

Transactions

American Laryngological Association

STANDARD COLLECTION

SEVENTY-THIRD VOLUME

"DOCENDO DISCIMUS"

TRANSACTIONS
OF THE
SEVENTY-THIRD ANNUAL MEETING
OF THE
American
Laryngological Association
1952

HELD AT THE ROYAL YORK HOTEL
TORONTO, ONTARIO, CANADA
MAY 23RD AND 24TH, 1952

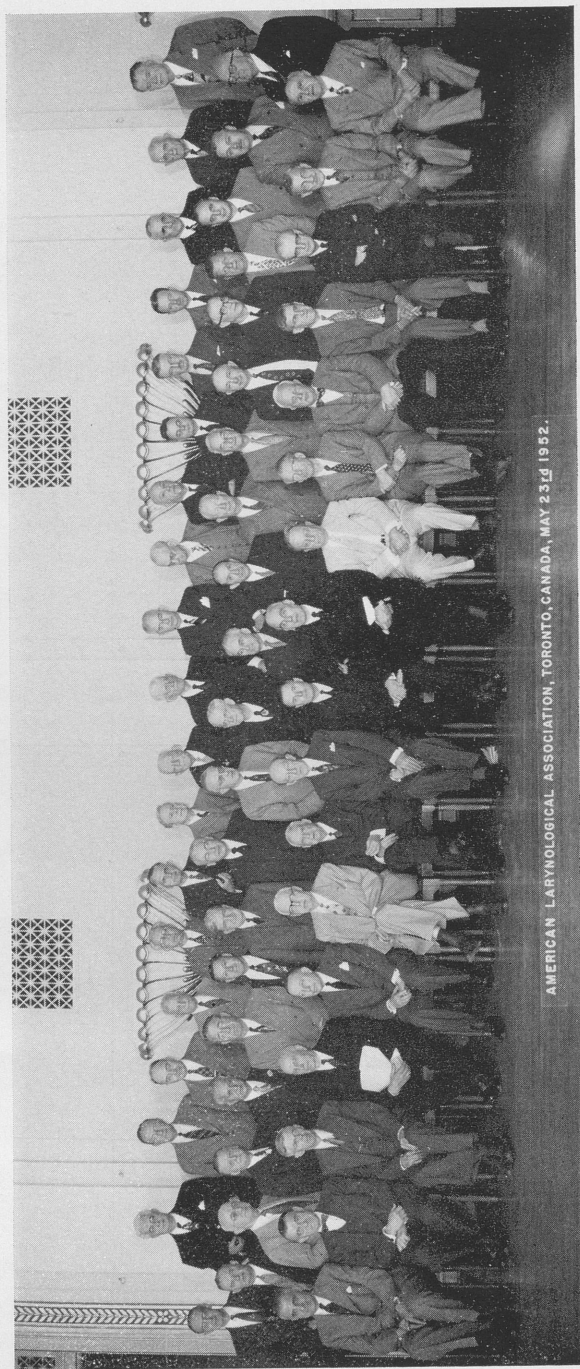
PUBLISHED BY THE ASSOCIATION
ST. LOUIS, MO.
1952

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THOSE IN ATTENDANCE AT THE SEVENTY-THIRD ANNUAL MEETING

Top Row: McMahon, Broyles, A. T. Smith, Hickey, Salinger, Ballenger, Whalen, Foster, Cody, Van Alyea, Lyman, McCall, Putney, W. W. Morrison, Heatley, Wishart, McCaskey, Cunnning.

Second Row: Davison, Tremble, LeJeune, Dixon, L. F. Morrison, Boyden, Miller, Robb, Williams, Maxwell, Shea, Kline, Weille, Johnston, Brown, Blassingame, Galloway, Lierle, Holinger, Ireland, Violé

Front Row: Schenck, Clerf, Hill, Fenton, Proetz, Harkness, Mosher, Taylor, McNally, Mr. Scott Stevenson, Beatty, Simpson, Orton, Coates, Jarvis, Beck, Theobald.

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MAY 23 AND 24, 1952

The Seventy-third Annual Meeting of the American Laryngological Association was called to order by the President, Dr. H. Marshall Taylor, at the Royal York Hotel, Toronto, Canada.

• REGISTRATION

The following Fellows were in attendance:

BALLENGER, HOWARD C.	CUNNING, DANIEL S.
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BECK, AUGUST LEO	DIXON, FRED W.
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CLERF, LOUIS H.	HICKEY, HAROLD L.
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MAXWELL, JAMES H.

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MCCART, HOWARD W. D.

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MCNALLY, WILLIAM J.

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MORRISON, LEWIS F.

MORRISON, WILLIAM W.

MOSHER, HARRIS P.

NASH, C. STEWART

ORTON, HENRY B.

PROETZ, ARTHUR W.

PUTNEY, F. JOHNSON

ROBB, JAMES M.

SALINGER, SAMUEL

SCHENCK, HARRY P.

SHEA, JOHN J.

SIMPSON, W. LIKELY

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TAYLOR, H. MARSHALL

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THE ADDRESS OF THE PRESIDENT

H. MARSHALL TAYLOR, M.D.

JACKSONVILLE, FLA.

It is with profound humility that I express to you my genuine appreciation of the honor you have conferred upon me by elevating me to the presidency of this Association. One who pauses to reflect, as have I, upon the galaxy of illustrious members who have preceded me in this office, could not feel otherwise.

When we speak of science in this advanced day we recognize that it has not changed any of the real laws of nature; but we know it has fathomed many of nature's intricate secrets. If we had no science, nature would present the same phenomena as at present. The heavenly bodies would move with equal regularity and preserve the same relative positions although no system of astronomy had been formed. The laws of gravity and of motion would operate in the same manner as at present if we had no such science as natural philosophy. Before the science of chemistry existed, the affinities of substances for each other were the same as they are now. It is an important truth, and one which cannot be too much impressed upon the human mind in all scientific investigations, that no effort on the part of man can change the laws of nature.

Nevertheless, by the faculties of the mind with which God has endowed us, we are able to examine the properties of the materials surrounding us. Sometimes we can offer opposing forces which retard and alter disease processes, thereby relieving suffering and prolonging life.

It is natural to suppose that the various forms of bacteria had relatively the same virulence and toxicity in the days of the Cro-Magnon man some 20,000 years ago, and in the time of Hippocrates and Galen, as they have today. Yet these poisonous enemies of mankind now have been robbed of much of their disease-producing power by medical science through the wealth of scientific investigation carried on in the modern laboratory by the bacteriologist, the pathologist and the cytologist. Likewise, the cancer cell most likely must

have possessed some of its present characteristics in the early history of human life. Sometimes, however, much of its malignancy has been altered or thwarted by the discovery of the roentgen ray and the splitting of the atom.

The scientist now tells us that 150,000,000 atoms in juxtaposition may measure only one inch. Furthermore, he avers that he can isolate this infinitesimal atom, divide it into its component protons, neutrons and electrons and measure the velocity of the electrons, which may exceed 100,000 miles a second. With this sobering information in mind, who has the temerity to predict the ultimate service to humanity of the beta and gamm rays of radium and the radio active isotopes, for example?

All these facts make us realize that the physician of today lives in an exciting and almost a magic world, with more and more avenues of research constantly being opened to students of medical science. With the phenomenal development of its various branches, where is medicine leading us today? While I am aware that I am speaking to a group of physicians primarily interested in rhinology and laryngology, I am convinced that all of us realize the term specialist properly becomes almost obsolete in some particulars.

Nevertheless, we contemplate the future of our particular branch of medicine with special interest and concern. We may think of it as one of the younger branches. Yet, we are told by the medical historians, Stevenson and Guthrie, in their recent "History of Oto-Laryngology" that the earliest physician known to us by name was a rhinologist. He was Sekhet'enanch, the medical attendant of a Pharaoh of about 3500 B. C. A limestone slab with a drawing of the physician was found in the tomb of the King bearing the inscription, "He healed the King's nostrils." Were we to speculate on the successful therapy employed by this early Egyptian rhinologist, how many of us would surmise that he advised the Pharaoh to discontinue the use of nose drops? As we review the past, the advances made in recent times, many of them by members of this Association, make us take stock of the present as we look ahead to greater achievements.

Like other sciences, the science of medicine has grown through the ages as a result of cumulative knowledge. But for Archimedes, Galileo, Roger Bacon, Newton and Descartes with their conspicuous achievements in the realm of synthetic thinking, Einstein would not

have evolved his theory of relativity. Such acts of synthesis are essential to the upbuilding of the scientific process. Thinking along these lines some 2,000 years ago, Hippocrates, when asked "Who is the physician that is an honor to his profession?" replied:

He who has merited the esteem and confidence of the public by profound knowledge, who has been led through the whole circle of sciences, who moreover neglects not to observe all the causes that may produce disorder in the animal economy.

Adam Politzer, known as the Father of Otology, once said that the essence of medicine could be summed up in these words, "Everything is connected with everything." In his presidential address before this Association in 1920, Dr. Harris P. Mosher observed, "A specialist should forage in every field of medical activity." Such expressions as these, coming from the pen of such savants, should stimulate every physician to keep his eyes and ears open and his mind alert. They serve to remind us of that sense in which the term specialist should ever be outmoded.

Recently, as a subject of discussion the future of otolaryngology has gained great popularity. More and more frequently it seems to find a place on the program of our medical meetings. In fact, it has become so much the vogue as to be almost a fad or a fashion. Unfortunately, some authors and editors have portrayed otolaryngology as "a shrinking specialty," subject to encroachment and erosion from other branches of medicine. The tenor of a recent editorial in a leading medical journal almost intimates that rigor mortis is setting in. If this fad does not soon run its course and this high degree of pessimism is allowed to grow, we may soon be hearing derogatory remarks from radio commentators and seeing disparaging presentations on television. Unless we take aggressive steps to arrest this trend, who knows but that we may yet be confronted with Milton Berle or Humphrey Bogart impersonating the otolaryngologist as the dying gladiator?

Although widely publicized, these gloomy forecasts express only the opinion of the individual essayists and editors who make them. Their import, however, is unfortunate both for our teaching institutions and our special hospitals. Such propaganda invites a dearth of applicants and a consequent let-down in the training program. It follows that students and interns would forego a sufficient training period to make them competent, and the public would inevitably

suffer. For the vociferous minority who seemingly delight to decry our branch of the healing art, let there be a more articulate majority to uphold it.

Even though the antibiotics have made less surgery necessary in some of the sinus and otitic infections, there nevertheless remains to the otolaryngologist today a wider field than many of the older members of the Association had when they began their professional career. It is my observation that the younger physicians now electing to cast with us their lot in medicine are working to capacity and utilizing their training in a broader field than many of their senior colleagues ever have done. Those who come from the best institutions with a background of internships and residences covering three or four years are not only well grounded in diseases of the ear, nose and throat but also versed in deep surgery of the neck, surgery of the salivary glands, bronchoesophagology, reconstructive surgery of the head and neck, allergy, audiology and physical medicine.

Certainly one is fascinated by the innumerable facets of the jewel of otolaryngologic research, especially so upon reading the "Forty Questions in Otolaryngology" posed by Dr. Arthur Proetz in his self-styled role of fool who asks questions wise men cannot answer (*The Annals of Otology, Rhinology & Laryngology*, September, 1951). As for the scope and practical aspects of the practice of otolaryngology, what thoughtful, fair-minded, informed analyst will gainsay its equally broad and intriguing appeal? Fritz Kahn has reminded us that in an opera an entire orchestra plays, but above it sings one human being with a small larynx, and the human voice triumphs over all artificial instruments. Likewise, the trained laryngologist and the skilled rhinologist will always transcend all others in the diagnosis and treatment of diseases of the upper respiratory passages. That is my considered judgment, in which I believe the members of this Association will heartily concur.

It has been said that it is very hard to press gold and diamonds and sapphires into the service of art, but the poor ball of clay which has been pinched by the fingers of Michelangelo may show the sign unmistakable and be treasured for centuries. And so it is with otolaryngology. Let us not lose the vision. Let us sharpen our perceptions. Let us search our hearts and minds and call forth the gift that is within us to mold the clay of our professional knowledge and ex-

perience into contributions that will leave the world better off. It is ours to participate in the synthesis of medical thinking which today ventures to cross the threshold of a new era.

Whither are Aesculapius and the savants of the ages leading us through the achievements of our modern day? In this dawning atomic age, who can visualize the future of the science and the art of medicine? Who indeed would narrow the concept of any branch of the healing art in this epoch of expansion? So great are the possibilities, so broad the concepts, that we may well be mindful of St. Augustine, who centuries ago sought to answer the question, "What was God doing before He made heaven and earth?" Wrote the eminent philosopher and divine, "He was making the hell for the persons who ask that kind of questions."

The questions which yet confound the wise men of otolaryngology are both fascinating and stimulating - and not unanswerable. The quest for the solution to the many problems assures and makes it bright. The Golden Age of Otolaryngology lies ahead.

LARYNGEAL TUBERCULOSIS

RALPH A. FENTON, M.D.

PORTLAND, ORE.

The purpose of this paper is to discuss briefly laryngeal tuberculosis, one of the most distressing complications of the disease, rather than to dwell exclusively on the accomplishments of streptomycin, which is the fashion at the moment in medical literature.

Pain in swallowing was formerly relieved by anesthetic lozenges, later by nerve block, while lactic acid locally, and the electric cautery, did away with much obstructive swelling, and silence gave local rest. Continuing pulmonary involvement led to continuing cough, while tracheal edema followed by stenosis caused still more interference with breathing, and thus further extension into uninvolved lung tissue.^{11, 12}

George Fetterolf reported to this Association in 1914 the autopsy findings on patients dead of tuberculosis in Philadelphia; of these, 83 per cent showed laryngeal involvement.¹ Sir St. Clair Thomson, himself in youth a victim of the disease, in 1919 discussed the prognostic importance of laryngeal tuberculosis and the value of proper laryngeal examination; he looked upon the appearance of this complication, occurring in at least 22 per cent of all sanitarium cases, as most dangerous.² In 1937 he stated: "Tuberculosis of the larynx is particularly interesting on account of its frequency, gravity, and the importance it bears to prognosis."³ But Ormerod has recently reported from London the results of a thirty year statistical study, showing a diminution from 25 per cent in 1921 to two and one-half per cent in 1939.⁴

Current opinion among American phthisiologists may be summarized about as follows: "It is difficult or impossible to obtain accurate statistics regarding the incidence of tubercular laryngitis. In the first place, the incidence seems to vary considerably in different parts of the country, being influenced no doubt by factors such as standard of living, nationality, race, education, etc. The incidence among admissions to tuberculosis institutions likewise varies according to the type of institution. For instance, some institutions will

draw only from the higher economic levels. On the other hand many state and county institutions, especially in areas where there is a bed shortage, select patients for admission on the basis of their prospects for recovery, their need or suitability for collapse therapy, surgery, etc. In institutions established primarily for segregation and custodial care, with many terminal cases, the incidence is naturally high. Thirty years ago, tuberculous laryngitis occurred as a complication of far advanced pulmonary tuberculosis in perhaps fifteen per cent of cases. In moderately advanced pulmonary tuberculosis not more than two or three per cent were so complicated; in minimal pulmonary tuberculosis not more than two or three per cent. At present, tuberculous laryngitis is thought to occur as a complication in only about four per cent of all cases.”^{5, 11, 12}

How has this beneficent change come about?

Several factors have been at work, notably control of coughing, by phrenicotomy and by various types of lung surgery—collapse therapy, rib resection, and removal of hopelessly involved lung tissue.¹¹ Early recognition of the disease through chest plates has of course contributed much to recent therapy. More recently still, the use of streptomycin, often coupled with paraaminosalicylic acid, has in spite of dangers to the auditory and vestibular nerves cleared up throat lesions rapidly. But it is not necessarily curative for extensive pulmonary damage.¹⁰⁻¹²

Most of all, freedom from the rasping, tearing assault of sputum upon the interarytenoid space, vocal cords and epiglottis has done away with the implantation of virulent germs into these regions. Particularly, when patients became so weak that it was hard for them to expel sputum, thus permitting drying and crust formation, the eventual removal of such crusts would tear off masses of mucosal tissue and leave sensory nerves exposed. Granulomatous masses, often partially occluding the glottis, were materially reduced by Spencer's technique of electrocautery;⁶ but the efforts involved in coughing often reinoculated such healing tissues. “Cauterization of tuberculous lesions of the larynx, pharynx, mouth, gums, etc. continues to hold an important place, but it is the only one of the older methods of treatment not displaced by the antibiotics. Of course, the tubercle bacillus soon becomes resistant to streptomycin, but less readily to the newer dihydrostreptomycin.” . . . “We believe the antibiotics at first

arrest the disease, and then cure it, except in long standing or late and advanced cases."⁷⁻¹²

However, long years before the use of streptomycin, attempts to put the injured lung at rest, or to remove its damaged or cavitated portions, were successful.^{5, 11} Thus coughing was cut down, and habitation to cough troches and narcotic medicines became unnecessary. Management of tracheal ulceration has been much improved by antibiotics, and the heavy stenotic scars resulting from strong silver nitrate or actual cautery are now rarely seen.

Chest films by public health services are now screening out many early cases which formerly became severe before coming to treatment. Use of BCG vaccine prophylaxis has, in Europe, Mexico, South America and Japan, apparently cut down the incidence of juvenile tuberculosis, and many epidemiologists hope it may soon be generally employed in the United States and Canada. Economic conditions, the privations of war, lack of essential protein food, all play a part in continued susceptibility to pulmonary tuberculosis.⁴

Nowadays, younger laryngologists, unless they have had residences in sanatoria, may never have seen a case of tuberculosis of the larynx; and the average practitioner rarely sees one in private practice.

Like diphtheria, it is a disappearing disease.

In the incidence of tubercular laryngitis, the reduction of 83 per cent in 1914 to some two per cent today is one of the outstanding achievements of modern medicine; and we may feel that one of the severest complications of tuberculosis has been practically wiped out, thanks to the control of coughing, and, more recently to streptomycin and to certain less toxic but potent drugs still under test, such as isoniazin hydrazide.

No older laryngologist will ever forget these pitiful patients who followed him with pleading eyes in clinics and sanatoria, asking voicelessly for relief of pain and difficulty in breathing.

1020 S. W. TAYLOR STREET.

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DISCUSSION

AUGUST L. BECK, M.D.: I would like to call attention to the fact that in tuberculosis the involvement of the larynx is not always a late manifestation.

I am one of three brothers. My younger brother contracted tuberculosis, a definite lesion, in his upper left lobe, cavitation and after between four and five years died after a violent hemorrhage.

My other brother one summer went off on a vacation. He had been over-doing. He had just finished in college and he began to cough. He developed what seemed to be an acute respiratory infection. Along with this he became hoarse. He came to me saying that he had a cold. I noticed he had a cough. I said, "Are you bringing up, are you raising anything?"

He said, "Yes, I am."

I gave him a bottle. I said, "All right, tomorrow or the next day you bring me some sputum that you cough up early in the morning." I instructed him as to how to do it. I examined his larynx and I saw hyperemic larynx, no specific areas of involvement, but he was definitely hoarse. I could not account for all of it but to make a long story short, the sputum report came back strongly positive, lots of acid-fast bacilli in it. He had rales in his chest.

I took him right up to Saranac. Dr. Baldwin, up there, said, after running around his chest with the stethoscope like a mouse running around on the floor, there was plenty of tuberculosis. This was before the days of x-ray. He spent a year in Saranac, the first six months of which he was flat on his back and made a complete arrested recovery.

I wanted to call attention to the fact that very early in pulmonary tuberculosis you may have laryngeal symptoms and see a hyperemic larynx in which case a sputum examination is immediately indicated.

MR. R. SCOTT STEVENSON (London, England): It is probably an impertinence for me to follow Dr. Ralph Fenton on this subject but it is one in which I have long taken a particular interest, and I am a laryngologist to three sanatoria in our country.

About six months ago I got a letter from the Chairman of the Management Committee of one of them asking what justification there was for a laryngologist to continue to attend regularly the sanatorium. I dug out my old arguments, put them very strongly to him and then I thought I will show him. I will go into all the cases I have seen and produce a paper on this subject. I had written the paper with Professor Heath of our country. I think in 1940 I published the paper of a series of something like 480 cases of tuberculous laryngitis seen in five years in this very same sanatorium. I went over the case sheets very carefully for the five years, just since the war and I found I only got 45 cases of tuberculous laryngitis, so I thought I better shut up. But it shows very plainly I think how true is what Dr. Fenton has said.

I said half in joke and half truly, not long ago at a meeting, that nowadays I don't diagnose tuberculous laryngitis. The assistant in the ward starts the patient off on streptomycin and p-amino hippurate. At the time I see it, it is healed. At the sanatorium I spend most of the time picking wax out of their ears instead of seeing the tuberculous larynges. It is very striking.

The other speaker mentioned another subject in which I was very interested. About 1932 I published a paper on, I think, five cases I had seen in which the first sign of trouble was hoarseness. They came to me with hoarseness and we found eventually that they had an involvement of the lung by tuberculosis. I thought I had gotten something new and was going to make the most of it but I thought I better do, as I usually do in such matters and see what Osler said about it. There it was in Osler all right.

RALPH A. FENTON, M.D. (closing): I am very grateful that Dr. Beck brought up the matter of the earliest sign being hoarseness. I did not go into that but it is very important to recognize the earliest appearances of lesions in the larynx.

I recall a similar case to that of his brother. This was a young married woman, who had been in vivid, good health, perfectly fine rosy cheeks and all that sort of thing, but she had noticed a little flushing up in the afternoons and that she had been a little hoarse. This was before the time when all the girls had begun to smoke; so we looked at her larynx, and I was not particularly surprised, but shocked really, to see that there was a little roughness, a little redness along both vocal cords; and it was discovered then, and that was before the days of mass x-rays. We did, however, have some made and it was found by Dr. Matson, a prominent tuberculosis man in our town, that she had a very distinct and extensive miliary tuberculosis, from which she promptly died. It was over both lungs, and she did not last long.

I am glad to hear from Mr. Stevenson. The paper that he mentioned was quoted by Ormerod in this survey that I cited, and that time is still running out in Britain, in spite of the marvelous achievements of the Health Service.

Now about Sir St. Clair Thompson: when he was here in 1919 he talked about himself, and he mentioned the fact that he kept quiet. During his whole year up in Switzerland, Sir St. Clair kept absolutely silent and he said, "That was quite a deprivation for me, but I did," and he said, "Part of the healing of my beginning tuberculous laryngitis I ascribe to the fact that I did keep absolutely silent."

BRANCHIOGENIC CARCINOMA

FREDERICK T. HILL, M.D.

AND

IRVING I. GOODOF, M.D.

WATERVILLE, ME.

At the 1946 meeting of the American Laryngological Association Blassingame¹ presented an excellent paper on "Congenital Cysts and Fistulae About the Head and Neck." Blassingame devoted considerable attention to the work of Winglowski who opposed the generally accepted theory of the branchial origin of these cysts and stated that these were more likely to be remnants of the pharyngo-thymic duct formed by the "anlage" of the thymus in its descent to the post-sternal space.

However, the term, branchiogenic cyst, by long usage, is generally accepted as applied to these cervical cysts of embryonic derivation. Of even more controversial nature than their exact origin would seem to be the question as to whether these cysts ever became primary sites of malignant changes. Since Lorenz in 1913 applied the term branchiogenic carcinoma to the condition first described by Von Volkman in 1882, this diagnosis has appeared from time to time in the literature and most text-books in pathology devote at least reference to it. Moore² states that epidermoid carcinomas are associated with branchiogenic cysts in many cases and "there seems no doubt that a carcinoma may take origin from the epithelium of a branchiogenic cyst. The presence of numerous lymphoid follicles serves as histologic evidence of their origin."

Crile and Kearns,³ in 1935, stated that this was a distinct pathological entity, although the mode of origin was obscure. While they found fewer than 100 cases reported in the literature they added 28 of their own. They described the clinical picture, stating it was one of rather rapid growth, that the tumor was adherent to the deeper structures of the neck but not to the skin, and that there might be involvement of adjacent cranial nerves as well as the cervical

sympathetics. Pain was sometimes referred to the face, occiput, base of the tongue, shoulder, or arm. The differential diagnosis included tuberculosis, Hodgkins disease, mixed tumor, lipoma, lymphadenitis, cystic hygroma, hemangioma, and aneurysm. The pathological findings were those of squamous cell carcinoma together with evidence of a cyst wall and often a definite capsule infiltrated by tumor tissue. In their opinion the pathogenesis was an embryonic rest stimulated to neoplastic activity. While the prognosis was poor they felt that a combination of surgery and irradiation offered the best results.

Hanover and Clark⁴ used the terms "adenolymphoma (papillary cystadenoma lymphomatosum) or branchioma" and stated that origin from rests of branchiogenetic entoderm or heterotopic rests of eustachian tube or pharyngeal entoderm was the most tenable theory.

Jackson⁵ states that "branchiogenic fistulous tracts may occur and undergo malignant change."

In 1949 Ward, Hendred and Chambers⁶ in a study of branchiogenic anomalies reported seven cases of branchiogenic carcinoma, three of which showed histologic evidence of originating in the cyst wall. They stated that the microscopic findings were those of squamous cell carcinoma together with a cyst wall thickened and infiltrated with tumor cells or, if small, almost entirely replaced with tumor. The tumor cells appeared arranged in whorls or branching cords with very little connective tissue stroma. Cell nests with pearl formations could be noted. Usually the tumor was undifferentiated.

On the other hand Martin⁷ in 1948, while granting the theoretical possibility of cancer arising in a branchiogenic cyst, stated that the diagnosis was rarely justifiable and that the condition was more likely to be secondary to a silent primary lesion in the pharynx or larynx. He doubted the validity of the diagnosis in the cases reported, saying that he had found no definite evidence that any such specific tumor existed and that the only acceptable proof of such would be the demonstration of cancer actually arising in the wall of a branchiogenic cyst.

In a later communication⁸ published in 1950, out of some 250 cases reported by 34 different authors, Martin found only three, in a series of 80 reported by Oliver⁹ from Johns Hopkins Laboratory of Surgical Pathology, which presented reasonable evidence of branchio-

genic malignancy. He stated that such a diagnosis must rest upon the assumption that there was no primary lesion elsewhere; that frequently the first, and for sometime the only, manifestation of a silent malignant tumor in the mouth, nasopharynx, pharynx, larynx or even below the clavicle, was a metastasis noted in the neck. If this occurred as a pathological entity he felt it should be found in the embryologically similar thyro-glossal duct cyst, yet no such cases had been reported. Commenting upon the difficulty in pathologically differentiating between metastatic and branchiogenic carcinoma, he stated that a definite diagnosis could not be made on a histologic basis and that the actual existence of branchiogenic carcinoma as a clinical entity was hypothetical.

It is rather difficult to reconcile these conflicting views. Obviously the clinical and microscopic findings in the cases reported confirm the presence of both branchiogenic cyst and carcinomatous tissue. It is then purely a question as to the point of origin of the malignant process. Is it primary branchiogenic carcinoma, or metastatic carcinoma coincident with branchiogenic cyst? While at first hand this question of origin might seem rather academic, inasmuch as the immediate management would be similar, it becomes extremely important when one considers the possibility of an overlooked or silent primary lesion elsewhere.

Further pursuing this question one might consider whether a metastatic process in the neck could invade the branchiogenic cyst, or conversely, could a primary lesion within the cyst itself extend to surrounding tissues, even conceivably into the tonsillar fossa.

The following case is reported—not as suggesting any solution to this controversy but merely for purposes of discussion:

REPORT OF A CASE

F. D., male, aged 39, was seen with a history of a tumor of the neck of some five months' duration. Examination of nose, nasopharynx, pharynx, and larynx was entirely negative. He had small tonsils which appeared normal. Operation revealed a characteristic cyst 4 cm in diameter with a stalk leading upward and apparently becoming obliterated in the region of the muscular bed of the tonsillar fossa.

The pathological report was as follows: "Sinus-like structure lined by papillary processes covered with stratified squamous and

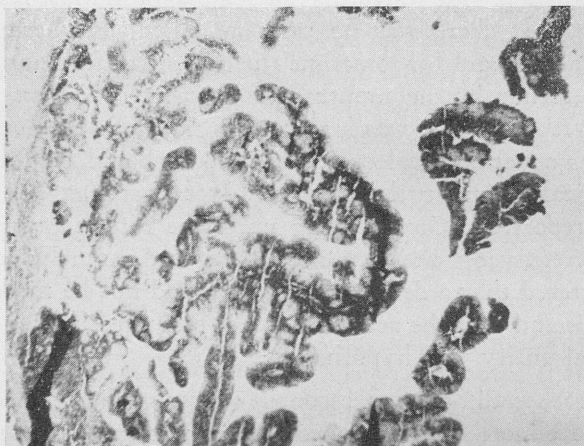


Fig. 1.—Specimen from last surgical procedure, showing papillary nature of the tumor.

transitional epithelium—marked lymphoid stroma. Wall dense and fibrous. Some areas show granulation tissue with cellular infiltration suggesting frequent or long-standing infection. Large collections of fat-laden macrophages in the walls. No evidence of malignancy. Diagnosis: Branchiogenic Cyst—Chronic Inflammation.”

