to have been to cleanse and medicate the mucous membranes of the upper air tract, to that which to-day may require the execution of the most skilful surgical measures, involving not only the nose, but also the more distant and intricate structures of the entire head. This evolution from the erstwhile rhinologist, who was a sprayer of noses to the present rhinologist, who must be a high type of surgeon, has in some measure been responsible for two sets of operators, viz.: those who believe that all, or nearly all, cases of accessory nasal sinus suppuration should be treated by the intra-nasal route, and those who hold

that, while the simple sinus empyemas may usually be thus successfully managed, when much actual pathological change has taken place in the structure within the sinus or its bony environs, external surgery should usually be chosen.

The discussion which continues between that class of surgeons who maintain, as does Ingals, that 95 per cent. of all cases of suppurative frontal sinus disease can be cured by the intra-nasal route, and that class which believe that the greatest certainty of cure and the least danger to the patient lie in the methods of operation by external surgery, puts forth some good arguments in support of these respective beliefs. The intra-nasal surgeon points out in support of his position the alleged facts that ample drainage, and the free admission of air into the diseased sinus will cure all except the worst cases, and that as a result of the concealed operative measures required, no unsightly scar remains. The chief argument of those who prefer external surgery, and the only one they consider absolutely necessary to make, is that thereby only can perfect drainage be secured and the diseased structures be completely removed. The more or less scar tissue which results from external surgery of the sinus, while always an objectionable feature, is by this class of surgeons regarded as a sequence absolutely necessary to a successful issue, and therefore entirely justifiable.

This paper is not, however, greatly concerned in the dispute between the two classes of surgeons, for both admit that a certain number of all cases of suppurative sinus affection must be operated by the external route, and that a certain percentage is also curable by intra-nasal procedure; but it is rather with the surgical principles essential to the cure of sinus empyema which should govern the method which is chosen, that we shall be most interested.

Good results from the practise of any method must depend largely upon the proper application of certain well-established surgical principles. Aside from an aseptic technic, which should be as perfect as possible in every case, the principles upon which all successful sinus work must depend, are, 1, the complete removal of all pyogenic structures; 2, provision for ample drainage of the operated parts; 3, healing the diseased cavity either by a

healthy, non-suppurative lining, or by the complete obliteration of the space by means of granulation tissue. The true value of either the external or intra-nasal method of treatment of suppurative frontal sinus disease can, therefore, be excellently judged by subjecting each plan to the standard of requirements of the above surgical principles. Of course in addition to these principles, the length of time the patient must suffer or be inconvenienced by the necessary treatment subsequent to each, and the actual danger to life resulting, although not surgical principles, are nevertheless often the result of the application of correct or faulty principles, and must, therefore, in justice be considered. Let us apply these principles first to the method of treatment by intra-nasal drainage.

The efforts of operators like Ingals, Halle, Good and others to cure frontal sinus suppuration by the intra-nasal route, and the ingenious devices which they have perfected for entering and draining the frontal sinuses, should receive the heartiest commendation. Undoubtedly a large percentage of cases in which structural changes have not been extensive, and in which the anatomical formation of the sinus is normal, can be and are cured by the simple provision thus made for good drainage. When, however, anomalies in the anatomic structure of the sinus are present, and when extensive pathologic tissues have been formed within, drainage alone is not often sufficient to bring about a cure; for in most instances it is impossible to remove this diseased pathologic content of the sinus, or to correct its anomalous structure through the comparatively small opening which it is possible by this method to provide for the drainage. Moreover, in case of the frontal sinus it has been considered a settled point that it is not possible or safe to enter the sinus with instruments intended for the enlargement of the infundibulum, in a considerable percentage of cases. Mosher (Candidate's Thesis for American Laryngological Association), after a study of the applied anatomy of the frontal sinus in 100 wet specimens, and 50 cleaned skulls, states that it is only by chance that the naso-frontal duct can be catheterized. Turner (*Accessory Sinuses of the Nose*) says the variations in the frontal sinuses which have been described sufficiently indicate that the passage of the probe through the anterior nares toward the frontal sinus may meet with some opposition.

Occasionally a narrow or tortuous duct, or the small size of the ostium frontale, may prevent the probe reaching the sinus. Beaman Douglass states concerning this point (*Laryngoscope*, May, 1904): "One is often mistaken in believing that he has entered the frontal sinus, when, as a matter of fact, his instrument is within an enlarged anterior ethmoidal cell." (*Bulla frontalis*). All who have extensively examined the frontal sinus either in the wet or dry state of the skull must have observed that in a considerable percentage of instances an anterior ethmoid cell projects itself into the frontal sinus either external or internal to the sinus orifice of the duct, and that the mouth of the duct lies partially under such cell. In this class of case catheterization of the frontal sinus, when successfully performed, must always result in the entrance of the instrument through the broken down ethmoidal cell wall, through which it has been forced. Douglass has, furthermore, pointed out the fact, which has also been observed by others, that the internal wall of the frontal sinus frequently lies so low above the orifice of the duct that any cutting instrument, even if successfully introduced through the naso-frontal duct, would immediately penetrate the cranial cavity and would most probably set up an infective meningitis. On this point Mosher states that if there is a sufficient basal relationship between the floor of the frontal sinus and the nose, a burr can be pushed up through the duct into the sinus, the burr running on a probe as a pilot. Concerning this plan of entering the sinus, Mosher observes that on the cadaver beautiful specimens can be made in this way, but that he is always afraid that the practice of the method will make cadavers of some of his patients. The literature of the subject, therefore, clearly indicates that few operators would be willing to place so high an estimate on the percentage of successful cases of intra-nasal enlargement of the duct as does Ingals, who, as previously quoted, states that 95 per cent. of all frontal sinuses may be safely and successfully entered and drained by means of intra-nasal instrumentation.

It should be stated, however, in justice to the method of Ingals that his latest instruments and plan of procedure for entering the frontal sinus through the naso-frontal duct are undoubtedly less dangerous than formerly (Ingals's paper before the New York



Academy of Medicine, Section on Laryngology, Dec., 1907), for this operator now passes a guide through the duct into the sinus, next by means of the X-Ray determines that the position of the guide is correct, and then, when certain that all is safe, drives the burr, which cuts only on its anterior face, along the guide until the whole course of the infundibulum is enlarged and the sinus is entered.

Unfortunately the most perfect drainage which it is possible to secure by operative enlargement of the naso-frontal duct, does not always thoroughly drain the entire frontal sinus, and this simple procedure of enlarging the naso-frontal duct will not, therefore, cure every case, even though of an uncomplicated, acute variety of sinus suppuration. As evidence of this, several anatomists have especially pointed out the fact that the frontal sinus is often partially, and more rarely almost completely, divided by osseous septa in such a way as to preclude the perfect drainage of every portion of the sinus, even though the naso-frontal duct be widely open. Thus Hoeve, in an examination of 100 heads (*Laryngoscope*, July, 1907), states that frequently an internal set of frontal sinuses was normally present, due to the middle sagittal septa being complete. Hoeve further states that occasionally a superior accessory frontal sinus was present in thick skulls, and that frequently posterior and external accessory sinuses were found during these examinations. The writer has seen one posterior accessory frontal sinus and two external accessory frontal sinuses in cases of secondary operations which he performed because of failure to cure by intra-nasal methods, and he has frequently found the external accessory sinus present during external operations in primary cases that seemed urgently to demand the surgical interference.

It must be clear to every operator of experience that intra-nasal methods of operation upon the frontal sinus are in accord with but one of the several essential principles of successful surgery, viz.: the provision for better drainage; and that even this principle is often violated because of the frequent pocketing of the sinus as a result of the presence of coronal or sagittal septa within. It would seem, therefore, that it may be justly stated that there are at least three classes of frontal sinus suppuration that cannot be cured by intra-nasal methods. These are, 1, Those in



which the inner table of the skull arches above the sinus orifice of the naso-frontal duct so closely that, to enlarge this orifice through the intra-nasal route by means of cutting instruments, would almost certainly endanger the life of the patient because of injury to the duramater at this point; 2, Those cases in which more or less complete sagittal or coronal bony septa within the sinus prevent good drainage, even though a large naso-frontal opening be provided; 3. The cases in which extensive pathologic changes have taken place in the mucous lining or osseous walls of the sinus.

Having briefly shown the limitations of the intra-nasal method of operations on the frontal sinus, when viewed from the standpoint of surgical principles involved, let us next estimate the value of the external procedure when subjected to examination by the same standard.

By following the external method only can the surgeon hope in any satisfactory measure to carry out the surgical principles of removing in an absolutely thorough manner all the diseased tissue involved. This is the method of certainty in this respect, and not one of guess work. No thoroughly trained surgeon is to-day at all satisfied that he has performed commendable work in the removal of diseased structure unless he actually sees, and therefore, knows, that he has in nowise disobeyed this first essential of all good surgery. Concerning the dictum that the most perfect drainage should be provided, it will probably be admitted by most operators that the external method of operating on the frontal sinus is superior in every way, for when either the anterior or inferior wall of the sinus is removed, or when both have been resected, as in the Killian operation, every means of access possible is provided for the removal of all the diseased structures which will subsequently interfere with the outflow of exudates. The external method furnishes, also, conditions which are more favorable than any other to the healing of the cavity by means of healthy granulation tissue, because, as will be subsequently stated, if this method of operation provides the best means of thoroughly eradicating the diseased structures and establishes a thorough drainage for the operated parts, it necessarily follows that the most ideal conditions are provided for a healthy growth of the necessary granulation tissue.

The application of well-established surgical principles to the external or intra-nasal methods of operation for the cure of frontal sinus empyemas seems, therefore, to offer little ground for good argument in favor of the latter plan of treatment after the disease has long since passed the acute stage, or after it is known in any case that the given sinus consists of a series of compartments rather than of a single, smooth cavity, all parts of which drain, funnel-like, toward the infundibular orifice.

Which of the several methods of external operation is most ideal when viewed from the standpoint of the application of correct surgical principles? The writer has had most experience with the Killian operation and with the open method. The procedure devised by Professor Killian seems more in accord with good surgical principles than any other method in which the external wound is immediately closed, and therefore if a primary union of the external wound is intended it should be the method of choice. This operation is also desirable from the reason that a minimum of scar is produced. Although the Killian operation is ideal in many respects, and especially is this true when the anterior ethmoidal cells are diseased, the writer believes, judging by his own limited experience, that when radical surgery is indicated in the cure of frontal sinus empyema, that the desired end is often most certain of attainment by following the so-called "open method" of treatment—that which has been advocated and extensively practised by Coakley—for by this method greater opportunity is offered the surgeon to provide and maintain throughout the whole course of treatment the most perfect drainage; to foster by the art acquired by his experience the natural growth of the necessary granulation tissue; and should perverse or freakish behavior of the granulation structures at any time become manifest, to correct at once and restore the same to a satisfactory and normal state.

The probable deformity resulting from the open method has, of course, been an insurmountable objection to its employment in the treatment of frontal sinus suppuration, but the writer believes that a careful and prolonged experience in dealing with the healing of osseous wounds, such as the accessory sinuses, by means of obliterating them with healthy granulation tissue, will,

in great measure, enable the surgeon to avoid ugly and greatly depressed cicatrices. Perhaps nowhere in surgery does experience count for more in securing esthetic results than in the case of closure of an osseous wound by the open method. The surgical principle will presently be stated that overpressure will stunt or kill granulation tissue. Hence, if the gauze packing, which is commonly used in the treatment of the sinus by the open method, is inserted with too much pressure, the filling-in process is lengthened to such an extent that the epithelial structures will probably cover the surface of the wound before the granulation tissue has risen to the skin level, in which event a depressed scar is the inevitable result. A similar outcome may likewise occur from an undue energy on the part of the surgeon in a too excessive or a too frequent repression of the granulation tissue by means of cauterization. A study of this class of wounds during the healing process and especially a study of the habits and behavior of granulation tissue is, therefore, a very necessary part of the surgical technic, and should provide a satisfactory means of avoiding ugly scars in many instances at least.

In a personal experience of more than 500 mastoid operations, and 11 frontal sinus cases treated by the open method, the entire after treatment of which the writer carried out in nearly every instance, opportunity was offered for rather conclusive observation concerning the behavior of granulation tissue under almost all conditions that are likely to occur during the healing of open wounds. The observations thus made have been of great service to the writer in securing a more rapid closure of the frontal sinus, and in avoiding to a great extent the resulting depression and ugly scars. The conditions under which healthy osseous granulation best thrives were noted, as well as the states of the wound which were most favorable to over-activity and necrosis of the granulation structures. The growth of this tissue in osseous wounds, as thus observed, seemed often freakish, and its behavior such that a useful interpretation of the same was frequently impossible. Nevertheless in the majority of instances its development was most natural and vigorous under the following conditions: (a) When the wound was sterile, or at least in a great measure approached a state of sterility. (b) When the

granulations were moistened only with the exudates which were present, but were neither submerged in secretion on the one hand, nor too greatly desiccated on the other. (c) When there was at no time a too pronounced or too prolonged pressure from the gauze or other dressing which had been inserted into the cavity. (d) When the primary operation on the sinus had been thorough in every respect and the operated osseous wound had been left smooth and free from any pockets capable of retaining subsequent secretion.

With a thoroughly prepared osseous wound as above described, clinical observation teaches that the subsequent behavior of the granulation buds may be almost entirely controlled at the will of the surgeon, who may by means of gauze packing or the application of caustics repress the growth of new tissues, if he so desires, until the whole cavity has been covered with epithelial structures, and the wound has entirely healed, leaving a maximum of depression and scar tissue. On the other hand, it is entirely possible, in most instances at least, to secure in a satisfactory way, a type of vigorous granulation structure which will rapidly obliterate the sinus by filling it in to a level with the skin structures, which latter are then, and not till then, allowed to bridge across the wound. Healing of the sinus by the open method of treatment will, therefore, leave a minimum of depression and scar tissue, provided the surgeon will exercise that control over the granulation structures which experience has abundantly shown may be exercised almost at the operator's will. The desired pliability of the new tissue is based almost entirely upon the surgical principles of efficient drainage, absence of undue pressure upon the granulation buds and a properly prepared wound to begin with.

Clinical observation of the processes of the healing of bone wounds by granulation also teaches that, if adequate drainage is provided, and the wound is secured against exposure to outside pathogenic bacteria, a sufficient degree of sterility will usually be secured to encourage rapid and successful filling-in of the cavity. The surgical principles involved in the provision of such ideal drainage for this class of wound will also insure against the accumulation of exudates and the consequent harmful flooding of the new tissue. In sinus surgery, therefore, successful restoration of the cavity, or its complete obliteration, need scarcely be

expected in any case in which the external wound is immediately closed, unless the operator is absolutely convinced that the drain canal he has provided is ample to insure against retention of fluids and the inevitable infection of the same, should they be permitted to stagnate. Even though such an ideal drainage is established at the time of the operation, if immediate closure of the wound is practiced, two things may speedily happen to cause failure to cure. These are, 1st, the too rapid closure of the drain canal by granulation tissue, and 2d, the formation of pockets through faulty behavior on the part of the granulation and the consequent retention of pus in the sinus, even though an adequate drainage canal remains intact. This latter condition is one which, in the writer's opinion, is responsible for the failure in many cases which have not improved, and in which secondary operations have, therefore, been necessary. It has already been stated that the behavior of granulation tissue, even when all conditions are apparently normal, is sometimes freakish and unaccountable. For example, the writer has often observed during the post-operative treatment of open wounds in the several head sinuses that occasionally, when all had been progressing perfectly well, if the patient be not seen for 36 or 48 hours, granulations of unaccountable size would spring up in some part of the wound and grow toward similar granulations in another part with such amazing rapidity that adhesion between the opposing buds took place, a wall was constructed across the healing cavity, and, before the surgeon was scarcely aware of what had occurred, some portion of the wound was already badly drained, the granulations rank and perhaps necrotic, and unless the faulty conditions thus established were promptly corrected, failure to heal the wound was inevitable. Of course should such an occurrence take place in an open cavity, every part of which can be inspected at any time, and failure should not result because the error is easily detected and corrected; but when the external wound has been immediately closed at the time of the operation, or when intra-nasal drainage only has been practiced, the surgeon can only surmise what is going wrong, and is, therefore, helpless to correct any freaks of healing by granulation, except by means involving a secondary operation.



THE FURTHER REPORT OF A CASE OF TRACHEAL  
SCLEROMA, PREVIOUSLY PRESENTED  
TO THIS ASSOCIATION.

BY EMIL MAYER, M. D.

The unusual occurrence of this affection, its still more unusual location in the larynx primarily, and its cure by a novel method of application of the Roentgen rays, impel me to present the further history of this case reported to this association two years ago. (Transactions 1906, p. 70.) At that time the subject of laryngeal scleroma was fully dealt with and these details may be omitted here.

The patient's history in brief then was: A female, aged sixteen, born in the province of Wilna, Russian Poland, three years in the United States, hoarse all the time she had been in this country and of late with increasing dyspnoea, presented herself at my clinic in the dispensary of Mt. Sinai Hospital. She had masses of secretion in her nose with crust formation, without offensive odor. There was no loss of weight. There was a subglottic mass below the right cord protruding into the larynx, grayish in appearance and diminishing its lumen by about one-half. Portions of the laryngeal growth were removed. These were of pale tissue, translucent to a degree, resembling soft adenoid tissue. The report of Dr. F. S. Mandlebaum, pathologist to the hospital, on the subglottic growth, is given in full in the former paper (which included the bacteriological examination) and was to the effect that the presence of hyaline bodies and Mikulicz cells, together with the bacilli obtained by culture, the diagnosis of scleroma (rhinoscleroma) was fully established.

After the removal of this growth endolaryngeally as far as possible, her condition both as to hoarseness and dyspnoea improved perceptibly, so that while there was still a small grayish exudate below the right cord, this gave rise to no symptoms.

In regard to the eventual outcome of these cases I then said:

"The prognosis as to recovery is bad. The general health may not be impaired save from such symptoms as the varying stenosis occasions. The disease may progress and suddenly remain stationary for several years. Sooner or later there is recurrence.



"As to treatment, the external conditions have been much benefited by the use of the Roentgen rays.

"For the laryngeal scleroma, removal endolaryngeally has at times benefited the patient, as has the pressure of an O'Dwyer tube both before and after tracheotomy, and the increasing sized Schrotter tubes have been useful for the time being. Slow dilatation may be of value by means of the writer's hollow introducer and the O'Dwyer tube.

"From a study of this subject we are led to conclude that in scleroma we have a chronic incurable affection, contagious to a certain degree."

With this as the sum of our knowledge as to the treatment of this affection, there was a serious problem before us when ten months later there was a recurrence of the growth, and she presented herself hoarse again and with dyspnoea very marked, gradually becoming worse. Noting the remarkable results obtained in other cases of external rhinoscleroma by the use of the Roentgen rays, it was felt that their application direct to the part through an opening in the trachea might have a curative effect. In any event, tracheotomy was becoming daily more necessary, and if the benefit was not obtained she would be compelled to wear the tube, as had others before her, to prolong her life.

The patient readily consented to the operation and subsequent direct treatment by the Roentgen rays, and on March 25, 1907, she was admitted to Mt. Sinai Hospital, service of Dr. A. G. Gerster. From the hospital records I present here her condition and the detail of treatment instituted.

A. W., admitted to Mt. Sinai Hospital, March 25, 1907, for gradually increasing dyspnoea, gasping for breath, and hoarseness. Examined March 28 by Dr. E. Mayer.

*Nose:* Half way back on the left is a grayish white mass which bleeds very readily when touched and is covered with thick secretion. This mass does not occlude the nose on that side. On the right side is another similar mass partially occluding the nose.

*Larynx and Trachea:* In the trachea, below the cords, on a level with the cricoid cartilage, is a mass attached to the wall of the trachea and extending downward. This is an extension;

and continuation of a condition existing for over a year under my observation, and is a scleroma of the larynx verified by the pathologist's report. The diagnosis is increasing tracheal stenosis, due to scleroma with secondary involvement of the intranasal mucous membrane.

I advised tracheotomy and X-ray treatment direct to the exposed growth.

The physical examination of March 30 showed a fairly well nourished young woman; facies somewhat dyspnoeic; breathes with some difficulty; gasping, and she is hoarse.

*Lungs:* Respiration stridulous. Respiration difficult and considerably prolonged in both phases. Percussion signs somewhat diminished, especially posteriorly at the bases. Apices free. Vocal fremitus diminished. Voice and whispering sound diminished below in front and over the bases posteriorly. There are numerous sonorous and whistling rales over both lungs, anteriorly and posteriorly. In the axillary regions and over the bases posteriorly there are numerous moist rales, increased after coughing.

On March 30 Dr. A. G. Gerster did an extensive tracheotomy. The incision extended  $3\frac{1}{2}$  inches long from the lower edge of the thyroid cartilage to about  $\frac{3}{4}$  inch above the sternal notch. The isthmus of the thyroid was tied off in two places and divided between the ligatures. The cricoid cartilages and tracheal rings were incised, bringing into view the tumefaction of the posterior and lateral aspects of the trachea. The new growth was grayish white in appearance, resembling soft mucous polypi readily breaking down, and extending  $1\frac{1}{2}$  inches below the larynx. A large tube was introduced and packing about it to keep the wound open.

On April 1 the packing and tube were removed, edges of the wound held apart by retractors, and under chloroform there was a five-minute exposure to high frequency current at a distance of eight inches.

April 3—Has had frequent paroxysms of coughing. Whenever she sits up, is fed, dressed, or in any way disturbed, these coughing spells occur, and frequently vomiting ensues. These phenomena were not present before X-ray treatment.

April 9, 12, 15 and 17—X-ray treatment by Dr. Stern, five to six minutes' exposure.

April 20—The growth has greatly decreased in size and the trachea looks much wider. Has had no more vomiting, and her tracheal reflexes are less active. It is now possible to dress the wound and clean the tube without exciting coughing spells as formerly. X-rayed 22d and 24th.

May 10—Patient has had three or four X-ray exposures a week. An examination of her larynx by myself showed an absence of any tumefaction, and I recommended permanent withdrawal of the tracheotomy tube and further treatment externally. Voice now normal and no dyspnoea.

On May 11 the tube was removed; she remained until May 17, when she was discharged from the hospital cured. During her stay in the hospital her temperature never rose above  $100\frac{1}{2}$  degrees. After the first few times no chloroform was given prior to X-ray treatment. Urinalysis frequently made showed neither albumin nor sugar.

Examination April 12, 1908. Until the present time the patient has been in normal condition, without dyspnoea, hoarseness or cough. She has been attending to her work with regularity, and only appears now at my request for examination. Her general condition is good, respiration normal, and she says that except for occasional nasal obstruction she is entirely well.

Examination of the nose. On the right side, about the center of the inferior turbinate, is a small pink mass, which probably interferes slightly with her breathing. On the left side, on the septum, at the floor, is a roundish mass, about the thickness of a small hazel nut. This was removed with a cold wire snare and was about one inch in length. There was but a slight bleeding.

Examination of the larynx and trachea showed perfectly normal conditions.

One year has now elapsed since her operation and subsequent treatment, and no sign of recurrence is visible within the larynx or trachea, and we may justly consider her as cured of her most threatening condition.

The benefits of the X-ray application to external scleromas have been known, and now we can show its utility in local conditions that we are able to reach in a direct manner. Should a recurrence be present we might reasonably expect good results

by external application of the X-ray over the cicatrix, this not presenting the same resistance as the normal tissues would; that failing, it would be easy to reopen the old wound and make local application again.

Finally, I wish to express my thanks to Dr. A. G. Gerster and to Dr. Samuel Stern for their cordial co-operation and active interest in the surgical and X-ray treatment of this case.

*Discussion.*

Dr. H. L. Swain: It is interesting to have the final results in this case, and I wish to thank Dr. Mayer for bringing it before us again. Too often we get only the beginning of cases and the final result is lost sight of.

A CASE OF ABSCESS OF THE LARYNX FOLLOWING  
PNEUMONIA.

BY T. MELVILLE HARDIE, M. D.

Joseph M., Italian, aged forty-three, street laborer, married, four children. Was admitted to St. Luke's Hospital April 16, 1907, service of Dr. H. B. Favill, suffering from a pneumonia of the upper right lobe. T., 102; P., 108; R., 40. He had always enjoyed good health. His habits are good, but he drinks beer freely.

April 18—T., 104.4; P., 128; R., 44. Dulness on right side posteriorly, about the level of the fourth dorsal vertebra; tubular breathing and a few crepitant rales.

April 19—Dulness and crepitant rales over right upper lobe anteriorly and posteriorly. Resolution April 25. T., 100.5; P., 120; R., 36.

April 26—Coughing frequent; some pain in throat, and Dr. Favill ordered steam inhalations and a cold compress, with 1/20 grain apomorphia and 1/24 grain of heroin.

April 27—An ice bag was substituted for the compress. As there was some dyspnoea, which was evidently increasing rapidly, I was asked to examine the larynx. The patient was very weak and in great distress. Examination showed oedema of the larynx; right and left ventricular bands, aryepiglottic fold on left side, posterior wall of larynx. I made several deep punctures and ordered adrenalin spray 1:4000, compound tincture of benzoin steam inhalations and continuance of the ice bag, with considerable relief to breathing for several hours.

April 28—No oedema in the upper larynx, but the swelling below the vocal cords was so extensive that dyspnoea was again very urgent. Intubation gave no relief, and tracheotomy, under cocaine anesthesia, was done. After exposure of the region of the cricoid and upper trachea it was seen that an irregular enlargement, more pronounced on the left side, prevented differentiation of the parts involved. Incision in the middle line opened an abscess discharging two or three teaspoonfuls of pus and at the

same time entered the trachea. A smear and culture showed diplococci (probably pneumococci) in pure culture. The tube was inserted and the patient returned to bed and stimulated. He was desperately ill for the next twenty-four hours, the high range of T. P. and R. being 104.3, 156, 40; but by May 1 his temperature was practically normal, and on May 31 the tube was removed and the larynx apparently in normal condition.

Some excerpts from the temperature, pulse and respiration record are appended.

The case is reported because of the unusual etiology of the abscess. I have been unable to find one reported following pneumonia. Two very interesting features were the extensive cedema in the upper larynx and the fact that the damage done to the cartilage was not severe enough to cause an obvious necrosis.

TABLE OF TEMPERATURE, PULSE, RESPIRATION.

|           |   |       |                |         |    |
|-----------|---|-------|----------------|---------|----|
| April 16, | 4 | P. M. | 100.4          | 114     | 26 |
|           | 9 | P. M. | 102            | 108     | 40 |
| April 17, | 2 | A. M. | 105            | 118     | 40 |
|           | 6 | A. M. | 105.8          | 128     | 44 |
|           | 1 | P. M. | 104.8          | 130     | 48 |
|           | 9 | P. M. | 104            | 120     | 44 |
| April 18, | 1 | A. M. | 103.8          | 112     | 48 |
|           | 1 | P. M. | 104.4          | 120     | 44 |
|           | 9 | P. M. | 103.6          | 128     | 44 |
| April 19, | 9 | A. M. | 103.8          | 138     | 48 |
|           | 9 | P. M. | 103.4          | 128     | 48 |
| April 20, | 5 | A. M. | 103.6          | 136     | 48 |
|           | 9 | P. M. | 103.4          | 152     | 48 |
| April 21, | 2 | P. M. | 100.2          | 128     | 38 |
| April 22, | 1 | P. M. | 98.2           | 100     | 30 |
| April 23, | 1 | P. M. | 100            | 110     | 36 |
| April 24, | 1 | A. M. | 97             | 100     | 32 |
|           | 1 | P. M. | 102.4          | 124     | 48 |
| April 25, | 1 | P. M. | 100.5          | 120     | 36 |
| April 26, | 5 | A. M. | 97             | 104     | 36 |
|           | 5 | P. M. | 100.6          | 106     | 32 |
| April 27, | 5 | A. M. | 97             | 100     | 38 |
|           | 3 | P. M. | Deep punctures | larynx. |    |
|           | 5 | P. M. | 102.6          | 116     | 38 |
|           | 9 | P. M. | 103.5          | 128     | 32 |



|           |         |                                  |     |    |
|-----------|---------|----------------------------------|-----|----|
| April 28, | 9 A. M. | 100.8                            | 120 | 36 |
|           | 5 P. M. | Tracheotomy. Evacuation abscess. |     |    |
|           | 6 P. M. |                                  | 136 |    |
|           | 9 P. M. | 102.4                            | 110 | 34 |
| April 29, | 5 A. M. | 103.7                            | 146 | 38 |
|           | 7 A. M. |                                  | 156 |    |
|           | 1 P. M. | 104.3                            | 142 | 38 |
|           | 5 P. M. | 102.6                            | 138 | 40 |
|           | 9 P. M. | 98                               | 128 | 38 |
| April 30, | 9 A. M. | 100                              | 128 | 30 |
|           | 1 P. M. | 100.4                            | 120 | 26 |
|           | 9 P. M. | 101                              | 124 | 36 |
| May 1,    | 9 A. M. | 98.3                             | 105 | 28 |
|           | 5 P. M. | 99.9                             | 108 | 32 |
| May 2,    | 1 P. M. | 99.6                             | 112 | 32 |
| May 3,    | 1 P. M. | 100                              | 100 | 24 |
| May 6,    | 1 P. M. | 99.4                             | 102 | 24 |
| May 9,    | 5 P. M. | 98.7                             | 98  | 20 |

## BLOOD COUNT.

|           |       |           |         |        |
|-----------|-------|-----------|---------|--------|
| April 17, | Reds. | 5,000,000 | Whites. | 16,500 |
| 18,       |       |           | Whites. | 16,150 |
| 20,       |       |           | Whites. | 22,500 |
| 24,       |       |           | Whites. | 26,000 |
| 26,       |       |           | Whites. | 29,400 |
| May 7,    |       |           | Whites. | 16,500 |

*Discussion.*

Dr. H. L. Wagner: It is interesting to see the different effects the pneumococcus germs have on the mucous membrane and adjacent tissues. I am reminded of a case in which the pneumococcus germ of Friedländer was found following a pneumonic attack in an acute swelling of the inside of the nose, both sides of the septum, and then an external infection giving quite a true picture of erysipelas. From this inflamed area a pure culture was obtained similar to that found in the nose and also on the septum of the convalescing patient.

## A SERIES OF PAPERS ON SINUS DISEASE.

### *First Paper.*

#### REPORT OF A CASE OF CYST OF THE FRONTAL SINUS, COMMUNICATING WITH THE FRONTAL LOBE.

BY CLEMENT F. THEISEN, M. D.

The following case, because of the apparent rarity of one feature of it, is considered of sufficient interest to be reported.

Cysts of the frontal sinus, particularly mucocoeles, are not so very rare, but it is fairly uncommon to find an absence of a large part of the posterior or cerebral wall of the sinus.

The patient, Miss M. B., aged thirty-four years, consulted the writer July 27, 1907. The only interesting point in her history is a severe fall, striking her head, when she was nine years old. She stated that she was unconscious for three days at that time. From that time she has been a sufferer from severe headaches, more intense on the left side. She is a worker in a textile mill.

In 1905 she had a nasal operation, the exact nature of which she could not tell me. This gave her some relief. For a number of years she had had a discharge from the left nostril. This, she believed, followed some work on the teeth on that side. About a year before consulting the writer her left maxillary antrum had been opened through the canine fossa, and for a time she thought she was a little better.

Her physician, Dr. J. B. Ledlie, of Saratoga, who referred Miss B. to me, told me that since July, 1907, her headaches had been unusually severe, particularly on the left side. She also complained of a pressure and fulness over the eyes. There was slight ptosis of the left eyelid, but no orbital swelling nor displacement of the eyeball. When she turned her head quickly to the left, or bent forward suddenly, she had great vertigo, and would fall if she could not get hold of some support. Pupils reacted to light and accommodation.

On examination nothing particularly wrong could be discovered in the nose. There was a small amount of a thin, mucopurulent secretion in the left nostril, which came from the antrum. On transillumination the left antral region was slightly darker than the right, but there was good illumination of both, and the pupils were also illuminated.

The left frontal region was darker than the right. This region was also slightly more prominent.

The headaches were becoming so frequent and severe that she was anxious to have any operation that would relieve her. While there was no direct evidence of a purulent frontal sinusitis, the result of transillumination, with the headaches confined to that side, appeared to the writer sufficiently suspicious symptoms to warrant an operation. This was performed in the Saratoga Hospital in August, 1907. The usual incision through the eyebrow was made and a portion of the anterior wall of the sinus carefully removed. A fluctuating tumor, having the typical appearance of a cyst, presented in the opening. It appeared to contain considerable fluid. An incision was made through the cyst wall and a large amount of a fluid very much like thin mucus escaped. This was, unfortunately, lost, so that no examination could be made of it. The anterior wall, as well as the floor of the sinus, was found intact, but a considerable portion of the posterior wall was missing, so that the pulsation of the meningeal vessels could be seen. The cyst wall, in fact, appeared to extend through to the frontal lobe.

The sinus was large and somewhat dilated.

The anterior ethmoid cells were then investigated through a separate incision, following the method employed by Coakley. No pus and very little bone necrosis was found. At this time the patient went into a condition of collapse, and it was with great difficulty that we succeeded in reviving her. We could not get a probe through into the nose from the frontal sinus, so that, as in cases of mucocele, the ostium was probably occluded. As much of the cyst wall as possible was removed, and the frontal wound, with the exception of a small opening in the inner angle, closed. A small wick of gauze was carried into the sinus for drainage, but in about ten days the gauze was no longer inserted and the

wound allowed to close. The patient made an uninterrupted recovery, and now, about ten months after the operation, is apparently perfectly well. She works twelve to fourteen hours a day and has no headaches.

I received a letter from her physician, Dr. Ledlie, a week ago, in which he stated that she was all right and able to do her work every day.

A. Logan Turner, in his interesting paper, mucocoele of the accessory nasal sinuses, reports two cases in which, as in the writer's case, absorption of part of the posterior wall of the sinus had taken place.

In six out of seven cases reported by him absorption of a part or the whole floor of the sinus had taken place.

I will briefly report the two cases in which part of the posterior wall was missing.

Case I.—C. S., a woman, aged forty-three years. During the operation it was found that a portion of the posterior, or cerebral, wall the size of a half-crown had been absorbed, so that the dura mater was seen pulsating.

Six months after the operation there was no discharge from the cavity. A drainage tube was carried into the nose at the time of the operation and worn for six weeks. It was then removed, and the patient was instructed to wash out the sinus with a curved canula.

Case II.—In the second case reported by Turner, a woman aged thirty-seven years, the dura was found exposed over an area as large as a sixpence. The right eye-ball was displaced downward, outward and forward. This was so in the other case also.

In the writer's case there was no orbital swelling, a common symptom of cyst of the frontal sinus. The anterior ethmoid cells are frequently involved. Patients, as a rule, do not complain of much pain unless infection of the sinus takes place.

In Turner's cases there was displacement of the eye-ball in seven out of ten.

The displacement depends upon the size of the orbital swelling. Turner states that when the affected sinus extends backwards for a considerable distance along the roof of the orbit, and the mucous

contents escape into the orbital cavity after the floor has been absorbed, the eye-ball is pushed forward and downward. A previous catarrhal condition of the nasal mucous membrane is the probable cause of such cases.

The ostium of the sinus becomes occluded, causing an accumulation of mucus in the frontal sinus and a gradual thinning and absorption of one or more of the bony walls.

Killian believes that traumatism is an important cause for the development of such cysts.

The writer was only able to find records of a few other cases of cysts of the frontal sinus, in which a large portion of the posterior wall of the sinus was absent, in the literature of the past ten years.

Casali has reported the case of a man, aged forty-one years, who had had a fall, striking the right side of the head, eighteen years before consulting him. There was a fluctuating swelling above the supraorbital ridge, extending down to the middle of the upper eyelid.

There was also a right exophthalmos.

A clear, sterile fluid was obtained on puncturing.

A radical operation was performed. After the anterior cyst wall was removed the pulsation of the meningeal arteries could be seen. Two hundred ccm. of clear fluid was evacuated.

Histological examination showed that this was probably not a cyst, but an enormously dilated frontal sinus. Another interesting case has been reported by Mayer.

This patient, aged fifty-three years, had been twice operated on for a cyst of the frontal sinus.

A large amount of a yellowish fluid had been evacuated, and it was found that the posterior wall of the sinus was missing.

The patient survived the first operation, but in a year there was a recurrence, and the second operation had a fatal termination. In the region of the right frontal lobe there was a deep depression produced by the cyst.

In a case reported by Zamazal of severe left-sided headaches for two years there was a sudden flow of pus and blood from the nose just before the patient died. There was an abscess in the

region of the frontal lobe which broke through into the frontal sinus.

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*Second Paper.*

## NOTES UPON TWO UNUSUAL FRONTAL SINUS CASES.

BY J. PRICE BROWN, M. D.

Case I.—Chronic Purulent Frontal Sinusitis, with External Fistula for Nearly Two Years—Operation—Recovery.

February 28, 1907, Miss M. F., was referred by Dr. Black, of Paisley, for treatment.

*History*—In September, 1904, two and a half years previously, while attending college in Toronto, she had an attack of fever, resulting in frontal abscess on the right side. This was lanced by the attending physician in consultation. There was a free discharge of very foul pus. Under treatment, healing took place in a month, and she went home to Paisley. Toward Christmas swelling of the forehead returned, and Dr. Black reopened it. From that time until the following March discharge was almost constant, and she was brought to the city again for further advice. The consultants decided that it was a case of frontal sinus disease, requiring immediate operation. Consequently, under general anesthesia, it was opened through the floor, the excision extending up into the superciliary ridge. An opening was also made downward through the region of the fronto-nasal passage into the nose, and an attempt was made to secure nasal drainage. The result was not satisfactory. The fronto-nasal passage closed and the external opening refused to heal. The discharge would sometimes almost cease and again for days come away more freely. Four months later the sinus was still open. Then, to secure more efficient drainage and lavage, the doctor inserted into the opening a short rubber tube, through which the cavity was washed out regularly. It was worn for nearly a year and then discarded. Subsequent to this the patient would sometimes probe the cavity to obtain a better outlet to the pus, or the doctor would make the

passage freer with the lance. Finally, after having almost continuous discharge for two years, a sudden aggravation of all the symptoms occurred and the patient was referred to me.

*Examination.*—Left eye almost closed, the eyelid swollen and inflamed, the swelling extending upward over the superciliary ridge and including the inner canthus. The surface beneath the ridge was irregular, pultaceous and darkly suffused in color, with pus exuding from a point immediately over the site of the sinus operation. There was a good deal of pain over the region, accompanied by headache. An X-ray picture, the one shown to-day, did not reveal much, save a darkened shadow on the affected side, and the opening in the bony wall from the previous operation upon the sinus. There was no shadow in the maxillary region.

Intranasally there was little if any pus visible, but the middle and inferior turbinals on that side closed up the passage. The patient was placed in the Western Hospital.

As preparatory operation, the anterior end of the middle turbinal and a portion of the inferior turbinal were removed. This had to be done under general anesthesia, as the young lady was too nervous to submit to any operative work under local anesthesia.

How to operate upon the sinus was the next question. I was unwilling to attempt a radical operation after Coakley's plan for cosmetic reasons. Killian had not yet visited the country, and at that time I knew little of his operation. At the same time, I was strongly impressed with Fletcher Ingal's intranasal treatment and use of gold tubes. As in his cases, the desideratum of nasal drainage was secured, something which Killian now insists upon as essential; the thought struck me that a gold tube inserted from the frontal sinus downward through an enlarged fronto-nasal passage might be equally effectual. The external wound could then be closed, irrigation would be practised through the tube, and the latter eventually removed through the nose. So an operation was done under general anesthesia. The eyebrow was not shaved. The incision was made through the centre of the eyebrow from the middle inward to the median line. The periosteum was then raised directly upward over the inner end of the sinus, and a rectangular piece of the outer table chiselled out

above the superciliary ridge, the long direction being upward. The object was, while leaving a minimum of deformity, to permit a straight drill to be passed downward to destroy the anterior ethmoid cells. The incision and the removal of the bony wall being entirely distinct from the site of the former surgeon's operation.

A quantity of pus and blood welled out on opening the sinus, the cavity was curetted and freely irrigated. Then, the infundibular point being found, successive hand drills were used, until this larger one could be inserted and a free entrance made into the nose, external to the septum and in the region of the anterior end of the middle turbinal. A curved forceps was then passed downward through the passage and after repeated irrigations this gold tube was inserted from above.

The external wound was at once closed. It healed in a very few days by first intention, but to my chagrin I found it impossible to irrigate the sinus through the tube as intended. The girl had passed through so many operations that she became hysterical the moment an attempt was made to pass any instrument, even the end of a syringe, into the nasal passage. Another point: while the discharge from the old fistula, which I had not disturbed at all, materially lessened, it did not cease, while the area still continued to be inflamed and tender.

Hence, twenty-three days after the insertion of the gold tube, I operated again. This time I made a similar incision to the former one, parallel to it, but below the eyebrow, and directly through the central point of discharge. From the opening two or three bits of dead bone were taken. The outer tissues were then raised over the entire extent of the original operation, the gold tube was removed from above and a large rubber tube inserted in its place; the upper end being curved on itself, to lie on the floor of the sinus, and the lower end extending beyond the nostril. The outer incision was then closed as before.

This time the result was all that could be desired. Although the tissues through which the incision had been made were inflamed, darkly suffused and spongy, the healing was again by first intention.

The sinus was for a time irrigated regularly twice a day with

warm sterilized water. The discharge gradually diminished and in a few weeks it practically ceased, when the tube was removed.

From then until now there has been no return of the disease and the patient is quite well. This I think is due to the fact



Case I.

that the wearing of the gold tube for twenty-three days secured permanent and effectual drainage from the affected cavity.

This photograph, taken last month, one year after the operation, indicates how small a deformity has resulted from the operation.

Case II. Traumatic Frontal Sinus Disease—Operation—Recovery.

*History*—In April, 1907, Mr. J. F., age 35 years, married, received a severe blow from the head of a horse; the region

struck was that of the right temple at the outer end of the frontal sinus. After some weeks this was followed by a swelling beneath the right eye-brow and tenderness on pressure. Later on both these symptoms subsided. In the following August, also, he had the misfortune to strike his forehead against a stone wall, resulting in an abscess which was lanced. In due time this healed, leaving a surface depression.

Early in January, however, his head began to be sore, followed by pains in the region of the frontal sinus, shooting backward toward the occiput. This was accompanied by the sensation of fullness and pressure in the forehead on that side, the condition being always aggravated at night time. Toward the end of January, swelling beneath the right eyebrow also returned, with marked tenderness on pressure over the central portion of the sinus, while sleep at night became almost impossible. During this period the patient was under Dr. Wilson's care; but medical treatment failing to afford relief, on February 22nd he referred him to me and he was placed in the Western Hospital for operation.

*Examination.*—Head feels hot continually, temperature ranges between 99 and 100, soils two or three handkerchiefs a day from right nasal hemorrhage, complains of intolerable bursting head pains and entire absence of sleep. There is also drooping of tissues beneath the floor of the sinus and much tenderness on pressure.

The right side of the septum was much thickened and spongy, the anterior end of the middle turbinal also was enlarged. Both were hemorrhagic, but there was very little pus visible.

My first effort was to give relief by intranasal treatment; so, under cocaine and adrenalin, I reduced, by operation, the hypertrophied tissues, but failed either to penetrate the infundibulum or give relief to the frontal sinus.

So, five days later, under chloroform anesthesia, assisted by Dr. Wilson, I did a modified Ogston-Luc operation; that is, after chiselling the usual opening into the frontal sinus above the superciliary ridge, instead of merely dilating the fronto-nasal passage and putting in a small drainage tube, I drilled a larger opening, destroying the anterior ethmoid cells; and placed a rubber drain-

age tube from the floor of the frontal sinus down through the nasal passage and out through the nostril.

On opening the sinus there was very free hemorrhage, accompanied by purulent matter. The blood vessels were enlarged. The mucous membrane, particularly on the anterior wall, was swollen and spongy, that on the cerebral side of the cavity being little affected. After curetting away freely all adventitious tissue, the sinus was washed out with hot boracic acid solution, then swabbed with peroxide of hydrogen, and the operation completed. The drainage tube was next inserted and lavage repeated both from above and below. Finally the wound was closed by silk sutures and padded and bound.

A swab was taken from the sinus at the time of operation. Examination revealed pus cells mixed with blood. Another swab taken ten days later from the sinus, quite free from blood, proved the disease to be one of pure pneumococcus infection.

Points with regard to temperature: Immediately before the operation the temperature was 99 1-5; three hours later it was 101 1-5, and still two hours later, at eight o'clock in the evening, it had risen to 104½ and was accompanied by delirium. Throughout the night the patient could with difficulty be kept in bed. In the morning the temperature dropped to 101 1-5, the delirium was over and did not again appear. Still the second night the temperature rose to 103 4-5, subsequent to which time it was almost invariably normal.

The respiration throughout was above normal, always between 20 and 25 per minute. At the time of operation it was 24 per minute. The pulse also from the first was rapid, running from 100 to 120 per minute.

Possibly the rapid pulse and respiration might be due to a general pneumococcic infection with local focus within the frontal sinus. With regard to after treatment, the intention was to wash out the sinus, regularly, through the wide drainage tube, by means of a small-tipped instrument. This was carried on effectually for some days; but possibly due to the high temperature of the first forty hours, the wound did not heal by first intention. The stitches loosened and were removed and the whole wound opened.



Granulation commenced and the lavage was then done from above; sixteen days after the operation the tube was removed through the nose, and as the sinus wound was closing a small rubber tube was placed at its inner end and the lavage continued. Latterly, no other treatment was followed, save to cover



Case II.

over the small opening of the wound and tube with adhesive plaster, to be replaced after each irrigation. The boracic solution came through the nostril freely and was soon free from pus. By April 16 the discharge from the sinus had ceased, so the little tube was removed, irrigation dispensed with, and the wound allowed to close—the patient being warned against all nose blowing until entire healing had taken place.

One point is worthy of note as brought out in this case, that is the advantage of a reversed Valsalva in cleansing the fronto-nasal passage during the process of healing. While the little

tube was in situ in the forehead, although there was no external discharge whatever, the air would whistle through the passage with each forcible effort to draw backwards; and with the effort, any mucus lodged within the passage would be removed. When the little tube was finally taken out the Valsalva drawing backward was forbidden, in order to favor surface healing. The present condition of the patient with a healed sinus is well marked in this photograph, which was taken only a few days ago.

In closing, I might make one other remark, which applies to both of these cases. Although the anterior ethmoid cells were engorged and the middle turbinal pressed tightly against the septum, yet in neither case was there any indication whatever of the antrum of Highmore being affected.

REPORT OF CASE: SINUSITIS-PYÆMIA—SEVERE  
CEREBRAL SYMPTOMS RELIEVED BY  
OPERATION—DEATH—AUTOPSY.

BY LEWIS A. COFFIN, M.D.

The following history was offered as a paper to be read at this meeting, while the patient still lived and offered some hope of ultimate recovery from a Killian operation for the relief of an empyæmic condition of all the accessory cavities of the right side of the head, from which the patient, on admission to the Manhattan Eye, Ear and Throat Hospital, showed such severe cerebral symptoms as to all but forbid operation:

*History.*—Mrs. K. S. was admitted to the Eye Ward of the hospital November 29, 1907. On admission, was unconscious, restless and delirious. Temp. 102 3-5 Fahr. Pulse 104. Resp. 24. She had two convulsive seizures, affecting principally the right side of the body shortly after admission, and the physician sending her reported that she had had three convulsions at intervals during the previous night.

Dr. S. M. Payne, of Dr. David Webster's staff, to whom the patient had been sent, diagnosed an orbital cellulitis, but, suspecting a more serious underlying condition, asked Dr. Webster and me to see the case with him. The right eye was slightly exophthalmic, conjunctiva injected, slight dilatation of the pupils of both eyes and the ophthalmoscope showed a neuro-retinitis. The entire right side of the head was swollen, the swelling extending downward, back of the ear, to about the middle of the mastoid process. Marked evidence of pain was shown when pressure was put upon the roof of the orbit. All tissues of the nose were swollen, and I could not see pus in that region. However, all agreed that an empyæmic condition of the frontal and ethmoidal sinuses was back of the ocular trouble. Dr. Edwin Zabriskie was asked to do a lumbar puncture and give an opinion as to the brain condition. He expressed the belief that the right half of the brain was about becoming infected, the infection taking the

form of a meningitis. This conclusion was based on the fact of the stupor, neuro-retinitis, unilateral convulsions and clear spinal fluid. Operation was decided upon and patient ordered ready. The previous history subsequently obtained from the patient and her sister was as follows: The patient was thirty-three years of age, married, and had three children. Has had, two attacks of pneumonia; otherwise has had no serious illness. There is no history of nasal catarrh, so far as sister knows, but for years she has complained of neuralgia over the right eye. Family history negative. On November 21, eight days before admission to the hospital, she complained of a severe frontal headache and neuralgia. Notwithstanding efforts of her physician the pain increased until, on November 28, the right eye began to protrude and there was considerable swelling of the orbital tissues. On the morning of November 29 she fell on way to bathroom and had a slight convulsion. Incision was made in the orbit, above and outside of the eyeball. No pus was found, and the patient was sent to Dr. Payne as above stated.

A Killian operation was done on the same afternoon. Nothing unusual was noted at the time, except the high pressure under which the pus was found to be both in the frontal sinus and the ethmoidal cells. It sort of popped out when the retaining walls were removed, due possibly to retained gas. A peculiar formation of the frontal sinus was a prolongation upward from about the middle of what would be the ordinary height of the upper border of the sinus of a process not larger than a slate pencil. This process was one inch in length and necessitated an auxiliary incision. It was filled with granulations. The bone of the posterior wall looked sound and healthy.

Patient was returned to ward in good condition. Pus and scraping showed a mixed variety of infective agents, streptococci being present in short chains.

During the nine days following the temperature ranged from 103.4-5 to 100, the variations less marked and the average lower toward the end of the period. This temperature was accounted for by moderately severe bronchitis and the feeling that probably her sinus involvement was a complication of an influenza. The swelling about head rapidly subsided, leaving two circumscribed swellings; one over right temple, broad and flat, and one just to

right of median line, at about the juncture of the frontal and parietal bones. This was about the size of a filbert and thought to be a wen. Exploration of swelling over temple by largest aspirating needle got no pus, and I put it down as a glandular swelling. Consciousness was gradually regained, and on November 30, the day after the operation, at about 5 P. M., she was conscious and quite rational. In the meantime she had had two convulsions. She complained, as she continued to do for many days, of severe pain in back of head, and neck and right leg. Was at times for three or four days delirious and irrational. Had cough and blood-streaked sputa. Five days after operation patient began to menstruate. However, on December 7, eight days after operation, everything looked better. Patient entirely rational. Cough had ceased. Was sleeping fairly well and taking nourishment. Not having operated on the antrum, and feeling that it might be partially responsible for the temperature which she now had, I did a radical Luc-Caldwell operation on that cavity on December 9, ten days after the Killian. It was filled with small, pedunculated polypi. They were thrown out in such a manner by a large spoon curette as to give one the impression that by turning the head over he could have shaken them out. The day following the nurse reported that the patient was still menstruating, rather more profusely. The patient told me that she had suspected that she was pregnant, whereupon I asked my friend, Dr. W. T. Gibb, to see and examine the case. As he made the examination, a two to three months' foetus was expelled into the vagina. The membranes were retained. Temperature after miscarriage shot up to 103, but came down almost immediately, and the following day, at noon, was normal. In the afternoon she complained of feeling chilly. Temperature on the following morning, December 11, was 101 F., and patient complained of pain in regions of coccyx and left breast. Induration and tenderness in both regions. Temperature that of general sepsis.

On December 12 Dr. Gibb cleaned out the uterus, and on December 14, under local anaesthesia, opened an abscess at coccyx, left breast, and, at my request, incised the swelling over right temple. Thick, hardly more than semi-fluid pus was found in all these locations. Dr. Gibb would not open swelling on fore-

head, as it looked like, and he believed it to be, a simple wen. At all times discharge from vagina was slight and without odor. Patient's condition remained same for several days. She complained of pain all through the right side. Temperature up and down irregularly. No chills. Occasional sweating. On December 17 a small, painful swelling appeared on left forearm, which I incised. I also incised small swelling at root of hair, which had never changed in locks. The same thick pus was found in both locations as had been found in other regions. Temperature curve remained that of a general sepsis, but the exacerbations became less and the remissions greater at longer intervals until, during the week beginning January 11, it at no time rose above 99.4-5.

Patient was under tonic and fresh air (sun-parlor) treatment. Abscesses were all healed. Patient sat up. Did some walking, but never had good use of right leg. She wanted to go home, but on January 17 she complained of severe headache. On the 18th eyelid was somewhat swollen and temperature went to 101 F. On the 20th some difficulty was experienced in the use of left arm. Was very quiet during the day and passed urine involuntarily at night. On the 21st there was complete left hemiplegia. On the 22d all food was refused and patient seemed dazed. Dr. Arthur Booth saw her in consultation and it was decided that an abscess had formed in the brain, for the relief of which operation was decided upon. When the right side of the head had been shaved there was disclosed, just back of the site of the small frontal abscess at the root of hair, a swelling of about the size of a half an egg, cut longitudinally. We decided, therefore, to enter the brain at this point. The bone was found necrotic for an area of about four to six square inches. The dura was inflamed and thickened. Pus was found by a puncture backward and downward, when a considerable amount was evacuated. A rubber tube drain was inserted into the abscess cavity and the wound dressed.

On the following days patient was at intervals conscious, but she had convulsions each day. No relief of hemiplegia. Temperature from subnormal to 103. Practically no nourishment, and she died on January 29, exactly two months after coming into the hospital. Autopsy was allowed upon the head only. It



showed a deficiency in calvarium at site of last operation of about four square inches. Also, in the posterior wall of the right frontal sinus, there was a deficiency about one-quarter inch in diameter as a result of necrosis. Dura was dull and thick and showed hemorrhagic infiltration. There were granulations and adhesions about deficiencies in calvarium. Lepto-meninges showed general hemorrhage infiltration and purulent infiltration in region of operative wound. Cerebro-spinal fluid turbid (purulent). Frontal sinuses, septum broken down and contained pus. Cribriform, normal. Ethmoid cells, filled with granulation; no pus. From operative wound a pus-discharging sinus extends into brain toward the ascending frontal convolution.

On examination of the brain it was found that the inner surface of the dura, over the entire right hemisphere, was covered with a thick, purulent exudate which was finally adherent to the pia arachnoid. Over the anterior pole of the right frontal lobe the adhesion was so intimate that part of the brain substance came away with it as it was stripped off. On the superior surface of the right frontal lobe, about midway between the anterior pole and the fissure of Rolando, and lying in the superior frontal fissure and involving the contiguous part of the superior and middle frontal convolutions, was the opening of the abscess cavity leading downward into the white matter. Horizontal section of the right hemisphere showed that the abscess cavity communicated directly with and burrowed along the superior frontal sulcus. About one inch internal to this were three oval hemorrhagic areas, evidently the sites of exploratory punctures.

Section at the level of the corpus callosum showed three small, irregularly shaped areas of necrosis, the most anterior of which was about one and one-quarter inch posterior to the cortex. It extended downward, becoming considerably larger and then, tapering off, extended to the level of the midplane of the corpus striatum. This area, grayish red in color, possessed no limiting membrane, but was differentiated from the surrounding tissue by an irregular line of necrosis. Anterior to this, at about the superior plane of the corpus callosum, there were three abscess cavities, the largest of which, about the size of an ordinary bean, was situated just beneath the cortex, was sharply walled off by a

limiting membrane and connected with the surface by means of one of the branches of the superior frontal fissure. The right lateral ventricle showed a hemorrhagic ependymitis, and the ependyma itself was covered with a thick, reddish gelatinous substance. The rest of the brain showed intense congestion, but no further abscess cavities.



Now, with the evidence all in, I think that when this woman was brought to the hospital the infective agent had entered the bone and an osteo-myolitis and pyaemia were already established, shown by the two abscesses on the head, at the time obscured by the general swelling, and the deficiency that appeared in the posterior wall of the frontal sinus, as well as the necrosis of the septum between the two frontals.

This case emphasizes again the causal relation of sinus and brain disease, and again we are reminded that, too frequently,

not only the gravity of sinus disease, but the disease itself is not recognized by the general practitioner. One can scarce think of a physician who to-day would fail to recognize an appendicitis or a diseased mastoid, and it is up to us, no matter how we may differ in opinion as to just how any certain case should be handled, to educate the general physician, yes, even the public, to recognize disease of the accessory sinuses of the head. And further, there should accompany every such recognition of the disease the same idea of danger that attaches to the recognition of a diseased mastoid or an appendicitis.

*Discussion on the Papers on Sinus Disease.*

Dr. W. E. Casselberry: With respect to the first paper, relative to cyst of the frontal sinus, I reported to this association some years ago three cases of what I designated then as "Serous Disease of the Maxillary Antrum" and in which I had difficulty in establishing a correct diagnosis. One must differentiate especially between (1) Serous Disease, *i. e.*, a free serous exudate confined in a bony cavity, its orifice being closed, and (2) the formation of a cyst with serous contents within a bony cavity, which may fill the cavity so compactly that the cyst wall appears like a lining membrane of the bone itself. In my report on cases of serous disease I detailed the means by which I logically deduced that diagnosis from the evidence and from the literature, but it was impossible to quite prove that they were not immense cysts filling the entire antrum. The same difficulty pertains to the question of serous disease, mucocoele or cysts of the frontal sinus. The literature is vague on this distinctive point, and we find the three names which do and should refer to different pathological conditions, used interchangeably by authors. It is, in fact, with immense difficulty that we can make the diagnosis between them. Perhaps this distinction is of greater importance in the antrum than in frontal sinus cases, as dental cysts more commonly encroach on the antrum than do similar cysts, perhaps originating in the nose, encroach on the frontal sinus, and yet I think the same point in diagnosis is pertinent to the frontal sinus. Singularly enough, although I saw and carefully studied these three cases several years ago, I have never seen one since in which the contents of the antrum or frontal sinus was of a serous or mucous nature, always since then encountering pus, so I have concluded that they must be comparatively rare.

Dr. H. P. Mosher: It is noteworthy that early in the history

of sinus disease mucocoele was one of the first conditions to be recognized. Since then cases of chronic suppuration seem to have been in the majority. To-day, however, mucocoele is coming to the front once more, and I feel that in the future it will be given a prominent place again.

Cases of sinus disease fall into two classes—cases of chronic retention and cases of chronic suppuration. I wish to say a word about the first class. It should be remembered that there are many grades of mucocoele from the case in which but few of the mucous glands are diseased and but little mucous is poured into the sinus, to the case in which all the mucous glands are diseased and the cavity of the sinus completely filled with their secretion. It is not usually recognized that a mucocoele may at any stage of its development turn into an hematocoele by the rupture of a vessel in the mucous membrane of the sinus. The larger the amount of blood which becomes mixed with the mucus the more easily will the mucocoele become infected. Many a case of chronic suppuration was originally a mucocoele, complicated with a partial or complete hematocoele.

In some cases the hematocoele replaces the mucocoele completely. When this happens the hematocoele gives all the signs of a mucocoele and the operating findings are the same except that fluid blood or blood which has become a jelly-like mass replaces the mucus. The blood like the mucus is usually sterile. I have had three such cases.

The case which Dr. Theisen has just reported seems to me to be a case of uninfected mucocoele.

Dr. H. L. Swain: There is a good deal of confusion in our terminology in conditions of this nature, and as the condition is purely pathological I do not see why it could not be simplified. As is the case in the ear, serous exudates take place within the middle ear and may never be anything but serous. We have the same true of mucus, which may be so thick and sticky that one has to pull it out with forceps. This is purely a secretion from the mucous glands. Then we have the cases where pus forms, where either serum or mucus or a mixture of the two has become infected and pus results. It is purely a matter of pathology—serous sinusitis, mucous sinusitis and purulent sinusitis may occur exactly as in the ear. The matter would then be perfectly clear, and I think we should have a clean terminology. I think in dealing with this subject of sinus disease the medical world has far from grasped the situation and they must go through the same education as we have had in mastoid conditions. We must make both physicians and patients afraid to leave such conditions without proper care.