The patient made an apparently uneventful recovery and was free of symptoms for 13 months. Then he noted a small swelling just below the scar of the previous operation. This had increased in size slowly when seen eight months later, or 21 months after operation. He was readmitted to the hospital where operation revealed a cystic tumor apparently similar to that previously removed but without a demonstrable stalk. This was markedly adherent to the underlying tissues making it necessary to ligate and resect a portion of the internal jugular vein.

The pathological report was as follows: “A cystic structure lined by transitional epithelium with suggestion of stratified epithelium in some areas—somewhat papillary type of architecture. Minimal anaplasia of some groups of cells. Diagnosis: Branchiogenic Cyst.

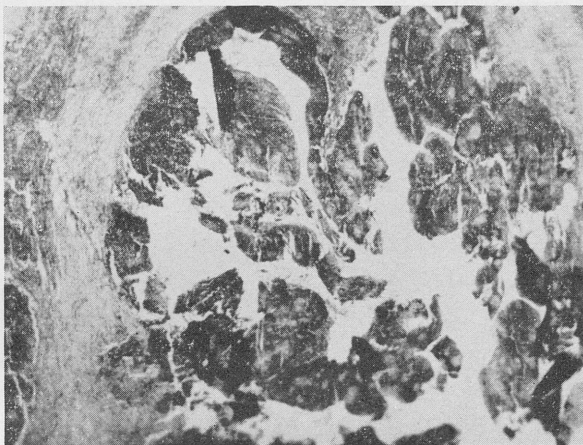


Fig. 2.—Specimen from last surgical procedure, showing papillary architecture, epithelial hyperplasia, and, at the top of the field, invasive tumor and lymphoid tissue.

Again the convalescence was apparently uneventful. Three months later, however, the patient reported, showing a large indurated tumor posterior to the original site and presenting under the margin of the sternomastoid muscle. Biopsy report was “branchiogenic cyst.”

Employing an inverted T incision as advocated by Mosher, a large indurated tumor surrounded by inflammatory tissue was found underneath the sternomastoid muscle. Following operation the patient developed a right recurrent laryngeal paralysis which persisted for 17 days before mobility was restored.

The pathological report at first was returned as branchiogenic cyst but careful re-examination of serial sections revealed nests of anaplastic cells and the diagnosis was changed to “epidermoid carcinoma, grade II-III, arising in a branchial cyst.”

Deep roentgen therapy was now instituted. The patient seemed again to be making satisfactory progress when ten months later, or 39 months after first noting the presence of the cyst and 35 months after the first operation, a small neoplastic-appearing mass was noted in the lower portion of the right tonsillar fossa. Biopsy reported was

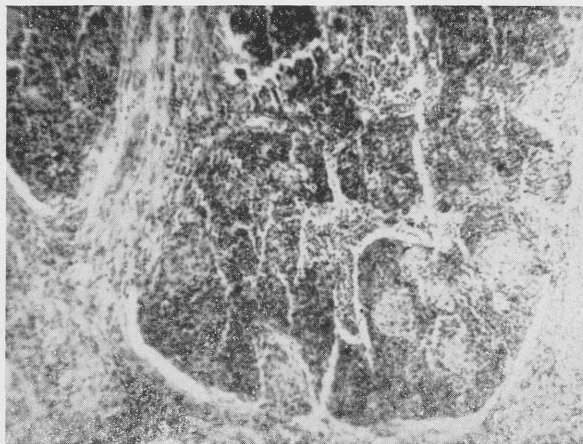


Fig. 3.—Biopsy of tonsil, showing epidermoid carcinoma growing in sheets, with epithelial pearls and some necrosis.

epidermoid carcinoma, grade III-IV. Interstitial irradiation, with a dosage of 3300 mc hours, was combined with a second course of external roentgen therapy. This resulted in complete regression of the pharyngeal tumor and to date (some two years now) there has been no evidence of recurrence in the neck.

The original specimen in this case was the only one which showed the characteristic architecture of a branchial cleft cyst. The epithelium, mainly stratified squamous in type, overlying active lymphoid tissue, with a scanty fibrous wall, provides incontrovertible evidence of the type of lesion involved. Subsequent specimens, while suggestive of this type of tissue, showed sufficient distortion of architecture as to be inconclusive in themselves. The last operative procedure produced a mass of tissue which grossly was not demonstrably cystic, but which microscopically showed a distinctly cystic architecture with papillary masses of epithelium which was definitely anaplastic and rich in mitotic activity. The stroma was fibrous, and showed pronounced lymphocytic infiltration, with some areas actually suggesting lymphoid tissue. This appearance is certainly suggestive of residual branchial tissue, or possibly, an attempt on the part of the tumor to form tissue resembling that of the respiratory tract.

The question of possible origin of carcinoma in a pre-existing branchial cleft cyst has been argued on both sides for some time. There is no denying the fact that all tissues of the body are susceptible to the development of malignancy in some degree. Many developmental anomalies show an even greater tendency to this type of change. Although it is true that branchial cleft cysts are a common site of lymph node metastasis from lesions of the mouth and throat, the demonstration of characteristic branchial cleft architecture on original examination certainly suggests consideration of this structure as the site of origin. The fact that the tumor recurred and finally involved the tonsillar area after three years, is still consistent with direct extension, especially since a silent carcinoma of the tonsil with spread to the neck would be expected to have involved additional nodes after this length of time. The occurrence of a primary carcinoma of the tonsil in a 39 year old man is also sufficiently unusual as to add weight to the opinion that the origin of the tumor was in some other area.

Many findings in this case are difficult to satisfactorily explain. The first operation revealed what was apparently a typical branchiogenic cyst except, possibly, for the histologic finding suggesting chronic inflammation. The apparent obliteration of the stalk distal to the tonsillar fossa may have been due to a technical error, resulting in leaving branchial tissue in that region. But the cyst proper was clearly developed and apparently removed, and the recurrence was below the level of the original incision and was not evident for over a year. Was the microscopic finding of minimal anaplasia in the second specimen of any significance? The third operation disclosed definitely carcinomatous tissue. Was the neoplasm which later made its appearance in the tonsillar region a primary lesion, long silent, or an extension from the pharyngeal end of the stalk? Removal of the tonsil and inversion of the stalk into the pharynx would have been advisable in the light of subsequent developments, although many authorities, including Lahey,¹⁰ no longer feel that this is necessary and it has not been the writer's practice to do so routinely.

The diagnosis of branchiogenic carcinoma was accepted as the best explanation of the recurrence of the tumor. At the same time we must admit the possibility of technical error as in both earlier operations we were limited by the type of incision commonly em-

ployed for the removal of branchiogenic cysts and only at the last, with the inverted T incision, was there really adequate exposure.

Obviously we cannot definitely determine in our case the primary site of the carcinoma. Considering the sequence of events, however, two possibilities come to mind. Was this the result of metastases from a silent neoplasm in the tonsillar fossa invading branchial tissue inadvertently left at the first operation? Or could embryonic tissue remaining in the pharyngeal end of the stalk, stimulated by chronic inflammation develop malignancy with the eventual appearance of neoplastic tissue in the pharynx? The first would seem in accord with Martin's views and, considering his extensive experience, should not be discarded, although rather at variance with our operative findings. At the same time it is interesting to speculate upon the second possibility, which would lend weight to the views of those who consider branchiogenic carcinoma a distinct pathological entity.

COMMENT

Reviewing our experience with the case, several points of practical value are suggested:

1. The necessity of wide exposure of the operative field in any case showing adherence to underlying tissue, or in which there is recurrence of the tumor.
2. The importance of serial section study in all suspected cases. Reliance on biopsies may be misleading.
3. Inversion of the stalk, at least in certain cases.
4. As so forcibly brought out by Martin, the persistent search for a silent primary lesion in all malignant manifestations in the neck.

THAYER HOSPITAL.

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DISCUSSION

I. I. GOODOF, M.D.: The first tissue received was a fairly characteristic branchial cleft cyst with a lining of stratified squamous epithelium in most areas and some respiratory epithelium elsewhere. Beneath the epithelium was a large amount of very active lymphoid tissue and a thin fibrous wall surrounding this. This is a fairly characteristic description of a branchial cleft cyst and reasonably incontrovertible evidence of the character of the original lesion.

The next specimen, being a second operation, was not a clear cut specimen. It did not show a definite cyst but here again there were areas where the lining was squamous stratified in type overlying lymphoid tissue and the only diagnosis I could make again was branchial cleft cyst with a fair amount of chronic inflammation surrounding it.

In the final specimen, after repeated search, we found a picture that was entirely different. If we can have the first slide (Slide) I think we can point this out a little more readily. As you see, now the character of the lesion is that of a papillary structure with primarily stratified squamous epithelium with a very thin wall, without very much in the line of lymphoid tissue. The papillary masses were large, and spread throughout a large portion of the specimen. We could not take them all in one field.

(Slide) The next slide will give you another view of this same thing under magnification. You see the pronounced piling up of epithelial cells with what might be interpreted as a relatively insecure basement membrane, but at least the papillary appearance is obvious.

(Slide) This slide shows the distinct invasion that has taken place. We now have the fibrous stroma infiltrated with lymphocytes with the solid masses of epithelial cells spreading down into the tissue and making a definite diagnosis of malignancy. There is some keratinization. There is a suggestion of epithelial pearl formation. The entire picture now is that of epidermoid carcinoma, which we graded somewhere between Grades II and III.

This is one showing the papillary appearance and again the irregularity of the basement membrane and invasion of the wall, deeper in the tissue.

The question of the origin of this lesion is one that has been argued, as Dr. Hill has mentioned, on both sides for a long time. The statements that have been made to the effect that no such lesion is possible in such a structure requires a little consideration, because I think everyone will agree that no tissue in the body is actually immune to the development of a malignant process. As a matter of fact, if we consider congenital anomalies in general, we might be inclined to feel that they are somewhat more apt to develop malignant change than the normal tissues. Here we can cite, for example, the tumors which arise in Rathke's pouch the cranio pharyngioma. The statement that malignant tumors have not been found to arise in the thyroglossal duct cyst is a debatable one. The most recent issue of *The American Surgeon* reports a case and a review of the literature with four instances of malignancy arising in thyroglossal cysts. We cannot rule out empirically this type of tumor developing in a branchial cleft cyst. There is no question but what the location of a branchial cleft cyst is a common one for metastases from a lesion in the pharynx. The area is one which is rich in lymph nodes and certainly metastases from a tonsillar tumor might be expected in this area. However, the fact that this is a 39 year old man and that nothing was found in the tonsil for over three years I think makes it relatively unlikely that this was a primary tonsillar carcinoma. We don't see them quite as often in 39 year old as we do in 59 and 69 year old patients. The fact that the tumor as we have found it is a papillary one primarily, that it shows active lymphoid tissue in the wall, that the structure is cystic, all add some evidence to the probability that this is a primary branchial cleft lesion. The tumors that we find in the lymph nodes of the neck as metastases in general are solid tumors with possible necrosis in the centers but not with a papillary type of growth.

HENRY B. ORTON, M.D.: This is certainly a most interesting case. As has already been brought out these branchiogenetic carcinomas are very rare indeed. I have yet to see a malignancy of a branchiogenic type. I have, however, seen a thyroglossal duct carcinoma.

The embryology of these cysts is important. A diagnostic point between a cyst and carcinoma, that in a cyst the light on transillumination would show through a cyst but would not in carcinoma.

As Dr. Hill presented this paper I made a provisional diagnosis at the time that this was probably an original carcinoma of the tonsil.

BERNARD J. McMAHON, M.D.: On reading Dr. Hill's paper, I realize more than ever there are no two such words in medicine as "never" and "always."

Dr. Hill has presented a most interesting and provocative case which he was fortunate in being able to follow through from its incipency.

He has established the fact that he was dealing with a branchiogenetic cyst, which apparently progressed from the benign to the malignant lesion. Whether this progression was the result of an activation of the epithelial cells of the walls or the cyst or the stalk, or of an isolated restive cell by surgery is conjectural. There is evidence that the malignant changes may have been present at the removal of the first tumor which revealed a minimal anaplasia of some groups of cells in groups. The recurrence of a larger tumor, malignant in nature, immediately adjacent to the site of the original tumor after three months certainly suggests a continuation of the growth as a fairly well differentiated epidermoid carcinoma, a type which theoretically would respond poorly to Roentgen ray therapy. This was corroborated by the appearance in the right tonsillar fossa ten

months later of the poorly differentiated neoplastic growth which did respond to Roentgen therapy as would be expected.

The pertinent facts are that the entire process was limited to the zone of the branchial cleft and the final appearance of the malignancy at its medial extremity. There was no infiltration or extension into the more distal tissues of the neck nor was there involvement of the lymph glandular systems even after five years. It would, therefore, seem to me that Dr. Hill is justified in considering this a true branchiogenetic carcinoma.

FRANCIS L. WELLS, M.D.: A few years ago I had an experience somewhat similar to that which has been described by Dr. Hill. I had a patient who had a swelling about 2 cm in diameter in the region of the left subdiaphragmatic lymph nodes. I thought it was inflammatory but I told the patient if it had not disappeared within three months that he would have to have it excised for biopsy. Removal of abscessed teeth and tonsils failed to affect this swelling and it was excised at the Deaconess Hospital in Boston. Dr. Shields Warren personally took the specimen to the laboratory and returned a diagnosis of a branchial cleft cyst. I was feeling very satisfied with myself at the moment and happened to see Dr. Frank Lahey in the Surgeons' Room. We discussed this case and he mentioned how difficult it is to make a diagnosis of tumor in the neck. Dr. Lahey also mentioned that malignancy was often unsuspected. A couple of days later Dr. Warren asked me to come by his laboratory and informed me that he had done a serial sectioning of this cyst. They take the cyst and go through it in a special manner so that they don't miss anything they say pathologically. He told me it was a papillary cyst adenocarcinoma of aberrant thyroid tissue.

A block dissection of the neck was done on that side and half of the thyroid was removed. The patient is well about five years later.

HOWARD W. D. MCCART, M.D.: I agree with Dr. Orton that most of these branchiogenetic cysts are not malignant. However, one should always be on his guard. Just recently I saw a patient from whom the general surgeons removed a branchiocyst. The diagnosis was malignancy and the patient was well for six months. I was asked to esophagoscope him and he had a carcinoma of the esophagus. I think that perhaps he had extension from the esophagus at the original operation.

FREDERICK T. HILL, M.D. (closing): With regard to Dr. McMahon's discussion, I would like to point out that the anaplasia first noted in this lesion was at the time of the removal of the second tumor, which was 21 months after the original operation. I think without doubt the original cyst as seen was a typical branchiogenetic cyst (clinically). It had every characteristic of that and we have this long period of 21 months before any difficulty developed. It was not transilluminated, Dr. Orton, but it had all the typical characteristics of a cyst.

I am very much interested that Dr. Orton has seen a thyroglossal duct carcinoma. Until Dr. Goodoff mentioned this report just recently we had not been able to find any reference to this condition.

I would agree with Dr. Wells most heartily of the importance of serial sections in all these suspected cases. That is what finally proved our diagnosis. We did not have a slide here of the section of the tonsil. Dr. Goodoff perhaps would be willing to give his appraisal of the section from the tonsillar neoplasm.

DR. GOODOFF: The same tumor of about the same grade, epidermoid Grade II to III.

DR. HILL: It was perfectly similar in every way.

XLIII

THE SUPRAHYOID APPROACH TO SURGICAL LESIONS AT THE BASE OF THE TONGUE

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MEMPHIS, TENN.

The feasibility of employing a suprahyoid approach to tumors situated on the base of the tongue has long been recognized. The anatomist, Vidal de Cassis,¹ in 1826, was the first to suggest an incision of the anterior wall of the pharynx as being the proper surgical approach to lesions in this area.

Malgaigne,² in 1834, recognized that the entrance to the hypopharyngeal space by an incision through the anterior wall of that space immediately above the hyoid bone should be a distinct advantage in dealing with lesions situated on the base of the tongue and the tip of the epiglottis. His deductions were based on studies of autopsy material. It was not until the latter part of the nineteenth Century however, that the anterior pharyngotomy approach was employed for tumors inside the pharynx. It would appear, from the literature on the subject, that the surgeons who first performed transverse pharyngotomies obtained their cues from observations of would-be suicides.

Jeremitsch,³ in 1895, reported an attempted suicide in which the cut, above the hyoid bone, was a true suprahyoid pharyngotomy. His description of the wound was as follows: "On the front of the neck, about 2 mm over the tongue bone and parallel with it, the cut is 12 cm, reaching the back part of the pharynx, it stretches from one nodding muscle to the other, gaps widely but shows clean edges. When he coughs and throws his head backward, the injury gaps still more widely and the uninjured epiglottis rises. In both corners of the injury we could see the inwardly situated jugular veins. No hemorrhage." So impressed was Jeremitsch with the findings in this suicidal case that he declared his intention of performing all future operations in the pharyngeal cavity in the area of the root of the tongue with the same type of incision as used by the suicide.

Grünwald,⁴ another influential writer of that period, observed that the suicides had been the teachers of the surgeons regarding transverse pharyngotomy. The experience gained from such cases, he said, may lead to professional application of the principles of suprahyoid pharyngotomy.

Jeremitsch emphasized two significant facts which were outstanding in his case: First, bleeding was minimum. This observation can be well understood, since in the suprahyoid incision no large blood vessels are encountered in the site of the incision. Parenthetically, neither are nerves encountered. Second the favorable disposition of neck wounds to heal. He quoted from the literature and cited statistics from Billroth's *Manual of General Surgery* which supported this thesis. This favorable tendency for neck wounds to heal was also observed by Thompson⁵ on soldiers whom he attended in English hospitals following the battle of Waterloo.

Dieffenbach⁶ emphasized that injuries on the neck, committed by a second person, healed quickly without complications.

Our purpose in reciting these data, some of which may at first appear irrelevant, is to emphasize that there was an early recognition of the apparent suitability of the transverse pharyngotomy approach to the pharynx. The operation, in spite of its merits, soon passed into eclipse and was little heard of until very recent times. This decline in the popularity of the operation was all the more strange since the suprahyoid pharyngotomy gives the most satisfactory approach to tumors at the base of the skull, in the area of the epipharynx, and to tumors of the choanal regions and on the posterior surface of the soft palate.

Hofmann,⁷ in a paper before the German Surgical Society in 1907, gave a comprehensive survey of surgical procedures dealing with tumors of the nasopharynx. He concluded that "the suprahyoid pharyngotomy proves excellent for observing the operative field in the nasopharynx. Since the walls of the pharynx are extremely extendable one can, by using retractors, pull the walls of the pharynx aside and the operation may be performed under the steady eye of the surgeon." He showed that by splitting the soft and hard palate in combination with the suprahyoid pharyngotomy, a broad approach can be easily obtained. This he contrasted sharply with the very bloody approaches of the other methods, such as, the Kocher⁸

method, the method used at the Bergman Clinic,⁹ Lagenbeck's¹⁰ Hertle's¹¹ and Garre's.¹² In evaluating the magnitude of the operation, he stated, since suprahyoid pharyngotomy involves no bone surgery, and does not cause much bleeding, one may consider it a minor operation. On the other hand, from the broad view of the operative field, one must judge it as radical as the other methods. Hofmann pointed out that, anatomically, the distance from the roof of the pharynx to the opening in the suprahyoid incision is no greater than that characterizing the Kocher operation where the septum and nasal cartilages are always in the surgeon's way. Also, when compared with Kronlein's¹³ experiments using the lateral pharyngotomy, the superiority of the symmetrical approach to the epipharynx was clearly apparent. Even in the Lagenbeck operation with the resection of the lower jaw added to a lateral pharyngotomy, the same free look into the epipharynx was not obtained. The technique described by Hofmann for the suprahyoid pharyngotomy is as follows:

"The head hangs comfortably over the end of the table. An incision is made above the hyoid bone 12 cm in length, dividing the skin, platysma and fascia. Then the mylohyoid and geniohyoid muscles are detached and drawn upward from the hyoid bone. These muscles, together with the medial parts of the hyoglossal muscle, are bisected near the hyoid bone, enough of the ends of the muscles being left to help join them together after the operation is performed. Both submaxillary glands and the hypoglossal nerves are pressed backward. The mucous membrane of the pharynx is then incised, the length of the slash being relatively small."

An extended discussion concerning the bisection and reuniting the median raphe of the mylohyoid muscle is also included in this article. Continuing his description, Hofmann says that by pulling on the end of the tongue or with the use of a proper retractor applied to the severed root of the tongue because of the extensibility of the

pharyngeal walls, an excellent view is obtained, especially of the nasopharynx. If one also uses a wide hook to pull the soft palate forward at the same time one can look unhampered over the whole of the nasopharynx, including the upper part of the spine and the lateral walls. One also has a good survey of the pharyngeal and faucial tonsils and the openings of the eustachian tubes.

Spizharniy¹⁴ did the suprahyoid operation and reported on his results in 1906. From his experience he concluded that a combination of the suprahyoid approach with a buccal incision would give an excellent and unblocked approach to the operative field. He

admonished that the lateral segments of the hyoglossus muscle be left uncut and enough of the muscle stump be preserved so that it may be sutured afterwards. He stressed that it is necessary to locate and unite securely the cut ends of the median raphe. His careful observation of these important points in technique undoubtedly accounted for his excellent results.

Grünwald,⁴ who performed the first successful suprahyoid pharyngotomy reported six cases in 1906. His results were unsatisfactory and it was due to his adverse criticism that the operation subsequently fell into disuse and little or nothing was heard of it until recent years. He insisted that, although the suprahyoid pharyngotomy furnished a splendid view of the base of the tongue, the epiglottis and faucial pillars, the end results were highly unsatisfactory. His criticism was based on one outstanding defect resulting from the operation, namely, sagging of the larynx. This unfortunate result, although in marked contrast with the experiences of Spizharnyi,¹⁴ Jeremitsch, Hofmann, and others who reported satisfactory results in their operations, nevertheless influenced Grünwald to raise his editorial voice against the operation. The superiority of the pen over the scalpel in that instance resulted in placing this operation in the low historical niche which it has occupied for a half century.

In the years following, other methods of outstanding merit for entering the hypopharynx have been developed and successfully used in dealing with a great variety of lesions in the hypopharynx. Trotter's¹⁵ operation for entering the hypopharynx laterally, so outstandingly employed by Orton¹⁶ and others; the peroral approach by means of the suspension technique as practiced by LeJeune,¹⁷ Simpson¹⁸ and others and the subhyoid approach are well adapted for specifically indicated procedures. Our own limited experience and observations lead us to believe that, despite its poor historical past, the suprahyoid approach is the method of choice for certain lesions situated on the base of the tongue. A new method of transverse pharyngotomy combining the basic features of both the suprahyoid and subhyoid operations, has recently been proposed by A. Rethi of Budapest. No doubt, this operation will also have merit in some specifically indicated procedures.

With the use of antibiotics to prevent infection, combined with layer suturing of the involved muscles with particular attention to the reuniting of the ends of the median raphe of the mylohyoid, I

am firm in the belief that Grünwald's objection to the operation, namely, that it results in a sagging of the larynx, is no longer valid. My interest in this operation was inspired by a recent case, a report of which I submit:

REPORT OF A CASE

A white male, 86 years of age, was admitted to John Gaston Hospital August 30, 1950, having a very large tumor which completely filled his oropharynx. It had a smooth surface, was soft in consistency and was movable. It was attached to the base of the tongue by a pedicle 1.5 cm in diameter, lateral to the tip of the epiglottis on the left side. Dysphagia and dysphonia were present to a marked degree. His general condition was remarkably good for a man of his age. The Kahn reaction was negative.

A lateral roentgenogram showed a soft tissue mass encroaching on the air passage and displacing the hyoid bone downward. There was no evidence of invasion of soft tissue or bone. Biopsies were done on two separate occasions and they were reported, the first as lymphoid tissue and the second as chronic inflammation. On the ninth day after admission, the tumor was removed employing the suprahyoid pharyngotomy approach. Nitrous oxide anesthesia was used, being administered through the stoma of a preliminary tracheotomy. The hypopharynx was sealed off from the larynx with gauze packing. The initial incision was made one-fourth inch above the hyoid bone, longitudinally, from the median border of one sternomastoid muscle to that of the other, extending through the skin, platysma and deep fascia. The incision was then carried through the suprahyoid group of muscles including the anterior bellies of the digastrics, the mylohyoid, the geniohyoides and the central portion of the hyoglossus. The mucous membrane of the pharynx was then incised, the incision being easily controlled by placing the index finger of the supporting hand through the corner of the mouth and into the valleculae, as each was alternately entered. The tumor was easily delivered through the opening which was maintained by suitable retraction. It was then dissected from its base. The tonsil, on the corresponding side, being closely related to the tumor at its lower pole, was also removed by dissection. The defect in the base of the tongue was closed with interrupted chronic sutures and the bisected tissues by layer suturing. Particular attention was given to reuniting the severed ends of the raphe of the mylohyoid muscle since this, in

my judgment, is the most important support of the hyoid bone upon which the larynx and upper end of the esophagus depend for anchorage. The patient was fed through a Levine tube and given antibiotics. The wound healed by primary intention and the patient was discharged from the hospital on the 18th day at which time he swallowed without difficulty and had no disagreeable sequelae.

The pathological report on this tumor was as follows: The tumor was 6.5 cm in diameter. It was well circumscribed and presented ulcerations. It was friable and soft in consistency and yellowish-gray in color. Microscopically, a diagnosis of hyperplastic lymphoid tissue, with chronic inflammation, was made, although a low grade early lymphoma could not be excluded.

In our department at the John Gaston Hospital, four other patients have been operated upon in recent years for tumors on the base of the tongue, using the suprahyoid pharyngotomy approach. These patients were operated upon by Dr. Ralph Braund Chief Consultant in Tumor Surgery in the hospital. Two of these patients had adenocarcinoma of the posterior third of the tongue, one had an enormous lingual thyroid and the fourth had a hemangioma of the posterior third of the tongue. In each of these operations the accessibility to the site of the lesion, afforded by the suprahyoid approach, was most excellent.

These experiences and observations have encouraged us to believe that in this technique we have a valuable procedure in the surgical management of tumors of the tongue.

SUMMARY

A historical review of the background of suprahyoid pharyngotomy as a means of approaching tumors on the base of the tongue is given.

The practicability of the operation is established as a means of approaching tumors in the epipharynx.

An unsatisfactory sequela of the operation, namely, sagging of the larynx occurred in the experiences of one man is mentioned. It appears from the literature that in his operations no attempt was made to suture the pharyngeal mucosa, nor to unite the ends of the sectioned muscles by suturing, the wound being left to heal by gran-

ulation. The influential writings of this one man discouraged the use of the operation for many years.

With the development of antibiotics to control infection and the assurance of the restoration of function of cut muscles by modern surgical technique, it is my belief that the objections to the operation which have formerly prevailed, can now be satisfactorily overcome. Suprahyoid pharyngotomy is an easy and satisfactory approach to the area of the root of the tongue and has a distinct value in dealing with specific surgical problems in this area.

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DISCUSSION

HUGH G. BEATTY, M.D.: I have nothing particularly to add to this excellent paper but to say that I liked it very much. I used this method in selected cases for probably 20 years, for quite a number of tumors in the base of the tongue and epiglottis and my main point in rising is the occasional difficulty in swallowing of some of these patients, fluid getting in the larynx.

We had one interesting patient in whom two fibrous bands arose apparently from the arytenoids above the larynx forming what on first appearance fooled our medical students who thought these were some of the vocal cords. They helped very much in closing the larynx and keeping the foreign material out.

DR. ROTENBERG (Toronto): I would like to ask the Doctor a question. I would like to ask him what is the preferred type of anesthesia for this procedure? Does he believe a pre-operative tracheotomy should always be performed?

THOMAS C. GALLOWAY, M.D.: I remember very well the brilliant presentation of Sir Norman Patterson here at our previous meeting of lateral pharyngotomy as a surgical procedure. The question was asked then about his statistical successes in treating tumors of this area by such surgery. The question was not answered at that time, nor was I able to find any published statistics of cures. I believe this is a fine thing to know but I would again like to ask what superiority this has over the use of surgical diathermy combined with suspension in the majority of lesions?

Young men forget how effective surgical diathermy is. They forget as a matter of fact that the tumors which are operable in that area are superficial tumors whose ancestral cells were probably squamous epithelium, usually arising from the pillars of the tonsils, or the surface of the epiglottis, that these tumors are quite accessible. They can usually be reached and removed with surgical diathermy. We had considerable experience with it in the days when we had no good irradiation. I think that we are likely to be again misled by a brilliant surgical procedure into doing something that could be done more safely and simply by a well accepted procedure. My experience has been that the infiltrating tumor at the base of the tongue and in the posterior third has a very serious prognosis when treated surgically. When treated by irradiation the prognosis is considerably better.

HENRY B. ORTON, M.D.: There can be no question about the exposure one has in the procedure of a suprahyoid pharyngotomy. Anyone doing laryngectomies and removing the hyoid bone en masse with the larynx from above, is fully aware of the wonderful opportunity this gives of viewing the hypopharynx.

This is one of the reasons why the larynx should be removed from above downwards in the extensive malignancies in this region, the visualization is excellent and gives one ample opportunity to completely circumvent the lesion.

Years ago when I was in London and visited Mr. Trotter, I was impressed with his lateral pharyngotomy. I think, especially in malignancies of this area it is far better to look at the growth from behind than trying to look up at it by going through a suprahyoid-pharyngotomy. A suprahyoid pharyngotomy in my opinion should only be used in a case of benign tumor.

I certainly would not use the suprahyoid approach for a malignancy at the base of the tongue, root of the epiglottis. The lymphatic drainage from the

tongue must be taken into consideration in all malignancies of this area. In all malignancies we must be radical before we can become conservative.

CHAS. D. BLASSINGAME, M.D. (closing): In regard to Dr. Rotenberg's question concerning the anesthesia and the pre-operative tracheotomy: In this type of case, a preliminary tracheotomy is certainly justified and in this particularly case, which I reported, it was entirely necessary for the purpose of providing adequate breathing space and at the same time allowing the hypo-pharynx to be packed off, thus making it easy to control the hemorrhage.

General anesthesia of some type is necessary. Sodium Pentothal should be satisfactory. Any form of inhalation anesthesia can be easily administered through the tracheotomy stoma. In our case, as stated in the paper, Nitrous Oxide was used. In dealing with a large tumor on the base of the tongue, the question of approach, both from the standpoint of the anesthesia and the surgery, must be considered from the mechanical factors involved.

I agree with Dr. Orton that in malignancies of the base of the tongue the supra-hyoid approach would not be considered as adequate. Only in benign lesions, such as, lingual goiter, cysts, granulomas, lymphomas and very early and circumscribed new growths would it be considered suitable.

RHINOLITH: A REPORT OF TWO CASES

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AND

CARL D. MAKART, M.D.

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The discovery of a nasal calculus is an unusual and interesting experience. It is regarded as a rare condition but its occurrence may be more common than is generally supposed, for over 350 cases have been recorded to date and many have been encountered but not reported. It is also reasonable to assume that not infrequently small calculi form but become dislodged and ejected before reaching sufficient size to produce symptoms.

The pathogenesis of calculi in the nasal cavity is similar to that of calculus formation elsewhere in the body, such as the gall-bladder, the urinary-bladder, the kidneys, and the salivary glands. In these areas the concretions are largely endogenous in nature with a matrix composed of bacteria, leucocytes or tissue elements around which salts of magnesium, calcium, and sodium are deposited.

In the nasal cavity, the calculus is often formed around some small foreign body which has been introduced into the nose. These have been variously identified as shoe-buttons, bits of string, wads of paper, pencil erasers, beads, and other items. In one of our cases, the matrix was found to be a portion of a rubber band.

In most of the articles dealing with rhinolithiasis, the possibility of the endogenous nature of the growth is mentioned. As far as is known, this is purely theoretical and no evidence that these calculi may form around a matrix of dried pus or other endogenous material has been presented.

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One wonders why, if that were the case, so few rhinoliths are encountered considering the vast numbers of individuals with chronic sinusitis whose nasal and post-nasal spaces are seldom free of collections of purulent material.

Most of the foreign bodies found in a rhinolith are comparatively large. When none is seen it could be because the foreign body is too small or has lost its identity in the substance of the growth.

A possible nucleus for some of these concretions could be small particles of coal or stone dust or grains of sand. In silicosis, calcium collects around such particles trapped in the lungs and could well do the same in the nasal space.

Jarvis,¹ who has practised for many years in the granite centre of the country, told me that commonly patients show him concretions they have coughed up. He had not, however, observed any such collections in the nose.

Rhinoliths vary in size and shape, from small bodies to massive growths which entirely fill the nasal chambers and invade the adjacent structures. Many of the calculi reported are of good size and range between 50 gm and 80 gm in weight. The largest observed to date was reported in 1951 by Abu-Jaudeh.² It occurred in a 50 year old Arab whose symptoms dated back ten years. The calculus weighed 116 gm which was 6 gm heavier than the next largest. It produced distortion of the facial bones with a widening of the bridge of the nose, encroachment on the maxillary and ethmoid sinuses, and displacement of the right eye.

The signs and symptoms of a nasal calculus are those of a foreign body, to wit, a unilateral, malodorous nasal discharge associated with nasal obstruction. An occasional symptom is recurrent epistaxis which occurs as the growth erodes through the septum or the naso-antral wall. Most of the cases are of long duration with a gradual progression of annoying symptoms. In some there is no record of a previous nasal examination or medical care, while in others there is a history of exhaustive efforts on the part of the patient to obtain relief. In Gorman's² case the patient received treatment to his nose and ear two or three times a week for over a period of nine months without disturbance of the rhinolith which came to within one-half inch of the anterior naris and could be palpated on digital examination of the nasopharynx.

Rhinolithiasis must be differentiated from chronic maxillary sinusitis, bone necrosis, and malignancy, but the diagnosis is fairly well established when a probe or other instrument scrapes against a rough, hard surface with an unmistakable grating sound.

Treatment consists in removal of the rhinolith and when this is accomplished, the symptoms disappear. The large stones, such as that reported by Abu-Jaudeh, may require a lateral rhinotomy for removal but in most instances, the extraction is accomplished by intranasal procedures. Some patients will not tolerate the travail incident to the manipulation required for extraction of the growth under local anesthesia and for these individuals and children a general anesthetic is indicated.

It is unlikely that any but the small concretions may be extracted in one piece because of the brittle nature of the calculus and the small area through which it must be removed. The plan usually followed is to break off the most anterior portions with a Knight forceps and push the remainder into the nasopharynx for recovery through the mouth. The final fragment is usually deeply embedded and it may be necessary to grasp the stone firmly and rock it back and forth as a dentist loosens a tooth for extraction.

Removal of the rhinolith is not always a simple procedure and several sessions may be required before the mass is completely eliminated. This becomes apparent after the visible portions of the rhinolith have been removed and the operation becomes a groping procedure with a large segment of the calculus still unaccounted for. Bleeding is usually the factor which terminates each session. The usual bleeding attending intranasal procedures is intensified by trauma to the highly vascular granulation tissue in which the stone is embedded. In the event that the procedure is temporarily abandoned, a further attempt at removal may be undertaken a week later and in the meantime x-ray studies should be made in order to reveal the nature and exact location of any remaining fragments. Again, after removal of all visible and palpable portions of the calculus, x-ray studies are made as an assurance that no fragments have been overlooked.

Two cases of rhinolithiasis have been seen by us in the past twelve months. They have in common the fact that both appeared in women, were of several years' duration and were complicated by maxillary sinusitis on the involved side.



Fig. 1.—Rhinolith as it appears in specimen from cadaver.

REPORT OF CASES

CASE 1.—Mrs. M. M., a 34 year old housewife was first seen on April 19, 1951, suffering from a nasal obstruction on the right side with a malodorous mucopurulent discharge, and pain and tenderness over the right cheek. The patient stated that her symptoms began four years previously, at which time she was treated by her family physician who later referred her to an otolaryngologist. Following x-ray examination she received treatment with nasal packs every week for four months. When this proved ineffective she was put on intensive penicillin therapy for ten days by her family physician. Subsequently she saw in order, another otolaryngologist, a chiropractor, who gave her cervical spinal adjustments covering a period of a year, an osteopath who carried out digital manipulations in the nasopharynx and finally, in desperation, a psychiatrist who referred her to us.

On examination the nose revealed a deviation of the external pyramid to the left with a septal deformity to the right which almost completely occluded that cavity. On posterior rhinoscopy there appeared to be a globular clump of glistening mucoid exudate filling the right choana. Transillumination revealed a dark right antrum.

Irrigation of the sinus produced a large quantity of foul smelling exudate containing strands and clumps of gray, putty-like material. Antral lavage was repeated a week later with a similar result. Following irrigation a nasopharyngoscope was passed into the right naris below the deviation of the septum. Progress of the instrument was stopped almost immediately as the tip scraped against a metallic-like object on the floor of the nose. Following application of local anesthesia the mass was grasped with a Knight forceps and a portion broken off. A few more bits of the rhinolith were obtained at this time, but the procedure was discontinued because of excessive bleeding. On the following visit several more pieces of the calculus were removed and the remaining portion was found to be moveable and could be rocked in situ. This final piece was not removed at this time but a few days later it became dislodged and was coughed out by the patient.

Subsequent examination of the nasal cavity revealed the space to be lined with normal appearing mucosa, free of pus and foreign bodies. This was verified by x-ray examination which also revealed a clear antrum.

On gross examination the calculus was brownish gray in color, irregular in contour with blackened margins. Reconstructed it was 45 mm long (the largest intact piece being 35 mm) 12 mm in its widest portion and 18 mm in its greatest vertical dimension. It displaced 3.5 cc and weighed 4.35 gm. In the center of the largest fragment was found the probable nucleus of the rhinolith, a portion of a rubber band. Microscopic study of the putty-like exudate, recovered from the antrum on irrigation, was reported as amorphous, homogeneous material characteristic of caseous degeneration, thus indicating the presence of rhinitis caseosa concomitant with rhinolithiasis.

CASE 2.—Mrs. M. P., age 72, presented herself on March 26, 1952, for relief from recurrent pain in the left cheek associated with a foul smelling purulent nasal discharge and blocked nasal cavity. This condition had existed for 14 years during which time the patient had been examined repeatedly by different physicians, mostly general practitioners. She was told there was a growth present which appeared to be a polyp but she refused to submit to operative procedures for its removal.

Anterior rhinoscopy revealed a grayish, globular mass wedged in the left middle meatus between the lateral nasal wall and the sep-

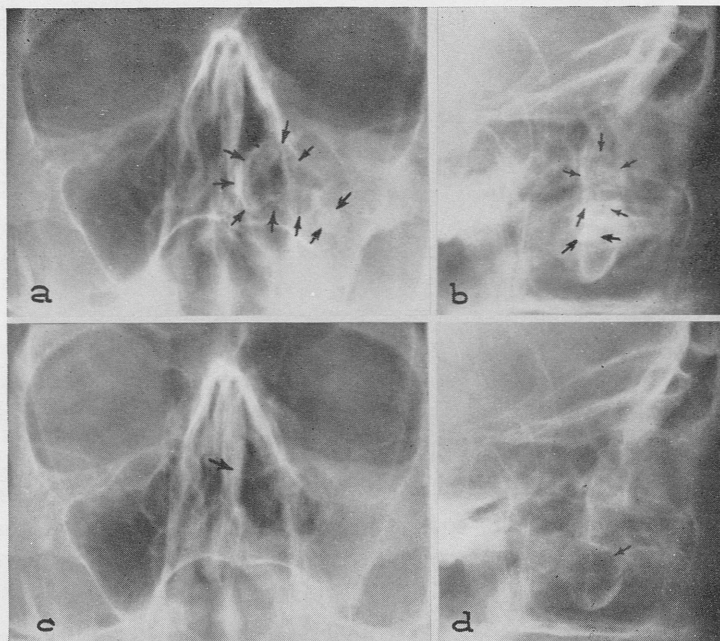


Fig. 2, Case 2.—A and B, Frontal and lateral views showing rhinolith in left nasal cavity. C & D, Same views, after removal of rhinolith.

tum. X-ray studies disclosed the presence of a slightly opaque mass in the left nasal cavity and a somewhat more dense object which appeared to be in the left maxillary sinus. When these were pointed out to the patient she consented to their removal.

Under local anesthesia the rhinolith was grasped with a Knight forceps. The mass disintegrated readily and seemed shell-like in structure. Considerable bleeding resulted and as the patient blew her nose minute particles of calcareous material were collected. X-ray films following this procedure showed absence of the cyst-like mass but continued presence of the small opaque object. On her next visit, a few days later, further manipulation in the nasal cavity dislodged this body and forced it into the nasopharynx to be hawked out by the patient. Irrigation of the antrum produced a thin, yellowish exudate extremely malodorous. Final x-ray films showed

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complete absence of the opacities previously visualized, although the left antrum appeared to be relatively hazy.

On a subsequent visit the patient related that the foul odor had disappeared and for the first time in many years she was able to breathe without a sense of obstruction. Antral irrigation at this time produced a clear return without odor.

Our reason for presenting this subject is to call attention to the fact that cases of rhinolithiasis exist and remain undiscovered for a number of years even though they present typical symptoms and are seen by many physicians.

Two cases were seen by us in the last twelve months. The concretions recovered in each case were not large as rhinoliths go, but nevertheless important to the patient's health. Both appeared in women, were of several year's duration and were complicated by maxillary sinusitis on the involved side. In one the sinusitis was of the caseous type and the rhinolith was formed around a foreign body which had become lodged in the nose, while in the other, there was a simple purulent sinusitis and no apparent foreign body formed the nucleus of the calculus. In both cases, as is common in most, the symptoms disappeared immediately upon removal of the foreign body.

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DISCUSSION

JOHN J. SHEA, M.D.: One of the reasons for foreign bodies in the noses of children is their playing habits.

It is our privilege to take care of a tribe of Irish gypsies who come to Memphis twice a year, once for matrimonial services and the other time for mass funerals of their dead of that year. During examinations of these children I have found a great many of them with beads in their noses, as it is with beads that they play. Going around the country they do not have the toys that other children enjoy and have the great habit of playing with beads. We always suspect a foreign body whenever there is a unilateral discharge of the nose.

Last year we had a girl of 18, who had a tremendous rhinolith, and when it was removed a small button was found in its center. The rhinolith had eroded into her maxillary sinus through the middle meatus. The child's mother reported that she had a severe nasal hemorrhage at the age of three, the cause of which was undetermined, but apparently it was from the insertion of the button into the nose.

I enjoyed the case reports of Dr. Van Alyea, as they exemplified the necessity of remembering, that a unilateral nasal discharge is frequently the result of a foreign body.

HARRIS P. MOSHER, M.D.: I will not discuss the first part of this paper, the tragic experiences that this patient had with the various types of practitioners, some legitimate in my opinion, and some not.

I have just a word or two to say about the paper as a whole. The essayist just used the word that I should use in connection with it. He is a good sport and I hope he will take this kindly. I know he will. I should summarize this paper by saying "Elementary, my dear Watson, elementary."

DR. O. E. VAN ALYEA (closing): I think I pointed out the fact that it might be a rather elemental subject to many of you, but the fact that during the past twelve months we have been confronted by two patients suffering for many years' duration, led me to believe that it was timely to bring the subject of rhinolith to the attention of the otolaryngologists again. Even the elemental subjects sometimes are hurried over. We get rather lax in our busy hours of clinics and office practice. We overlook post-nasal examination. Quite often we pass up x-ray studies for economic reasons and for speed. A complete examination of the nose should have been carried out by the otolaryngologists who saw our patients previously. It is for that reason that I think there is a justification for this elemental subject.

THE USE OF CORTISONE AND CORTICOTROPIN IN THE FIELD OF OTORHINOLOGY AND LARYNGOLOGY

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The first rush of enthusiasm for the use of cortisone and corticotropin (ACTH) resulted in their indiscriminate and haphazard use in medicine. This enthusiasm has now abated to a large extent. We have been allowed time to assess the results of therapy with these substances and found them generally disappointing in the field of otorhinology and laryngology.

Conditions which seem to derive temporary benefit from the use of these substances appear to be those in which allergy appears to be an etiologic factor, especially if allergy is defined as being a localized type of autonomic dysfunction in which an antigen-antibody mechanism may or may not be present.¹

UNFAVORABLE EFFECTS OF TREATMENT WITH CORTISONE AND CORTICOTROPIN

For the purposes of this presentation it seems to me that a sounder basis for the use of cortisone and corticotropin will be established if the unfavorable effects of these substances are considered first and then the favorable effects on otorhinologic conditions.

Euphoria.—Sprague and co-workers² mentioned the frequent occurrence of euphoria especially during the early stage of treatment with cortisone. This may be a distressing complication, sometimes amounting to an acute mania. It is also possible that in some instances the good results attributed to treatment of conditions of the ear, nose or throat with these substances may depend to a considerable degree on the production of euphoria.

From the Section of Otolaryngology and Rhinology, Mayo Clinic.

Effect on Carbohydrate Metabolism.—Diabetes may be greatly aggravated by corticotropin or cortisone. Sprague and co-workers² stated that it is apparent that neither cortisone nor corticotropin in the doses which are usually employed produces any conspicuous alterations in the tolerance of carbohydrate. It is equally apparent, they said, that more striking changes in carbohydrate tolerance might be expected if the hormone was administered to persons having latent or frank diabetes.

Effects on Appearance.—The same authors² also stated that rounding of the facial contours or a Cushing-like appearance was one of the earliest signs of hormonal excess. Pigmentation of the skin also occasionally occurs after prolonged treatment with corticotropin.

Disturbances in Water and Electrolyte Metabolism.—Thorn and co-workers³ found that retention of sodium chloride and water occurs with great frequency during treatment with corticotropin, but it rarely complicates treatment with cortisone.

Clinically, a progressive gain in weight is observed which pre-sages frank edema. It is wise, therefore, to restrict the intake of sodium chloride and fluids of patients receiving corticotropin.

Thorn and his associates also found that a hypokalemic hypochloremic alkalosis might occur after treatment with either corticotropin or cortisone. This condition might be independent of retention of sodium chloride and water. It is associated with easy fatigability, muscular weakness and characteristic electrocardiographic changes. Oral administration of 1 or 2 gm of potassium chloride in enteric-coated tablets three times daily often will prevent and certainly will ameliorate these changes.

In passing, it is of interest that Thorn and his associates also found that abnormal elevations in blood pressure are seen only infrequently in the course of treatment with corticotropin and practically never in the course of treatment with cortisone.

The Danger of Infection During Prolonged Treatment With Cortisone or Corticotropin.—Both corticotropin and cortisone will suppress the resistance mechanism by which the body meets invasion from without by pathogenic bacteria and also the usual signs and symptoms of the infection.⁴ As a consequence, patients who are

being treated with these agents may have a serious infection, especially septicemia, without showing the signs and symptoms by which such infections are ordinarily recognized. The physician must be on the alert for the development of such a complication.

The Danger of Surgical Procedures on Patients Who Have Been Receiving Corticotropin and Cortisone.—Sprague and his associates² presented evidence which suggested that cortisone acetate administered in ordinary therapeutic dosage would cause a depression of adrenal cortical activity. It is recognized, therefore, that the response of patients who have been recently receiving cortisone to the stress of surgical procedures resembles that of patients with Addison's disease. Many patients who have been receiving cortisone recently neglect to mention this fact to the surgeon. Because the period of recovery of the adrenal cortex is variable and may take from three to six weeks or more, it has become important that patients be queried specifically on this point before surgical procedures are undertaken. Patients who recently have received cortisone or corticotropin must be treated preliminary to and after operation in much the same way that a patient who has Addison's disease is treated.

OTORHINOLOGIC CONDITIONS WHICH APPEAR TO BE FAVORABLY
AFFECTED BY CORTICOTROPIN OR CORTISONE

Otorhinologic conditions which it has been suggested might be favorably influenced by corticotropin or cortisone include vasomotor rhinitis with or without polyposis, angioneurotic edema and idiopathic (lethal) granuloma of the midline facial tissues.

Vasomotor Rhinitis and Its Sequelae.—Carrier, Koelsche and associates⁵ observed the nasal mucosa of patients with vasomotor rhinitis associated with hay fever and asthma during treatment with cortisone. The secretions tended to disappear first, then the color of the mucosa changed toward normal, and after a delay, the edema of the nasal mucosa receded. The following year Koelsche and associates⁶ observed a less marked effect from further treatment of many of the same patients with cortisone.

Rose and associates⁷ observed 8 patients with vasomotor rhinitis and polyps treated with corticotropin or cortisone. Six of these were improved temporarily, and in all 6 shrinkage of the mucous membranes and usually of the polyps occurred. The polyps, however,

never entirely disappeared. A small residue remained even in the most favorable cases and within two months of the cessation of treatment the polyps had returned. Rose and his associates stated, however, that the feeling of well-being "associated with the sense of smell and easy nasal breathing" created a marked change in the outlook of these patients. It seems probable that the elevation of mood observed in these patients resulted at least as much from the euphoria produced by these medicaments as it did from improvement of nasal breathing.

Rose and associates⁷ found that for nasal polyposis the average effective dose of corticotropin was 100 mg per day in divided doses for the first three days after which the dose could be reduced to 50 or 60 mg per day. For some patients 100 mg per day were required continuously to produce a clinical effect. The dose of cortisone equivalent to 100 mg of corticotropin was found to be 200 mg per day.

Rose and his associates⁷ observed that a second course of treatment with corticotropin or cortisone might not be as effective as the first, even though doses were the same and both courses were administered in the hospital. The reasons for this were obscure, but these observers suggested that a change in the time of the year or climate might have been a factor. Rose and his associates also noted that the condition of a number of patients appeared to be resistant to the use of both corticotropin and cortisone. These patients may have responded well to a first course of corticotropin, somewhat less effectively to a second course and not at all to a third or may have failed to respond to any but the first course. The same held true for cortisone.

Lake and Williams⁸ treated 5 patients with severe polyposis with a total of 200 mg of cortisone a day given by mouth in divided doses, every six hours night and day. They found that 2 of the 5 patients failed to show any response as far as recession of the polyps was concerned even when at the end of three weeks the total dose was increased to 400 mg per day and treatment was continued for another week.. Three of the 5 patients had rather marked recession of polyps when they were receiving 200 mg of cortisone per day, in divided doses. The polyps of all 3, however, were as large as before treatment a month after treatment was stopped. In 2 of these patients treatment was repeated without any diminution in response. In 1 of these patients an attempt was made to delay the return of polypoid tissue by a maintenance dose of 50 mg of cortisone per day,

25 mg at night and in the morning. This treatment delayed the return of polyps slightly but had relatively little effect.

Dill and Bolstad⁹ reported the treatment of 25 patients with allergic rhinitis with a spray of cortisone acetate (cortone acetate) 1 to 4 in saline solution. Each side of the nose was sprayed four times a day with this solution. Of the 25 patients treated, 12, or 48 per cent, obtained marked relief of symptoms. Six of the patients had some improvement and 7 none. In a second paper Dill and Bolstad¹⁰ stated that of the 12 patients obtaining good results 5 continued under treatment and have been under observation for more than a year. In addition 10 new patients were treated. Of the entire 35 patients 14 had polyps on initial examination. The effect of cortisone spray was considered negligible in this group except that the polyps of 1 patient disappeared. The assessment of improvement in this group seemed to be based solely on subjective improvement such as return of the sense of smell and relief of nasal stuffiness. When relief was obtained, it lasted only as long as the spray was continued.

Lake and Williams⁸ attempted to reduce nasal polyposis by the injection of 0.2 cc of cortisone acetate (cortone acetate) solution into all visible polyps daily. This treatment was continued for two weeks without an observable effect on the appearance of the nasal polyps.

Comment.—Perennial vasomotor rhinitis is a persistent allergic reaction affecting to a large extent the collagen of the tunica propria of the nasal mucosa. Nasal polyposis is essentially a collagen disease. Nasal polyps may produce more or less obstruction to nasal breathing. As a result of interference to drainage and the effect of allergic edema in lowering local resistance to bacterial invasion, infection of the sinuses may take place, producing a condition known as "hyperplastic sinusitis." Adenomyxomatous polyps or cysts in their early stages may be reversible phenomena. After long standing, however, they become fibrosed and will not recede even though the stimulus to allergic response is completely removed.

Heretofore nasal polyps and hyperplastic sinusitis have responded best when a combination of surgical, medical and specific anti-allergic treatment was given. Although these measures have not been entirely successful, their effectiveness in producing prolonged symptomatic relief is far greater than that of cortisone or corticotropin.

Idiopathic (Lethal) Granuloma of the Midline Facial Tissue.—Idiopathic granulomas of the midline facial tissues are being reported with increasing frequency. It seems probable that previously many of these cases were not reported, either because of the difficulty in making a definite diagnosis or because in such a long, lingering illness the patient frequently goes from physician to physician in hope of relief and consequently one physician is not often able to observe the total course of the illness. The use of cortisone and corticotropin for this group of patients seems to produce a healing tendency in these lesions.

Clinically the picture in idiopathic granuloma is one of progressive destruction of the nose, face and pharynx. The duration of the illness may be from a few months to five years or more. The most striking feature of the disease is the apparent complete absence of resistance on the part of the patient to the progress of the disease.

Hoover¹¹ described the typical pathologic features of a lethal granuloma of the face and nose. He stated that the center of the lesion was an amorphous necrotic mass. Peripheral to this there was an infiltration by inflammatory cells chiefly lymphocytes and macrophages with occasional plasma cells. A few polymorphonuclear leukocytes were also present. The large vessels in the tissue showed a definite perivascular cuff of lymphocytes and macrophages with degeneration of arterial walls in some regions. While there were focal accumulations of infiltrating cells in the tissues, the lesion was not characteristic of tuberculosis, syphilis or leprosy. To Hoover the histologic picture of the lesion was suggestive of that of the Arthus phenomenon.

Weinberg¹² in discussing idiopathic granulomas added what appears to be an important point. He found that these lesions were not strictly limited to the midline tissues of the face but that here and there throughout the body a pathologic picture of periarteritis nodosa developed. Weinberg stated that if all organisms which might produce a similar histologic picture were eliminated in so far as possible, there is left a picture which clinically consists of ulcerations of the upper respiratory structures associated with evidence of focal alterations in the lungs which pathologically consist of multiple granulomas of unknown origin. These may involve one or more organs with terminal development of periarteritis nodosa. He concluded that the constancy of the history of antecedent chronic infection in

these cases lends support to the belief that hypersensitivity may be a factor in the development of the lesion. From the viewpoint that allergy is fundamentally a localized hyperimmune reaction of the peripheral vascular bed, it seems reasonable to suppose that cortisone and corticotropin would tend to ameliorate this disorder. Williams and Hochfilzer¹³ reported a preliminary trial of cortisone on a patient with a lesion which seemed to be of this nature. The effect on the lesion seemed to be encouraging although clinical trial was too short to be conclusive.

Moore and associates¹⁴ reported the use of cortisone and corticotropin for a patient who had a relatively early lesion of this type with necrotic lesions in the nose, pharynx and larynx. Later in the course symptoms developed which suggested the presence of periarthritis nodosa. Under treatment with corticotropin and cortisone the lesions in the nose, pharynx and larynx healed completely and the general symptoms also subsided. At present their patient appears to be in a complete remission. This is the first report in the literature in which a patient with idiopathic granuloma has made an apparent recovery.

The dosage schedule used for this patient appears to have been inadequate to produce the most rapid and effective result. If dosage is based on the findings of Shick and co-workers¹⁵ in the treatment of periarthritis nodosa, an initial dose of 300 mg of cortisone followed by 100 mg per twenty-four hours in divided doses given every six hours and continued for a minimal period of six weeks might be expected to produce a more rapid recession of the symptoms and signs.

COMMENT

The use of cortisone and corticotropin (ACTH) in diseases of the ears, nose and throat seems to be as yet in the stage of clinical evaluation. With the exception of idiopathic (lethal) granuloma, the conditions can be treated with reasonable success in most instances by other medical or surgical measures. For idiopathic granuloma, however, these hormones offer the only hope of saving the patient's life. It might be well, therefore, if the otorhinologist would delay a little in the indiscriminate use of these substances.

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DISCUSSION

D. S. BOLSTAD, M.D.: I have been the junior author of the paper Dr. Williams mentioned. We did have this experience with approximately 35 patients treated with cortisone as a local spray in the nose.

First of all, I wish to say I agree with Dr. Williams in the use of this parenterally. I think the effects are by far too numerous, too severe to warrant the use of cortisone or ACTH for the use of many of these allergic things. I also agree with Dr. Williams when he brings up the point, as we mentioned in our paper that relief of the symptoms is present only as long as the patient continues to use the cortisone spray. I refer to the nasal symptoms. However, this same thing is true also of allergic treatment. Patients who have undergone extensive allergic studies and who have been begun on the desensitization program will have relief of their allergic symptoms usually only as long as they continue with their allergic desensitization. They have to continue with their injections at intervals of three to four weeks for an indefinite period of time or their symptoms will come back. This same thing is true with the surgical removal of polyps. How many of you have removed polyps in the past only to have them come back repeatedly. Some men will argue you have not cleaned out every thing adequately, you have not done ethmoidectomy, or not followed with x-ray therapy. I have seen patients who had very adequate ethmoidectomies and radium treatment used in their nose and whose polyps in spite of this continued to recur.