Dr. C. P. Grayson: With reference to the two cases reported by Dr. Price Brown, I would only question the necessity of the introduction of tubes after the sinus operation in order to procure permanent drainage. It has seemed to me that this latter is most often secured by the adoption of Laurens's operative methods. These consist in the opening of the sinus and the removal of its anterior wall, and then, after thorough curettage of the cavity, to extensively enlarge the fronto-nasal duct by breaking down the ethmoidal cells adjacent to it. In packing the cavity one end of the strip of antiseptic gauze is first introduced into the nose through the enlarged duct and carried down to the anterior naris, and upon the completion of the packing the external wound is immediately closed by sutures. The gauze, allowed to remain for thirty-six or forty-eight hours, is then removed through the nose. Drainage is now amply provided for, and in addition, should it seem necessary, the sinus can be readily washed out. If excessive growth of granulation tissue be restrained, I have observed no such degree of contraction of the duct as would demand further operation.

Dr. J. H. Bryan: I can hardly agree with Dr. Grayson that there is not much tendency to closure of the opening between the frontal sinus and the nose; it has been my experience that there is a very great tendency to such closure. I have made very large openings with this burr, such as Dr. Brown has shown, and I believe you can get away most of the ethmoidal cells to the back, but the tendency is for the opening to close and then it is that you get your recurrence, and we may then have to resort to a secondary operation, either an Ogston-Luc or Killian, or remove the nasal process of the superior maxillary bone in order to gain access to the ethmoidal cells.

Dr. T. P. Berens: The success of any operation on the frontal sinus depends upon complete drainage, and this can be secured only by the complete removal of the anterior ethmoidal cells. In order to have complete removal of all of the anterior ethmoid cells we must always remove a large portion of the nasal process of the superior maxillary bone. We then get a perfectly clean wound that is drained by a perfectly clean, bony tube that is large enough to insert the little finger at least one-half inch in diameter. In the small number of these cases (eighteen or twenty), which I have had I have not seen this bony tube close before I wanted it to, *i. e.*, before the frontal sinus has healed. I do want the whole wound to close the same as I want the mastoid wound to close, and apparently it does this partly by adhesion of the periosteum to the bone, partly by blood-clot formation, and the rest fills in by granulation tissue so that finally we get complete occlusion of this sinus and of the very much enlarged naso-frontal duct.



Dr. J. Price-Brown: With regard to the point of introducing tubes: We have been taught that when an opening is made from the frontal sinus into the nose for drainage the tendency is for it to close again, and Dr. Coakley, in introducing his method of treatment, has said that in the majority of cases they have closed again. The point in the introduction of these tubes in the first case was that: there had been so many operations done upon this patient that I thought that the gold tube would answer the best purpose in keeping the passage open. Apparently it succeeded in doing so, and the reason I performed the second operation was because of the old wound which had never healed; and the fact that the patient would not submit to intra-nasal treatment through the gold tube. Hence I reopened, removed dead bone, and, after taking out the gold tube, inserted the rubber one in its place in order that I could properly drain and get the cavity thoroughly healed. This was accomplished in forty-eight days from the time of the first operation, each tube being in position for about half that time, when the rubber one was finally removed. In the second case I thought it was better to retain drainage for some time at least, and the tube was kept in position for two weeks. The excellent results in these cases seem to me to warrant this method of treatment. My object in presenting this paper was to show that a less severe operation than Killian's could be attended by equally as good results.

Dr. L. A. Coffin: I want to report the case of a large retention cyst in a colored girl who had, on account of it, a marked exophthalmos. On opening the anterior ethmoids by resection of the frontal process of the superior maxilla, I found a cavity as large as the inside of an English walnut. The contents were sponged out in one gelatinous mass. The orbital wall was necrotic and the cavity connected with the orbit. The picture of this case previous to operation and the plate are to be seen in the X-ray exhibit. In the Killian operation there is no need of drainage tubes. I have been obliged to do a secondary operation but once. In this case I found at the time of the secondary operation that I had passed by a very narrow supra-orbital recess.



## PAPILLITIS ATROPHICANS BILATERALIS LINGUAE.

BY HENRY LEWIS WAGNER, M.D.

The following is interesting as illustrating a locally well defined atrophy of the upper stratum of the tongue. The patient was a woman, fifty-four years of age, who had passed her meno-



Papillitis Atrophicans Bilateralis Linguae.

pause some years previous to her coming under observation. She was somewhat anemic and very nervous. She gave no history regarding lues or any other predisposing disease (malaria). Nothing was especially noticeable about the mouth other than

that she wore a well-fitting dental plate. The interest in the case centers in the primary affection which attacked principally the left side of the tongue and which gave rise to small, whitish vesicles—similar to herpes—occurring at regular intervals and accompanied by general malaise. This eruption would continue from one to two days and then subside. A few days subsequent to the subsidence a subacute inflammation would set in with only slight swelling.

The bilateral atrophy gradually followed. Local applications with general treatment seemed only to alleviate the condition, but a prolonged iodide treatment prevented a recurrence up to the present time—covering a period of nearly a year. The present atrophic appearance of the sides of the tongue is best shown in the accompanying illustration (p. 174).

Microscopical analysis of our pathologist, Dr. E. A. Victor, gives the following result: The epithelial lining is thin and dense. A deeper layer corresponding to the rete mucosum is almost discernible. The fibrous structure of the corium is loose and secondary papillae are almost obliterated. But few mucous glands are found.

It is worthy of note that in the case just given there was no lump or nodule in the substance of the tongue such as Butlin has nearly always observed in parenchymatous hemiglossitis. This vesicular eruption might have been looked upon as pointing to herpetic hemiglossitis, as described by Butlin and Gueterbock, were it not for the subsequent atrophy.

## HYPERPLASIA LATERALIS LINGUAE.

BY HENRY LEWIS WAGNER, M.D.

Of equal interest is the following case, showing a true hyperplasia of the superficial structures of the side of the tongue accompanied by similar pathological conditions of both lower tur-



Hyperplasia Bilateralis Linguae.

binated bodies in the nose. The patient, otherwise healthy, was about forty years of age—a cigarette smoker, whose teeth were in an unusually good condition. The mouth, however, was congenitally defective, the defect consisting of a somewhat narrowed lateral diameter of the upper jaw and the absence of the upper lateral incisors. The close proximity of the teeth to the tongue,

especially during sleep (the patient being a mouth breather) may have been the causative factor in bringing about the localized hyperplasia.

The patient sought advice, not for this hypertrophic condition, which, according to his statement, had existed for years, but for a lancinating pain caused by a very small ulcer in the center of the hyperplastic tissue. A thorough extirpation of all diseased tissue restored the tongue to a normal condition.

Microscopical analysis of Dr. E. A. Victor shows a true hyperplasia of all the structures of the mucous membranes; the epithelial covering is thickened and on the apices of papillae shows a tendency to the horny variety. The vasa of an ulcer dipping into the deeper structures is seen. The corium shows no alteration in its elastic and connective tissue fibres other than a marked overgrowth of the primary papillae. In some specimens a branching of the secondary papillae is shown. Many mucous glands are seen scattered through the connective tissue.

#### *Discussion.*

Dr. J. Payson Clark: I should like to ask whether or not Dr. Wagner considered it possible that his first case might be one of pemphigus of the tongue. I have seen a case of this rare affection recently, and some points in the history of this case brought it to my mind.

Dr. J. H. Bryan: I do not think one gets atrophy of the tongue with pemphigus.

Dr. J. W. Farlow: I have seen several cases of pemphigus of the mouth; but in that disease the eruption is bullous and not vesicular, as in the case just reported. I have not seen atrophy follow the bullae, so that I should not be inclined to consider this a case of pemphigus of the tongue.

Dr. H. L. Wagner: The examination at the time of the eruption would not really coincide with all the desiderata required for the diagnosis of pemphigus, and I simply left the matter open. Certainly I would be glad to have light thrown upon it.

*Candidate's Thesis.*VARIATIONS OF THE OSTIUM FRONTALE AND  
THEIR BEARING ON INTRA-NASAL OPERATIONS  
ON THE FRONTAL SINUS, WITH A NOTE  
IN REGARD TO THE TOPOGRAPHY  
OF THE CRIBRIFORM PLATE.

BY J. GORDON WILSON, M.B.

(Hull Laboratory of Anatomy, University of Chicago.)

The importance of the frontal sinus in diseases of the nose has attracted much attention to its anatomy. In this paper the primary purpose is an examination of the relation of the ostium frontale to the neighboring parts, especially its relation to the anterior fossa of the skull. This relationship has not attracted the attention it deserves. So far as I am aware, there have not been published reliable statistics which would enable one to arrive at an understanding in regard to the variability in position of the ostium. It is obviously important to ascertain in what proportion of cases this opening is so placed as to render it dangerous in an intra-nasal operation. One must know where the danger lies in order to avoid it.

In pursuing this investigation it was necessary to ascertain with which part of the nasal cavity this opening (the ostium frontale) communicates, whether with the infundibulum or with the middle meatus. These results have been tabulated. Incidentally there was collected a number of cases in which an intra-nasal opening would not be capable of draining the frontal sinus because of depressions in the anterior inferior part of the sinus.

The examination was made both of osseous preparations and of preparations met with in the anatomy room during the students' dissections. In neither case was a selection made; the skulls were taken indiscriminately out of the collection made for general use. In the dissecting room all the available parts were examined during one quarter, together with cross sections prepared for special study. To examine the parts accurately sections were made in more than one plane; in most the sections

were made in both the sagittal plane and in the nasion-inion plane or parallel to these planes, and where necessary the perpendicular plate of the ethmoid was removed. In the dissecting room preparations, in addition, the anterior part of the middle turbinate was taken away to expose the infundibulum.

In each case the diameter of the ostium was measured and its relation to the anterior fossa of the skull ascertained. Where the opening was small, 2.5 mm. or less, and where it lay adjacent to the cribriform plate or ethmoidal fossa, with only thin bone intervening, especially if it approximated the plate at an acute angle, there was recognized a source of danger and the skull was so marked. Where one or more ethmoidal cells intervened between the ostium and the middle fossa of the skull, or where the ostium, though of small diameter, lay more than 1.5 mm. below the floor of the fossa, the skull was marked as not dangerous. In each case of interest or of doubt an explanatory note has been appended to the table.

There appears to be such a lack of agreement in the text books with regard to the meaning of the terms applied to structures in this neighborhood that I shall define the terms which I propose to use:

(1) The infundibulum is the depression lying between the uncinat process and the bulla ethmoidalis.

(2) The hiatus semilunaris is the narrow slit-like entrance which leads to this depression.

(3) The ductus naso-frontalis is the narrow, closed in passage which leads from the upper end of the infundibulum, or from the middle meatus, to the sinus frontalis.

(4) The ostium frontale is the narrow communication of the sinus frontalis with either (a) the nose directly, or (b) with the upper end of the naso-frontal duct.

(5) The bulla frontalis is an inconstant osseous cavity closed above, which projects into the frontal sinus. It is of varying size and position, and usually thin walled. It represents, I believe, an upward dilatation of an anterior ethmoidal cell or possibly of some other depression in the anterior superior part of the middle meatus. Its presence dates from the cartilaginous stage of the



nasal cavity and the varying origin accounts for the varying position of its ostium.

(6) The fossa ethmoidalis is the cranial depression which lies immediately over the cribriform plate and which is bounded laterally by the orbital plate of the frontal bone.

As the investigation proceeded it was soon evident that the cribriform plate was the first and most important part likely to be injured (Figs. 1, 2). In addition, in only a few cases would the lateral wall of the fossa ethmoidalis run any chance of injury (Fig. 3). The importance of this dangerous area is the more impressed on one when it is remembered that the ethmoidal fossa varies considerably in its depth and breadth, especially at its anterior extremity (compare Figs. 2 and 3). Further, the dura mater is here fastened firmly to the bone, and there is the probability that in this area the intra-cranial lymphatics communicate with the nasal lymphatics.

In the normal frontal sinus the ostium may be said to be at the down-pointed apex of a pyramidal-shaped cavity which opens into the anterior superior part of the middle meatus. The normal ostium frontale lies at some distance below the cribriform plate; thus, some of the narrowest ostia are so far below the plate as to be no source of danger, *e. g.*, osseous preparations V. and VIII. The depth of the ostium beneath the cribriform plate depends, in the first place, on the mode of communication of the sinus with the nasal cavity, whether directly with the middle meatus or indirectly through the infundibulum; and in the second place on the presence or absence of a distinct naso-frontal duct. Thus, in osseous preparation VI. it was 12 mm. below the level of the plate and frequently it was 8 mm. From this level in an inferior superior direction the ostium was found to vary considerably, in one case reaching 11 mm. above the cribriform plate (Fig. 3). As the level of the ostium approximates to or passes above the cribriform plate the length of the naso-frontal duct increases correspondingly. There was, therefore, in my specimens a variation of 23 mm.

The transverse variation is equally striking. The ostium may be regarded normally as surrounded by ethmoidal cells. It may, however, be displaced medially to the cribriform plate or laterally

toward the orbital plate; anteriorly toward the nasion or posteriorly for a considerable distance along the cribriform plate. (Skull XIX.) This deviation varies with the size and position of the neighboring ethmoidal cells, and is especially marked if a bulla frontalis be present; thus in some (*e. g.*, Fig. 1) the ostium is thrust back to the fossa ethmoidalis. In others it is pushed over to the orbital border.

The variation in size of the diameter of the ostium, especially near the level of the cribriform plate, is usually associated with an upward projection of anterior ethmoidal cells into the floor of the sinus. An exception to this was found in skull XIX. (see note), and in Fig. 3.

The examination of these osseous and moist preparations clearly showed that the great source of danger in opening the frontal sinus by an intra-nasal route, especially where the attempt is made to dilate the normal channel of communication between the sinus and the nose, is the frequent approximation of the ostium or even of the naso-frontal duct to the cribriform plate. In a few cases the prolongation of the naso-frontal duct up the side of the ethmoidal fossa adds the danger of injury to the orbital wall of the anterior fossa of the skull (Fig. 1). The differences in the percentages of danger between the osseous and the moist preparations is sufficiently accounted for by the narrowing of the ostia by mucous membrane, *e. g.*, osseous preparations XIX. The figures are sufficiently close to allow one to conclude that the average of danger lies between 20 and 24 per cent.

It is obvious that this percentage is much higher than that met in routine practice. The anatomical explanation of the discrepancy is that as a rule the character of the bone surrounding the ostium in the dangerous area varies; that towards the cranial cavity, though thin, is firm and compact, that towards the ethmoidal area is thin, parchment-like in consistence, and yields easily to pressure. This was markedly so on both sides of osseous preparation XIV. In this skull, were equal pressure exerted on each side of the duct, the part first to yield would be the wall adjacent to the ethmoidal cells. Recognition of this fact is of the utmost importance in dilation of the naso-frontal duct. In addition, were the anatomical position of the ostium and its relation to the

cribriform plate (see below) noted previous to operation, to this difference of structure of the bone would be added the adoption of a path directed away from the source of danger in the anterior cranial fossa—a result which would facilitate operation and ensure greater safety.

While this easy yielding condition of bone towards the region of safety exists in the great majority of cases it cannot be regarded as universally true. In contrast to this is Fig. 3. In this preparation the slit-shaped duct has a diameter of 1.5x2mm. at a height of 8mm. above the cribriform plate; and of 2.5x3 at 11 mm. above the plate. Above this point it dilates to form the sinus cavity. Here the duct is separated from the ethmoidal fossa by a very thin plate of bone (scarcely  $\frac{1}{4}$  mm. thick) and from the nasion by a very dense bone. In this case an examination previous to operation would have enabled one to ascertain that an anomalous condition existed from the presence of a naso-frontal duct extending far above the level of the cribriform plate.

The ostium of the frontal sinus may communicate with the nose:

1. By a naso-frontal duct of varying length opening into the infundibulum. The duct may appear to be a direct continuation of the infundibulum or may join the infundibulum at an angle. In the latter case the infundibulum may communicate with an ethmoidal cell, or may end blindly.

2. The ostium or its naso-frontal duct may communicate with the anterior superior part of the middle meatus independent of the infundibulum. The infundibulum may at its upper end communicate with an ethmoidal cell, or may widen into a shallow groove opening in the anterior superior part of the middle meatus.

3. In rare cases communication of the ostium or its duct may be with the anterior part of the nasal cavity or with the superior meatus. Such cases, especially the latter, must be regarded as pathological.

It will be noted that while in the osseous preparations double openings were present in 3 out of 36 (20 skulls), none was met with in the series of moist preparations. In my experience double patent openings in moist preparations are relatively rare as com-

pared with double openings in osseous preparations. This it appears to me should be ascribed not to a breaking down of the bone during the preparation of the skull (though such might easily occur and may easily be distinguished), but to the presence of a membrane covering the second opening, or—as it would be better to term it—defect in the bone.

Accepting the definition of infundibulum given above, it is as a rule not difficult to decide whether the sinus opens into the infundibulum or into the middle meatus. In one or two cases there was some difficulty, such, for instance, as IX, where the infundibulum is represented by a shallow canal, and the ostium is a narrow slit, one end of which lies directly over the upper end of the canal. As here, however, the greater part of the ostium communicates with the middle meatus and only a small part with the infundibulum, it can scarcely be said to open into the infundibulum.

Our preparations show that the number of sinuses in which the ostium frontale forms a direct continuation of the infundibulum is relatively small. Including sinuses which enter the infundibulum at an angle with those which form a continuation of it we find that in 20 skulls with 36 sinuses there are only 19 or 52.8 per cent which fall within this class. In the other 17 or 47.2 per cent, while the sinus may open in the neighborhood of the infundibulum it cannot be regarded as opening into it, but must be regarded as opening into the middle meatus. In the moist preparations the figures are equally striking. In 20 heads with 39 sinuses the ostium opened into the infundibulum in 19 or 48.7 per cent, in 20 or 51.3 per cent into the middle meatus. In some of these cases the infundibulum ends in an ethmoidal cell or blindly. In many one finds that the canal of the infundibulum at its superior extremity becomes extremely shallow and widens into a bay-like indentation of the ethmoidal bone where the frontal sinus as well as the anterior ethmoidal cells communicate with the nasal cavity.

The more carefully one examines a series of preparations the more one becomes convinced that it would be better were our anatomical text books to describe the communication of the frontal sinus as being with the anterior superior part of the middle

meatus rather than with the infundibulum—a description which would appear to agree with known embryological facts.

It is interesting in regard to this to note the opinion of A. Hartman:\* “Als Grundtypus glaube ich das Verhalten betrachten zu müssen, dass überhaupt kein Nasofrontalcanal vorhanden ist, sondern die Stirnhöhle sich bis zum vorderen Ende der Mittleren Muschel erstreckt und frei durch eine breite Spalte in den äussern Theil des mittleren Nasenganges mündet.”

In the sinuses which are marked “incapable of drainage,” there are only grouped such as have well marked pockets projecting into the naso-frontal spine and whose depth lies considerably below the level of the ostium frontale or shut off from it by a ridge of bone. The osseous preparations were most carefully examined with a view to incapability of drainage. I found that in the 36 sinuses there were five which might very easily be placed within this list. There were others in which the depth of the depression was not great, 3 or 4 mm., or with a cavity so formed that slight inclination of the head would drain it.

Sinuses incapable of drainage after intra-nasal dilatation of naso-frontal duct:

- VIII. Right. 6. On both sides ridges of bone shut off depression from ostium frontale.
- Left. 5.
- XI. Left. 9. Depression is separated by ridge of bone from ostium frontale and also extends to an inferior level.
- XV. Right. 7. Depression lies 6mm. below level of ostium.
- XX. Right. 10. Depression is separated by ridge of bone from ostium and also extends to a lower level. (Fig. 4).

Since the part of the anterior cranial fossa most likely to be injured is the cribiform plate, it would be of the greatest importance to indicate extracranially the plane in which it lies. By such means it would be possible in an X-ray picture to form correct conception of the relation of the ostium frontale to it. In the literature I can find no reference to such landmark; it does not seem to have attracted the attention of, among others, such painstaking observers as Jacobson, Cunningham, Zuckerkandl or Logan Turner.

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\*From Zuckerkandl's *Anatomie der Nasenhöhle* 1893, Bd. i, S. 96.

When we remember the wide range of variation to which the bones of the skull are subject, and especially the varying range of the speno-ethmoid angle of which the cribriform plate forms part of one of the sides, we could not expect to find any extracranial line which would indicate absolutely the plane of the cribriform plate. But to find a line which would be sufficiently close to form a guide would be of no small importance. It appears to me that such a line exists.

In examining a series of skulls cut in the sagittal plane I found that the cribriform plate had a close relation to the nasion-inion line and that the anterior part of the plate lies very close and parallel to this line. A closer approximation to the plate is got by taking the line which passes from the nasion to the depression which exists immediately below the inion. This I take as my line of measurement and refer to it as the N-ID line. In some skulls the anterior part of the cribriform plate lay in this line, in most it lay, 1 to 2mm. below. I have tabulated the results in a series of 20 preparations—14 skulls (in Table 1-14) and 6 dissecting room preparations (in Table 15-20). In the dissecting room preparations the line of attachment of the dura mater to the bone was measured. One advantage of this N-ID line is the facility and accuracy with which it can be marked out on the head. The transverse plane in which it lies is for practical purposes sufficiently ascertained by a line drawn through the inion depression and the nasion having equal relations to the rim of each orbit. Though the examination was chiefly made on adult skulls, it was ascertained that the relation holds for all ages above 12; below that age I have as yet had no opportunity to test it.

The variation in position of the inion or of the length of the nasal bone does not alter the relation to such an extent as to prevent the line being of use in an approximation of the surface marking of the anterior half which is the most important for our purpose. From the sulcus chiasmatis (optic groove) the speno-ethmoid bones in the middle line form a shallow curve, concave upwards. The deepest part lies about midway between the anterior edge of the sulcus and the nasion about the middle of the cribriform plate. In the table where necessary this point is



referred to as the mid-point. Where no such figure is given, no extra depression is present.

| Skull. | Relation of<br>Cribriiform Plate<br>to N-ID Line. | Remarks.  |
|--------|---|---|
| 1.     | 2 mm. below                                       | Greatest depth below N-ID. at mid point is 3 mm.  |
| 2.     | On level with                                     |   |
| 3.     | 2 mm. below                                       |   |
| 4.     | 1.5 mm. below                                     | Greatest depth at mid point is 3 mm.              |
| 5.     | On level with                                     |   |
| 6.     | On level with                                     | At mid point 1 mm.                                |
| 7.     | 1 mm. below                                       |   |
| 8.     | 1.5 mm. below                                     |   |
| 9.     | On level with                                     |   |
| 10.    | 2 mm. below                                       | Greatest depth 4 mm. lies posterior to mid point. |
| 11.    | 1.5 mm. below                                     |   |
| 12.    | 2 mm. below                                       |   |
| 13.    | 1.5 mm. below                                     |   |
| 14.    | On level with                                     | Skull of child of 12-14                           |
| 15.    | On level with                                     | Anterior part slopes very gradually to plate.     |
| 16.    | On level with                                     |   |
| 17.    | On level with                                     | 2 mm. below at mid point                          |
| 18.    | 1.5 mm. above                                     | On level at mid point.                            |
| 19.    | 1.5 mm. above                                     | On level at mid point                             |
| 20.    | On level with                                     | 1 mm. above at mid point.                         |

*Conclusions.*—The great source of danger in intra-nasal operation directed to opening the frontal sinus is injury to the cribriform plate and fossa ethmoidalis.

In a large proportion of cases the intra-nasal operation is safe from the size of the ostium or its distance from fossa ethmoidalis. In a still larger proportion the operation is safe if one remembers that the walls of the ethmoidal cells are thin and that pressure exerted in this direction will yield easy and rapid dilatation.

The nearer the naso-frontal duct is to the orbital border the safer the operation.

Operations on sinuses in which the ostium is large are devoid of danger.

Operations on sinuses in which the constriction is found to lie below the N-ID plane, are safe.

Those cases are especially dangerous in which the ostium lies in the plane of or above the cribriform plate and the more dangerous the nearer the sagittal plane.

The plane of the cribriform plate lies in the transverse plane passing through theinion depression and the nasion or sufficiently close to it to make the N-ID plane useful for all practical purposes.

In a small proportion of cases the operation cannot give satisfactory drainage.

The length and diameter of the naso-frontal duct usually varies with the development of the ethmoidal cells. If the ethmoidal cells be well developed, and especially if a large bulla frontalis is in close apposition to it, the duct may be 10-12mm. long and small in diameter throughout (2mm.). The angle which the duct forms with the cribriform plate also varies considerably, in some being as acute as 45 degrees. An exception to this is seen in Fig. 3, where the long, narrow duct is surrounded by compact bone, which is especially thick anteriorly.

## OSSEOUS PREPARATIONS.

|   |             |
|---|-------------|
| Number of skulls examined.....  | 20          |
| Number of frontal sinuses present.....  | 36          |
| Number in which <i>no</i> frontal sinus present (IV and X).....                     | 4           |
| Number in which there would be danger from intra-nasal operation .....              | 7 or 19.5%  |
| Number in which there would be no danger from intra-nasal operation .....           | 29 or 80.5% |
| Number of frontal sinuses opening directly into infundibulum .....                  | 19 or 52.8% |
| Number of frontal sinuses opening into anterior superior part of middle meatus..... | 17 or 47.2% |
| Number of sinuses with double openings.....   | 3           |
| VI—Left (a) Superior meatus.  |             |
| (b) Middle meatus.  |             |
| IX—Right (a) Middle meatus.   |             |
| (b) Ethmoidal cell.   |             |
| XII—Left (a) Middle meatus.   |             |
| (b) Middle meatus.  |             |

## OSSEOUS PREPARATIONS.

| <i>No. of skull.</i> | <i>Diameter of Ostium in mm.</i>  | <i>Relation to Cribri-form Plate.</i>                                | <i>Nasal Opening.</i>                             | <i>Danger.</i> | <i>Remarks.</i>   |
|----------------------|-----------------------------------|--|---|----------------|---|
| I.                   | R. 4x8<br>L. 2x5                  | Adjacent<br>Adjacent   | Middle meatus<br>Infundibulum                     | o<br>x         | Size of ostium excludes danger.<br>Narrow diameter is separated from ethmoidal fossa and cribriform plate by thin bone. Naso-frontal duct to infundibulum.  |
| II.                  | R. 4x6<br>L. 3x5                  | Below and anterior<br>Below and anterior                             | Middle meatus<br>Middle meatus                    | o<br>o         | Nasal opening would not drain because of deep pocket in floor of sinus.<br>Sinus lies in orbital wall; its position would make it difficult to locate in external operation.                                      |
| III.                 | R. 2x3<br>L. 10x3                 | Surrounded by ethmoidal cells<br>Adjacent to lateral border of plate | Infundibulum<br>Middle meatus                     | o<br>o         | Safe because of size.   |
| IV.                  | R.—None<br>L.—None                | — — — — —  | — — — — —   | — —            | No sinus.<br>No sinus; very slight depression in orbital plate of frontal bone.   |
| V.                   | R. 1x2<br>L. 3x3                  | 5 mm. below<br>5 mm. below   | Infundibulum<br>Infundibulum                      | o<br>o         | Separated by ethmoidal cells.<br>Situated at orbital border.  |
| VI.                  | R. 4x3<br>L. 2 mm.<br>1.5x1.5 mm. | 12 mm. below<br>Adjacent<br>Slightly below and toward orbital border | Middle meatus<br>Superior meatus<br>Middle meatus | o<br>x<br>o    | Pyramidal shaped apex opens low down into middle meatus.<br>Double opening.   |
| VII.                 | R. 2x2<br>L. 2x2.5                | Separated by ethmoidal cell<br>Adjacent and slightly above           | Infundibulum<br>Infundibulum                      | o<br>x         | A broad depression into orbital plate; bone toward cranial fossa comparatively thick and at level of ostium has a small ethmoidal cell intervening.<br>Makes angle of 45 degrees with cribriform plate. (Fig. 2.) |

OSSEOUS PREPARATIONS—(Continued).

| No. of skull. | Diameter of <i>Ostium</i> in mm. | Relation to Cribri-<br>form Plate.  | Nasal Opening.                                   | Danger.              | Remarks.  |
|---------------|----------------------------------|---|--|----------------------|---|
| VIII.         | R. 1.5x2<br>L. 2x3               | 8 mm. below<br>6 mm. below  | Infundibulum<br>Infundibulum                     | o<br>o               | Would not drain.<br>Would not drain.  |
| IX.           | R. 4x10<br>3x3<br>L. 1.5x1.5     | Below<br>Below<br>Near orbital border<br>separated by eth-<br>moidal cells  | Middle meatus<br>Ethmoidal cell<br>Middle meatus | o<br>o<br>o }<br>o } | Double opening.<br>Difficult to locate from external surface be-<br>cause passes into orbital medial wall. In-<br>fundibulum simply a groove.                   |
| X.            | R.—None<br>L.—None               | —<br>—  | —<br>—   | —<br>—               | No sinus.<br>No sinus.  |
| XI.           | R. 4x4<br>L. 4x4                 | Below; close to orb-<br>ital wall<br>Below; close to orb-<br>ital wall  | Middle meatus<br>Infundibulum                    | o<br>o               | Crista galli hollowed out into a mere shell<br>and forms part of sinus. Nasal opening<br>would not drain.   |
| XII.          | R. 4x4<br>L. 1.5x1.5<br>2x2      | Below; separated by<br>ethmoidal cells<br>Below; separated by<br>ethmoidal cells  | Middle meatus<br>Middle meatus<br>Middle meatus  | o<br>o }<br>o }      | Double opening.   |
| XIII.         | R. 1.5x3<br>L. 2x3               | Below<br>Below  | Middle meatus<br>Infundibulum                    | o<br>o               | On each side naso-frontal duct is pushed<br>toward ethmoidal fossa by thin-walled eth-<br>moidal cell and is separated from cribri-<br>form plate by thin bone. |
| XIV.          | R. 2.5x3<br>L. 2.5x3             | 1 mm. below<br>Adjacent   | Infundibulum<br>Infundibulum                     | x<br>x               | Fronto-nasal spine hollowed out and ostial<br>opening would not drain cavity; ostium<br>partly separated by ethmoidal cell.                                     |
| XV.           | R. 3x5<br>L. 2x2                 | Adjacent to anterior<br>part of ethmoidal<br>fossa, on level with<br>cribriform plate<br>Adjacent to eth-<br>moidal fossa | Middle meatus<br>Middle meatus                   | o<br>x               | Infundibulum ends in ethmoidal cell; well-<br>marked bulla frontalis. (Fig. 1.)   |

## OSSEOUS PREPARATIONS--(Continued).

| No. of skull. | Diameter of Ostrum in mm. | Relation to Cribri-form Plate. | Nasal Opening. | Danger. | Remarks.  |
|---------------|---------------------------|--------------------------------|----------------|---------|---|
| XVI.          | R. 4x4                    | 4 mm. below                    | Middle meatus  | 0       | No naso-frontal duct; ostrum opens directly into infundibulum.  |
|               | L. 2x2                    | 8 mm. below                    | Infundibulum   | 0       |   |
| XVII.         | R. 4x2                    | Below                          | Infundibulum   | 0       | Separated by ethmoidal cell. Separated by ethmoidal cell.   |
|               | L. 3x5                    | Below                          | Middle meatus  | 0       |   |
| XVIII.        | R. 1.5x3                  | Adjacent                       | Infundibulum   | x       | Ostrum surrounded by ethmoidal cells.   |
|               | L. 3x3                    | On same level                  | Infundibulum   | 0       |   |
| XIX.          | R. 3x2                    | Adjacent                       | Infundibulum   | 0       | On each side sinus is a large scooped-out cavity extending over whole orbital surface of the frontal bone and separated from ethmoidal fossa by very thin bone.                                     |
|               | L. 2x2.5                  | Adjacent                       | Infundibulum   | x       |   |
| XX.           | R. 3.5x2.5                | Below                          | Middle meatus  | 0       | On the R side the ostrum had a diameter of only 1.5 mm., but on stripping off the mucous membrane the ostcal opening was found to be 3x2. On both sides the ostrum was 2 c. m. posterior to nasion. |
|               | L. 2.5x3                  | Adjacent and slightly below    | Infundibulum   | 0       |   |

Would not drain; infundibulum opens into a wide space at anterior superior part of middle meatus, into which also open the anterior ethmoidal cells.

Would drain with difficulty because of ridge.

(Dissecting Room Preparations, see p. 194.)

# DISSECTING ROOM (MOIST) PREPARATIONS.

| <i>Diameter of Ostium Relation to Cribri-<br/>frontale in mm. form Plate.</i> |                | <i>Nasal Opening.</i> | <i>Danger.</i> | <i>Remarks.</i>  |
|---|----------------|-----------------------|----------------|--|
| 1.  | R. 1x1         | Infundibulum          | o              | No danger because of large size of ostium frontale, which is displaced up and back by bulla frontalis projecting above nasion into the floor of sinus frontalis. The diameter of ostium of bulla frontalis at level of and midway between plate and nasion is 1.5 mm. Infundibulum enters bulla frontalis. |
|   | L. 3x3         | Middle meatus         | o              |  |
| 2.  | R. 1.5x2       | Infundibulum          | o              | The adjacent internal plate of frontal bone was comparatively thick. Infundibulum leads into ethmoidal cell.   |
|   | L. 2x3         | Middle meatus         | o              |  |
| 3.  | R. 1.5x5       | Infundibulum          | o              | Infundibulum is a shallow channel. Naso-frontal duct opens into a wide bay at anterior superior part of middle meatus. Narrow diameter adjacent to plate.  |
|   | L. 1.5x5       | Middle meatus         | x              |  |
| 4.  | R. 1.5x3.5     | Middle meatus         | x              | Infundibulum ends blindly.<br>No sinus frontalis.  |
|   | L. not present | —                     | —              |  |
| 5.  | R. 3.5x3.5     | Middle meatus         | o              | No danger because large ostium frontale. Infundibulum passes into ethmoidal cell. Narrow diameter parallel to plate. Infundibulum passes into ethmoidal cell.  |
|   | L. 2.5x4       | Middle meatus         | x              |  |



## DISSECTING ROOM (MOIST) PREPARATIONS—(Continued.)

| <i>Diameter of Ostium Relation to Cribri-<br/>frontale in mm. form Plate.</i> |            | <i>Nasal Opening.</i> | <i>Danger.</i> | <i>Remarks.</i>  | <i>No.</i> |
|---|------------|-----------------------|----------------|--|------------|
| 6.  | R. 2x3     | Below                 | 0              | Infundibulum passes into ethmoidal cell.<br>naso-frontal duct.   |            |
|   | L. 2x3     | Below                 | 0              |  |            |
| 7.  | R. 1x2     | Adjacent              | x              | Long naso-frontal duct.<br>Long naso-frontal duct. (Fig. 2.)   |            |
|   | L. 2x3     | Adjacent              | x              |  |            |
| 8.  | R. 2x2     | Immediately below     | 0              |  |            |
|   | L. 3x4     | Immediately below     | 0              |  |            |
| 9.  | R. 8x4     | 10 mm. below          | 0              | On each side the large sinus frontalis appears<br>to be a direct continuation of the anterior<br>superior part of middle meatus. |            |
|   | L. 8x2.5   | 9 mm. below           | 0              |  |            |
| 10.   | R. 6x3     | 6 mm. below           | 0              |  |            |
|   | L. 6x3     | 8 mm. below           | 0              |  |            |
| 11.   | R. 2.5x2.5 | Adjacent              | x              | A long, narrow duct at no point of greater<br>diameter than 2.5 mm.<br>No danger because of large ostium frontale.               |            |
|   | L. 3x3     | Adjacent              | 0              |  |            |
| 12.   | R. 2x2     | 8 mm. below           | 0              | Sinus frontalis exists as slight depression in<br>orbital wall of frontal bone.<br>Small sinus in orbital wall.                  |            |
|   | L. 3.5x3.5 | 7.5 mm. below         | 0              |  |            |
| 13.   | R. 3x3     | 8 mm. below           | 0              | Ostium frontale lies at pyramidal-shaped apex<br>of sinus frontalis and immediately over up-<br>per end of a deep infundibulum.  |            |
|   | L. 5x7     | 6.5 mm. below         | 0              |  |            |

DISSECTING ROOM (MOIST) PREPARATIONS—(Continued.)

| <i>Diameter of Ostium Frontale in mm.</i> |            | <i>Relation to Cribriform Plate.</i> | <i>Nasal Opening.</i> | <i>Danger.</i> | <i>Remarks.</i>  |
|---|------------|--------------------------------------|-----------------------|----------------|--|
| 14.                                       | R. 4x1     | Adjacent                             | Middle meatus         | x              | Infundibulum ends blindly; narrow diameter of ostium adjacent to plate.  |
|   | L. 2.5x2   | 3 mm. below                          | Middle meatus         | o              | Sinus extends 7 mm. to right of middle line. Infundibulum passes into ethmoidal cell.  |
| 15.                                       | R. 3x3     | 4 mm. below                          | Infundibulum          | o              | Separated by ethmoidal cell.   |
|   | L. 2.5x2.5 | 4 mm. below                          | Infundibulum          | o              | Cone-shaped apex of sinus lies over infundibulum. No duct.   |
| 16.                                       | R. 1.5x4   | Slightly below                       | Middle meatus         | o              | Separated by ethmoidal cell. Infundibulum is a narrow groove.  |
|   | L. 5x2.5   | Below                                | Middle meatus         | o              | Pyramidal-shaped apex of sinus opens directly into middle meatus.  |
| 17.                                       | R. 2x4     | 2 mm. above                          | Middle meatus         | o              | Naso-frontal duct separated from ethmoidal fossa by ethmoidal cell. Infundibulum passes into ethmoidal cell.                       |
|   | L. 1x3     | On level with plate                  | Infundibulum          | o              | Naso-frontal duct separated from cribriform plate by ethmoidal cell. Narrowest diameter of ostium is parallel to cribriform plate. |
| 18.                                       | R. 2x2     | 7 mm. below                          | Infundibulum          | o              | Depression in naso-frontal spine 7 mm. deep.   |
|   | L. 1.5x2   | Adjacent                             | Middle meatus         | x              | Ostium separated from cribriform plate by ethmoidal cell.  |
| 19.                                       | R. 2x3     | Level with                           | Infundibulum          | o              | Ostium at anterior end of ethmoidal fossa.   |
|   | L. 2x2     | Adjacent                             | Middle meatus         | x              | Infundibulum ends blindly.   |
| 20.                                       | R. 4x3     | 6 mm. below                          | Infundibulum          | o              | Ostium frontale is a slit 3 mm. above cribriform plate, adjacent to anterior part of ethmoidal fossa.                              |
|   | L. 1x4     | 3 mm. above                          | Middle meatus         | x              |  |

## DISSECTING ROOM PREPARATIONS.

|   |             |
|---|-------------|
| Number of heads examined.....   | 20          |
| Number of frontal sinuses present (including Specimen 12) .....                     | 39          |
| Number in which no frontal sinus present.....                                       | 1           |
| Number in which there would be danger from intra-nasal operation .....              | 10 or 25.6% |
| Number in which there would be <i>no</i> danger from intra-nasal operation .....    | 29 or 74.4% |
| Number of frontal sinuses opening directly into infundibulum .....                  | 19 or 48.7% |
| Number of frontal sinuses opening into anterior superior part of middle meatus..... | 20 or 51.3% |

*Explanation of Plates.*

|                        |                           |
|------------------------|---------------------------|
| O. Ostium frontale     | F. S. Sinus frontalis     |
| C. P. Cribriform plate | I. Infundibulum           |
| C. G. Crista galli     | P. U. Processus uncinatus |
| N. Nasion              | E. Ethmoidal cell.        |

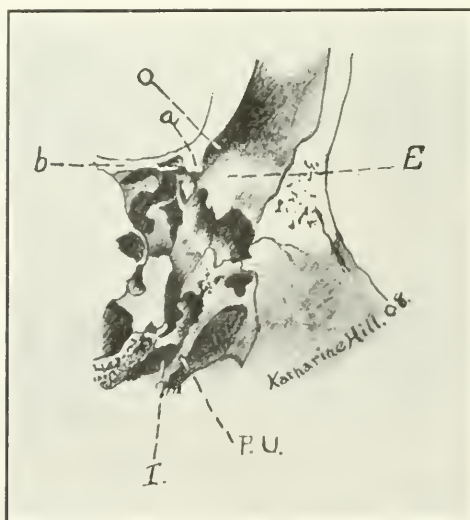


Fig. 1.

Fig. 1. Skull XV. A large ethmoidal cell (E) has raised the floor

of the left frontal sinus and pushed back its ostium (O) towards the ethmoidal fossa. The skull was cut 1.5 mm to left of middle line. The ridge of bone (b) represents the lateral border of ethmoidal fossa. The thin plate of bone (a) separates the narrow ostium (O) from the cribriform plate. (See Notes Skull XV.)

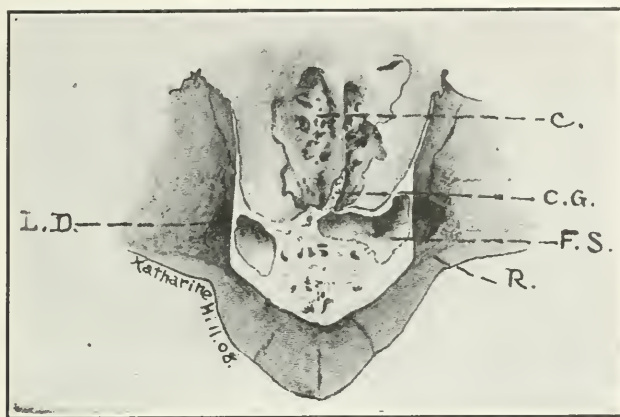


Fig. 2.

Fig. 2. Skull VII. Sectioned in plane which passes through nasion and the point midway between inion and lambda. On left side, naso frontal duct makes angle of  $45^{\circ}$  with cribriform plate and ostium lies slightly above the plate. (See Notes Skull VII.)

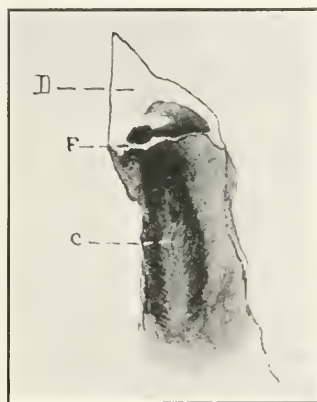


Fig. 3.

Fig. 3. (Not in tables.) Cut in plane which passes through nasion and slightly above midpoint betweeninion and lambda. Shows thick bone (d) anterior to duct; and thin bone towards anterior cranial fossa, about  $\frac{1}{4}$  mm thick. F lies 11 mm above anterior part of cribriform plate. The naso frontal duct at 8 mm above plate is  $1.5 \times 2$  mm, in diameter; at 11 above plate, where it opens into the right sinus, it is a slit  $2.5 \times 3$  mm.

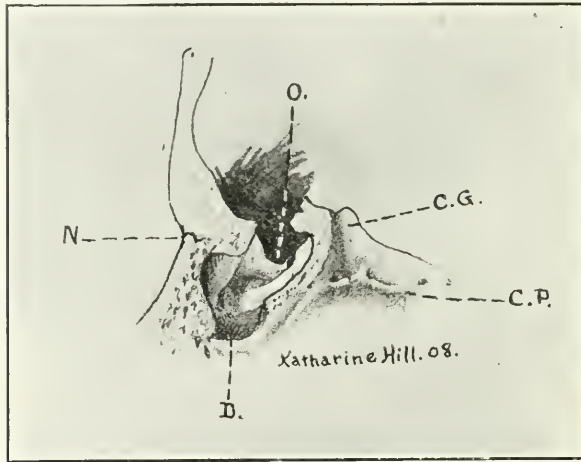


Fig. 4.

Fig. 4. Skull XX. Drainage impossible. Right frontal sinus has passed over to left of middle line and hollowed out nasal spine and part of perpendicular plate of ethmoid. D lies 10 mm below ridge of bone at O.

*Symposium.*

RECURRENT AND ABDUCTOR PARALYSES OF THE  
LARYNX.

*First Paper.*

Introductory Remarks on Anatomy and Physiology, Etiology of  
Paralyses of Central Origin.

RY JOSEPH W. GLEITSMANN, M.D.

The subject selected by your Council for general discussion is in my opinion the most interesting and at the same time the most difficult one in the whole domain of Laryngology. It is interesting, because it leads the Laryngologist at once beyond the sphere of his specialty, and it is difficult, as it deals with the most intricate questions of Neurology, about which in many instances the investigators entertain conflicting opinions.

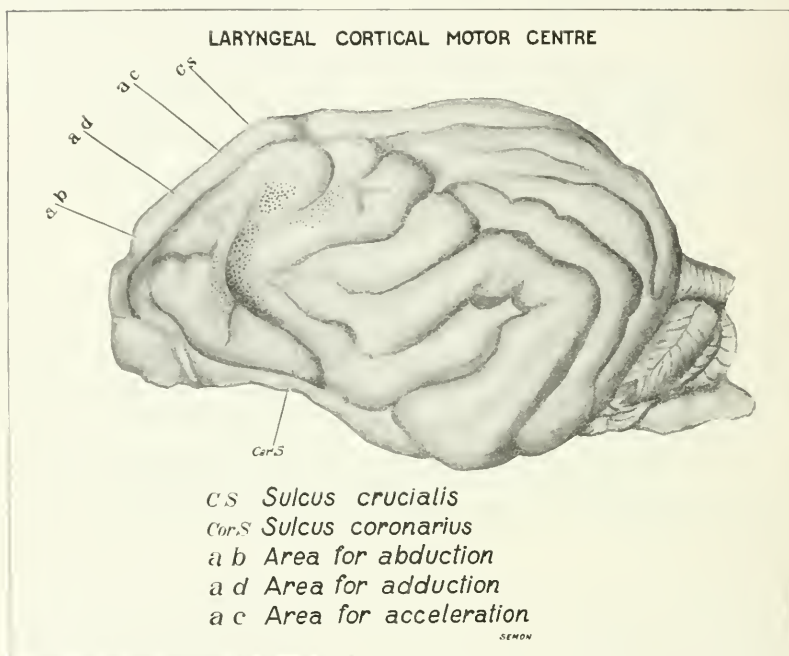
For a full understanding of the part assigned to me—the central causes of Recurrent and Abductor Paralyses—a clear conception of the motor centres in the cortex and bulbus, of their predominant functions, their connecting strata, is absolutely necessary. With the approval of your presiding officer I shall briefly analyze the anatomical features and add such physiological and pathological data, as have a direct bearing on our subject.

*I. Remarks on Anatomy*

The Larynx has to perform two functions, diametrically opposite to each other, viz.: phonation and respiration. The chief centre for phonation, adduction of the cords and a voluntary act, is in the cortex, for respiration, abduction of the cords and a vital process, in the bulbus. In addition there are subordinate centres in reverse order and of lesser dignity for phonation in the



bulbus, for respiration in the cortex. The connecting nerve fibres run from the cortical to the bulbar centre and vice versa through the internal capsule. The appended diagram from Rethi (1) shows very plainly the different motor centres and their relationship to each other.

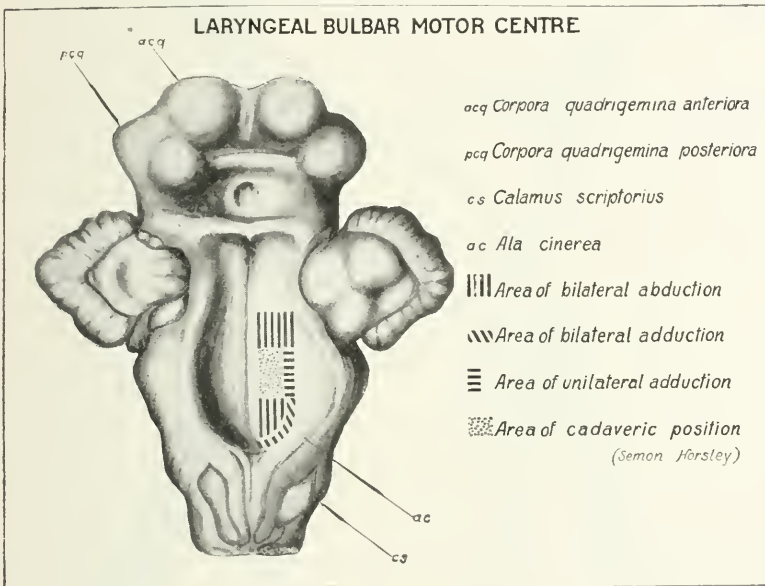


The cortical motor centre for phonation was discovered by H. Krause (2) to be located at the descending surface of the prefrontal convolution. The result of his investigations was confirmed by Semon and Horsley (3 and 4), who by extending their researches found additional motor areas in the cortex for acceleration and for deepening of the respiration. By ingenious experiments Risien Russel (5) succeeded in finding below the

1. Die Krankheiten des Kehlkopfes, 1901, p. 13.
2. Archiv fuer Anatomie und Physiologie, 1884, Volume Physiology, p. 203.
3. Phil. Transact. Royal Society of London, Vol. 181, 1890, p. 187-211.
4. International Medical Congress, 1890, Vol. IV., Part 12, p. 132.
5. British Medical Journal, August 24th, 1895, p. 481.

phonatory centre a locality from which abduction of the cords can be elicited. The illustration of the different cortical motor centres is a copy from Semon's treatise in Heymann's handbook of Laryngology and needs no further comment.

The bulbar representation of the Larynx is in the fourth ventricle, and we find in the *alea cinerea* two localities, irritation of which produces typical abduction of the cords, the respiratory



act, further smaller areas for bilateral adduction, one for unilateral adduction and one for the cadaveric position of the cords, all of which are graphically shown in the illustration, which is also taken from the same treatise by Semon.

The existence of the subordinate centres, mentioned above, has been proven by repeated experiments on animals, of which one only will be cited. Acephalic monsters, animals whose brain has been extirpated, are able to utter a cry, due to the subordinate phonatory centre in the bulbus, but their respiratory movements are not materially influenced, confirming the bulbus to be the main centre for respiration.

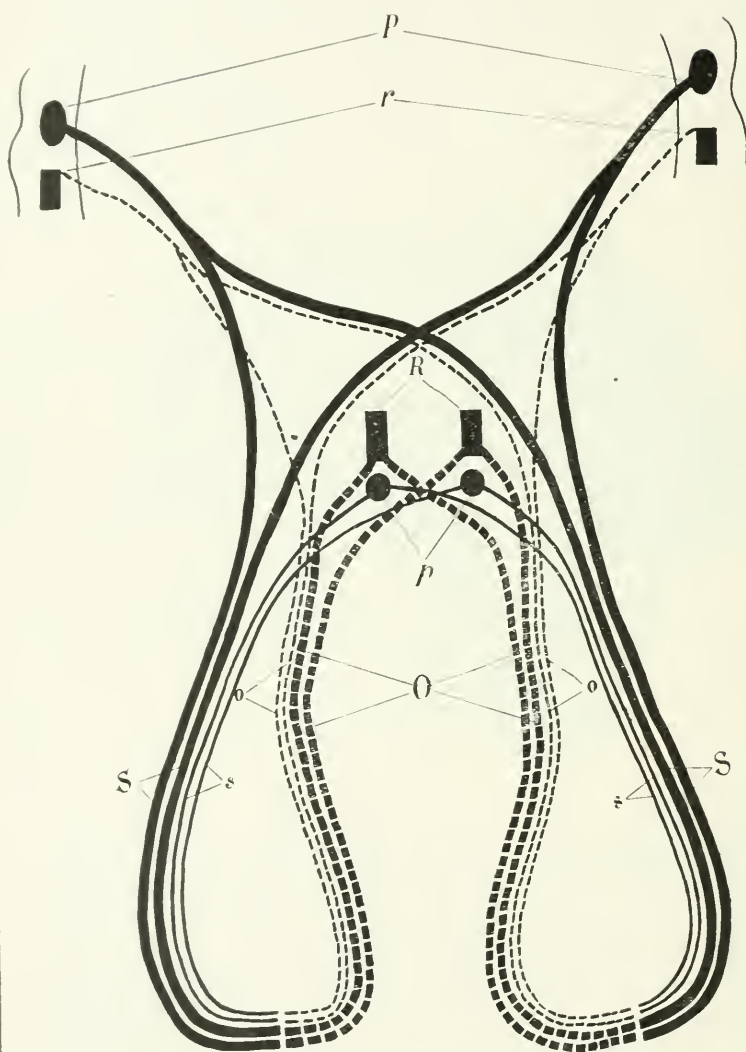


DIAGRAM OF  
**Phonation and Respiration in Cortical P-p and**  
**Bulbar p-R Centre with tracts in internal**  
**Capsule : full Lines for Adductors — S-s**  
**broken Lines for Abductors == O-o**

These elementary anatomical data, condensed within the narrowest possible compass, have almost universally been accepted; the first real discrepancy of opinions we meet is about the question of the root of the motor nerve of the larynx, which is not definitely decided at the present day.

After the careful experiments of Schech (6) the laryngeal motor nerve was for many years considered to have its source from the inner branch of the accessory Willisii, till Onodi (7) and Grabower (8 and 9) brought forth forcible arguments in favor of the pneumogastric, exceptions to which were taken by some authors. But a recognized authority, H. Oppenheim, says (1905) in his book (10) there is at present no certainty in regard to the participation of the accessory nerve in the innervation of muscles supplied by the pneumogastric.

The illustration from Edinger shows very clearly the intimate relationship of the nerve nuclei, the motor fibres being in black, the sensory fibres in red color.

Another much debated issue relates to the nature of the recurrent nerve. Whilst many investigators strongly believe it to be exclusively a motor nerve, others offer good reasons, based on animal experiments, to consider it a mixed nerve, carrying also centripetal fibres. But if the latter view should prove to be correct, we cannot yet draw any definite conclusions from it in regard to the human system, as even in animals a differentiation exists in the response in different species.

The last and a most important anatomical fact is the discovery by Risien Russel (11) of the existence of special nerve fibres for adduction and abduction, which run in separate bundles throughout the whole course of the recurrent, those for the abductors on the inner, those for the adductors on the outer side of the nerve trunk. They can be dissected to such an extent that one set of fibres responds to electrical irritation exclusively by adduction, the other exclusively by abduction of the vocal cords. By his work

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6. Experimentelle Untersuchungen ueber die Funktion der Nerven und Muskeln des Kehlkopfs, Wurzburg, 1873.

7. Archiv fuer Laryngologie XII., 1902, p. 70.

8. Ibidem, II., 1895, p. 144.

9. Ibidem, N., 1900, p. 320.

10. Lehrbuch der Nerven Krankheiten 4 Auflage 1st Band, p. 511.

11. Proceedings of the Royal Society, Vol. 51, 1892, March 31st, p. 102.

the formerly vexed question was solved, how a supposedly homogeneous nerve could effect such directly antagonistic movements.

## *II. Remarks on Physiology and Pathology.*

The cortical motor centres of the larynx differ from other cortical motor centres in the essential point that irritation of either one is always followed by symmetrical bilateral adduction of the cords, phonetic movements, which also take place when one cortical centre has been destroyed by disease or experimentally removed. This physiological observation had enjoyed universal acceptance till Masini (12) maintained to have found a locality in the cortex from which movements of the opposite side of the larynx could be produced. Subsequent experiments by others did not verify his statements, but recently Katzenbach (13) obtained by an improved method similar movements of the corresponding as well as opposite side.

The influence of the bulbar centre on respiration and on the position of the glottis has been carefully studied by Semon (14) in his endeavor to ascertain if the vocal cords play an active part during quiet respiration, and to determine the cause of the difference between the width of the glottis during life and its cadaveric position after death or after severance of the pneumogastric or recurrent. He found by numerous and carefully made measurements with graduated mirrors that in over 80 per cent. of adults the glottis remains stationary during quiet respiration and is considerably wider than in the cadaveric position. Its average width in men during life is 13.5 mm.; after death 5 mm.; in women 11.5 and 4 mm. respectively. Without entering into a detailed statement of his experiments and arguments, he is led to the conclusion that the greater width of the glottis during life is the result of a permanent activity of the abductor muscles, due to tonic, rhythmic impulses, received by their ganglia centres in the medulla from the neighboring respiratory centres through centripetal fibres of the pneumogastric. He calls this physiological action of the abductor muscles the abductor tonus.

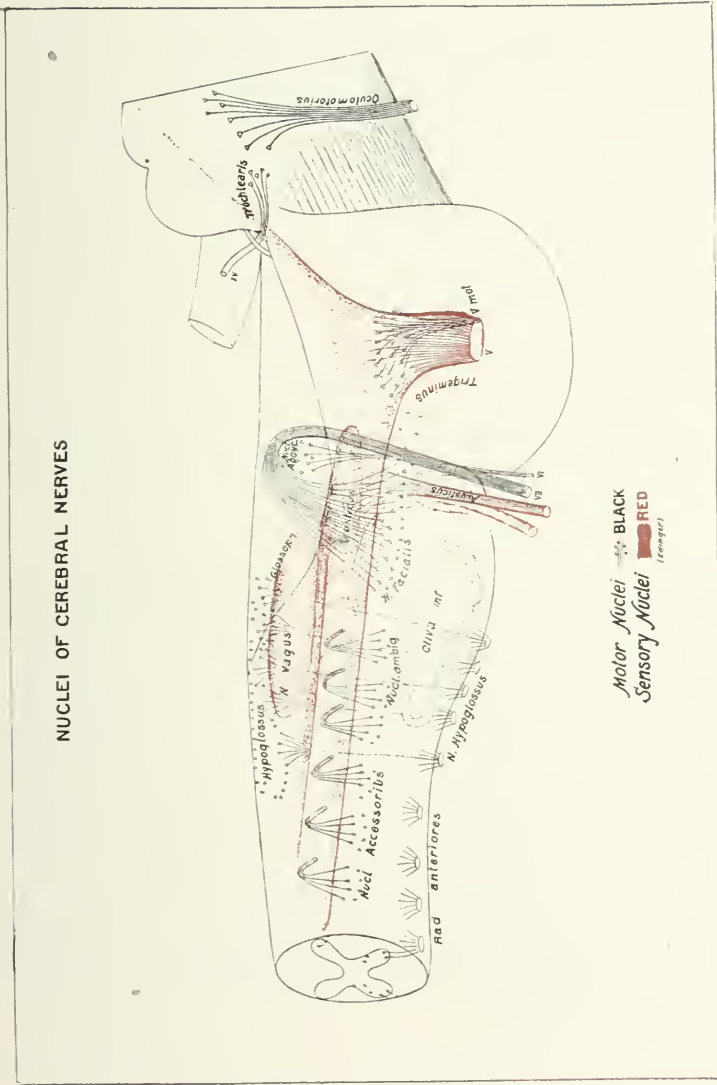
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12. Bolletino d. Mal. dell' Orecchio, 1893, July, p. 151.

13. Archiv fuer Anatomie und Physiologie, 1905, Heft 3 and 4, p. 396.

14. Proceedings of the Royal Society, Vol. 48, 1890, p. 403.

I cannot conclude my introductory remarks without calling to your attention and emphasizing the far-reaching importance of



the proclivity of the abductor muscles to paralysis in progressive organic lesions.



Whilst Rosenbach (15) on the strength of a case of cancer of the oesophagus, accompanied in the beginning by paralysis of both abductors, later of both recurrent nerves, was the first to proclaim that the abductors are the first to suffer from compression of the recurrent, the credit belongs to Semon to have proven that this important pathological condition is seen not only in local lesions, affecting the nerve along its course, but also in central, cerebral as well as spinal diseases of an organic character.

His first publication (16) appeared (1881) in an American journal of high standard, devoted to our specialty, but meeting with a premature death on account of the indifference of the Laryngologists at that time, viz.: the *Archives of Laryngology*. His statements met with a host of opposition and elicited controversies, sometimes of an acrimonious character and not always of the highest scientific standard. By his incessant labors, extending from 1881 to 1897, and by publications too numerous to quote, he had finally the satisfaction to have the so-called Semon Law universally acknowledged.

It is beyond the scope of these remarks to enumerate the arguments he put forth to support his assertion, the truth of which all of us had undoubtedly frequent occasion to confirm.

The ultimate, true cause of the proclivity of the abductors to paralysis has not been definitely determined yet. Grabower (17) sought not long ago to solve the problem by anatomical, instead of experimental researches, previously employed. He examined the innervation of the laryngeal muscles in regard to the configuration of their last nerve terminals, and counted the number of nerve fibres in each muscle. He found that the branch for the abductor does not divide into smaller ones, radiating in different directions, as in the adductors, and that the number of nerve fibres is less in the former than in the latter. The *posticus f. i.* has in the average 281 terminal fibres, the external and internal thyroarytenoid 485, the transversus 279, and the sum of all the adductor fibres is 680.

I have so far not been able to find reports confirming Gra-

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15. Breslauer aerztliche Zeitung 1880, Nos. 2 and 3.

16. *Archives of Laryngology*, Vol. II., 1881, No. 3, p. 197.