The patient he mentioned during his paper, who in our series had regression of his nasal polyps, had complete regression of the polyps. There were none visible in his nose at all on two occasions. Now he has discontinued completely the use of cortisone and in each instance the polyps have come back. In each instance when the cortisone has been resumed the polyps have again disappeared.

The one argument for the use of topical cortisone, as compared to parenteral use of cortisone is the fact that it has absolutely no side effects. It can be used indefinitely over prolonged periods of time without any side effects of any kind. There is no change of the blood picture, no influence upon any of the body's physiological functions. There is no atrophic change present in the nasal cavity and our experience is also very similar to that of the Department of Ophthalmology who have used it in the conjunctival sac for prolonged periods of time with absolutely no side effects at all.

THOMAS C. GALLOWAY, M.D.: In the first place I am curious to know why Dr. Williams calls this "midline lethal granuloma" as the cases I have seen have not been midline.

We have had a very interesting experience with this condition. A man had soreness beginning around the second molar tooth. He got intense pain, a maxillary sinusitis, the sclera and the conjunctiva of the eye on the same side were involved, involving the inner canthus and there was ulceration of the palate. Because of pain and a foul discharge cultures were taken and a micro-aerophilic osteomyelitic involvement of his maxilla was found. I removed the floor of the maxilla, the medial wall and destroyed the granulation tissue with electrocoagulation. The diagnosis was a little in doubt. This man had had previously an erythema multiforme type of lesion. He was put on ACTH and cortisone and this lesion completely healed except for one area over the soft palate of about 5 mm. The internist on the case then stopped the cortisone. He had gotten down to 50 mg dosage, once a day. The patient had a recurrence and we found it

extremely difficult to control the lesion until penicillin was given at the same time.

SAMUEL SALINGER, M.D.: While this may not be strictly germane to the subject, nevertheless I think it is worth mentioning. We have had two experiences with the use of cortisone in the treatment of penicillin reactions. Both of these cases following surgery were very severe but cleared up promptly within a period of two or three days after the parenteral use of cortisone. I think that when used for that short period of time one is not likely to encounter the ill effects that were mentioned.

DR. FRANCIS L. WEILLE: I would like to ask Dr. Williams a question, that is, does he have any opinion or does the Mayo Clinic have any opinion as to the basic action of cortisone and ACTH? The reason I ask is this: Hastings, the chief biochemist of Harvard Medical School, has done some experiments with alanine marked radioactively and has found that the protein synthesis is cut down 50-60% in tissue cultures from animals treated with ACTH or cortisone. He believes that is the common denominator in these preparations. I would like to ask Dr. Williams if he can enlighten me further.

JULIUS W. MCCALL, M.D.: At the suggestion of Dr. Clerf recently when we had two amyloid deposits of the larynx we used cortisone as a local injection, 25 mg. This we repeated eight times on each patient. There has been very little visible improvement in the larynges of these two women, but one girl has had a marked improvement in the voice, though from a clinical picture there has been no improvement. It was just used as an experimental study of amyloid deposit in the larynx.

DR. MARKS: I have had experience with only one case but I feel this worth reporting here. This was a case of hyperplasia of the epipharynx and secretory otitis. This child was six years old, had had an adequate T. and A. and control of her allergy with antihistamines. She had also had as I remember a course of x-ray therapy for the post-nasal pharynx. In spite of this she persisted to run about a 40 to 50 db loss in both ears. Because of the marked hyperplasia we decided that perhaps it might be worth while to do a secondary T. and A., removing as much excess tissue as possible and to use cortisone to perhaps try to control the reformation of this tissue. The endocrinologist was consulted, Dr. Lawrence Consell. He agreed that perhaps a postoperative dose of approximately 50 mg a day would be adequate over two months. We accomplished the T. and A., and bilateral myringotomy; using a small suction, a No. 5, we were able to get out quite a bit of stringy mucous material from the middle ear in both ears. In ten days we did an audiogram and the audiogram had returned to about almost normal, to around 10 db loss. This case is about seven weeks postoperative and she has maintained this hearing gain to this day. We don't anticipate any miracles. However, we do feel that it was worth while considering.

DR. HARRY P. SCHENCK: I would like to ask Dr. Williams whether he has used Compound F. Compound F is a more recent compound and very little of it has been available. I had a small amount last summer for the treatment of seasonal pollenosis and the manufacturer, as well as those doing research in arthritis thought that this compound would be a very valuable drug when used locally. I only had sufficient material to treat eight patients and four of them received the material in the form of a nasal spray and I had the temerity to inject the material into the turbinates of the other four. I am sorry to say they all became very much worse, very much worse than they have ever been before. It did

absolutely no good. In fact, they would not consent to continue with the treatment or repeat it after their first flare-up occurred. I was then told that the method of administration was incorrect and that it should be used in a water-soluble base, which would liberate this material very slowly and give a more or less progressive action. I have now had the opportunity to spray it on some early pollen cases and again this material has proven to be extremely irritating. None of them have received the slightest improvement.

I have also been interested in these peculiar granulomas. Ours have always been in the midline of the face or at least been intranasally and more or less in the midline. I think everyone realizes that this is actually a systemic disease. At autopsy one finds vascular lesions in all the vital organs, not as extreme as those we deal with locally, but in the first case we had no ACTH or cortisone, because it was being used for other research purposes. In the second case we did. That patient died in spite of ACTH and cortisone. In this patient, no virus was isolated, no molds were isolated, no bacteria were isolated and we were curious to find out whether we could find etiologic agents because we were thinking in terms of allergy. We could get a lot of necrotic tissue from the upper respiratory tract. We obtained some of the necrotic tissue, ground it up and put it through a Berkefeld filter and that filtrate was used for skin testing. The patient reacted to that material. We used the same material on six other individuals presumably normal and they obtained practically no reaction at all. I thought this was very significant, but after I talked it over with my colleagues they found that the test was so complicated, and involved so many uncontrolled factors it was not significant. I just mention this in case anyone else encounters one of these rare cases so they may carry the intradermal testing a little farther and perhaps get some valuable information.

HENRY L. WILLIAMS, M.D. (closing): I want to thank the discussers very much indeed for their kindness in regard to this paper.

In regard to Dr. Bolstad's remarks, it is quite true that cortisone spray used as he suggested does not have any side effect, but when one remembers that he found that only one out of 14 patients with polyposis showed any reduction of the polyps one is not very much encouraged as far as the use of this substance for nasal polyposis is concerned. In the other patients they secured about the same relief as is secured from the antihistamines in patients who react favorably to them. I see no objections to using it except for the inordinate expense.

In Dr. Galloway's discussion, I think he brought up a question of differential diagnosis. He spoke of the presence of micro-aerophilic or anaerobic organisms; in these lesions that we studied we find one of the most difficult differential diagnoses to make is that between the necrotic lesions produced by the anaerobes and also the so-called Krompecher type of epithelioma, both of which attack the midline facial tissues. You may remember that Dr. New advised the use of peroxide for washing these lesions and reported good results. That is another incidence of incorrect differential diagnosis. Necrotic lesions produced by anaerobes which will respond to peroxide are distinctly different from the midline lethal granulomas.

Naturally we have cultured these lesions, both for anaerobic and aerobic organisms and used all methods of study for fungi and viruses with negative results.

As far as the Krompecher epithelioma is concerned, that differential diagnosis is extremely difficult. This epithelioma starts in the midline facial tissues and advances through the nose. It is very difficult to get a positive biopsy in these

lesions because of the tremendous amount of granulation tissue which surrounds the malignant cells. Sometimes apparently the diagnosis is helped by the use of ACTH or cortisone owing to the fact that these substances tend to cause the granulations to disappear and make it possible to get a biopsy which will include malignant cells.

I was very much interested in Dr. Salinger's remarks about the penicillin reaction.

In regard to Dr. Weille's question about the action of ACTH and cortisone, I am sorry that I am unable to give the official opinion of the Mayo Clinic. In talking it over with the men that are interested in ACTH and cortisone, it appears to be their feeling although not officially expressed that cortisone and ACTH have a local action on the cells, that they act somewhat as a chemical mediator to reaction. I am not familiar with Dr. McCall's case of amyloid disease and have not tried any treatment of that condition. I have used cortisone and ACTH in one case of secretory otitis media. This gentleman in addition to the secretory otitis had eczematoid dermatitis and vasomotor rhinitis. The eczematoid dermatitis and secretory rhinitis cleared up but the other did not. It was my opinion since this patient had rather marked cloudiness of his mastoid, that this represented an infectious rather than an allergic type of secretory otitis media.

I have used Compound F in two types of patients, those that are receiving the medicament by mouth and that which we use to inject the polyps. The Compound F did not seem to have any different effect than the Compound E which we had been previously using. We did not make any of the patients worse, but, as I said, in injecting the polyps it had no effect whatsoever, and using it by mouth it produced just the same effect indistinguishable from that of cortisone itself, or Compound E.

BRONCHOGRAPHY IN CHILDREN

D. E. S. WISHART, M.D.

TORONTO, CANADA

Bronchography in children can be performed easily and safely. At the Hospital for Sick Children Toronto it has been carried out for many years; latterly with complete satisfaction to the paediatric and surgical services of this hospital.

However in the *Annals of Otology, Rhinology and Laryngology* September 1951,¹ an article stated that the technique of bronchography in young children is particularly difficult and the results poor. This may be the situation in certain hospitals but it does not need to be. This work is being done regularly by six members of the otolaryngological staff and is being taught satisfactorily to each resident in otolaryngology. There are thousands of bronchograms available for inspection.

The first report on our work² was made in the *Journal of the American Medical Association* of December 12th, 1942. The technique was modified and a second report³ published in the *Annals of O. R. & L.* in June 1947. Both these papers discussed technique and results obtained in the old hospital in a room and under conditions which were far from ideal. The objections must have seemed so formidable to a casual observer that he may have found it difficult to appreciate that the work had been carried on as stated over many years with increasing satisfaction without any trouble of a serious nature.

In February 1951 we moved to our present new building where relatively ideal conditions for the carrying on of our work have been set up.

The pediatric staff of this hospital and the surgical staff are enthusiastic about every part of our procedure and it is because we believe that everything can be copied safely and readily in hospitals for children throughout the country that this subject is again being reported before this Society.

As stated in the other papers this work in our hospital commenced in 1927 and ever since has steadily increased. There is a gradual increase in the number of lobectomies. At present we perform bronchography regularly on two mornings a week, three or four bronchographies being performed at a session. The work is the combined and closely co-ordinated efforts of the staffs of otolaryngology, anaesthesia and radiology. The details of the technique employed have been slightly altered from time to time but the essential bronchoscopic method has been used since 1930. Because a new multiplane fluoroscope was provided in February 1951 certain modifications in the former technique became necessary and these will be described.

Bronchography by endo-bronchial injections of iodized oil through the bronchoscope under general anaesthesia and under fluoroscopic observation has been performed approximately 2,500 times. As from 3 to 5 bronchograms are made at each session we have approximately 10,000 films for inspection. The resultant bronchograms have led to about 111 lobectomies and pneumonectomies for bronchiectasis in the period since 1945 alone.

There have been no serious post-anaesthetic complications since the early years of our work. These were reported in the first paper from this hospital.

The object of the bronchography has been to provide accurate anatomical diagnosis of all portions of the bronchial tree. This is done primarily at the request of and for the information of the paediatric service. Where the abnormalities discovered are deemed suitable for surgical treatment the medical service enlists the interest of the surgical service. The bronchograms are studied in concert, and the requisite surgery performed.

In adults bronchography is performed adequately by the catheter technique. Occasionally this catheter technique can be used in suitably conditioned co-operative children who are relatively well. It is impractical to use it with miserable, coughing, sick, apprehensive, unconditioned children. It cannot be used at all in tiny children. On the other hand the technique at present in use in this hospital has made bronchography possible even in an infant one month old.

The conquest of bronchiectasis by the surgeon has made bronchography of great importance. Bronchography in children in this

hospital is of major importance to our surgical service. Failure to map a small portion of a lung has occasionally occurred as will be commented upon later in the paper but these occasions have been exceedingly rare.

Modern chest surgery can cure bronchiectasis. The thoracic surgeon excises every diseased portion of the bronchiectatic lung. Before he commences the surgical treatment he must know from the bronchogram the condition of every part of the lung. In the early days adequate mapping of the lingula was rare, of the right middle lobe uncertain and of the two upper lobes far from satisfactory. Our present technique enables us always to get the formerly difficult portions of the lung completely and adequately pictured. Our surgical service knows that at operation the lingula may seem normal by inspection and by palpation yet they have such confidence in our lung mapping that they unhesitatingly remove the lingula where our procedure has shown it to be diseased. The bronchograms which 5 years ago we might have considered good are no longer considered adequate. The chest surgeon knows that he cannot see all branches of the bronchial tree completely in any one picture. He requires exposures in certain positions for the outlining of special bronchi and exposures in other positions for the visualization of other branches.

Therefore, today, before he will operate upon a bronchiectatic patient, a chest surgeon requires for study bronchograms of all lobes of each lung in several positions. If he is not satisfied with the delineation of some part of the bronchial tree he will demand more bronchograms of that particular portion, for he must plan his surgical attack and he cannot do that until the bronchography is complete to the last segmental bronchus.

Technique. The preparation of the patient for bronchography varies with the age and condition of the patient. In some patients postural drainage is carried out for a period.

Bronchography in our hospital is performed in the bronchoscopic theatre of the department of otolaryngology. It is performed through the bronchoscope under general anaesthesia. The bronchoscopist is in charge of the whole procedure. But the operation is performed by a three man team, the anaesthetist, the bronchoscopist, and the radiologist. Each member in turn plays a major role. When the other members are playing their main parts the bronchoscopist

either co-operates with them by not interfering or by obeying their orders.

As the bronchoscopist is in charge of the procedure he observes the condition of his patient before the anaesthetic is started.

The bronchography is not preceded by sedation. Any sedative will interfere with the cough reflex if the dose is sufficient to diminish fear. The success of the procedure depends, in part, on the rapid return of the cough reflex once the anaesthetic has been stopped. No local anaesthetic is used.

The excitement of coming for operation excites coughing. The patient is turned on his side and the coughing is encouraged in order to free the bronchial tree of as much of its purulent material as possible. The anaesthetist attempts by gentleness and reassurance to gain the child's confidence. When he has achieved this he commences the anaesthetic.

The bronchoscopic operating room is 18 feet x 16 feet and has a high ceiling. There are many windows and a large door. Fresh air is delivered at the ceiling and the stale air is removed by suction through a ventilator, close to the floor and close to the head of the operating table.

The incoming air passes through filters. It enters at a rate which allows of complete change of air 7 times an hour. The rate is regularly tested once a month just before the regular monthly cleaning of the filters.

Relative humidity of the incoming air is 70%. The door of the operating room opens into a small corridor which is closed by doors from the main corridor. Keeping the windows and doors of the operating room closed for one hour before operation is necessary for maintenance of the humidity. The humidity is not constant but varies with the temperature of the room.

The result of this ventilation is that no concentration of explosive anaesthetic occurs and even if electrical sparks were produced they would do no harm.

However we take particular precautions to see that electrical sparks shall not occur. The table used is shock proof and completely grounded. The use of wool in the draping of the patient is prohibited. The view boxes and all the electrical outlets are flash proof.

The anaesthetic used is ethyl-chloride-ether sequence. The child is placed in the lateral position with the face turned slightly down towards the table to permit saliva and discharges to flow out of the cheek rather than to collect in the pharynx. This position makes maintenance of the airway easier as anaesthesia progresses and relaxation occurs. Suction is available at all times and is used as necessary. The induction stage is not hurried: it being our belief that some coughing at this stage is helpful in clearing secretions from the airway.

The open mask method is used until sufficient depth is obtained to allow the introduction of the bronchoscope without damage to the patient or embarrassment to the bronchoscopist. The mouth and pharynx are then cleared with a suction tube and the patient turned on his back for the exposure of the larynx.

A bronchoscope suitable to the size of the patient is now introduced and from this point anaesthesia is maintained through the bronchoscope by means of a rubber tube delivering oxygen and ether vapor produced by the AINMED gas anaesthetic apparatus.

At this point the depth of the anaesthesia is diminished to allow slight coughing to occur. The purpose of this is to promote the expulsion of secretions from the inaccessible distal portions of the bronchial tree to the main bronchi from which the secretions can be sucked by the bronchoscopist. The attempt is made to clear the bronchial tree as far as possible. Iodized oil will not enter a plugged bronchus. Much of the success of the whole procedure depends upon this evacuation of secretions from the main bronchi by suction. (The material aspirated is collected and sent to the laboratory for microscopic and bacteriological investigation.) When there are no more bronchial secretions to be removed by suction the bronchoscopist notifies the anaesthetist who then proceeds to increase the anaesthesia. Deep anaesthesia has to be reached and maintained for about five minutes. The anaesthetist knows from experience, when his patient is ready for the next stage.

The next stage is the introduction of cool iodized oil into the bronchial tree to be outlined. This is done through the bronchoscope via the metal cannula.

At this point the radiologist is summoned. (The radiological department immediately adjoins the bronchoscopy room.)

For bronchography of the right lung, assistants turn the patient's body to the left anterior oblique chest position and use sand bags to maintain the position.

(The patient's body is rotated 45% to the right. The left shoulder is uppermost and therefore close to the film which is in the carrier above it. In this position the right main bronchus and its branches are dependant, show up individually and are in the best position for filling. Our radiologist has found that no particular advantage is gained when filling the bronchial tree by lowering the chest or elevating the feet.)

The oblique positions are desirable because the bronchial tree divides in a direction oblique to the horizontal (when the patient is in the horizontal position). In consequence the oblique position permits clear separation and identification of the secondary bronchial divisions. The iodized oil is delivered through a special metal tube into the mouth of which a modified Burrell's syringe is fitted and locked by the nurse. The room is now darkened and the radiologist using the fluoroscope directs the bronchoscopist until the bronchoscope and the oil tube are in the requisite position, just proximal to the opening of the bronchus to the right upper lobe. The beveled end of the bronchoscope is directed laterally. At this point the radiologist asks the nurse to inject the iodized oil slowly. He watches the upper lobe bronchi fill until they are well delineated. At his command the injection of the oil is stopped and at that moment a "spot" film is taken. This film is invariably excellent and without any flooding of alveoli.

The radiologist then directs the bronchoscopist to push the bronchoscope and the oil tube slightly further into the right main bronchus and directs the nurse to inject more iodized oil. This outlines the right middle lobe and the right lower lobe and when the optimum filling has occurred a second "spot" film is taken. At this moment the "spot" film will show a clear outline of the middle and lower lobes but the upper lobe will now commence to show flooding or alveolarization.

Following this the patient is returned to the horizontal position and a postero-anterior "spot" is taken.

At this point it is our custom to pull out the oil cannula and pull up the bronchoscope so that its lower end is just above the carina.

Then the patient is turned to the left side into the right lateral position and a lateral "spot" is taken.

The oil is never forced in by the syringe—it is drawn into the smaller bronchi and from there into the bronchioles by inspiration. These distal parts fill only if sufficiently clear of secretions and if that part of the lung expands with inspiration.

The key note of good results lies in the absolute co-operation of bronchologist and anaesthetist and radiologist. All the pictures must be taken as rapidly as possible. Deep anaesthesia until the last exposure is essential.

At the conclusion some of the iodized oil is removed by suction and when the anaesthetist is satisfied with the patient's condition the bronchoscope is pulled out.

The patient comes rapidly out of the anaesthetic. Because there has been no pre-operative sedation the cough reflex returns rapidly and the iodized oil is expelled. It is extremely rare that there is any embarrassment to those watching the post-anaesthetic recovery. The work is performed in the morning between 10 and 12:30 and the child is usually sitting up by 4:30 p. m. and enjoys his supper and is discharged almost invariably the next morning.

Bronchography of the left lung is done on a subsequent occasion, after an interval of several weeks. The main consideration is that most of the iodized oil injected at the first bronchography should have vanished so that the delineation of the left bronchial segments is not impaired by overlying iodized oil in parts of the right lung.

For bronchography of the left lung the patient is first placed in the right anterior oblique position. After the filling of the left upper lobe great attention is paid to the proper mapping of the bronchus leading to the lingula. The successive positions of the patient are the exact reverse of those used in the mapping of the right lung.

Delineation of all parts of the lung is very satisfactorily accomplished by our present technique. Occasionally it happens that some segment of one lung will not expel the material filling its smaller bronchi and consequently it cannot be filled with iodized oil. Occasionally injection of iodized oil is followed by more adequate emptying of the bronchial tree and a second bronchography some weeks

later is successful in outlining the segment which formerly would not fill. There have been occasions when after lobectomy the unmapped segment of the lung has been found almost completely filled with semi-solid material which could not possibly have been expelled.

In the two papers already published modifications of the above technique have been mentioned. All of these have now been discarded.

In the literature bronchography in young children is described as accompanied by certain theoretical dangers. Four of these warrant comment. The first is the risk of the anaesthesia. We have described our methods of procedure under general anaesthesia. We have at the hospital a staff of ten certified anaesthetists and in their hands we do not believe that we now have any risk whatsoever. In our long experience we have been asked to bronchograph many very young children, many with extreme degrees of bronchiectasis, and other patients with grave conditions for carefully considered medical reasons. We do not approach such work haphazardly but on the contrary with great caution and always with every step mutually discussed by the three co-operating services. We can say without reservation that what we can do and have done at our hospital can be done elsewhere with equal satisfaction, if similar co-operation is obtainable.

The second risk put forward is that of the technical procedure of the introduction of the iodized oil. We use as stated a bronchoscope. We use always a bronchoscope relatively small for the child's larynx. We do not know of a single instance in which trauma has resulted. As already stated we teach this procedure to our residents and have done so without any damage to our little patients. We do not believe at all the statement that—"it seems nevertheless advisable to resort to bronchoscopy only when other techniques cannot be applied or are inadequate." Between us we have inspected techniques in vogue in other hospitals. In a few instances we have seen a technique neatly and dexterously performed with good bronchography but we have not seen in any other hospital bronchography performed by such a variety and such a large group of people nor in anything like the volume performed at our hospital. The results that we have seen elsewhere have been occasionally good but we can maintain without exception, that all our cases are essentially good and the great majority are excellent at the first trial.

In our hands, the third danger mentioned, the risk of respiratory trouble due to the filling of the tracheo-bronchial tree has never occurred. Our filling is done under the direct vision of the radiologist using the fluoroscope, consequently excessive filling is not allowed to occur.

The fourth danger, the risk of intoxication due to iodine we have never seen.

Certain other objections are to be found in the literature. One of these was that general anaesthesia slows down the spontaneous expulsion of the oil suspension through coughing and expectoration. This statement is simply untrue. Ether anaesthesia stimulates spontaneous expulsion of the oil suspension through coughing and expectoration. As described above we use this ether-aroused expulsion to evacuate bronchial secretions and then we, for a very short time, have the anaesthetic deepened so that the iodized oil may remain in the distal bronchioles for a long enough time to enable the radiography to take place. Anaesthesia beyond this point is unnecessary and is not carried on. The termination of the anaesthesia permits expulsion to be rapid and in most cases the bronchial tree is quickly cleared.

In certain hospitals a notice can be found posted in the bronchoscopy room to the effect that no general anaesthetic is to be given when the radiological unit is to be used. And yet in the same hospital in a room of similar size exactly the same radiological apparatus is used many times daily while patients under general anaesthesia are having work performed by surgeons treating fractures and dislocations.

I do not want to be misunderstood. We recognize dangers of all sorts and we never neglect these dangers. We take every precaution that we know of to avoid trouble.

There is always danger to the patient when an instrument carrying an electric bulb is inserted into a patient's pharynx or bronchial tree when the patient is under anaesthesia. This danger is present every day in every hospital in the land where anaesthetists putting a patient under a general anaesthetic use a laryngoscope for the insertion of the endotracheal catheter. There is always danger from a possible electric spark when the patient is being given an anaesthetic of an explosive nature.

The way to avoid trouble is to avoid the use of the more explosive mixtures when a laryngoscope or a bronchoscope has to be used, and always to make sure before inserting it that the electrical attachments are in perfect working order.

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DISCUSSION

DR. LOUIS H. CLERF: Dr. Wishart's presentation exemplifies what can be done anywhere and is being done here in Toronto at the Hospital for Sick Children in cooperation with the pediatrician and the thoracic surgeon, to learn about the condition of the tracheobronchial tree.

My interest in bronchography goes back to 1922 when I became associated with Dr. Jackson in Philadelphia. In those days the aids were not numerous. We used bismuth compounds, and anyone who used them knows that with powder in the presence of secretions one cannot visualize smaller bronchi because secretions prohibit its entrance into the smaller bronchi. I did some experimental work with sodium bromide and iodide. They were two radiopaque substances with which we were familiar but the results were not satisfactory. It was indeed a step forward when iodized oil was introduced by the French workers.

Dr. Wishart emphasizes the use of the bronchoscope. I certainly would not deprecate its use because that is the way we started instilling iodized oil in the bronchial tree. I have been weaned away from it but I have no objection to its use. I still believe the bronchoscope is an excellent aid in getting the iodized oil where desired; that is one of the fundamental things in bronchography to map out a particular portion of the tracheobronchial tree.

Secondly, he uses inhalation anesthesia. We have discussed this at great length and I must admit that I felt for a time it was inadvisable to administer inhalation anesthesia to a patient suspected of having a chronic bronchopulmonary infection. His results have been so conclusive, however, that now we have changed our routine and employ inhalation anesthesia. I still use the catheter introduced pernasally. Using inhalation anesthesia in my estimation is much safer than using sedation in large doses and attempting to use local anesthetics. You can mention any of the local anesthetics you will but I say they all are hazardous in children. Inhalation anesthesia as he has described it is in my estimation the safer way.

Bronchiectasis is a disease of young people. There is no point in waiting until children with bronchiectasis grow up before one makes a diagnosis. It is obvious that the diagnosis must be made early. Waiting for long periods may resolve an operable case into an inoperable one or bilateral disease may occur. The problem

is not simplified but usually is complicated by waiting. I feel that Dr. Wishart has done a magnificent piece of work in the field of bronchography. He has pursued this for many years and he has given the matter a great deal of thought and I want to go on record that I think he is absolutely correct in wanting to do bronchography in the young and in using inhalation anesthesia and I wish to congratulate him.

PAUL H. HOLINGER, M.D.: In observing Dr. Wishart's technic of bronchography and certainly in seeing his hospital, his bronchoscopic unit and the entire setup, one cannot help but be tremendously impressed with not only the teamwork that he has developed but the results that he has obtained. Nevertheless, it has been our practice to do almost everything exactly opposite to what Dr. Wishart has mentioned and what Dr. Clerf has possibly seconded. And yet we feel that our results still are very good from the standpoint of the information that can be obtained and given to the thoracic surgeon.

I do disagree with Dr. Wishart in his statement that infants cannot have a bronchography with the use of the catheter. We use small ureteral catheters in the newborns, and in the tiny infants, inserting the catheter through the larynx with the help of the laryngoscope and taking the child to the X-ray Department and inserting the oil in the same manner that Dr. Wishart mentioned: Fluoroscopy and spot films as the procedure progresses. We feel that there is less possibility of injury while turning the child for fluoroscopy and spot films if a catheter rather than a bronchoscope is in place.

Dr. Wishart and I have discussed the problem of explosion hazard and certainly, in his new setup he has gone into every possible precaution in avoiding explosion in that way. Nevertheless, our building code in Chicago will not permit us to use explosive anesthetics in a room, not only in which x-ray is being used but in which x-ray is installed unless the prohibitively expensive "explosion-proof" equipment is used; so that we are not allowed to use an ether anesthetic in the x-ray department. This is a building code which we cannot change. Other procedures, as Dr. Wishart mentioned, are done under general anesthesia in the x-ray department; these are done without the use of explosive anesthetics. The orthopedic procedures, for example, are done with nitrous oxide, and so forth.

The problem of personnel is an important one in the technic Dr. Wishart has described and he has solved it in his institution by having an adequate nursing and anesthetic team for the procedures he has mentioned.

In our children we use sedation and a catheter, the catheter depending upon the size of the child. The catheter is inserted with the laryngoscope and taped in place, the child taken immediately to the x-ray department, the oil instilled under fluoroscopic vision, first into the affected side. Spot films are made during instillation of the oil and then the anteroposterior and lateral films are taken. The opposite side may then be filled if desired and another anteroposterior film made followed by oblique films. If only one side is to be done we follow the same plan of film studies Dr. Wishart has made.

Again, these remarks merely show a difference in technic and are not intended in any way to criticize the excellent work that Dr. Wishart has demonstrated.