17. *Archiv fuer Laryngologie*, XVI., 1904, p. 189.

bower's investigation, but without feeling competent to pass on its merits, I am free to say that I would consider it a most acceptable solution of the question at issue. It would explain also in a natural way by an anatomical anomaly the first and only exception to Semon's statement during the long period of 25 years, observed by Saundby (18) in a case of oesophageal cancer, the analysis of which I have to leave to the gentleman treating of peripheral causes.

Semon (19) admits frankly the facts in Saundby's case, but is not able to offer an explanation. On the occasion of his visit to the United States (1904) I called his attention to Grabower's publication, but the same seemed not to appeal to him. He adheres to his belief in the biological differences in the structure of the laryngeal muscles and nerves, which is evident from experimentation in young and in old animals as well as in different species, and still more from the differential influence of superficial and deep narcosis on the recurrent, which our late member, Hooper (20) was the first to investigate.

### *III. Etiology.*

Recurrent and abductor paralyses of central origin are caused by organic affections, whilst cortical paralysis of a functional character occurs in hysteria, psychical emotions, shock, etc., with concomitant paralysis of the adductors, but not of the recurrent proper.

According to the location and the nature of the diseases we can distinguish paralyses due (1) to cortical lesions, (2) to disturbances of the nerve tract in the internal capsule between cortex and bulb, (3) to bulbar processes, (4) to syphilis, (5) to tumors, exostoses at the cranial base, pressing upon the pneumogastric, (6) to spinal diseases, (7) final paralyses of the Avellis and Hughling Jackson type.

Beginning with paralyses, due to cortical lesions, we enter at once a much contested field.

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18. British Medical Journal, 1904, March 12th.

19. Internationales Centralblatt fuer Laryngologie, 1904, p. 225.

20. Transactions American Laryngological Association, 1887, p. 41, and 1888, p. 163.

A number of cases have been published in which their authors claim that an unilateral cortical affection had caused an unilateral organic paralysis. Whilst in many instances the history, the laryngeal examination, the post-mortem reports, are incomplete or missing, and therefore the proof of the contention made is lacking, there are cases in which the symptoms in the living corresponded with the lesion found after death. Garel (21), for instance, saw a woman 73 years of age, with hemiplegia of the right side and complete paralysis of the left vocal cord. The autopsy showed softening of the left hemisphere and two similar foci in the right one at the base of the third frontal convolution, to the latter of which Garel attributed the laryngeal paralysis. A microscopic examination of the bulbus had not been made, and consequently its integrity cannot be vouchsafed.

On the other hand, cases which appeared during life to belong to this class show bulbar diseases after death. Our member Delavan is quoted amongst the above authors when relating the history of his patient seen during life (22), but in his comment on the result of autopsy he says (23) that his case was a direct proof that paralysis of the larynx of central origin is generally due to bulbar lesions and not to disease of the cortex.

In my opinion, there are at the present time extremely few believers in an unilateral laryngeal paralysis brought about by unilateral cerebral disease; otherwise the number of cases would certainly be much larger than we find in the literature as unilateral cortical lesions, for instance, hemorrhage, softening, etc., are not infrequent. Semon in his article in Heymann's handbook (1898) could collect only 25 cases in which such a coincidence was claimed by the authors. The strongest argument against the occurrence of these paralyses is the anatomical fact that laryngeal movements can be excited by each of the two cortical centres, independent of the loss or destruction of one of them; further, that the experimental extirpation of the cerebral hemispheres down to the fourth ventricle does not interfere with the respiratory movements of the cords, the integral function of the abductor.

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21. *Annales des maladies d'Oreille, etc.*, 1886, p. 218.

22. *Transactions American Laryngological Association*, 1884, p. 82.

23. *Ibidem*, 1888, p. 195.

Affections involving both cortical centres, for instance hemorrhages, are as a rule not confined to this area alone, and are of such a serious character that the laryngeal condition is overshadowed by the grave general symptoms. But cases are reported in which the connection between the cortical and bulbar centre through the internal capsule became obliterated by the morbid process with resulting aphasia.

Eisenlohr's (24) patient had general motor disturbances, was absolutely speechless, and the post-mortem showed destruction of the posterior third of both thalami optici, partial degeneration of the posterior part of the internal capsule and of the pyramidal tract of the medulla and spinal cord. Pathological changes of a similar nature were found after death and reported by Pitt (25 and 26) in a case of chronic nephritis with two apoplectic attacks in quick succession, the second one being followed by loss of voice. In both cases bulbar and cortical lesions were absent and the patients experienced no respiratory difficulties during life.

Similar laryngeal symptoms, associated with lingual and labial pareses, are observed in pseudobulbar paralysis, due to disseminated cerebral lesions, in which disease of the medulla and respiratory troubles are absent, and the laryngeal muscles undergo no fatty degeneration.

The subsequent chapters can be considered more briefly, as they deal mostly with well-known data, and offer no field for widely dissenting views. Under bulbar paralyses I have included all cerebral diseases, irrespective of the primary seat being in the bulbus or in the hemisphere. Further, the etiological features of recurrent and abductor paralysis will not be treated of separately, as both are liable to be caused by the same affection, and as the latter is in many instances only a precursor of the former. Prevalence of one or the other type in special diseases will be mentioned in its proper place.

Progressive bulbar paralysis, being due to a degeneration of the motor nuclei in the medulla, represents a genuine type of the bulbar origin of laryngeal paralysis. As a rule paralysis of other organs, tongue, lips, precedes the laryngeal symptoms, which ap-

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24. *Deutsche Zeitschrift für Nervenheilkunde*, Vol I., 1891, p. 388.

25. *British Medical Journal*, 1893, May 20, p. 1058.

26. *Transactions Clinical Society*, London XXVI., 1893, p. 250.

pear sooner or later, dependent on how early the laryngeal nuclei are attacked. The laryngoscopic picture varies in different patients, according to the focus and the intensity of the process. Complete recurrent and unilateral as well as bilateral abductor paralysis has been observed, whilst some authors claim to have seen also adductor paralysis.

Another bulbar affection occupying the same or also adjacent areas is the apoplectic form of bulbar paralysis, caused by disease of the blood vessels of this region, producing hemorrhages, thrombosis with subsequent softening, also embolism of the vertebral or basilar arteries. Many reported cases of cerebral hemorrhages and encephalomalacia with concomitant laryngeal disturbances are undoubtedly due to such lesions, which have been described already as early as 1879 by Eisenlohr (27 and 28). If the patient survives the attack and its sequelae, we can expect to find a recurrent paralysis, if all the motor fibres are destroyed, otherwise an abductor paralysis.

In the different forms of meningitis we observe anomalies of sensation, but seldom paralysis of the larynx. Our late member Major (29) saw a patient with bilateral abductor paralysis after cerebro-spinal meningitis, tracheotomy was performed and the patient's condition remained stationary for seven years, when he died by an accident.

Cases of paralysis from multiple cerebro-spinal sclerosis are rare, and the laryngeal symptoms depend on the extent of the disease. Loeri (30) reported several cases, one of which had recurrent paralysis, J. Solis-Cohen (31) (1886) a case of unilateral, later bilateral abductor paralysis, shown to this Association seven years before. From the carefully made autopsy he concludes that the paralysis has been caused by an ascending sclerosis. Rethi (32) collected in a recent publication forty-two cases of

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27. Archiv fuer Psychiatrie, Vol. IX., 1879, case 3 and 4, p. 19 and 22.

28. Ibidem, Vol. XIX., 1888, p. 316, containing also report of autopsy of cases 3 and 4 of 1879, p. 315 and 316.

29. N. Y. Medical Journal, 1890, June 28.

30. Die durch anderweitige Erkrankungen bedingten Veränderungen des Rachens, Kehlkopfs & der Luftröhre, 1885, p. 13.

31. Transactions Am. Laryng. Assn., 1886, p. 42.

32. Die laryngealen Erscheinungen bei multipler Sclerose des Gehirns und Rückenmarks, Wien, 1907.

sclerosis from the literature and gave a full description of the subject in all its bearings.

Although infectious and toxic paralyses do not belong to the part assigned to me, I cannot refrain from mentioning syphilis of the brain as a cause of recurrent or abductor paralysis. M. Mackenzie (33) published already (1873) one case, and Lefferts (34) (1878) two cases of bilateral abductor paralysis due to syphilis, all three cured by specific treatment.

Tumors at the base of the brain are liable to produce paralysis by pressure on the pneumogastric. The symptoms are generally not confined to the larynx alone, as the extension of the growth is apt to implicate other nerve roots also. Nothnagel (35) communicated an exceedingly interesting case of an extended unilateral paralysis, involving the recurrent of the same side, which was due to pressure from a large abscess at the cranial base, found at the autopsy. Similar symptoms we must be prepared to meet in affections of the cranial bones, exostoses, fractures of the base of the skull, etc.

The importance and the deleterious influence of locomotor ataxia on the larynx is too well known to all of us, as to need but brief remarks. General medicine is indebted to laryngology because in more than one instance the laryngologist detected abductor paralysis in an otherwise healthy person with a perfectly clear voice, and suspicion being aroused, the examination of the patient revealed locomotor ataxia. By far the most prevalent type is abductor paralysis, of which again the bilateral form is more frequent than in any other disease. Burger (36) collected in his excellent monograph seventy-one cases of paralysis in tabetics, covering the period from 1866 to 1891, forty-three of which had abductor paralysis. Still more striking is Sendziak's (37) supplementary table of fifty-three cases of paralysis published between 1892 and 1898. Of these six only had recurrent, of the balance twenty-two unilateral and twenty-five bilateral abductor paralysis.

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33. Diseases of the Nose and Throat, London, 1880, vol. i., p. 429, case 6.

34. N. Y. Medical Journal 1878, December.

35. Wiener Medizinische Blätter, 1884, No. 9, February 28, p. 256.

36. Die laryngealen Störungen der Tabes dorsalis, Leyden, 1891.

37. Klinische Vorträge an dem Gebiete der Otologie und Pharyngo-Rhinologie, 1901, vol. iv., No. 3, p. 170 and 171.



In the remaining spinal diseases we find only isolated cases of laryngeal paralysis, a few of which may be mentioned. Lori (38) observed a case of bilateral recurrent paralysis in amyotrophic lateral sclerosis, and one in acute spinal lepto-meningitis, Schroetter (39) and Gavello (40) each a case of unilateral recurrent paralysis in syringomyelia, and Koschlakoff (41) a bilateral abductor paralysis in progressive muscular atrophy.

Although finding scant consideration in the text-books, a series of unilateral laryngeal paralyses, associated with such of other organs is of sufficient interest to be mentioned.

Avellis (42) was the first to draw attention to the occurrence of simultaneous unilateral paralysis of the larynx and soft palate—the genuine Avellis-syndrom. In another form the muscles of the neck are also paralyzed. If hemiplegia of the larynx, pharynx and tongue exists, we have the Hughling-Jackson type. A typical case of the latter with abductor paralysis was demonstrated in the London Laryngological Society by Semon (43), who, in consideration of the general symptoms, believed it to be of central origin, due to a meningeal thickening at the base of the brain. Tapia (44), who observed unilateral paralysis of the larynx and tongue, called it the fourth type, and finally Pohli (45) gave a complete description of the subject in the Italian Archives, 1906.

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38. *Loco Citato*, pp. 19 and 16.

39. *Krankheiten des Kehlkopfs*, Wien, 1892, p. 413.

40. *Arch. Ital. di Otol., Rinol. e Laring* XVII., 1935, Novbr., p. 25.

41. *Internationales Centralblatt fuer Laryngologie* II., 1885-6, p. 229.

42. *Berliner Klinik*, 1891, vol. 40, October.

43. *Journal of Laryngology*, 1907, February, p. 50.

44. *Annales des maladies d'Oreille*, August 1906, p. 134.

45. *Arch. Ital. di Otologia, etc.* XVII., 1906, March, p. 215.

*Symposium—Second Paper.*

THE ETIOLOGY OF PERIPHERAL PARALYSIS OF THE  
RECURRENT LARYNGEAL NERVES.

BY D. BRYSON DELAVAN, M.D.

Reviewing the history of this subject, we find that at a period centering about twenty years ago much interest was shown in its study. Led by Prof. Hermann Krause in Europe, and by the late Prof. Franklin H. Hooper in this country, excellent laboratory work was done and some important facts were demonstrated. Of the main questions at issue, however, many were unanswered, in spite of the earnest efforts of a number of well-qualified investigators and of controversies which, in some instances, assumed the proportions of bitter feuds. Of late years not much has been advanced on the subject, and that little has not been illuminating. Possibly the difficulty of the study and the lack of success attending its investigation have discouraged progress. Possibly again, in view of the present popularity of other adjacent fields, the larynx has received less attention recently than it deserves. The serious nature of recurrent paralysis and the not inconsiderable number of patients affected by it should certainly stimulate us to more earnest effort toward the better understanding and explanation of the causes which bring it about.

In many varieties of the affection the cause is clear. In others it is obscure, as witness the number of cases in which the diagnosis has either not been made or has been clouded by conflicting opinions as to the actual effect of the supposed cause.

I shall be able to do little more than call attention to some of the questions which the subject suggests and ask you to interest yourselves in taking up their investigation.

Indeed, in spite of all that has been written on the subject, it may be said that the study of this department is in its infancy, for it has not even been determined whether certain toxic paralysees

of the recurrences are due to central causes, to changes in the nerve elements themselves, or to neuritis from severe surface irritation.

Recurrent paralysis may be either unilateral or bilateral, and only the posterior fibres of the nerve may be involved, as in abductor paralysis, or the paralysis may be complete.

Shurly (1) quotes Avellis to the effect that of one hundred and fifty cases the cause was discovered in only eighty-five. Of the above one hundred and fifty cases, plus nineteen collected by Sendziak (2) one hundred and thirteen were males and fifty were females. In the same series of cases the paralysis was found forty-six times on the right side, ninety-two times on the left side, and twelve times on both sides. In forty-one cases of Sendziak five were right, fifteen left and twenty-one bilateral. Unfortunately, little reliability can be based upon these statistics. Three points became evident from them, however, as has been suggested by Lermoyez, namely, the preponderance of peripheral paralysis over that caused by central lesions; the excess of cases in which the paralysis has been of the left side, and, finally, the greater liability of recurrent paralysis among men.

As to age, of nineteen cases fifteen occurred between the twentieth and sixtieth years. The youngest was a child of two years, the eldest was fifty-nine.

Sendziak claims to have collected one thousand and seventeen cases which he has classified according to the form of paralysis, as follows:

|   |      |            |
|---|------|------------|
| 1. Abductor paralysis .....               | 526  |            |
| 2. Paralysis of recurrent.....            | 491— | 1017       |
| <hr/>                                     |      |            |
| 1. a. Unilateral abductor paralysis.....  | 283  |            |
| b. Bilateral abductor paralysis.....      | 243— | 526        |
| 2. a. Unilateral recurrent paralysis..... | 397  |            |
| b. Bilateral recurrent paralysis .....    | 94—  | 491        |
|   |      | <hr/> 1017 |

Among the earliest observations noted with regard to them was the fact that if the recurrences were injured dyspnoea was a prompt result. This was made apparent through the effect upon

them of wounds of the throat and neck. Later on the possibility of paralysis from pressure was recognized. Finally, within recent years, a few observers have called attention to the occurrence of paralysis from toxic causes. Many cases have been reported in which the movements of the larynx have been affected by peculiar conditions of the blood, due to disease, or to the ingestion into the system of certain well-known poisons. Thus gradually it has become apparent that paralysis of the recurrences of peripheral origin may be due to many causes.

The consideration of paralysis due to traumatism and to pressure are very interesting topics. Moreover, the subjects are far from having been exhausted. Many things remain to be explained regarding both of them.

Paralysis due to toxic agencies, however, is a subject which has been hitherto little studied. It is replete with problems of many kinds, and these again depend upon questions of surpassing interest. Few fresher or more attractive fields could be offered to the investigator, whether physiologist, pathologist or chemist.

In studying the reports of cases by various authors the writer has been impressed with the belief that some of them were probably not cases of actual paralysis, but of ankylosis (3). This is especially likely to be true in such diseases as typhoid, erysipelas and other conditions attended with severe local inflammation adjacent to the joint, or, as in rheumatism and gout, with inflammation of the joint itself.

The possible myopathic origin of loss of motion in the larynx must also be remembered. This condition may be present in wasting diseases, muscular atrophy, rheumatism and carcinoma.

The possibility of error in deductions based upon the statistics of reported cases of paralysis, some of which may have been wrongly diagnosed, must therefore be taken into account.

While first asking attention to the better known departments of my subject, it is to the toxic conditions that I shall finally bespeak especial attention and interest, believing that the time has come when light should be thrown upon the obscurities which surround them and hoping that ere long more complete and definite knowledge may be secured.

The causes of recurrent laryngeal paralysis of peripheral origin, as far as now known, may be divided into three general groups, namely.

1. Cases due to traumatism or laceration of the nerve trunks.
2. Cases due to mechanical pressure upon the nerve.
3. Cases due to toxic influences, either of the nature of poisons ingested into the body or of toxæmias or infections generated by disease.

As has already been said, careful distinction must be made between actual paralysis and simple loss of motion due to myopathic causes. Also from loss of motion from ankylosis of the crico-arytenoid articulations.

Of the various causes of paralysis of the recurrents, I shall first consider traumatism.

Laceration of the nerve trunk may occur in any part of its course and from a variety of accidents, and the injury may be inflicted from without the larynx and trachea or from within them.

The list includes incised, gun-shot and punctured wounds of the neck, self-inflicted or accidental; injuries to the nerve made in the course of surgical operations done upon neighboring parts; foreign bodies in the larynx, trachea or oesophagus and unsuccessful efforts made at their extraction; injury to the nerve from attempts at strangling and from blows upon the larynx.

Division of the recurrent happens occasionally in cases of cut throat. The possibility of its existence should be recognized before attempting the complete closure of such wounds, as the effects of sudden urgent dyspnoea would of course be very serious. Lefferts (4) has reported a case of paralysis of the right recurrent nerve due to a punctured wound made by the point of a blade of a pair of scissors.

The recurrents are liable to injury in operations for the removal of various tumors, especially of the thyroid gland. This is particularly true of goitre, as a number of recorded instances will attest.

In view of the increasing popularity of the operative treatment of goitre this should be carefully remembered.

Operations for the removal of new growths of the neck in which the necessary dissections approach the vicinity of the recurrents may easily be attended with injury to them, as also may attempts at the extirpation of diseased lymph nodes lying near to the larynx or trachea.

The writer has seen cases of paralysis of the right side of the larynx following extensive extirpation of the cervical glands in recurrent carcinoma of the right breast.

The position usually occupied by the left recurrent behind the inferior thyroid artery renders it liable to injury during ligation of the latter.

We will now consider the subject of recurrent paralysis due to mechanical pressure.

The list of known causes includes: Diseases of glands and of lymph nodes; new growths; aneurisms; diseases of the heart; diseases of the pleura; scoliosis.

Clinical experience seems to prove that this division presents by far the largest number of the cases that come under observation. Scientifically speaking, they are more interesting than the cases due to traumatism, for while some are relatively common others are rare, and while some—fortunately, as a rule, the more common ones—are generally rather easy of diagnosis, there are no small number which are very difficult. So obscure are they that a positive diagnosis with the means of examination at present available to us seems to be impossible.

Many cases hitherto obscure have of late years been made clear by the aid of the Roentgen ray. Perhaps nowhere among the varied uses of that splendid contribution to science have more brilliant successes been obtained.

We will begin with the consideration of the diseases of the glands and of the lymph nodes.

That a diseased condition of the thymus gland has ever been the cause of laryngeal paralysis I have not been able to learn.

Disease of the thyroid gland, however, is a common cause, occasionally from malignant involvement, but far more often from goitre.

Remembering the anatomical relations of the thyroid with the trachea it is easy to understand how the recurrences might be encroached upon by almost any swelling of that gland.

The size of the thyroid tumor seems to have little effect upon the degree to which it may endanger the recurrences. Thus a very large goitre may cause little or no dyspnoea. On the other hand, one of small size may prove fatal.

The most striking instance of this which the writer has ever



seen came to his notice when he was Assistant Pathologist to the New York Hospital, some years ago.

A middle aged lady, of rather full habit, while shopping, was suddenly seized with urgent dyspnoea. She was hurried to the New York Hospital, which was near by, and within a few minutes after the onset of the attack tracheotomy was performed. Before the cannula could be inserted she died.

At an autopsy made by Dr. George L. Peabody the thyroid appeared to be slightly enlarged. Upon exposing it by dissection a goitrous tumor, parenchymatous in nature, was found, the base of which did not measure quite four inches in diameter. It was so situated with regard to the trachea, however, that both recurrents had been pressed upon sufficiently to cause paralysis, or, as is more probable, spasm of the larynx and asphyxia.

Disease of the tracheo-bronchial lymph nodes has been the cause of recurrent paralysis in a number of recorded cases. These may have become enlarged from syphilis, tuberculosis, malignant infiltration or other infections.

In a case seen by the writer at St. Luke's Hospital, New York, a child two and one-half years old suffered from urgent dyspnoea requiring a prompt tracheotomy which failed to give complete relief. Death followed in a few days. The child was found to have general tuberculosis, and the cause of the dyspnoea was a large lymph node pressing upon the middle section of the trachea. Great obscurity must often attend the recognition of these cases. The fact of their possibility, the presence of a condition capable of causing them, the absence of other discoverable cause, and, above all, the use of the X-ray may all aid in arriving at the truth.

Malignant disease of the thyroid, the oesophagus, the neck and the mediastinum is a common, if not the most frequent, cause of recurrent paralysis.

In all of these but the oesophagus it is generally due to direct pressure upon the nerve trunk. While sometimes due to direct pressure from disease of the oesophagus, it may also, in the latter case, be caused indirectly through the extension of the disease when located in the upper part of the oesophagus to the larynx or trachea, so that both recurrents are surrounded by cancerous masses and thus totally paralyzed. Or the paralysis

of the nerves may be direct, from the pressure of degenerated cancerous glands upon them.

Sandy (5) has reported a case which has become famous for two reasons, first, because as a result of oesophageal carcinoma adductor paralysis was first observed. Later this disappeared and a condition of purely abductor paralysis took its place. Finally the cords assumed the cadaveric position, thus proving the complete paralysis of the nerves. A second point of great interest in this oft-quoted case is that it seems to contradict the so-called "law" of Semon as to the supposed greater vulnerability of the abductor filaments of the recurrent nerve.

In the minds of most practitioners the discovery of fixation of the left side of the larynx is almost proof positive of the presence of an aortic aneurism. Very often this view is correct. In the experience of the writer with the cases that have come under his observation aneurism of the aorta has been the most frequent cause of left recurrent paralysis. The mechanism of this is too familiar to require special explanation. It must be remembered that bi-lateral paralysis, with its attendant dangers, may be developed by the growth of the aneurysmal tumor in its late stages.

The position of the right recurrent with relation to the right subclavian artery is such as to render it vulnerable in case of aneurism of that vessel. Such cases are very rare in comparison with those of left recurrent paralysis due to aortic tumor.

Aneurism of the innominate has been said to be an occasional cause.

Cases have been reported in which recurrent paralysis has been caused by various diseased conditions of the heart, especially valvular disease in the form of mitral insufficiency and of pericarditis. By the use of the term valvular disease in this connection we are probably meant to understand the effects of it in the form of dilatation or hypertrophy, the increase in the size of the organ causing it to press upon the recurrences, as would be the case in pericarditis. Apropos of this subject the case reported by Baumler (6) presents features of extraordinary interest.

A young man suffering from syphilis and an old mitral insufficiency lost his voice synchronously with the appearance of a pericardial exudation. Examination of the larynx showed a bi-

lateral abductor paralysis. With beginning resorption of the exudate the voice gradually returned, at first rough and hoarse. Two weeks later it became entirely normal and remained so until the patient's death.

From its position with regard to the apex of the right lung the right recurrent may be pressed upon under certain morbid conditions of that part.

Thus, in fibrous phthisis, such cases have been found. They have also been caused by thickening of the pleura covering the right lung apex. It is said that the same thing has been seen in the case of the left lung.

Finally a few cases are reported in which paralysis was attributed to pneumonia, pneumothorax and pleurisy. That the paralysis was actually due to pressure and not the result of severe peripheral inflammatory conditions remains to be proved.

By far the most interesting group of cases which come within the scope of this article are those due to toxic influences. They are less frequently observed than those caused by traumatism or by pressure, so that opportunity to study them has not so often been given.

Moreover, the physiological and pathological processes by which they are brought about are far too little discussed and much less well understood. It is proper, therefore, that particular attention should be called to them both by reason of their pathological importance and because they offer one of the most interesting and valuable fields for original research to be found within the range of medicine.

The ingestion of certain drugs and chemicals has been believed to produce in some cases paralysis of the recurrent laryngeal nerves.

In like manner recurrent paralysis occasionally accompanies certain acute infectious diseases. If it be true that the poison acts directly upon the nerve elements through some especial affinity of the one for the other, then the variety of toxics capable of so acting is remarkable, and it suggests a vulnerability of the recurrences to toxic effects which is, to say the least, unusual and peculiar. Other sets of nerves may be similarly acted upon, as in poisoning from lead, alcohol or diphtheria. The number of

different toxins which have been said to affect the recurrents, however, seems unusually extensive.

The list of causes of toxic paralysis of the recurrents should be divided into two groups: First, mineral and vegetable poisons; and secondly, toxins generated by certain acute infectious diseases. To the former belong lead, arsenic, antimony, copper, iodide of potassium, iodoform, and, it has been alleged, cyanide of potassium and phosphorus.

Recurrent paralysis due to other mineral poisons must be very rare. A few cases have been observed which seem fairly to have been due to the influence of arsenic, iodide of potassium and iodoform.

Those referred to antimony, copper, cyanide of potassium and phosphorus are apparently of doubtful nature.

The first case on record in which the influence of lead upon the recurrents was observed by the aid of the laryngoscope was seen by Sir Morell Mackenzie (7) in 1865. He reported a case of complete paralysis at the right side of the larynx in a young man suffering from general lead poisoning. The paralysis disappeared after two months' application of electricity.

Our fellow member, Sajous, (8) in 1882 reported a case of bi-lateral paralysis of the larynx due to lead poisoning, and Seifert follows in 1884 with three well observed cases. A few others have appeared in literature up to the present time. The cases reported in which other minerals have been thought to produce recurrent paralysis have been so few in number as to make deductions impossible. They suggest the possibility of coincidence quite as strongly as they do that of cause and effect.

Paul Heymann (9) collected over fifty cases of mineral poisoning of the larynx. He found lead to be the most common cause. Usually the abductors were affected.

Of the vegetable poisons the following have been alluded to in the literature of the subject: Alcohol, opium—morphine, belladonna—atropine, cannabis indica, cocaine.

Among the vegetable poisons mentioned alcohol properly heads the list. Hippocrates (10) calls attention to that form of aphonia of the drunkard which comes upon him in his cups and does not disappear when he has become sober. Several good authorities have quoted the co-existence of recurrent paralysis with alco-

holism. Cases have also been reported as being due to opium and to belladonna, and it has at least been suggested that similar results may arise from cannabis indica and from cocaine.

The questions raised by the action of the drugs and minerals just described upon that always peculiar pair of nerves, the recurrent laryngeals, are perplexing. In what manner do they inhibit the action of these most active parts?

1. Is there any truth in the supposition that there is an elective affinity existing in these particular nerves in favor of certain drugs, through the influence of which the one exercises an inhibitory effect upon the other?

2. Does the drug produce changes in the nerve tissue itself which result in the loss of its activity?

3. Is the lesion a neuritis, induced by pressure from congestion of neighboring parts or otherwise?

4. Are the effects upon the recurrences due to central causes, the latter induced by the drug?

Turning now from the mineral and vegetable poisons which may affect the recurrences, we take up the consideration of the alleged results produced by the toxins of some of the acute infections. These include typhoid fever, acute articular rheumatism, influenza, diphtheria, typhus fever, acute croupous pneumonia, puerperal fever, erysipelas, measles, scarlet fever, gonorrhœa, serum therapy.

Of seventy-three cases collected by Sendziak twenty-six occurred in typhoid fever, eighteen in acute articular rheumatism, eleven in influenza, nine in diphtheria, three in typhus fever, two in croupous pneumonia and one each in puerperal fever, erysipelas, measles, scarlet fever, gonorrhœa and following the use of serum therapy.

Of the infectious diseases in which recurrent paralysis may be found, typhoid fever seems decidedly the most common. The paralysis may be unilateral or bilateral. It may occur early in the course of the disease, but it generally appears toward its close during the third or fourth week.

Osler (11), agreeing with the generally received opinion, attributes it to a neuritis of the recurrent and states that the general activity of the toxins in a given case has more influence upon the nervous system in causing the neuritis than has the temperature.

In other words, the neuritis is not so apt to be developed in a case where the temperature is running a high course as it is in one in which the typhoid intoxication is profound.

Laryngeal paralysis in a severe case of typhoid is, of course, an exceedingly grave complication, less so in the milder cases of fever and where the paralysis is unilateral.

Regarding the cause of paralysis, the question again arises as to whether, after all, the nerve irritation is not brought about by the violent surface inflammation commonly met with in and around the larynx in this disease.

While most writers state that the paralysis of the recurrents in typhoid is generally due to neuritis, certain writers believe that the loss of motion is sometimes myopathic. Degeneration of the laryngeal muscles may take place in like manner with the degeneration of the abdominal muscles in typhoid.

Moritz Schmidt (12) says that recurrent paralysis has occurred in typhoid patients suffering from complicating pleuro-pneumonia, glandular swelling and muscular degeneration.

May it not be possible that some of the instances reported as being due to typhoid toxæmia have, after all, been caused by such complicating conditions?

In typhus fever, as in typhoid, recurrent paralysis may supervene. It is said to be more frequent in typhus than in typhoid; it is likely to occur at an earlier period of the disease and to be of a more serious nature.

In acute articular rheumatism recurrent paralysis has been found in all its phases: unilateral, bilateral, abductor and complete.

Here again the causes are variously stated, neuritis and rheumatic affection of the laryngeal muscles having the preference.

Clinton Wagner and others have reported cases of gouty inflammation of the crico-arytenoid articulation with perichondritis, necrosis, abscess and sloughing of the arytenoid cartilage. The possibility of such a case should suggest the possibility of rheumatic affection of the laryngeal muscles and admit of the assumption that not every case of loss of motion in the larynx is necessarily neuropathic.

Moritz Schmidt (12) says that he has not infrequently seen recurrent paralysis following influenza, and other writers have



reported a few such cases. This does not seem improbable in a disease capable of affecting so profoundly the nervous system in general.

Moritz Schmidt (12) also calls attention to the fact that in diphtheria it is still questionable whether the resulting paralyses are due to central or to peripheral causes. In the opinion of the writer it is not necessary that fixation of the larynx following diphtheria should be due to paralysis at all, since under condition of inflammation as violent as those often found in laryngeal diphtheria, ankylosis of the crico-arytenoid articulation might easily be produced.

In several cases seen by the writer of unilateral loss of motion present many years after an attack of diphtheria this has seemed the true explanation. The question is rendered still more difficult, however, when we remember that following a genuine paralysis from any cause the joint may ultimately become ankylosed through disuse.

Apparent paralysis of the larynx may also accompany the severe infections of the throat non-diphtheritic in type, as, for example, the Staphylococcus and Streptococcus. It seems more probable that the loss of motion is due rather to the effects of inflammation upon the muscles and joints of the larynx than to its effects upon the recurrences.

One case each is reported of recurrent paralysis from erysipelas, puerperal fever, scarlet fever, measles, serum therapy and gonorrhœa. In all but the first, the paralysis was unilateral.

The last named case has been elaborately described in an extensive and scholarly article by Lazarus (13).

With regard to toxic paralysis some very interesting questions arise:

1. If it be true that there is such a thing as a "selective affinity" between the recurrences and a given poison, in a case of poisoning we would expect to find both recurrences affected. In point of fact, mineral poisons seem almost invariably to affect but one.

Again, if the toxins of infection cause paralysis by exciting a recurrent neuritis, we would expect the result to be bilateral, whereas a considerable proportion of such cases have been

unilateral. Both of these facts seem to contradict the affinity doctrine.

2. Is it possible that toxins, whether inorganic or organic, produce special structural changes in the nerve tissue itself which result in recurrent paralysis?

For the answer to this question we must look to the pathologist, although it would seem that a simple inflammation of the nerve would be all that was necessary to inhibit its activity.

3. Are the effects of toxics upon the recurrences due to central causes? Here again the fact that bilateral paralysis is rare and unilateral the rule would throw doubt upon the probability of an affirmative answer.

4. Is the lesion a neuritis, induced by pressure from congestion of neighboring parts or otherwise? It seems probable that in many cases it is, since this would explain the presence of recurrent paralysis from so many different varieties of causes, most of which are associated with congestive conditions of the throat, and for its occurrence in so many different forms.

It is easy to suppose that an inflammatory condition severe enough to cause a crico-arytenoid ankylosis might cause a paralysis of nerves lying as near the surface as do the recurrences.

It is impossible to escape the conviction that some of the cases of recurrent paralysis found accompanying violent inflammations of the throat have been due to the effects of superficial irritation.

Nothing is more common than to find a paralysis of the posterior arytenoideus muscle accompanying an acute laryngitis. The position of this muscle near the surface, and at a part of the larynx where the inflammatory conditions are apt to be most intense, would render its nerve filaments peculiarly liable to irritation and injury.

Percy Kidd (14) reported a case of bilateral paralysis of the larynx, cadaveric position, following a severe laryngitis, lasting about three weeks and ending in recovery.

Such cases are not unique. If they suggest anything, it is that surface irritation may be the cause of recurrent paralysis. In many of the diseases in which recurrent paralysis is reported to have occurred inflammation of the throat is a marked symptom.

If it can be caused by the irritation of a simple laryngitis, it certainly is possible that the still more violent inflammations ac-

companying severe acute infections may produce it. Doubtless, among both groups of toxic causes there are some which may act in this way.

These are some of the questions that remain to be explained and finally answered. If my presentation of them has been imperfect and my deductions inadequate, it must be remembered that they have thus far baffled the efforts of all who have attempted their solution. With the advance of knowledge of the nervous system in general and with the increased accumulation of clinical material it is to be hoped that better progress may ere long be made.

A study of the literature of the subject shows that it leaves much to be desired. Considering the large variety of conditions which may cause it and the vast number of the cases which many of these conditions create, as, for instance, traumatism, tumors, cardiac diseases, tuberculosis, typhoid, diphtheria, influenza, etc., it is strange that relatively so few recurrent paralyses have been recorded. Doubtless more careful observation aided by laryngoscopic examination would demonstrate many additional ones. Here, as is the case in many other departments, many of the histories have been too incompletely recorded to be of any value.

The latest writer, Sendziak (2), claims to have found 1017 cases, 201 of which, he says, are his own. Where he found them he does not say. Unfortunately he does not give the references to them so that his statistics cannot be verified nor can the articles from which he derived his material be studied. His own articles being published in the Polish language and in obscure provincial journals make them almost impossible of access.

The article of Lermøyez (3) is classic and well worthy of study. Paul Heymann's (9) monograph on recurrent paralysis due to the effect of poisons is excellent. It contains a full and very valuable bibliography.

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*Symposium—Third Paper.*

## SYMPTOMATOLOGY.

BY CLARENCE C. RICE, M.D.

The division of this most interesting subject which has been allotted to me would at first sight seem a less difficult task than that of the gentlemen who have just read their admirable papers; but when it is appreciated that several varieties of either spasm or paralysis of the vocal bands may co-exist, the tabulating of an exact classification of symptoms is not an easy one. It is desirable to select the commonest forms of paralysis of the laryngeal muscles and give their usual appearances and symptoms, rather than to complicate the description by stating the symptomatology of a combination of conditions. A discouraging amount of literature has been written upon the subject of paralysis of the muscles of the larynx, discouraging because we have often found it confusing, and it has been a relief to write, for the most part, from our own observations.

Let us, therefore, endeavor to portray a picture of the clinical phenomena which are present in paralysis of the several muscles and groups of muscles of the larynx. It seems desirable to present the subject of symptomatology under two grand heads or divisions, namely: First, the symptoms, laryngeal and general, of the several temporary disturbances in the efficiency of the laryngeal muscles; disturbances not caused by grave or permanent pathological conditions of the nerve centres, but by injuries inflicted either upon the peripheral nerve filaments or upon the muscular tissue itself; the so-called "myopathic" disturbances occurring in the various forms of catarrhal laryngitis, the acute and the chronic, either of the moist or dry variety, and the "traumatic" type of laryngitis, a name properly used in describing the crippled vocal conditions of singers and professional people generally. We suggest this as the first division of our sub-

ject, and since it has not been asked of us to describe the impairment of efficiency of the anterior muscle which is supplied by the superior laryngeal nerve, we must first speak of the laryngeal image and symptoms which are exhibited in paralysis of the "internal" and "transverse" muscles. I should like to be permitted to use the term "transverse" for the arytenoideus muscle, "internal" for the thyroarytenoideus, "lateral" for the large adductor, and "posterior" for the solitary abductor.

The second division of the subject deals first with the symptomatology of paralysis of the large adductor; second with that of the abductor of the vocal cords, and third with that of total paralysis of the recurrent nerve.

We suggest that it will simplify matters if we keep this division widely apart in our minds, because in the first or myopathic class the laryngeal image of disturbance is constantly changing, and may present abnormal laryngoscopic appearances for but a short time, because the tone of the muscles is apt to be rapidly restored by rest and intelligent remedies. It is of interest to radically separate the fatigued appearances of the internal and transverse muscles from the widely different exhibition presented by a permanently disabled lateral adductor, caused by lesions of nerve trunks; a paralysis in the full sense of the term, a condition usually not to be improved because the lesion is permanent and often progressive.

In our first division, paralysis of the internal muscle is by far the most common. We can say that it is nearly always one of the manifestations of a severe grade of laryngitis. The college boy attending a single game of football can so abuse his vocal apparatus as to become aphonic the following day and his vocal bands present the characteristic elliptic opening, extending from vocal process to the anterior commissure. The picture is familiar to us all. The degree of hoarseness or loss of voice depends upon the diameter of the ellipse as well as upon the accompanying general inflammatory conditions. The history of the case is so simple as to make the diagnosis itself. The disturbance in these acute cases is nearly always bilateral. If such injury as shouting and screaming is inflicted upon a perfectly healthy larynx, the vocal bands of the aphonic patient will next



day present the elliptical opening, and the margins of the bands will be markedly concave, but there will not necessarily be any accompaniment of congestion and swelling. In some cases the wider separation of the vocal bands, at attempts of phonation, would indicate that the lateral adductors had also been injured by the unusual strain. When such injury as we have described is imposed upon a larynx which has been the seat of a chronic catarrhal condition, especially of the dry variety, congestion and perhaps thickening of the bands will be associated with the concaved margins of the vocal bands.

Again, we sometimes see cases where, in addition to the elliptic concavity symptomatic of paresis of the internal tensors, we find the bands very strongly abducted; bands not in the cadaveric position of recurrent paralysis, but resembling the picture, which theoretically should indicate a combination of internal and lateral paralysis. The patient in these cases is entirely without voice. I have seen vocal cords completely abducted to the sides of the larynx and concave throughout in cases of chronic laryngeal catarrh associated with a far advanced atrophic condition of the nose, pharynx and larynx. I recall no such cases in private practice; they have been in dispensary patients, most commonly in women and, strange to say, in fat women who have usually been cooks. I recall two such cases, and in both there was a severe dry catarrh of the larynx and trachea. The lesion in these cases I have always supposed to be peripheral, due to inflammation of nerve terminations. Both of these cases made a fair recovery.

We have seen unilateral paralysis of the internal muscle, as evidenced by a moderate concavity of one vocal band, in chronic laryngitis where pathological injury was greater on one side than the other. The left cord or its nerve or muscular supply is undoubtedly the weaker of the two. It is not unusual to see concavity of that band with some degree of failure of adduction, associated with such singers' injuries as congestion, general thickening, and perhaps a localized nodule. This impairment of muscular tone is not so easily repaired as in the sub-acute cases where both internal muscles are temporarily enfeebled, because continued abuse has produced a chronic impairment of muscular power.

The symptoms of all disturbances of the internal muscle, whether unilateral or bilateral, aside from the well-known laryngeal image, are those of some degree of vocal disturbance, from simple hoarseness to entire loss of voice, depending upon the degree of separation of the cords during phonation. Even if the power of the internal muscles should remain permanently impaired, the compensatory power of the other adductors would pretty largely approximate the cords sufficiently for the ordinary speaking voice, but as some of the fibres of the internal muscle are attached to the edge of the vocal band, and as they very nicely regulate the tension of the cord, frequent disturbances of this kind will do great and lasting injury to the singing voice. The vocal bands are the tendons of the internal muscles, and when these muscles are paralyzed the bands become more narrow and humped up. The singing voice is frequently impaired when the laryngeal mirror shows no apparent loss of power of any of the laryngeal muscles, and in such cases we are led to conclude that the fault is due to weakness of that portion of the internal muscle which is attached to the ends of the cords. Paralysis of the internal muscles is sometimes called "paralysis of the vocal cords themselves," in contradistinction to all other forms of paralysis.

Paralysis of the short transverse or arytenoideus muscle is much less frequently seen than that of the internal; rarely, I believe, in ordinary catarrhal laryngitis, although the posterior triangular aperture may frequently, to a slight degree, accompany impairment of the "internal" muscle. Where the triangle is large and characteristic, it has been my experience to find it in those cases of local disease of or near the arytenoid cartilages, such as tuberculous infiltration in the posterior commissure. The diagnosis of this condition, if it exists alone, is very easy, and the degree of impairment of voice is in proportion to its degree and to other disturbances of the larynx. The speaking voice is almost normal, but in singing there is phonatory leakage and the voice becomes quickly fatigued. It is interesting in cases of paralysis of the transverse muscle to notice, with the laryngeal mirror, the jerky motions of the arytenoid cartilages during phonation. I suppose this is because this muscle often receives its nerve supply from two sources. I have not frequently

seen this triangular paralysis associated with the concavity of the cords, which indicates inaction of the internal muscle, and it has been our experience that paralysis of the transverse muscle is much more frequently associated with that of the anterior or thyro-cricoid. It is not necessary to dwell longer upon this division of our subject because the laryngeal picture is easy to comprehend.

It is much more difficult to give an intelligent symptomatology of the second division of our subject, which in reality is the entire topic of our discussion to-day. "The symptoms of recurrent and abductor paralysis of the larynx," is the text printed on our programmes.

It seems to us unfortunate that the word "recurrent" has been employed in describing the nerve supply of the larynx. Why should not the names superior and inferior laryngeal nerves be sufficient? This nomenclature would be less confusing.

It will simplify our position if we state that there are practically but two forms of laryngeal paralysis, exclusive of those which we have described in the early part of our paper, and they are, first, "abductor paralysis," and second, "recurrent laryngeal paralysis," and we should perhaps add to these cases of adductor spasm which simulate abductor paralysis. There may possibly be a few cases of solitary adductor paralysis on record, but such a high authority as Semon says not. Such a condition would show vocal bands abducted to the sides of the larynx, would produce complete aphonia, and a hoarse, low-pitched noise when the patient coughed or laughed.

Theoretically, there should be a time in cases of irritation of the recurrent nerve when a laryngeal picture of spasm of the powerful adductors is in evidence. When we consider that there are three and even four strong adductors of the bands to the one solitary abductor, we may reasonably conclude that irritation of nerve centres or trunks insufficient to cause entire loss of muscular power would naturally produce approximation rather than separation of the bands. This picture would simulate that of paralysis of the abductors. Let us for a moment consider the points of symptomatology in spasm of the adductors and paralysis of the abducting power of the bands. This is fitting because when irritative spasm has been succeeded by true

paralysis it is generally conceded that the abductor fibres are the first to give way. The anatomical reason for this has already been ably stated in the papers preceding me. We believe it can be truthfully said, then, that so far as the laryngeal image is concerned there are three stages in the course of nerve and muscular impairment. First, adductor spasm; second, abductor paralysis, and third, total inaction of all the muscles of the larynx supplied by the inferior laryngeal nerve, that is, Recurrent paralysis or laryngo-hemiplegia. The condition of spasm of any set of muscles is not a permanent one, as is that of paralysis, but occurs at periods of irregular intervals. It is possible that some of the cases of so-called laryngeal chorea associated with general chorea, vertigo and stridor are really cases of adductor spasm, which eventuate later in true paralysis. It does not belong to this paper to speak of functional hysterical spasm which may by extreme and prolonged adduction produce unconsciousness, for a brief period, until relaxation ensues. We read, too, of rare cases where patients make most unusual noises associated with forcible closure of the glottis. It is difficult to give a precise picture of the differential laryngeal images seen in adductor spasm and in abductor paralysis. Theoretically the vocal bands should be much more taut in adductor spasm. We use the term adductor spasm rather than laryngeal spasm, since irritation of the nerves, which should produce both adductor and abductor spasm, only accentuate the action of the adductors owing to their greater number and strength. It is well to bear in mind that adductor spasm of the vocal cords may produce what is called a laryngeal crisis and give such symptoms as the forceful cough seen in whooping cough. Irregular and intermittent adduction of the bands will produce irregular inspiratory dyspnoea, and many different pitches in stridulous breathing, but these symptoms are all temporary, in contradistinction to those of a permanent abductor paralysis.

If the spasm is severe it is said that the adduction may be so forcible as to produce overlapping of the vocal bands, and pain in the larynx like that of cramp. If spasm of the adductors continued for any length of time, stenosis would be so complete as to destroy life.

Theoretically what has been said regarding the symptoms of spasm of adduction may be of importance, but clinically we know little of such cases.

If due to functional causes the symptoms soon disappear, if caused by actual nerve lesion—the spasm is succeeded by abductor and recurrent paralysis. A symptom which is sometimes of value as indicating central involvement of the inhibitory centre is a persistently rapid pulse—and this with normal heart and temperature.

We must next call your attention to the symptoms of abductor or “posticus” paralysis; that of the great inspiratory muscles, the glottic openers, which yield to injury sooner than the opposing lateral muscle. It is perhaps well to remember a point, which we fear we have repeated many times, and that is that a lesion which eventually produces recurrent paralysis may first cause paralysis of abduction alone. Since central lesions are more rare than those affecting the nerve trunks, unilateral paralysis of the abductor and its consequent symptoms, are more common than those of bilateral paralysis. Not much time is needed to describe the laryngeal picture of unilateral paralysis of the abductor, nor its objective symptoms. The affected cord is seen in the median line, even in deep inspiration; phonation should not be markedly interfered with, and in unilateral cases the normal abduction of the other band should give ample breathing space.

Unilateral abductor paralysis rarely gives symptoms to attract the attention of patient or of the physician—unless the abductor paralysis has been preceded by spasm and abnormal phonatory symptoms. The affected vocal band remains in the median line during inspiration, because the three adductors are, at the commencement of the nerve injury, in their full integrity. Breathing may be slightly affected during great exertion. The voice is so little affected that, if our diagnosis was correct, we recall the case of a singer who retained a church position, never knowing that he had one vocal band which remained immovable in the median line. While the voice may be at times normal, it is apt to be uncertain, and break in pitch, because one cord may lie at a lower level than its fellow. In sopranos and tenors singing would be almost impossible. The voice would become more and more uncertain, because the tendency of an abductor paralysis is



towards a recurrent disability. It is quite possible that many cases of unilateral paralysis escape the notice of the laryngologist, because they often give no marked symptoms until a laryngo hemiplegia is present. It is for that reason that most observers believe that unilateral recurrent paralysis is more frequent than unilateral posticus paralysis. Much more has been written about the symptoms of bilateral abductor paralysis, because they are so much more noticeable and dangerous than in the unilateral lesion. Semon says that in bilateral posticus paralysis the position of the bands may pass through two stages. First, they are in the cadaveric position of recurrent paralysis, but later, if the abductors recover their tone, the paralyzed cord openers, which remain incompetent, leave the bands in the median line. When we remember that the abductor muscle is solitary, and opposed by several and by more powerful antagonistic muscles, it is easily understood why the bands remain immovable in the median line. The glottis presents the appearance of an antero-posterior slit between the bands. The cords are blown slightly apart during expiration; and if the adduction is extreme, the inspiration may be prolonged—high-pitched and stridulous. The air current may actually turn the edges of the bands downward as it passes through. In posticus paralysis, dyspnoea comes on by degrees, and may steadily increase, and is aggravated by physical exertion and mental excitement. Cases are recorded where the onset is so sudden that the patient is awakened in the night struggling for breath. In children the dyspnoea is much more distressing, because the larynx is smaller, and breathing is rendered more difficult by crying and struggling. In children there may be noticed a disproportion in length of time between the acts of inspiration and expiration. Inspiration requires much longer time than usual, because of the glottic obstruction. Expiration relatively requires less time. The voice may be nearly normal, but is apt to be breathy and uncertain. Confusion in the diagnosis of paralysis of any one group of laryngeal muscles of the larynx is rendered easy by the spasmodic contraction of any opposing group which are at the same time responding to nerve irritation. Some writers speak of the offensive breath in cases of abductor paralysis, which they believe to be due to the decomposition of laryngeal secretions; by reason of improper drainage and ventilation.



It is remarkable through how small an aperture a quiet, calm patient may breathe without showing apparent dyspnoea. We have seen cases in which the inspiratory current actually turned the vocal bands inward, as it sucked downward between the vocal cords. This in the case of bilateral abductor paralysis. We have seen recently a case of tubercular swelling of the larynx in which hardly any breathing space could be seen, and the patient complained not at all of respiratory difficulty; but this is a digression.

The symptoms of a solitary adductor or "Lateral" paralysis, as it may well be called, can theoretically be easily stated. This must necessarily be a very rare condition, because the lateral muscle possesses so much vitality that, as we have seen, the posterior is the first to succumb, and a more extensive lesion paralyzes all the muscles supplied by an inferior nerve, and a laryngo-hemiplegia, not a lateral paralysis, is the result. But if the fibers of the "lateral" are alone affected, the vocal band, if the lesion is unilateral or both bands, if it is bilateral, would be seen to be about in the cadaveric position during attempts at phonation, for the internal and transverse muscles would probably be competent to draw the bands to this position. In a state of inspiratory and phonatory rest, when the posterior muscle was not in action, the cords would be in the cadaveric position. The essential diagnostic point of time would be during deep and forced inspiration, when the unopposed abductors would draw the bands outward to the extreme sides of the larynx. If the lesion is one-sided, the affected cord makes a more outward journey than its fellow. But during rest and phonation, the three other adductors are competent to keep the disabled band as near the median line as the cadaveric position, which is the position of a recurrent paralysis. The diagnosis, therefore, must be made during forced inspiration. Theoretically, the margins of the bands would assume a straighter line in Lateral paralysis than in Recurrent, because the smaller tensors would be still exerting their power, but the voice would be weaker—there would be larger phonatory leakage because on account of the larger separation of the bands the normal cord would not be able to get so close to its fellow as it does in a one-sided Recurrent paralysis;

sometimes even coughing is impossible and the voice is completely lost. The bilateral laryngeal image of a Lateral paralysis is almost identical, therefore, with that of a bilateral Recurrent paralysis, except at the period of forced inspiration, when there is wider abduction in the former than in the latter. The speaking voice, in all varieties of paralysis, commonly improves, because of the marvellous ability of the uninjured muscles to render compensatory aid to the disabled ones. If there was time we should like to cite examples of such compensatory action, but we have already presented to this Association a paper on the subject.

Lastly, we must speak of the symptomatology of Recurrent paralysis, that is, a total paralysis of all the muscles supplied by the inferior laryngeal nerve. This is the form most commonly observed by the laryngologist, because, as has been frequently said, if the other forms, excepting bilateral posterior paralysis, precede it, they usually furnish so little evidence of their existence that the nerve lesion has produced a Recurrent paralysis by the time the patient consults a physician. The band or bands are in the cadaveric position, and respiration is little affected during rest. In one-sided paralysis the speaking voice steadily improves, as the sound vocal cord is able to go a longer distance, but the voice at first is lower pitched, uncertain, becomes tired easily, and the nervous patient dreads to use his voice because of its liability to make ludicrous breaks in pitch. In bilateral Recurrent paralysis there can be no marked dyspnoea, but the voice is lost or very breathy or uncertain, and because of non-approximation of the false cords, the pitch in coughing is lowered, and instead of terminating with a short explosion it is prolonged, sounding like a hoarse fog horn, until the phonatory leakage empties the lungs. The sound arytenoid is sometimes pulled in front of the disabled one and pushes it backward, and the weaker band sometimes lies at a lower level than the opposite one. Since aneurism or neoplasms in the neck or upper chest are most frequently the cause of unilateral Recurrent paralysis, they themselves sometimes furnish symptoms, which help materially to determine not only whether the laryngeal condition is one of paralysis rather than one of ankylosis, but such symptoms may also differentiate between paralysis and spasm—Watson Williams says a peculiar

brassy cough may be one of the earliest symptoms of thoracic aneurism.

I believe it is not in my task to speak of the differential diagnosis between the forms of paralysis and the immobility of a band, by reason of an ankylosed crico-arytenoid joint. As a rule this joint, when affected, shows inflammatory signs, but authors speak of a degenerative change here, which does not affect the gross appearance of these cartilages, but a careful history of the case should make its character plain. Most authors note the fact that in unilateral ankylosis the sound arytenoid is only drawn up to and not in front of the disabled one, and in ankylosis, too, the band is not in any known paralytic position, neither in that of abduction, adduction, nor in the cadaveric position. Theoretically, in non-paralytic conditions the bands are tense, while in paralysis they are limp.

In closing I would say that it is difficult to tabulate any statement of symptoms of the motor neuroses of the larynx, which renders the diagnosis positive, without a careful examination with the laryngeal mirror. So far as the voice is affected there is present every degree of impairment—from slight hoarseness to complete aphonia—a lesion which shows no abnormality in quiet conversation would be apparent if the patient endeavored to use the voice in a forcible way, or tried to sing. The quality of the voice may even change from day to day, because the compensatory aid given by unaffected muscles constantly varies in efficiency. In unilateral conditions, it may be stated that, as a rule, the voice shows a gradual and permanent improvement if the vocal chord muscles proper, the internal tensors on the affected side, retain their integrity; but if loss of power here allows the margin of the band to remain concave, there is little hope of vocal improvement. This is an important point in considering the prognosis of voice behavior—a careful study of the conversational pitch from day to day would show whether the impaired muscles were improving in tone. One unaffected lateral muscle can make approximation sufficiently good for ordinary conversation, provided the margin of the affected band is straight and not concave. We do not see so many concave bands even in unilateral Recurrent paralysis, because, I suppose, the anterior muscle is, as

a rule, not involved, and so the margin of the band is kept fairly straight and taut. As to dyspnoea, there are many degrees of this. It has already been said that if the loss of power develops slowly, and, consequently, if the respiratory space is but gradually narrowed, the patient accommodates himself in a remarkable manner to a larynx almost entirely closed, without showing visible distress. Other symptoms, such as vocal fatigue, laryngeal cramps and cough, depend much upon the individual case and upon conditions apart from the neurosis. There are many cases where it will require numerous and most painstaking examinations to determine what muscle or group of muscles is at fault, because, perhaps, the state of partial paralysis will be masked by a condition of jerky spasm; a weak solution of cocaine sometimes aids by quieting spasm, and by emphasizing the image of real loss of power. The symptoms of no two cases seem to be exactly similar, and it is most interesting to carefully study the behavior of each.

*Symposium—Fourth Paper.*

## DIAGNOSIS AND TREATMENT.

BY WILLIAM E. CASSELBERRY, M.D.

In the diagnosis of unilateral recurrent nerve paralysis which, when complete, results in the fixation of one vocal cord in the cadaveric position, the cracked, softly rauous voice is suggestive rather than distinctive, but it is usually the one small symptom of an inversely grave condition which first causes the patient to seek advice. It is nature's distress signal, full of meaning to him who reads the code. The same is true of that partial stridor which may or may not usher in an unilateral abductor paralysis in which, the dilator muscle alone being powerless, the vocal cord is retained in a position closing over one-half of the lumen of the larynx. Either symptom demands a prompt resort to laryngoscopic inspection, but even then, by reason of the fact that patients are liable to present themselves for the first time during "a cold" or other aggravating influence, confusing departures from the typical image may be encountered, and one turns next to the really vital phase of the diagnosis: a search of the chest, neck and central nervous system, for the cause and significance of the vocal paralysis. The tortuous course and complex composition of the recurrent nerves should be kept well in mind. Branching from the pneumogastric, the left one takes a turn under the arch of the aorta, *between it and the left bronchus*, upward to the larynx, while the one on the right passes under the subclavian artery; both being liable to compression by aneurysm, mediastinal and cervical tumors and, on the right, by disease at the apex of the lung or its pleura which is crossed by the nerve. The similarity of lettering of the two words abduction and adduction is unfortunate, for, in addition to typographical errors, a conscious mental effort is necessary to avoid confusing one with the other, but, as applied to the movements of the vocal cords, they are

more exact in meaning than any other available terms. The opposing muscles which effect the to and, fro motion of the vocal cords, adduction for vocal vibration, abduction for breathing space, are innervated by different fibres which merely travel in the same recurrent nerve trunk, either set of fibres alone to any degree or both sets together being subject to paralysis. Semon's law refers to the fact that the abductor fibres and muscles are the more vulnerable of the two, yielding first to an equal pressure on

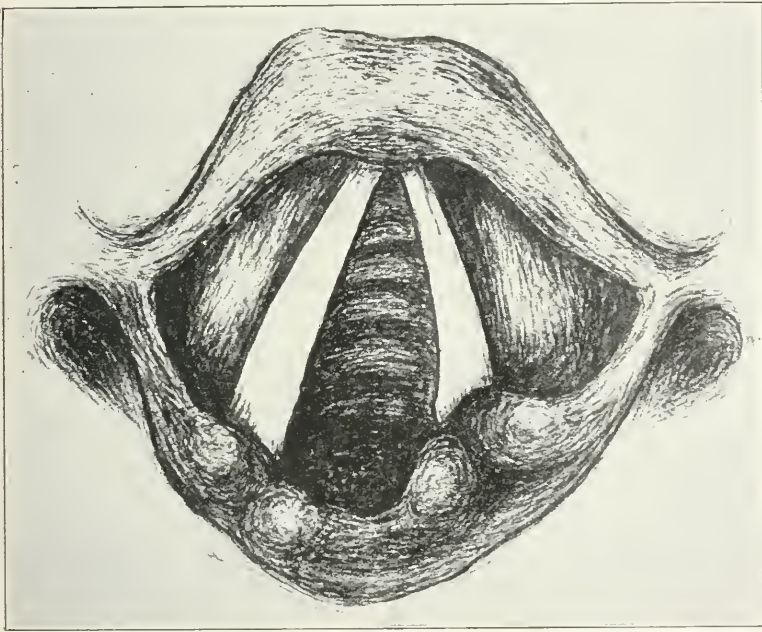


Fig. 1.

The typical image of complete recurrent paralysis. The vocal cord, in the cadaveric position, appears relaxed, narrowed, shortened and depressed; the arytenoid "fallen in," not swollen.

#### *Aneurysm Group.*

the trunk and, in consequence, every complete paralysis of a vocal cord by pressure must have passed through a preliminary stage, however brief, of abductor paralysis alone.

The typical image of complete unilateral recurrent nerve pa-



ralysis illustrated in Fig. 1 is introduced as a standard from which to describe the diagnostic variations. It represents the larynx of a middle aged salesman whose voice, he said, "Failed all at once, one day, while selling goods." Not only is the left vocal cord motionless in the cadaveric position, but it is relaxed, tends to bay outward, and its arytenoid, which is *not swollen*, has "fallen in," i. e., collapsed forward, which shortens the vocal cord and permits the cord to sink to a plane slightly below the level of its fellow. On phonation, the left arytenoid may give a twitch, but fails to straighten up or convey more than a mere tremor to the cord, while the opposite arytenoid and vocal cord, in an effort to compensate, overreach the centre line, the sound arytenoid crossing a trifle, usually in front or possibly behind, the paralytic one and the healthy cord coming nearly, but not quite, over to the paralyzed one. These are not insignificant details, for their presence collectively, rather than the cadaveric position alone of the cord, insures the fact of a complete recurrent nerve paralysis. Its cause in this instance was equally evident. There was substernal pain and a radial pulsus differens, but no other symptom, excepting that in the fluoroscope a small pulsating aneurysm of the arch of the aorta was visible. Five months later, while walking on the street, this patient fell dead with a gush of blood from the mouth—the aneurysm had burst, presumably into the left bronchus.