D. E. S. WISHART, M.D. (closing): One of the great advantages of such an Association as this is we have our own ideas and develop our own ideas and hear the ideas of other people. It would be a terrible thing if we all had to think the same way.

It is perfectly marvelous to have other people get good results under different techniques. We all learn from differences.

Dr. Holinger mentioned the way in which he used the catheter for little infants. I am very interested. I did not, as I listened to him, hear whether he used any sedation with it or not, and I hope afterwards he will tell me about that because a little infant involves certain problems. In the first place, as you know, to open a little infant's mouth and depress its tongue is very difficult and the doctor has to see the larynx and put in the catheter and then he has to take that child to the operating room. I want him later on to tell me how he handles that child and achieves that necessary service. I appreciate very much what he said and when I was designing our department he gave me every possible help. He showed me the designs of his operating room. I went to Chicago and saw it and I owe a great debt of gratitude to Dr. Holinger for his help.

To Dr. Clerf, I am just overwhelmed to have a master say as nice things about me as he said.

THE INFLUENCE OF THE CHEMOTHERAPEUTIC AND ANTIBIOTIC DRUGS ON THE INCIDENCE AND COURSE OF DEEP NECK INFECTIONS

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That the antibiotics have remarkably influenced the incidence and course of bacterial infections in all parts of the body is universally recognized. The object of this paper is to evaluate their influence on infections in and near the neck. Such infections in the past, while not occurring in great numbers, were not uncommon.

For purposes of comparison two groups of cases will be used, namely, one group of 78 cases reported in 1939 before chemotherapy,¹ and a group which consists of cases observed since 1940, during which time the sulfonamides and, later, the antibiotics were used.

The comparison demonstrates a recognizable change in the incidence of the entrance portals and the regions of involvement.

In the first group of 78 cases seen before 1940 there were 56 cases in which the portal entrance was the pharynx, tonsils and adenoid. In the second group of 55 cases the infection entered through the pharynx, tonsil and adenoid region in 18 cases. The percentages are 71 and 32 respectively.

There is also a striking change in the relative and total incidence of the dental group. In group 1 there were 16 cases of dental origin, 20 per cent, as compared with 19 cases, 34 per cent, in group 2. Another significant observation may be made, namely, the increased frequency of salivary glands as a portal of entrance. There were no salivary gland cases in group 1, while in group 2 there were 11 cases, nine in the submaxillary and two in the parotid. This is a fairly large percentage, 20 per cent. The only explanation that occurs to me is that at that time I had not fully realized that salivary gland infections are actually infections of the neck from the start. What

TABLE I.—PORTALS OF ENTRANCE.

Groupe 1.—78 Cases.		
Pharynx and tonsils	56 cases	71%
Dental	16 cases	20%
Lingual tonsil	1 case	
Petrous temporal bone	1 case	
Gingival Trauma	1 case	
Sialitis	none	
Not determined	3 cases	

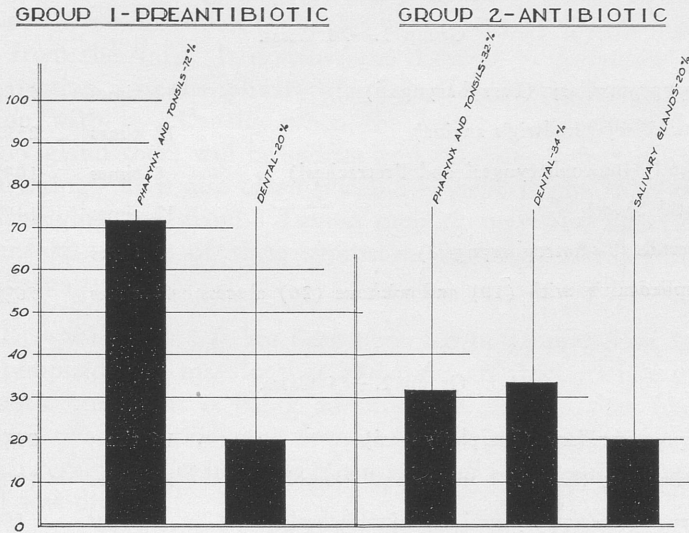
Group 2.—55 Cases.		
Pharynx and tonsils	18 cases	32%
Dental	19 cases	34%
Submaxillary sialitis (9)		
Parotid sialitis (2)	11 cases	20%
Lingual tonsil	1 case	
Scalp furuncle	1 case	
Not determined	5 cases	

few there may have been should have been included. Later, in 1942, I wrote a paper on salivary gland inflammations, and in that paper stated that they are in reality neck infections.²

Infections from the various other portals of entrance occurred in each series sporadically and the occasional appearances are so few as to make comparisons insignificant.

When the infection is seen while it is still mostly confined to the portal of entrance, and treated promptly with antibiotics, further extension is apt to be stopped and the infection soon eradicated. How many of these would have extended to the neck if they had received no antibiotics is impossible to estimate, but it may be reasonably assumed that the number would have been larger. The pediatricist and the general practitioner today treat cases which in former times

TABLE II.—PORTAL OF ENTRANCE.



were treated by the otologist and the rhinolaryngologist. A short period of treatment with the antibiotics terminates the infection at the portal of entrance, and there can be no doubt that many deep neck infections are being prevented. Occasionally there appears a patient who has a well developed infection because early medical advice was not sought. Also occasionally there appears one whose infection does not yield readily to antibiotic treatment, and this type will be discussed later. I have in the past stated that I believe that infections arising in soft parts seem to yield to antibiotic medication more readily and more rapidly than infections having their origin in bone tissues such as dental-alveolar and the temporal bone.³

Since there have been these significant changes in the incidence of the portals of entrance it may be appreciated that there will also be changes in the incidence of the regions of involvement. The same two periods of grouping are again used.

Pharyngomaxillary Infection. In group one pharyngomaxillary infection occurred 42 times or 53.+ per cent. In group two it occurred 16 times or 29.+ per cent. This indicates a very considerable

TABLE III.—REGIONS OF INVOLVEMENT

Group 1.—78 Cases.		
Pharyngomaxillary (Parapharyngeal)	42 times	53%
Submaxillary (Ludwig's angina)	18 times	23%
Visceral (Buccopharyngeal and Pretracheal)	8 times	10%
Carotid Sheath	11 times	14%
Prevertebral (Retropharyngeal)	3 times	
Lymphadenitis, with (18) and without (26) abscess	44 times	56%
Group 2.—55 Cases.		
Pharyngomaxillary (Parapharyngeal)	16 times	29%
Submaxillary (Ludwig's angina and Parotid)	32 times	58%
Visceral (Buccopharyngeal and Pretracheal)	4 times	5%
Carotid Sheath	2 times	3%
Prevertebral (Retropharyngeal)	1 time	
Lymphadenitis, with (11) and without (13) abscess	24 times	43%

reduction of pharyngomaxillary infections. It is my considered opinion that the antibiotics have brought about this reduction in the occurrence of pharyngomaxillary infection.

Submaxillary Type Infection. In group one the submaxillary type occurred 18 times, or 23.+ per cent. In group two it occurred 32 times, or 58.+ per cent, the largest single type. It is thus disclosed that the most significant change is this predominance of the submaxillary type. It is my belief that one of the reasons for this is that infection from the dental region has not been reduced, or if so, not to the same extent as has the pharyngomaxillary type. The most probable explanation is that dental infection does not usually receive antibiotic medication until the infection is already beyond the dental areas in the neck; whereas pharyngeal, tonsillar and upper respiratory infections usually receive antibiotic medication while the infection is still limited or confined to the portal of entrance. Another reason is that sialitis, parotid or submaxillary, was the portal of en-

trance in 11 cases in group 2 resulting in submaxillary region localization. When infection originates in the salivary glands it is in the neck from the start. It is sometimes difficult to determine whether the infection is in the salivary gland itself or in the lymph nodes in relation with it. Usually when the infection is primarily in the salivary gland there will be sudden and very uncomfortable swelling when eating. It is also often possible to express pus from the duct by massaging the gland. Lemon-sucking may also aggravate the discomfort. None of these symptoms and signs are present with lymphadenitis alone.

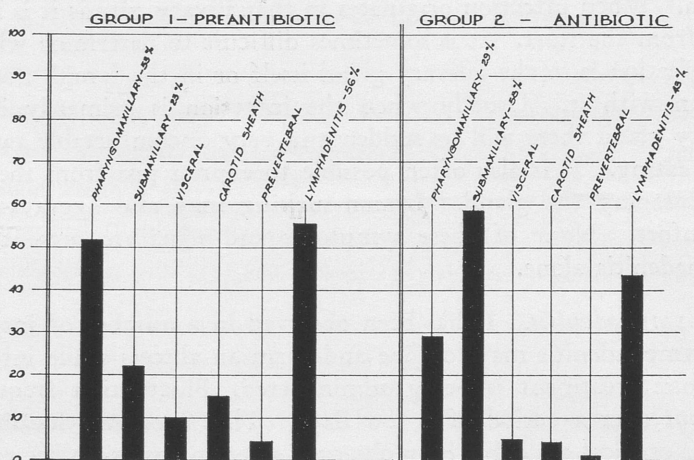
Lymphadenitis. It has been observed in a number of instances that lymphadenitis may localize and form an abscess while intensive antibiotic treatment is being administered. Fluctuation frequently develops over a period of a few days. This secondary localization in deep cervical nodes has been observed after an abscess in the parotid gland was drained.

Lymphadenitis, with and without abscess, was present 44 times, or 56 per cent, in group 1, 18 times with abscess, and 26 times without abscess. In group 2 it occurred 24 times, 11 times with abscess and 13 times without abscess. This discloses a lower percentage, 43 per cent, in group 2, and while no dogmatic conclusions may be drawn one may at least assume that despite antibiotics the lymphatic system is still very actively concerned, in a protective sense, in the presence of infection.

Visceral Fascia Infection. Infection along the visceral fascia occurred eight times in group 1, or 10 per cent, as compared with four times in group 2, or 5 per cent. It is reasonable to attribute this reduction to antibiotic therapy, not only when applied before the infection has extended into the neck and into the walls of the viscera, but also for the reason that more endoscopists are trained in skillful foreign body removal. Antibiotic therapy is very efficacious in preventing infection from minor traumas incident to foreign body removal, especially foreign bodies in the esophagus.

Prevertebral Fascia Infection. Prevertebral fascia infection, or retropharyngeal abscess, occurred three times in group 1, or 3.+ per cent, and once in group 2, or 1.+ per cent. Because of the small number of cases percentages here have no significance, and it may be concluded that no change has been brought about. Retropharyngeal

TABLE VI.—PURE CULTURES.



abscess occurs usually during the first two years of life, and therefore may be expected to remain about as formerly for the reason that the infection in infants is fully developed before it is recognized and before antibiotics have been administered.

Carotid Sheath and Blood Stream Infection. Carotid sheath infection with or without blood stream infection occurred 11 times in group 1 or 14 per cent, and twice in group 2, or 3 per cent. In one of these two cases the patient had a number of chills with excessive sweating, but the blood cultures were negative. She was treated with sulfonamides before penicillin was available and was the only death in group 2. She was extremely septic when first seen and the death was from septicemia.

This shows a significant reduction in blood stream infections in the antibiotic group, and is evidence that the antibiotics exert a pronounced protection against sepsis. This is markedly reflected in the appearance and general demeanor of the patient. Under intensive antibiotic therapy it is rarer for a patient to have chills. There is, however, some diaphoresis but much less in evidence than when no antibiotics were available. Therefore dehydration is also much less in evidence than formerly. Moderately high septic blood counts are present in the beginning, but after a few days of antibiotic therapy

the total white cell count and the neutrophile percentage are reduced. The non-segmented neutrophiles, or young forms, are not so much increased when the antibiotics are given. The shift is still there, but less.

In pre-antibiotic days the eosinophiles promptly disappeared from the blood count in the early intense stage of the infection, and their reappearance was interpreted as a sign that the infection was no longer so severe. When antibiotics are administered early in the infection the eosinophiles frequently do not disappear from the count, but remain throughout the course of the disease.

Sometimes, particularly when the sulfonamides are being given in full dosage, there will occur leucopenia and neutropenia, and, perhaps, agranulocytosis. In such cases the sulfonamides should be discontinued, but penicillin may be continued, and folic acid administered, an initial dose of 100 mg and thereafter 20 mg daily for adults. In these cases the temperature is apt to remain high until the folic acid is added to the therapy.^{4, 5}

If the infection has been prolonged there may develop an anemia which requires appropriate medication, but in infection whose duration is not prolonged anemia is usually of mild degree only.

The absence of general symptoms and signs of sepsis makes it necessary for the clinician to rely more on the local manifestations at the site of the infection, and when these are obscure and not frank he may be faced with quite a dilemma in evaluating the course of events. The optimum time for drainage and the very indications for any drainage at all are thus much more difficult to determine. At such a time it will be necessary to penetrate or see through "the penicillin fog," an expression I first heard used by Dr. H. P. Mosher. Until the infection is really under full control and practically cured the sedimentation rate will remain high. In the presence of a blood count which is not much increased considerable diagnostic help is derived from the sedimentation rate. If this is normal the sepsis may be regarded as mild or absent.

Under penicillin medication the total white cell count is lowered sooner than the neutrophile percentage. This is also true after a drainage operation. Penicillin does not disturb the blood count as much as do the sulfonamides. With either there may be expected a

lowering of the leucocytosis and the neutrophile percentage, but with the sulfonamides this is apt to be excessive and is largely a toxic manifestation in that there occurs an abnormal leucopenia and agranulocytosis. This toxic effect is not often, if ever, caused by penicillin which lowers the total and neutrophile counts indirectly by removing the infection, and not by any toxic action on the hematopoietic organs.

BACTERIOLOGY

The information which is derived from routine bacteriological investigation in these cases of neck infection is quite confusing, and the results as recorded on the chart often seem inconsistent. In those patients who have recovered without surgery the culture has not been taken from the inflamed tissues of the neck, but from the portal of entrance which may have been the pharynx, tonsils, dental region or mouth. It is therefor usually a mixture of organisms, any one of which may be the invader of the neck. The evidence is therefor highly inferential. Sometimes organisms from an abscess do not grow on cultures and a report "no growth" is received, and this even when a large quantity of pus from a large abscess is transferred to the culture medium. The organisms may have been rendered incapable of growing by the antibiotic therapy. However, it must also be remembered that such "no growth" reports were encountered before the antibiotics came into use. Also, pathogenic organisms may be cultured from a perfectly normal mouth and throat. A bizarre combination of organisms is often reported; in fact these combinations comprise a majority of the throat culture reports. After drainage we look for and hope to get a pure-culture report, for then we may be reasonably certain that we have adequately reached the infectious location. In this connection, however, it must be remembered that even though a pharyngomaxillary abscess has been drained high up, there may still occur an abscess of the deep lymph nodes lower down on the sheath.

Chemotherapy has, if anything, added to the difficulties by inhibiting the growth of organisms on culture. If there has been sufficient time bacterial sensitivity tests will sometimes help by indicating the selection of the most effective antibiotics. The tests should be started as soon as cultures are available. It has been customary to use the throat and mouth cultures for this purpose. Sensitivity tests are not mathematical certainties. I have seen infections yield to

TABLE V.—PURE CULTURES.

Group 1.—Preantibiotic			
78 Cases	39 Times	50%	
Streptococcus hemolyticus	24 times		
Streptococcus non-hemolyticus	3 times		
Streptococcus viridans	2 times		
Streptococcus, not typed	3 times		
Streptococcus hemolyticus in blood stream	1 time		
Staphylococcus aureus, not typed	1 time		
Staphylococcus albus, not typed	3 times		
Staphylococcus albus hemolyticus	1 time		
Pneumococcus	1 time		
Group 2.—Antibiotic			
55 Cases	16 Times	29%	
Streptococcus hemolyticus	3 times		
Streptococcus non-hemolyticus	2 times		
Streptococcus viridans	1 time		
Staphylococcus aureus hemolyticus	5 times		
Staphylococcus aureus, not typed	4 times		
Staphylococcus albus, not typed	1 time		

penicillin therapy when sensitivity tests were reported resistant to penicillin. When the neck abscess has been reached by surgical drainage recovery is usually so rapid and satisfactory that sensitivity tests will not be necessary.

A comparison of pure cultures in the two groups is noteworthy and somewhat striking. In group 1, 78 cases, pure cultures were found 39 times, or 50 per cent, of which 24 cases were streptococcus hemolyticus. In group 2, 55 cases, pure cultures were found 16 times, or 29 per cent, and the streptococcus hemolyticus occurred only three times. No growth was reported as often, four times, in the 55 cases of group 2 as in 78 cases of group 1.

Several conclusions seem justifiable: (a) there is not the predominance of the streptococcus hemolyticus in the antibiotic group that there is in the pre-antibiotic group: (b) the antibiotics do exert a definite inhibitory effect on the growth of organisms on culture; (c) the antibiotics, particularly penicillin, have had their best effect on the streptococcus hemolyticus; and (d) the other organisms now encountered, such as the staphylococci, are more resistant.

CURES WITHOUT SURGICAL DRAINAGE

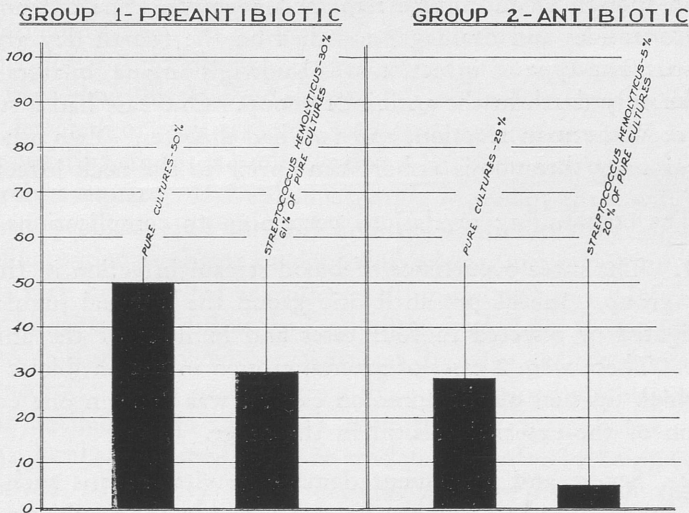
In the pre-antibiotic group 1 there were five cases in which there was subsidence of the infection without surgery, 6.+ per cent of the 78 cases. One case, not included in these, had an abscess which ruptured spontaneously through the tonsil fossa and recovered without surgery.

In the antibiotic group 2, seven cases were cured by the antibiotics without surgery. The longest stay in the hospital was ten days and the shortest, three days. There were five cases of the pharyngeal and parapharyngeal type in one of which the hypopharynx and extrinsic larynx were also involved. The remaining two were the submaxillary type, both of dental origin, one developing a severe well-marked Ludwig's angina which at first was fully expected to require surgical drainage. She was discharged cured on the tenth day without having had any surgery. Those seven cases comprise 12.+ per cent of the total 55. Therefore, it may be justifiably stated that between 10 and 15 per cent of neck infections may be terminated with antibiotic therapy if seen early.

Since writing this paper I have treated another case with supra-hyoid dentoalveolar infection which recovered under penicillin therapy and without surgical drainage. There was extreme trismus, and a frank Ludwig's angina was already present. If surgery had become necessary a preliminary tracheotomy would have been unavoidable.

How often an incipient infection is aborted at the portal of entrance when therapy is administered at such a time it is impossible to estimate, but there must be a great number. Wanamaker et al.⁵ reported on the effect of penicillin and aureomycin on a series of 475 cases of pharyngitis and tonsillitis. Results were better than without treatment. Suppurations were unusual in all groups. Seven cases later developed acute rheumatic fever, five in the untreated control group. Comparatively the results with penicillin and with

TABLE IV.—REGIONS OF INVOLVEMENT.



aureomycin were about the same, but penicillin prevented the carrier state while the aureomycin did not. To me it is surprising that there were not more cervical lymph node infections and complicating suppurations. They still occur.

COMPLICATIONS

It will be more revealing to mention these in the absentee sense. In the 78 cases of group 1 there were 54 cases with no complications, or 69 per cent. When complications did occur they were severe and dangerous. In the 55 cases of group 2 there were 34 cases with no complications, or 61 per cent. Accordingly complications have not been reduced, but have changed in kind and degree. In group 2 there were two cases of leucopenia and two cases of toxic nephritis caused by the sulfonamide medication, and there were three cases requiring preliminary tracheotomy for restoration of the airway despite sulfonamide medication in one case and penicillin in the other two. The two which received penicillin were dyspneic early, before admission to the hospital. The sulfonamide case was in an advanced stage of Ludwig's angina, and tracheotomy was necessary before anesthesia could be administered. There was only one case with blood

stream infection in group 2. She had severe chills even before admission though blood culture remained negative. She did not yield to sulfonamides and drainage, and died on the fourth day after admission. The type of infection was Ludwig's angina, bilateral. This was the only death in the antibiotic group. One case had a strongly positive Wasserman reaction, and two had diabetes. Two others had had coronary thrombosis a short time prior to the neck infection.

The outstanding revelations pertaining to complications are:

1. The rarer occurrence of blood stream infection in the antibiotic group. In the pre-antibiotic group the internal jugular vein was ligated or resected in four cases and in none of the antibiotic group. There were 2 cases of arterial erosion in the antibiotic group, for which ligation of the common carotid was done in one case and ligation of the external carotid in the other.
2. Severe and permanent damage to vital organs such as the heart, lungs, liver and kidneys did not occur in the antibiotic group. The importance of this cannot be overestimated.
3. The necessity for tracheotomy has been increased because of the greater incidence of the Ludwig's angina type of infection.

DIAGNOSTIC DIFFICULTIES

Formerly the local and general manifestations were frank and severe. The patient was very uncomfortable locally and very ill generally. The antibiotics change such a picture very soon after administration is begun. The local manifestations are much less uncomfortable and the general manifestations are so much ameliorated that the patient seems not nearly so ill. Frank blood stream infection is now a rarity, and while there may be some diaphoresis, it may be very easily overlooked if no inquiries are specifically made. Digestion is not so much deranged; chills are not so commonly present. For all these reasons dehydration is not the pronounced problem it formerly was. The changes in the blood count are not so pronounced, at least not after antibiotic medication has been well established, for which reason lower blood count readings must receive more consideration than similar counts would have received formerly. The sedimentation rate should be more frequently done than formerly for it may continue to be increased when the count is hovering around the upper normal levels.

If an abscess deeply situated is already in existence progressive enlargement or extension may be checked, and a more or less static condition established. In other words it will remain as a small abscess which is difficult to detect. Surrounding tissue inflammation and edema may show marked subsidence. If at such a time an abscess can be detected, or even strongly suspected, surgical drainage will be rapidly curative, and it is probable that a less extensive operation will be entirely adequate. If frank fluctuation is present there will be no diagnostic problem, and surgical drainage is immediately indicated. When fluctuation is absent or doubtful, edematous pitting on finger pressure may be regarded as evidence of underlying pus. In cases in which the process is diffuse and cellulitic, and there is not yet any localization, antibiotics without surgical drainage are often curative. In the necrotic tissues of an abscess there is no blood circulation and therefor antibiotics do not reach such tissues. Both horns of the dilemma, local and general, must be rendered harmless by being capped or removed. Formerly in an extremely severe infection the imminence of blood stream infection justified surgical drainage long before any evidence of fluctuation was present. In many cases no free pus was encountered, and the operation prevented blood stream infection. If the disease does not in these days yield to antibiotic therapy in a reasonably short time, two to four days, surgical drainage is indicated as in preantibiotic times. When in doubt drainage is still the safest course. Diagnostic acumen is now in much greater demand.

ANTIBIOTIC THERAPY

All of the cases in group 2 were treated with either the sulfonamides or penicillin, or both. Only in one or two cases were any of the others used, chiefly for the reason that until 1943 only the sulfonamides were available to us, and later, because the infections yielded so quickly and so conclusively to penicillin, with and without drainage, that there were no further indications for any kind of medication. No cases were encountered in which penicillin could not be administered. There were none that showed any of the allergic manifestations we now encounter more frequently. Because large numbers of patients have received penicillin for previous illnesses of many kinds a greater number of patients in the future will have developed a sensitivity to penicillin. The chief concern or hazard with penicillin therapy is the possible development of penicillin-

resistant organisms. During the course of any one illness this possibility is not common because the course is usually of short duration and organisms are not apt to become resistant in so short a time. In the event of resistance of organisms other antibiotics are now available. The same may be said with regard to allergic sensitivity, but penicillin is to be regarded as the most effective and least toxic of all the antibiotics now available. When a frank abscess is already existent, and prompt drainage is applied it is frequently unnecessary to give any antibiotic therapy. Antibiotic therapy alone, without drainage, does not cure such cases. If no abscess is reached by drainage, penicillin therapy is very important. Penicillin G is now the commonly used preparation. In cases of allergic sensitivity it will be necessary to look for substitutes. A newer preparation, penicillin O has been evolved, which is designed for such contingencies. Others are also becoming available. While penicillin administered orally has its advocates⁷ who show proof of adequate blood levels, I have not used it, fearing that allergic sensitivity is more likely to be produced. It is now well known that penicillin lozenges frequently cause very marked stomatitis and gastrointestinal disturbances. In these extremely acute and fast-traveling neck infections hospitalization and intensive parenteral medication are prime necessities. If the patient has had no antibiotic medication it is well to start with one dose of penicillin G in saline by vein and at the same time one dose intramuscularly; then continue with an intramuscular dose of the same every 3 hours around the clock. This is continued until conditions show marked improvement, perhaps after drainage if that becomes necessary, or after the improvement indicates that drainage will not be necessary. After that one of the newer repository-type preparations may be substituted, one or twice daily to lessen the number of needle punctures and to avoid disturbing the patient during sleeping hours. The original aqueous penicillin G appeals to me greatly because of its rapid absorption and effects. The average dose of the aqueous solution is 100,000 units every three hours for adults, and that of the repository-type 300,000 or 400,000 units once or twice a day according to the severity of the illness. Until now I have not had to give penicillin O in neck infection cases.

During the war when penicillin was very scarce and strictly allocated, results seemed very striking even with the small doses of the crude preparations which were then available. At that time the ether every two to four hours. The strength of this solution is more

usual dose was 20,000 units for adults. Can it be that organisms have acquired increased resistance, and that larger doses are now necessary? Or have antibiotics which patients have repeatedly received rendered the natural immunizing mechanisms of the body less active? It could be partly both. It is now believed that the ordinary head cold is instigated by a virus infection which in practically all cases sets the stage for invasion by bacteria secondarily. If the lungs are immediately involved the treatment is usually started with one or another of the newer antibiotics such as aureomycin, chloromycetin, terramycin or streptomycin. This seems logical though penicillin is also indicated for the secondary bacterial invasion. Not many neck infections are preceded by this type of virus head cold onset, and in this minority the initial phase has passed before the neck is involved. During the height of the neck infection these newer antibiotics, other than penicillin, are rarely needed. By the time one ascertains the specific type of organism or organisms present, and sensitivity tests are made, the patient is usually well on the way to recovery. Occasionally I have used an aqueous solution of penicillin locally in the wound, introduced through an indwelling catheter which protrudes through the dressings and bandage. The nurse is instructed to inject five, ten or 20 cubic centimeters of the solution, containing 500 to 5000 units to the cubic centimeter, into the cath- or less arbitrary as it is non-irritating. I have not yet observed any allergic sensitization from this procedure.

In extremely severe and, perhaps, late cases when time-saving is important it may be advisable to add streptomycin to the penicillin therapy for a few days because streptomycin is more effective against gram-negative organisms.⁸ Preparations combining penicillin and streptomycin are now available. None of the antibiotic drugs are wholly innocuous, and it may become necessary to discontinue administration and very carefully select substitutes. One must be constantly on the watch for evidences of toxicity which evidences must not be confused with the clinical manifestations of the infection or its complications.

HORMESIS

This term is used to describe the stimulating effect of an antibacterial agent on the growth of otherwise sensitive organisms. It may also be used when organisms which are ordinarily not numerous or pathogenic become so when a pathogenic penicillin-sensitive or-

ganism is eliminated by penicillin therapy. The development of moniliasis is an example. Ordinarily the monilia organisms are not numerous or pathogenic, but may become so when the commonly present pyogenic bacteria disappear as a result of the antibiotic therapy. The constituents of the bacterial flora normally present are thrown out of balance, and a severe unopposed monilia infection may result. This may occur anywhere in the alimentary canal and cause severe illness. Also, the natural antagonism of one type of pathogenic organisms, e.g. Gram positive, for another type of pathogenic organisms, e.g., Gram negative may be eliminated by the antibiotic. It is when the removal of the pathology in the portal of entrance, e.g., diseased tonsils, must be delayed for some reason, that there may occur relapses of the original infection after the infection in the neck has been drained and healed. Resumption of the antibiotic therapy in such cases may bring about these hormetic effects. One case in this series is an example of this tendency.