Life's duration in aneurysm, however, is seldom so brief. Dating from the early period of vocal paralysis, it has often surpassed my expectation; in one 10 years, in another 5, while others are now living at 2, 3 and  $4\frac{1}{2}$  years. The seemingly prolonged course, last mentioned, the patient being still active, is explained by the fact that it dates from the earliest demonstrable stage in the development of the aneurysm, as evidenced by the presence of unilateral abductor paralysis, which is the first definite effect of pressure on the recurrent laryngeal nerve. A sketch of the larynx made at that time (Fig. 2) represents the typical image of unilateral abductor paralysis and is intended to show not only the vocal cord, motionless, in the median or closed position, but also that the arytenoid has not yet "fallen in," hence the cord is but little depressed or shortened, and not very much relaxed. Paralysis

limited to abduction is not the only possible interpretation of the median position of a vocal cord, for one of the most deceptive of the variations from the typical image of *complete* recurrent paralysis is a fixation of the cord, not exactly in but quite near to the median line instead of in the usual cadaveric position. Either form of paralysis is suggestive of an aneurysm, but with paralysis of the abductor fibres alone, indicating an earlier stage and more

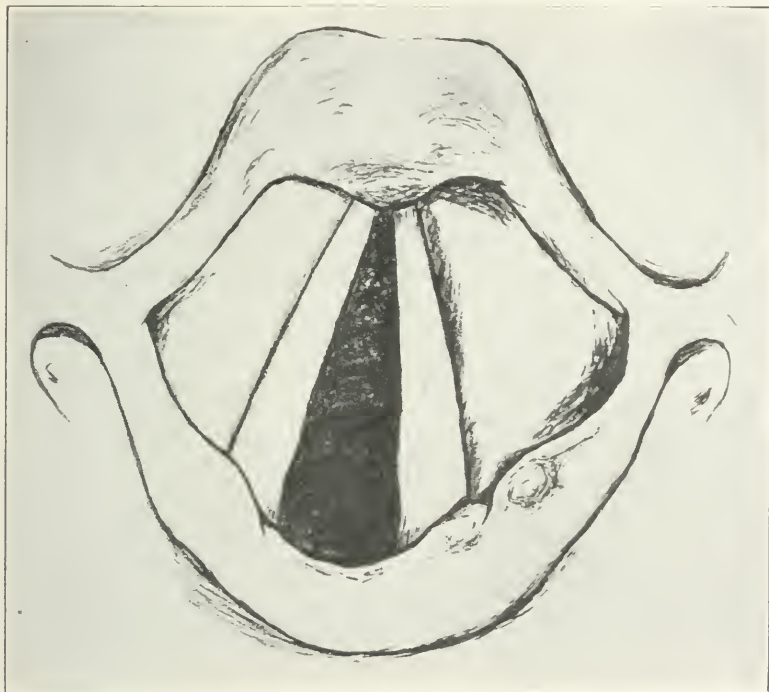


Fig. 2.

The typical image of unilateral abductor paralysis. The vocal cord is in the median or closed position. The arytenoid is only slightly "fallen in"; hence the cord is but little depressed or shortened.

limited development of the aneurysm, a longer course is to be expected and an "arrest" or even a retrogression might not be impossible. Hence, in confirmation of the memorandum and sketch made of abductor paralysis in this case I will quote the additional notation inscribed that "the vocal cord was not much relaxed" and

that the arytenoid, although standing a little forward, had not yet "fallen in," a combination of conditions which I interpret to indicate an incomplete degree of paralysis of the recurrent nerve, which, in connection with the median position of the cord, could be no other than an abductor paralysis. His X-ray print I find somewhat blurred by the effects of time; still it distinctly shows, representing the aortic arch, a broadened shadow which rises in the back above the normal limit of the fourth rib, with a bulge to the left which centres near the second rib and a smaller bulge to the right at the fourth intercostal space. A fluoroscopic examination also by Dr. John Kales and the author was recorded at that time as follows: "A distinct but small pulsating bulge in the descending part of the arch of the aorta could be plainly seen."

[Since writing the above description with its deductions relative to the four and a half years duration of an aortic aneurysm, this patient, known to have remained vigorous so long as to suggest a shade of doubt in the diagnosis, has returned, thus enabling me to complete the evidence to date with respect both to the laryngeal paralysis, of which he is conscious only when further disabled by a "cold," as at present, and to the aneurysm which does not even yet cause any subjective symptoms other than the vocal cord paralysis. Re-examination confirms all of the original findings, with natural progression. The position of the left vocal cord has advanced from the typical abductor or first position, partly, but not wholly, into that of complete recurrent paralysis. It now exemplifies that variation from the typical image of complete recurrent paralysis described as: "a fixation of the cord not exactly in, but quite near to the median line instead of in the usual cadaveric position." Intensified tubular breathing, the physical sign emphasized as coming next after vocal paralysis, is now heard in the back at the left above the angle of the scapula and, as might be expected with compression of a bronchus, the vocal resonance and fremitus are diminished at the same point. A blurred, widely transmitted first sound stands for the aneurysmal bruit and the subclavian impulse is in excess of the natural. The fluoroscope and X-ray plate show clearly defined sacculated aneurysm, with a diffused distension of the rest of the aortic arch.]

It requires but a slight expansion of the aorta at a given spot to compress the left recurrent nerve, hence the frequency of even a complete recurrent paralysis at comparatively an early period, when perchance one may search in vain for any other classical sign of the aneurism, which, in fact, can still be so small that even a radiograph will leave a doubt behind, as in Fig. 3. Here there was neither bruit, thrill, tracheal tug nor protrusion, yet



Fig. 3.

Aneurysm so small that the radiograph alone would have left it doubtful. Complete paralysis of the left vocal cord and exaggerated bronchial breathing present.

there was paralysis of the left vocal cord and there was, moreover, a noteworthy accessory physical sign, i. e., an exaggerated bronchial breathing transmitted to the back, a symptom which, coming early, *pari passu* with vocal paralysis, should be of special diagnostic value to the laryngologist, for an aneurysm which compresses this nerve must, at its very next step, compress the

left bronchus. An appreciable diminution in vesicular expansion comes only later.

It has been noted that in the typical image of complete paralysis the "fallen in" arytenoid is not swollen; but exceptionally, as a departure from the typical image, a coexisting swelling of the arytenoid is found, which is liable to disguise its paralytic aspect and simulate a crico-arytenoid ankylosis with fixation of the cord. Figure 4 represents this important variation. When first observed,

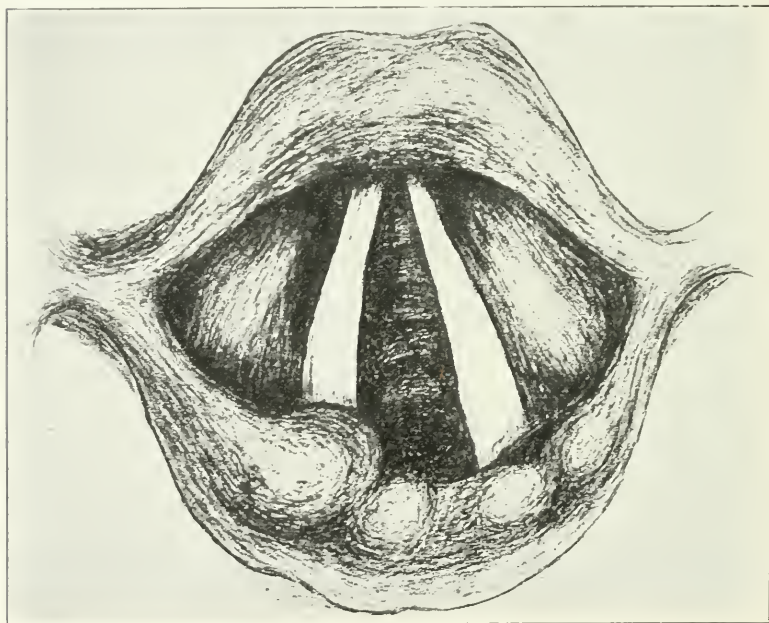


Fig. 4.

A variation from the typical image of recurrent paralysis. The arytenoid is swollen.

the right cord was stationary, but in a doubtful position, the arytenoid and subglottic tissue was swollen and the left cord, also, was sluggish in movement. It was only after some weeks of detergent local treatment that the cadaveric position of the right cord could be accurately determined, the left cord, meanwhile, having definitely weakened into the first position, that of



abductor paralysis, which, in conjunction with the other lesions, sufficed to cause alarming respiratory stridor on exertion, or on "taking cold." Aneurism was now evidenced by other signs and its existence was later verified in Berlin by Dr. Krause, who, in explanation of the swollen arytenoid, wrote: "The small tumefaction of the arytenoid is a little bit unclear, but may be caused 'by a prolapsus of the cartilage.'" Hooper (1) also once drew our attention to certain "anomalous muscular fasciculi (2) innervated from other sources which, when present, in conjunction with recurrent paralysis would serve to pull the arytenoid forcibly forward." The possibility, thus, of a partial dislocation of the arytenoid, amounting to a prolapse instead of merely a collapse, is a striking point in diagnosis to be remembered, especially with reference to the differentiation of paralysis from ankylosis, which is rendered thereby all the more difficult.

The bilateral inflammatory form of paresis of the internal tensors with its familiar ellipse, is in no danger of being mistaken for recurrent nerve paralysis, but I have twice observed in clergymen an unilateral internal and external tensor paralysis, induced by vocal strain, but of so severe a grade as to simulate and, perhaps, in a way, to partake of a recurrent nerve paralysis. Assuming for it a neuromuscular rather than a strictly myopathic basis, it is the nearest approach to a direct impairment of the nerve endings which my series affords. The level of the cord is lowered, its range of movement to and fro lessened, and the arytenoid disposed to collapse, but it is distinguishable by a predominance of the flaccid tensor element, absence of lead, alcohol or other toxæmia, absence of any cause of pressure, and it finally recovers by rest to the voice.

Of a total of forty cases upon which this study is based, the left recurrent nerve was paralyzed by an aneurism in eight and the right recurrent in six, including in both numbers those in whom the opposite cord was likewise but less affected. Concerning the treatment, in only a few of the aneurysm group was local medication indicated, and then not for the paralysis itself, but rather to aid in strengthening the voice or in diminishing the

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1. Franklin H. Hooper, *Trans. Am. Laryng. Assoc.*, 1886, page 25.

2. *Thyro-arytenoidei Superiores Muscles.*



stridor by controlling the inflammatory complications. Moderation in voice use is enjoined on all and soon has its reward in distinctly improved tones, the voice in time, through compensation, often becoming fairly good for conversational use. The prognosis of aneurysm, in general, is not so absolutely bad, and, discovered at an early stage, it should be better. Syphilitic arteritis in other situations, for example, in the brain, elicits and yields to a vigorous specific treatment; why not so in an early stage of aortic arteritis?

#### *Pleuritis Group.*

Four additional paralyzes of the right cord compose the pleuritis group. All were tuberculous and in none could any other cause be assigned than an assumed circumscribed pleuritis. In one of them, whatever might have been the exact cause of a recent and complete unilateral right recurrent paralysis, which showed a typical image, a perfect restoration of power to the cord was gradually attained contemporaneously with a treatment by fresh air and rest for the incipient tuberculosis. Our means of certifying a diagnosis in this group are, as yet, sparse. The X-ray should be of service for the purpose of excluding or establishing the presence of enlarged mediastinal lymphatic glands on the left side, which, in tuberculous subjects constitute a possible cause of pressure on the recurrent nerves; but on the right side enlarged glands so located would scarcely be beyond the reach of deep palpitation. However, a radiograph was taken of this patient, who recovered, and no abnormality was shown. Her reaction to the tuberculin test was positive.

#### *Tumor Group.*

Another departure from the typical image of complete paralysis is presented in Fig. 5, in that the ventricular band is hypertrophied and almost covers over the paralyzed cord in compensation; it vibrates with the sound cord and produces a fair voice. It is selected from my next group of eight cases, in each of which a tumor was the cause, and in all but two of which, unlike in the aneurysm group, the vocal disability appeared only as a late and gradual development. In this patient a primary malignant

growth had been removed from the chest wall three years previously, a fact which he considered so irrelevant to vocal trouble that he deliberately falsified when I asked about the scar, saying he had been struck by a shell during the war. A grimly humorous episode served, in the interest of science, to complete my record of this case for, the autopsy having been held in my absence, the widow, who, it appears, had fervently disapproved

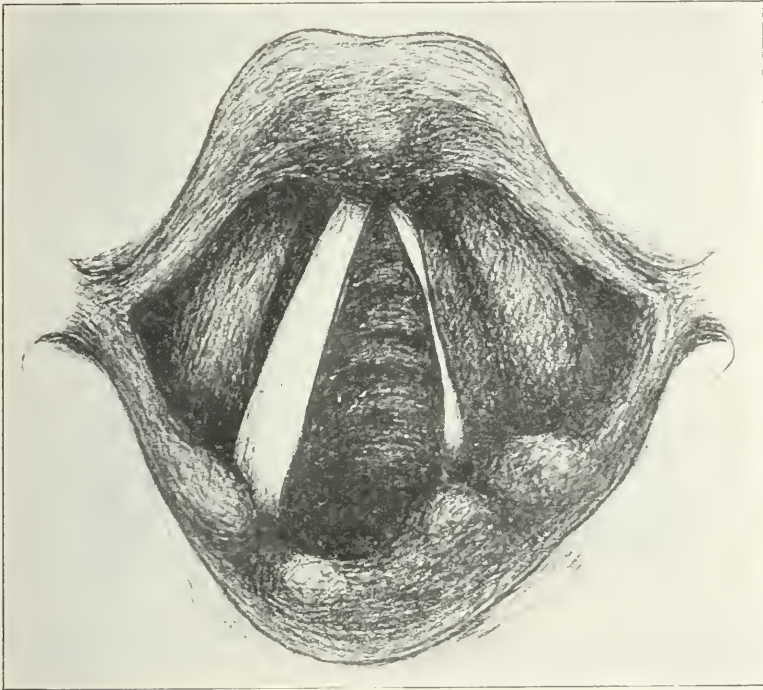


Fig. 5.

A variation from the typical image of recurrent paralysis. The ventricular band is enlarged.

of his falsehood about the scar, entered one day and dramatically deposited a large package before me on the desk, stating exultingly, "There, that's what he had." It proved to be a sarcomatous tumor from the mediastinum.

In this group, also, I find another observation:—that late in the course of a carcinoma of the oesophagus, an irritable cough and



inverted recumbent position after each meal, which, by gravity, emptied the pouch, has afforded a gratifying relief to the throat symptoms, which two years ago were quite distressing. Meanwhile, the obstruction in the oesophagus, whatever its nature, remains still in statu quo. Having the appearance of a neoplasm, it is of interest in this connection because it is differentiated from an aneurysm by the fact that it does not pulsate as observed in the fluoroscope, and because it is in line with the dictum that vocal paralysis, unlike in aneurysm, is not an early symptom of neoplasm of the oesophagus unless the latter is located high up.

We recognize in the so-called "lazy cord" a symptom of carcinoma of the larynx, but I think it is little known that total immobility of a cord in the cadaveric position may be the first visible evidence of carcinoma of the larynx. Through a surprise of this sort at autopsy I am able to duplicate the single observation of it which Semon (3) records in these words: "*Einmal habe ich eine bösartige Kehlkopfneubildung in Form der Unbeweglichkeit einer Stimmlippe in Cadaverstellung beginnen sehen.*"

The other exception to a late development in the tumor group occurred with a high carcinoma of the oesophagus and involved both cords, the left a cadaveric, the right an abductor paralysis. The end was then approaching, but aphonia two years before had been the very first symptom, the reason being that high up the recurrent nerves lie so close to the oesophagus that not only one but both nerves are liable to be affected by pressure or to be incorporated in the carcinoma almost at the start.

Goitre was responsible but once for a complete paralysis of a vocal cord. It was a small, firm enlargement of the left lobe, which was treated by surgical removal, but the nerve had already atrophied. In goitre, when the voice begins to weaken, the cords should receive the closest scrutiny, for surely the first definite evidence of paralysis would be an additional incentive to surgical interference. This principle pertains equally to other cervical tumors and to enlarged lymphatic glands. Its value was brilliantly evidenced in one of this group by a complete restoration of voice which followed the excision of three tuberculous glands in a school teacher whose left vocal cord previously had been motionless in the cadaveric position.

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3. Felix Semon—Heyman's Handbuch, Band I. S. 744.

The large variety of conditions capable of inducing recurrent nerve paralysis is emphasized by an error in diagnosis of which I became apprised only when tracing the patients for the purpose of this report. The image of complete paralysis of the left cord was typical and without tumefaction inside the throat. A swelling in the neck at the site of the thyroid gland was naturally mistaken for a goitre, the subject being a girl of 18 years. Goitre was accepted as the cause of the paralysis and its surgical removal recommended, but not done. Signs of tuberculosis at the right pulmonary apex seemed to explain her anaemic or cachectic appearance. The supposed goitre turned out to be a malignant neoplasm which terminated as usual about a year later.

### *Centric Bulbar Abductor Group.*

Like other respiratory muscles, the abductors of the vocal cords (dilators) being impelled by reflex action, have their main centres in the medulla oblongata (bulbus), so near together that naturally the symmetrical double abductor paralysis predominate in my bulbar group. The mere fact of *symmetry* in persistent abductor paralysis is indicative of a bulbar lesion. When uncomplicated the appearance in the mirror is pathognomonic. The flaccid cords which will not separate, their edges flapping up and down like a sail in the wind, are unmistakable. The question of a possible overpowering spasm of the opposing adductor muscles did not need consideration in any one of this class, although it may arise, especially in connection with the intermittent stridor of an incipient stage of abductor paralysis and also in the unilateral type. The differentiation from ankylosis may require the same degree of critical observation as recurrent nerve paralysis. A paralytic immobility is liable to lead into a sort of ankylosis or stiffness of the joints, and there is at least one form of arytenoid arthritis, previously described by me under the name of "Arthritis Deformans of the Larynx," (4) which causes a symmetrical ankylosis without much tumefaction.

An energetic course of mercurial and iodide medication is indicated whenever the centric degeneration is connected with tabes

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4. Casselberry, Trans. Amer. Laryn. Assoc.—1893, page 18.



or with any other suspicion, however remote, of syphilis, for exceptionally one finds recorded a practical recovery. Eight cases of abductor paralysis compose this bulbar group, four of them being of the persistent symmetrical double type. Unfortunately, in all of these four the varied treatment did naught but eventuate in tracheotomy which, although far from an ideal remedy, remains still the best life-saving device. For eight years one of them had been wearing a tracheal tube of the sort which is fitted with a ball and socket valve which enabled him not only to inspire with freedom but to talk with ease, as the obstruction of abductor paralysis is inspiratory only, the downward air pressure forcing the vocal cords even closer together, while the outward air pressure balloons them slightly apart. Thus, by a skilfully modulated use of the expiratory current which served momentarily to close the tubal valve, diverting the air through the chink of his glottis, he could uninterruptedly intone and articulate sentences of considerable length. Neither this patient nor another, who had been dependent for his life, during several years, upon a simple tube which, in order to effect conversation, required its stoppage by the usual conspicuous method of holding the finger at its opening in the neck, would, on account of the risk to speech involved, consider any effort to widen the glottis to the small extent which would be adequate for ordinary breathing, either by means of an excision of one cord or by the method of deliberately causing it to assume the cadaveric position by intentionally cutting the recurrent nerve. The latter method, supplemented perhaps by mobilization of the crico-arytenoid joints if found stiffened, would seem worthy of a trial considering that prolonged dependence upon the wearing of a tracheal tube is in itself a menace to life. Neither of them suffered from tabes, but both gave a history of syphilis.

Not only the parasyphilitic degenerations, but also the degeneration incidental to advanced age may happen to strike these minute centres in the medulla. It was the only apparent lesion, without other cause, in a woman of 75 years, in whom a treatment by emollients and mild vascular astringents sufficed, under careful observation, to defer, for some months, the ultimate tracheotomy. The same line of treatment in conjunction with



antisyphilitic remedies was finally successful in another one of this group, but of a different type, the condition being a partial and unsymmetrical abductor paralysis in which the stridor became serious only during periods of acute exacerbation by "a cold."

The remaining representation in the bulbar group is of that combination which anatomically is far from clear, now that the spinal accessory nerve is not considered contributory to the recurrent nerve, in that the abductor paralysis, being unilateral and devoid of symptoms, was discovered incidentally, as the complaints had reference to difficulty in swallowing and articulating, in consequence of paralysis of the palatal, pharyngeal and lingual muscles, together with atrophy, in one instance, of the trapezius and sterno-cleido-mastoid muscles. One, the victim of a generalized arteriosclerosis without syphilis, was not responsive to any form of treatment and succumbed from inanition, while another, a syphilitic, was benefited by energetic specific medication to the extent of regaining an ability to swallow liquids without a nasal reflux and to articulate with fair distinctness.

#### *Centric Cortical Group.*

Owing to the bilateral laryngeal action which ensues from a unilateral cortical stimulus, it is supposed that paralysis of one vocal cord from a lesion of the opposite side of the brain really does not occur and that some fallacy has underlain the few apparent cases which have been recorded (5). Delavan (6) reports, in convincing detail, one case, previously published as cortical, in which at autopsy there was found a conjoined bulbar disease; but probably the fallacy also lies in mistaking the "stroke" of a syphilitic endarteritis for that of true hemorrhagic apoplexy. Laryngeal paresis can, of course, ensue from organic cortical lesions which affect the brain centres of both sides or interrupt the nerve fibres in transit from both sides, as in syphilitic degenerations or arteriosclerosis; and the six cases which compose my centric cortical group may have been either of this nature or else similar to Delavan's case. In three of them the vocal paralysis occurred

5. Lewin—Berliner, *Klin. Woch.*, 1874, page 10—Cited by Semon.

6. Delavan—*Trans. Am. Laryng. Assoc.*, 1884, pg. 82, and 1888, pg. 195.

in connection with attacks of so-called apoplexy, but as they were men under 50 years of age, with venereal histories, did not lose consciousness at the time of the "stroke" and had recovered in some measure, the attacks doubtless were due, not to unilateral hemorrhage, but to syphilitic cerebral arterio-thrombosis, which is often unequally bilateral. In none was a typical image of paralysis of any particular group of laryngeal muscles presented although the left cord in one was nearly, but not quite, motionless in the cadaveric position, the condition in two being an incomplete bilateral paresis which predominated in the larynx on the side of the paretic arm and leg, as evidenced by a feeble closure of the flaccid cords in a slanting position. Either an enfeeblement of the pharyngeal muscles, or impairment of articulation, or mild aphasia, was associated with the laryngeal paresis in one or another of this subgroup. One of them has remained under observation, receiving antisyphilitic treatment during a period of seven years, and is now well.

The others of this group were persons somewhat advanced in years who presented symptoms, in general, of cerebral degeneration, the paresis of the vocal cords being the cause only in part of the difficulty in speaking, although hoarseness and weakness of the voice were particular complaints. The laryngeal image was atypical, and represented an unequal paresis of both cords rather than a complete paralysis of either cord. The local treatment was mildly palliative, only, but proved far from useless. The detergents and emollients applied by simple methods served to lessen the friction of vocalization and thus represented to the aged sufferers the difference between misery and contentment.

#### *Discussion on Symposium.*

Dr. E. Fletcher Ingals: There is one difficulty to which I would like to call attention in making diagnosis of aneurysm by means of the X-ray. In two cases I have seen a shadow shaped like half a circle about  $1\frac{1}{2}$  inches in diameter protruding about half an inch to the left from the ordinary shadow of the aorta, about the level of the third dorsal vertebra. In one, a man who had past sixty years of age, there had been pain under the sternum and symptoms of angina pectoris. I thought that I had discovered the cause of his angina in this little tumor.

which I took to be a circumscribed aneurysm of the aorta. I do not recollect the history of the other case, but the shadow was the same. The first case died within thirty-six hours after I saw him, but the autopsy revealed no aneurysm whatever. In the other case I finally came to the conclusion that there was no aneurysm present. I am inclined to think the shadow may have been caused by the transverse portion of the arch of the aorta. I would like to know whether others have seen similar shadows.

Dr. G. Hudson-Makuen: An interesting point in connection with this discussion is to determine the exact location of the lesion causing the paralysis. Three possible locations have been suggested, namely, the peripheral organs, the medulla oblongata and the cerebral cortex. All the speakers showed very clearly that the seat of the lesion might be in the peripheral organs and that this is a common situation, but a positive diagnosis of the lesion in either of the other locations is not so easy. Dr. Casselberry referred to eight cases of supposedly bulbar origin, but he did not seem to make quite clear the reasons for this conclusion. The paralysis was bilateral and therefore probably of bulbar origin. As to the third possible location, namely, the cortical region, I think that there is even more uncertainty. I do not recall having seen a case of abductor or adductor paralysis of the cords that was of undoubted cortical origin. The fact that a mere stimulation of a cortical center will cause to-and-fro movements of the vocal cords does not prove to my mind that a lesion in this location will necessarily cause paralysis of the cords. It merely means that such stimulation may incite to action the bulbar motor centers and thus cause the to-and-fro movements. I have examined the larynges of upwards of 2,000 patients with gross defects of speech and functional cortical disturbances of almost every variety and in only one case do I recall actual paralysis of the vocal cords, and it was abductor and unilateral paralysis.

Dr. B. Alexander Randall: As to cortical lesion as mentioned in connection with the abductor paralysis of the larynx I would call attention to an excellent case published last year by Dr. Mills and Dr. John L. Atlee, of Lancaster, in the *Jr. Amer. Med. Assn.*, Dec. 28, 1907. The patient, a physician, had been under my observation with total paralysis of the cord as the result of a cortical tumor, the removal of which was successfully accomplished, with immediate, although as yet only slight return of power in that cord. A more perfect demonstration of a cortical lesion as the cause of this disorder could hardly be seen, although mediastinal pressure was not wholly

excluded. This case was reported last year before the American Medical Association.

Dr. H. L. Swain: It seems to me that the emergencies of our life very often bring to a test our ingenuity as well as our diagnostic ability in connection with such cases as paralysis of crico-arytenoideus posticus muscles. Three times I have been suddenly confronted with the problem of why a person could not breathe and yet could talk perfectly clearly and who had inspiratory dyspnoea due to paralysis of the abductor muscles. In all of the three cases it was a pure muscular defect; in all of them it turned out to be that there was some peripheral cause. In two cases it was a syphilitic immobilising of the crico-arytenoid joint on both sides in the median position and in one which eventually came to autopsy it was due to a complete necrosis of the cricoid cartilage. I noticed in a specimen in the beautiful pathological exhibit in the adjoining room an exact duplicate of my own case, where the cricoid cartilage was found lying loose in an abscess of the larynx. This was in a case of typhoid fever running an ordinary course in the hospital. I received an urgent summons to attend him and found him talking perfectly freely, but unable to breathe. Under examination I found the cords in the median position. Immediate tracheotomy was performed. He lived to recover entirely from his typhoid and was about ready to leave the hospital, though unable to exist without the tracheotomy tube, when one night he suddenly expired with a gush of blood, and the autopsy proved it to be a rupture of an abscess cavity into the trachea and he died of suffocation. The two cases of syphilitic trouble responded very nicely to the intermittent use of the intubation tube, together with anti-syphilitic remedies. The large tube was kept in the larynx in one case for three days, then a shorter interval, then left out for a whole day, then two days, etc. He got so that he could run to catch a car without the tube and without inconvenience. He wore the tube always at night, however, and this allowed him to go about all day without it and with perfect comfort.

Dr. John O. Roe: A few years ago an exceedingly interesting case of paralysis of the abductors of the larynx, due to fusiform enlargement of the arch of the aorta, came under my observation. The case from the beginning of the trouble was an exceedingly puzzling one, and I had a number of clinicians examine the man to discover, if possible, the cause of the trouble, but without success until I took him to Dr. Alfred L. Loomis, of New York, who made the diagnosis of fusiform enlargement of the arch of the aorta, which the autopsy, about two months later, confirmed. The man had great dyspnoea, both from pressure on the trachea and on the recurrent laryngeal

nerve, affecting the abductors. The dyspnoea was so great that he was only kept alive during the last three weeks before he died by the use of oxygen.

It is interesting to note in connection with this case that the beginning of the trouble dated back to a strain from heavy lifting. As he was one day assisting in moving his wife's piano he felt something in his chest give way. This was followed for a time by a slight discomfort in his chest, which later on was followed by the dyspnoea from which he succumbed.

I have seen a number of cases of incomplete paralysis of the left side of the larynx due to tuberculous deposit in the apex of the lung, involving the recurrent laryngeal nerve as it passes through the upper part of that side of the chest. In fact, I have an interesting case of this kind at the present time. On account of the tumefaction and some ulceration of the left side of the larynx attended with marked hoarseness, a diagnosis of cancer of the larynx was made in this case by two laryngologists. When he was brought in to me for an opinion the conditions were too masked for a positive diagnosis without further study of the case. I believed the disease, however, to be tuberculous, which opinion was confirmed by subsequent developments.

Dr. C. P. Grayson: It may be of interest in connection with the etiology of the peripheral class of laryngeal palsies to speak of a case of double abductor paralysis brought quite recently to the Hospital of the University of Pennsylvania—an Englishman, aged sixty-eight, who, with no apparent disturbance of his general health, was suddenly seized in November last with dyspnoea. It was not very severe at first, but continued in a somewhat intermittent manner until March when, within a few days, it developed an alarming increase. When he was admitted to the hospital, early in April, his dyspnoea was so extreme in degree that he was able to take but a few steps without stopping to regain his breath. Previous to operation his chest was carefully examined by Dr. Fussell with absolutely negative results, and a skiagraph made shortly after the tracheotomy was equally negative in its findings. Later, also, Dr. Spiller conducted a rigid investigation to detect if possible a central nervous cause, but with no better success. It was not until a week or two subsequent to the operation that persistent questioning of a member of his family discovered the fact that the patient during the three or four months preceding the onset of the dyspnoea had had three attacks of suppurative peritonsillitis. In the absence, therefore, of other discoverable causes we were forced to the conclusion that this case of double abductor paralysis was due to a peripheral neuritis occasioned by the series of peritonsillar



abscesses. Whether it should be termed infective or toxic would depend upon one's conception of the relative activity of the pathogenetic factors involved.

Dr. C. F. Theisen: X-ray examinations are sometimes misleading in reaching a conclusion as to the cause for the compression of the recurrent. I recall one case in which there was a complete left-sided abductor paralysis and this man dropped dead on the street; left recurrent laryngeal compression was diagnosed during life though from an X-ray examination made in another city, the diagnosis being returned as aneurysm of the arch of the aorta. On making a fluoroscopic examination a mass which appeared to pulsate was seen back of the aorta. A very careful autopsy showed that not only the left recurrent nerve was involved in a mass of retrosternal glands, but the pneumogastric as well, and sudden compression of this last probably caused the sudden death. There was no aneurysm.

Dr. J. W. Gleitsmann: In closing this discussion I would wish to point out one or two facts of interest which presented themselves to me when writing my paper. First, that it is highly desirable that more definite expression in publications, as well as in verbal communications, be used when referring to laryngeal paralysis. In examining the literature when preparing my paper the paralysis described by the author could in many instances, only be inferred by carefully examining the whole context of the article and weighing the evidence by the history of the case; in more than one case such an analysis alone enabled me to find out whether an author meant by laryngeal paralysis an adductor or a recurrent paralysis, two widely different affections.

If I understood correctly, one of the speakers in the discussion stated as his opinion that there was no cortical paralysis. I would say that bilateral adductor paralysis in hysteria, emotion, sexual conditions, etc., is of the cortical type. In the remarks made in my paper I merely emphasized that abductor or recurrent paralysis, especially of the unilateral type, based on cortical lesions, was not proven by the cases published.

I agree entirely with the speaker who stated that the presence of a recurrent or abductor paralysis did in many cases not assist us in the diagnosis of the central lesion producing it. The difficulty in finding the true cause is unfortunately more frequent than we would desire and the best clinicians and laryngologists encounter and report cases which baffle their skill and experience to discover an etiological factor.

With regard to recovery from the paralysis; disappearance of the paralysis is an extremely rare occurrence, though it has been reported in bilateral abductor paralysis due to syphilis, as



in the cases of Mackenzie and Lefferts, referred to in the text. Of recovery from bilateral recurrent paralysis only one case is known. Here the paralysis had been caused by the pressure of a large pericardial exudate on both recurrent nerves, and disappeared with its absorption. The paralysis being of peripheral origin I shall not enter into an analysis of the case except that this was a case of tuberculosis with pericarditis, and with rest in hospital the pericardial exudate, which had been pressing against both recurrents, became absorbed with relief to the pressure and consequently the recurrents regained their tone.

I was very much interested in the conclusion of Dr. Delavan's paper with regard to toxic influence on abductor and recurrent paralysis, and I am in perfect harmony with the doctor when he says that this action of drugs as well as of infectious diseases, is a subject requiring further elucidation and offering a most fruitful field for original research.

Dr. D. Bryson Delavan: Few seem to realize the variety of causes which may produce paralysis of the recurrent by pressure, and so mistakes in diagnosis are often made which are discreditable. For instance, aneurysm is so well known and recognized as a cause, and other causes are so little talked about that in many cases a left abductor paralysis, when recognized, is attributed to the presence of aneurysm. With the introduction of the X-ray we have been given most valuable aid, and even if at times the skiagraphic picture may be misleading, nevertheless it is of exceedingly great value and no obscure case should be dismissed without recourse being had to it. The indistinct knowledge on the part of the practitioner as to just what this condition means must be taken into account. In the histories of cases reported we are constantly reminded of the fact that the writer has not been very clear in his mind as to the exact nature of the case.

The fact is we have of late been tempted to wander away from this field; nevertheless the larynx still exists and as long as it exists it is going to need our care and we cannot neglect its study. It is interesting to compare the literature of the day on the subject of this paper with that of fifteen or twenty years ago. Then the journals were filled with interesting material relating to paralysis of the larynx, recurrent paralysis in particular. It would be well if more attention were paid to this subject, since the larynx is still a very interesting organ.

Dr. W. E. Casselberry: With reference to X-ray examination, the doubts expressed are valid and I have found it difficult to differentiate aneurysm by the plate alone. The prints which I have shown exemplify this doubt, one an aneurysm, the other a tumor. (Figs. 2 and 6.) Fluoroscopic examination

should invariably follow the X-ray plate exposure, as the expansile pulsation of the aneurysm in connection with a distinctive shadow renders the diagnosis as nearly certain as possible. Add paralysis of a vocal cord, when an early symptom, and the margin for error becomes still narrower. The pulsation of a normal aorta or its throb transmitted to a contiguous neoplasm has not the same centrifugal character as the expansion of an aneurysm. Dr. Roe's case of enlargement or dilatation of the aorta might not have been distinguishable in this way from an ordinary aneurysm, but for all practical effects it was an aneurysm. The shadow drawn by Dr. Ingals in outline, if as narrow as he depicts it, I could not account for satisfactorily, but if somewhat wider and of more obtuse curvature it could represent the bulge of the normal aorta on the left side, which may appear a little more pronounced in one person than in another. The method of substantiation of a diagnosis of bulbar paralysis is another very pertinent inquiry. We can judge by having knowledge of similar cases which have come ultimately to post mortem and in which degenerated areas were found in the medulla oblongata. Again, the mere fact of symmetry in bilateral abductor paralysis in the absence of a demonstrable cause of pressure is presumptive evidence of a bulbar lesion. Symmetry indicates an equal degree of paralysis of both nerves, and to be of peripheral origin, the aneurysm or tumor would necessarily be gross and readily found. In one of my cases of aneurysm there was double paralysis, but of unequal degree, a cadaveric position on one side and an abductor median position on the other. One can scarcely conceive of an absolutely symmetrical abductor paralysis arising from peripheral pressure unless the lesion is located so high up as to compress in an equal degree the trunks of the nerves, therefore, located not far from their exit from the medulla. An absence of any indication of traumatism at the base of the skull or of thickening of the meninges would tend to exclude even this possibility.

I am opposed to intubation in cases of abductor paralysis, because it is more hazardous to rely permanently upon an intubation than a tracheotomy tube. It was suitable in Dr. Delavan's case as the patient was not in extremis. If the tube is accidentally expelled, ordinarily, suffocation would be likely to occur before the operator could be summoned to re-introduce it. In the other case cited as having been cured of the symptoms of abductor paralysis by intubation, the progress of the case suggests to me that it might have been one of partial ankylosis instead of paralysis. However, the point is well taken if it is meant that by forcibly keeping the paralyzed cords apart for a long period by means of an intubation tube they would ulti-

mately become shaped or fixed to maintain the space between them, permitting of a permanent withdrawal of the tube. If this effect can be reasonably expected, the extra hazard of intubation is justifiable.

CYSTS, ABSCESSES AND OEDEMA OF THE  
EPIGLOTTIS.

BY HENRY L. SWAIN, M.D.

Swelling of the epiglottis occurring suddenly and independently of any of the more chronic and systemic conditions, such as syphilis and tuberculosis, is apparently due to a number of causes, and three peculiar instances having come to my notice with this condition at about the same time, I was led to give the matter enough thought to look over my own records and to find out the opinions of others as to the origin and causation of such cases. The results of this consideration I venture to present to you under the above title.

Two patients with swelling of the epiglottis came to me in which there was a history of previous attacks similar to the one in which I first saw the patient, and each of the attacks was apparently not connected with any previous inflammation of the tonsils or other parts of the throat.

In 1895 I was consulted by Mr. L. C., forty-six years old. He had had difficulty in swallowing for at least two years. At all times there was a sense of fullness in the throat. Frequently the difficulty in swallowing solids was very great. Rarely until the present time had he had any actual pain. Has in the past suffered from frequent attacks of tonsillitis, which have presented no other difficulties than is usually the case with tonsillar inflammations.

Examination showed him to have simple enlargement of both faucial tonsils. In the lower part of the throat the laryngoscope discovered a large swelling, a little to the right of the median line, on the lingual surface of the epiglottis, pressing it so far backward toward the posterior pharyngeal wall as to completely conceal any view of the interior of the larynx. The surface of the swelling was red, and it was evident from appearances that we had to do with a cyst, which was in a state of inflammation. Under simple local anesthesia a large slit was made in the wall of the cyst and a very thick, gelatinous, greenish-colored fluid

was evacuated. The sac seemed to be fairly thick-walled, but on seeing the patient five days later the cyst had so contracted that it was impossible to get into it again. My idea had been to cauterize the lining membrane of the sac.

Nothing more was heard of the patient until January, 1896, some three months later. The sac had filled up again, to a small extent. Puncture was made and a bead of nitrate of silver on a probe was pushed through the opening into the interior of the sac.

In October, 1898, the patient again presented himself, with a large swelling in the same location, this time weighing down the epiglottis to such an extent that there was considerable difficulty in breathing, and severe choking when liquids were swallowed in large amount. Considerable more pain and constitutional symptoms were also present at this time than in the previous attacks. A liberal slit was made rapidly into the superior wall of the sac, and a large amount of mucus, with more pus in it than before, was evacuated. The collapse of the sac was so complete, owing to the gagging of the patient, that it was impossible for me to get into the opening again with a probe in order to cauterize the interior. Examination a month later showed no evidence of any cyst wall remaining.

The patient reports at the present writing that he has had no trouble from a recurrence of any inflammation in the sac, and he attributes his immunity for these years to the fact that he had his tonsils cauterized. As none of his attacks of enlargement of the cyst have followed tonsillitis, directly or indirectly, I do not take seriously his suggestion as regards the reason of his immunity.

The second case of abscess formation in the epiglottis, of the recurrent type and of cystic origin, occurred in a young man, about twenty-six years of age, who was first seen by me in 1894. He was then recovering from what he now supposes to have been abscess in the region of his epiglottis. His history at the time of his first appearance was that he had "an awfully sore throat," which he considered to be quinsy, that it had broken in the night, and, that as he had had two previous attacks of it, one in 1890 and one in 1892, he wished me to ad-

wise him as to some method of relieving him of such recurrences in the future. He was then a student in the university, and I saw him a number of times, but as his tonsils were not very large, I did nothing for him further than the ordinary treatment for the catarrhal conditions which existed in his nose and throat. At that time the existence of a cyst in his epiglottic region was unknown to us. He had an interval of immunity of about eight years, during which time he had so much cough and bronchial trouble that he suspected himself about to be the victim of tuberculosis, and passed his time in a warmer climate, being principally engaged, in one way or another, in business in the Southern States.

In 1904 he came to me with exactly similar symptoms and conditions as depicted in case 1. A large cyst or abscess cavity was found at the base of the epiglottis, a little to the left of the centre, which drove the epiglottis downwards and backwards over the entrance to the larynx. He was in the greatest possible distress when he came to me, having been in his present condition for three or four days, and having sought relief at the hands of various physicians, all of whom, however, had failed to use the laryngoscope. Immediate relief was given by puncture and evacuation of the cyst, and the same glairy, thick, yellow mucus in large mass mixed with small flocculi of pus was evacuated as in case 1. There was so much blood and discharge that it was impossible to do anything toward further opening of the sac or cauterizing its interior that night. The patient failed to come back until the third day, when the epiglottis had so righted itself and the parts had so shrunk down that it was impossible to get a probe into the opening as one would have expected to do had there existed a large cystic cavity.

His description of his feelings and symptoms in the three previous attacks left but little room to doubt that he had had a similar state of affairs on those occasions, with spontaneous rupture of the sac.

A three-year interval elapsed, and in August of last year he again had a sudden attack of violent pain and swelling and choking, following the use of his voice in a lecture. He had had absolutely no previous inflammation in his throat or any



cold. He was then in a distant part of the country, and the sac was opened by a skilled hand, and an attempt was made to curette the interior of the sac, which resulted in an enormous amount of swelling and discomfort, and the thickening went down very slowly, not having entirely subsided a month thereafter.

He had another attack in December last, coming on in exactly the same way as before, and it was again opened, this time, however, by simple incision. The sac collapsed completely, and since that time it is impossible to see any evidence other than that of the scar, which is situated at the base of the epiglottis, entirely off from the base of the tongue.\*

He reports that his mother and two sisters have had abscesses in their throats, which on two different occasions have been diagnosed as being somewhere at the root of the tongue. None of these patients was ever examined with the laryngoscope.

These two cases, it seems to me, are to be diagnosed carefully from those of abscess of the lingual tonsil, which cases of lingual quinsy are like the faucial type in that they are more or less peritonsillar and do not as a rule involve the epiglottis at all, except as the latter becomes oedematous. Of the abscesses in this region which I have seen, and which number only five, three were on the left side of the base of the tongue, just at the entrance of the pyriform sinus. At least that is the region in which the greatest swelling occurred and where I made the incision to evacuate the contents. This means, of course, just beyond the lymphoid tissue of that region. The other two abscesses were nearly central, just to the right of the ligamentum glosso-epiglotticum at the root of the tongue. These five cases were all after well developed acute, follicular inflammation of the lingual tonsil. The two cystic swellings first mentioned were so situated as to be certainly clearly of epiglottic origin. The fact of the recurrences and the character of the contents in both instances, not being simply pus, but thick, glairy, greenish

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\*Since writing the above the patient has been through another attack and a strenuous endeavor was made to make a radical clearing out of the sac, but with no better promise of success than on previous occasions.

mucus, have led me to the conclusion that they must be cysts of the branchial cleft type. I am more impressed that this is the explanation because of the character of the matter contained in the cysts, which was exactly similar to that which I once saw in a recurring one which opened externally in the neck in the thyro-hyoid space.

This was a most interesting case of this type, and was where we could study it more carefully than when occurring within the throat. It was in a girl of about twenty years. When I first saw her it had been opened frequently, evacuated and washed out with various solutions, all to no effect, for in a shorter or longer time, the secreting mucous membrane not being destroyed, it would again fill up. When I opened and carefully probed the cavity, it went down to within so short a distance of the interior of the larynx that the separating membrane was apparently as thin as thin note paper. That this was the case was definitely demonstrated when a surgeon, rather against the advice of the laryngologist, attempted to dissect the sac out under general anesthesia. Notwithstanding that the membrane was well defined, it was impossible to loosen and remove it over a circular area of about 3-16 of an inch in diameter because of the fear of producing a fistula by opening into the interior of the larynx at the base of the epiglottis. The unremoved area of mucous membrane was lightly touched with a pure stick of silver nitrate, and the edges of the wound sewed up. For some years there was no return and the case has passed entirely from my knowledge.

As is well known to all, in the depths of the branchial clefts of the embryo, epiblast and hypoblast, as seen in serial section under the microscope, are separated by the merest network of a single layer of intervening fibres. Whether the inclusion cyst comes on the one side of this partition or on the other is probably purely accidental, but when, after the habit of these cysts, after years of quiescence, something starts them to secretion, in the one instance the swelling will be external and in the other internal, at the base of the epiglottis. The clinical phenomena are the same in both cases. Dermoid cysts and supernumerary thyroid glands are also recorded as occurring in the region

which we have under consideration, so that our cases of branchial cysts fit in with the universal experience. How small these cysts may have been in the quiescent period one does not know. Branchial cleft cysts often grow to very much greater size, even as large as an orange being reported. When inflammation starts, as in the two cases which I report, the sac becomes as large as a small English walnut. It should be remembered that on several occasions in both cases the attack came on suddenly and a large amount of pus was evacuated, making it appear that the infection had taken place in an already large cystic cavity. In these instances there seemed to have been no previous inflammation in the throat, at least none of which the patient was conscious until the feeling of discomfort resulting from the swelling of the cyst and the distortion of the epiglottis came to pass.

This explanation for these cases seems to be fairly clear, and the indications for treatment are evident and plain, the only trouble being the difficulty of destroying the lining membrane of the sac at the time of the incision.

During the last year three patients with simple, what might be called "idiopathic," oedema of the epiglottis came to my attention, which also were not connected with previous inflammatory action in the tonsils or throat. Two ended with distinct abscess of the epiglottis, and the third, between a morning and an evening visit was so much improved, during a nap which the patient had taken in the middle of the afternoon, as to lead one to the only probable supposition that perhaps here also a small abscess had broken. The histories are, very briefly, as follows:

A student of the university came in to me one evening complaining of a swollen and tight feeling in the lower part of his throat, with difficulty in swallowing. It had come on during the last two days, being very much worse to-day. He had had no previous sore throat or cold. Examination of his throat showed the left side of his pharynx somewhat congested, no active tonsillitis in evidence, but an enormously swollen epiglottis, with a greater prominence of the right half, on its lingual side. As there was no marked swelling of the aryepiglottic fold

or the arytenoid cartilages, it did not seem to me to be urgent and I made no punctures at that time, but simply sprayed the throat and ordered a steam atomizer, with Seiler tablet solution, and adrenalin every two hours through the night. He came next day with a distinct white point on the more swollen right side of his epiglottis, which on puncture developed a cavity as large as an orange seed. This evacuated itself, with complete collapse of the walls. The swelling promptly went down in two or three days, and he made an uninterrupted recovery.

This case was scarcely off my hands when I was called to an elderly woman who, for a chronic ear trouble I have known professionally for a good many years. I had seen her throat at various times, and had been particularly conversant with the base of the tongue, for she had had enlargement of the lingual tonsil, which frequently produced cough. She sent for me one day with the statement that she had a sore throat in a different position than ever before, and that it was exceedingly distressing to her, and that she had a queer suffocating feeling that was very disturbing. Examination showed an epiglottis very similar to the one I had seen a few days before, with the exception that there was more swelling on the left side than on the right, and that the ary-epiglottic fold around to the arytenoid was very considerably swollen. Here the indications were certainly for more active treatment, and paying but little attention to the epiglottis, I punctured the ary-epiglottic fold, and was gratified that in a few hours the swelling went down in response to the puncture and the steam atomizer, used as in the other case. The next day a white point was seen upon the most swollen portion of the epiglottis, and on the following day it was punctured, with the evacuation of a considerable amount of pus, with a quick subsidence of the symptoms. In this patient I was able to put a pledget of cotton dipped in strong carbolic acid directly into the cavity itself. There had been to my certain knowledge in this patient, as I had seen her within a few days of the time she called me to the house, no active inflammation in the throat such as one would usually expect to find in an oedema case.

At a time not very distant from these two instances, I was called suddenly in the night to a patient who, being a physician,

diagnosed his own case. He said he believed he was having oedema of the glottis. He had been having a leptothrix of both tonsils, and had had considerable irritation in this region from the foreign organism. During the twenty-four hours previous to his calling me his throat had been a little sorer than usual and a different sort of soreness than he had been having during any of the time. I found a well swollen epiglottis, with the ary-epiglottic fold on the left side also markedly swollen back on the arytenoid itself. The swelling was of the very baggy type on the epiglottis, but not so deeper down, so that I felt no alarm that the patient would immediately be troubled for breath, but, nevertheless, I made multipuncture and started the steam atomizer. The patient was very uncomfortable and had a good deal of pain and trouble during that entire day. The next morning the swelling had gone over decidedly on to the other ary-epiglottic fold. No evidence of any pointing as of abscess in the epiglottis was seen, although it remained distinctly swollen. From the morning of the second day the patient began to take moderate doses of salicylic acid and oil of wintergreen. He had a nap in the afternoon, from which he awoke very much relieved, and to my great surprise, when I examined him, the swelling had disappeared by more than half in all the parts that were affected, and I have felt fairly positive that there must have been some small abscess which had ruptured, which had been so situated that I was unable to see it.

The explanation of the causation of the two first cases in this group was rather difficult. The third and last may be said to be due to the leptothrix of the tonsils or to gout, owing to the sudden improvement after taking the salicylates. The simplest explanation to give of the phenomena in the first two cases is that we had perichondritis with oedema and the formation of a focus of pus. In both the pus formation was spontaneous and not the result of any infected wound of the particular region where the abscess occurred, for no punctures were made in that area.

Concerning the probability of such a perichondritis, I was interested to run through such literature as I happened to have at hand, with the following results: D. Braden Kyle speaks of

an acute epiglottiditis as occurring, and that it might sometimes be of a malarial or miasmatic origin. This last was referred to by several other authors, among them being Shurly, of Detroit, and at considerable length by Arnold, of San Francisco, in Burnett's older system. Newcomb, in the text book, "Burnett, Ingals and Newcomb," speaks of abscesses probably occurring but very rarely. Casselberry, in Buck's Handbook, appears to have seen just such cases as those described in this last group, mentioning very similar conditions. When the abscess occurs on the lingual side of the epiglottis, he speaks of the inflamed condition as causing much dyspnoea, but no great amount of dysphagia. Morell Mackenzie, who saw about everything that was ever observed, mentions abscess of the epiglottis as sometimes occurring.

Some of these authors speak of the matter under the general head of perichondritis of the cartilages of the larynx; others under the head of affections of the epiglottis. Lenox Browne said he had never seen a case of pure and simple perichondritis of the epiglottis, and mentions the fact that the fibro-elastic cartilage, with no very well developed perichondrium, is not such as would be conducive to the production of perichondritis.

Unless we arbitrarily presuppose a quiescent cyst to be present in these two cases of pure abscess formation, and that the condition resulted in oedema and pus formation, we are obliged to come back to one or two suppositions as regards their formation—either a perichondritis or an epiglottiditis. Neither of these two patients had ever had an attack of sore throat which could possibly be construed as being the same as the one described, and the existence of old branchial cysts cannot be rung in.

Incidentally a small retention cyst from the stopping of some mucus gland could be the origin of the trouble similarly as we so often see it in an anatomically similarly constructed part of the body, viz., the eyelid when a styne forms. In styne formation we often have a simple Meibomian cyst existing for a long time previous to infection, and then we have what is usually called the styne, with the formation of pus following the infection. The analogy is very good here, and I have never



seen a case of chondritis of the lid except from infection after injury or infection of a cyst. It would only remain, then, to know how often these simple retention cysts of small diameter occur to make the picture complete. These, however, have never been reported. I have looked rather diligently for them since the idea occurred to me, and I have not found them as yet, but these oedema cases with abscess are also very rare, these three being the only ones I have ever seen in twenty-three years' experience. So that we may reasonably assume that one would not often find the stopped-up mucus glands which we are supposing to exist as a preliminary to the formation of abscesses. Furthermore, racemose mucous glands are not numerous on the lingual surface of the epiglottis. Not giving credence to this explanation, we are driven back to the condition of idiopathic inflammation of the epiglottis, a very difficult thing to understand in these patients where no previous inflammation has existed, and yet it is the one adopted by numerous writers. Balenger, in his recent book, gives a separate heading, "Acute Infectious Epiglottitis," apparently taking the term from Theisen, whose admirable paper had escaped me. Both these observers are confident that such cases as I have here detailed belong under this heading.

These three instances differ very markedly in my observation from certain others where the oedema, for example, follows a simple tonsillitis or other inflammatory action. Difficulty in swallowing is the first symptom. Take, for example, a certain student whom I once saw, where, following the mumps, he had had a sore throat with very marked swelling and oedema of the left arytenoid and aryepiglottic fold nearly to the epiglottis proper. The oedema was of the baggy type, and fell over into the larynx on inspiration, so that it entirely covered over the false and true vocal cords. Multiple puncture and the steam atomizer again proved of the utmost relief and speedily reduced the swelling on this side, but the next day it appeared on the other, and remained there with the greatest persistence and in very threatening amount for two whole days, when it yielded to treatment and promptly recovered.

I have had a similar experience in perhaps ten other cases

where the oedema followed previous inflammation in other parts of the throat and was not associated with any real perichondritis of the adjoining cartilages or inflammation of the crico-arytenoid joint. In every case it did not affect, as in the instance related, the opposite side, but usually did, although only once were both sides simultaneously attacked. In these cases we have apparently a simple inflammatory affection of the mucous membrane with oedema, exactly as we see it sometimes in the uvula and soft palate, with no false membrane and no other manifestations.

At present writing there seems no good explanation for this phenomena, or for the spreading and more or less self-limitation of the inflammation. I have thought that in most of these fugacious cases there was some connection with the grippe, but was only twice able to positively connect the two. Three of the ten were certainly at a time of the year when grippe was not in evidence.

No abscess formation in the arytenoid region has been noticed in my cases except in that form of genuine perichondritis which follows severe scarlet or typhoid fever. In one instance of typhoid perichondritis there resulted the exfoliation of a small piece of an arytenoid cartilage, and in another the whole cricoid was involved and became necrotic. The one case resulted in complete immobility of one arytenoid, with fixation of it near the median line. The other case produced the same fixation of both cords in the median line as might have resulted from a pure and simple paralysis of the crico-arytenoideus posticus muscles. Both cords being in the median position, it was necessary to do an immediate tracheotomy. These cases belong to a very different group from the fugacious type, which latter, while they may be exceedingly threatening at the time, seldom leave any complications. One has to act quickly and rapidly in the premises, but the ultimate outcome is good. Also we have to distinguish again general oedema of the glottis as it occurs as a result of some systemic condition such as disease of the kidney or purpura haemorrhagica.

I have seen one such instance of what one might call general edema of the glottis, such as is shown in Gruenwald's Atlas as published by Saunders. This oedema involved, as is shown in

the picture, not only epiglottis and glottis and the interior of the larynx, but also the upper part of the trachea. In the case shown, as in my own, no operation was of any avail.

To recapitulate, then, we certainly have several distinct types of inflammation; the branchial cysts with recurring enlargements, edema of the epiglottis with abscess formation, due, perhaps, to small retention cysts of glandular origin, and then the fugitive oedema of the simple or local type following inflammation in the throat, which latter is not to be confused with the two types which result from pure perichondritis as after typhoid fever, or from the firm swelling and slight oedema with which we are all so familiar as it exists in tuberculosis and syphilis. These are all to be distinguished from the general oedema resulting from systemic causes, from burns and from other trauma.

### *Discussion.*

Dr. W. E. Casselberry: Dr. Swain has grouped together two classes of diseases in this paper which have received but little attention in the literature. Cysts of the larynx, the epiglottis, aryepiglottic folds, base of the tongue and palatal folds, are frequent and they are summed up as a rule under the name of retention cysts. Branchial cysts had not occurred to me as occurring in the region of the epiglottis, although I have recognized them on the dorsum of the nose and in the neck. If laryngeal cysts are of branchial origin it explains their obstinate nature and frequent recurrence, notwithstanding attempts to obliterate the sac. I recorded a case many years ago under the name of cystoma of the arytenoid region of the larynx which involved this and the aryepiglottic fold. I had made a preliminary tracheotomy under stress of immediately impending suffocation, and later proceeded to extirpate by snare and cautery dissection when the growth appeared like a broadly pedunculated neoplasm about the size of a walnut. It proved to be a cyst with a thick wall and pale greenish-yellow mucilaginous contents and was permanently cured by thorough cauterization of its base. I have given up trying to curette through an evacuation puncture as impracticable on account of the mobility of the parts, gagging of the patient, etc. In all of my cases I have succeeded after an evacuation by incision, in extirpating the whole cyst by galvano-cauterization, by firmly pressing the hot electrode through the cyst wall from the outside, making three or four deep lines by cautery incision through it. I believe this to be

the simplest and best method. The inflammatory reaction is not dangerous, but enough in connection with the eschar to destroy the secreting remnants of the cyst wall.

Concerning "idiopathic inflammations" of the epiglottis I think we should designate them differently. The term "idiopathic" is nearly obsolete. We now know that nearly everything grouped under it in former times was of an infectious, microbic origin. I have seen cases similar to those described by the essayist, accepting them as infectious by some one of the species of germs which commonly cause inflammation elsewhere in the throat. Formerly, when epidemics of diphtheria were more prevalent, it was not unusual to see several affected in one household, some with a typical exudate and others with redness and swelling, but with little or no visible exudate, one of whom, as I recall a particular case, requiring intubation to relieve stenosis of the larynx, but did not exhibit an exudate, and produced a culture of uncertain identity because similar to but not identical with the typical diphtheria bacillus, this organism at that time not having been fully classed in all of its seventeen or more varieties and attenuations. Only last Winter, during an accidental epidemic in a hospital, one of the internes approached me for diagnosis; he had severe pain on swallowing, with sore throat. The pharynx showed a general redness, which might have passed for a severe pharyngitis, being without an exudate. The epiglottis showed in the mirror great oedema, and near by, but not on the epiglottis itself, was just a suggestion of an exudate in two irregular spots so slight that if he had not told me there was diphtheria in the hospital I think I would have overlooked the exudate. A culture, however, showed diphtheria bacilli. Also, I have observed pronounced oedema of the epiglottis in a young woman which I feared for days would necessitate operation, which was not diphtheritic, according to the culture, and which I first thought might be tuberculous. It lasted three weeks, recovered, recurred once in minor degree some months later, again recovered and has remained perfectly well for two years or more. The culture showed nothing more definite than streptococci and staphylococci, but no other cause for the oedema could be determined either then or since.

Dr. J. E. Newcomb: One point suggested by Dr. Casselberry recalls a conversation which I had with Dr. William H. Park of our Public Health Department of New York, to whom we turn in matters of opinion in pathological diagnosis and especially in questions of diphtheria. We were speaking of the subject of diphtheria without membrane, and I understood Dr. Park to say this, that there is membrane in every case of diphtheria, not always visible and in some instances the only deposit

would be a very small area deep down in some single bronchus. And in every instance these cases would give a positive culture reaction, though the throat might not show the slightest sign. In the present light of our knowledge diphtheria without membrane is not to be expected.

Dr. Emil Mayer: I have recorded a case of the second class presented by Dr. Swain of a patient whom I was asked to see suffering from dyspnoea. He was in the anaesthesia room of the hospital, and the surgeon simply waited for my opinion as to the condition which existed before he did a tracheotomy. I found that the epiglottis only was involved, a firm, congested thickening of the epiglottis itself, and I suggested that we had to deal with an inflammatory condition, and that it was not such as to require tracheotomy, and that he be returned to the ward, hot inhalations used with regularity and applications of 1-10,000 of adrenalin given. Following that treatment the patient's condition subsided as far as his dyspnoea was concerned, and at the end of one week's time the swelling had almost entirely disappeared. A fortnight later, when I came to see the patient before his discharge, I found that there was destruction of more than one-half of the epiglottis and the remaining portion stood out like a finger extended in the air, with ragged edges about the site where the ulcerative destruction had taken place. It seemed to me, however, that this had nothing to do with the present condition, and my final conclusion was that we had an acute inflammation of the remainder of the mass of the epiglottis that had already been destroyed.

Dr. E. Fletcher Ingals: Just a word with reference to the treatment of these cases. I had, during the last year, a cyst of the left side of the larynx, extending from the vocal cord upward. It appeared to run similar to a branchial cyst. I succeeded in curing it by aspirating the fluid through a large strong needle, and without withdrawing the needle injecting the cavity with carbolic acid and glycerine. I had to repeat the operation two or three times. The cyst was permanently cured.

Dr. J. M. Ingersoll: I have had two cases which I thought when I saw them were simple retention cysts of the epiglottis; one was discovered by accident and had caused no symptoms; the other caused considerable discomfort. After cocaineizing I grasped the cyst wall itself and simply cut off the whole top of it. This treatment was all that was necessary in these two cases. They did not recur.

Dr. H. L. Swain: I would like to ask Dr. Casselberry what the contents of his cyst were, because the distinguishing feature of cysts is the character of the matter found in them, to separate them from the ordinary retention cysts which occur in

various parts of the throat. As to the use of the galvanocautery, it is an ideal method when it is possible to adopt it to the circumstances as you find them in any given case. In regard to the possibility of diphtheria in the cases of the second group, I am sure that had nothing to do with them. It is rather difficult even when you have a membrane to determine which micro-organism is the cause of the trouble. These micro-organisms may grow anywhere, so that unless you get directly at the pus of an abscess without contact with any other part of the throat the character of the process, whether due to this or that germ, cannot always be determined. As to the term idiopathic, I think that the grouping given by Dr. Ballenger is more acceptable.

Dr. Newcomb, in speaking of diphtheria reminds me of a case which we had recently of laryngeal stridor in a child of very unusual origin apparently. The child's larynx was examined perfectly and there was absolutely no trace of membrane anywhere, but deep down in the trachea I got just a fleeting glimpse of something which seemed to me possibly to be membrane, and a diagnosis was made of primary laryngeal diphtheria of the subglottic space. Later on a bit of membrane was coughed up. He developed membrane all down the inside of his trachea and away down in the bronchi of one side. But in the membrane coughed up we could find no Klebs-Löffler bacilli. Just before I left to come here, the little fellow developed a paralysis of one arm and leg, which had come on two weeks after he was supposed to be entirely well. This settles the diagnosis of its having been a diphtheria, even though we found no membrane until after the symptoms were well established. Dr. Ingals's idea of keeping the needle in the cyst after puncture and injecting medicaments at that time is an ideal treatment, when the circumstances are such that you can carry it out. Dr. Mayer's case was certainly very interesting.



## SOME SURGICAL EMERGENCIES ASSOCIATED WITH THE TUBERCULOUS LARYNX.

BY CHARLES PREVOST GRAYSON, M.D.

I am quite aware that the small group of cases of which I shall speak represents a series of conditions that, while they can lay no claim to novelty, yet possess a clinical interest which arises largely from the very fact of their frequency. My purpose in reporting them is to give support to my assertion that they are of much too frequent occurrence, that they are almost, if not always, preventable, and that the responsibility for their occurrence is to be laid elsewhere than at the door of the laryngologist. As compared with others of the laryngeal complications of pulmonary tuberculosis, these cases of mine may not, perhaps, be regarded as among those most common, but I wish to submit to you the question as to whether it is not extremely seldom that there is any excuse for their happening at all. Speaking for myself, I have no hesitation in saying that there should be no such thing as emergency surgery in connection with the tuberculous larynx, and I say with equal emphasis that there would be no such thing if the general practitioner acquired the habit of placing the larynx of each of his tuberculous patients under the protection of the specialist either at the moment that there was reason to suspect its implication in the disease, or, better than this, even before such reason became apparent. I shall venture to tax your patience with no more than simply the essential clinical details of three comparatively recent cases in my own practice. The first, a man of forty with but moderately active involvement of the upper portions of both lungs, who had been under treatment for five months, and who had developed laryngeal symptoms something more than a month previous to my seeing him. The request for my assistance arose from a rapidly increasing dyspnoea, which, by the time I reached him in one of the suburbs of Philadelphia, was so extreme as to momentarily threaten death.

A hurried glimpse of the larynx disclosed a general infiltration moderate in degree everywhere but in the interarytenoid space. Here, however, it was of such prominence that the cords were widely separated and through the interval between them I could see that the seriously obstructive condition was a sub-glottic oedema or infiltration. Under the circumstances I would have preferred a low tracheotomy, but there seemed scarcely time for a careful dissection or anything that would have prolonged the dyspnoea by a single unnecessary moment, and so the high operation was performed. The patient is still living, the enforced rest has greatly benefitted the larynx, but the outlook for recovery is quite hopeless. If, a few weeks earlier, the larynx had been properly cared for I am confident that no such condition would have developed within it and that the ultimate result of the case might have been a happier one.

The second of these patients was a lad of nineteen, whose pulmonary disease was of about two years standing. Fully three months before my summons, laryngeal involvement had been announced by vocal impairment, and this had been allowed to progress without any interference until there was complete suppression of the voice. Ten days previous to my visit pain had been added to the aphonia, had rapidly increased, and within another day or two dysphagia and dyspnoea made an almost simultaneous appearance. It was the seemingly imminent necessity for tracheotomy that called me to his bedside. The mirror disclosed an enormous swelling of the tissues enveloping the left arytenoid, the color of the tumor being sufficient of itself to indicate its purulent contents. With the patient recumbent and his head lowered, the abscess was freely incised. The following day the subsidence of the swelling permitted the remnants of the deeply ulcerated cords to be seen, and it became clear, I thought, that a perichondritis of the arytenoid had, as a result of a secondary pyogenic infection, eventuated in the abscess formation. Am I over-credulous in believing that this condition also could not only have been prevented, but easily so by timely treatment of the affected cords?

The last case of the three was that of a woman of twenty-eight in whose larynx there had been interarytenoid ulceration

of sufficiently long standing to have developed a prominent mass of granulations sprouting from its edges, and, in addition to this, extreme infiltrative swelling of both aryteno-epiglottic folds. As the result of a wetting and subsequent chill, due to a sudden shower while she was out driving, an acute inflammatory oedema encroached still further upon the already narrowed glottic space and compelled an immediate tracheotomy. Again I contend that the necessity for surgical intervention in this case, as in the others, would not have arisen had systematic inspection of the larynx been adopted earlier in the course of the disease.

Surely, "he who runs may read," the lesson that should be taught by such cases as these, yet their continued daily occurrence not only in the country districts but in the very midst of our large cities shows that it is still unlearned. I fancy that few, if any, of us will doubt that if early co-operation between the general practitioner and the laryngologist became the rule rather than the exception, the large percentage of secondary infections of the larynx occurring during the course of pulmonary tuberculosis would be very materially reduced. The frequency of such involvement, as shown by the records of the post-mortem room, is at this day inexcusably large, and inferentially it suggests the surprisingly small proportion of these cases that are permitted to enjoy either the prophylactic or the curative aid, so far as the larynx is concerned, that the laryngologist might extend. This proportion, as it relates to the large number of charity cases that find their way to the post-mortem table, is, I am inclined to think, but very little larger than that that would be shown by a strict record of the patients if such were obtainable. For various reasons, more or less open to criticism, many of these unfortunates are allowed to develop laryngeal lesions which, receiving no better treatment, perhaps, than that afforded by cocaine and orthoform lozenges, medicated inhalations of doubtful utility, and gargles, concerning the harmfulness of which there can be no doubt, steadily progress to the stage at which pain and difficulty of deglutition seriously cripple nutrition, and the patient loses his one and only chance of recovery. I maintain that the physician who, without incontestable reason, permits this sort of thing to happen deserves the pillory of professional criticism no less than

he who incompetently temporizes with a purulent median otitis complicating an infectious fever until the mastoid and the cranial cavity are invaded.

*Discussion.*

Dr. H. L. Swain: I would like to inquire if any of the gentlemen present have had any experience in the use of the modified tuberculin that is being quite extensively applied to the treatment of tuberculosis; whether they have had these laryngeal emergencies about which Dr. Grayson has just spoken. I have seen three patients in which œdema of the epiglottis has suddenly come on as a result of treatment by injection where there was no other apparent cause.

Dr. W. E. Casselberry: While the general trend of the paper is admirable and timely, I feel that perhaps it conveys a wrong impression concerning the proportion which the cases capable of arrest or recovery bear to those which are inevitably fatal. Surely, all cases of laryngeal tuberculosis should be accorded the amelioration of skilled treatment, with its better chance of recovery, but there is no assurance that recovery will result or of our being able actually to control the septic and other complications of the original tuberculosis process to which the essayist refers. I have recently recorded a series of cases of tuberculosis of the larynx which had received local, general and climatic treatment, followed by recovery or "arrest" in 20 per cent. of the number, which had endured for a period of from six to nine years. But on the other hand, I especially recall one example of another group in which, in spite of my most diligent efforts for several months to forestall the oncoming stenosis, I was not able to prevent the final need of tracheotomy and an ultimate fatal ending.

However, the 20 per cent. of "arrests" just mentioned is an encouragement. On tracing and counting them, I was surprised as well as gratified to find even that many, so forcibly had I been impressed by what is still an appalling mortality. The local treatment had been an essential aid, but general treatment and a mode of life which tended to augment their natural resistance were the major factors in promoting a practical recovery or "arrest" in the 20 per cent. of cases mentioned. Therefore, as it is usually impracticable over a length of time to employ two physicians, it should become the accepted routine for the laryngologist to assume the whole treatment of laryngo-pulmonary tuberculosis. Otherwise the most dangerous feature is neglected, for the generalist is unprepared to cope with the laryngeal phase. It was formerly customary for the laryngologist to cul-

tivate a continuance and development of his general medical education by its practical use in this connection, and a return to this custom, in the interest of these unfortunate sufferers, might well be made a plea for emphasis in the present anti-tuberculosis crusade.

Dr. J. Price-Brown: It seems to me that the treatment Dr. Grayson gave in his cases is the very treatment required for these patients, namely, absolute rest. I reported a case of tuberculosis of the larynx in which the infiltration was so great and the emergency so urgent that I had to perform tracheotomy; that was five years ago, and the man is alive to-day. One-half of the epiglottis is gone, and the upper part is contracted down to a very small orifice, but he still works eight hours a day and is free from disease. In this case the rest was given by the tracheotomy.

Dr. J. O. Roe: I thoroughly agree with the speakers that often in these cases we are at our wits end to know what to do to give the patient relief from the suffering attending tuberculosis of the larynx.

I consider rest of the utmost importance in the treatment of these cases, although I cannot advocate tracheotomy for this purpose only. It, however, becomes necessary in cases of great dyspnoea, or to prevent strangulation from odema of the larynx. The simplest and most effective way of giving the larynx rest is to prohibit talking and even whispering, compelling the patient to make all his communications in writing or other means. Two or three years ago Dr. Porcher of Charleston, S. C., read a paper on "The Importance of Rest in the Treatment of Tuberculosis of the Larynx" by the interdiction of talking and whispering. While in Charlestown about that time Dr. Porcher showed me some of these cases, manifesting the remarkable results derived from this method of maintaining rest for the larynx. In this manner the rest given the larynx is just as complete as after tracheotomy, and we thereby avoid the added misery attending the tracheal wound.

There is one remedy that I have used with marked success in these cases, but which seems to be but little known or little used. It is iodide of starch. It is used by being blown into the larynx with a powder blower. It is not only non-irritating but very soothing, and in many cases it has given most excellent results. In these cases I teach the patient to insufflate the powder into his larynx himself. This is easily done by putting a small quantity of the powder into a glass tube about three-eighths of an inch in diameter and three or four inches long, one end of which the patient places well back into his throat; then by closing the mouth tightly around the tube and giving



a sudden inspiration through the tube, the powder is deposited in and about the larynx quite as thoroughly as by having it blown in with a powder blower in the usual way.

Dr. J. W. Gleitsmann: I would like to say a few words with regard to tuberculin preparations as to the possible dangers of its use. I have used these preparations in tuberculous laryngitis—and it is immaterial which one you use—for ten or twelve years, and I have never met with any unpleasant after effects. I have always been very much on my guard, and I have never treated a patient with any of these preparations unless he resided in my own district, because one is never sure, and it is well to have these cases always within reach. The only reason I can give that I have never had any unpleasant accident is that I have been very careful in selecting the dose. Any preparation will do, but the dose must be increased gradually. I usually make a careful examination of the chest, and then increase or repeat the same dose, or leave it off, etc., according to my examination, and so I have never had any reactive local condition in the larynx or anything which was annoying, or which gave forebodings of danger. About the throat condition, in regard to tuberculosis, it is my firm conviction that if you put your patient under ordinary good hygienic circumstances, give him proper food, with plenty of fresh air during day and night, together with tuberculin preparations, there is certainly some chance of relieving that patient. I have a case of a young girl cured of tuberculosis for six years now. There is no fever, cough or expectoration, and she has steadily increased in weight and has never lost a day at her work. With the careful use of the tuberculin I have successfully treated quite a number of other patients who could not be sent away. Only strict hygienic rules were followed, and the tuberculosis was arrested. This treatment has been extended over six or eight months with some slight interruptions.

Dr. Emil Mayer: I agree with the reader of the paper and feel that this point is not sufficiently accentuated in the discussion; namely, that the laryngologist should be called in much more frequently than he is in cases of tubercular conditions associated with hoarseness. I think that is a point which demands recognition, and I am sure there is not one of us who cannot recall cases of laryngeal conditions associated with tuberculosis where early surgical intervention would have been of incalculable value.

Dr. C. P. Grayson: It was not my intention to precipitate a discussion upon the treatment of laryngeal tuberculosis, nor did I wish to appear over-sanguine as to the curability of these cases. That which I particularly wished both to justify and emphasize was my belief that if the tuberculosis larynx were placed under



proper hygienic and therapeutic control at the moment its implication was recognized, the occurrence of such surgical emergencies as those that I have reported would become much less frequent. There is scarcely any diseased condition in which prevention is more easy than cure than it is in laryngeal tuberculosis. If in its early stages approximate functional rest is secured by the enforcement of silence, and if catarrhal or irritative hyperemia is kept within bounds by the employment of appropriate cleansing and astringent sprays, by nebulized oils and, perhaps, intratracheal injections, it is only in the more intractable and hopeless cases that serious laryngeal lesions are apt to develop. It has seemed to me that the aid of the laryngologist is little, if at all, less essential to the general practitioner in his cases of pulmonary tuberculosis than is that of the surgeon in cases of appendicitis.

## PAPILLOMA OF THE LARYNX IN CHILDREN—A FURTHER CONSIDERATION OF THE SUBJECT.

BY J. PAYSON CLARK, M.D.

Three years ago I had the privilege of reading a paper on this subject before this association. I wish now to report the results in the four cases which were then incomplete, to report four new cases, to review the literature which has appeared in the past three years and, in the light of more experience and thought, to modify somewhat the statements which I then made in regard to treatment.

In reporting the subsequent histories of cases 9, 10, 13 and 14 in my first paper, I will begin with a brief resume of each case as they were then reported.

Case 9. Boy, six years old, when first seen in February, 1901, with symptoms of a year's duration. Tracheotomy for severe dyspnoea. Papilloma removed. Eight months later larynx again filled with growth. Papilloma again removed. In February, 1902, larynx free from growth above vocal cords, but large mass below glottis. Nothing done. Seen from time to time, growth diminishing. In October, 1904, only a small growth below glottis growing from anterior wall. This was the condition of the case when last reported. On June 27, 1905, he was admitted to the hospital, and, under ether anesthesia, a small growth was removed from the larynx through the Killian tube-spatula and a small mass from the tracheal wall through the tracheal opening. On July 5 no papilloma was visible in larynx or trachea. September 11. No papilloma. Tube removed. The patient reported again on March 17, 1906, when the larynx was normal. Under ether anesthesia the fistula left by the tracheotomy tube was dissected out and the wound sewed up. Recovery was uneventful.

Case 10. George W., nine years old, when first seen in August, 1904, with history of hoarseness for six months and dyspnoea for two weeks. Tracheotomy done. Growth removed in September, 1904, and again in February, 1905. It recurred rapidly and further operative interference was abandoned. Such was the state of the case when I reported it. On May 15, 1905, the record says: "Papilloma not increased in size since last visit. Rises from

right side and anterior commissure." July 10: "P. appears smaller." August 28: "Growth about the size of half a split pea in anterior commissure." December, 1905: "The same." May 11, 1906: "Papilloma having remained quiescent for ten months, it was decided to remove what remains." The patient was etherized and placed in the Rose position. By means of the tube-spatula a good view of the larynx was obtained. A growth the size of a pea was seen on the anterior tracheal wall below the vocal cords and was removed with alligator forceps and curette. On May 24, there being no evidence of papilloma anywhere, the tracheotomy tube was removed. It had been worn for one year and nine months. On July 18, 1906, the edges of the tracheal fistula were refreshed by dissecting out the scar tissue and the edges brought together and sutured.

July 22, 1907: "Larynx free from papilloma, vocal cords normal in appearance, voice good. Tracheotomy wound has healed nicely."

Case 13. Nellie S., aged six, entered hospital on July 5, 1904, with a large papilloma, completely hiding the glottis. Tracheotomy was done, and on September 26 the growth was removed. It recurred rapidly and over a greater area than before the operation. Under these conditions non-interference was decided on.

Note on May 5, 1905: Large papilloma visible through tracheotomy wound, apparently attached above. No change in larynx."

September 22: "Comes in with history of having been ill with pneumonia since June 28. Papilloma is much diminished. None visible through tracheotomy wound. Breathes fairly well with tube closed and can speak aloud. Examination of chest shows dullness and diminished breathing over lower half of right side, front and back, slight dullness at left base behind. X-ray showed opaque shadow from fifth space to base of right lung with less dense shadow at base of left lung. It was thought that there was fluid on right side, but a needle was inserted with negative results. The patient's general condition rapidly improved in the hospital. Her temperature became normal on October 4, and she felt perfectly well. She was discharged "relieved" on October 12. Diagnosis: Thickened pleura and chronic bronchitis.

On November 10 the papilloma had increased somewhat, and her general condition was poor. Her temperature was 100, pulse 160, and respiration 40. Physical examination of the chest gave practically the same condition as in October. On November 24 the condition on the right was about as before, but in the left axillary line there was a dull area about  $2\frac{1}{2}$  inches in diameter with fine moist rales. Broncho-pneumonia? The patient had a temperature of 102, chilly feelings, poor appetite, pain in back and sides on cough, which was frequent. The temperature

dropped to normal on night of the 24th and remained so. The sputum at this time was large in amount, containing a few influenza bacilli, many pneumococci, some in long chains, all extracellular, and many other bacteria, but no bacilli of tuberculosis. On November 29 she was discharged "relieved." The diagnosis was croupous pneumonia. She died at home on February 12, 1906. A letter from the family physician, dated June 16, 1906, contains the following: "The condition of Nellie S. during last few weeks was one of exhaustion and pyemia. She took little nourishment, vomited more or less and raised through tube large quantities of fetid pus, as much as four ounces at a coughing spell. She complained of some pain in region of heart during cough. She had not been able to lay down for a couple of months. Sputum showed no bacilli after repeated examinations during last few months, although tuberculosis is in her immediate family."

Case 14. Jennie M. C., age two years, was admitted August, 1904, on account of increasing hoarseness and dyspnoea. An examination showed the larynx almost filled with papilloma. Tracheotomy was done, and in October the growth was thoroughly removed. In February, 1905, the larynx was again full and it was decided not to attempt further removal. On September 8, papilloma was seen presenting at the tracheal opening, as well as in the larynx. On January 19, 1906, while changing the tube, the child was nearly asphyxiated by the closing of the tracheal opening by a papilloma. A portion of this growth was removed. On March 1st a small papilloma springing from the tracheal wall was removed through the tracheal opening. On May 21st the growth had diminished. On July 9th the remaining growth was removed through a Kierstein autoscope. February 13th, 1907: "Very small papilloma, 1 to 2 mm., in diameter on anterior portion of left vocal cord." February 18th: "Small papilloma removed with snare through tracheal opening." April 10th: "Growth on cord nearly disappeared. No recurrence in trachea. Tracheotomy tube left out." September 25th: "Growth has gone. Left vocal cord slightly reddened at site of growth."

In April, 1908, the larynx being normal in appearance, the patient was admitted to the hospital, the tracheal fistula dissected out and the wound sutured.

Since the above cases were first reported there have been four other cases in the Massachusetts Hospital throat clinic, which I will report.

Case 15. Alphocena M., girl age six, was first seen on January 10th, 1905. She had convulsions a year before and lost

her speech a few days after. Speaks now only in a whisper. The larynx appeared to be two-thirds full of papilloma. On January 14th a small piece was removed. On February 11th the growth was seen to have extensive base but no large pieces. There is no record after this, and a letter written to the parents on March 11th, 1908, was returned stamped by the Post Office authorities, "Removed. Address unknown."

Case 16. Herbert H., age 10, came to the clinic on March 14th, 1905, with the history of hoarseness and slight cough for a year. On examination a papilloma was seen apparently protruding from the left ventricle, and a smaller one in the anterior commissure. On March 21 the Hooper laryngeal curette was passed twice but no growth obtained. On April 13th there was no material change in the size of the papilloma. On May 25th the growth had nearly disappeared and the voice was fairly loud. The patient was written to in March, 1908, and the following reply received: "The boy's throat has been very well since the last visit. He has grown rapidly, and Dr. B., our local doctor, thinks he has outgrown the trouble."

Case 17. Annie F., age seven, was seen in August, 1906. There was a history of mouth breathing, choking at night and inspiratory stridor. The patient was aphonic and there was marked supraclavicular retraction on inspiration which was whistling. An examination showed a large papilloma on the right cord, causing marked obstruction. Tracheotomy was advised. The patient did not return, and a letter sent on March 11th, 1908, to the address given was returned stamped "Unclaimed."

Case 18. Charles B., age three, was seen first on May 22d, 1907, with the history of having lost his voice eight months before. The laryngeal obstruction was sufficient to cause aphonia and dyspnoea. Examination showed a large mass growing from both sides of the larynx. On May 24th I performed tracheotomy and the patient was discharged on the 31st, wearing the tube. After over three months of waiting, the papilloma remaining apparently the same size, the patient was etherized on September 6th and as much as possible of the growth removed with alligator forceps by direct laryngoscopy. On September 30th an examination under ether anaesthesia with the tube-spatula showed a very small growth on the right side of the larynx. An examination on March 23d, 1908, in the same manner, showed the larynx absolutely free from papilloma. The tracheotomy tube was removed and the tracheal fistula will be closed after waiting two or three months to be sure that there is no recurrence.

The outcome in case 14 adds one more death in the group

first reported, making five fatal cases out of ten in whom tracheotomy was done. The fatal termination in this case does not alter my belief that the method pursued was the right one. If the growth had been removed at first without preliminary tracheotomy and there had been no occurrence at the time requiring an emergency tracheotomy, the growth recurred so rapidly and extensively that tracheotomy would have eventually been necessary. If the growth had been removed by laryngo-fissure there is no reason to believe, from the history of similar cases, that there would not have been an equally rapid recurrence. It is obvious, however, that children wearing tracheotomy tubes should have good home care. The parents should be instructed in the care of the tube and in reasonable precautions against catching cold, and that measles should be guarded against as particularly dangerous.

It hardly seems necessary to speak of the advantages of a preliminary over an emergency tracheotomy. In the former, the site of the incision is carefully chosen and each step of the operation is done in a thoroughly surgical manner, while in the latter the importance of opening the trachea as soon as possible makes a careful dissection impossible. John Rogers has said something about tracheotomy which I shall quote later.

Cases 15 and 16 illustrate the type of case which gets well without tracheotomy after one removal of the growth. In these cases the papilloma was evidently in a retrogressive condition. In fact, Case 16 may be said to have recovered without treatment, since the one attempt to remove the growth failed. Although Case 15 is incomplete, it seems fair to assume from the diminution in the growth in the month between the first and last examination that this papilloma disappeared without further treatment in the same way that those in Case 16 did. A bad prognosis in Case 17 without treatment seems perfectly reasonable. Case 18 illustrates well a successful result of tracheotomy, and the removal of the growth later when it had ceased to increase.

The literature of this subject during the past three years contains some interesting and valuable articles. A. Neubauer reports five cases on which he operated, three after a preliminary tracheotomy, and two without. Four of these cases were cured



and one died. This case, a girl of two years of age, had tracheotomy for dyspnoea. The papilloma was removed two weeks later. After five weeks the child had measles, followed by bronchitis and broncho-pneumonia, to which she succumbed.

One case, a girl of three years, had had laryngo-fissure a year before, followed in six months by hoarseness and dyspnoea, caused by a recurrence of the growth. This patient wore the tube for over two and a half years. The other tracheotomized patient wore the tube a year and a half. A boy, age not mentioned, had a papilloma on the right vocal cord, causing aphonia. This was removed without tracheotomy and three months later the larynx was still free from growth. A girl of three, with hoarseness for a year and dyspnoea for two weeks had a papilloma on the left vocal cord, size of a bean. This was removed without tracheotomy and the patient discharged in two weeks, relieved. Neither of these cases can be considered complete, as in neither has sufficient time elapsed to warrant assurance of non-recurrence. Neubauer removed the papilloma in all these cases with the modified LÖRICH catheter, an instrument with which it would seem to be impossible to effect a thorough removal of the growth, except by a happy chance.

KOELREUTTER reports a case in which he thinks the administration of iodide of potash in small doses caused the disappearance of the papilloma when it had recurred once after removal. It does not seem reasonable to draw any conclusions from one case when we know that papilloma sometimes disappears without treatment. GAREL reports two cases operated on through Killian's tube-spatula without previous tracheotomy. Both cases exhibited marked dyspnoea, and tracheotomy instruments were ready. One case was operated on four times in eighteen days, and the child discharged five days after the last operation, with the larynx free. In the other case there was an attack of suffocation the night after the first operation, for which intubation was done. This child was operated on three times in nine days. Then operative interference was interrupted on account of an attack of rheumatic fever. Neither of these cases is of value statistically, because reported too early.

McCREARY reported the case of a girl, two and a half years old,

in which intubation gave no relief. In fact, the condition was worse. Tracheotomy was done, and the child is still wearing the tube. In discussing this case, John Rogers said that a high tracheotomy with a long curved tube was very liable to make a "spur" behind, or a stricture, if the opening was made to one side. If the wound is median and not too high the chance of stricture is slight. Everything depends on where the incision is made. This is a strong argument against an emergency tracheotomy. In not one of my cases was there the slightest evidence of a tracheal stricture. Rogers reported that in one case he had opened the larynx and trachea and cut through the hyoid bone, excising all the papillomata and then put in an intubation tube and sewed the wound up tight. He does not mention how long the intubation tube was kept in, or the condition of the voice afterwards. This operation had been done nearly a year before and there had been no recurrence. He expressed the opinion that recurrence was probably due to deficiency of excision. It is surprising that he could make such a statement as this on the strength of one case in the light of the literature on this subject.

Logan Turner showed before the Laryngological Society of London the larynx of a boy, aged ten, who died suddenly while eating, apparently choking to death. He had had hoarseness, but no history of dyspnoea or choking before. Almost the whole larynx above the glottis was filled with papilloma.

Wingrave is inclined to consider laryngeal irritation due to mouth breathing caused by the presence of adenoids a factor in pathology. He states that papillomata have been reported not only as co-existing with adenoids, but as having spontaneously disappeared after the removal of the latter. The writer is obliged to disagree with this statement. While he has had cases in which adenoids were present and were removed before the papilloma was touched, it has never been his good fortune to see any diminution of the papilloma following this operation, or to find any such cases reported in literature.

Baumgarten has published an interesting and instructive article which I shall quote rather freely, because his conclusions coincide closely with my statements made three years ago, and with my present position on the subject. He propounds an inter-

esting theory as to the etiology of papilloma in adults, which I will not go into, as it is not germane to the subject of this paper. He suggests the possibility of a gonorrheal discharge from the mother at birth being a factor in etiology. As another factor he mentions bad hygienic surroundings. He also states that blondes are more susceptible than brunettes, and that adenoids stand in no causative relation. There comes a time, he says, with every child who has papilloma when the power of the papilloma to recur after operation ceases. In rare cases it ceases after the first operation. In most cases, on the other hand, it loses this power only after months or years and it is immaterial whether one operates endolaryngeally or by laryngofissure and removes most carefully the minutest growths and destroys the base. He says further that all cases that have measles die of pneumonia. It is my opinion that, while measles is a very grave complication, it is not necessarily fatal. Case 4 in my series recovered from pneumonia following measles.

Baumgarten keeps the tube in for six months after the larynx is free. He advises tracheotomy for children under ten years of age, even when dyspnoea is slight, recognizing the disadvantages of an emergency tracheotomy. After tracheotomy one can operate unhampered by any thought of interference with the respiration. Children who are tracheotomized bear the operation better. Hygienic surroundings, nutrition and age affect the prognosis.

In children under six years, if symptoms of suffocation are present, or come on during examination, tracheotomy is done.

A case reported by B. Robinson illustrates the evil of not leaving the tracheotomy tube in until one can be reasonably sure that there will be no recurrence. A girl of five years had tracheotomy on account of cessation of respiration during examination on March 19th, 1904. The papilloma was removed in two operations and the tube removed a week after the second operation. The patient returned on May 14th with severe dyspnoea, for which she had to be intubed. The growths were removed intralaryngeally in July. On August 27th the tracheotomy wound had to be reopened and the tube inserted. In September thyrotomy was done, the growths removed with scissors and the base

cauterized. The tracheotomy tube was removed in five days. In January, 1905, the papilloma had recurred and was partially removed intralaryngeally. In February laryngotomy was again done for suffocation. The tube was removed in a few weeks. The patient was admitted to the hospital in June with bronchopneumonia. She recovered from this disease but the larynx was found to again contain papilloma. These were removed intralaryngeally from time to time. An intubation tube was occasionally inserted for dyspnoea. During 1906 the growths were removed nine times, the last being in November. Breathing became bad in December and she died suddenly on January 2d, 1907, of suffocation, before relief could be given, nearly three years after the case was first seen. One cannot help feeling that if the tracheotomy tube had been left in uninterruptedly and no attempt at removal made until the papilloma had evidently ceased to grow the result might have been a different one.

Van den Wildenberg reports two cases seventeen and eighteen months old respectively, operated on through the Killian tube spatula. These cases are interesting on account of the youth of the patients, but are too recent to be of value otherwise.

Zuppinger advises intubation instead of tracheotomy when interference for dyspnoea is called for. His preference does not seem well taken, because of the many cases in which the papilloma grows so high in the larynx that it would obstruct an intubation tube, but no less because the presence of the tube would act as an irritant to stimulate the papillomatous growth. Ferreri favors laryngo-fissure for papilloma even in adults. He reports three cases, two adults and one child. It seems very strange that anyone can still advocate this operation for laryngeal papilloma. In T. J. Harris's case, reported before this Association in 1905 and 1906, in which the removal of a fibroma from the larynx of a child by laryngo-fissure was followed about a year later by the growth of a papilloma, the second laryngo-fissure for the removal of this growth was more justifiable than in the ordinary case of papilloma. But even in this case I should have been inclined to proceed as in any case of papilloma.

As to the cases which C. G. Coakley reported before this Association a year ago, he said himself that they were too recent to

tell whether there would be a recurrence or not. Consequently, it must be admitted that his results, as then reported, cannot be considered as proof that one would be justified in operating without previous tracheotomy in every case. I think that he would admit that, at least in cases where dyspnoea was marked, preliminary tracheotomy is the safe procedure.

What has been added to our knowledge of this subject during the past three years? As to the etiology and pathology, practically nothing. Wingrave's suggestion of the presence of adenoids as a causative factor seems to lack sufficient support to be worthy of consideration. Baumgarten's, that a gonorrhoeal discharge from the mother at birth may be an etiological factor, is obviously, for many reasons, a difficult question to investigate satisfactorily. That bad hygienic surroundings favor this affection is certainly borne out by its comparative rarity in the children of well-to-do parents. Wingrave states that papilloma is found more frequently in the anterior half of the larynx. In nine of my cases it is stated that the papilloma sprang from somewhere in the anterior of the larynx, but was limited to that region in only five of these. In five cases the site was one or both sides of the larynx, and in three others the site is not definitely stated. In one case the papillomata grew from the interarytenoid space as well as from other parts of the larynx. On the whole, my experience bears out this statement of Wingrave.

*Treatment.*—In treating these cases our aim is to get rid of the papilloma in the shortest time and with the least risk to the patient. Secondary desiderata are that the voice should not be impaired, and that there should be (in girls at least) no external scar. It is plain to anyone who has studied this subject, that no one method of treatment is applicable to every case. How, then, should one decide in a given case what method to pursue? The operation of laryngo-fissure I dismiss at once as never applicable to the treatment of this affection. The first question, then, which confronts one on seeing a case for the first time is shall we do a preliminary tracheotomy. Mackenzie advises tracheotomy as soon as complete aphonia is present. Garel operates without tracheotomy, even when marked dyspnoea exists. Others take positions between these extremes. I stated in my first paper that

"these growths will not yield to any form of treatment which has been attempted, however radical, until their period of active growth has passed," or, in other words, until they have lost their power of reproduction. The age of the patient offers us little, if any, assistance in deciding whether the papilloma is still growing and has not lost its power of reproduction, or whether it is in a quiescent state and will not recur if removed. If, therefore, a case is seen before dyspnoea has developed, a careful record should be made of the site and size of the growth and the patient should be kept under observation for a few weeks. If, in that time, there is no increase, removal without preliminary tracheotomy may be attempted. The various instruments used for operating with the aid of the laryngeal mirror must now give place to straight instruments and direct vision. This is obtained under general anaesthesia, by means of the Jackson laryngeal speculum, the Killian tube-spatula or the Kirstein autoscope. I have had less experience with the first mentioned instrument than with the other two, but find it offers distinct advantages where it can be used, not the least of these being the better illumination. For very small children the Killian instrument is best on account of its size. After one of these instruments is introduced the larynx is sprayed with a one per cent solution of cocaine to quiet the laryngeal reflex. Tracheotomy instruments should be ready for emergency. Great care should be exercised in removing the papilloma not to wound the mucous membrane, as any such trauma may be the site of a new papilloma. The advisability of operating without a previous tracheotomy in cases where there is dyspnoea must be largely a matter of judgment in the individual case. If only a short time has elapsed since the first symptom, and dyspnoea is already present, showing a rapid growth, a preliminary tracheotomy should be done. When the dyspnoea is marked, or when without marked dyspnoea, the growth is found to fill more than half the glottis, tracheotomy should also be done before any attempt at removal is made. After tracheotomy the papilloma should not be removed at once, but the larynx should be examined from time to time to determine if the papilloma is growing or not. If, after a reasonable time, usually a number of weeks, there is no apparent increase, the papilloma may be



removed. If it recurs the operator should again wait until it has apparently ceased to grow before attempting a second removal. Repeated operations at short intervals are inadvisable, not only because they are useless when the papilloma is still growing, but also because they appear to stimulate the growth. While a cure is possible in a large number of cases by simple tracheotomy and time, as some of my cases show, and as shown in a series of cases reported by G. Hunter Mackenzie, quoted in my first paper, removal of the papilloma when it has become quiescent hastens the cure and shortens the time of wearing the tube. Apart from the curative value of tracheotomy there can be no question that it makes the subsequent removal of the growth easier and safer. The risk of broncho-pneumonia following, immediately or eventually, tracheotomy is a real one. But in the case of a large and rapidly growing papilloma, or of a papilloma recurring rapidly after a removal without tracheotomy, this operation offers the only alternative to suffocation. Intubation I consider inadvisable in any case for reasons already stated. Proper hygiene, undoubtedly, lessens very much the dangers of bronchial complications. The home surroundings of the child should be very carefully considered. I feel confident that the outcome in two of my cases would have been different if the patients had had better home care. On the other hand, I am inclined to believe that the presence of laryngeal papilloma alone makes the child more than ordinarily susceptible to bronchial troubles.

The total length of time which the tube has to be worn varies very much. Among my cases the shortest time was ten months, the longest time was four years and seven months. The shortest time between removing the tube and closing the tracheal fistula was two months, and the longest time was one year. It is not necessary to leave the tube in long after the papilloma has disappeared, because after the tube has been worn a few months a fistula is formed which will remain open until closed by operation, so that a tube can be readily reinserted, should it become necessary on account of recurrence. No fixed rule can be laid down as to the length of time which should elapse between the disappearance of the papilloma and the closing of the tracheal fistula.

I am inclined to make the interval at least six months. The voice is normal in all my cured cases.

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*Discussion.*

Dr. A. Coolidge, Jr.: I agree with Dr. Clark in his conclusions. Papilloma in a child who can not tolerate intra-laryngeal operating without a general anaesthetic is a serious condition, and one in which proper treatment may make the difference between life and death. With experience the tube spatula may do away with the necessity for tracheotomy, but if a tracheotomy tube is put in it should stay in until all danger is passed of a second tracheotomy on account of recurrence. Frequent operating and laryngo-fissure should be condemned. Denuded surfaces are liable to be the starting point for new papillomatous growth, as is sometimes seen on the epiglottis after operating. It is important not to wound healthy mucous membrane.

Dr. D. Bryson Delavan: The fact that Dr. Clark has been studying this subject for a great many years independently and that he was formerly associated with our late colleague, Dr. Franklin H. V. Hooper, who was also deeply interested in

it and an original observer regarding it, would cause us to treat with great respect anything that Dr. Clark has to tell us. In the first place there are different views as to when and how we should remove these particular neoplasms. The views set forth by Dr. Rogers, of New York, deserve consideration. The question of tracheotomy has this to be said for it that while it has great advantages there are some dangers which should not be minimized. Great care must be exercised in applying it, for many of the dangers with which we are familiar may be traced to ill-fitting tubes producing pressure upon the trachea. The trachea of the child is extremely fragile and small and it is very easy in making the incision to seriously injure it. No objection can be made to the proper introduction of the tube temporarily in cases of dyspnoea.

Dr. Harmon Smith: I have had three cases of papilloma of the larynx in children since last October. Two of these were multiple and one was single.

The case of single papilloma is in a boy of seven years and has been under the observation of my colleagues for about three years. The growth has been frequently removed with forceps and the use of cocaine. The tumor is now more fibrous than formerly, but is growing less rapidly.

The other cases are multiple. The one in a girl of ten, the other in a boy of five. Both have had tracheotomy tubes inserted, as they were cyanotic when brought to the clinic. In the boy's case the growths are particularly virile and grow larger and more quickly after each removal. In both cases the growths have been frequently removed through the Jackson tube, but after each removal there have sprung up papillomata in new places and in greater quantities than before.

This fact has led me to suppose that there might be some pathological difference between single and multiple papilloma, which has been unobserved, and which makes them spread more rapidly than the single variety.

In the case of the single papilloma there have been some abrasions of the laryngeal mucous membrane and some spreading of blood from the removal of the neoplasm, yet there have been no secondary growths; while in the other two cases new growths spring up apparently wherever the blood of the papilloma comes in contact with the mucous membrane. I firmly believe that interference occasions more harm than good in cases of multiple growths in young children.

Dr. Emil Mayer: I would like to ask Dr. Clark whether he has had any experience with the use of alcohol as a local application to the growth after tracheotomy had been done. I think Dr. Delavan made that suggestion some time ago and in some cases

that were inoperable it seemed to have this effect of producing a condition which would enable the final removal of the growth to be undertaken.

Dr. H. L. Swain: I am glad that Dr. Clark, speaking of removal of these growths, bore down on the fact that we have all observed, and I think the physician fails to recognize, namely, that the removal of these growths frequently causes new papillomata to occur in the exact point where we have injured the tissue. I remember distinctly in a young lad of about 22 where the papillomata had been recurring as fast as I could remove them, that I had the misfortune to grip the well cord directly in the forceps, and within two weeks a papilloma had started on that very spot. You have all observed it in a general way, but we do not often have such clear evidence. Thinking over Dr. Delavan's suggestion of some years ago of the alcohol spray, I started on the theory that I would only remove the growths when they got so far that the patient could not talk out loud or there was interference with respiration. In one particular case this was done, the parts sprayed with alcohol, and for now nine months the growth has not been touched nor has it grown. I lay a little of the patient's improvement to the fact in recent work on her larynx I have used the instrument such as is attached to this straight tube, made by Pfaut, which I now pass around. With it one can get down close to the healthy membrane, removing the growths much more effectively than with any of my older instruments. In three cases of papilloma of adults, none have recurred since I have had this instrument, made up with the straight cannula for use in the Killian tubes. The case I had sprayed with alcohol certainly seemed to get good effect from it from the very first of its use. Regarding a case referred to last year, in reference to Dr. Coakley's paper, I was waiting when the time of the physiological cessation of the growth should take place, not only has the papilloma not decreased in size, but it has actually increased and the child is still wearing the tracheotomy tube, the papillomata are spreading in number as well as increasing in size. I shall here refrain from interference, as was my original intention, until the growth ceases to increase. I have never had any experience with iodine. Carbolic acid in one case seemed to cause cessation of growth when pure nitrate of silver had failed.

Dr. E. Fletcher Ingals: I feel like urging a tracheotomy in all cases when there is dyspnoea before attempting the laryngeal operation. I would like to ask Dr. Clark whether he does tracheotomy in children with severe dyspnoea under cocaine alone, or must he use a general anaesthetic. I referred yesterday to a little patient with papilloma of the larynx in which I had applied to the growth a solution of tr. iodine, 2; carbolic

acid, 4; sp. vin. rect. glycerine aa., 10; aq. q. s. ad, 30. It did not recur, but whether or not the application prevented it I cannot say. Dr. Shurly told me years ago that he thought he derived benefit from the use of fl. ext. Thuja occidentalis in preventing a recurrence of these growths. I have used it in a number of cases of papilloma without sensible effect excepting in one patient, when these growths came in the nose. In that case the curette, acids and the galvanic-cautery had been used without avail, but a cure followed after a few weeks of local and internal use of this remedy.

An experience which I had some years ago may be of interest in this connection. A child of four years was brought to me suffering from papilloma of the larynx. The obstruction was marked and because of conditions at the moment I decided to try intubation for temporary relief. The tube was worn with so little discomfort that trial of its retention for pressure effects upon the papilloma was decided upon.

The easy tolerance of the tube—the ordinary O'Dwyer metal tube—led to a serious error. The growth indeed disappeared but the erosion of the cords from the pressure of the metal tube caused denudation of the free border, with subsequent adhesion of the anterior third. This patient lived at a distance and was so comfortable that she was not brought in for observation as frequently as she should have been. Had a hard rubber tube been used, and more care exercised in the after treatment, the results I am sure would have been entirely satisfactory.

Dr. J. Payson Clark:—With regard to Dr. Coolidge's remarks I should say that where the child is perfectly tractable and allows the passage of an instrument by indirect vision one may attempt, as in the adult, to remove the papilloma in that way. With regard to the scars following a tracheotomy, of course, as Dr. Rogers's statements show, a tracheotomy done in emergency is not performed in a very careful way and scars are much more apt to follow than where the tracheotomy is done at leisure. This is one of the arguments for doing tracheotomy in all cases where dyspnoea exists, not waiting until it becomes a necessity. As regards the use of intubation in an emergency, to relieve dyspnoea, it has never been my experience to see a case which was actually in a state of suffocation. Cases have shown severe dyspnoea, but I have been able in all cases to do a tracheotomy. The objection to intubation is that in cases where the papilloma fills the larynx and mounds up like a cauliflower above the false bands it is more than likely that the intubation tube would be obstructed by the papilloma above it. There may be cases, of course, where it would work satisfactorily. As to the nature of the single and multiple papilloma

I think they do not differ essentially in their structure. Until we know more about the nature of papillomata we cannot say what it is that makes them recur, but we do know in individual cases that papillomata show marked differences in this respect. I do not know that the power to recur is limited to the multiple variety, but the fact that papillomata are multiple shows a greater hold on the tissues and a greater activity of growth. I have never had any experience in the use of alcohol in this condition. I have used it in the nose with the idea of destroying the remains of polypi, but I cannot say anything as to its effect on papillomata. I was much interested in Dr. Swain's case where he saw the papilloma recur on the site of a slight trauma. I am sure this has occurred in one or two of my cases, but I have never been able to definitely locate the seat of injury as he did.



*Address.*

## THE ESSENTIALS OF VOICE PRODUCTION,

BY WESLEY MILLS, M.A., M.D.

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I wish to assure you that I much appreciate the honor you have done me, in spite of the fact that I have long been out of the ranks of practising laryngologists, in inviting me to prepare a paper for this meeting.

I was led to enter on my short career as a laryngologist almost entirely from a love of a study of the voice and the desire to be of assistance to those who use the voice in public. As a matter of fact, such persons constituted the majority of my patients. Having from the age of sixteen made a practical study of voice production and worked out many results wholly by experiment on myself unbiassed by the theories of others, I was in a position to judge of the causation of many of the troubles for which speakers and singers came to me though sometimes I went to hear some of these patients speak in public purely for my own satisfaction, that I might feel more sure on the question of causation.

My interest increased; I began to read all I could lay hands on that bore on the subject of voice production, and I must say I never spent the same amount of money on books badly as on those bearing on the voice as it was treated twenty years ago by other than physiologists and laryngologists, speaking generally. Being enthusiastic and determined to know the truth, I carried out some laborious investigations and practised auto-laryngoscopy industriously, and I think very profitably. Up to the present hour my interest has kept increasing, especially as I have always been a practical musician, and music is to me as daily bread. I have preferred to give you a little of my own history rather than attempt to prescribe what shall be the attitude of the

laryngologist to this subject so far as his own studies are concerned.

That his patients will appreciate real knowledge of the voice, my experience gives ample warrant, but to influence them much he must show a sympathy with and some knowledge of music sound as far as it goes. He may choose to say nothing to his patients that bears on the subject of voice production—he may simply draw his own conclusions as to the methods of the speaker or singer after the necessary inquiries as to his teachers and methods, or he may take a bold stand and feel it his duty to prescribe a change as to both. Were I myself to again return to the practice of laryngology, I suspect that I would often do both, though aware that this might not put money in my purse. It produces, in my mind, an indescribably miserable feeling to know that the methods a young person of a naturally good voice and sound vocal organs are pursuing, must inevitably lead to the ruin of that voice. In such a case there would be for me but one course to take. I do not prescribe for others.

In dealing with the essentials of voice production I think your confidence in what I have to say may be largely in proportion to the fewness of the principles on which I insist as essential. When one reads or hears accounts of the "methods" of teachers, and finds that they differ, or seem to differ so much, one naturally asks: Whom of all these professed experts are we to believe?

Oliver Wendell Holmes said the medical was the most sensible of the professions, so I presume that if I am to prove worthy of your confidence I must so present what I consider either as essential or very important in such a way that it shall at least seem sensible.

I remember when a youth hearing a man of considerable attainments and ability say that he could tell all he knew about any subject in five minutes. This statement caused me to marvel then, but it is not so mysterious now. Although I have been studying the subject theoretically and practically all my life, I feel satisfied that I can state all the essentials in five minutes or less. Such statements might, however, be so imperfectly comprehended that they would serve no enlightening purpose. But just to put the matter in this brief form for the moment, I should

say that to realize that speaking and singing were in essentials the same—that physiological adjustment of the various parts of the vocal organs was the keynote to the whole—that practically all respiration exists for the expiratory blast—that the principal adjustment was between the expiratory action of the respiratory mechanism and the vocal bands, known as “the attack”—that this is completed by a further adjustment of the upper or resonance chambers to the parts below—that one speaks with the tongue—that one must on no account speak with the throat—that he must speak from the throat into the resonance or upper chambers, that is, he must speak towards his face—that he must especially speak and sing with that part of his mouth cavity lying between the soft palate and the teeth—that he must keep the back of the throat and the front boundaries of the mouth cavity out of the way—that he must never strain or use more force in phonating than the organs can bear without suffering—that artistic speaking and singing are always physiological—that vowels alone are musical and that consonants are necessary nuisances, but deserving of close attention.

Having said this, what remains further to be said that is really vital? Now perhaps I cannot do better than attempt to illustrate, as I would do for a body of intelligent medical students who had completed their second year and who were at once musical and interested in the voice. I think I should tell them that the key to the understanding of the respiration is to remember the great importance of the diaphragm and the lower chest; that the key to the complex movements of the larynx is found in the relations of the vocal bands to the arytenoid cartilages—the swivel-like action of which is especially to be borne in mind.

Most voices, especially in America, are somewhat lacking in resonance. It is important therefore that the student of the voice understand this subject, and I find some such simple illustration as the following at once interesting, easily remembered and convincing.

[At this point a violin, with and without a mute, an imitation violin and a cylinder and tuning fork were used to illustrate resonance. The voice of the speaker was used to illustrate other points and many of the principles of the paper were expanded considerably.]

The fact that vowels alone are musical and that consonants cannot but be the reverse follows from the simple fact that the former all call for the open mouth and the latter all close the mouth more or less and interrupt the breath stream. Vowels are learned first, the infant cries on a vowel sound, and consonants come later—a hint to teachers of voice production.

Many persons in their zeal for good consonantal utterance forget that there is a danger in dwelling on the initial consonant of a word, the effect of which may be to keep the mouth too long closed or to leave it imperfectly opened on the vowel, so that I urge that the voice student bound off the initial consonant and get the mouth open on the vowel. This is especially important for singers who prolong sounds to a much greater extent than the speakers. Indistinctness does not usually arise through inattention to the initial consonant of a word, but to the final one; and in a large building it is better for the speaker to dwell only moderately on final consonants and to make frequent and somewhat lengthy pauses so that the listeners may be enabled to hear not only the last word, but to grasp the entire phrase and not feel himself under constant tension in order to follow the speaker at all.

It is much to be regretted that speakers are not equally convinced with singers of the importance of the study of technique or the movements of the vocal organs necessary to the best results. An enlightened singer thinks it necessary to spend years in acquiring technique, but any such conviction on the part of a speaker is of the rarest, and with corresponding results.

The time was when a man could go to the theatre and hear our noble English tongue nobly uttered, but that is only a memory. Where are the Booths, the Macreadys, the Modjeskas of to-day? Although the American nation perhaps excels all others in the amount of energy it puts forth, it is positively lazy and indifferent as regards utterance. The negro is proverbially indolent and his speech corresponds to his nature. In some cases his consonants are all but non-existent; but many white people make fairly good seconds to the despised black in this respect, and the nation from one end to the other, and Canada must be included in this indictment, is given to nasality, owing to the laziness that

allows the tongue to roll up and back and the soft palate to drop and meet it so that the breath stream must pass through the nose.

Thus bad utterance is so common that it passes unnoticed, indeed almost anything passes even with educated people among us so long as it be not ungrammatical. There is an almost universal vitiation of taste as regards vocal utterance, and an equally widespread indifference—and I only refer to what you all know in the hope that we may be more fully resolved to set our faces against this and to cry out for reforms which must begin in the home and in the school.

I lay great stress myself on exclusive mouth breathing in phonating. Some have made a fetish of nose breathing, because it is "natural." It is, under ordinary circumstances—but speaking and singing are only natural processes in a secondary sense. The lower mammals do not employ them, nor does man all the time. In mouth breathing the tongue flattens and the soft palate tends to rise and the totality of the mouth parts are put into the best form for the utterance of pure vowel sounds. Nasality becomes relatively difficult under such circumstances, while in the absence of mouth breathing it is very difficult of cure.

There is no real objection to mouth breathing, except when the atmosphere to be inhaled is either very cold or very impure. It is not to be adopted for outdoor speaking. I find that it greatly facilitates good results in both singers and speakers, while in some instances bad habits can only with great difficulty be overcome if nasal breathing be employed.

It will be seen then that the whole question of good voice production hinges on physiological adjustments of a certain kind, so that there is economy of energy and ease on the one hand and pure tones on the other. Nearly all faulty methods tend to produce venous congestion with a corresponding starvation and poisoning of the tissues of the vocal organs, owing to the simple fact that the blood is kept too long in the capillaries. One may say that all proper use of the voice is physiological while all methods giving bad artistic results are physiologically unsound.

This is another way of saying that one part of nature is not in antagonism with another. Good speaking or singing is at once natural and unnatural. Unnatural because it is never attained by the untrained man—natural because the processes must be in harmony with the laws of the body and the mind.

## THE ESSENTIALS OF SPEECH PRODUCTION.

BY G. HUDSON-MAKUEN, M.D.

Speech has been called articulated voice. Voice is the material of which speech is made. The first essential of speech production is adequate voice production.

I have ventured to define voice as a moving column of breath set in vibration by its own impact with the vocal bands and reinforced by its diffusion through the various resonance chambers into the surrounding atmosphere. The best methods, therefore, of setting the column of breath into vibratory motion and diffusing it through the resonance chambers into the surrounding atmosphere may be regarded as the essentials of voice production.

Two separate and distinct mechanisms are used in the production of voice, namely, the respiratory and phonatory mechanisms, and not only must the different parts of each work in harmony, but there must be also a complete co-ordination of the mechanisms themselves.

The voice production for speech differs somewhat from the voice production for song. In the first place it differs subjectively. To the speaker the vocal effort is secondary to the articulatory effort, while to the singer the idea of vocality is paramount. The speaker is inclined to minimize the importance of voice production and the singer the importance of articulation. The former often uses too little voice and the latter too little articulation. This tendency of the speaker to neglect his voice production leads to serious difficulties. In fact, the great developmental defect of speech, which we call stammering or stuttering, has been shown to be due in the majority of cases to inadequate voice production, so that stammering may be called hypophonia rather than hyperphonia, a term that Professor Scripture has given to it in a recent article.

Moreover, the voice production for speech differs somewhat from that for song in still another respect. The voice for speech varies more frequently than the voice for song, and it requires for its production more numerous and more rapid adjustments of



the mechanisms. The sustained voice of song is rarely used in speech, although it may be approximated in the strongly oratorical and dramatic roles. A greater volume of voice may be required for song, but more numerous changes are used in speech. In song the voice is sustained and voluminous, while in speech it is inflected and more irregularly accented and emphasized.

The difference is more marked perhaps in the use of the larger muscles of the respiratory mechanism. The diaphragm, for instance, in singing is held in strong contraction during a greater period of time and the respirations are more prolonged, while in speaking the diaphragm and other respiratory muscles are constantly changing with the varying degrees of inflection, accentuation and emphasis.

But speech is something more than voice production. It is the product of the combined co-ordinate action of the two vocal mechanisms with a third, namely, the articulatory mechanism. In the production of speech we have combinations and complexities of finely co-ordinated movements such as are required in the exercise of no other bodily function. Each of the mechanisms employed has its own delicately co-ordinated action, and for the finished product the three mechanisms themselves must be so accurately co-ordinated that there shall be complete harmony of action. To add to the complexity—and perplexity oftentimes—of the act of speech production, we have the fact that the co-ordinate action of each mechanism, as well as their combined co-ordinate action, is largely involuntary and automatic.

This brings us to a consideration of the so-called central mechanisms of speech, as distinguished from the peripheral mechanisms. The central mechanisms are those situated in the brain and spinal cord, and their function is to set in motion and to control the peripheral machinery of speech and to co-ordinate its various parts.

The cerebral speech mechanisms are situated in the left hemisphere, and they are designated as follows: The auditory centre, in which are stored the memories for spoken words; the visual centre, in which are stored memories for written and printed words; Broca's center, in which are stored memories for motor speech impulses, and the graphic centre, in which are stored memories for the movements of the hand in writing. Of these four centres the first two have been called sensory and receptive, and the last two motor centres, although, as we shall see later on, there are good reasons for supposing that they are all sensory centres, and that the motor centres are situated in the bulb and spinal cord.

The chief receptive avenue for speech is through the hearing. It is through this sense that the child gets his first impressions

of speech, and it follows that when the organs of hearing are defective, the development of speech will be correspondingly impaired, and when there is absolute deafness, there will be absolute dumbness, until the child learns through special training to substitute for the sense of hearing the sense of sight, and to some extent also the sense of touch.

But a child who is not absolutely deaf may be mute, because the auditory speech centre constitutes only a part of the general zone of hearing in the left hemisphere of the brain, and if only the speech centre is defective or undeveloped there will be deafness only for speech sounds, or word deafness, as it is called, while other sounds are distinctly heard.

Moreover, no two individuals hear speech sounds exactly alike, because no two brains are exactly alike, and for the same reason no two individuals develop exactly the same kind of word images or speech habits.

Next in importance to the auditory speech centre, and closely related to it, is the visual centre, the region of the brain in which are stored memories for written and printed words. This centre is situated in the angular and supra-marginal convolutions, and it is connected by commissural fibres with the auditory centre, and also with the two so-called motor centres, which I am about to describe.

The chief motor, or Broca's centre, as it is called, is situated in the posterior portion of the third frontal convolution, and in it are stored memories for the various movements used in the production of speech. The graphic centre is supposed to be situated in the posterior extremity of the second left frontal convolution, and in it are stored memories for the movements used in writing.

The fact that Broca's center shares with the auditory and visual centers the function of serving as a storehouse for sensory impressions has led Bastian and others to conclude that it is not a motor center at all, but a sensory center, and that the motor centers for spoken words are represented in the medulla oblongata. Similarly it is claimed that the graphic center, being a storehouse for the memories of movements of the hand and arm in writing, is also a sensory center.

This disposition of the cerebral mechanisms of speech, placing the sensory centers in the cerebral cortex and the motor centers in the bulb and spinal cord, seems to be the more scientific one, because it accords more fully with both the physiologic and pathologic phenomena of speech production. Moreover, it has long been known that the centers for the co-ordination of the muscles and mechanisms employed in speaking and writing are situated in the bulb and spinal cord, and it is only reasonable to suppose that the motor centers of these mechanisms should be

in the same region of the nervous system. Bastian also gives to these hitherto called motor centers names that are suggestive of their functions. To Broca's center he gives the name of glosso-kinaesthetic center, and to the graphic, cheiro-kinaesthetic center.

As I have said, it is through the ear that one receives his first impressions of speech and it is in the auditory center that memories for spoken words are stored. The first step in the production of speech is a revival of these word memories in the auditory center. This is effected either from without, by afferent impulses coming through the auditory and other nerves of special sense, or from within, by a process of silent thinking. These revived memories are immediately transmitted through commissural fibers to the glosso-kinaesthetic center, where they in turn arouse previously stored memories for movements employed in the production of speech. Simultaneously there may be revived also in the visual center memories for the movements of the vocal and articulatory muscles as seen by the eye, and this center is also connected by means of commissural fibers with the glosso-kinaesthetic as well as with the auditory center.

Although it is in the auditory center that the revival of word images primarily takes place in most people, it occasionally happens that visual images are the first to come into the consciousness, and less often perhaps the glosso-kinaesthetic images. To the three classes of individuals thus characterized, Charcot gives the names, *auditifs*, *visuels* and *moteurs*.

In the revival of the word images, an important mental faculty is required, namely, that of attention, and in their externalization by means of articulate speech, the faculty of volition is employed. The exact anatomical connection between the willing to do a certain act and the act itself has never been satisfactorily explained. We only know that when one wills to speak, for instance, the act follows as a natural consequence, unless it is inhibited by other more powerful nerve impulses.

The order of cerebral activities in the production of speech, therefore, is, first, the revival of word images in the auditory center (and in some cases in the visual center) by means of afferent impulses from without through the special senses, or by means of impulses from the higher intellectual centers from within the brain; second, a transmission of impulses from these revived images in the auditory and visual centers through commissural fibres to the glosso-kinaesthetic center; and, third, a revival of the kinaesthetic word images, which in turn incites to action the motor centers in the bulb.

A word as to the development of speech in young children may

throw some light upon this interesting subject. The first cry of the infant is the beginning of speech development. It is entirely automatic and reflex and its immediate purpose seems to be to expand the lungs and aerate the blood. It is not expressive of pain, because there is as yet no susceptibility to pain, and there is no psychic element in it, because the brain is wholly undeveloped. The young infant does not even hear himself cry, but as he grows older the nerve tracts leading to the brain gradually develop myelin sheaths, which give to them the power of conveying sensations to their respective end stations in the cortex, and the child begins to feel, to see and to hear. The crying now gives place in a measure to other sounds, such as babbling, crowing, mimic reading and echolalia, all of which are largely automatic and reflex, but more or less expressive of states of feeling. When the auditory nerve has become medullated the child begins to hear. He hears, for instance, the word *mama*, and he may repeat it in a mimic way, but it means no more to him than any other sound until he has learned to associate it with the individual for whom it stands, at which time it becomes enregistered in a portion of the general auditory center as a word image, and this forms the beginning of the differentiation of the auditory speech center.

When the nerve tracts leading from the motor center in the bulb to the Rolandic area of the brain become medullated, the sensations produced by the musculature of the peripheral organs employed in crying, babbling, prattling, etc., begin to make their impressions in a definite portion of the general kinaesthetic area, which portion is destined soon to become the glosso-kinaesthetic center. In a similar manner the visual word center, and the cheiro-kinaesthetic center, are developed.