REPORT OF A CASE

Case 55, a 22 months old female child, was admitted to the hospital with a history of three days of upper respiratory infection consisting of acute nasopharyngitis, tonsillitis, an infected left lower molar tooth, gingivitis, acute bilateral tubo-tympanic catarrh, and cervical lymphadenitis on the left side. The patient was given penicillin and aureomycin by the family physician with prompt subsidence of all the inflammation except in the cervical nodes of the left side. These broke down and became fluctuant, and a large abscess was drained on the seventh day. The recovery was then uninterrupted, and the patient was discharged on the fourteenth postoperative day, the discharge from the hospital being delayed because of respiratory infection at home. This case is an example of what I have repeatedly observed, namely, that a lymphadenitis may go on to abscess formation after the infection has subsided at the portal of entrance under intensive antibiotic therapy. It is also an example of "no growth" culture reports from the abscess. The throat culture on admission was *M. catarrhalis*. Two weeks later the patient was readmitted to the hospital with recurrent infection of the upper respiratory tract, given antibiotic therapy and discharged eight days later. Several days later she developed a severe attack of chicken pox from which she made an uncomplicated recovery. When seen in the office a week later the tonsils were spotted with white patches of monilia which was attributed to hormesis. The patient was then readmitted to the hospital for intensive treatment so that the tonsils and adenoid could be removed. Penicillin was again given and the throat was swabbed three times a day with 1 per cent solution of gentian violet. A marked improvement was immediately recognizable, and when the throat was comparatively clean the tonsils and adenoid were removed, following which the condition went on to complete recovery. Before and after the operation folic acid injections were given for neutropenia.

This case emphasizes the fact that the antibiotics may enable clinical problems to develop with which we were not familiar in the past.

In summing up the present and future status of the antibiotics I feel that I cannot do better than to quote directly from the last chapter of the Welch and Lewis book on Antibiotics.⁸

"Properly used these drugs are life saving, perhaps more so than any other agents available to the clinician. Improperly used they can be dangerous drugs. The inherent toxicities of certain of the antibiotics such as the eighth nerve damage caused by streptomycin, the nephrotoxicity of bacitracin, and the possible sensitization that may occur with any of them, are all obvious and can be controlled through judicious use. Other effects of these drugs are less obvious, and perhaps present more serious problems for the future. The development of tubercle bacilli resistant to streptomycin already portends a future public health problem. Indiscriminate use of this drug will accentuate this dangerous possibility. The development of increasing numbers of staphylococci resistant to penicillin and other antibiotic drugs is a problem that will increase in importance as time goes on. Perhaps more important than any of these is the ability of the antibiotics to stimulate rather than retard the growth of microorganisms in vivo."

CONCLUSIONS

1. Blood stream infections have been reduced by the antibiotics.
2. Tracheotomy will still be necessary occasionally because of the frequency of the Ludwig's angina type of infection.
3. Pure cultures of streptococcus hemolyticus are now less frequent. The staphylococci and the alpha streptococci are more frequently present.
4. Moderate masking of symptoms and signs is to be expected under antibiotic therapy.
5. The visceral fascia type of infection has been reduced from 10 per cent formerly to 5 per cent now. Some of this reduction may be attributed to fewer infections from foreign bodies.
6. Lymphadenitis, with or without abscess, occurred in 56 per cent formerly and 43 per cent now—a moderate decrease.
7. The regions of involvement have changed from a predominance of pharyngomaxillary, or parapharyngeal type formerly to a predominance of submaxillary, or Ludwig's angina, type now.

8. The portals of entrance have changed from a predominance of pharynx and tonsils formerly to a predominance of dental and salivary glands now.

9. It is not yet time to throw away our scalpels, and I feel it never will be.

421 HUGUENOT ST.

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DISCUSSION

FREDERICK T. HILL, M.D.: This paper is most timely, when we think of the bacteriological confusion that we are in at the present time due to the indiscriminate and often abused use of antibiotics.

I am very pleased that Dr. Beck has mentioned the danger of masking of symptoms resulting in a false sense of security, because I am sure all of us have many times seen these cases where treatment has been postponed through this false security; and only later, when the consultant has been called in, has a serious complication been avoided.

I was very much interested in the change in the percentage regarding the tonsillar portal of entry and the dental. It seems to me that before the period of antibiotics, as a rule, the dental type of infections were less virulent as compared to those from the tonsil, and perhaps this may in some way explain the difference.

The matter of salivary glands has been mentioned as a portal of entry. I don't quite see why the increase unless they were not considered in the cases in

the earlier period. Certainly I think that we saw as many salivary gland infections prior to the use of antibiotics.

JAMES M. ROBB, M.D.: In the care of deep infections of the neck what was said about the use of penicillin and penicillin mist is important.

When we are concerned about the extensions of deep infections in the neck we should always keep in mind the value of a tracheotomy. I remember at the Receiving Hospital in Detroit having seen a young man with involvement of the aryepiglottic area with some obstruction to respiration. I advised that he be let alone. This was a grave error. A tracheotomy should have been done then.

With the suction we have at present, with chemotherapy; and with the antibiotics, if a good airway is established, with proper care almost every deep infection of the neck is brought to good conclusion.

PAUL H. HOLINGER, M.D.: I enjoyed Dr. Beck's paper very much and only want to mention one point to record a warning of one of the problems that has arisen in foreign body work. Dr. Beck mentioned the decreased incidence of infections that occur following foreign body accidents due to the administration of antibiotics. He, himself, mentioned the problem of the masking of symptoms of infection by the use of antibiotics. This is extremely important but sometimes is completely forgotten. Certainly the use of antibiotics routinely following the extraction of a foreign body from the esophagus is to be very definitely condemned unless one has evidence of infection or perforation; should such evidence be apparent, the antibiotic should be administered, of course, to the fullest extent. But the administration of the antibiotic for the twenty-four hours the patient is in the hospital should be avoided since it is inadequate to treat infection and only adequate enough to mask infection if it is present. For example, we have found, to our chagrin, that some residents give antibiotics immediately following routine foreign body extraction for no real reason; and then the patient is discharged the following day because his temperature is normal; under these circumstances no accurate index of the patient's condition is available and this routine is certainly one which is in error.

Our routine at present is to avoid the routine use of antibiotics following foreign body extraction unless there is obvious infection at the time of extraction.

LOUIS H. CLERF, M.D.: I am highly in accord with what Dr. Holinger said about the use of antibiotics in potential cases of infection. If you are going to use them because there is an indication, well and good. It is a grave mistake to discharge a patient until after the use of the antibiotics has been discontinued for a day or two. It is amazing what may happen 24 to 36 hours after an antibiotic has been discontinued. Secondly with regard to tracheotomy if there is any question about obstruction to the airway, one should resort to tracheotomy. I have seen some very unhappy results where the decision was made not to have tracheotomy done because it was not considered necessary or if necessary that it always could be done later. That is presumptuous. One can always decannulate a living patient, and if the tracheotomy was needless the most one will get is a scar and a good plastic surgeon can take care of that.

FRANCIS W. DAVISON, M.D.: Dr. Beck has given us much valuable information. I wish he had been a little more definite about which antibiotic was used and the dose that was selected.

As time goes on I think we have less and less faith in aureomycin and terramycin, despite the culture reports that I have previously given. We know they

are primarily bacteriostatic. Penicillin if given in adequate dose is bactericidal. Streptomycin if given in adequate dose is bactericidal. I think we are all seeing more penicillin resistant infections. Of course we all know when pus has formed the antibiotics are inadequate and that surgery must be done. I am of the opinion it is more blessed to prevent than evacuate pus, so if we can see these serious neck infections early, I think our job is to give adequate amounts of penicillin. My dose at present is five million units a day. If the infection really serious, I give ten million units daily. It is not expensive at the present time and it is easily given. I give two million units of fortified procaine penicillin intramuscularly every four hours and omit the four a.m. dose.

AUGUST L. BECK, M.D. (closing): I don't remember having had a great number of salivary cases. When I began to use the first antibiotics I did not use them for neck infections. I think there would have been some that would have made a better lamp post but we will have to do with what we have.

I agree with Dr. Holinger 100 per cent that on the problem of masking for 24 hours, you will not get symptoms when you hit them with large doses of penicillin after taking out a foreign body. You won't get symptoms until after you discharge the patient. If you are going to give antibiotics you have to keep the patient in for a while and watch him very carefully.

There is a possibility I find—I had one such instance not so long ago—of sensitizing a patient with just a few doses of penicillin when one uses a lot. When one uses a lot of penicillin one always has to ask, even in cases where given prophylactically, if they are sensitive.

I had one unusual case where an acquaintance playfully stuck a wire into a young man's ear. It went through the ear drum and the hearing was very much impaired. I did not know whether it had dislocated the stapes or perforated the inner ear wall, or what, and I put him in the hospital right away and got him under antibiotics, heavy doses of antibiotics, as fast as I could. I let him go home in a week or ten days' time and after he got there he developed the most severe case of sensitization I have ever seen, urticaria all over the body. He had arthritic symptoms. He could not even walk from the bed to the bathroom. That was given prophylactically. We did prevent a labyrinthitis and meningitis in that case but we did sensitize him too.

Dr. Clerf's remarks on tracheotomy are appreciated, and I am glad to be supported on this because I feel exactly the way he does about it. In fact, in a great many cases it is a terrible expense. In acute infections of the neck you don't have to do much thinking to make up your mind to do tracheotomies. It is absolutely imperative that you do them.

I reported a case last year where a two and one-half months' old infant came in with the neck swollen, dyspneic, who had a history of antibiotics over a period of several weeks by the family pediatrician and the infection going merely on, no reduction in temperature. The patient came in almost in the last stages. I could not even examine the mouth. The tongue was terribly swollen, trismus was terribly extreme. The only thing that enlightened me at all was lateral neck x-ray. I expected to find a retrolateral pharyngeal abscess. This child had not even erupted a tooth. I did not think of a dental origin but there was a real frank Ludwig's angina, with the tongue pushed backwards and upwards causing the intense dyspnea. I could not do anything until I had done a tracheotomy. I did it under local anesthesia. At the same time I did a midline approach to the submaxillary region toward the mouth through the median raphe of the mylo-

hyoid muscle and I evacuated what seemed to me an inordinate amount of pus. It did not seem possible that such a small tongue could hold so much pus. The child got well. It is about four or five years old now, and last month I took out the tonsils and adenoids and the child is still going strong.

Dr. Davison's remarks with regard to the antibiotics, are answered in the completed paper. As it is going to be published there will be more on the details of that part of it but I would like to make a point, so long as it has been asked, in these very acute, fulminating conditions I like to go back to the aqueous penicillin in saline. I give one dose, in the case of an adult, 100,000 units intravenously and at the same time 100,000 units intramuscularly around the clock every three hours until that patient is better, whether drained or not. As soon as I have the temperature down some and the blood count better and the patient feels better, then I change to the repository types, giving three to four hundred thousand units twice a day in the beginning and once a day and taper off, stopping. It is a good idea to give them some streptomycin in the beginning along with it for a couple of days because the penicillin takes care of the gram negative organisms.

Sensitivity tests are also covered in the complete paper. I just mentioned it in one sentence today. In these cases of neck infection the process is so acute you don't have time, or when you get an opportunity to do sensitivity tests on the organisms which are actually causing the neck infection, you have already drained it. Then it is no longer necessary. The cultures that you get from the mouth and throat are such that you rarely get a pure culture from that region. You don't know which of these is the invader of the neck or going to be the invader of the neck, or if none of them are. So sensitivity tests in acute neck infections are not always applicable. By the time you get around to doing them the patient is probably better.

CARTILAGE HOMOGRAFTS IN RHINOPLASTY: A CRITICAL EVALUATION

SAMUEL SALINGER, M.D.

CHICAGO, ILL.

Of all the materials that have been employed for the correction of defects in the contour of the nose, none has stood the test of time as well as human cartilage. This does not necessarily imply that cartilage is ideal in every case nor does it mean that it is 100 per cent effective but, by and large, in the majority of cases it can be relied upon to accomplish a lasting result. Theoretically, the ideal material should be one that approximates as closely as possible the tissue it is supposed to supplant, should be capable of being retained without change in its new environment, should be readily obtainable and easily molded to the desired shape. Furthermore, it should possess sufficient density to withstand the normal minor traumas to which the nose is exposed, and should not be so rigid as to be unyielding under normal stress.

On the basis of the standards thus set forth, it is readily apparent that neither cartilage nor bone alone, and certainly no foreign body material, possesses all of these qualifications. The nearest approach to this ideal would be a composite autogenous bone and cartilage graft taken from the rib. While the difficulties of obtaining such a graft are not too great, the problem of shaping is more complex because of the difference in density between the two components. A smoothly sculptured contour is essential inasmuch as the least irregularity is visible through the overlying skin.

Autogenous costal cartilage alone is almost ideal but has certain disadvantages which must be reckoned with. The most annoying of these is its tendency to curl after being in situ for some time. Despite the most careful cutting and shaping, this phenomenon makes its appearance in a certain percentage of cases within a few weeks to a number of months. In an effort to prevent this complication New and Erich¹ suggested boiling the cartilage. This pro-

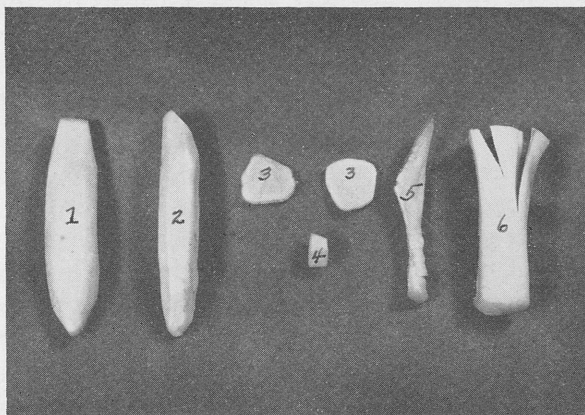


Fig. 1.—(1) Dorsal implant, top view; (2) side view. (3) Thin discs cut from full thickness of rib. (4) Small "button" for implantation into nasal top. (5) Strip showing tendency to bend. (6) Full thickness of rib cartilage sliced to show tendency to curve in direction of cortex.

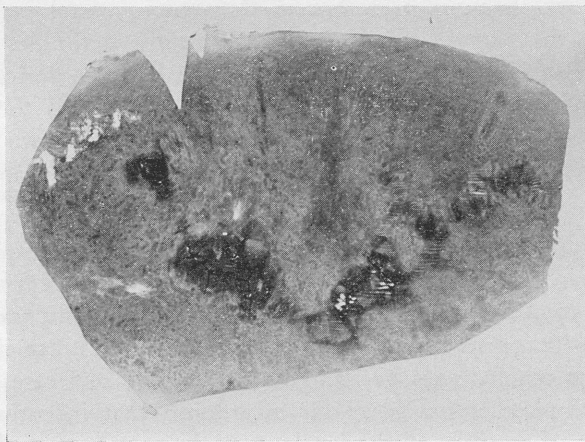


Fig. 2.—Section of full thickness of rib showing differing densities of cortex and medulla.

cedure, however, means that the viable cells are destroyed, thus depriving the implant of its viability and losing any chance it might have had to survive as a living structure. It reduces the implant to the status of refrigerated cartilage and makes the resection of a rib an unnecessary operation.

My observations on the tendency of costal cartilage to curl lead me to the conclusion that this phenomenon is due to the difference in density between the cortical and medullary portions as well as to the persistence of fine strands of perichondrium. This can be easily demonstrated by slicing off a thin strip from the cortex of a piece of rib cartilage and noting how promptly it forms an arc in the direction of the denser layer (Fig. 1 and 2). How to minimize this tendency is a matter that I shall discuss later in the paper.

Another difficulty with cartilage lies in the texture of the particular piece in question. Cartilage of uniform density is extremely difficult to obtain, since it is found only in young well-developed adults. Cartilage obtained from children and adolescents is incompletely chondrified and, being partly fibrous, is more likely to be absorbed. On the other hand, cartilage obtained from adults approaching middle life and beyond is partly ossified and experience has shown that the lacunae of the ossified portions are soon invaded by tissue juices and various wandering cells which absorb a portion of the implant, leaving conspicuous irregularities in contour.

The fact that our preference is for young costal cartilage does not imply that septal cartilage is not equally desirable. Peer² has shown that this material will retain its shape for long periods of time. However, the chief shortcoming of septal cartilage is the fact that its bulk is not adequate for most of the saddle noses we have seen. There is no question that for minor defects it is a most suitable material.

In addition to the items just mentioned one has to take into consideration also the possibility of postoperative infection and foreign body reaction. These complications, though comparatively rare since the widespread prophylactic use of antibiotics, are nevertheless a possibility which must be recognized. In the few cases which we have encountered it has been our impression that infection was not due to lack of sterility of the implant or the solution in which it was kept, but rather to the presence of pathogenic organisms in the operative field. One must, therefore, be extremely careful to avoid

operating on a patient who has recently suffered an acute upper respiratory infection, or in the presence of an active sinusitis.

In a further effort to avoid contamination from within the nasal cavity, it has been our practice in most cases to introduce the implant through an incision in the midline of the columella near the tip of the nose. The advantages are that the field can be completely sterile, which is not always the case with intranasal incisions, and the pocket which receives the implant is completely shut off from the intranasal cavity. Another advantage of this approach is that the pocket can be tunnelled more accurately since it is approached directly in the midline, whereas an intranasal incision must be in one or the other vestibule, which involves displacing the tip in order to maintain the midline dissection. If the incision is accurately placed in the midline and carefully sutured without tension, the resulting scar will be scarcely noticeable even on close inspection.

As for foreign body reactions, it is difficult to account for this occurrence inasmuch as in the instances where they were observed, the cartilage came from the same batch where the preponderance of successful takes would indicate that intolerance was not due to the cartilage itself. It is significant to note, however, that foreign body reactions were more common in columellar implants than in the dorsal. I believe this can be accounted for by the fact that the columella is subjected to greater stress than the nasal dorsum, due to traction of the upper lip in talking and eating. Also, the columella is more exposed to trauma. These factors also account for the more frequent absorption of columellar struts than is the case with dorsal implants. Stress and tension due to the scars of previous surgery or trauma will certainly predispose to rapid absorption of a cartilage implant in most cases.

The situation as regards dorsal implants is different. Here a properly shaped implant that is not introduced under tension can become firmly attached to the underlying bone and remain intact for an indefinite period of time. I have found it advantageous to elevate the periosteum over the nasal bones up to and beyond the nasion and to roughen the bone with a rasp. A firm fibrous union takes place between the roughened bone and the cartilage, which fixes it so that it is immobile. At the same time the lower portion of the graft which is less firmly attached is capable of being bent from side to side in conformity with movements of the mobile portion of

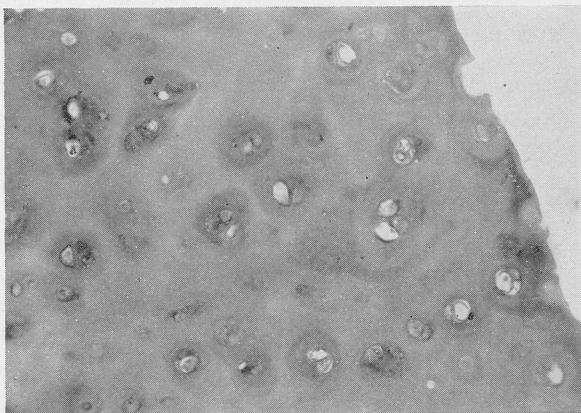


Fig. 3.—Cartilage removed after having been in situ over five years.

the nose. This makes it more desirable than a bone implant, which is too rigid if it is long enough to extend below the lower end of the nasal bones. Furthermore, experience is beginning to accumulate to show that a bone implant which is not in contact with bone is likely to be absorbed.³

With regard to the prevention of curling, I have found this complication extremely rare when the implant is cut from the core of the full thickness through its long axis, thus balancing the cortical and medullary portions. This is illustrated in Figure 1, which also demonstrates how the upper end of the graft is bevelled so as to fit closely into the curve of the nasion.

In order to insure fixation of the implant exactly in the midline, it has been my practice to apply mild pressure with a well-padded molded copper splint for a period of at least four days.

As to permanence of these dorsal implants, I have observed cases for as long as twenty-five years without noting any appreciable diminution in their size or shape. Examinations of specimens removed after a long period of time show a remarkable degree of preservation of the cartilage stroma despite loss of viability of the cells (Fig. 3).

In this connection, the case reported by Brunner⁴ is particularly significant, at least to me. I recognized the photos as those of a



Fig. 4.— Patient operated upon in 1924.



Fig. 5.—Same patient re-operated upon in 1946 (Brunner).

patient upon whom I had operated in 1924, and I am grateful to him for permission to reproduce them (Figs. 4 and 5). Brunner reported the histologic status of the cartilage which I had implanted and which, for good reasons, he found it expedient to remove. He found the columellar portion in a good state of preservation, whereas the dorsal portion was broken up and partly absorbed. It may be that his explanation of the latter circumstance is correct, because it is likely that with the passing of years the nose may have been the site of some trauma. However, it has been my experience that dorsal implants when properly affixed to the underlying abraded bone survive much longer than do the columellar.

In addition to its use in the nasal dorsum and columella, I have found that small bits of cartilage can be successfully employed to fill minor depressions which seem to annoy many patients (Fig. 5). In these cases it is best to use thin slices taken from a cross-section of the whole cartilage. Even though such thin segments are likely to be absorbed, experience has shown that the fibrous tissue which replaces them is adequate for the maintenance of the desired contour. I have used implants of this nature to restore the contour of the alae and columella where irregularities have resulted from trauma or previous surgery. I have also found a small segment of cartilage useful in filling out a flat nasal tip as described by Sheehan.⁵

In conclusion, I wish again to emphasize the points which were elaborated in the paper and which will make for a higher percentage of successful takes. These are:

1. Employment of young adult cartilage only.
2. Proper cutting and shaping of the implant.
3. Scrupulous care in the preparation of the bed which is to receive it.
4. Under no circumstances to introduce the implant under tension or in areas where excessive scarring is likely to cause pressure, thus favoring absorption or distortion of the graft.

25 E. WASHINGTON STREET.



Fig. 6.—Irregularities in alar margin from previous surgery filled in with thin discs of cartilage.



Fig. 7.—Distortion due to infection: corrected by single dorsal implant of cartilage.

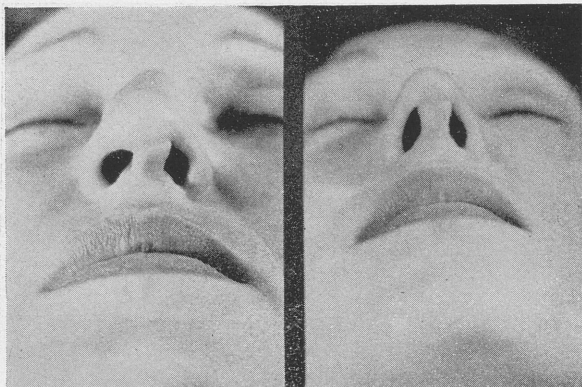


Fig. 8.—Distortion of columella and nares due to trauma: correction by repositioning of lower end of septum. Cartilage implant entire length of columella introduced through the horizontal scar which was opened and resutured.

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DISCUSSION

JAMES M. ROBB, M.D.: Samuel Salinger always presents a clear paper and all I have to say is this, that this paper should be read to the residents in the hospital once a month.

AUGUST L. BECK, M.D.: I don't understand why the external incision or dorsal incision that Dr. Salinger has used is not used more. If you have a clean case, such as he has presented here, you put in a subcuticular suture very carefully with the finest kind of a needle, and you have very little scarring. In most of those cases it is so slight you could not notice it readily; even though you are not presbyopic you could not notice it two or three feet away. I also found it useful in one case which I will tell you about that was infected. One of my medical friends at home wanted the hump taken off his nose and I put him off for several years. I did not want to tackle it at all. I saw the difficulties that might arise when you work on a doctor. He pestered me so much that I finally said to him, "I will tell you, if you come up to Boston with me, I will put you in touch with Dr. Kasanjian and there you will get the best possible job you can get in this part of the country."

This was some years ago. I must have had a premonition but as happens many times with doctors, this thing got infected. Dr. Kasanjian did the stereotyped rhinoplasty but as happens many times this thing got infected and he did not stay up there very long and came home and was under my care. He developed an abscess over the dorsum of his nose and I had to incise it. There was no choice. I made a midline incision in the dorsum of the nose, put in a little drain and you would be surprised, after a few months' time, how difficult it was to see that fine scar, even in that infected case. So I have also done the columella externally. I like the external approach better than via the nostril. I don't think there is a dirtier place in the body than the nostrils, except perhaps at the other end of things.

VACUUM FRONTAL SINUSITIS—ABSORPTION OF AIR FROM THE FRONTAL SINUS

AN EXPERIMENTAL STUDY ON DOGS

JOHN J. BALLENGER, M.D.

AND

HOWARD C. BALLENGER, M.D.

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The medical literature contains scant information as to what happens to gases trapped in the middle ear by closure of the eustachian tube or in a paranasal sinus by closure of the sinus duct. Sluder and many others have written about a "vacuum headache" which presumably is due to a relative vacuum in the diseased paranasal sinus but the literature contains no sound observations on the possibility of such a condition. On theoretical grounds it would seem that air could be absorbed via the blood stream as in the case of atelectasis and other conditions. Hilding has proposed a theory that ciliary action in the sinus ducts or eustachian tubes could remove quantities of mucus and encased air.

The frontal sinuses of dogs were chosen for the experiments because of their relative large size; an important factor when ease and accuracy of gas measurements in a closed cavity are essential. What was needed was the following: 1. An air-tight closure of the duct leading from the sinus to the nose and 2. Another air-tight connection between the closed sinus and a manometer which could measure changes in volume and pressure of the gases trapped within the sinus.

The precise procedure followed is given below but a prior word concerning the most difficult problem, technically, encountered might

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be given. The problem of securing an air tight obstruction of the nasofrontal duct was difficult to solve. Obstruction of the duct from the sinus end required a sufficiently solid plug to preclude any movement of mucous or other secretions by ciliary action as well as being air tight. None of the various substances tried such as petrolatum-saturated gauze packs, dry gauze packs, liquid paraffin dropped into the duct orifice, or a shaped soft rubber plug inserted into the sinus end of the duct to form a tight seal could be done successfully. Either bleeding occurred when the plug was forced into the duct or the plug was not sufficiently tight to prevent the passage of air. Blood in the sinus was undesirable because possible diffusion of the gas in the sinus directly into the capillaries would be altered and the blood itself would cause volume changes.

It was finally found most feasible to drill a small trephine hole through the nasal bones over the naso-frontal duct just inferior to the floor of the sinus and to obstruct the duct by external pressure by packing through that portal. It would have been ideal if the duct could have been obstructed by dissecting it out and then ligating but this proved technically impossible. A satisfactory arrangement, detailed below, was found by snugly inserting dry gauze packing through the trephine hole directly on top of and around the naso-frontal duct until it was completely obstructed.

The experiments were made on 15 dogs in which 23 different frontal sinuses were used. The average weight was 22.3 pounds and the average volumes of the frontal sinuses were for the right and left respectively, 3.1 and 2.6 cc. Each animal, before being used, was carefully inspected for evidence of disease. Particular attention was paid to the respiratory tract when searching for evidence of disease. Anesthesia was secured by the intravenous injection of 6.5% nembutal until a sufficient depth of anesthesia had been reached. From time to time additional amounts of intravenous nembutal were needed to maintain the anesthesia. The animals were observed from 60 to 240 minutes before terminating the experiments.

In preparing the animal after anesthesia had been obtained a trephine hole was made on the animal's nose over the nasofrontal duct. The hole was approximately 1 cm. in diameter and usually was located approximately $\frac{1}{2}$ cm below the floor of the frontal sinus. In removing the bone the mucous membrane was not excised as occurs when a trephine opening is made into the frontal sinus (de-

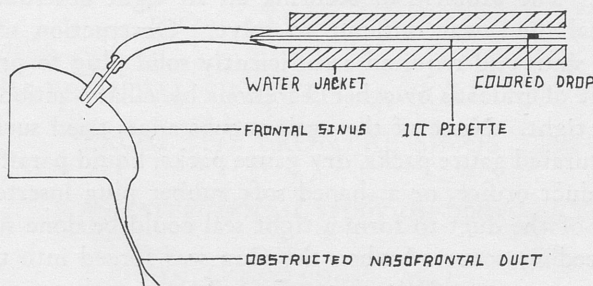


Fig. 1.—The apparatus used to measure volume changes.

scribed below). Rather the mucous membrane was elevated from the under surface of the nasal bone and the ascending process of the maxilla over an area corresponding to that occupied by the fronto-nasal duct. Obstruction of the duct was done by packing $\frac{1}{2}$ inch gauze snugly around and on top of the naso-frontal duct.