Just prior to, and synchronously with, the development of these special speech centers, however, we have the development of the higher intellectual centers of the brain. The child begins to understand speech before he begins to make use of it in a voluntary manner, but it is only after he begins to make use of it that its full import dawns upon him, and his intellectual faculties become thoroughly aroused.

An important period in the child's development is that of the transition from the involuntary babbling and prattling of meaningless words to the voluntary use of intelligent speech. The child now begins to assume a conscious control of the mechanisms of speech, and this involves certain mental faculties hitherto almost wholly unused. It is true the child begins to think before he begins to speak, but his thoughts are very superficial and rudimentary. Thinking and speaking are so closely related and so interdependent that the one can scarcely be carried on

without the other. "To think," says Max Muller, "is to speak low, and to speak is to think aloud." However, the higher intellectual centers now take command, as it were, of the lower speech centers, and through them also of the peripheral mechanisms of speech, and actually train them into more or less healthful physiologic activity. This task of training the speech centers is conducive also to healthful activities in the intellectual centers, and thus they react beneficially the one upon the other, and the highest development of either is impossible without a corresponding development of the other. Defective speech, therefore, is naturally suggestive of defective mentality, and defective mentality is always accompanied by defective speech.

My purpose in presenting this paper has been, not to herald any new or original theory, but rather to give a brief resume of the anatomy, physiology and psychology of the mechanisms employed in the production of speech, and in summing up the essentials of speech production, therefore, I would say, first, that the mechanisms must be structurally normal and free from pathologic encumbrances, such as dental, alveolar and palatal irregularities, enlarged tonsils, nasal obstructions and cerebral tumors; second, the various parts of the individual mechanisms must be accurately co-ordinated, and there must be a similar accurate co-ordination of the mechanisms themselves; and, third, this accuracy of vocal and articulatory musculature must be largely automatic, and this in turn is brought about mainly through psycho-physical development and training.

*Discussion on the address of Prof. Mills and the paper of Dr. Makuen.*

Dr. J. Payson Clark: I was very much interested in these papers of Professor Mills and Dr. Makuen. I have seen a number of patients, voice users, whose soreness of throat or hoarseness was entirely due to improper use of the voice and instruction in its proper use cured the condition. My difficulty has been to find voice teachers who are able to teach an adult the proper use of the organ. I have been in the habit of illustrating to patients the difference between speaking with the voice, using the resonating chambers, and speaking in the throat where one is straining the organ of the voice. If one can throw the voice forward in the face one gets the resonance and speaks with much more ease. It is very difficult for people to understand this and appreciate it and get it into their minds, and I should like to ask Professor Mills if he has any method by which he can convey the idea to patients.



Dr. ————: One point of interest in this connection is in removal of the tonsils, whether after such an operation the voice would be affected. I am frequently asked by singers who require that operation whether their voices would be changed and I have invariably told them that if at all it would be a change for the better. Still, I have had one case in which the pitch of the voice was actually changed, and I think this is a point we ought to consider before removing large tonsils in singers. Of course the pathological tonsil, if it produces symptoms, should certainly be removed. I should like to ask Dr. Makuen what his experience is in that direction.

Dr. J. Price-Brown: I would like to ask Professor Mills if we appreciate as well as we should the value of the salivary secretion in vocalization. We know that in using the voice the constant action of the muscles increases the secretion. When we open the mouth and breathe without moving the muscles of the mouth the throat gets dry and sore; but in singing or speaking this is not so, as the muscular action induces secretion from the salivary glands to the full extent required for oral respiration during the act.

Dr. Emil Mayer: I would like to ask how soon instruction in voice production may be begun? This is very often asked, and I would like to know if any definite limit has been agreed upon. In order to get proper and good speech it is necessary to begin with the growing child, but often is this neglected; especially are Americans prone to pay very little attention to pronunciation, intonation, etc. The English, on the other hand, often pay very much attention to it and the result is that their voice is often much better modulated than the average American in this respect.

Dr. E. Fletcher Ingals: I wish to thank both of these gentlemen for their excellent papers. What a blessing it would be if, as Professor Mills hopes for, proper speech training in our universities could be realized so that a student could not graduate without a thorough knowledge of proper speech. This is a new idea, but it seems good. It would not take me long to tell all that I know about the voice. I would like to ask for an explanation of the voice in a patient from whom I had the larynx entirely removed. The trachea is attached to the skin so that there is no communication whatever between the lungs and the oral cavity. Yet the man learned to talk as loudly and clearly as anyone. I cannot understand how he does it, but suspect that the air comes from the oesophagus. With regard to Dr. Makuen's statement that the cry of the new-born child is simply reflex and that it does not feel pain, I would like to know at what age



it would realize that something had happened if the nurse should happen to stick a pin in it.

Dr. J. W. Farlow: One great difficulty I have found in trying to instruct patients in the proper use of the voice is that people do not hear their own voices as others hear them. If any of you have ever spoken into a phonograph you will often be surprised at the tone of voice which is reproduced. Often you would hardly recognize it as your own. I did this once as a matter of curiosity and I did not recognize my own voice; several others with me did the same thing, none of us having any idea whether we spoke high or low. It seems to me that if we close up the ears and hear through the bones of the head we would have an entirely different conception of our voice in that way. A number of years ago I had a patient, a young man in the high school at Lawrence, Mass., who was brought to me for operation on account of a very high voice which had never gone down. The young man was to deliver an address at a school exhibition, and on this account his friends wanted to see if something could be done for this high voice. I found that this high speech was unknown to the patient, and he could not see how he spoke differently from anyone else. I tried to imitate him and finally trained him by having him hold his larynx down, so that he could speak at a very low pitch and also in a rather creditable way. He said, "that voice sounds very harsh to me," and he added that if he spoke his valedictory in that way his classmates would all laugh at him. He did not really know that he spoke differently from others. After some instruction and getting him to speak and hear his own voice, in two or three weeks he spoke very well and he made his address at the school with much credit. He told me afterwards that he had never known how his voice sounded, and although he now spoke exactly like the other boys, it was not until he became accustomed to it that he realized that that was the proper way. This is a subject I think that we have not thought much about, and it is worthy of consideration. Another thing is the importance of starting young. I think we cannot start too young. I should not be disposed to give set exercises or lessons or so-called elocution, but I think it is of most importance that children should have some nurse who was properly educated and trained, and although a higher salary would be required for such a nurse it is worth more to the child to have a good, acceptable voice. There is not one in a thousand of us who can sing well or use our voice for the pleasure of our friends or of ourselves. A good nurse, educated and capable, together with a school in which the teachers pay some attention to the speech of the pupils and do away with the injurious practice of yelling and this sort

of so-called comic slang, would work wonders. I believe with Dr. Mills that these comic operas and miserable stage darkies are degenerating, for the thing is made amusing by being spoken in a so-called comical way. It seems to me that the point which Professor Mills has brought out is the importance of speaking in a correct way, and that we cannot start too early and that we should take infinite pains in showing others the correct way of speaking.

Dr. H. L. Swain: While we are imbibing wisdom at the expense of Professor Mills I want to ask him a very simple question. I would like to know if he can give me a simple word picture of the difference between the speaking and the singing voice, giving it so that the ordinary musical student may understand it.

Dr. D. Bryson Delavan: The efforts now being made to reduce the art of voice production to a somewhat scientific basis are certainly to be encouraged. It is unfortunate that the methods of the masters of the old Italian school should be lost with their passing out of the world. It may be impossible to separate the artistic from the scientific side of this question. On the other hand we should welcome heartily the efforts that are being made by the very few who are studying after the manner of the gentleman who has just addressed us. The number of singers who come to us for advice and help is very great. A singer whose whole career is in peril is certainly in a very serious position, and not only singers, but public speakers as well, ought to find help and relief from us. It is very disappointing to believe that we have found teachers who are properly qualified to help our patients only to find the results as bad, if not worse, than before. The subject is a very broad one, but it is interesting and important. No period is too young at which the child may be and ought to be instructed in the proper use of the voice. With regard to operations upon the throat, in particular the removal of tonsils, it is said of Patti that after her first visit to this country she suffered a considerable enlargement of the tonsils. The tonsils were removed, after which she sang much better than before. The throat should be put in order without question before the singer takes up his vocal lessons, and if the case of the patient promises ill, then the candidate should be disappointed at the outset rather than at the end of a long and arduous course of training. There are some throats that are physiologically wrong and not calculated to make the art of singing a success, and persons having such throats should at least be advised to that effect and warned of the danger of their ultimate failure. Defects of speech are very general the world over. The "nasal voice," however, is not confined to the United States, as many

of our friends seem to think. It is also to be found in the south of England, where the type of intonation and the pronunciation is exactly what is found in some American localities peopled by emigrants from England. Nasality is also prevalent in Sweden, France and Italy. If we could have a phonograph in our offices and allow our patients to listen to their own voices from it this would perhaps be the best way for bringing before them pointedly the necessity for improvement.

Dr. G. Hudson-Makuen: In discussing Dr. Mills' address I may say that it gave me great pleasure some months ago to write a brief review of his excellent book on Voice Production, and I came here to-day with some foreknowledge of his views on this interesting subject. I had only one real criticism to make of the book, and that was the fact that he did not explain the actual use of the larger respiratory muscles in voice production, and, to my mind, curiously enough, that is the most essential of all the essentials of voice production. In his address to-day Dr. Mills seemed to me to dismiss this part of the subject in the same summary manner, and I hope that, in his closure of the discussion, he will give us his views on the action of the larger respiratory muscles in the production of the voice. It seems to me to be very important, because upon the action of these muscles depends to a very great extent the action of the smaller muscles which control the vocal cords and other parts of the vocal and articulatory mechanisms. If the action of the larger muscles is not artistically correct, the smaller muscles cannot properly perform their function.

Professor Mills: I am delighted that my paper, together with Dr. Makuen's, has interested you and at all events has led to such remarks as have been enlightening and encouraging. I will make one practical suggestion which occurred to me when Dr. Delavan was speaking. The subject of joint meetings and of joint research is very much in the air just now, and I think it would be a very good thing if societies of vocal teachers could have a joint session with the laryngological societies at least occasionally. A great deal of the controversy about the voice among teachers grows out of the use of a variety of terms, the exact meaning of which is not well defined. Another difficulty is the tendency to unduly appreciate the influence of great teachers, which can only, after all, be but traditions. I am acquainted with some of these great teachers. For instance, Madame Marchesi, while knowing the old traditions through Garcia, found she had to modify them somewhat to suit the changes in the times. We may profit by the old traditions of one hundred and fifty years ago, but we must suit them to our times by finding briefer ways of obtaining results. The principles were arrived at largely empirically, but they had a chance of

testing them in a way that the modern teacher has not. And I would suggest here the advisability of the laryngologists and the more prominent, at least, among the vocal teachers getting together for joint discussion. Dr. Makuen in his paper brought out the differences between speaking and phonating very nicely, as I thought; but I think it still remains clear that speaking and singing are essentially the same. It is, I think, somewhat thus: The singer speaks whenever he addresses the public, and the speaker sings. There is no difference between singing and speaking, as I understand it, that is essential.

As to the question of how I would put the matter before a student, I may say that I had occasion to do this the day before yesterday in the case of a student of the second year who has a good baritone voice. I talked to him of how he had been using his voice. We got to understand each other, but he had the difficulty that he did not hear himself properly. The first thing that I had to make him understand was that he was judging his own voice incorrectly, because he was not taking a proper standard for his ideal tone. His standard was easy of production. He said, "My lowest tones are the easiest produced," and argued that they were his best. In reality they were his worst. We got to soon understand each other and I convinced him that he had been singing in his throat instead of into his mouth cavity.

Dr. Curtis thinks it is very important to begin the attack on a syllable, the first letter of which is a consonant, in order to avoid a forcible *coup de glotte*. I have not had that fear myself, because I give the individual to understand distinctly that he must not feel that he has a throat at all. He must only be aware of that when he directs attention to it, and if he *must* be conscious of it he is singing incorrectly.

One portion of Dr. Makuen's paper is eminently physiological and psychological, and I may say that it was lack of time which prevented my dealing with the psychological side of the question. There are some points in the most recent physiology of the cortex which seems to me rather against the view that Broca's convolution can be sensory in the first place. The work of Sherrington and Grünbaum, done on apes, is the most recent. These investigators are strongly of the opinion that all this region is motor and motor only, and that the sensory does not extend further forward than the fissure of Rolando, nor the motor further back. If Broca's convolution were to be considered sensory it would be entirely an exception to the most recent investigations.

At the recent International Congress of Neurology a number of prominent investigators took up this question of aphasia and they are all modifying the view presented by Dr. Makuen, and

the trend of discussion went to show that the present localization will not answer at all and that the difficulty is a mental one and can be explained by various defects of the brain. Therefore I think we must be prepared for a change of view on this whole subject of phasia and aphasia.

Dr. Clark has touched on a difficult subject: How to find one who will carry out methods that commend themselves to those of physiological tendencies. My ideal of a voice teacher is an individual who would combine in himself in a large measure the physiologist and the physician, the laryngologist and the musician; this is the only complete solution of the question. At present it would be well if there were frequent conferences between the laryngologist and the vocal teacher. The world is in a mood to be enlightened and educated by physicians, provided they show some sympathy with art and music. The laryngologist must try to be a musician to some extent, at least, in order to gain the sympathy of those who are teaching, especially singing.

As to the salivary secretion, I have been astonished at the difference between singers' throats and those of the average individual in the comparatively more perfect build of the parts and the fact that usually they are, as compared with many throats, comparatively dry. Much secretion is, I think, rather a hindrance than a help to the singer, especially that on the vocal bands; and I remember the case of a man who, in consequence of having been trained by bad methods was so troubled with a tenacious secretion that he could not continue to sing for any length of time. Treatment of such cases I found very unsatisfactory. Hence it would seem that just a moderate amount of secretion suits best.

As to how early to begin voice production, I think we must all agree with the remarks that have been made as to the desirability of having the child's attention called to correct and beautiful utterance; that he should be correct first and musical second; have him dislike bad speaking as a little girl would hate ugly clothes. As has been remarked, while few can sing all can be trained to speak acceptably and many very beautifully. There are many beautiful voices in America, but we learn wrong methods of speaking by unconscious imitation. I do not know that I have ever listened to a body of medical men in America among whom I had heard as little nasality as I have heard in this audience to-day. As to singing, I think the proper thing is to keep the child who is musical in a musical atmosphere, but except in special cases I do not think it is worth while to have rigid training, because the vocal organs often change so much at puberty. At the same time I should allow a musical child to sing a little if he would, but I should certainly make sure that it



was by a correct method. In choirs a good voice suffers because of the bad ones.

A point which has been brought out is that the ear is the controlling sensory centre. All muscular movement is determined by sensory impulses and the ear is the organ providing the controlling sensory impulses in this case, and the great difficulty in training people, especially adults, is owing to the fact that they do not, as has been said, hear correctly. Why do you pronounce 'hat vowel as you do? I said recently to one of my assistants; it should be thus (illustrating). He assured me that he could not observe the difference on which I was insisting. Often the first and chief preparation for speaking or singing is the cultivation of the ear. In fact, when I began my lectures this year at the McGill University Conservatorium of Music I had on one side the singers and on the other the speakers, and I began the work with the speakers by recommending them a simple exercise on the piano for the cultivation of the ear, because, unless one can discriminate fairly well through hearing there is no place for elaborate exercises. One must recognize or know good sounds before he can make the movements of the vocal organs necessary to produce them. But one must be very cautious in discouraging people about the ear. A great deal of apparent lack of hearing is simply due to inattention. But I think it would be but seldom that one accustomed to listen to his own voice would fail to recognize it when reproduced by an instrument; especially is this true of the singer. It is with those who are not used much to listening to their own voices that this is noticed. Often the very first thing I have to do in teaching people is to insist on their talking less. A good rule is to diminish the quantity by about 50 per cent and increase the quality by 200 per cent. Even with singers I have been obliged to insist on this and to convince them that they do not hear what they produce. Only the best singers really notice certain defects in their own tones.

As to the practical point of removal of the tonsil, I think I can understand why a singer may think there should be a change of pitch in consequence, just as singers often believe that they cannot sing a vowel pure all the way up the scale. That simply means that they have not sufficiently mastered either exact listening or enough technique; and in the case of the person who thought that the pitch had changed owing to the removal of the tonsil I would say that he had not sufficiently modified his method of voice production. I do not see how the removal of an obstruction must necessarily interfere with production, rather should its removal improve it. The case of the gentleman with the high voice just enforces what I have been saying; first of all



he had no ideal tone in mind, and, secondly, he got used to his own voice and it pleased him well enough.

With regard to the criticism of Dr. Makuen of my book, I would explain that I did not lay greater stress than I have done on diaphragmatic respiration, because many teachers of singing have reduced "diaphragmatic breathing" to mere cant to a great extent; they talk about it but they do not know very well what they mean. It is difficult to find in works on the voice written by teachers of singing a real good account of respiration. You will find them good up to a certain point, but as a whole they are inadequate; they do not go far enough. Now, inasmuch as every one of these accounts was either actually wrong or defective, I tried to lay stress on the point that one must learn to breathe with every part of the chest. Now, for example, there are times when one speaks or sings very rapidly and he simply takes a rapid, shallow breath that fills the upper chest and that can hardly be called diaphragmatic breathing; but certainly in very rapid speaking or singing it is all one has time for. But I do most thoroughly believe that as it is the lower part of the chest that always works with the diaphragm, it is the most important part, but this is not the whole truth. I have actually known a case of a pretty good singer who thought that breathing began below and extended up, and one of her pupils had never really expanded the chest above because, of course, one cannot begin below and fill the chest. It was various facts of this kind that led me to avoid what I considered an undue and unwise use of the expression "diaphragmatic breathing," and to point out that one must be able to fill the whole chest or to use one part more than another as circumstances demanded.

Dr. G. Hudson-Makuen: As to hypertrophied tonsils. I always advise their removal and I have had only good results from the operation. I can understand how the vocal technique of singers may have to be slightly changed after the removal of very large tonsils, but there should be no difficulty about that. The best time for the operation, however, is in early youth before the vocal technique has been established.

As to Dr. Ingals' question, I should say that the child feels a pin stick only after the affected nerve has developed its myelin sheath, which enables it to transmit the sensation to the brain.

In reply to Dr. Farlow's remarks, would say that in a paper before the Medical Society of the State of Pennsylvania last year I endeavored to show that we do not hear ourselves as others hear us, and also that this fact has an important practical bearing upon the correction of vocal and speech defects. To paraphrase a remark once made by Dr. Oliver Wendell Holmes under similar circumstances, I may say that the best time to

begin speech training is three or four generations before the child is born. In other words children inherit bad speech tendencies, which are fostered and developed later on by bad examples. The endeavor to correct faulty speech should begin as early as possible, and the development of speech being so largely imitative, greater care should be taken to give the child good examples of speech.

As to the use of the diaphragm and other large respiratory muscles I would refer you to my own articles on the subject\* and also to Dr. John Howard's book on "The Physiology of Artistic Singing," in which he explains fully the use of the diaphragm in the production of the voice. I may say that I did not get the idea from John Howard, but from a vocal teacher with whom I studied some years before reading his book. The important point to remember is that for purposes of voice production the diaphragm is an expiratory muscle and that it should not be used in inspiration.

Professor Mills: The last remark is so radically opposed to my notion of physiology in general and to what I have seen in respiration that I really must protest against it. For example, I must ask Dr. Makuen to explain how he gets the respiratory effect from the diaphragm if it is already up. By bringing it down it has all the distance from which it came and still more through which one can push it in order to gain the desired effect. The fact that in inspiration the diaphragm descends, that in expiration it ascends—that the abdominal muscles work, or may be made to work, with the diaphragm—that the muscles of the lower ribs act always in conjunction with the diaphragm—that the will must control respiration in phonation, at least till correct habits are formed, are among the leading truths of this subject.

Dr. G. Hudson-Makuen: That is explained in this way. The diaphragm covers about 1-6th of the lung surface, the thoracic walls covering the remaining 5-6ths, and when the diaphragm is contracted its surface diminishes and tends to draw the ribs downward and inward. The slight lengthening of the thoracic cavity by the depression of the diaphragm is more than made up for by the narrowing of the cavity in its lower half.

It must be remembered that when we use the diaphragm it contracts, and when it contracts strongly, instead of occupying a broad surface about the size of a large platter, it becomes about the size of a small saucer, and in this strong contraction it tends to diminish the size of the thoracic cavity by helping to pull the ribs downward and inward. This being true, it follows that in

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\*"Breathing Movements in Relation to Voice Production" *Education*, December, 1890, and "Artistic Breathing," *Philadelphia Med. Jour.*, September 3d, 1898.

full inhalation the diaphragm is always relaxed, and therefore it should be relaxed during the inhalation of voice production and contracted only during the exhalation.

Professor Mills: I think we must distinguish between the sudden action and the quiet action of the diaphragm. The diaphragm itself behaves in just the same way, but the ribs are pulled in when the action of this muscle is sudden and forcible. I have seen this action in the cat in asphyxiation when the abdomen was opened and the diaphragm exposed to view, and I do not think it is different in singing.

HEMORRHAGE FOLLOWING QUINSY—LIGATION OF  
THE COMMON CAROTID ARTERY—RECOVERY.WITH A STUDY OF 51 CASES OF HEMORRHAGE IN CONNECTION  
WITH PHARYNGEAL SUPPURATION.

BY JAMES E. NEWCOMB, M.D.

The title of this paper has been so worded as to include cases which have been reported under various headings and to permit a proper grouping of them. The majority of the hemorrhages have occurred in connection with ordinary quinsies. The subject was brought to the attention of this Association in 1900 by Dr. W. F. Chappell, who reported a case under the title of "Hemorrhage from a Circumtonsillar Abscess," and stated that he had found ten similar cases recorded. (Transactions 1900, Page 213.) The personal experience of the writer hereinafter described has led him to make an extended study of the subject, and some fifty-one cases in all have come to light. In proportion to the vast number of cases of this malady which run their clinical course without untoward incident, the number of those presenting hemorrhage is, of course, extremely small, but the alarming possibilities which the occurrence of this complication presents render it obligatory upon us always to bear it in mind and to have a well-considered plan of procedure in case it happens.

Through the courtesy of Dr. John D. McBarron I am able to report the history of the following case seen in consultation with him.

The patient was a stout German, 55 years of age, married and engaged in mercantile pursuits. He lived very comfortably and was a rather large consumer of alcohol. His vessels showed distinct arterio-sclerosis. There was no history or evidence of specific disease, and his urine, which had been examined within six months, showed no evidence of renal disease. For a period of between thirty and forty years he had had numerous quinsies, and in the intervals between his attacks much treatment with reference to the prevention of throat infection. This had been so

far successful that he had enjoyed an immunity for ten years. In none of the previous attacks had there been any clinical features out of the ordinary.

On Sunday, Feb. 3rd, of the present year, a new attack came on and Dr. McBarron found both tonsils inflamed and suspected commencing suppuration. On Feb. 6th local conditions indicated a pointing of pus on the left side, rather far out into the soft palate. An incision was made, the knife going through brawny tissue for about three-eighths of an inch and then apparently entering a cavity which, on withdrawal of the blade, voided about a dram of healthy-looking, odorless pus, and then partially collapsed, but almost immediately became distended with blood, some of which was spat out and was apparently venous. The incision was enlarged with a stiff probe, the clots turned out, various astringents used, peroxide injected and the bleeding ceased.

Some thirty-six hours later, just as Dr. McBarron was in the house visiting his patient, the bleeding suddenly recurred, and this time the color of the blood was distinctly arterial. It was at this stage that the writer was called in. The blood was coming from the original pus cavity. Bleeding was not severe, but persistent. The cavity was cleansed and packed with a strip of gauze dipped in aceto-tartrate of alum. Bleeding was again checked. The gauze was removed without incident the next morning. Some fourteen hours later bleeding again recurred and now was more persistent and in increased amount. Another attempt was made to pack the cavity, which was mainly in the soft palate, when it was discovered that the suppurative process had perforated the posterior surface of the palate and that the gauze was all going into the pharynx. It was all withdrawn and recourse had to ice internally and externally, together with the usual local applications, but all to no avail. It was impossible to localize the bleeding point. Hemorrhage continued at intervals, although the total amount lost was not enough to exhaust the patient, but he had become pretty well demoralized from loss of sleep during the days of his illness and from the fact that he had been unable to swallow but scanty nourishment.

It was now evident that something more radical must be done, for we were unable to feel any security against the occurrence of

what might be an alarming hemorrhage. It was impossible to locate the exact source of the bleeding, and the sloughing condition of the entire area affected rendered any plan of suturing out of the question. The question of ligating one of the large arteries came up, and Dr. Charles N. Dowd, a general surgeon of our city, was called in. After a full consideration it was decided to ligate the common carotid artery on the side involved, and this was done about 9 A. M. on the morning of Feb. 9th. The patient's habits with reference to alcohol made the giving of an anaesthetic a very difficult procedure, but it was carried out in a masterly manner by Dr. Thomas L. Bennett. Chloroform was used, and after the man was fairly under its influence stiff rubber tubes were passed through the nares, their proximal ends loosely wrapped with gauze on which the chloroform was poured and the pharynx stuffed with gauze. This prevented the leakage of blood down the throat during the ligation of the artery. The latter was done by Dr. Dowd without incident. Two ligatures were passed, one with single strand and one with double, about half an inch apart. As soon as they were tightened the bleeding ceased and did not return at all. The patient made a good recovery after operation. There was no disturbance of cerebration, though he complained of considerable neuralgic pain all over the head and great difficulty in sleeping at night. These symptoms were combatted with the usual remedies, and the man made a perfect recovery. About three weeks after the operation a pulse was palpable in the left temporal region.

A careful and prolonged search through the literature of this subject has brought to light fifty-one cases which may properly be included under the title selected for this paper. As is always the case in a collation of cases spread over a long series of years, classification is somewhat difficult, as every reporter has followed his own ideas in setting forth the particulars of his individual case or cases. It seems logical to make of the material here presented the following subdivisions:

*Group 1.* Spontaneous opening of the abscess, with immediate hemorrhage.

#### A.—FATAL CASES.

Caytan, male, 38 years; no operation; ulceration of the internal carotid.



Grisolle; no particulars given; no operation; ulceration of the internal carotid.

Lefort; no particulars given; no operation; bleeding believed to have come from the internal carotid.

Méry; no particulars; no operation; abscess led to ulceration of the internal carotid.

Macgregor, male, 20 years; no operation; tumor appeared in right side of neck, running down to the mastoid process; autopsy revealed a circumtonsillar abscess communicating with the internal carotid by an opening the size of a small bougie.

Monod, male, 38 years; no operation; probable ulceration of internal carotid.

Norton, female, 4 years; no operation; incision of abscess was refused by parents; autopsy revealed ulceration of internal carotid.

#### B.—RECOVERIES.

Dunn, child,  $3\frac{1}{2}$  years; bleeding immediate and recurred twenty-four hours later; cervical haematoma; ligation of common carotid, after which tumor gradually decreased in size; bleeding was seen to come from the posterior faucial pillar.

Luke; no particulars; bleeding immediate and recurrent; ligation of common (?) carotid.

Messiter, male, 24 years; immediate bleeding, profuse and recurrent; initial hemorrhage on fourth day of a quinsy and recurrence on the nineteenth day; ligation of common carotid.

Pircher, male, 22 years; bleeding immediate and recurrent; ligation of common carotid; definite statement that no bad effects followed the operation.

Summary of Group I—II cases, 7 fatalities and 4 recoveries.

*Group 2.* Spontaneous opening of the abscess; secondary hemorrhage.

#### A.—FATAL CASES.

Bourget, male, 20 years; bleeding recurrent; no operation.

Brewer, male, 25 years; small immediate and later recurrent hemorrhage; no operation; an effort was made to secure pressure against the posterior surface of the soft palate by means of a small roll of gauze to which were attached tapes coming out through the nares; arrest of hemorrhage for a while, but later fatal recurrence; supposed rupture of a small abscess on the posterior surface of the soft palate.

Clayton; no particulars; no operation; ulceration of internal carotid.

Hall, male, 26 years; bleeding came on two hours after the abscess burst; fatality probably due to rupture of a carotid aneur-

ism, as a few minutes previously the patient had complained of a "balloon-like" feeling in his throat; no operation.

Müller, male, 44 years; recurrent pus and blood; no operation; autopsy showed facial artery thinned and ulcerated cavity full of pus at bottom of left tonsil.

Reeves; few particulars; hemorrhage same day as opening of abscess and recurrent; operation proposed but refused.

Vergeley, male, 34 years; blood and sphacelus on bursting of abscess and again a few hours later; operation refused; autopsy showed in retro-tonsillar space a cavity the size of a pigeon's egg, full of clots and sphacelus; internal carotid trunk cleanly severed about two inches from bifurcation of the common trunk.

Walker; few particulars; hemorrhage forty-eight hours after abscess burst; tumor formed along right side thyroid cartilage, communicating with abscess; no operation.

#### B.—RECOVERIES.

Ehrmann, male, 22 years; hemorrhage on the eighth-ninth day; ligature common carotid; supposed lesion of internal carotid.

Moizard, male, 40 years; large hemorrhage in twenty-four hours, causing syncope, which checked bleeding; clot seen in situ in vessel wall; no operation; ether, ergotin hyperdermically, rest and carotid compression.

Marotte, male, 30 years; hemorrhage in forty-eight hours; no operation; large doses of quinine given for some days.

Postempski, male, adult; hemorrhage twenty-four hours after bursting of abscess; ligature of common carotid artery; suspected lesion of internal carotid.

Stoupy, male, 22 years; hemorrhage on eighth day; ligature of external carotid; massive inflammation had appeared in region of sub-maxillary gland; incision for ligature encroached on this area and gave exit to considerable clotted blood, and in spite of ligature external bleeding continued for a while, but patient eventually recovered.

Veillard, male, 26 years; recurrent hemorrhage some hours after abscess burst and recurrences; no operation; ice and rest.

Weinlechner, male, 37 years; hemorrhage two days after abscess opened; ligature of common carotid, with immediate recovery, but evidences of disturbed muscular power appeared on the opposite side of the body; in six weeks this had nearly disappeared; later death from apoplexy.

Summary of Group 2—15 cases, 8 fatalities and 7 recoveries.

Group 3. Abscess opened by incision; immediate hemorrhage.

#### A.—FATAL CASES.

Duke, male, 32 years; pulsatile tumor had appeared in tonsillar region; ligature of common carotid immediately stopping

pulsation; ligature came away on the sixteenth day, and on the twenty-first day the man appeared perfectly well and was dismissed; one week later he went on a debauch and had a fatal hemorrhage.

Lebram (second case), male, 2 years; no operation; bleeding also from ear.

Fehleisen, male, 34 years; after incision clots were turned out of abscess cavity, but threatening dyspnoea necessitated tracheotomy, during which patient died; swelling had appeared at angle of jaw following angina, with fluctuation over it; internal structures were pressed toward middle line; no bruit or pulsation in pharynx; carotid vessels were found lying in purulent foci and perforated at bifurcation.

Liston; incomplete report; ligation of common carotid, which did not control hemorrhage.

#### B.—RECOVERIES.

Lebram (first case), female, 9 years; hemorrhage from pharynx, nose and ear; ligature of common carotid; hemiplegia followed ligature.

Long; incomplete report; free bleeding followed incision; later patient was found choking and was resuscitated with great difficulty; no operation.

McBarron (case reported in this paper), male, 55 years; hemorrhage immediate and recurrent; arterio-sclerosis; ligature common carotid.

Summary of Group 3—7 cases, 4 fatalities, 3 recoveries.

Group 4. Abscess opened by incision; secondary hemorrhage.

#### A.—FATAL CASES.

Pitts, male, 39 years; hemorrhage twenty-four hours after incision and recurrent; ligation common carotid; autopsy showed left tonsil replaced by cavity and the internal face of the internal carotid bathed in pus, with a perforation the size of the tip of the little finger.

Watson; incomplete report; hemorrhage forty-eight hours after incision; ulceration of lingual artery found.

(In this category it seems proper to include two cases reported by Kaplan, although they did not present the usual clinical features of an ordinary quinsy. The incisions in these cases were made externally, but both cases have been reported as hemorrhage due to circumtonsillar abscess. The surgical conditions, and especially the problems they offered, make this classification here followed logical. For a summarized history of the cases taken from their original source of publication in one of the Russian journals I am under obligations to Dr. S. M. Jacobs of New York.)

Kaplan (first case), male, 22 years; a fluctuating swelling the size of an egg appeared at the angle of the jaw externally, tonsil red, and inflamed; incision revealed a deep pus cavity leading toward throat; wound did well for thirteen days and then bled most profusely; ligature of common carotid; paralysis of arm and leg of opposite side; ten days later paralysis much better, but patient lost consciousness and died with slight convulsion of paralyzed side; hemorrhagic pleurisy.

Kaplan (second case), male, 19 years; tumor at angle of jaw; incised and pus found burrowing toward pharyngeal wall; same day cyanosis choking; tracheotomy, with temporary improvement; forty-eight hours later sudden hemorrhage from wound, nose and mouth; ligature of common carotid immediately checked it; patient regained consciousness, but soon became comatose and died; perforation found in internal carotid.

#### B.—RECOVERIES.

Bobone: incomplete report; hemorrhage the day following incision.

Chappell, male, 27 years; hemorrhage fourth day and recurrent, controlled by packing; no operation.

Gleitsmann, male, 24 years; hemorrhage eighth day and recurrent; styptic packing controlled; no operation.

Schmiegelow, male, 30 years; hemorrhage fourth day; ice, rest and morphine controlled; probable lesions of a twig of the tonsillar or ascending pharyngeal artery.

Summary of Group 4—8 cases, 4 fatalities and 4 recoveries.

As far as the reports show all of the cases in the foregoing groups were quinsies. Additional cases have been reported which may be grouped as follows:

*Group 5.* Hemorrhage following retro-pharyngeal abscess.

Carmichel, male, 5 weeks; immediate hemorrhage following spontaneous opening of abscess; no operation; death; abscess found to connect with posterior tonsillar cavity; bleeding from a branch of the external carotid.

Clutton, male, 28 years; spontaneous opening of abscess, with immediate and recurrent hemorrhage; ligation of external and internal carotid arteries; recovery; blood thought to have come from the internal jugular vein; abscess appeared to focus above right tonsil.

Erichsen; incomplete report; phlegmon in pharynx discharged pure pus on seventh day; fatal bleeding from abscess cavity; autopsy showed abscess bordering wall of internal carotid, which had perforated.

Franklin, male, 8 years; spontaneous opening of abscess; no operation; recovery; pulsating swelling was evident on outer side

of neck; evidences of general sepsis; subsidence of external swelling; aphonia not clearing up for several months; long and tedious convalescence; one year later slight weakness of arm and pes equino-varus; probable embolus of middle cerebral artery, a terminal branch of internal carotid.

Chassaignac; incomplete report; immediate hemorrhage following incision; ligation of common carotid; recovery; probably false aneurism due to carotid erosion.

Summary of Group 5—5 cases, 2 fatalities and 3 recoveries.

*Group 6.* Hemorrhages in connection with scarlatinal suppuration.

Immermann, male, 17 years; spontaneous opening of abscess; sudden buccal hemorrhage; no operation; death; deep tonsillar abscess found, which had perforated carotid; much blood in bronchi.

Pepper, male, 30 years; spontaneous opening, recurrent hemorrhage; ligation common carotid artery; recovery; blood regarded as having come from internal carotid; left vocal cord paralyzed before operation.

Mahomed, in discussing Pepper's paper, reported six cases without particulars except that some were fatal. From lack of data these cases are not included in this summary.

Lyot and Petit, female, 19 years; elastic swelling suddenly appeared in left mastoid region with systolic bruit; left tonsil pushed toward median line and compressible; puncture externally withdrew blood; spontaneous subsidence; no internal bleeding; recovery.

Summary of Group 6—3 cases, 1 fatality, 2 recoveries.

*Group 7.* Hemorrhages in connection with gangrenous tonsillitis.

Craigin, male, 45 years; spontaneous opening and immediate hemorrhage; no operation; death; right tonsillar space and posterior pharyngeal wall showed partly detached slough, exposing open mouths of two small arteries, possibly twigs of ascending pharyngeal.

Terganowan, male, 20 years; spontaneous opening two or three months after initial tonsillar symptoms; no operation; death from hemorrhage; autopsy showed complete destruction of parotid gland and gangrenous softening of entire circumference of external carotid, with fistulous opening into throat.

Summary of Group 7—2 cases, both fatal.

Two other cases have been reported under the heading of hemorrhage from circumtonsillar abscess, but they do not seem to the writer to belong to this category.

Wulf, female, 6 years; suspected aneurism of internal carotid, but stopped by compression of the external trunk; ligation of

common trunk, with cessation of pulsation and bruit in pharynx; recovery; sac filled up with blood clot, but later suppurated and puncture then gave fluid blood; eventual recovery.

Savory; apparently a case of abscess in the neck, involving internal structures with external hemorrhage; no operation; death.

Excluding the last two cases, we have then fifty-one well authenticated cases of hemorrhages dependent on suppuration in connection with pharyngeal structures. Of this number twenty-three recovered and twenty-eight died, a mortality rate of 54 per cent. These figures show the gravity of this complication. Sex is stated in thirty-nine, of which number thirty-five were males and four females. This fact is of no special significance, as circumtonsillar and other pharyngeal abscesses are more common in males. Age is given in thirty-seven cases. Seven occurred in the first decade of life, five in the second, eighteen in the third, six in the fourth and one in the fifth. The maximum and minimum ages were fifty-five years and five weeks, respectively.

Operations were performed as follows: Ligation of the common carotid, sixteen times, with eleven recoveries and five deaths; of the external carotid once, with recovery, and of the common trunk with both branches, once, with recovery.

As regards the result of ligation on brain function, no statement is made in eleven of the sixteen cases of ligation of the common trunk; in three cases paralysis followed and in two it is definitely stated that no bad effects followed. No statement with regard to this point is made in the case of ligation of the external branch, nor in the case of ligation of the common trunk and both its branches.

Autopsy records are given in thirteen cases. In two the bleeding came from the external carotid in 2, ascending pharyngeal in 1, "carotid" in 1, lingual in 1, at the bifurcation in 1, facial in 1. In 9 cases in which no autopsy was held the blood was supposed to come from the internal carotid and from the internal jugular vein in 1.

Many interesting questions suggest themselves in connection with this class of cases. We have time to consider but one or two of them.

The first relates to the invasion of arterial structures by suppurative processes. That this does happen is now universally be-



lieved, but we must remember that until comparatively recently it was a mooted point. It was but forty years ago that Bouchard observed that while an artery in a purulent focus did sometimes rupture, he was not prepared to admit that the latter accident was referable to an ulceration in the proper sense of the latter term. In 1873 Cauchois stated that such rupture occurred as the result of the following pathological sequence: First, denudation of the artery by the destruction of the cellular wall; second, suppurative inflammation of the external tunic; third, disassociation and disappearance of the muscular fibres of the middle coat, and, fourth, rupture of the external tunic. In 1878 Ehrmann called attention to the patient's general condition as bearing on vascular rupture in pharyngeal suppuration, but did not admit the existence of any diathetic condition as indispensable to the rupture. In 1888 Monod noted that one of the most constant effects of arterial inflammation is the disappearance of the muscular tunic. The muscular and elastic fibres, he says, give place to connective tissue which often unites itself with that of the external tunic and the vegetations of the internal tunic. There results, consequently, a weakening of the vessel wall. Often the external tunic hypertrophies and so supplements the deficiencies of the middle.

Now all these processes are apt to assume a peculiar virulency in the region under consideration, owing to its proximity to the air passages. The mouth is a hotbed of all sorts of bacterial life, and the parts are constantly flushed with the air of inspiration and expiration. Pus near the air passages rapidly decomposes. The fetid characteristics of mouth abscesses and their virulence are well known. Hence the pus and gas confined in the pharyngo-maxillary space cause a rapid death of the arterial wall before any compensating hypertrophy of the latter can be brought about by nature's conservative processes.

Granting the existence of a weakened vascular wall, we have further to recognize the presence in some cases of adjuvant causes of rupture, such as cough and violent muscular effort which momentarily increase the pressure in the vessel. These patients are especially liable to cough from the irritation of the initial blood which trickles down the pharynx, and a slight leakage may become a formidable rent. Another cause following the incision of the abscess is the sudden diminution of pressure on the

outside of the vessel wall produced by the accumulation of pus under pressure, which acts as an elastic cushion. The weakened vascular area is thus between the internal force of the blood pressure and the external force of the pus sac. If the latter is suddenly lessened by incision the sudden unbalancing of pressure is too great for the further resistance of the weakened area, and it gives way. Here arises a point of great significance for the surgeon, for the hemorrhage which immediately follows incision is apt to be referred by the patient or his friends to unskilful intervention. Such was the experience in the case reported in this paper. The patient had been having quinsies for over thirty years. They had been opened repeatedly. No hemorrhage had followed. All this long time his habits in regard to consumption of alcohol had been leading up to a condition of distinct arteriosclerosis. In the last attack the vessel—whichever one it was—had become vulnerable, and when the escape of pus removed the counter-pressure on the vascular wall it gave way. In a sense, it is true, the surgeon does cause the bleeding, but how much better that it should occur under such conditions than that the destruction of the vessel wall should increase in extent and the rupture of the abscess come on at a time when no relief is at hand and an immediately fatal hemorrhage may follow. In those cases in which the bleeding has been controlled by various means we must assume the formation of a thrombus which acts as a plug, and which later becomes organized permanently, stopping the leak.

Even in the case of an aneurism (of which more later), if the opening is not large it may be closed by a thrombus. At this stage incision is far less dangerous, even if after the emptying of the abscess cavity the thrombus becomes loosened and bleeding recurs. It is possible for the thrombus to involve the contents of the aneurismal sac and become organized. Under these circumstances spontaneous healing occurs. (Case of Lyot and Petit.) The chances of such a favorable outcome, however, are lessened if the thrombus is surrounded by pus. The occurrence of a false aneurism depends also on the stability of the abscess wall. If it is friable and ruptures, probably a fatal hemorrhage will occur. The same untoward result may also happen if the abscess has already opened before the arterial wall is perforated.

We have all opened many quinsies, and will open many more. We must not forget that some of the most severe and even some of the fatal cases of hemorrhage have come like the thunder clap out of the clear sky. Patients who have presented only the signs and symptoms of an ordinary quinsy have died from this complication. Naturally, therefore, we are faced with this question: Leaving out those instances in which a swelling in the throat, with pulsation and bruit suggesting aneurism, gives us a definite course to pursue, are there any signs or symptoms which will advise us that we are confronted with the gravest of dangers? In other words, what quinsies are harmless and what potentially harmful? This aspect of the matter has received the careful attention of Lebram, who discusses with considerable detail the matter of differential diagnosis of swellings in the tonsillar region. We are called upon to decide between an inflammatory process, a tumor of the tonsil with metastases in the cervical glands, and aneurism. Decision is further complicated by the fact that carotid aneurism sometimes presents distinct evidences of inflammation, suggesting a quinsy. Certain aural lesions are accompanied by an extension of suppuration down into the circumtonsillar region. An abscess may form and a pulsation be communicated thereto by some neighboring vessel, though the latter itself is intact. Syphilitic processes with glandular involvement rarely confuse us. The question then reverts to the possibility of involvement of an artery. If the abscess has already opened, has blood appeared or can clots be turned out of the cavity? Autopsies have shown that hemorrhage in these cases may come from the internal carotid, a branch of the external carotid, the palatine branch of the ascending pharyngeal, the lingual or the inferior palatine branch of the facial. If incision evacuates clot and fluid blood escapes later in greater or less quantity, we must regard it in the highest degree probable that we are dealing with hemorrhage from the carotid, which has led to the formation of a false aneurism. Even with unruptured abscess wall, inspection, palpation and auscultation would enable us to make a diagnosis if they were uniformly present, but, unfortunately, they are not, as the case of Fehleisen clearly shows. Under such conditions compression of the artery above and below the abscess would make a difference in the physical characteristics

of the latter. It has been found that when inflammation develops over an aneurism pulsation and other usual features may be lacking. Under these circumstances we assume a partial or complete clotting of blood in the aneurismal sac. The general feel of the parts would not differ from that of an abscess. We would find merely a softness and indefinite fluctuation. Some cases of aneurism have presented a tumorlike swelling of the cervical glands.

A point of anatomy mentioned by Chassaignac should be borne in mind. He states that after middle life the internal carotid at this level describes a curve with its convexity directed inward. (Cf. paper by Connal, *Jour. Laryngology*, March, 1908, on "Abnormal Pulsating Vessels in the Pharynx.")

Extra-cranial aneurisms of the carotid of traumatic origin are rare. Lebram found only three instances recorded.

Further factors to be taken into account are advanced age, gout, arterio-sclerosis and lues. Growth of an aneurism under such circumstances would be slow and gradual, while aneurism caused by suppurative erosion develops rapidly.

In regard to diagnosis by needle of swellings in this locality, we are left in doubt if the swelling contains thick pus or clotted blood. A suspicion of aneurism would, of course, negative a diagnosis by puncture. If a tumor has appeared in the neck and we suspect aneurism we may excise a piece of the former for submission to the microscope.

The brief notes in the cases reported show that hemorrhage has been checked by or has followed the use of ice, rest, morphine, packing, etc. Previous to opening a suspicious swelling in a case of severe bleeding, the artery may be exposed as a provisional measure. Experience seems to show that in these severe cases the safest plan is to ligate the common carotid. But rarely can we determine the bleeding point. This fact is Clutton's justification for advising the ligation of the common trunk together with its two terminal branches. It may be asked, Why ligate the common trunk and not its external branch? It is now considered the correct procedure in post-tonsillotomy hemorrhage requiring ligation to select the latter, but it must be remembered that different local conditions prevail in the latter emergency. Pepper states that the ascending pharyngeal, which is undoubtedly the

source of the bleeding in some of the quinsy cases, comes off from the bifurcation of the carotid, and ligation of the external branch, therefore, would not help us. The anatomies, however, state the origin is not at the bifurcation, but half an inch above. Moreover, ligation of this branch would lead us into sloughing tissue and its efficacy would be very doubtful. We are in a very different environment here from that of the non-suppurating tissue of an ordinary tonsillar hemorrhage. Pitts declares that ligation of the common trunk alone is not safe, as anastomosis is very common through the branches of the external. Morris expresses considerable doubt as to the possibility of the anastomosing flow through the arteries within the skull ever reaching down as low as the origin of the internal carotid in the neck. While traumatic hemorrhage in this region generally involves the external carotid or ascending pharyngeal, a large repeated hemorrhage secondary to abscess is far more likely to involve the internal. Under these circumstances, if Morris is right, ligation of both common and external would prevent returning hemorrhage. The dangers of ligation of the common are not to be underestimated, but this procedure relates to the casuistry of general surgery and need not be discussed here.

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 Since this paper was finished another article on the subject



has come under the author's notice, viz., that by Nettebrock (*Zur Casuistik der Blutungen nach Tonsillotomie und ihre Behandlung*: Inaugural Dissert., Kiel, 1906). This writer reports two cases:

Case I.—Angina incised in a man of thirty years, with slight bleeding which recurred later and was controlled by tampon.

Case II.—Tumor formed in the region of the tonsil and was incised on the fifth day of throat symptoms on the supposition that it was a quinsy. Fever was lacking and no pulsation was made out. Incision was followed by escape of blood but no pus. Bleeding recurred, requiring ligation of common carotid and tracheotomy. This proved sufficient and the tumor gradually disappeared. Its exact nature was not determined. Some hemiplegia of the tongue followed ligation, but this soon disappeared.

### *Discussion.*

Dr. W. E. Casselberry.—In an emergency as described by the essayist the main question lies between ligation of the common carotid and ligation of the external carotid artery. He passes lightly over the danger of the ligation of the common carotid artery, but that is the salient point which must influence us in our decision. In one of my patients, a case in which a general surgeon ligated the common carotid artery for the purpose of facilitating the extirpation of a malignant tumor of the pharynx, the patient thereafter was paralyzed on one side; he survived four years, during which time he regained ability to walk but did not regain a practical use of the paralyzed arm. This has happened only once in my practice but I have heard that evidence of lack of circulation in the brain following ligation of the common carotid is not so very uncommon. I have here a sketch of the carotid and its branches, taken from Gray's Anatomy which I have long carried with me to refresh my mind in case of emergency. I observe that the branches of the external carotid do come off very low down but the ascending pharyngeal seems to come off at a working distance above the bifurcation. And unless the ascending pharyngeal happened to branch unusually low it would be included in a ligation of the external carotid, as well as all the other branches which we would need to check. Therefore, it seems to me that ligation of the external carotid would be adequate and this is the practical experience of Jackson, who reports seventeen cases of tonsillar hemorrhage in which he ligated the external carotid with excellent results

and I surely think from this date that we might at least first try it and then if the hemorrhage did not cease, the common carotid could be tried also.

Dr. D. Bryson Delavan:—If I recollect correctly, the literature of the subject ligation of the common carotid has not by any means always been successful; in several instances, at least, on record it has resulted in immediate failure. The ligation of the carotid as described in my paper (*Trans. American Laryngological Assoc.*, Vol. X.), seems to me a very much better procedure. If we may believe what we hear of some of these cases, they have not acted as kindly as that described by Dr. Newcomb. For there has been a gush of blood which has caused the death of the patient in a very few minutes. In such a case where it is possible, the point referred to by Dr. Casselberry is very important.

Dr. W. L. Ballenger:—I have had some experience which may justify me in making a few remarks, although I have never had a hemorrhage requiring the ligation of either the external or common carotid artery. In my experience of the removal of tonsils with the capsule intact, I have learned something about hemorrhages in this region. Some of the hemorrhages that have occurred in the cases reported by Dr. Newcomb may have been due to the technique employed for the evacuation of pus in a tonsillar abscess. I know this has been true in a great many cases. I have learned that in dissecting tonsils if you avoid injuring the musculature of the pillars and of the bed of the tonsillar fossa you will not have severe hemorrhage, whereas if you injure either the muscle of the anterior pillar or the posterior pillar, or injure the superior constrictor muscle of the pharynx, which is the bed of the sinus tonsillaris, you are apt to have very severe hemorrhage. The tonsillar artery is a single branch, just external to the superior constrictor muscle, and as it passes through the superior constrictor muscle, it divides into three or four branches. When the incision is made so as to avoid the superior muscle the hemorrhage is not so severe as if the muscle is injured. If peritonsillar abscess is operated on through the anterior or posterior pillar, without reference to whether you are injuring the musculature, as I have just said, and you happen to cut through the muscle of the anterior pillar, you are liable to have serious hemorrhage; if that muscle is avoided, the artery will not be injured. I have devised a method of operating upon peritonsillar abscess which avoids just this injury, and may, in a good many cases, prevent hemorrhage where the artery is not necrosed. The method I have employed is to separate the anterior pillar from the tonsil just as though going to remove the tonsil and then after separ-

ating it to push the pillar back out of the way and separate it from the superior constrictor muscle and you will absolutely always locate your abscess. You cannot fail, provided you can carry out the technique. Of course the mouth may be so that you cannot do this, but from here you can always open the abscess and you will not injure any of the arteries and this can be broken down by blunt dissection. Even if injured, the arteries are not so liable to bleed, and it seems to me that we should take preventive measures at the time of operation rather than depend too much upon ligation of these very important blood vessels. I hope some present will try this method of operating, it is a clean, accurate surgical procedure.

Dr. J. O. Roe:—I never had any serious hemorrhage on opening peritonsillar abscesses due, doubtless, to the method I have for a long time employed of opening the abscess through the tonsil, thereby avoiding wounding the pillars. As we all know, the danger from incising a tonsil is in injuring the vessels of the pillars, the glosso- and palato-pharyngeal arteries. The former being the one usually injured when serious hemorrhage occurs.

In his book on "Rest and Pain," many years ago John Hilton described his method of opening deep abscesses with a trocar and scissors. By thrusting the trocar deeply into the tissues and then introducing a pair of sharp pointed scissors along a groove in the trocar, a wide opening is made that will give free exit to the pus. In this manner he never encountered any hemorrhage or other trouble in opening deep abscesses.

For the opening of peritonsillar abscesses I modified this method by dispensing with the trocar and using a pair of sharp pointed angular scissors. In opening the abscess with the scissors the point of the scissors is thrust into and through the tonsil and the scissors opened, the scissors are then closed and thrust in a little further and again opened and so on until the abscess cavity is reached. In this manner we avoid any cutting and at the same time an opening is made that does not readily close, sufficiently large to give free escape of the pus and drainage of the cavity.

Dr. J. E. Newcomb:—The closing sentence of this paper reads, "The dangers of ligation of the common carotid are not to be underestimated but this procedure relates to the casuistry of general surgery and need not be discussed here." The point that I desired to bring out was this, that if we are going to consider the question of ligating the common carotid artery, it is a question we should approach not simply from the standpoint of laryngologists but from the standpoint of the general surgeon as well, because any conclusions as to the ultimate danger which might result from ligation of such a large vessel, must

obviously include ligations made for all causes. Now in so far as this paper represents conclusions they are from material here collected. My own experience is confined to this one particular case. What Dr. Casselberry has said is, of course, absolutely true and in this paper, in the series of cases reported, it is very frankly stated that in some of them the untoward result to which he made reference did happen. Thus, for instance, in the case reported by Lebram, it is distinctly stated that hemorrhage followed ligation. In another case reported by Stoupy it is said that ligation of the external carotid was made, and in spite of this ligation, the bleeding continued for a while but the patient eventually recovered. In still another case ligation of the common carotid brought on severe hemorrhage which was recovered from, but evidence of impaired muscular power was found on one-half of the body.

If you care to go into these things in detail you will see that there is a considerable number of cases, as verified by autopsy, in which ligation of the external carotid would not have met the necessity because it was the internal carotid that was severed as the post-mortem showed. I do not mean to take the position that the operation for these cases is invariably ligation of the common carotid, but the experience set forth in the material here reported would go to show that it probably met the necessities of the various cases better than ligation of any other vessel would have done. This subject is a very interesting one to me and I was moved to go into it in detail in the summaries of these cases with the thought in mind that it would probably save somebody else a good deal of trouble in looking the matter up.

## THE FORM OF THE HARD PALATE.\*

BY HARRIS PEYTON MOSHER, M.D.

The subject of this paper is the form of the hard palate. I shall discuss it under the following headings:

- 1.—The four types of the hard palate.
- 2.—The normal form of the palate.
- 3.—The part played by the alveolar process in making the normal arch.
- 4.—The causes of variations from the normal form, which are:
  - a.—Mal-occlusion of the teeth.
  - b.—Unequal growth between the parts of the septum.
  - c.—Low descent of the antra.
  - d.—Faulty shrinkage and faulty readjustment of the pre-maxillae.
  - e.—Asymmetry of the halves of the palate.

The most important, and so the central idea of the paper is that asymmetry between the halves of the palate is due not always to asymmetry of the head as a whole, but is due nearly as often to asymmetry confined to the bones of the face. The teeth are the chief cause of this asymmetry of the face. Accompanying the inequality in the two sides of the palate there is almost always a vomer spur and a deviation of the septum.

Asymmetry between its halves is one of the most important variations in the form of the palate. Since in fifty per cent. of the cases this is part of a general asymmetry of the head, it is necessary by way of introduction to refer briefly to the subject of asymmetry of the skull as a whole.

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\*The specimens which furnish the foundation of this paper are from the Laboratory of the Anatomical Department of the Harvard Medical School. The clinical cases, the impressions of the palate and the X-rays are from the Throat Department of the Massachusetts General Hospital.

For help in making the casts and impressions of the palate and the brow, I here express my thanks to Dr. Isabella D. Kerr.

The illustrations were drawn by the writer.

ASYMMETRY OF THE SKULL

The most frequent irregularity in the form of the skull is a want of symmetry. Probably no skull is perfectly symmetrical. The condition which has been observed to exist most frequently with irregular forms of skull is premature closure or obliteration of certain sutures. The cranial bones increase in size mainly at

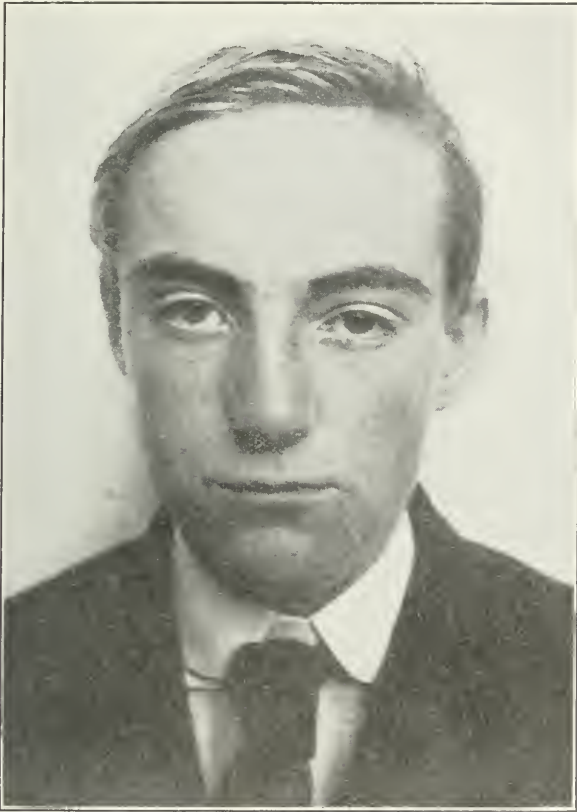


Fig. 1.

Congenital occlusion of the right choana, resulting in failure of full development of the right side of the head and face. (Courtesy of Dr. Coolidge.)

their margins, and when a suture is prematurely closed, the growth of the skull in the direction at right angles to the line of suture is checked and increased growth takes place in other



directions in order to supply the defect. For instance, in cases of obliteration of the sagittal suture the transverse growth is prevented and compensatory growth takes place in a vertical direction, resulting in the scaphoid or boat-shaped skull. Similarly, in obliteration of the coronal suture compensatory growth takes place mainly upwards. Oblique deformity of the skull



Fig. 2.

Congenital occlusion of the right choana. Side view.

occurs with premature closure of one-half of the coronal or lambdoid suture. A like deformity may be produced by rickets or wry-neck.

Not only may premature closure of sutures cause asymmetry of the skull but pressure artificially applied in early life may do so. This is best shown in the skulls of Flat Headed Indians. These tribes had the well known custom of compressing the

heads of their children by an apparatus of boards and bandages. Distortions likewise occur in long buried skulls which have been subjected to pressure and moisture. Asymmetry is illustrated in a slighter degree by individual instances where undue pressure has been employed unintentionally (Quain, Vol. II., Part I., p. 85).

DIFFERENCES IN THE SKULL ACCORDING TO AGE.

In order to properly appreciate asymmetry of the growth in the



Fig. 3.

Photograph of a case of Asymmetry of the Palate. The right side of the palate is higher than the left. See figures 4, 5, 6, 7.

skull it is necessary to bear in mind the normal periods of growth and to know the differences found in the skull according to age. The most striking fact is that at birth the head is chiefly skull and

but little face. At birth the face is scarcely an eighth of the bulk of the cranium, whereas in the adult it has risen to one-half. In the first seven years the skull grows rapidly so that during this time certain parts of it, namely: the circumference of the occipital foramen, the body of the sphenoid, and the petrous portion of the temporal bone have attained practically



Fig. 4.

Photograph of a case of Asymmetry of the Palate. Side view of the case shown in Fig. 3.

their adult size. The other regions increase but little until the period of the second dentition. With the coming of the second teeth the accessory sinuses begin to enlarge and in consequence of this the face begins to grow. The growth of the face is

due, in part however, to the increased height of the nasal fossæ, to the enlargement of the alveolar arches and the growth of the teeth. By puberty the difference between the size of the skull and the face has, in a great measure, disappeared. X-ray plates show that the sinuses can be of adult size at puberty.

In old age the skull becomes lighter and thinner. Often it is a little smaller. In some cases, however, it increases in thick-



Fig. 5.

Impression in modelling compound from a case of asymmetry of the two sides of the palate. In this instance the right side of the palate is higher than the left. See figures 3, 4.

ness and weight owing to the deposit of bone on the interior of the brain case. The proportion of the face to the skull is diminished by the loss of the teeth and the absorption of the alveolar processes of the jaws. In consequence of this the upper jaw recedes and the lower jaw projects. (Quain, Vol. II., Part I., p. 82.)

## HOW ASYMMETRY OF THE SKULL IS SHOWN.

Asymmetry shows itself in the face by unequal width of the two halves, by unequal height of the eyebrows and by deviations of the nasal bones and of the nose. In the internal structure of the face asymmetry appears in unequal size of the antra, in in-



Fig. 6.