A second trephine opening of approximately 1 cm in diameter was then made through the anterior bony and mucosal wall of the frontal sinus, picking the highest dome of the sinus as the site for the hole. As soon as entry to the sinus was gained a careful inspection for blood was made and if more than a little blood-tinged fluid was seen the experiment was terminated. If the sinus was clean of blood a rubber stopper carrying a No. 16 blunt needle in its center was immediately fitted snugly into the hole. The needle was then connected by means of rubber and glass tubes to a 1 cc pipette graduated in $\frac{1}{100}$ s. This pipette was held horizontally on a level with the sinus and contained a colored drop of alcohol as an indicator. This volumetric scale was insulated against changes in volume produced by variations in room temperature by enclosing the horizontal 1 cc pipette (except for the tip which received the rubber connection) in a water jacket.

A closed system of the frontal sinus, connecting tubes, and horizontal 1 cc pipette was thus created and any change in volume of the gases within the sinus was reflected in the motion of the colored drop.

After conclusion of the measurements the efficiency of the naso-frontal duct-block was tested by substituting a water manometer for

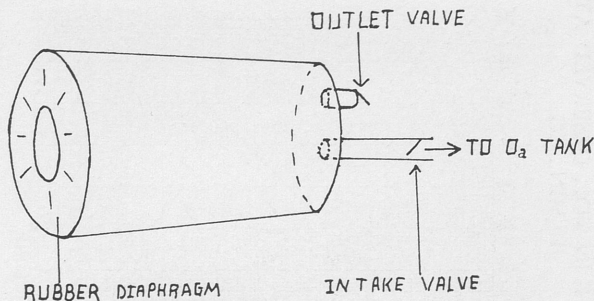


Fig. 2.—Diagram of the snout mask used during experiments when oxygen was administered.

the 1 cc pipette. Only those cases in which the block withstood the weight of 20-50 cm of water were included. The frontal sinus of the animal was finally opened and examined for blood, mucus or other contents. If time and the animal's condition permitted first one and then the other frontal sinus were tested.

There were three series of experiments, the first using the set-up described above, was designed to measure changes in volume. The second series of experiments was designed to measure changes in pressure. The apparatus necessary in this case was the same as that described above with the addition of a second manometer-tube, held vertically, interposed between the dog and the horizontally held 1 cc pipette. Then either of two things was done. Either (a) the volume was followed for a short period and when it began to fall indicating beginning reduction in the volume of gas the pressure tube was opened and the volume tube blocked off or (b) the pressure was followed throughout. The tip of the vertical pressure tube was immersed in water and measurements of the height in mm of the water column were made.

The third series of experiments was done using either the volume-measurement or the pressure-measurement apparatus. After following the animal for 15 to 45 minutes commercially pure oxygen was administered to the animal in an effort to reduce the pN_2 of the blood and thereby theoretically enhance the speed of absorption of the air trapped in the frontal sinus. The gas was administered by a snout mask containing an intake valve for the oxygen and an out-

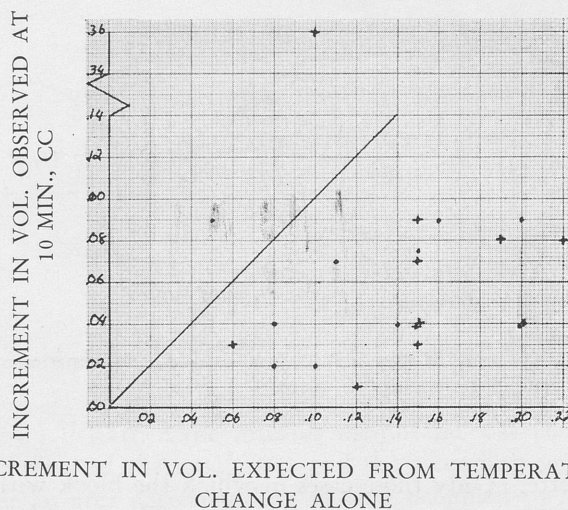


Fig. 3.—The + points indicate an experiment in which the volume continued to rise after the first 10 minutes, the black dots an experiment in which the volume fell after the first 10 minutes.

let valve for the expired air. On inspiration the former valve would open, permitting oxygen to enter the mask, and on expiration the latter valve would open, permitting expired air to be forced out. Thus the re-breathing of carbon dioxide was held at a minimum.

Post-mortem examinations were done on all the sinuses.

RESULTS

1. *Volume changes in closed sinuses.*

Of the 23 sinuses studied the *volume increased in every case for the first 10-20 minutes*. To what extent this increase in volume of the trapped gases can be attributed to changes in temperature was investigated by calculations based on Charles' law which states that the volume of a gas varies directly with the absolute temperature, the pressure remaining constant. The air entered the sinus at room temperature but after closure of the trephine openings, thus trapping the air in the frontal sinus, the temperature of the trapped air rose to near that of the body temperature and, according to the law

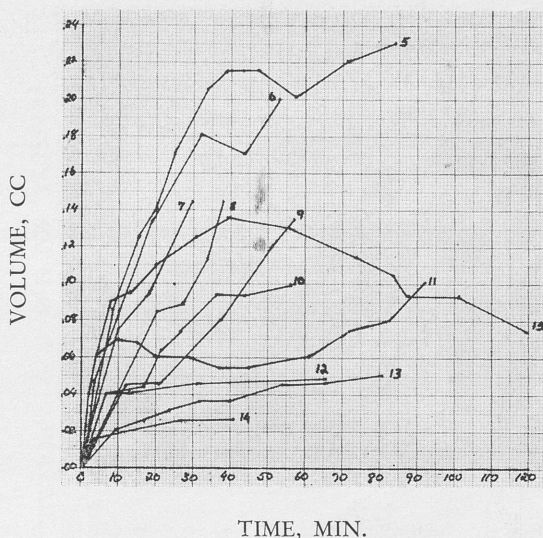


Fig. 4.—Experiments in the gas volume continued to rise after the first 10 minutes.

of Charles the volume must have increased. To calculate how much the volume changed the following formula was used:

$$V_1 = \frac{VT_1}{T} \times \frac{B - p_{H_2O}}{B - p_1H_2O}$$

where V_1 the new volume of the trapped gases

V the original volume of the trapped gases

T_1 the absolute temperature of the trapped gases after they have increased to near body temperature

T the absolute room temperature

B the barometric pressure, assumed to be 740 mm mercury.

p_1H_2O the partial pressure of water vapor at intrasinus temperature

p_{H_2O} the partial pressure of the water vapor at room temperature

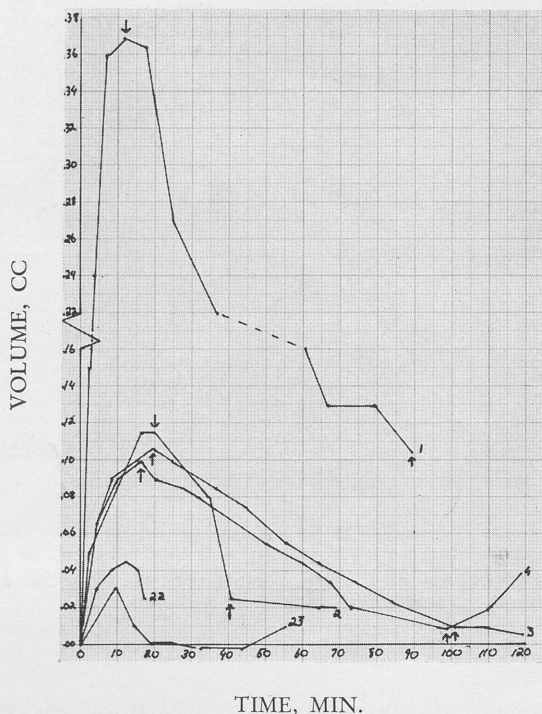


Fig. 5.—Six experiments in which a fall in volume was observed. The points between the arrows were used in calculating the mean rate of fall. (Also Exp. 21)

The intrasinus temperature was measured simultaneously with the rectal in four dogs and, after a period of approximately 20 minutes, was found to average 4°C lower. In the above calculations T_1 is taken as the rectal temperature minus 4°C . The value for the initial intrasinus temperature, T , is taken as room temperature. This is not strictly accurate, inasmuch as the initial intrasinus temperature probably lies somewhat above room temperature.

The observed increment in volume of the trapped gases at 10 minutes is plotted against the calculated increment due to the increase in the temperature of the gases. If there were a perfect equality between the observed increase in volume of the gases at the end of 10 minutes and the theoretical increase due to increased temperature of the gases the plots would fall on the solid line. The fact

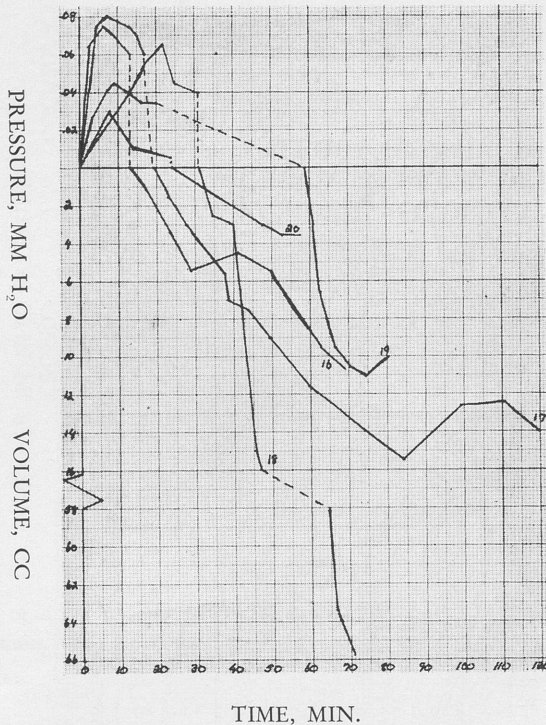


Fig. 6.—The five experiments in which a fall in pressure was observed.

that all but two of the 19 points are below the solid line indicates that although the temperature change must have contributed to this initial increase in volume other factors operating in the reverse direction may also have been involved. The error made in the calculations in assuming the initial intrasinus temperature to be identical to room temperature, as pointed out above, may be enough in itself to cause the points to fall below the solid line. If the initial intrasinus temperature were taken as midway between the room temperature and the final intrasinus temperature then the points do lie close to the solid line.

Following the initial rise in volume of the trapped gases, in 11 of the 23 sinuses experimented upon, the volume continued to rise for the duration of the experiment and in the remaining 12, the volume decreased. The courses of the volume changes are indicated

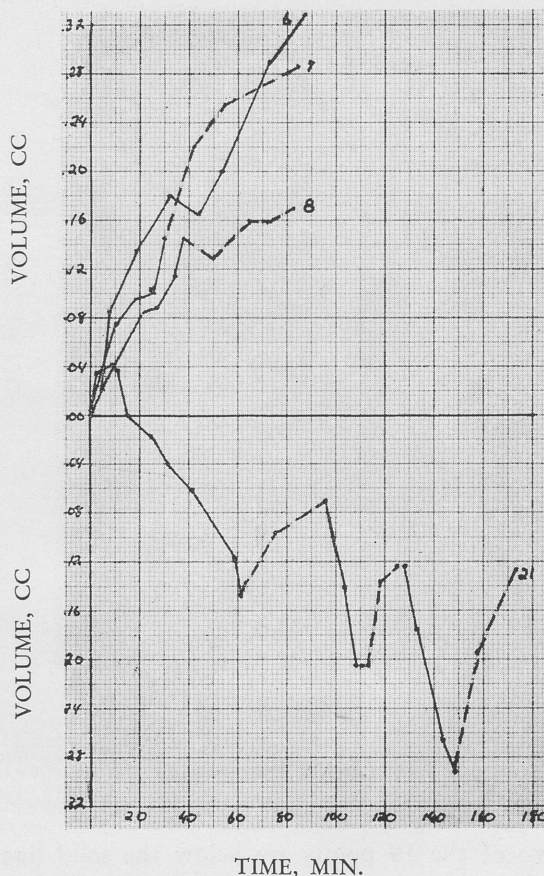


Fig. 7.—The effect of O₂ (dash) inhalation on the volume of the gases in the frontal sinus.

separately for those which continued to rise and those which reversed and began to fall.

In calculating the average rate of rise in volume the plots from 10 minutes to the termination of the experiment as given in experiment numbers 5, 6, 7, 8, 9, 10, 11, 12, 13, and 14 were used. The average rise in volume was found to be .001841 cc/min.

To obtain the average rate of decrease in volume plots between the two arrows in experiments numbers 1, 2, 3, and 4, and 21 were

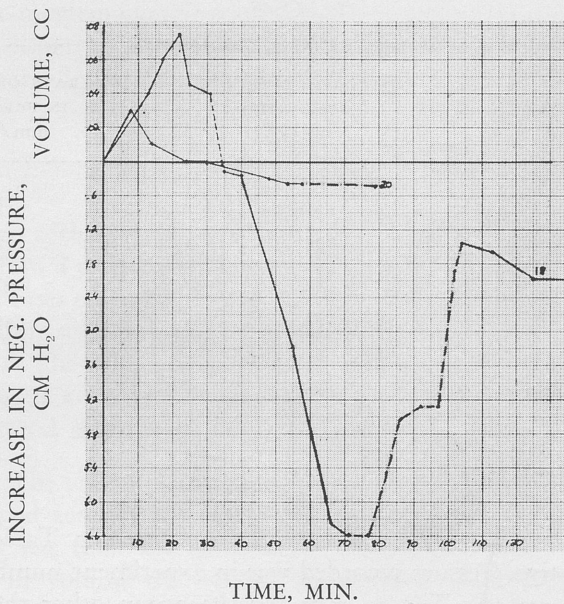


Fig. 8.—The effect of O₂ inhalation (dash) on pressure within the frontal sinus.

used, the average rate of decrease in volume was found to be .00252 cc/min.

Only one experiment, number 21, showed a reduction below the original volume. However, if the expansion of the trapped gases due to temperature changes is excluded 12 of the 21 showed a reduction below the original volume. In making this calculation the increase in volume due to the temperature change was subtracted from the observed volume at the time the volume measurements were terminated. Those experiments showing a fall below the original volume were numbers 3, 4, 11, 15, 16, 17, 18, 19, 20, 21, 22, and 23. Those not showing this fall below the original volume were numbers 1, 5, 6, 7, 8, and 10. Experiments 12, 13, and 14 showed essentially no volume change after the first ten minutes.

2. Pressure changes in closed sinuses.

In the five sinuses experimented upon in this group the volume had begun to fall, before the volumetric apparatus was closed and the

TABLE E.

EXPERIMENT NUMBER	NEGATIVE PRESSURE DEVELOPED mmH ₂ O	RATE OF FALL IN PRESSURE mm/min
16	10.5	.184
17	15.5	.250
18	66.	1.65
19	11.	.687
20	.35	.010
mean	20.67	.556

manometer opened. The detailed data are given in Table E. The greatest negative pressure recorded was in experiment number 18 and was 66. mm water. The average for the group other than experiment 18 was 9.34 mm water.

3. *The effects of oxygen inhalation on volume and pressure changes.*

In experiment number 6, 7, and 8 the volume of the gases trapped in the frontal sinus was increasing when the inhalation of oxygen was instituted. In none of these three experiments was the increase in volume of the trapped gases reversed by the inhalation of oxygen. This was contrary to expectations, since by inhaling oxygen the pN_2 is reduced and absorption of the trapped nitrogen should be thereby enhanced. In experiment 21 the volume of the gases had fallen below the original volume when oxygen-inhalation was started. Instead of the expected increase in the speed of absorption of the trapped gases, the volume change immediately reversed itself and began to increase. On the replacement of oxygen with air the volume again began to fall, only to increase again after the reinstatement of oxygen-inhalation.

In experiments 18 and 20 oxygen-inhalation was inaugurated after a fall in pressure was being recorded on the manometer. Rather than the expected further decrease in pressure, the pressure increased after the onset of oxygen-inhalation by the animal.

Post-mortem examinations carried out on each sinus experiment-ed upon displayed a variable accumulation of small amounts of blood and mucus. In no case was a gross edema of the mucous membrane seen.

DISCUSSION

From a theoretical standpoint air, or other gases, should be absorbed from a paranasal sinus where they have been trapped the same as in the cases of trapped gases in atelectasis, pneumothorax, pneumo-peritoneum, subcutaneous emphysema and gaseous distension of the intestine. The evidence obtained in these experiments which indicates that there is a partial absorption by the blood of gases trapped in a dog's frontal sinus is as follows:

a. The values recorded for the observed increment in volume of the trapped gases at the end of 10 minutes, with two exceptions, are regularly less than the theoretically predicted values on the bases of increase in volume due to the temperature change. This indicates that some other factor, possibly absorption, was operating to decrease the volume of the trapped gases.

b. In 15 of the 21 sinuses reported in these experiments (No. 2 and No. 9 excluded because of incomplete data) the volume at the termination of the volumetric recordings was below the original observed volume. In five of these experiments the pressure was measured and found to be negative.

c. These observations suggest that gases were absorbed from the closed sinus cavities. The rate of absorption in five successful experiments (Numbers 1, 2, 3, 4, and 21) averaged 0.00252 cc/min or about 0.09% of the total volume per minute which is a rate considerably in excess of the ability of the lung to absorb gases as pointed out below.

There are several factors which indicate that absorption of gases trapped in a dog's frontal sinus should be a much slower process than occurs for example in the lung alveoli.

a. In the dog's frontal sinus the surface area available for absorption of the trapped gases is small compared to the lung alveoli. To calculate the surface area of the sinus it is assumed the shape of a sphere which would under-estimate the surface. Then, knowing

the average volume of the 23 sinuses examined to be 2.8 cc, the surface area of the average sinus is obtained as follows:

$$\begin{aligned}
 \text{Volume of a sphere} &= 4.189r^3 \\
 r^3 &= 2.8/4.189 = .666 \\
 r &= .873 \\
 \text{Surface of Sphere} &= 12.57r^2 \\
 &= 12.57 \times .873^2 = 9.58 \text{ cm}^2 \\
 \text{Surface of sinus} &= 9.58 \text{ cm}^2
 \end{aligned}$$

Comparing this with the surface of the lung alveoli, 70 square meters (73), reveals how much more efficient the lung should be in absorbing gases.

b. The sinus mucosa is approximately 1 mm thick in contrast to the alveolar walls which are only one cell layer in thickness.

c. The sinus mucosa is relatively avascular compared to the alveolar walls. The result of b. and c. is that the average distance the trapped gases would have to diffuse is relatively greater than is the case of the gases contained in the lung alveoli. A comparison can be made here between the surface area of the lung alveoli related to the thickness of the alveolar walls (assumed to be .01 mm) and the corresponding figure for the sinus.

Alveoli:

$$\text{Area (A)}/\text{Thickness (t)} = \frac{A}{t} = \frac{7 \times 10^5}{.01} = 7 \times 10^7$$

Sinus:

$$\text{Area}/\text{Thickness} = 9.58/1. = 9.58$$

Thus the lung alveoli should be, on this basis, $7 \times 10^7/9.58$ $.729 \times 10^7$ times as effective in the absorption of gas as the sinus.

In experiments Numbers 1, 2, 3, 4, and 21 the observed average value for the absorption of gas was 0.00252 cc/min. This figure multiplied by the value representing the greater efficiency of the lung as compared to the sinus gives a product of 18370.8 cc/min which is far greater than the known capacity of the lung for absorption.

Thus though several factors have been pointed out which indicated that the sinus absorb gases slowly compared to the lung the observed values indicate the opposite.

There are a number of uncontrollable factors which may account for the inconsistency of the results.

a. The changing permeability of the sinus mucosa to the absorption of gases may be due to a variable covering surface layer of fluids, blood, mucus, etc.

b. Variable vasodilation and vasoconstriction may cause changes in the thickness of the sinus mucosa and thus decrease and increase respectively the volume of the sinus.

c. The presence or absence of secretions, hemorrhage, transudates in the sinus lumen, may alter the sinus volume.

The most surprising part of the work was relative to the respiration of commercially pure oxygen by the animal in an effort to influence the speed of absorption of the trapped gases from the frontal sinus. Theoretically the respiration of oxygen should have rapidly lowered the pN_2 of the arterial blood and thereby greatly increased the absorption of the nitrogen fraction (approximately 79%) of the trapped air. Instead of the expected result respiration of commercially pure oxygen lead to an opposite one. In those cases where the volume of gas was decreasing the inhalation of oxygen by the dog immediately lead to an increase in the gas volume. Similar unexpected results were obtained when a manometer was used instead of the volumetric apparatus. In this case instead of the theoretically expected accelerated fall in pressure there was a decrease in the negative pressure after the institution of oxygen respiration.

Possible explanations which relate to these unexpected results after respiration with oxygen include the following:

a. The possible diffusion of oxygen into the sinus lumen. If the inhalation of commercially pure oxygen by the animal increased the pO_2 of the blood perfusing the sinus mucosa above that of the oxygen in the trapped air there would be a diffusion of gas from the blood to the trapped air, thereby increasing the trapped gas volume. Against this possibility is the fact that tissue oxygen tensions are little affected by oxygen inhalation in the absence of arterial anoxemia, because the extra dissolved oxygen amounts to only a fraction of the

oxygen consumed by the tissues, so that venous oxygen tensions remain essentially unchanged. It is unlikely, although possible in principle, that the utilization of oxygen by the sinus mucosa is so small that it is bathed essentially by arterial blood: if this were true, oxygen inhalation might raise the mucosa pO_2 and permit diffusion of oxygen into the sinus cavity.

b. Possible changes might occur in the absorbing qualities of the surface of the mucous membrane. For example a sudden exudation of blood or serum concomitant with the inhalation of the oxygen would decrease the speed of absorption of the trapped gases as well as encroach on the sinus lumen. To explain the reversible effect of oxygen inhalation, a sudden absorption of this blood or serum would have to be visualized and this does not seem reasonable.

c. Possible changes might occur in the caliber of the mucosal blood vessels. If oxygen inhalation were in some way able to produce a reversible vasodilation in the mucosal blood vessels, with a resulting change in the mucosal thickness, the observed results could be explained. Perhaps this is the most likely explanation of the phenomenon.

The question arises as to whether the absorption of trapped gas in a paranasal sinus can explain the so-called "vacuum headache." In the five experiments in which pressure was measured an average -20.67 mm water was obtained and this does not seem of sufficient degree to produce a sinus pain of that nature.

SUMMARY AND CONCLUSIONS

1. In 15 normal dogs under nembutal anesthesia a trephine opening was made through the anterior wall of the frontal sinus and the normal ostium occluded. Changes in pressure or volume of the gases enclosed within the sinus were then recorded from the trephine opening.

2. Of the 23 frontal sinuses in which volumetric measurements were made, all showed an initial 10 minute increase in volume of the trapped gases, but this increase was of less magnitude than expected on the basis of the temperature change alone.

3. After this initial 10 minute period the gas volume decreased in 12 of the 23 and increased in 11. The average rate of volume decrease was .002520 cc/min; the average rate of volume increase was .001841 cc/min.

4. Substitution of a manometer for the volumetric apparatus in five cases where the volume was falling indicated the development of a negative pressure. The greatest negative pressure recorded was 66. mm water and the average for the group was 20.67.

5. These results indicated that if absorption of trapped gases occurred in the sinus, the rate was very slow and only very small negative pressures developed. However, even this slow rate was greater than expected on the basis of a comparison with the lung. The inconsistent behavior indicates that other processes, such as vasomotor changes, secretion, bleeding, edema probably complicated the results.

6. The inhalation of 100% oxygen instead of accentuating the absorption of gases as expected, produced a readily reversible effect interpretable as either an increase in volume of the trapped gas or as a decrease (vasomotor) in volume of the sinus cavity.

DISCUSSION

ARTHUR W. PROETZ, M.D.: It is a great satisfaction to hear reports of such a carefully conducted series of experiments on this old question of sinus pressures.

Without having read the paper carefully I will not attempt to go into detail. It occurs to me that the one factor which Dr. Ballenger mentioned, namely, changes in vasomotor pressure in the sinuses, has a good deal to do with the results. This was not overlooked by him.

Just before this experiment was conducted, there was surgical interference with the duct and a trephining of the opening. This would set off a series of reactions which might affect the blood vessels. Probably the swelling of the blood vessel has more to do with the vacuum than anything else. These pressures were, as I understand, from 2 to 66 mm of water, which is at its maximum six times the pressure of normal breathing. About 10 mm constitute the normal cycle of breathing and in an inflammatory condition these vascular changes are much greater than they are under experimental circumstances.

I have talked about absorption with physiologists and they say that tests must be made on individual tissues because the absorptive qualities of various tissues for various gases differ through the body. The swim bladder of a fish, for example, contains almost pure oxygen. That is a particularized function of that membrane. Nothing of that sort has ever been shown in the nose and the chances are that the absorption of gases is constantly complicated by variations in the blood system. We have shown some time ago that the mucosa lining the sinus can swell to several times its normal thickness in a matter of minutes or hours changing the air volume in the sinus to a considerable extent without the patient's being conscious of it. Whether that is enough to produce headache under some circumstances I don't know but I should think so.

HOWARD C. BALLENGER, M.D. (closing): I have no further remarks other than in our opinion the varying reactions were probably vasomotor. Occasionally a hissing sound as of air entering the frontal sinus may be heard when inserting a small sized silver cannula into the frontal nasal duct, into the frontal sinus. It would be strange indeed, I think, to find the frontal sinus the only cavity in the body in which absorption of gas or air does not occur.

SURGICAL TREATMENT OF CHRONIC FRONTAL SINUSITIS

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PORTLAND, ORE.

It is my purpose in this paper to bring before you a method first described by E. C. Sewall¹ in 1935 at the Western Section of the American Laryngological, Rhinological and Otolological Society meeting and subsequently published in the Archives of Otolaryngology in June, 1935, for the cure of chronic frontal sinusitis. I believe all are agreed that the handling of the nasofrontal duct is the greatest obstacle in the cure of this disease.

Many procedures have been described, such as mucous membrane flaps, skin grafting the newly made opening, the use of various tubes to be retained in the new opening for various lengths of time, such as the Ingalls² gold tube, the use of rubber tubes left in for various lengths of time, the use of tantalum foil as described by Goodale³ and the acrylic obturator as described by Erich and New.⁴ Also mention should be made of the work of Walsh⁵ in which he described a method whereby the nasofrontal duct is left intact. However, we believe that in chronic frontal sinus disease, at least, the anterior ethmoid sinuses are diseased and, if so, they should be removed. With their removal it certainly is a difficult problem to leave the frontal duct intact.

Brown⁶ in 1941 mentions the mucosal flap of Sewall and states, "Mucosal grafts as advocated by Sewall are of distinct advantage."

Lothrop⁷ thirty-seven years ago stated, "Frontal sinusitis is caused and kept up by infection and obstruction; but if very free drainage is established and the sinus is made easily accessible to intranasal treatment all fistulas will close, all subjective symptoms will disappear, and the discharge from the sinus will cease or will be reduced to a minimum."

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Kernan⁸ states, "The most popular and successful operation is one that removes the floor only, without disturbing the anterior wall, which in a great measure eliminates the possibility of osteomyelitis of the skull. To Jansen⁹ belongs the credit of first describing in 1894 the cardinal principles and technique of such an operation. Knapp,¹⁰ in 1899, reported a successful case, but it was Lynch in 1921¹¹ who vigorously put forward the advantages of this type of operation. Sewall,¹² in 1926, published his technique which has been amplified and refined by Ferris Smith,¹³ and others."

However, it was not until 1935 that Sewall published an article in which he described in detail a method of using a mucoperiosteal flap to insure a permanent opening into the operated frontal sinus. Very little mention in literature is made of this procedure except to state that it had been tried but had been found unsuccessful. I hope to show in reporting fifty-seven successful operations done by the staff of the University of Oregon Medical School, and twenty of these were operated by myself, that in our hands it has been very successful. In fact, all have been symptom free following the operation. My oldest case was operated upon in the summer of 1935. He was seen by me a few months ago and was symptom free and had a good opening into both frontal sinuses. Some of you have seen a motion picture of this procedure which I showed at the American Academy in Chicago in 1950.

R. L. Goodale has stated, "After reviewing one hundred and ninety frontal sinus operations that the following seemed to be the cause of failure in the Lynch type of operation:

- "1. Complete removal of the floor of the frontal sinus,
2. Removal of the spine of the frontal bones,
3. Removal of enough of the ascending process of the superior maxilla to give access to the anterior ethmoid region.
4. Complete ethmoidectomy and opening of the sphenoid sinus for drainage if the latter is involved. But even where this technique had been observed, a large number of operations had been failures because of adhesions. The prevention of scar tissue at the site of the nasofrontal duct is difficult to obtain. This region is involved in the ethmoidectomy so that the nasofrontal duct is removed as an anatomical structure. The area becomes merely a wide open passage."