View from above of the impression figured in plate No. 5. Notice that on the right side, which is the side which corresponds to the higher half of the palate, the dental arch has lost its normal curve and has become almost a straight line.

equality of the nasal fossæ, and in the unequal height of the two halves of the hard palate.

In many cases the asymmetry includes each half of the head as a whole, in other cases, casts show that the lack of parallel

growth is found only in the bones of the face. My observations go to show that asymmetry confined to the bones of the face is common, and but a little less so than asymmetry of the head as a whole. When you stop to consider the conditions which can cause asymmetry of the face, you find that there are more which have to do with the face than have to do with the skull. The development of the face depends upon the expansion of the accessory sinuses, and the development of the sinuses depends upon the teeth. The face is made for the teeth and by the teeth.



Fig. 7.

Tracing from an X-ray of a case of Asymmetry of the Palate. The plate is looked at from behind. The right side of the palate is higher than the left. In this instance there is no asymmetry of the skull. Figures 3, 4, 5, 6, 7 are from the same case.

There are two sets of teeth and the bones of the face are concerned with the development and eruption of these two sets for the first twenty years.

I wish now to show an example of each form of asymmetry of the skull.

I.—Asymmetry of the two halves of the head (see Figs. 1 and 2).

Figure 1 is a photograph taken from a boy of seventeen who had



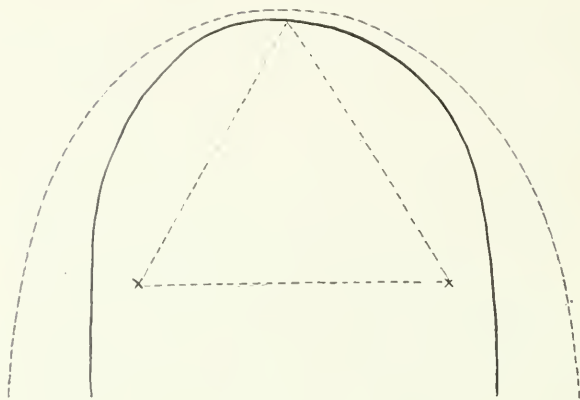


Fig. 8.

The dark line represents the dental curve of the normal palate. A palate which has this curve is classed as Type No. 1. The dotted line represents diagrammatically the wide form of palate, or Type No. 4.

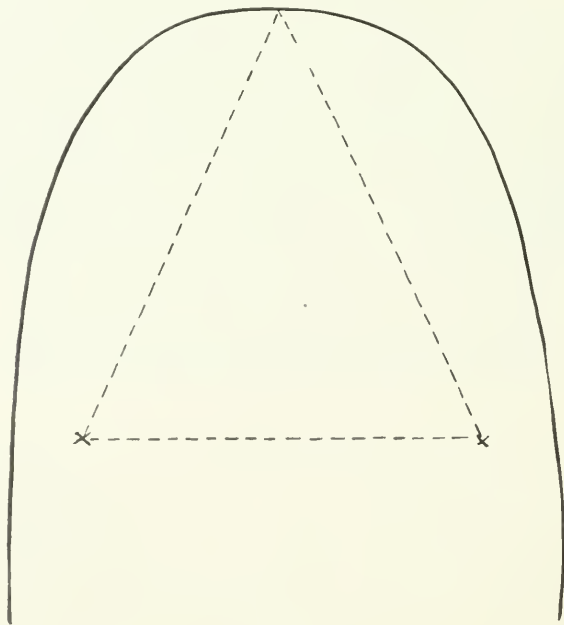


Fig. 9.

Type No. 2. The Quadrilateral Palate.

congenital occlusion of the right choana. He is a patient of Dr. Coolidge. The picture shows that the right half of the head and the right half of the face are smaller than the left half of the head and face. A cast made of the brow and one made of the occiput show the same difference between the right and left sides of the head, as are shown in the photograph. An impression of the hard palate shows that the right side is higher than

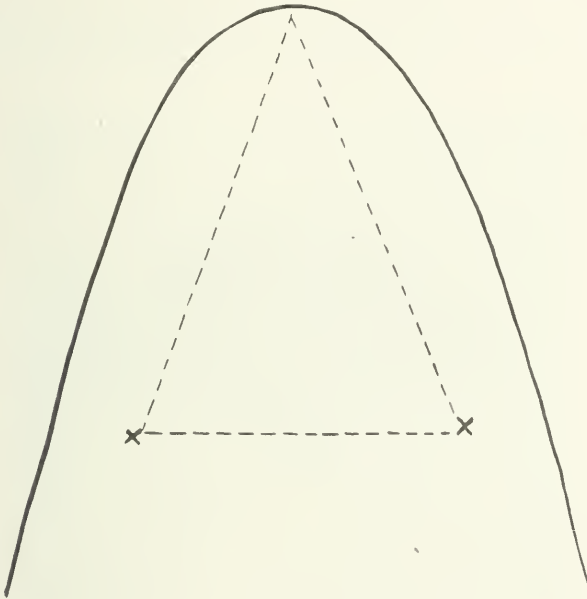


Fig. 10.

Type No. 3. The V-shaped or Angular Palate.

the left. The septum is deviated to the right with a marked vomer spur on the left.

2.—Asymmetry of the two halves of the face.

In this case the right half of the face is less developed than the left. (Figs. 3, 4, 5, 6, 7.) The impression of the palate shows that the right half is higher than the left, and the X-ray shows that the right nasal notch is higher than the left. In the cast of the forehead the right frontal sinus is more prominent than the left, but the two frontal eminences are equal. In other words, the asymmetry stops with the frontal sinuses. The frontal

sinus is really a part of the face. It commences to enlarge at the beginning of the second dentition, at the same time that the antrum starts to enlarge, and its growth from now on is intimately related to the growth of the antrum. Usually in asymmetry of the face, the superciliary ridge on the side of the palate which is the higher is more prominent than the other, and the frontal sinus of this side is usually larger than the other frontal sinus.

What I have said about the general subject of asymmetry of the head has been necessary because asymmetry figures so largely



Fig. 11.

Side view of the impression of a palatal arch of a boy of twelve years. Notice how the line of the palate, after reaching its maximum height about a third of the distance back, slopes gently downward for the rest of its course.

in certain important variations of the form of the hard palate. I come now to speak of the form of the palate. In the palate we have to do with two arches, the arch made by the palatal processes of the palate bones and the superior maxillæ and the arch made by the teeth. Both the palatal arch and the dental arch are equally important in determining the form of the palate. Of both there are four types.

## THE FOUR TYPES OF THE DENTAL ARCH.

1.—The curve described by the dental arch (Fig. No. 8) is variable. These variations have been classified according to temperament. This is a rather loose, unscientific method, but it is quaint and expressive, and undoubtedly has a certain amount of truth in it. In the first type the dental arch is well rounded anteriorly, the circle being continued backward through the region of the molars. The distance in a straight line from the centre of the second molar on one side to the centre of the cor-



Fig. 12.

Side view of the impression of the palate from a negro boy thirteen years of age. Notice that the highest point of the arch comes about half way back instead of a third of the distance, as is usually the case. Behind the highest point the line of the palate slopes downward very markedly. The steep slope of the posterior half of the palate is an infantile characteristic.

responding molar tooth on the other side is about equal to the distance from either of these points to a point in the median line between the central incisors. Such an arch is found according to the old phraseology in connection with the sanguine temperament.

2.—The second type of arch is narrower than the first and

is shaped more like the three sides of a parallelogram. It is almost straight across in front and the sides run backward with very little curve. The line between the molars is much reduced in length. The old name for this is the bilious type (Fig. No. 9).

3.—The third type of the arch is the angular. In this the two



Fig. 13.

Front view of a noticeably square form of arch from a young man eighteen years of age.

sides of the arch are straight lines, not curved or bowed, and they almost meet in a point in front. The segment of the arch made by the anterior teeth is much smaller than in the other two types and amounts to but little. The line between the molars is

much shorter than the line to the space between the incisor teeth. The old name for this is the nervous type (Fig. No. 10).

4.—The fourth type of arch is an arch made like the first, of two equally bowed halves. It is simply a wider and larger form of the first type. The arch is well rounded and broad, the segment being that of a much larger circle than any of the other types. The inter-molar line is longer than the molar-incisor



Fig. 14.

Side view of the same arch shown in Fig. 13.

line. The old name for this is the lymphatic type (Bromell, p. 106. Fig. No. 8).

#### THE PALATAL ARCH.

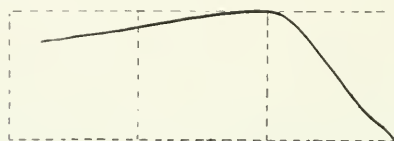
The palatal arch is practically a duplicate of the dental arch. It occurs in the same four types. If the dental arch is reproduced in wire and this wire is placed upright like a wicket, the upper half of the wire would represent the palatal arch and the lower half the alveolar process and the teeth.

#### THE NORMAL ARCH.

Heads group themselves into two classes: the high, narrow head and the short, broad head. In the high, narrow head the arch of the palate tends to be the second or third type, that is,



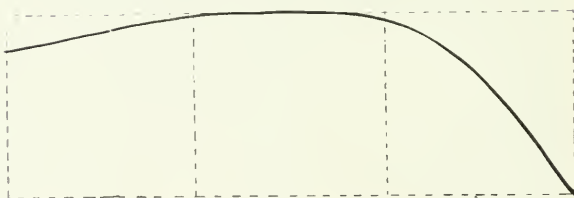
it is high and narrow and has the parallelogram or the acute angle for its geometrical basis. The short, broad head has a wide, flat, equally curved arch, more like type four. The ideal



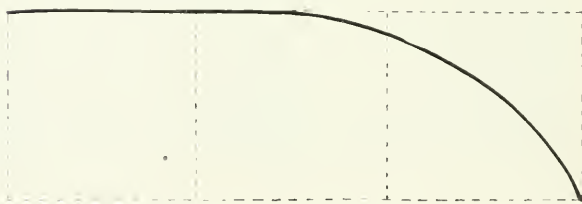
Side view of the palate at birth.



Front view of the palate at birth.



Side view of the palate at twelve years of age.



Side view of the adult palate. These figures were taken from large drawings. The diagrams are all larger than life size.

Fig. 15.

and so the normal, arch is a smaller form of the fourth type. Such an arch is represented most nearly by type one. This type, as you remember, is supposed to be associated, according

to the old and prettily descriptive phraseology, with the sanguine temperament. In a way, however, each of these four types is normal for a certain type of head. (Fig. No. 8).

#### THE PALATE SEEN IN LONGITUDINAL SECTION.

A longitudinal section of the hard palate at birth shows that the median line gradually rises and reaches its highest point about a third of the distance back. From this point it slopes a little downward. This downward slope of the posterior two-thirds of the median line of the palate is found up to ten years of age. In the adult the highest point of the palate occurs between the first and second bicuspid teeth or between the second bicuspid and the first molar. From the highest point the normal adult palate does not slope downward as the infantile palate does, but continues in a horizontal line. The highest point in the arch corresponds in the adult to the anterior limit of the fully developed antrum. In herbivorous animals there is a toothless space between the incisor teeth and the premolars. This gap is not conspicuous in carnivora but it is never wholly obliterated until man is reached. This gap, or the diastema, as it is called, corresponds in man to the highest part of the palate. Roughly speaking, the portion of the palate in front of the highest point is made from the premaxillæ, and the portion of the palate behind is made from the superior maxillæ. If you start with the flat animal palate and trace the changes which have occurred in the palate down to man, you find that one of the greatest changes is the tilting downward of the premaxillæ. The bending downward of the premaxillæ and their proper shrinkage is accomplished, to a certain extent, in the monkeys, but is not accomplished fully except in man (Figs. No. 11, 12, 13, 14, 15).

#### THE PART PLAYED BY THE ALVEOLAR PROCESS IN THE FORMATION OF THE NORMAL ARCH.

In the normal palate the alveolar process is responsible for much of the arch effect. When the teeth are lost and the alveolar process is absorbed, the arch, in a great measure disappears. When the teeth are long and the amount of the alveolar substance about their roots is small, the arch of the palate appears

to be high. In such a palate the roof rests upon the half naked tooth roots as if supported upon stilts, and the lines made by the half-bare tooth roots are clearly seen on the sides of the arch.

The mounding of the alveolar process about the teeth may be compared to the mounding of earth about celery stalks. The higher the earth is piled about the celery the deeper the furrow between the rows. When the celery is pulled up the earth falls in, the ridge which it formerly made is partially obliterated

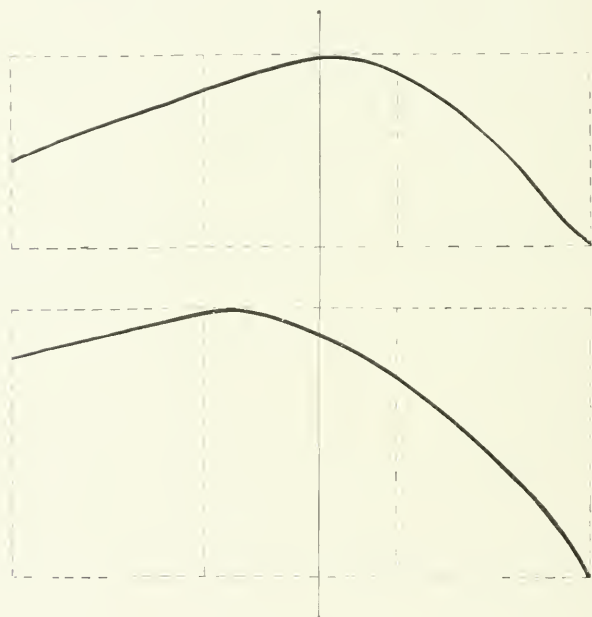


Fig. 15 (Continued).

The upper drawing represents the side view of the palate of a negro boy thirteen years old. The lower drawing represents the side view of an adult negro palate. This palate is a marked example of the V-shaped arch.

and the furrow between any two rows becomes less deep and the sides less steep. The likeness between the behavior of two rows of celery and the halves of the alveolar processes of the superior maxillae and the premaxillae is almost perfect.

In certain animals we have a chance to see how much of the

arch of the palate is made by the alveolar process. The great anteater, as you know, is distinguished by his long cane-line snout and the absence of teeth. The palate of the ant eater is absolutely flat. The palate of the bear with the teeth extracted is flat, except for a slight tendency to arch formation where the alveolar process mounds down to support the very large canine teeth. In herbivorous animals who have large and complicated molars, there is posteriorly a distinct transverse arch to the palate. This is made by the mounding down of the alveolar process about the molar teeth, so that they may be firmly supported for the heavy grinding which they are called upon to per-

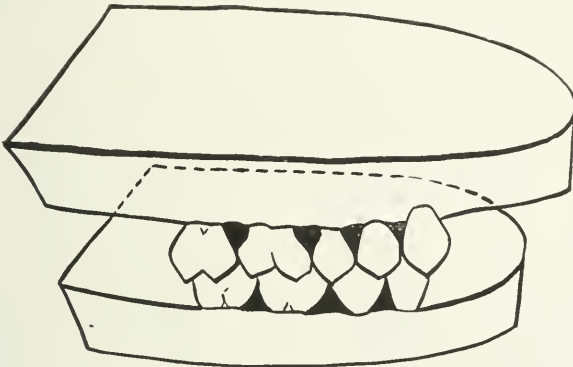


Fig. 16.

Diagrammatic drawing to illustrate the occlusion of the teeth.

form. There is no arch about the anterior teeth. The teeth of a bear and a dog are used mostly for grasping and tearing, and have but little mounding of the alveolar process about their roots, and so the palate in these animals is not arched.

#### THE PALATE AT BIRTH.

There are no teeth at birth, nevertheless the palate has a very marked arch. It would not seem as if the alveolar process could play any part in the formation of this. However, it does, though a bit indirectly. There is no such thing as a toothless baby. Only an adult can be really toothless. At birth the alveolar process is filled to bursting with two sets of tooth germs. Owing to this the alveolar process mounds down into a ridge on either

side of the palate and makes its share of the palatal arch just as much at birth as in the adult. The teeth at birth are inside of the alveolar process instead of outside of it, nevertheless they are responsible for its form (Fig. No. 15).

So far I have described the usual types of the alveolar and palatal arches. I have described the characteristics of the nor-



Fig. 17.

Tracing from an X-ray of a case of V-shaped palate due to narrow nasal fossae and to low descent of the antra.

mal arch and brought out the importance of the alveolar process in making the normal arch. This brings me to the next division of my subject, the causes of variation from the normal type. These, as I stated at the beginning of the paper, are:

- 1.—Mal-occlusion of the teeth.
- 2.—Unequal growth between the parts of the septum.

- 3.—Low descent of the antra.
- 4.—Faulty shrinkage and faulty readjustment of the pre-maxillæ.
- 5.—Asymmetry of the halves of the palate.

I.—MAL-OCCLUSION OF THE TEETH.

The dental arch made by the upper teeth is larger than the dental arch made by the lower teeth. On this account the upper teeth project a little beyond the lower teeth, and the upper in-



Fig. 18.

Tracing from a cross section of the head and face showing a V-shaped palate due to low descent of the antra. In this case the nasal fossae are of normal width, not narrow, as is usually the case when the antra are low and the arch of the palate V-shaped. Tracing seen from behind.

Out of a hundred heads from the dissecting room only three examples of the V-shaped arch were found. Two of the subjects were white and one colored.



cisors overhang the lower ones. With the rest of the teeth each upper tooth fits between or over two lower teeth, articulating with two teeth instead of one. Unless the cusps of the teeth fit accurately there is a wedge-like action between the two sets. When this is excessive it is capable of producing marked deformity. This deformity shows in the alveolar process and in the shape of the arch. It has long been known that the normal

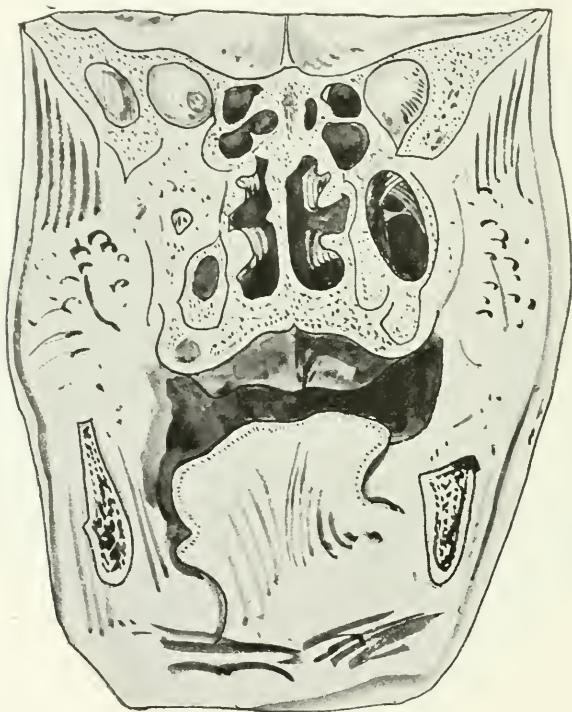
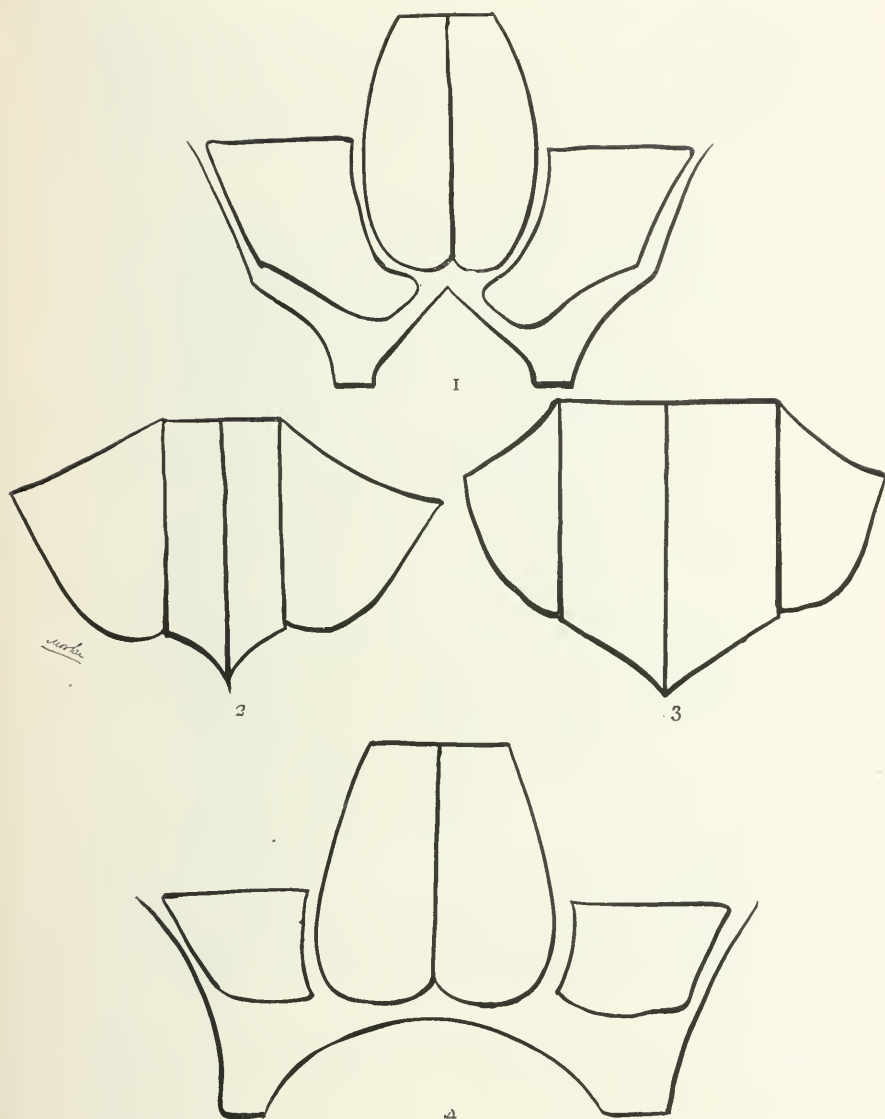


Fig. 19.

Tracing from a cross section of the head and face showing inequality in the size of the antra. The left antrum is much smaller than the right antrum. The antra do not descend below the floor of the nasal fossae, so that their inequality in size has not affected the form of the palate. Tracing seen from behind.

The arch of the palate is flat and wide. This is due to the thickness of the alveolar processes, not to excessive width of the nasal fossae.

growth and placing of the molar teeth depends upon the normal growth of the antra. It has been shown also that the normal growth of the premaxillae depends upon the normal growth of



1. V-shaped palate, due to narrow nasal fossae and to low descent of the antra.

2. Horizontal section of Fig. 1, showing the narrowness of the nasal fossae.

3. Broad, flat palate, due to wide nasal fossae and to small high antra.

4. Horizontal section of Fig. 3, showing the width of the nasal fossae.

Fig. 20.

the incisor teeth. Mal-occlusion is one of the most common deformities of the teeth, and so one of the most important methods of changing the form of the hard palate (Fig. No. 16). The formation of the hard palate, therefore, is dependent in a very great measure upon the teeth.

## 2.—LOW DESCENT OF THE ANTRA.

Where the arch is high and pointed, in a majority of cases the X-ray shows that the antra descend well below the floor of the nose into the alveolar process. Normally the antrum descends from an eighth to a quarter of an inch below the floor of the nose, but in cases of high arch the floor of the antrum may be

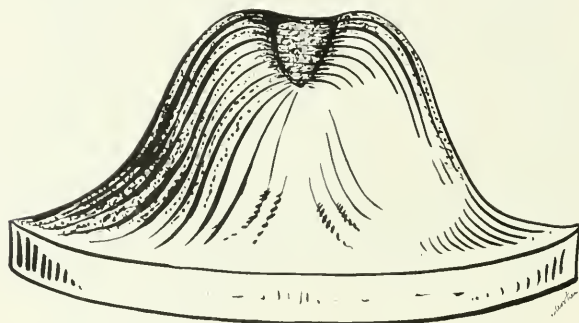


Fig. 21.

Diagrammatic drawing of an impression of a palate with a median ridge.

one-half inch below the floor of the nose (Fig. No. 17). Out of one hundred heads from the dissecting room I was able to find only three examples of the V-shaped arch. In all of the three specimens the nasal fossae were narrow and the antra descended well below the floor of the nose. In one case the nasal fossa at its floor was only a quarter of an inch wide. Normally, it is nearer a half of an inch wide, while in extreme cases it may be three-quarters of an inch. Where the nasal fossae are narrow and the antra descend well below the floor of the nose, the slope of the inner side of the floor of the antrum gives the slope of the sides of the palatal arch (See Figs. 18, 19). Numerous examples show that the converse of this is true; that is, where the nasal fossae are wide and the antra are on a level

with the floor of the nasal fossae or above them, the arch of the palate is wide and flat (See Fig. No. 20). The two prominent, if not the two chief, factors in determining the height of the palatal arch are, therefore, the width of the nasal fossae and the degree of the descent of the antra.



Fig. 22.

Impression from the palate of a boy of twelve years. In the centre of the palate there is a pronounced median ridge. The side view of this impression is shown in Fig. 11.

### 3.—UNEQUAL GROWTH BETWEEN THE PARTS OF THE SEPTUM.

I have seen the statement that a marked median ridge of the palate is a characteristic of Peruvian skulls. The impression that it occurs chiefly in early races is incorrect, because

it is common also in present-day races. I have found it frequently in skulls from thirteen years on. Modern dentistry has taught us what slight but continuous pressure will accomplish in changing the form of the alveolar arches. Some such explanation is the best one which I am able to offer for the occurrence of this peculiarity of the palate. At birth the premaxillary wings and the nasal spine are very small. When the



Fig. 23.

A wide arch with a plateau-like mound in the centre. The width of the arch is due to wide nasal fossae, small, high antra, and possibly, in addition, to retention of the canine teeth.

second teeth appear the premaxillary wings grow rapidly in order to keep control of the tip of the vomer. The nasal spine grows as rapidly as the premaxillary wings. At birth there is no crest on the palatal processes of the palate bones and

the superior maxillae, but with the growth of the premaxillary wings the crests of these bones likewise develop and grow upward. If there is a disproportionate growth between the crests of the palate bones and the superior maxillae and the downward growth of the upper and larger part of the septum, the median line of the palate might be forced down into a median ridge. More than this happens in some animals (See Fig. Nos. 21, 22). In certain whales, for instance, the lower edge of the

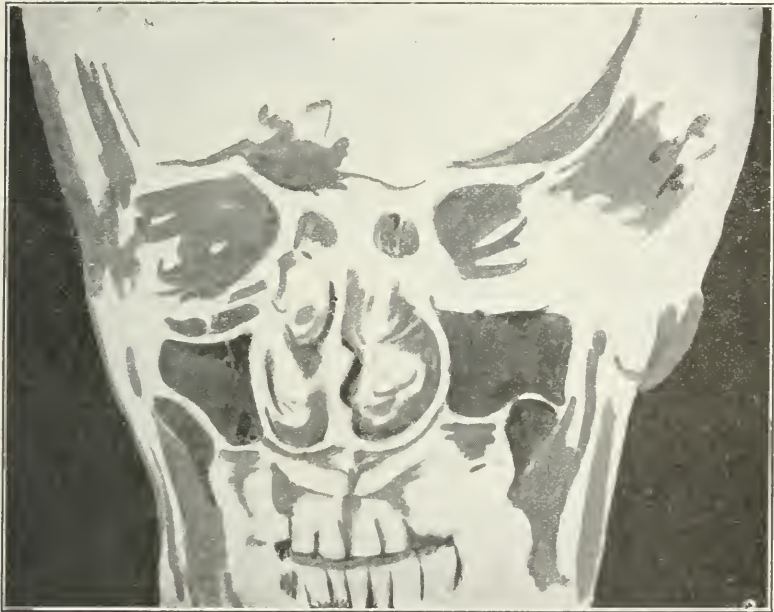


Fig. 24.

Tracing from an X-ray of a case of wide, flat arch, with a plateau-like mound in the centre. The width of the arch in this case is due to wide nasal fossae, to small high antra, and possibly, in addition, to retention of the canine teeth. See Fig 23.

septum makes a free ridge in the centre of the palate, the palatal processes not uniting with it. Sometimes, instead of a median ridge, the whole central part of the palate mounds down in a plateau. I have one example of this formation. The X-ray shows that the nasal fossae are excessively wide. From this it would look as if excessive outward and downward growth of the



nasal fossae was responsible in this instance for the peculiarity of the palate (See Figs. Nos. 23, 24). There is another explanation for this median ridge. It can be due to exostoses along the central suture line. Partial ridges due to this cause are common. In one skull which showed elsewhere very marked arthritic changes I found a large median ridge. How often this

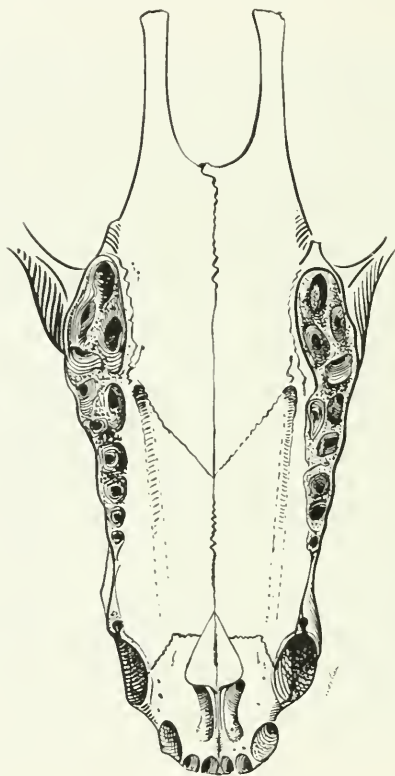


Fig. 25.

The palate of a bear.

median ridge accompanies rheumatism I do not know. It would be interesting to determine if it is common.

4.—FAULTY SHRINKING AND FAULTY READJUSTMENT OF THE  
PREMAXILLAE.

The arch with projecting premaxillae is characteristic of the negro race. It is an animal peculiarity and gives a very distinc-

tive form to the face. The proper shrinkage and readjustment of the premaxillae plays a very important part in the formation of the palate. In the evolution of the skull of man from the skull of his animal predecessors there are two great changes, the increase in the size of the cranium in order to accommodate the larger brain, and the disappearance of the snout and the shrinkage of the jaw owing to the lesser importance in man of the sense of smell and the fact that the teeth are no longer important for obtaining food and for defence. The enlargement of the cranium is a much easier mechanical problem than the obliteration of the snout and the proper readjustment of the parts of the hard palate. In order to realize what must be accomplished before man is reached, take, for instance, the hard

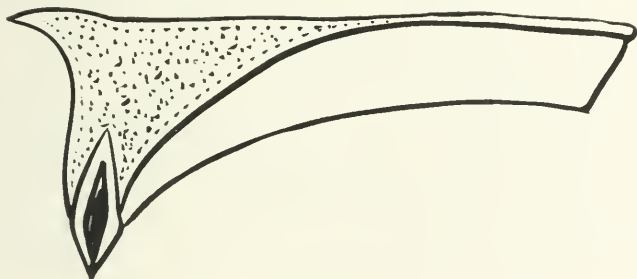


Fig. 26.

This figure represents a palate in which there has been normal shrinkage and readjustment of the premaxillae.—*See Fig. 26 (Continued.)*

palate of a bear (See Fig. No. 25). In the bear the largest part of the hard palate is made by the palatal processes of the palate bone. The palatal processes of the superior maxillae make the next largest part, and the premaxillae the smallest. The part made by the premaxillae, however, is but little smaller than that made by the superior maxillae. When the palate of the bear is compared with the palate of man, the bones which have changed the most are found to be the palatal processes of the palate bones and the two premaxillae. The palate shrinks from before and from behind, the middle changes but little.

In man the premaxillae not only shrink in size, but they turn downward so that the incisor teeth become nearly vertical. In order to have a normally formed palate, these two changes,

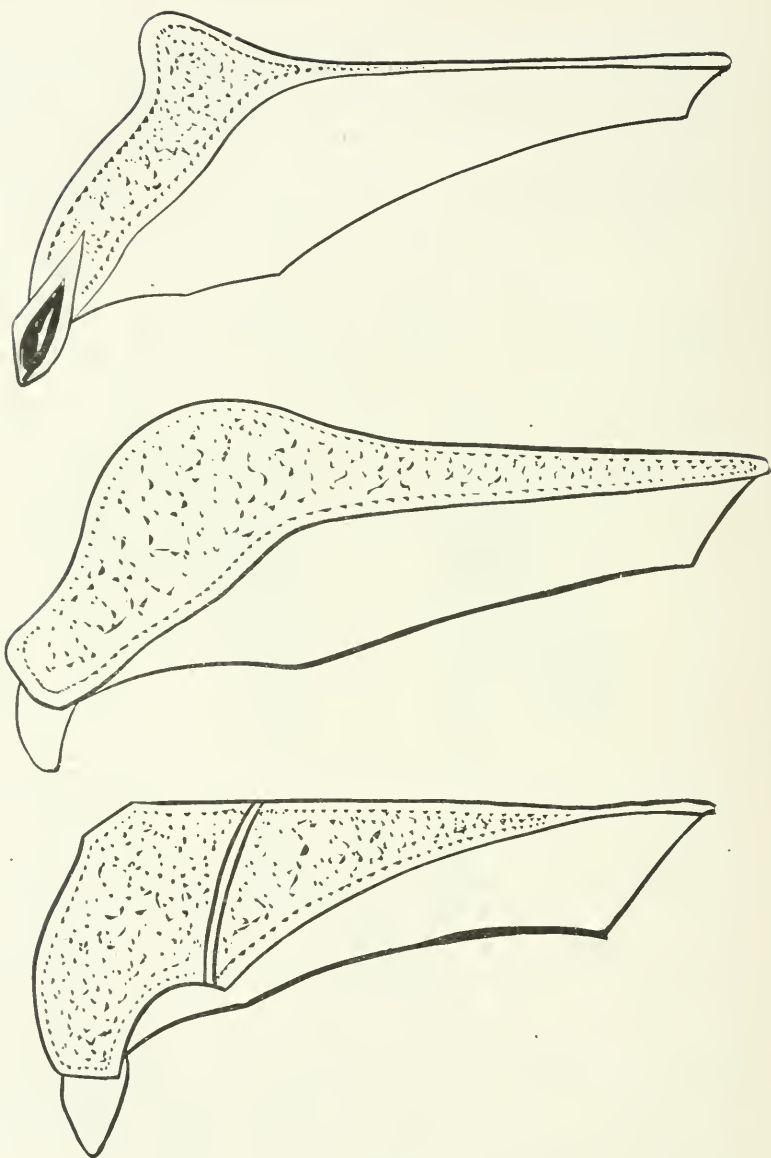


Fig. 26 (Continued.)

These figures are from cases in which the changes have been faulty.  
The figures are larger than life size.

shrinkage and downward tilting of the premaxillae, must both be fully accomplished. Often, however, the premaxillae fail to accomplish one or both of these changes. When both of these changes fail to occur the negro form of palate results. In this case the premaxillae are large and shoot markedly forward. In other respects the arch is very symmetrical. In other cases the premaxillae remain large, but they change their position properly and bring the incisor teeth to a vertical line, so that there is only the normal overhang. It is a peculiarity of the dentition of man that when all the teeth are erupted they reach down

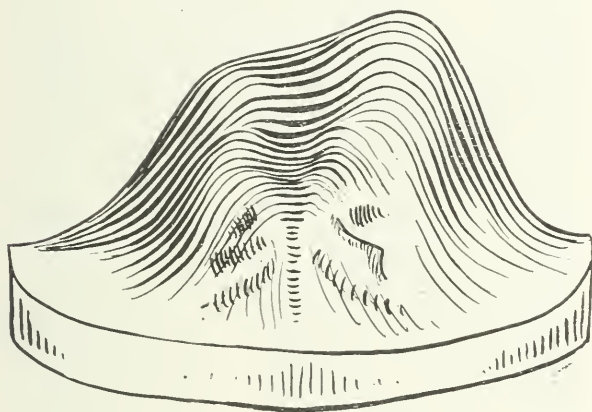


Fig. 27.

Diagrammatic drawing to illustrate asymmetry of the palate. The left side of the palate is higher than the right.

to the same level. The anterior teeth accomplish this first and the molars last. In other words, the anterior teeth give the level which the other teeth must attain. If the premaxillae are longer than they should be, owing to lack of proper shrinkage, the palatal arch will be deep anteriorly, and when growth is completed and the rest of the teeth have reached the level set for them by the teeth in the premaxillae, the arch will be deep posteriorly as well. Non-shrinkage of the premaxillae, therefore, is one cause of the high arch. Such an arch is not usually pointed, but has a narrow symmetrically curved dome (See Fig. No. 26).

## 5.—ASYMMETRY OF THE PALATE.

Asymmetry of the two halves of the palate is seen very frequently in the clinic. It is found from ten years of age onward. It is demonstrated prettily by impressions of the palate taken in modeling compound and by the X-ray. (Figs. 5, 6, 7, 27, 28.) On examining carefully a palate where this deformity occurs one side



Fig. 28.

Impression from a case of asymmetry of the palate. The left side of the palate is higher than the right.

is seen to be higher than the other. One antrum also usually is higher than the other. I have, however, found the reverse of this. The asymmetry of the two sides of the palate may be a part of asymmetry of the two sides of the head as a whole, or the asymmetry may be confined to the face. In the cases of

asymmetry of the palate which I have collected, facial asymmetry and asymmetry of the halves of the head have occurred about the same number of times. In one case the inequality of the two sides of the palate was due to asymmetry confined to the superior maxillæ, the rest of the face and the head being perfectly symmetrical.

In cases of asymmetry of the palate the impression taken in modelling compound shows that one side is higher than the other, and the X-ray shows that one nasal notch is higher than the other. If a cast is taken of the brow, the superciliary ridge on the high side of the palate is more prominent than the one on the other side. If the asymmetry includes the whole half head the frontal eminence on the side of the higher half of the palate is larger than that on the other side. When the asymmetry is confined to the halves of the face, the frontal eminences are equal, but the swelling of the superciliary ridges is not, the ridge on the high side of the palate is more prominent than the other one. Growth in the frontal sinus seems to take place forward instead of downward. The frontal sinus, as I said before, begins to enlarge when the antra enlarge at the coming of the second teeth. From this time on there is a marked parallelism between the growth of the two sinuses. The frontal sinus is really a part of the face.

#### THE EFFECT OF ASYMMETRY ON THE DENTAL ARCH.

When one half of the palate is higher than the other, the line of the teeth on the side of the palate which is the higher loses its curve and becomes straight. On the other side the curve of the dental arch is rounded and more nearly normal. (See Fig. No. 6.)

The most frequent cause of asymmetry of growth of the two halves of the face I believe to be some fault in the eruption or placing of the teeth. In the upper jaw ten milk teeth have to erupt, perform their function and then disappear. They are replaced by sixteen permanent teeth. Twenty-six separate bones therefore are developed in connection with the upper jaw. There are consequently twenty-six chances for development to go wrong. It goes wrong so often that a separate specialty has arisen in dentistry to deal with deformities of the teeth due to faulty development.



In a paper which I published about a year ago I brought up an observation which had long lain dormant. This observation, due to Potiquet, was that faulty descent of the central incisor teeth was associated with deviation of the septum. On reviewing the subject, I satisfied myself that this was true, and came to the conclusion that unequal descent of the central incisor teeth, and the consequent disturbance of the premaxillary wings, was one of the chief causes of deviation of the septum. In that paper I stated that unequal descent of the antra resulting in inequality of the two halves of the palate was another cause of deviation of the septum, and especially that it was a cause of vomer spurs. Continued observations during the last twelve months have convinced me of the truth of these statements. At first I did not recognize the importance of asymmetry of the palate in causing spurs and deviations. I do now, because I have found that whenever there is an inequality in the two halves of the hard palate there is a vomer spur on the side of the septum corresponding to the low side, and with the spur there is, in the majority of cases, a deviation of the septum toward the high side. The septum attempts to follow down and to attain its full growth, and in so doing it slips out of the vomer groove on the low side and bends outward one leaf of the vomer and makes a spur. I now would place asymmetry of the two sides of the hard palate, due to unequal descent of the antra or to unequal growth of the superior maxillae, as of equal importance with faulty eruption of the incisor teeth in causing deviations of the septum.

I believe that we have in faulty eruption of the teeth, of the anterior teeth as well as the posterior, and in their faulty occlusion, the explanation of fully a half of the cases of asymmetry of the hard palate. In a measure this paper is a continuation of my paper of last year. With the results of that paper and of this paper in mind we have, then, as causes of deviations of the septum, trauma and faulty eruption and occlusion of the teeth. The practical application of this paper is obvious. It is first: To avoid trauma. This we all do instinctively. It is second: To look after the proper eruption and spacing of the teeth. In the past pardonable vanity and toothache have sent many people to the dentist. In the future there should be a more scientific rea-

son, namely, the attempt to avoid the harmful effect of poorly erupted and placed teeth upon the septum and upon nasal respiration.

### *Discussion.*

Dr. H. L. Swain. Having on a previous occasion called the attention of the Association to the necessity of the care of the teeth, as based upon the idea that it influenced the shape of the palate, it gives me the utmost pleasure to hear Dr. Mosher express, in this very delightful and scientific way, this same opinion on further study, such as his experience has made him thoroughly competent to make. I think he has laid none too great stress upon the matter. The evidence of the fulcrum action as the arch breaks down, and how it draws with it the antra into this lower position, the plates, here presented, illustrate most clearly.

Dr. D. Bryson Delavan. Some years ago I spent a good deal of time in the study of this subject. I am therefore able to appreciate what Dr. Mosher has done and I would congratulate him and ourselves for having brought this subject before us so admirably. There is one point, however, that has never been clear to me and that is, why do we find the high arch palate so early in life? There are many instances where it is present before dentition, some even at birth. It is attributed to various causes, sometimes even to thumb sucking. It occurs before we are able to interfere in any way to prevent it; I have seen it present in children but a few months old.

Dr. A. Coolidge, Jr.: A few years ago I read a paper before this society as a protest against some of the theories of asymmetrical development which had been brought forward. The face contains three sets of cavities which have obvious and very important functions—the single cavity of the mouth and the double nasal cavities and orbits. The functions of these cavities demand accurate adjustment of certain of their parts, the teeth must fit accurately for mastication, the nasal respiratory slits should be of uniform breadth, and the orbits must fit a spherical eyeball. These cavities are so closely packed together that deformity of one tends to deform the others. In the complicated growth of the skull there are several factors which may tend to asymmetry, of which, as the writer has shown, trauma and irregular descent of the teeth are evident examples. But asymmetrical accessory sinuses I believe to be often a secondary effect rather than a primary cause of deformity, a correcting rather than a deforming factor.

These sinuses fill in the unused space between the three important sets of cavities, and are thus able to aid these cavities in

adjusting themselves to accidents of development. The point which I wish to make is, that whatever departure from typical growth we may prove, or guess, to be due to trauma, irregular eruption of the teeth, obstruction to breathing or defective heredity, we often can trace a counteracting asymmetrical growth attempting to minimize the disturbance of function which deformed jaws or a deviated septum or a flattened orbit would cause. These correcting asymmetries, of which the large turbinates and ethmoid cells opposite a concave septum are good examples, may be as prominent features as those caused by the deforming factors.

Dr. B. Alexander Randall: It seems to me that a word might be said of that often disregarded, as well as overworked, element, heredity; for as children often present the facial configuration we term likeness to one or both sides of the family, they may combine such elements disastrously in the internal configuration. In one family I had my attention called to this particularly. On one side there was a broad, full face and Roman nose, and in the other the narrow, aquiline high configuration. In the older generation there was little or no nasal trouble on either side, and in only one known instance a little deafness. In the younger generation there was the ample turbinal structure within narrow noses, and without exception all had trouble with the nasal chambers and with the ears. I think this element should be borne in mind and it is not without its influence in relation to the form of the palate.

Of course, it is hardly necessary to speak of the effect of mouth breathing in its relation to the pressure of the tongue in changing the conformation of the hard palate, as the open mouth eliminates this important influence in broadening of the arch and we have the high irregular arch unmodified. While mouth breathing may be oftener the consequence than the cause of the high-arched palate, they are probably parts of a vicious circle.

Dr. J. M. Ingersoll: Dr. Mosher spoke of the development of the forebrain. In most of the lower animals the cribriform plate of the ethmoid bone is at right angles to the floor of the nose, and the perpendicular plate of the ethmoid extends forward instead of downward, as it does in man. The long snout is covered by the thin nasal bones, and there is little or no pressure over the septum. As the forebrain develops it carries the frontal bones and cribriform plate forward over the nasal chambers, and the cribriform plate, in its new position, forms the greater part of the roof of the nose in man. The septum is thus held firmly in one position along its superior border. I should like to ask Dr. Mosher if he does not consider this one element in the causation of deviations of the septum.

Dr. J. O. Roe: Jackson in his work on "Orthodontia and

Orthopædia of the Face," and Pullen in the Dental Cosmos, as well as other dentists writing on the subject, recognize the open mouth in children, caused by nasal obstruction, adenoids and enlarged tonsils, as the principal etiological factor in the causation of irregularities of the teeth and deformities of the jaw, particularly in the production of prognathism. Associated with this condition we have the V-shaped or high dental or palatal arch. This condition, associated with nasal obstruction and mouth breathing, was, I believe, first called attention to by Jarvis of New York many years ago.

The effect produced by the open mouth is a narrowing of the jaws by the lateral pressure of the muscles of the face that are naturally drawn more or less tense. There is also an absence of the opposing pressure of the teeth against each other, which greatly assists in maintaining their regularity during the period of eruption. In the closed mouth the tongue also plays an important part in keeping the jaws, particularly the upper jaw, normally expanded.

The different types of faces naturally have much to do with the different forms which the jaws assume, as has been mentioned by Dr. Randall, but very much can be done, even when the distortion of the jaws, irregularity of the teeth and prognathism are very great, by proper appliances so arranged as to correct the deformity.

Dr. D. Brayden Kyle: Irregularity in development seems to be confined largely to the upper jaw. Outside of injuries, any such irregularity is rarely if ever observed in the lower jaw. This would seem to indicate to me that nutrition plays an important part in the early development of these two structures. In other words, why is it that we have this irregularity of development in the upper jaw and do not have it in the lower jaw? The blood and nerve supply of the upper jaw comes largely through bony foramina. Is it not possible that up until a certain age these bony openings are sufficiently large to permit sufficient nerve and blood supply for proper development, and then, owing to some slight injury or malformation, the size of the foramina is interfered with and does not allow for increased blood and nerve supply? The irregularity in the arch thus started will no doubt explain or account for any nasal irregularities, such as slight deflection of the septum and inequality of nasal openings. But I certainly think that many of the cases of irregularity in the upper arch are caused by blocked-up nasopharynx, interference with nasal respiration and defective blood and nerve supply, caused by bony malformation involving the foramina.

Dr. H. P. Mosher: Dr. Delavan asked for the explanation of the high arch when it is found in children before the period of

second dentition. The probable explanation, I think, is that it is due to faulty shrinkage of the premaxillæ. In certain cases the premaxillæ swing down into position as they should, but they do not shrink. Owing to this the vertical diameter of the premaxillæ is greater than normal. Therefore, in order for the posterior teeth to attain the level set for them by the anterior teeth, they must grow downward further than usual. The arch for these reasons is high from the start.

I agree with Dr. Ingersoll that the change in the plane of the cribriform plate from the roughly vertical position which it has in animals to the horizontal position which is found in man may play a part in causing deviations of the septum.

## METHODS OF OPENING THE MAXILLARY ANTRUM WITH PRESENTATION OF A NEW INSTRUMENT.

BY JOHN O. ROE, M.D.

It is a well recognized fact in medicine that the more obscure the etiology of a disease the more intricate the diseased structures, and the more complicated their anatomical relations to other parts the more numerous and varied will be the remedial measures proposed for the relief or cure of the disease. This is illustrated in the varied medical and surgical measures that have been brought forward for the treatment of accessory sinus diseases. It is also a matter of daily observation that if the exciting cause of a disease is removed the disease itself will speedily disappear—*sublata causa tollitur effectus*. Nature will effect a cure if the normal functions of the part are not too seriously interfered with. This restorative tendency, this *vis medicatrix naturae* is to be observed in the case of diseases of the maxillary antrum as well as in diseased conditions of any other part of the body. This is accounted for by the fact that diseases of the antrum are almost invariably secondary to diseases of the associated structures in the nose that obstruct its ostium and interfere with its normal functions, causing infections to extend to its cavity.

It is, therefore, of the utmost importance, before attempting an operation on the antrum itself, for the cure of an empyema, to carefully investigate the structures in the immediate vicinity of the hiatus semilunaris and the infundibulum, which may be regarded as the gateway to the different sinuses. This region has been appropriately termed by Ballenger "The Vicious Circle," although, as there is no circle involved, I think the term vicious centre would be a name quite as appropriate for this region. This is particularly a vicious centre, for there is no region of the body in which the anatomical conformation is subject to such infinite variations as the cellular structures of



the nose. It is this variation which accounts for the fact that a disturbance in the same location will in one case produce most serious results, while in other cases the effect will be slight.

Moreover, on account of these great anatomical variations, the system of drainage of the different cells is by no means constant, and, accordingly, may be very readily diverted from its proper channel. It has been shown by Zukerkandl, Logan Turner, Wright, Myles, Cryer, Killian, Jansen and others that obstruction of these channels, and particularly of the infundibulum, may divert the discharges from the frontal sinus or ethmoidal cells directly into the antrum. It is, therefore, readily seen that in cases of disease of the frontal or ethmoidal cells the antrum may become the innocent receptacle of infectious discharges. When the antrum has been so long the recipient of such infections as to become the seat of an independent disease which Nature is unable to cure on the removal of the exciting cause, and is not readily cured by irrigation and medication through the ostium maxillare, the opening of the antrum becomes a necessity.

The four well-known routes through which such an opening is made are through the alveolar process, the canine eminence or canine fossa, the hard palate and the naso-antral wall.

In those cases in which the exciting cause of the antral trouble is a diseased tooth penetrating the floor of the antrum, the extraction of the tooth and the free opening of the antrum through the tooth socket, through which opening the antrum can be thoroughly irrigated and medicated, will, in many cases, if the disease has been existing for but a short time, speedily cure the antral affection. In all cases, however, in which the disease does not speedily yield, other methods should be employed. The very old and common practice of inserting a permanent cannula through the maxilla to drain the purulent discharge into the buccal cavity is not only ill advised but can not be too strongly condemned.

The opening through the roof of the mouth is only advisable in certain cases for the removal of growths so located as to make this route the most direct method of reaching them.

In making the opening through the anterior wall of the an-

trum the Küster operation, which consists in taking away a sufficient portion of the wall to permit a free examination or exploration with the finger, and the Denker operation, which is simply an enlargement of the Küster, opening forward to the anterior angle of the sinus so as to render this portion of the cavity entirely accessible, are those commonly employed.

In those cases in which the entire chain of cells is diseased, the antrum, the ethmoidal cells, the frontal sinus, including in many cases the sphenoidal sinus, Jansen has proposed the extensive external operation of laying open the entire chain. This operation is only called for and only warranted in extreme cases, in which the cavities are the seat of myxomatous or other growths. In all ordinary cases of empyema of the antrum and ethmoidal cells, associated with nothing more than a degenerated condition of the mucous membrane that has resulted from a prolonged maceration in pus, these external operations are, in the opinion of the writer, unnecessary, for the reason that diseased conditions of the maxillary sinuses, and also of the ethmoidal cells which are commonly associated with an empyema, can be successfully treated by the nasal route. I simply allude to these methods for the sake of comparison, in order to emphasize the advantages of the nasal route over all other methods in the treatment of such conditions.

As a combination of the external and internal route, Caldwell, of New York, in 1895 described a new method by which an opening is made into the antrum through its outer wall sufficiently large to inspect the cavity and for the removal of any growths that may be found. When this is done the opening is continued through the naso-antral wall into the nose, through which the subsequent cleansing and treatment of the cavity are carried on, the external opening being immediately and permanently closed. Two years later Luc, of Paris, without a knowledge of Caldwell's operation, performed and described exactly the same procedure and so strongly advocated the operation that it has since been called "the Caldwell-Luc operation."

This same operation is also advocated by P. Watson Williams; but instead of using a chisel for making the opening through the different bones, he employs a six-penny trephine

with a gimlet-pin, making an opening into the antrum corresponding to the canine fossa. A circular opening is then made with the same trephine through the naso-antral wall just behind the anterior end of the inferior turbinated body close to the antral floor, removing at the same time the corresponding portion of the inferior turbinal. Through this opening the necessary treatment of the antrum is applied, the buccal opening being allowed to close at once.

Various methods of technique are employed by different surgeons for opening the antrum through the nose, an operation which was first proposed by Gooch and Réthi, and also advocated by John Hunter. The simplest method is by puncturing the wall with a Krause trocar and cannula, which is sometimes used for the purpose of diagnosis and washing out the antrum, and is often sufficient in recent or sub-acute inflammations to effect a cure.

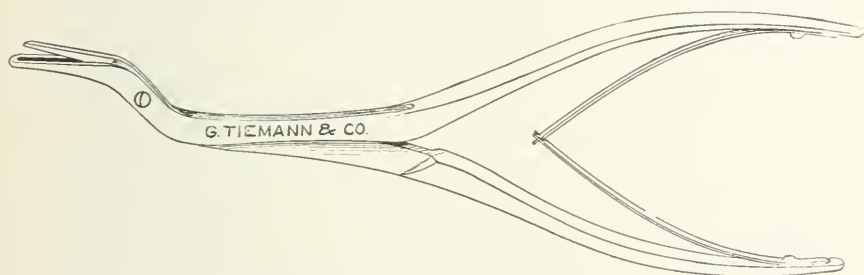
Vail proposed making a circular or oval opening by means of curved saws after having punctured the bone with his perforator, which process necessitates at the same time the removal of the anterior half of the inferior turbinated body.

Ballenger's operation consists in cutting out a portion of the wall either square or of any desired shape or dimension with his right angle knife. His method he describes as follows:

"The knife should be introduced through the naso-antral wall at the posterior limit of the antrum near the floor of the nose; then make an upward cut, a forward and downward cut. The upward and forward are made with the blade of the instrument at right angles to the naso-antral wall. When the forward cut is made the blade should be turned down, parallel with the naso-antral wall and pulled through it. The inferior incision remains to be made, and is done with the reverse knife, the knives coming in pairs. The knife is introduced into the posterior perpendicular incision at the floor of the nose and drawn forward along the floor of the nose to the anterior perpendicular incision, thus completing the removal of the naso-antral wall. Should the knife fail to remove the thickened lower portion of the wall it may be removed with the Grünwald or other bone forceps."

It has not been the experience of the writer to encounter naso-antral walls so readily pared away with the knife as has been my friend's experience, judging from the above description; on the contrary, a large majority of naso-antral walls are very firm below the attachment of the lower turbinate, and require, in many cases, considerable force to puncture with a drill or chisel.

The success of the operation by the naso-antral route in the treatment of chronic empyema of the antrum depends upon the freedom of the opening, so as to allow perfect drainage into the nose and free access to every portion of the cavity for thorough medication. For this purpose I have devised a pair of cutting forceps (*vide cut*) with sufficient strength to meet the require-



Cutting forceps for removing the naso-antral wall.

ments in every case, however firm and resisting the bony wall may be found.

The technique of my operation is as follows: Owing to the great variation in the size, position and attachment of the lower turbinate, it frequently becomes necessary to remove the lower anterior portion of this body in order to make room for the operation. Therefore, when the turbinate is attached very low down or enlarged or curled so as to greatly limit the space between it and the floor of the nose, the lower portion must be either resected or removed. If removed it should be done from the under side in a slanting manner, slanting toward the centre. This may be done with a slender, probe-pointed knife introduced beneath the turbinate along its attachment, cutting downward and inward towards the septum so as to catch the scroll as it rolls outward, or with a

pair of scissors or a pair of Grünwald's cutters. When this is done a small angular or curved pointed knife is introduced and an incision made through the periosteum down to the bone from behind forward, covering the entire length of the antrum, and as close to the attachment of the lower turbinate as possible. By examining the conformation of the jaw assisted in doubtful cases by the X-ray, we can judge quite accurately as to the position of the antrum and its forward projection, at which point the puncture should be made. At this point the incision through the mucoperiosteum is turned downward to the floor of the nose, the incision at the posterior end being also carried downward to the floor of the nose. This flap is then raised from the bone, carrying the periosteum with it throughout this sub-turbinal portion of the wall and rolled and turned into the centre of the nostril out of the way of injury. An opening is then made into the anterior portion of the antrum as close to the floor of the antrum as possible. This opening may be made with the writer's curved drill, a trocar or chisel. The most satisfactory instrument I have found to be Myles's curved chisel, particularly if the bony wall is very thick. A chisel may be used of the size we wish this preliminary opening to be, or the opening can be afterward enlarged with a gouge or chisel sufficiently to admit the blade of the punch forcep. This instrument is now introduced and the whole wall below the turbinal attachment removed by nipping it off, a piece at a time. It is quite important that the lower stump of the wall be levelled off so as to be on a plane with the floor of the nose and the floor of the antrum. When this wall has been completely removed the cavity is carefully explored and any growths that may be found are removed. The cavity is then thoroughly irrigated and carefully dried out. The periosteum and mucous membrane that has been raised from the inner wall is trimmed if necessary and turned down over the stump of the wall and carefully held there by thoroughly packing the entire antrum, and also the nasal cavity, with iodoform gauze. It is unnecessary to deal especially with the upper border of the incision, as it is thin, drains freely and readily heals. By thus covering the lower stump of the wall with mucous membrane we have left a uniform mucous surface and a perfectly free opening.

into the antrum through which we can deal with any growths or abnormal conditions which may be present and medicate the cavity as we would any other diseased mucous surface. The practice of vigorously curetting the antrum and destroying its mucous membrane so often recommended in the treatment of empyema of the antrum cannot be too strongly condemned. It is an operation that only should be done as a part of the radical operation of obliterating the antrum by the complete removal of its anterior wall and its lining membrane.

In three cases which I recently operated upon in the manner above described, the lining membrane of the cavity had become degenerated from long maceration in muco-pus until it was a boggy mass and in many places the bone had become denuded. By giving the antrum free drainage, keeping it clean by frequent irrigation, and stimulating the diseased mucous membrane by frequent applications of stimulative solutions, such as nitrate of silver, the tissues have regained their tone, the periosteum and mucous membrane have become restored, and at the end of four or five weeks the patients are well.

The advantage of this method over other methods is the freedom and ease with which the cavity can be irrigated and medicated, the patient being very readily taught to do the washing himself. Moreover, perfect drainage is maintained, giving a permanent and free ostium to the cavity at its most dependent point, thereby removing the danger of a recurrence of the empyema, regardless of the infection to which the cavity may be subjected. Finally the necessity of an external operation is avoided except in the case of growths, which may require the most radical measures for their eradication.

#### *Discussion.*

Dr. Harmon Smith: When the empyema of the antrum is not of long standing I have had very favorable results from using two instruments devised by Dr. Abrahams for giving drainage to the cavity, and likewise enabling the operator to pack it with gauze.

The first is a pyramidal shaped burr at right angles to the handle, which can penetrate the antrum beneath the inferior turbinate. The second is an olive-shaped burr which will enlarge



the former opening to any desired extent, and will lower the bony wall to the level of the floor of the nose.

So far as the mucous membrane lining the cavity is concerned, I find that it is ordinarily so lacerated that it serves no particular purpose and, in fact, on withdrawing the initial dressing it is frequently pulled away, and I believe that it retards healing more than it aids it. Another point favorable to the recovery is the treatment by the dry method, that is, packing the cavity with gauze rather than by irrigating it. Just as in cases of mastoid operation, the firm dry packing serves to promote healing quicker than irrigation.

Dr. W. E. Casselberry: Dr. Roe's forceps cut backward and seem satisfactory for that purpose; but I have felt the need of one has entered into the antrum. The Ostrom forceps and the one that will enlarge a chisel perforation by cutting forward after Spiess forceps are designed to cut forward, but do not always work effectively.

Dr. J. Price-Brown: It seems to me that Dr. Roe's method would be an excellent one in certain cases of chronic sinus disease; but I doubt whether it would be wise to follow it in all, especially in young people. In opening a sinus perpetually into the nose, would there not be great danger of atrophy taking place? The anterior part of the nasal passage and the cavity of the antrum do not secrete much moisture; and are you not by this operation going to produce an arid condition there, in the case of a young person, for the remaining thirty or forty years of life? In elderly people it may be an excellent plan, but in young people I have always been able to make a cure through the canine fossa, closing the wound in as short a time as possible, and leaving no permanent artificial opening anywhere.

Dr. Emil Mayer: I am very glad to know that Dr. Roe has presented this, to my mind, most ideal method of operating on the antrum of Highmore through the nose. My own experience in a number of cases leads me to protest very vigorously against the very unclean habit of having our patients' mouths bathed with this pus, all of which must be very disagreeable. Chronic cases have been entirely cured by a method in which the wall of the maxillary sinus was drained through the nose. The form of cutting I have been able to do by using the instruments presented by Dr. Myles, which have a large end and pull forward, and I have one case of my own in which I have made an opening that is fully  $\frac{3}{4}$  of an inch in its diameter that remains patent and that has cured a most obstinate case.

Dr. J. H. Bryan: There are one or two criticisms of this operation, and that is if this opening is made in the anterior part of the inferior meatus, or in the anterior extremity of the inferior

turbinate you are going to leave an opening which may cause a very great embarrassment to the patient. In the other operation the patient will have much less trouble and there will not be a gush of mucus every time a little congestion occurs in the antrum.

This operation will not cure all cases. Frequently the seat of a chronic maxillary sinusitis is in the upper anterior angle. You cannot curette the area involved by going through the inferior meatus. Yet that area must be curetted thoroughly and every vestige of diseased tissue removed. This can be done only by opening the anterior wall of the sinus.

Dr. T. P. Berens: Dr. Roe has not clearly defined the kind of disease of the antrum which he selects for his operation. There is a confusion of terms, resulting in a confusion of ideas, as to the relative value of the different operations. Many cases of acute sinusitis with involvement of the antrum recover without surgical measures. Some few, not more than 10 per cent—and that percentage is probably too great—run a subacute or chronic course and require operation. In the acute and subacute forms of nasal origin almost any drainage will suffice for a cure. In the chronic forms, many of which are dependent on disease of the frontal or ethmoidal, and in which the antrum acts simply as a reservoir for the pus, intra-nasal drainage will not cure. Dr. Mosher's specimens illustrate very clearly why intra-nasal drainage is insufficient, even in those cases where the antrum alone is diseased. The operation through the canine fossa is quite as easy of performance, quite as painless; it can be performed under cocaine and affords this important advantage over intra-nasal methods—it allows of a free inspection of the cavity and each step of the operation is directly in sight of the operator.

Dr. J. W. Gleitsmann: I am very glad to hear Dr. Bryan's remarks about the conservative treatment. What Dr. Bryan has said is exactly what I have thought all along, viz.: that if we open an antrum with decayed pus, possibly granulation tissue, we cannot get the diseased membrane out of this antrum by any large opening in the anterior wall whatever instrument you may use. As to those instruments, I would say that they are not new, but only modifications of modifications and of the original devised by Krause. It is the same with a frontal sinus. The intra-nasal operation of suppurating frontal sinuses is to me, in a surgical sense, impossible for cure. What is needed is a more thorough knowledge of the etiology and diagnosis of these conditions, and to cure them when so diagnosed in as simple a way as possible.

Dr. L. A. Coffin: I rise to put myself on record as being in agreement with Dr. Bryan, Dr. Berens and Dr. Gleitsmann. We differ very widely sometimes in the treatment of these cases and yet, on general principles, we must agree. Frequently the antrum

acts as a reservoir only, and if that be the case radical operation on the frontal or the ethmoidal sinus relieves the trouble with the antrum. One washing may be all that is necessary. My habit in multiple sinusitis of one side is to do a radical frontal and ethmoid, and leave the antrum for secondary operation if necessary, and as Dr. Bryan has suggested do the radical through the canine fossa. This enables one to follow the method of Killian in removing the middle portion of the inferior turbinate instead of its anterior portion. Dr. Killian made the statement that he never removed the anterior portion because of the nuisance from the dropping of mucus experienced by the patient when bowing his head. I had never had that complaint brought to my notice, but on inquiry one patient did make that complaint, and another, on being asked if he was troubled in that way, said: "Oh, I should think so." Another point that Dr. Killian made last year was that he would not operate on the frontal and ethmoid until he has done a radical on the antrum, feeling that there is danger of infection from a diseased antrum through the operated field. I believe he had had two deaths from an infective process, brought about, he thought, because he had operated the frontal and ethmoid sinuses and left the antrum for future operation. The acute cases will, as a rule, get well with simple treatment through cannula and trocar washings. Failing to do so I prefer to do the radical at once. Of course, if the patient refuses the radical operation, something must be done, and any of the procedures that have been described may be tried. Perhaps Dr. Roe's method may give the best results.

Dr. E. Fletcher Ingals: From what has been said about operations on the sinuses, I am impressed with the feeling that many men want to see how great an operation they can do on these patients without killing them. We do not need a sledge hammer to drive a tack. I presume that 80 per cent of the cases of antrum disease and more than 80 per cent of the cases of frontal sinus disease can be cured by a comparatively mild operation through the nares. I believe it is very much better in nearly all cases to do these comparatively mild operation and give them a reasonable time to get well; but if this does not succeed, then adopt more radical measures.

Dr. J. H. Bryan: I hope I do not belong to that radical class which is being so vigorously condemned. I would merely say that you cannot cure a chronic maxillary abscess of long standing without doing a radical operation. In the majority of cases it is impossible to remove the disease until you can get at those recesses by inspection, and the one most difficult to get at is the anterior orbital angle of the maxillary sinus. In a number of

cases I have not succeeded in my operation because I had overlooked one of these recesses.

Dr. Emil Mayer: I do not wish to be understood as objecting or endorsing the operation; I only rise and express my objection to the method of operating upon the maxillaries by way of the mouth alone.

Dr. W. L. Ballenger: I, with Dr. Ingals, believe that nature can be trusted somewhat, even in the most aggravated cases, to cure diseases. I do not believe that the surgeon has to do the whole job, and I have proved, in a large number of sinus cases, that this is true. I believe, taking the anterior group of sinuses that drain into the infundibulum or the meatus of the nose, that a large per cent of them, even chronic cases, can be cured without the so-called radical operation. I have proved this by confirming my diagnosis in about 100 cases with skiagraphs, and in another 100 which I diagnosed without the aid of the skiagraph. I have had 200 cases operated on by the intranasal route with just as good results in the frontal sinus and the anterior ethmoidal sinuses. The fundamental principle to be observed in the therapeutics of infection of the sinuses is drainage and ventilation, and even in the presence of granulations and bare bone, and even of necrotic bone, the case may go on to complete cure with conservative surgery of the sinuses. In the 200 cases I only found it necessary to do the so-called radical operation in five; this represents  $2\frac{1}{2}\%$  of the cases in which I found it necessary to do a radical external operation. I have been amazed at the number of reports of cases of radical operations at the hands of some of my confrères, and from my own experience have come to the conclusion that they may have operated in a large percentage of cases which would perhaps have been equally well cured by conservative intranasal operations. This is a point where you can usually attack frontal sinus disease and get good results, not at the sinus end of the sewer system of the frontal and anterior ethmoidal cells, but at its exit or infundebulum.

Dr. W. E. Casselberry: To harmonize these opposing views we should first agree upon what is to be considered as a "cure." Many cases that have been "cured," by radical or conservative methods, really practically cured, the patient being immensely improved over his previous condition, are still not absolutely free from a secretion of pus. The patient himself is usually contented with this improved condition, and if it were myself I think I would be contented with such a relative cure if obtainable by intranasal methods rather than to endure one of the more serious external radical operations. Cases are classified as acute or chronic, but that is an incomplete classification. It is conceded that a strictly acute case will usually recover with very moderate

treatment, and the chronic cases in which the cavity is filled with polypi will not recover by intranasal drainage alone, but between the two are many other grades; subacute, i. e., slow to recover, but finally passing away, perhaps never to return; chronic, but inactive for months at a time; or chronic, but of mild degree, with little pus excepting during acute exacerbations, which may be few or many. The majority of all of these grades can be practically cured, while perhaps only a minority are absolutely cured by intranasal methods; but even so, it has seemed to me best to avoid deforming external operations, excepting: (1) In well established chronic conditions of constant or intermittently severe degree; (2) in the presence of symptoms which menace life; (3) when business, convenience and economy demand an expeditious course, as in cases from a distance admitted to hospital for the purpose of being cured, under which circumstances the principle of "the greatest good to the greatest number" probably justifies the hospital management and the patient in encouraging those methods which are the quickest to get the patient out and back to work.

Dr. J. O. Roe: No one will deny the fact that in some cases the extreme operation known as the radical operation for opening the antrum is required; and also it will not be denied by those familiar with diseases of the antrum and their surgical treatment that the radical operation is done in many cases where it is unnecessary, and where the disease would be better treated by milder methods.

Dr. Gleitsmann speaks of removing every cell, whether diseased or not, when operating on the accessory sinuses, as in the case of radical operations of the mastoid. It is encouraging in this connection to observe that a reaction has taken place against both the radical mastoid operation and the radical operations in the treatment of chronic accessory sinus diseases, milder and safer measures being advocated. In these cases, as well as in other diseased conditions, surgeons are ceasing to see how near they can come to killing the patient without doing so, as Dr. Ingals has so very aptly remarked.

I am pleased to hear Dr. Berens discourage the use of the curette, an instrument with which so much mischief is capable of being done. To go into the antrum and curette out its entire mucous membrane simply because this membrane has become diseased and degenerate from maceration in pus is, to my mind, unscientific, unwarrantable, and should be strongly condemned. In place of this destroyed mucous membrane we have, when the surface has healed, if it heals at all, a cavity lined with cicatricial tissue that is quite liable to break down at any time on the slightest provocation. When this has taken place the diseased



cavity can then be only successfully treated by the radical operation of obliterating the cavity, necessarily followed by more or less sinking in and deformity of that side of the face.

The rational and scientific method of treating these cases is that which gives complete drainage to the cavity and affords ready access to every part, for not only the removal of growths or other conditions that need to be dealt with surgically, but for thorough medication as well, which, if carried out thoroughly, will in many cases restore the diseased membrane to its normal condition, thereby effecting a cure where the radical operation seemed the only solution of the problem.

In all ordinary cases, at least, the patient should be given the benefit of the doubt by making an exploratory opening through the anterior portion of the naso-antral wall and carefully examining the cavity for growths, ulcerations or other abnormal conditions. When such conditions are found the operation which I have described, of completely removing the lower half of the naso-antral wall, thereby converting the antrum into a recess of the nasal cavity, will enable us to cure a large proportion of patients on which is now performed the so-called radical operation.

In regard to the instrument which I have presented for opening the antrum in this manner, I am sure you will find it as serviceable and satisfactory as it has been to me in the treatment of these cases.



## THE MORPHOLOGY OF THE TURBINALS.

BY JOHN M. INGERSOLL, M.D.

The turbinal bones may be classified, in a general way, according to their function, into respiratory turbinals and olfactory turbinals. They are also classified as maxillary, nasal and ethmoidal turbinals, according to the bones which support them.

FISH: The nasal organs of all the fishes except Dipnoi (lung fishes) are used only for olfaction, and so contain only olfactory turbinals or tissue which corresponds to the olfactory turbinals in other vertebrates. Respiration is, of course, carried on entirely thru the gills. In the white fish of the Great Lakes (*Coregonus Clupeiformis*) the nasal organs are two irregularly spherical shaped cavities separated from each other by a thick septum. They are situated in the anterior part of the head. Each cavity opens externally thru two orifices in its external lateral wall, but it does not communicate with the mouth. The posterior orifice is supplied with a valve which, when opened, permits the water to circulate thru the nasal cavity.

On the internal lateral wall of each cavity there are twelve finger-like projections, or lamellae, radiating from a common center, which are covered with olfactory epithelium in which the filaments of the olfactory nerve end (Fig. A), and they constitute the olfactory turbinals of the fish.

These lamellae, like the turbinals in other vertebrates, increase the expanse of mucous membrane exposed within a limited space and so increase the acuteness of the sense of smell.

REPTILES: The character and structure of the nose in reptiles is modified by the change in the method of respiration. The nasal fossae become differentiated into a respiratory and an olfactory portion. The pterygopalatine arch is well developed and the shelf-like palatine processes of the superior maxillae unite with the palatine bones and form a secondary roof to the mouth. The fossae are thus prolonged backward, below the brain, and open into the nasopharynx.

The Crocodilia, to which order the alligator belongs, are the most highly developed of the reptiles, and the nasal organs are much more complicated than in the fishes.

In the alligator the two nasal cavities are separated in the



Fig. A.

Lake Whitefish (*Coregonus Clupeiformis*). Longitudinal section thru the right nasal fossa looking toward the septum. 1. Nasal fossa, showing the olfactory lamellae radiating from a common centre on the internal lateral wall. 2. The mouth. 3. The eye.

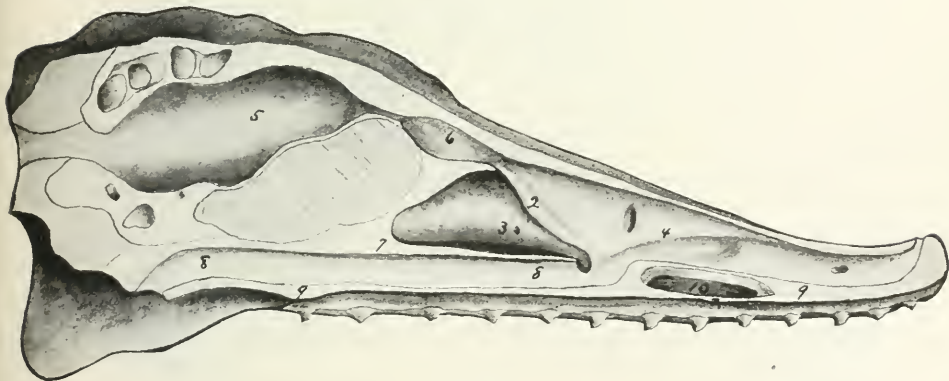


Fig. B.

Alligator. Longitudinal section, showing the left nasal fossa. 2. Olfactory turbinal. 3. Pseudo turbinal. 4. Maxillary turbinal. 5. Brain cavity. 6. Olfactory lobe of brain. 7. Lateral wing of vomer. 8. Respiratory portion of the posterior part of the nose. 9. Palate. 10. Maxillary cavity.

median line by a rather thick, smooth septum (Figs. C and D, 1). Each cavity opens externally thru a small elliptical orifice in the extreme anterior superior surface of the snout. The anterior

half of each nasal cavity is the respiratory portion of the nose. There is a round ridge, projecting downward from the roof of this part of the nose (Figs. B and C, 4), which is a true turbinal and is homologous with the inferior or respiratory turbinal in man. The posterior half of each nasal cavity is subdivided into



Fig. C.

Alligator. Transverse section thru both nasal fossae, in the anterior or respiratory portion of the nose, looking anteriorly. 1. Nasal septum. 4. Maxillary turbinal. 9. Palate. 10. Maxillary cavity.

two superimposed cavities, of which the upper one is much the larger of the two and may be designated as the olfactory region, for it contains the olfactory turbinals and mucous membrane.

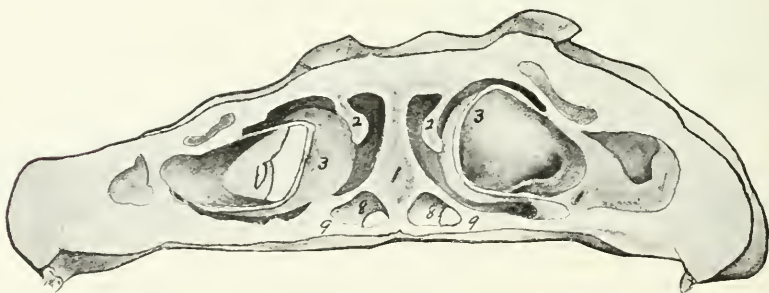


Fig. D.

Alligator. Transverse section thru the olfactory portion of both nasal fossae, looking posteriorly. 1. Nasal septum. 2. Olfactory turbinal. 3. Pseudo-turbinal. 8. Respiratory tract. 9. Palate.

The inferior cavity is simply a round tube which conducts the inspired air backward from the respiratory portion of the nose into the nasopharynx (Figs. B and D, 8).

The olfactory turbinal is a curved process attached to the roof of the olfactory portion of the nose (Figs. B and D, 2). The posterior end is simply a ridge projecting downward into the nasal fossa but along the middle portion the turbinal increases in size and its inferior border curves outward toward the external wall of the nose. The anterior portion is much larger than the rest of the turbinal and contains a distinct cell. The olfactory lobe of the brain extends forward and ends just behind the posterior portion of this turbinal, and the olfactory nerves are distributed over it and in the surrounding mucous membrane (Fig. B, 6). The general characteristics of this turbinal correspond quite closely to the middle turbinal, its homolog in man.



Fig. E.

Loon. Transverse section thru the anterior part of both nasal fossae, looking posteriorly (twice natural size). 1. Nasal septum. The free communication between the two nasal fossae in this part of the nose is shown. 2. Vestibular turbinal.

BIRDS: In most birds the anterior portions of the nasal fossae communicate with each other, the septum being incomplete (Fig. E, 1). Posteriorly both fossae open together into the mouth thru an elliptical fissure in the middle of the posterior part of the palate. The turbinals are more highly developed than they are in fishes or reptiles.

The Loon (*Colymbus*) is a web-footed swimming bird which lives in the cold waters of the northern hemisphere. It has a long beak and the nasal fossae extend forward in it about one-half

of its length. Each fossae contains three turbinals. The most anterior one is attached to the roof and external lateral wall of the fossa by a slender pedicle which gives off five distinct branches. It is called the vestibular turbinal on account of its location in the vestibule or extreme anterior part of the nose, but it is covered with stratified pavement epithelium and does not correspond to any of the turbinals in man (Fig. E, 2).

Just above the posterior end of the vestibular turbinal a second turbinal begins and extends backward thru the rest of the fossa. Its anterior part is attached to the roof of the nasal cavity and its inferior border is curved outward. As the turbinal extends backward its attachment shifts from the superior to the lateral

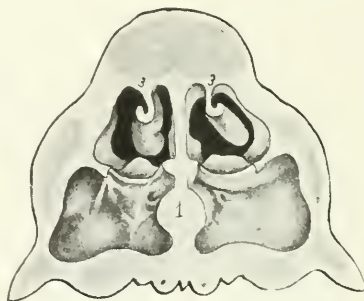


Fig. F.

Loon. Transverse section thru the middle portion of both nasal fossae, looking posteriorly (twice natural size). 1. Nasal septum. 3. Maxillary turbinal.

wall and it increases in size and becomes more and more rolled, so that in its posterior part it is decidedly scroll-shaped and occupies the greater part of the nasal fossa (Figs. F and G, 3). This turbinal is the homolog of the inferior or maxillary turbinal in man, and it is covered with ciliated epithelium.

The third turbinal is situated in the extreme posterior superior part of the nasal fossa. It is a simple, ridge-like structure supported by a broad base which is attached to the external lateral wall. Its anterior end contains a small closed cavity (Fig. G, 4). The olfactory mucous membrane is distributed over this turbinal and the roof of the nasal fossa in this region. This turbinal corresponds to the ethmoidal turbinal in man.

**MAMMALS:** The turbinal structures in mammals exhibit great variations. In microsmatic mammals (animals with an acute sense of smell) the turbinals attain their highest degree of development and are very complicated structures.

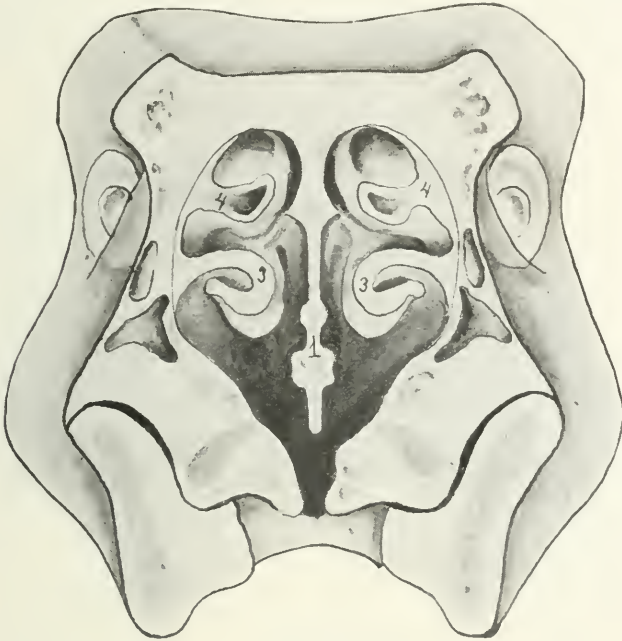


Fig. G.

Loon. Transverse section thru the posterior portion of both nasal fossae, looking posteriorly (twice natural size). 1. Nasal septum. 3. Maxillary turbinal. 4. Olfactory turbinal.

The turbinals in the American panther (*Felis Concolor*) are a good example of this high degree of development. The nasal fossae are separated from each other by a thin, straight septum, which unites with the cribriform plate posteriorly. Inferiorly the septum is formed by the vomer and presphenoid, and superiorly by the descending plates of the frontal and nasal bones. Along the articulation of the perpendicular plate of the ethmoid with these descending plates, a lateral process extends out at right angles into each fossa and supports a considerable portion of



the olfactory turbinals and partially subdivides the olfactory portion of the nose into two superimposed cavities (Fig. 1, 1).

The maxillary turbinal is in the anterior inferior part of the nasal fossa (Figs. H and I, 2). It springs from the superior maxillary bone by a slender pedicle which extends out into the fossa, nearly parallel to the floor of the nose, and divides into two branches. The superior branch curves upward, making one complete coil. The inferior branch curves downward and makes

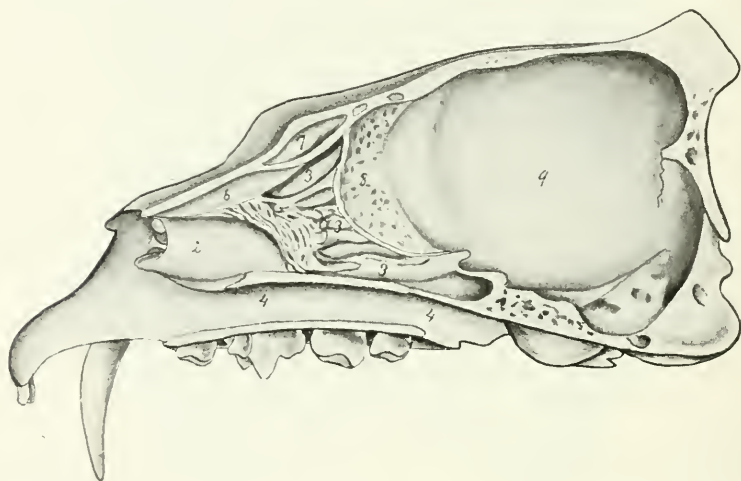


Fig. H.

Section showing the right nasal fossa of the American Panther (one-half natural size). 2. Maxillary turbinal. 3. Ethmoidal turbinals. 4. Respiratory portion of the nose separated from the olfactory portion by the lateral wing of the vomer. 6. Nasal turbinal. 7. Ethmoidal turbinal in frontal sinus. 8. Cribriform plate. 9. Brain cavity. 10. Sphenoidal sinus.

two turns within itself. The whole structure is a typical example of the double coiled turbinal which is present in many mammals. It is the homolog of the inferior turbinal in man.

The naso-turbinal is a rather simple structure which projects downward from the nasal bone and overlaps the maxillary turbinal and the anterior part of the ethmoidal turbinals (Fig. H, 6). Its homolog in man is the *ager nasi*, a small bony ridge anterior to the middle turbinal.

The ethmoidal or olfactory turbinals are very complex structures. There are six of these turbinals in each fossa. They radiate from the convex surface of the cribriform plate and the extent of their surface is tremendously increased by almost innumerable folds and branches which make an intricate labyrinth occupying the posterior part of each nasal fossa and constitute the olfactory portion of the nose (Figs. H, I and J, 3). Branches from these turbinals extend into all of the accessory sinuses.



Fig. I.

Panther. Transverse section thru the anterior part of both nasal fossae, looking posteriorly. 1. Nasal septum. 2. Maxillary turbinal. 3. Ethmoidal turbinals.

The posterior portion of each nasal fossa is subdivided into two superimposed cavities by the lateral wings of the vomer (as it is in the alligator). The superior cavity is the olfactory portion of the nose and contains most of the olfactory turbinals. The inferior cavity connects the respiratory portion of the nose with the nasopharynx (Figs. II and J, 4).

APES: The relative position of all of the turbinals is changed in apes and in man. Especially in man the tremendous develop-

ment of the fore-brain pushes the ethmoidal and frontal bones forward and the nose appears to recede into the head. The cribriform plate, in its new position, forms the greater part of the roof of the nose, and the ethmoidal turbinals are situated more directly above the maxillary turbinals.

The turbinals in the gibbon or long-armed ape (*Hylobates*) are comparatively simple structures, but they are less degenerated

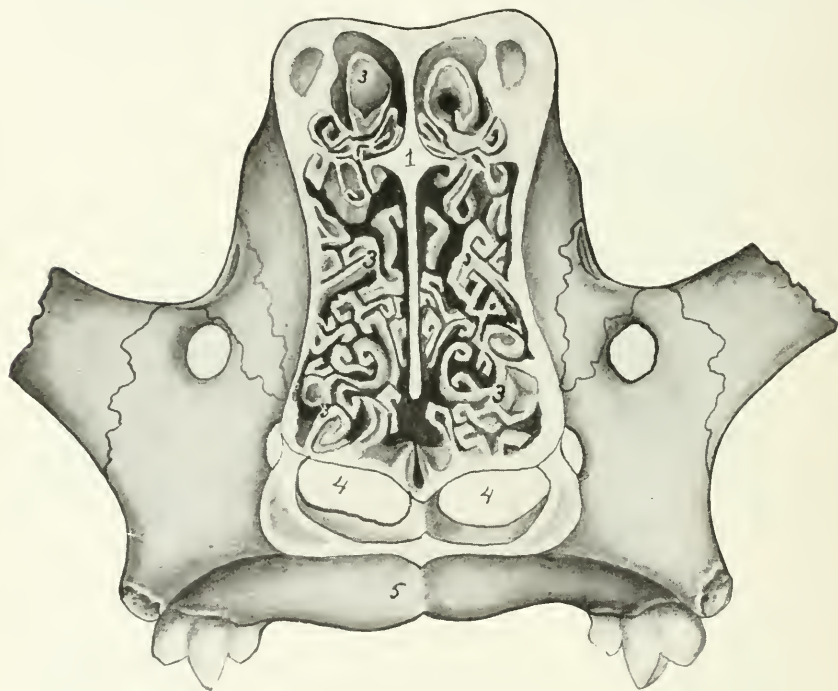


Fig. J.

Panther. Transverse section thru the middle portion of both nasal fossae, looking anteriorly. 1. Nasal septum. 3. Ethmoidal turbinals. 4. Respiratory portion of nose separated from olfactory portion by the lateral wings of the vomer. 5. Palate.

than those of some other apes. The maxillary turbinal has a double coil, but the superior coil is much smaller than the inferior one and thus shows the tendency toward degeneration and disappearance of this part of the turbinal in the higher animals (Figs. K and L. 2). The ethmoidal turbinals show a much

more marked degeneration. There are two in each fossa and their position and general appearance is the same as the ethmoidal turbinals in man (Figs. K and L, 3 and 4). They have entirely lost the numerous fine branches which are present in some of the lower animals and their number has decreased from five to two. Immediately in front of the ethmoidal turbinals, on the external lateral wall of each nasal fossa, there is a bony ridge,

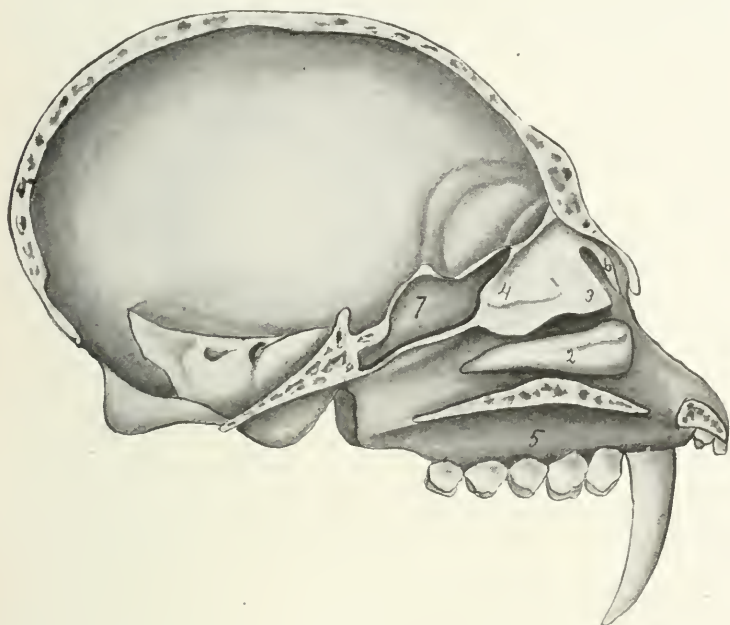


Fig. K.

Ape. Longitudinal section showing the left nasal fossa. 2. Maxillary turbinal. 3. Ethmoidal turbinal. 4. Superior ethmoidal turbinal. 5. Palate. 6. Nasal turbinal. 7. Sphenoidal sinus.

the agger nasi, which is the rudiment of the nasal turbinal (Fig. K, 6).

MAN: The characteristics of the turbinals in man are too well known to need a description here. They are comparatively rudimentary structures which have reverted to the more primitive types which are present in the lower vertebrates.

To recapitulate briefly, the turbinal structures of fishes (ex-

cept Dipnoi) are used only for olfaction and are simply ridges covered by olfactory mucous membrane. In reptiles, owing to the changed manner of respiration, the nasal organ functionates both in respiration and olfaction, and respiratory and olfactory turbinals are found. They are, however, quite simple structures. In birds, the importance of the respiratory function of the nose increases, and the respiratory turbinals show a high degree of development. The expanse of respiratory mucous membrane is very largely increased by the coiled structures of the turbinals. The olfactory turbinals are simple ridge-like structures.

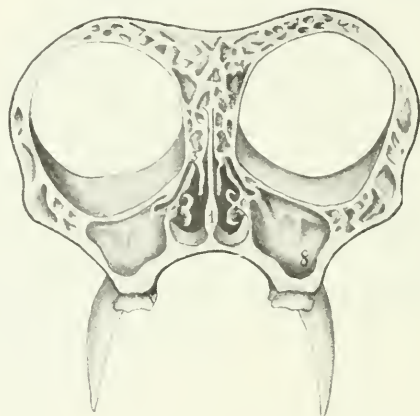


Fig. L.

Ape. Transverse section thru both nasal fossae, looking anteriorly. 1. Nasal septum. 2. Maxillary turbinal. 3. Ethmoidal turbinal. 8. Maxillary sinus.

In microsmatic mammals, all of the turbinals exhibit a very high degree of development. Their numerous fine branches and coils enormously increase the amount of mucous membrane exposed within a comparatively small space and thus increase the efficiency of the nasal organs. In apes, all of the turbinals have degenerated or reverted to some of the more primitive types. In man, the turbinals are quite similar to the turbinals of the ape. They are all rather simple structures. Rudiments of the fourth and fifth ethmoidal turbinals are sometimes found. The agger nasi is the rudiment of the nasal turbinal. Such rudiments

are much more frequently present in the embryo than in the adult.

*Discussion.*

Dr. H. L. Wagner: I would like to ask the doctor if he has ever found something similar in the lamb or sheep.

Dr. D. Bryson Delavan: In operating upon the septum where the section is carried deeply inward toward the median line we sometimes come upon a slightly oval space which is peculiar and may be the remains of this so-called organ of Jacobson. Its origin and the line of its communication through the hard palate with the mouth are not easy to explain.

Dr. W. E. Casselberry: Although familiar with Zuckerkandl's literature on Jacobson's organ, I have never been able to identify with certainty the furrow which he describes as leading into it in the human being. While operating on the septum narium I have many times encountered the intercartilaginous space to which Dr. Delavan refers, and also would like to know if it represents Jacobson's organ, for I always considered the usual explanation of this space as being a defect in the developmental attachment of the two septal plates to be inadequate because it fails to account for the presence of the epithelial lining within the space.

Dr. H. L. Wagner: Some years ago I had the good fortune to be working with Zuckerkandl and he showed me just exactly what Dr. Delavan has described as the degenerated Jacobson's organ, and it is just as Dr. Delavan has described in front of the septum. We certainly cannot find it in all cases, but certainly in the specimens which Zuckerhandl showed me it was well marked.



## THE X-RAY AND PATHOLOGICAL EXHIBITS.

In addition to specimens presented during the reading of papers, the exhibits above referred to were notable features of the Congress. Through the kindness of Dr. Birkett printed catalogues of both were furnished to all in attendance and copies of them have been incorporated in the permanent records of the Association.

The X-ray exhibit consisted of skiagrams illustrative of diseases of the accessory nasal sinuses. Dr. Coakley showed five plates illustrating normal sinuses, eight of absence of one frontal sinus, one of absence of both frontals, seventeen of unilateral sinus disease, and nineteen unclassified plates. Dr. S. Cummings, of Toronto (by invitation), showed seven of normal sinuses, one of absence of both frontals, and eight unclassified. Dr. St. Clair Thomson, of London, England, Corresponding Fellow, showed three photographs illustrative of, respectively, the value of the X-rays in sounding and washing out the frontal sinus and the absence of the left frontal. Dr. Casselberry showed one plate of disputed interpretation. Dr. Birkett showed fifteen plates of normal sinuses, one with absence of one frontal, fourteen showing unilateral disease and eight unclassified. Prof. G. P. Gridwood (by invitation) showed one plate illustrating the application of Dr. Bela Alexander's "Plastic Roentgenography" for demonstrating accessory sinuses by lantern slides. Dr. Birkett expressed in the catalogue his indebtedness to Prof. G. P. Gridwood, Dr. Robert Wilson and Mr. J. C. McNeill, of Montreal, in the arrangement of the exhibit. The plates were arranged in a small room adjoining the meeting hall and were illuminated by electric lights. The catalogue embraced 100 titles.

In a corresponding room was an exhibit of pathological specimens in the arrangement of which Dr. Birkett had been assisted by the following persons: Prof. J. G. Adami, director of the Museum at McGill University; Dr. Maude Abbott, who prepared the catalogue, and Mr. E. L. Judah, who skilfully mounted the specimens shown. The catalogue contained thirty-eight entries.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

*Business Meeting.*

The Thirtieth Annual Congress of the American Laryngological Association met at the Windsor Hotel, Montreal, under the Presidency of Dr. Herbert S. Birkett, of that city, on Monday, May 11, 1908, at 10 A. M. The following Active Fellows were registered during the various sessions:

Herbert S. Birkett, Montreal, President.  
J. Payson Clark, Boston, Mass., First Vice-President.  
J. Edwin Rhodes, Chicago, Ill., Second Vice-President.  
J. E. Newcomb, N. Y. City, Secretary and Treasurer.  
J. H. Bryan, Washington, D. C., Librarian.  
C. C. Rice, N. Y. City, Member of Council.  
J. W. Gleitsmann, N. Y. City, Member of Council.

And in addition:

W. L. Ballenger, Chicago, Ill.  
T. P. Berens, New York City.  
J. Price-Brown, Toronto, Canada.  
W. E. Casselberry, Chicago, Ill.  
F. C. Cobb, Boston, Mass.  
L. A. Coffin, New York City.  
A. Coolidge, Jr., Boston, Mass.  
D. Bryson Delavan, New York City.  
J. W. Farlow, Boston, Mass.  
C. P. Grayson, Philadelphia, Pa.  
T. H. Halsted, Syracuse, N. Y.  
T. M. Hardy, Chicago, Ill.  
F. E. Hopkins, Springfield, Mass.  
E. Fletcher Ingals, Chicago, Ill.  
J. M. Ingersoll, Cleveland, Ohio.  
D. Braden Kyle, Philadelphia, Pa.  
G. Hudson Makuen, Philadelphia, Pa.  
Emil Mayer, New York City.  
H. P. Mosher, Boston, Mass.

B. A. Randall, Philadelphia, Pa.

W. S. Renner, Buffalo, N. Y.

J. O. Roe, Rochester, N. Y.

Harmon Smith, New York City.

H. L. Swain, New Haven, Conn.

C. F. Theisen, Albany, N. Y.

H. L. Wagner, San Francisco, Cal. Total, 33.

After the roll call at the opening session the President, Dr. Birkett, welcomed the Association to Montreal and delivered an historical address entitled "A Brief Account of the History of Medicine in the Province of Quebec from 1535 to 1836." The scientific programme was then taken up, and as time permitted the papers scheduled for the session were read and discussed. The Association then went into executive session and first proceeded to the election of new Fellows. The candidates were as follows: For Corresponding Fellow, Dr. Gustav Killian, Freiburg-im-Br., Germany, and for Active Fellows, Dr. John F. Barnhill, Indianapolis, Ind., proposed by Drs. King and Swain. Thesis, "Some Surgical Principles Essential to the Cure of Frontal Sinus Empyema;" Dr. B. Alexander Randall, Philadelphia, Pa., proposed by Drs. Freeman and de Roaldes. Thesis, "Some Neglected Points in the Anatomy of the Pharynx;" Dr. Harmon Smith, New York City, proposed by Drs. Chappell and Farlow, Thesis, "Multiple Papilloma of the Larynx in Children;" Dr. J. Gordon Wilson, Chicago, Ill., proposed by Drs. Rhodes and Shambaugh. Thesis, "A Study in Variations of the Frontal Sinuses with a Note on the Topography of the Cribriform Plate." The tellers, Drs. Theisen and Hopkins, reported that all the candidates were elected.

The President appointed Drs. Ingersoll and Renner as a committee to audit the books and annual report of the Treasurer.

An election of a Committee of Five to nominate officers for the coming years resulted as follows: Dr. Swain, Chairman; Drs. Bryan, Casselberry, Coffin and Coolidge.

Dr. Farlow presented for inspection a nasal gouge and a myxo-fibroma removed from the naso-pharynx.

At 1 P. M. the Congress adjourned and the Fellows, with their ladies and invited guests, were conveyed by special cars to the

Montreal Hunt Club, where they were entertained at lunch by the President. Following this, the scientific programme was resumed, and all returned to the city shortly after 6 P. M.

In the evening a reception was given by the President and Mrs. Birkett at the galleries of the Art Association, Phillips Square, which was largely attended by the physicians of Montreal with their families and by prominent citizens. A musical and literary entertainment of a high artistic order was also provided.

On the second day, Tuesday, May 12th, the Congress met at 9.30 A. M., opening in executive session. The following reports were read and accepted:

*Secretary's Report.*

Since the last Congress Dr. W. Peyre Porcher has resigned from Active Fellowship. The death of Prof. L. von Schroetter again leaves the Honorary Fellowship class vacant. The death, in December last, of Prof. Moritz Schmidt left a vacancy in the Corresponding Fellowship class. The elections of yesterday reduce our vacancies to 12 in the Active class and to 1 in the Corresponding class.

In the name of the Association, letters of appreciation and condolence were sent to the respective families of our deceased colleagues, and acknowledgments were received.

The Transactions of 1907, a copy of which is, by precedent, herewith presented, were issued in October last at a cost of \$1.87 per volume. Somewhat over 200 copies were distributed through the usual channels. At the request of the Medical Library of Dallas County, Texas, its name was placed on our mailing list.

The Association received an official invitation to participate in the Tuerck-Czermak Congress in Vienna in April of this year. As no one asked for credentials as delegate, the following letter was sent:

Prof. Dr. Ottokar Chiari, President Tuerck-Czermak International Laryngo-Rhinological Congress, Vienna, Austria.

The American Laryngological Association extends its hearty congratulations to its colleagues assembled in International Con-

gress in Vienna to celebrate the semi-centennial of the establishment of laryngological teaching and to honor the memories of those illustrious men who were the pioneers in now well-trodden paths.

No more fitting place than Vienna could have been selected for this celebration. In this city these men fought their early struggles and won their early victories.

The Association regrets that it cannot be represented by a personal delegation, but tenders its best wishes to the International Congress, expressing the hope and confident expectation that the review of our knowledge which will here be made and the scientific progress which will here be heralded will mark a distinct advance in the history of laryngology and rhinology.

Finally, to you, Honored Sir, one of its esteemed Corresponding Fellows, it extends the assurance of its personal congratulation and continued good will.

By order of the Council,

(Signed)

DR. HERBERT S. BIRKETT, Montreal, President.

DR. JAMES E. NEWCOMB, New York City, Clerk.

The Secretary calls attention to the fact that the Transactions of this year will complete the records of the third decade of the Association's existence. Following precedent, the volume should contain an index of the issues from 1899 to 1908 inclusive. To be of service, this work must be done carefully by one accustomed to indexing, and will materially enhance the cost of the issue of the current year. The expenditure is justified, however, by the increased utility it will give to the volumes.

Two meetings of the Council have been held during the past year. (Minutes read).

This report is respectfully submitted.

JAMES E. NEWCOMB, M.D., Secretary.

#### *Treasurer's Report.*

Shortly after the close of the last Congress, the Treasurer received from Dr. Arthur W. de Roaldes the sum of \$500 for the establishment of the de Roaldes Prize Fund. In accordance

with the vote passed at the Congress, he assumed custody of this fund pending further direction by the Council. The money was deposited in the Franklin Savings Bank of New York City in the name of the American Laryngological Association, James E. Newcomb, Treasurer, where it now lies, drawing interest at four per cent, Account No. 202,169.

The current transactions of the treasury for the year have been as follows:

*Receipts.*

|                                   |          |
|-----------------------------------|----------|
| Balance from old account .....    | \$387.56 |
| Sale of dinner tickets, 1907..... | 306.00   |
| Sale of Transactions .....        | 32.50    |
| Exchange on checks .....          | .60      |
| Loan, Corn Exchange Bank .....    | 225.00   |
| Dues for 1907 .....               | 80.00    |
| Dues for 1908 .....               | 770.00   |

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Total receipts ..... \$1,801.66

*Expenditures.*

|   |          |
|---|----------|
| Annual dinner, 1907 .....   | \$333.00 |
| Stenographer, 1907 .....  | 100.00   |
| Publishing Transactions, 1907 .....   | 467.38   |
| Repayment of bank loan .....  | 225.00   |
| Interest on loan .....  | 3.45     |
| Stenographer, extra service .....   | 7.50     |
| Programmes and distribution for 1908.....   | 33.50    |
| Printing and stationery .....   | 49.11    |
| Incidentals of Secretary and Treasurer, telegrams, telephones, expressage, postage and sundries ..... | 15.55    |

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Total expenditures ..... \$1,234.49

Balance to new account ..... 567.17

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\$1,801.66



*Statement of Assets and Liabilities.*

|  |          |
|--|----------|
| Assets: De Roaldes Prize Fund, \$500; accrued interest \$10.00 to Jan. 1, 1908; total..... | \$510.00 |
| Current: Unpaid dues .....   | 120.00   |
| Postage in office .....  | 4.72     |
| Bank balance .....   | 567.17   |
|  | <hr/>    |
|  | \$691.89 |
| Liabilities: Due treasurer .....   | \$6.91   |
|  | <hr/>    |
| Net assets .....   | \$684.98 |
| A gain in assets over one year ago of \$201.18.  |          |
| Audited and found correct.   |          |

(Signed) W. SCOTT RENNER,  
J. M. INGERSOLL,  
Auditing Committee.

*Report of the Librarian.*

Your Librarian has to report that at the last meeting of the Association 61 pamphlets and 4 bound books have been added to the library. Respectfully submitted,

J. H. BRYAN, Librarian.

*Report of the Nominating Committee.*

Through its Chairman, Dr. Swain, the Nominating Committee presented the following report:

For President: Dr. A. Coolidge, Jr., Boston, Mass.

For First Vice-President: Dr. Emil Mayer, New York City.

For Second Vice-President: Dr. Francis R. Packard, Phila., Pa.

For Secretary and Treasurer: Dr. James E. Newcomb, New York City.

For Librarian: Dr. Joseph H. Bryan, Washington, D. C.

For Member of the Council (to serve four years): Dr. Herbert S. Birkett, Montreal, Canada.

For Delegate to the Executive Committee of the Triennial Congress of 1910: Dr. William K. Simpson, New York City; Alternate, Dr. Joseph H. Bryan, Washington, D. C.

Place of meeting for 1909: Boston, the date to be selected by the Council.

The following items of business were then taken up:

The Secretary reported the receipt of the following letter from Dr. W. H. Carmalt, New Haven, Conn., Secretary of the Congress of American Physicians and Surgeons:

Dr. James E. Newcomb, Secretary of American Laryngological Association, New York City:

Dear Doctor: The following members of the American Laryngological Association were, according to the list you furnished me, present at your Washington meeting in May last. They did not register, as notified that the rules of the Congress required, consequently did not pay the registration fee of five dollars (\$5), their share toward the local expenses.

In consequence of this neglect on their part, the assessments to the various Societies and Associations are correspondingly increased and the other members must make up the deficiency.

It is for you and your council, or other executive body, to take whatever action you deem fit. I simply report it in explanation of the assessment which the Treasurer will shortly call upon your society to make on the pro rata basis according to the by-laws of the Congress. I am, very respectfully yours,

(Signed) W. H. CARMALT.

Accompanying the letter of Dr. Carmalt were the names of eight Fellows in attendance at the Triennial Congress of 1907 who had failed to pay the registration fee referred to. After some discussion of the matter it was

Voted: That the Treasurer be and hereby is directed to forward to Dr. Carmalt the sum of forty dollars, and to collect said fees from the delinquent Fellows as far as may be possible.

A communication was received stating that it was proposed to form, at the coming meeting of the American Medical Association at Chicago, a national organization of Secretaries of the various medical societies of the country and of medical editors and inviting this Association to send a representative to participate in the organization of the new society. No action was taken.

The Secretary stated that he had been informed that it was pro-

posed that the American Otological Society, the American Laryngological, Rhinological and Otological Society and this Association should take over the management of the Archives of Otology with reference to having it become the official organ of the bodies named. After considerable discussion, it was voted that Council of this Association, together with Dr. Ingals, be appointed a Committee to consider this matter and report at the Congress of 1909.

The Secretary further stated that this Association had been invited to send representatives to the Fifth Pan-American Medical Congress, to be held in Guatemala, Central America, in August, 1908. No action was taken.

The Secretary further stated that he had been advised that the American Laryngological, Rhinological and Otological Society had appointed a Committee on Nomenclature, and that it was desired that for purposes of joint conference a similar committee should be appointed from this Association.

Voted: That such a committee be appointed by the President, to report at the next Congress. The President appointed as said committee, Drs. Coolidge, McKernon and Kyle.

The Secretary stated that he had received a letter from Prof. J. Pease Norton, of Yale University, Executive Secretary of the Committee of One Hundred of the American Association for the Advancement of Science on National Health, the substance of which was as follows:

"The sub-committee of the Committee of One Hundred on Co-operation with other societies appointed by the American Association for the Advancement of Science to advocate a national health organization, begs to call the attention to the Secretary of the American Laryngological Association to its work, and respectfully requests your endorsement of it.

Voted: That the endorsement of this Association be given to the work of said organization.

At the close of the executive session, the scientific programme was resumed, and the remainder of the morning session devoted to papers and discussion. Adjournment at 1 P. M.

The Congress reconvened at 3 P. M. and listened to an address by Prof. Wesley Mills (see page 300). Following the address

came Dr. Makuen's paper on a related topic, and both the address and the paper were discussed. Adjournment at 6 P. M.

In the evening the annual dinner of the Association was held at 7.30 at the Mount Royal Club, Sherbrooke Street, West. Twenty-eight Fellows were in attendance and two guests, Dr. T. G. Roddick, Dean of the Medical Faculty of McGill University, and Prof. Wesley Mills. Illness prevented the attendance of Mr. William Harrison Bradley, U. S. Consul-General at Montreal, who had received an invitation from the Council.

The final session of the Congress convened at 10 A. M. on Wednesday, May 13th. The reading of papers and discussion thereon was concluded and the Association went into executive session. The matter of publishing those papers, the authors of which were not present and which had not been forwarded to the Secretary, was taken up and discussed. It was

Voted: That the papers, the titles of which appear on our programme, in the absence of their authors and which are not now in the hands of the Secretary, be referred to the President and Secretary with power to include said papers in the Transactions or not as, according to the circumstances, they may deem best.

Voted: That hereafter no paper shall be read by title and published in the Transactions that is not in the hands of the Secretary at the time of the annual Congress, except by vote of the Council.

The following motions were offered and adopted:

That the Secretary be directed to convey the Medical Board of the Royal Victoria Hospital and the Medical Board of the Hospital Hotel Dieu the deep sense of obligation felt by the Fellows toward the gentlemen named for courtesies tendered at their respective institutions.

Voted: That the thanks of the Association be transmitted to respectively, the governors of the Montreal Club, the Mount Royal Club, the St. James Club and the University Club for the hospitality tendered to the Fellows of the Association in connection with the present Congress.

That the thanks of the Fellows be tendered to the retiring President, Dr. Birkett, for his unbounded hospitality, shown in so many ways, and for the able manner in which he has presided

over the deliberations of this Congress, and that he be authorized to convey to Mrs. Birkett the compliments and best wishes of the Fellows of the Association.

Dr. Birkett expressed his thanks for the sentiments thus expressed and bespoke a share of them for his associates in the preparation of the X-ray and pathological exhibits.

A vote of thanks was extended to Prof. Wesley Mills for his interesting address, and to the Secretary for his work during the year now closing.

The report of the Nominating Committee was again read by the Secretary, and he was instructed to cast one ballot for the gentlemen named. This was done and they were declared elected. At the request of the retiring President, Drs. Ingals and Bryan escorted the President-elect, Dr. Coolidge, to the chair, and Dr. Casselberry rendered a similar courtesy to the First Vice-President-elect, Dr. Mayer. Dr. Birkett then retired. On taking the chair, Dr. Coolidge expressed his thanks for the honor conferred upon him and the hope that the attendance next year at Boston might be large and participation in the programme hearty.

At 1 P. M. the Congress adjourned.

JAMES E. NEWCOMB, Secretary.

Following adjournment, the Fellows were entertained at lunch at the Royal Victoria Hospital by the Medical Board, who afterward conducted the visitors through the institution. Several post-operative cases of malignant disease of the throat were exhibited.

## ACTIVE FELLOWS.

1909.

1906. BALLENGER, WILLIAM L., Columbus Memorial Building,  
Chicago, Ill.
1908. BARNHILL, JOHN F., 323 North Delaware St., Indianapo-  
lis, Ind.
1880. BEAN, C. E., Germania Life Bldg., St. Paul, Minn.
1904. BERENS, T. PASSMORE, 35 Park Ave., New York.
1893. BIRKETT, HERBERT S., 252 Mountain St., Montreal.
1893. BLISS, ARTHUR AMES, 117 South 20th St., Philadelphia,  
Pa.
1878. BOSWORTH, FRANCKE H., 41 Park Ave., New York.
1901. BROWN, J. PRICE-, 37 Carlton St., Toronto, Can.
1892. BROWN, MOREAU R., 34 Washington St., Chicago, Ill.
1891. BRYAN, JOSEPH H., 818 17th St., Washington, D. C.
1889. CASSELBERRY, WILLIAM E., 34 Washington St., Chicago,  
Ill.
1896. CHAPPELL, WALTER F., 7 East 55th St., New York.
1897. CLARK, J. PAYSON, 71 Marlborough St., Boston, Mass.
1902. COAKLEY, CORNELIUS G., 53 West 56th St., New York.
1899. COBB, FREDERIC C., 11 Marlborough St., Boston, Mass.
1905. COFFIN, LEWIS A., 156 West 58th St., New York.
1878. COHEN, J. SOLIS-, 1824 Chestnut St., Philadelphia, Pa.
1893. COOLIDGE, ALGERNON, JR., 613 Beacon St., Boston, Mass.
1882. DEBLOIS, THOMAS AMORY, 48 Gloucester St., Boston,  
Mass.
1881. DELAVAN, D. BRYSON, 1 East 33d St., New York.
1892. FARLOW, JOHN W., 234 Clarendon St., Boston, Mass.
1901. FREEMAN, WALTER J., 1832 Spruce St., Philadelphia, Pa.
1905. FREER, OTTO T., 34 Washington St., Chicago, Ill.
1879. FRENCH, THOMAS R., 150 Joralemon St., Brooklyn, N. Y.
1897. FROTHINGHAM, RICHARD, Sydenham Bldg., Madison Ave.  
and 58th St., New York.
1907. GIBB, JOSEPH S., 1907 Chestnut St., Philadelphia, Pa.



- 1880. GLEITSMANN, JOSEPH W., Sydenham Building, Madison Ave. and 58th St., New York.
- 1898. GOODALE, JOSEPH L., 258 Beacon St., Boston, Mass.
- 1905. GRAYSON, CHARLES P., 251 South 16th St., Philadelphia, Pa.
- 1906. GREENE, D. CROSBY, JR., 483 Beacon St., Boston, Mass.
- 1905. HALSTED, THOMAS H., 831 University Block, Syracuse, N. Y.
- 1896. HARDIE, T. MELVILLE, 34 Washington St., Chicago, Ill.
- 1903. HARRIS, THOMAS J., 117 East 40th St., New York.
- 1878. HARTMAN, JACOB H., 5 West Franklin St., Baltimore, Md.
- 1888. HINKEL, F. WHITEHILL, 581 Delaware Ave., Buffalo, N. Y.
- 1881. HITCHCOCK, URBAN G., 122 West 49th St., New York.
- 1907. HOLMES, CHRISTIAN R., 8 East 8th St., Cincinnati, Ohio.
- 1893. HOPE, GEORGE B., 133 West 72d St., New York.
- 1895. HOPKINS, FREDERICK E., 25 Harrison Ave., Springfield, Mass.
- 1895. HUBBARD, THOMAS, 205 Ontario St., Toledo, Ohio.
- 1901. HYATT, FRANCK, 1022 14th St., N. W., Washington, D. C.
- 1878. INGALS, E. FLETCHER, 36 Washington St., Chicago, Ill.
- 1904. INGERSOLL, JOHN M., 318 Euclid Ave., S. E., Cleveland, Ohio.
- 1882. IVES, FRANK L., 117 East 30th St., New York.
- 1907. JACKSON, CHEVALIER, 428 Park Building, Pittsburg, Pa.
- 1878. JOHNSTON, SAMUEL, 204 West Monument St., Baltimore, Md.
- 1901. KING, GORDON, 624 Gravier St., New Orleans, La.
- 1885. KNIGHT, CHARLES H., the St. Hubert, 120 West 57th St., New York.
- 1878. KNIGHT, FREDERICK I., 195 Beacon St., Boston, Mass.
- 1898. KYLE, D. BRADEN, 1517 Walnut St., Philadelphia, Pa.
- 1880. LANGMAID, SAMUEL W., 277 Dartmouth St., Boston, Mass.
- 1878. LEFFERTS, GEORGE MOREWOOD, 212 Madison Ave., New York.
- 1894. LELAND, GEORGE A., 354 Commonwealth Ave., Boston, Mass.
- 1903. LINCOLN, WILLIAM, 210 Lennox Bldg., Cleveland, Ohio.
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1886. MACCOY, ALEXANDER W., 216 South 15th St., Philadelphia, Pa.
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1898. MAKUEN, G. HUDSON, 1627 Walnut St., Philadelphia, Pa.
1896. MAYER, EMIL, 25 East 77th St., New York.
1899. MCKERNON, JAMES F., 62 West 52d St., New York.
1904. MOSHER, HARRIS P., 828 Beacon St., Boston, Mass.
1901. MYLES, ROBERT C., 46 West 38th St., New York.
1893. NEWCOMB, JAMES E., 118 West 69th St., New York.
1903. PACKARD, FRANCIS R., 1836 Pine St., Philadelphia, Pa.
1906. PIERCE, NORVAL H., 31 Washington St., Chicago, Ill.
1878. PORTER, WILLIAM, 3886 Washington Ave., St. Louis, Mo.
1908. RANDALL, B. ALEXANDER, 1717 Locust St., Philadelphia, Pa.
1903. RENNER, W. SCOTT, 361 Pearl St., Buffalo, N. Y.
1897. RHODES, J. EDWIN, 100 State St., Chicago, Ill.
1884. RICE, CLARENCE C., 123 East 19th St., New York.
1905. RICHARDS, GEORGE L., 84 North Main St., Fall River, Mass.
1902. RICHARDSON, CHARLES W., 1317 Connecticut Ave., Washington, D. C.
1893. ROALDES, ARTHUR W. DE, 624 Gravier St., New Orleans, La.
1879. ROE, JOHN O., 44 Clinton Ave. South, Rochester, N. Y.
1907. SHAMBAUGH, GEORGE E., 100 State St., Chicago, Ill.
1878. SHURLY, ERNEST L., 32 Adams Ave. W., Detroit, Mich.
1892. SIMPSON, WILLIAM K., 952 Lexington Ave., New York.
1908. SMITH, HARMON, 44 West 49th St., New York.
1889. SWAIN, HENRY L., 232 York St., New Haven, Conn.
1903. THEISEN, CLEMENT F., 172 Washington Ave., Albany, N. Y.
1892. THRASHER, ALLEN B., The Groton, 7th and Race Sts., Cincinnati, Ohio.
1892. WAGNER, HENRY L., Pacific Union Club. San Francisco, Cal.

- 1892. WATSON, ARTHUR W., 126 South 18th St., Philadelphia, Pa.
- 1908. WILSON, J. GORDON, Dept. Anatomy, University of Chicago, Chicago, Ill.
- 1906. WINSLOW, JOHN R., 114 West Franklin St., Baltimore, Md.
- 1905. WOOD, GEORGE B., 129 South 18th St., Philadelphia, Pa.
- 1890. WRIGHT, JONATHAN, 44 West 49th St., New York.

## CORRESPONDING FELLOWS.

- 1885. BRUNS, PAUL, Tübingen, Württemberg, Germany.
- 1896. CHILARI, OTTOKAR, Bellariastrasse 12, Vienna, Austria.
- 1901. COLLIER, MAYO, 145 Harley St., Cavendish Square, London W., England.
- 1890. DE LA SOTA Y LASTRA, R., 7 Calle de Torqueros, Seville, Spain.
- 1893. DESVERNINE, C. M., Cuba 52, Havana, Cuba.
- 1904. FRAENKEL, B., Lennestrasse 5, Berlin W. 9, Germany.
- 1901. GRANT, DUNDAS, 18 Cavendish Square, London W., England.
- 1902. HEYMAN, PAUL, 60 Lützowstrasse, Berlin W., Germany.
- 1894. HOLDEN, E., 13 Central Ave., Newark, N. J.
- 1908. KILLIAN, GUSTAV, Freiburg-im-Breisgau, Germany.
- 1892. KRAUSE, H., Hotel Bellevue, San Remo, Italy.
- 1881. LABUS, CARLO, Piazza Castello 28, Milan, Italy.
- 1902. LERMOYEZ, MARCEL, 20 Rue de la Boetie, Paris, France.
- 1897. LUC, H., 54 Rue de Varenne, Paris, France.
- 1903. MCBRIDE, P., 16 Chester St., Edinburgh, Scotland.
- 1896. MACDONALD, GREVILLE, 85 Harley St., Cavendish Square, London W., England.
- 1894. MACINTYRE, JOHN, 179 Bath St., Glasgow, Scotland.
- 1892. MASSEI, F., 4 Piazza Municipio, Naples, Italy.
- 1887. MOURE, E. J., 25 Cours du Jardin-Public, Bordeaux, France.
- 1896. MYGIND, HOLGER, 31 Havnegade, Copenhagen, Denmark.
- 1901. NATIER, MARCEL, 12 Rue Caumartin, Paris, France.
- 1903. ONODI, A., o'Uctza 12, Buda-Pesth, Hungary.
- 1894. SAJOUS, C. E., 2043 Walnut St., Philadelphia, Pa.

1896. SCHMIEGELOW, ERNEST, ix—2 Mariannengasse 3, Copenhagen, Denmark.
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1902. THOMSON, ST. CLAIR, 28 Queen Anne St., Cavendish Square, London W., England.
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- 1896. Marshall R. Ward, Pittsburg, Pa.
- 1879. Whitfield Ward, New York City.
- \*1886. Benjamin F. Westbrook, Brooklyn, N. Y.
- 1896. Greene V. Woollen, Indianapolis, Ind.

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\*Deceased. †Corresponding Fellow. ‡Honorary Fellow.



INDEX OF THE  
TRANSACTIONS  
OF THE  
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ASSOCIATION  
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