I would add that a thorough removal of the frontal sinus lining is also essential.

As has been said before, probably the greatest obstacle to the cure of chronic frontal sinus disease is the problem of handling the nasofrontal duct, and, in my opinion, all efforts to preserve the nasofrontal duct are the chief causes of failures in frontal sinus surgery.

We prefer local anesthesia. One hour before operation we give nembutal $1\frac{1}{2}$ grains. One-half hour before operation, we give morphine sulphate $\frac{1}{4}$ grain and atropine sulphate $1/150$ grain. Cocaine crystals and adrenalin 1-1000 are brushed throughout the nasal cavity. Then we use regional anesthesia consisting of one or two per cent novocaine into which is added ten drops of adrenalin to the ounce.

Sewall advocated the complete removal of the duct and an opening made large enough and covered by a mucoperiosteal membrane from the nose to prevent granulations from forming causing adhesions, which are the causes of failure.

True, it is an operation that must be done with the utmost care in order to preserve the flap; it is a time consuming operation because to save the mucoperiosteal membrane for a flap and to remove every vestige of the floor and the sinus lining, especially in a large or shallow sinus, taxes the ingenuity of the surgeon. It, however, can be done. Also it can be accomplished successfully on cases in which sinus surgery has been previously done.

Most writers speak of curetting the lining from the sinus but this should never be done. The lining should be separated from the bone by dull periosteal elevators. It is desirable to have remote portions of the sinus cavity obliterated by granulation tissue and this cannot take place unless the original lining is entirely removed. If one is not sure that every portion of the membrane has been removed, picric acid-acetone solution may be used. This consists of 5 per cent picric acid in 35 per cent acetone solution, as described by Ferris Smith¹³ in 1934. This toughens the membrane and makes its removal much easier. It is our belief that at least the anterior ethmoid sinuses and usually all the ethmoid sinuses are diseased where we have chronic disease of the frontal sinuses. Consequently we advise removal of at least the anterior ethmoids in order to obtain satisfactory results.

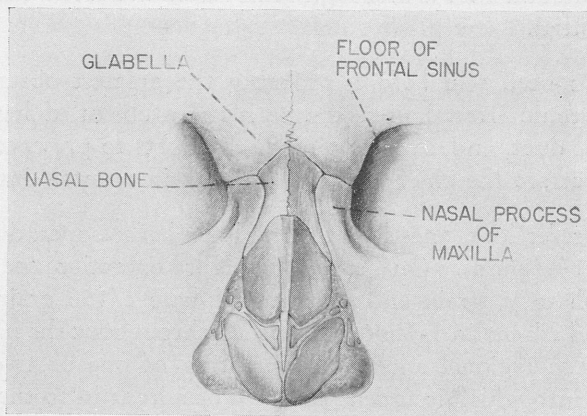


Fig. 1.—Anatomy of areas from which mucoperiosteal flap is obtained.

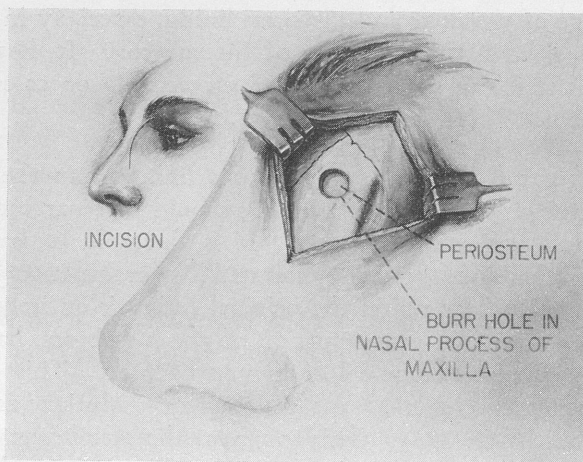


Fig. 2.—Showing incision and the burr hole uncovering the mucoperiosteal membrane.

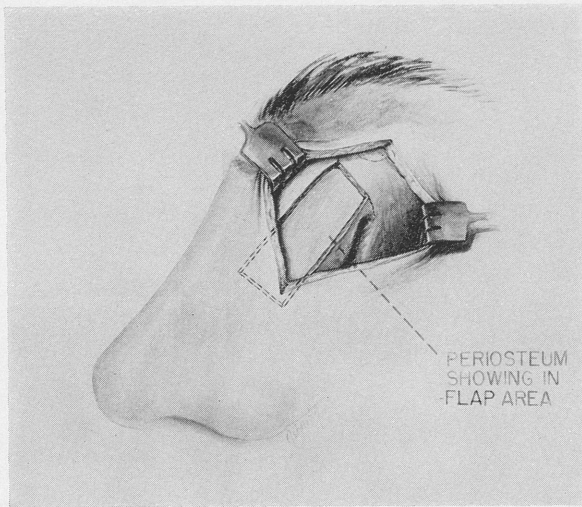


Fig. 3.—Showing the entire mucoperiosteal flap uncovered.

In performing the fronto-ethmo-sphenoid operation, the following technique should be followed. The skin incision is a curved one, following the inner angle of the orbit extending not farther than the supra-orbital notch and downward on the nasal process of the maxillary bone. Usually the incision is from 3 to 6 cm in length, which gives sufficient room because of the flexibility of the soft tissues. This usually suffices. If insufficient, an incision may be made at the outer margin, in sinuses which extend to the outer margin of the orbit.

However, in my experience I have never had to use the second incision. If, in the sinuses which extend to the outer margin of the orbit, the narrow part is thoroughly cleaned and the floor is taken entirely down, the periosteum will allow the soft tissues to push up and obliterate any remaining space. The elevation of the periosteum exposes the antero-inferior wall of the frontal sinus, the frontal process of the maxillary bone, and a portion of the nasal bone. The lacrimal sac is now raised from its bed, and the periosteum of the lamina papyracea is separated for a few millimeters. At this stage one should identify the anterior ethmoid artery and it should be tied and cut. This can be done by grasping the artery with long pointed forceps

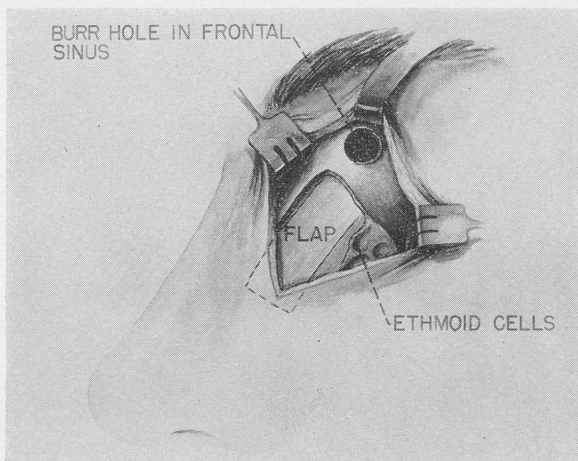


Fig. 4.—Showing the cut flap and the beginning of frontal sinus operation.

and tying, as is done in any deep cavity, or by using some tying instrument such as the ones described by Sewall or Ferris Smith. The bony window is started just below the nasal fissure which is the suture joining the anterior wall of the frontal with the superior borders of the nasal bone, nasal process of the superior maxilla and the superior process of the lacrimal bone. The bone, which uncovers the mucoperiosteal flap and which is principally beneath the nasal process of the maxilla, but usually extends a millimeter or two onto the nasal bone, may be now removed with a mastoid gouge by carefully shaving down to the periosteum, making an opening large enough to use rongeurs to enlarge the opening. However, we found in our cadaver work that it is much simpler to remove the bone to make the bony window at least at the beginning, with a large sized cutting electrically driven burr. This is much faster and there is very much less danger in tearing the flap.

After the bone is removed for a few centimeters, the membrane or the periosteum, beneath the bone, is carefully separated a few millimeters at a time, and the overlying bone removed with rongeurs and the flap uncovered. The membrane is separated by passing variously curved periosteal elevators downward as well as laterally and upward to the nasal fissure to obtain a flap large enough to cover the inside of the glabella. At this point in the operation, two vertical incisions

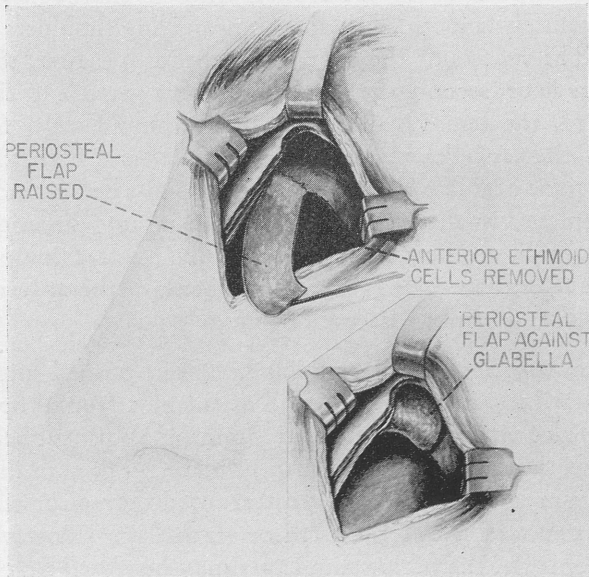


Fig. 5.—Showing the final step of the removal of the floor of the frontal sinus, the lining removed and the mucoperiosteal flap hinged at the nasal fissure and then turned into the glabella.

are made downward from the nasal fissure, wide enough apart to make a flap of the proper width. The horizontal or lower edge of the flap can now be cut by going through the nostril which is the inferior border of the flap. This sturdy piece of mucoperiosteum from 1 to 3 cm in width and from 3 to 6 cm in length is to be turned upward to line the medial wall of the new nasofrontal opening and more or less of the frontal sinus. This flap will now cover the inside of the glabella after preparation and prevent the formation of granulations and so prevent adhesions, which may cause closure of the new frontal nasal opening. At the same time it will extend into the sinus, and epithelium will grow out from it, covering the granulations that are formed within the sinus cavity. This flap is now pushed to the front of the nose and the fronto-ethmo-sphenoid operation started. The bony window is now enlarged upward, removing all of the heavy cancellous bone of the lower inner part of the glabella. If this is not removed, the cancellous bone will be an ideal place for the formation of granulations and consequently close the newly

made duct. The floor of the frontal sinus is thoroughly removed, leaving absolutely smooth edges and no projecting shoulders or spicules of bone. The success of the operation depends on: first, the removal of the entire floor; second, the removal of every particle of the mucosal lining; third, the removal of the diseased ethmoid cells; fourth, the removal of the cancellous bone of the glabella; and fifth, the making of a muco-periosteal flap of proper size. The diseased ethmoid sinuses, under complete visualization and accessible in any direction back to the sphenoid wall, are now removed carefully. After completing this part of the operation, it is fairly easy to remove the anterior wall of the sphenoid and remove its entire lining.

The mucoperiosteal flap, which has been pushed aside, is now turned upward to line the medial wall of the new frontal opening and is kept in place either by a cigarette drain or a soft rubber tube extending into the nose to hold the flap in place. The skin incision and the periosteum is closed by a few interrupted dermal or silk sutures, which are removed about the fifth or sixth day. However, if you prefer, as some do, the periosteum edges may be closed first by catgut sutures and then the skin incision closed. The tube holding the flap in place should be removed about the fourth or fifth day and the skin sutures removed at the same time. It is well to remember that gauze of any type should not come in contact with the flap or it will be apt to adhere and remove the flap along with the tube. If the flap has been well made and of sufficient size, the after care is very simple and the new frontal opening will remain open permanently. We have found that in all sinus surgery the less we do in after treatment, the better. Let the tissues recover with very little washing. Crusts may have to be removed a few times and the nose may be cleaned by gentle suction rather than by irrigations.

The original dressings used by us consists of painting the closed incision with tincture of benzoin compound, then cotton placed over it and more tincture of benzoin added.

1735 W. WHEELER ST.

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DISCUSSION

J. MACKENZIE BROWN, M.D.: Mr. Chairman, I have seen Dr. Boyden do this work. I have seen and can subscribe to his results. However, we do this operation differently. First of all, a Mosher operation on the anterior ethmoids is done. I don't believe it is always necessary to do a complete ethmoidectomy. This operation I learned from Dr. Mosher some 40 years ago. I have not altered it since. Next the frontal sinus is entered through the Lynch incision. We do not extend the incision as far down on the nose as Dr. Boyden. The opening in the frontal sinus is made of sufficient size for complete inspection. The pathological conditions in the sinus are then removed, any normal appearing membrane is left. Then the nasofrontal opening is enlarged by a small curved Kerrison forceps. Next a polyethylene tube is inserted, this tube is very easy to manipulate. The tube is made half to three-quarters of an inch in length. If it is made funnel shape at the top it will not slip into the nose. We formerly used tantalum but it is rather difficult to manipulate and Dr. H. L. Williams experience decided us to use polyethylene. I think the advantage of this procedure is its simplicity and the nose is left in a normal condition. We have not done as many as Dr. Boyden but we have done sufficient operations over the last ten or twelve years to demonstrate that this is a satisfactory operation.

RALPH A. FENTON, M.D.: I can attest to the brilliance and skill with which Dr. Boyden has maintained this type of surgery for a good many years past. He is perhaps our most radical operator, and his results speak for themselves. Most of us are not quite so radical but he follows the very excellent technique of the late Dr. Kistner and of Dr. Sewell, and insists on taking down any suspicious looking cells in the ethmoid.

Some of us agree with Dr. Brown that this is not always necessary and we try to preserve the turbinates; particularly by going up under them in the fashion advised so long ago by Dr. Mosher and followed by some of us. But I must say that the results that Dr. Boyden has brought about, particularly in intractable cases in our University of Oregon Clinics are most surprising and most gratifying, and I want to congratulate him on his procedure.

ROBERT C. McNAUGHT, M.D.: I would like to congratulate Dr. Boyden for bringing this method again before the group because I think it has real merit. Dr. Sewall and I were associated for ten years before his retirement so that I have fallen heir to the cases which he did.

Just a side remark on the reputations that people get; Dr. Sewall, apparently from conversations I have had throughout the country, was thought to be a radical surgeon. Such was not the case. He was very slow to decide that the indications were proper for this sort of a procedure, with the result that there is not a large series of cases that were subjected to this. But in reply to those who say that it fails, it does not work, etc., I can say that most of those patients are still around and most of them are symptom-free. An occasional one comes in to see me. I don't think they have drifted very far and the patients that Dr. Sewall selected for surgery were all intractable chronic cases. Many of them had had previous operations and by and large it has been a most satisfactory procedure.

Just before coming here I had occasion to redo or revise a patient on whom I had done this sort of procedure with a slight modification. This fellow was done 15 years ago and had ten years of complete absence of symptoms and then slowly developed a displacement of the orbit on one side from mucocele. Apparently the naso-frontal opening had closed over. It did not bother him too much so he did not seek advice until four years later when he came in with a very obvious mucocele. It was interesting in reopening it to find a very beautifully epithelized cavity. This particular one as I mentioned before was done with a modified technique.

The pedicle flap is not a free graft as somebody seems to think. Dr. Boyden showed it is a pedicle flap rather than a free graft. In Dr. Sewall's method with the flap being detached from the lateral wall of the nose, it must be swung medially and cover the denuded interfrontal partition. The modification in the case I mentioned was to leave it attached on the lateral wall, detach it from the nasal septum and swing it laterally out over the orbit to permit removal of the interfrontal partition. This was a case of bilateral disease and I wanted to take advantage of Lothrop's procedure of throwing both frontal sinuses into one cavity and taking away a little bit of the upper part of the nasal septum. We have done several that way and most of them have worked. This particular patient dropped from sight and the opening closed over.

Just one other remark, and that is that Dr. Sewall made a very strong point of leaving the middle turbinates. Someone suggested, and the inference was that in this procedure the nose was completely gutted inside, which is not the case at all. Excellent exposure of the supra-orbital extensions of the ethmoidal cells is had by this external approach. The entire floor of all the ethmoidal cells and the orbital wall are removed. The cells are eliminated but the middle turbinate is completely unattacked.

GUY L. BOYDEN, M.D. (closing): We all know Dr. Brown. We all love him. I cannot see any sense though in using tubes when you have perfectly good membranes in the nose to use instead of tubes.

Then in not removing the lining, many years ago when I took my examination for the National Board, Dr. Mosher examined me. One of the things he asked me was how I liked the operation that was done in the previous morning, and he happened to know that I was standing beside him, knowing I was there. I hesitated, being a young fellow. "No, this is just between us. It won't go any further. What do you think?"

I said, "I did not like it."

He said, "Why?"

"Because he did not remove the lining after going into the frontal sinus. He did not remove it completely."

Dr. Mosher said, "That is absolutely right. I did not like it either." So I think he will bear me out in that.

RECURRENT LARYNGEAL NERVE PARALYSIS

A REVISED CONCEPTION BASED ON THE DISSECTION OF

ONE HUNDRED CADAVERS

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The purpose of this paper is to present a revised conception of the causes and mechanism of recurrent laryngeal nerve paralysis. The explanation is based on the fact that, at times, these nerves divide into two or more branches prior to entering the larynx. This division may occur at any point in the recurrent course from the clavicle to the cricothyroid articulation. The statement that the recurrent laryngeal nerve has been found to divide in the extra laryngeal areas could not be news to those familiar with the literature on this subject. This is a report on the dissection of 100 cadavers. It confirms the division of the recurrent laryngeal nerves in the extra laryngeal space. The information obtained from these 100 dissections not only offers a simple, practical and tenable explanation of the elementary and bizarre type of traumatic laryngeal paralysis but should assist in avoiding these paralyses.

There is experimental evidence^{25, 26, 27, 28} to show that the initial division of this nerve produces a separation of the abductor and adductor fibers. It is logical to believe that since this separation occurs in an area that is commonly subjected to surgical intervention one should anticipate that there will be occasions when one or the other of these divisions will receive damaging trauma or actual severance. The relative vulnerability of the divisions will be influenced by their respective anatomical relationships. Clinical findings demonstrate that a certain percent of patients who have undergone thyroid surgery or other major neck surgery do have laryngeal muscle paralyses as a result of nerve injury. The importance of this fact

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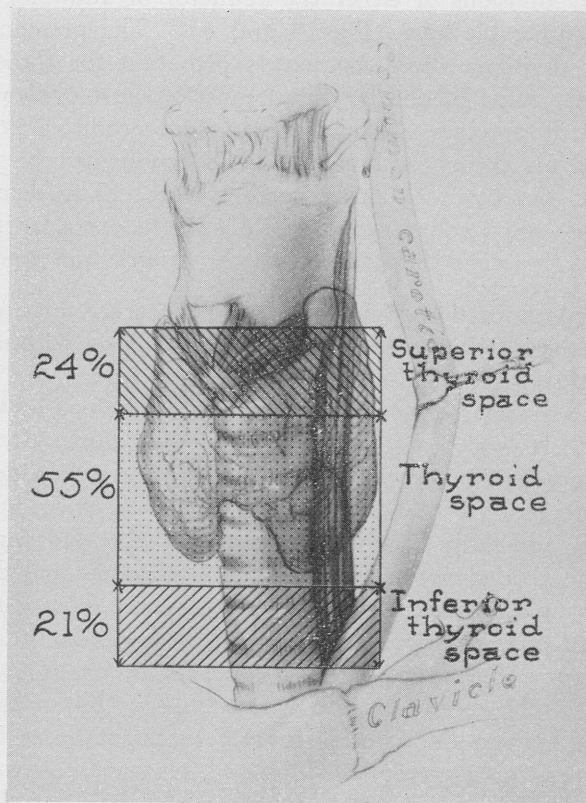


Fig. 1.—An arbitrary division of the extra laryngeal area into Superior Thyroid and Inferior Thyroid Spaces. The location of the divisions of the 86 nerves is shown in relative percent.

was not recognized nor the significance appreciated until recently.⁴⁴ The general acceptance of this knowledge will come slowly but surely. It is hoped that concurrently with its acceptance will be the recognition of one further point, namely, that there are at times secondary and tertiary divisions of either the abductor or adductor trunks in the same vulnerable area (Figs. 3 and 4). The presence of these subsequent divisions offers adequate explanation for those instances wherein only some of the laryngeal muscles show evidences of paralysis. Such previous explanations as were produced were postulates, based on theory and could not be confirmed by controlled experiment, dissection or microscopic section. The demonstration of secondary and tertiary nerve divisions in the extra laryngeal areas offers an explanation that satisfies all the criteria for proof.

It was illuminating to find that the main trunk of the recurrent laryngeal nerve divided bilaterally prior to arriving within $\frac{1}{2}$ cm of the cricothyroid articulation in 24% of the cadavers, and that either the right or the left nerve was found similarly divided in 43% of the dissections. It is also of interest to note that of the divided nerve trunks, such divisions were not confined to the right side, contrary to the statement of Meckel⁴ in 1832. This is evident in the instances of bilaterally divided nerves. In the cadavers showing unilaterally divided nerve trunks there is almost equal distribution of division on the two sides. The exact figures show a ratio of 20 on the right side as compared with 18 on the left side. A summation of these figures shows that of the 200 nerves, 86 or 43% were either unilaterally or bilaterally divided nerve trunks. If one deletes those that divide high in the superior thyroid spaces, some 21 in number, there still remain 65 examples of divisions in areas frequently invaded during neck surgery. In other words, more than one-third of the patients can be expected to have one or more divided nerve trunks in the thyroid or inferior thyroid spaces and therefore one-third of the patients carry potentials for injury of one or the other of the main trunk branches of the recurrent laryngeal nerve.

The fact that the recurrent portion of the laryngeal nerve divides in the extra laryngeal area in more than one-third of the dissections has been conclusively demonstrated on a purely anatomical basis. The application of this knowledge has a practical value.

Paralyses of the larynx have been a controversial subject for many years. The literature is bulky and richly endowed with con-

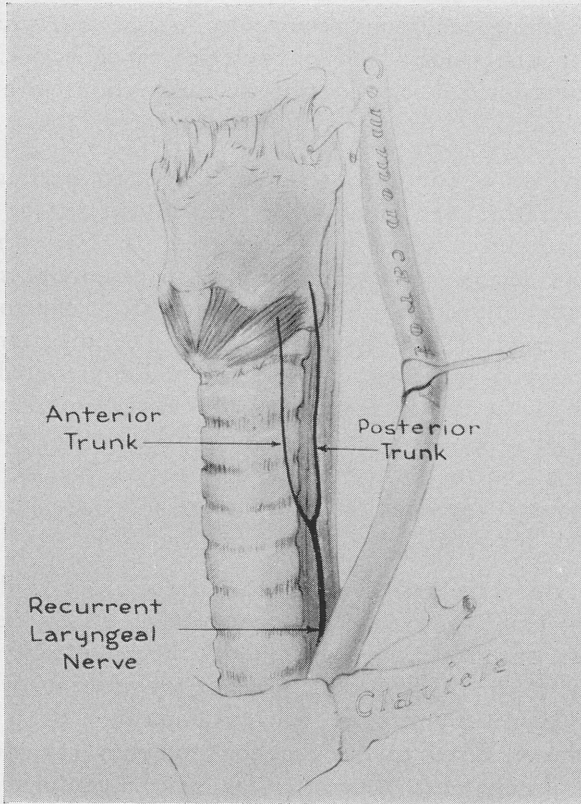


Fig. 2.—A simple divided left recurrent laryngeal nerve.

tributions that are of little value. There are many demonstrations of how active the mind can be in presenting pseudo-logical, abstract, or philosophical deductions, even though the explanations had no counterpart in any other nerve muscle relationship. These are excellent examples of attempts to construct anatomy to fit a clinical picture. This is seldom a satisfactory approach especially when it entails establishing conditions peculiar to a single area which violate fundamental embryologic and physiologic principles. Conversely, several present lucid descriptions of their dissections to explain the clinical pictures.

The division of paralyses of the larynx into central and peripheral types is rather well established and accepted on the basis that upper motor neuron lesions produce spastic paralyses whereas peripheral nerve lesions or lower neuron damage produces a flaccid paralysis with ultimate fibrosis and contracture, following varying or variable periods of atrophy and fibrosis. The importance of this sequence of events is apparent but not too well appreciated, as far as the muscles of the larynx are concerned. A description of the accepted central origin of the fibers that make up the vagus nerve would tend to confuse rather than add to the purpose of this paper. There is a general agreement among authors and texts which is satisfactory in all respects.

The vagus nerve has the most extensive course and distribution of all of the cranial nerves. It is composed of visceral sensory and motor (para sympathetic pre-ganglionic) fibers as well as somatic and sensory fibers. The laryngeal paralyses of central origin are adequately described and presented satisfactorily. That portion of the vagus nerve, distal to the ganglion nodosum (ganglion of the trunk) does interest us. Our attention will be confined to two of the three branches of distribution that originate in the neck—the superior and recurrent laryngeal nerve. The third branch or group—the superior cardiac branches (rami cardiaci superiores: cervical cardiac branches) do not contribute to or interfere with paralyses of the motor or sensory function of the larynx.

The site of origin of the superior laryngeal nerves has not been a subject for comment or discussion. This does not hold true for the recurrent laryngeal nerve. Hérard⁵ describes an anomalous origin of the right recurrent laryngeal nerve and in 1846 suggested "that we ought to be prudent in denying existence of the recurrent lary-

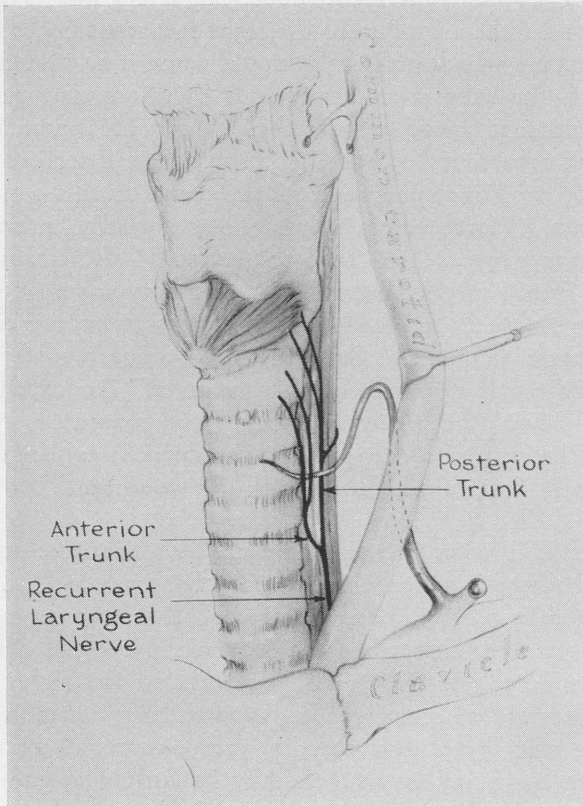


Fig. 3.—A four divisioned recurrent laryngeal nerve. In this specimen the anterior trunk of adductor fibers divided in the thyroid space anterior to the inferior thyroid artery. The posterior trunk of abductor fibers divided in the superior thyroid space posterior to the artery. A fifth branch is shown going to the esophagus.

geal nerve if we did not find it in its proper place." On rare occasions and in association with congenital developmental anomalies of the fourth pair of aortic arches wherein the right member atrophies, is misplaced or is absent, the right subclavian artery has its origin more caudad. Under these conditions the right recurrent nerve is cephalad to any structure which ordinarily would cause it to descend into the thorax with the subclavian artery and therefore does not have to assume a recurrent course to enter the larynx. This anomalous course of the right recurrent laryngeal nerve was first described by Steadman² in 1823. His explanation of the cause of this condition was not adequate. Hart³ whose views are not wholly in accord with modern embryology, stated that the secret of the circuitous route was to be found in the natural law of embryologic or embryonic development. Reid⁶ in 1847 gave the correct explanation of the origin of these anomalies. Several other authors^{2, 3, 5, 6, 8, 9, 29} have reported additional examples of this anomaly. An excellent article by Work²⁹ covers the historical background, presents a typical case and refers the reader to Piersol⁴⁸ for the detailed explanation of the developmental factors which produce this condition.

Thus far, all cases wherein there has been aberrant origin of the recurrent laryngeal nerve, the anomaly has occurred on the right side. This may have induced several of the later investigators to make the statement that when the recurrent laryngeal nerve divides in the extra laryngeal areas, it only occurs on the right side. The question naturally presents itself as to what may occur in those rare individuals who have dextrocardia and transposed viscera. This question was answered in an article by Berlin²⁴ who cites Weatherford of Harvard, who, in 1934 presented findings to show that when such a condition occurs in these rare individuals it then occurs on the left side.

The historical background of interest in the function of the recurrent laryngeal nerve extends into antiquity. An outline of abstracts made while reading the earlier authors is incorporated under the heading "Chronological Data." It is offered for review by those who have covered this field, as a working and time saving reference for those who are less familiar with the literature and primarily in the hope that it will intrigue and perhaps stimulate others to find time to peruse this background when leisure permits or the conscience demands. Few authors who have presented reviews of the literature

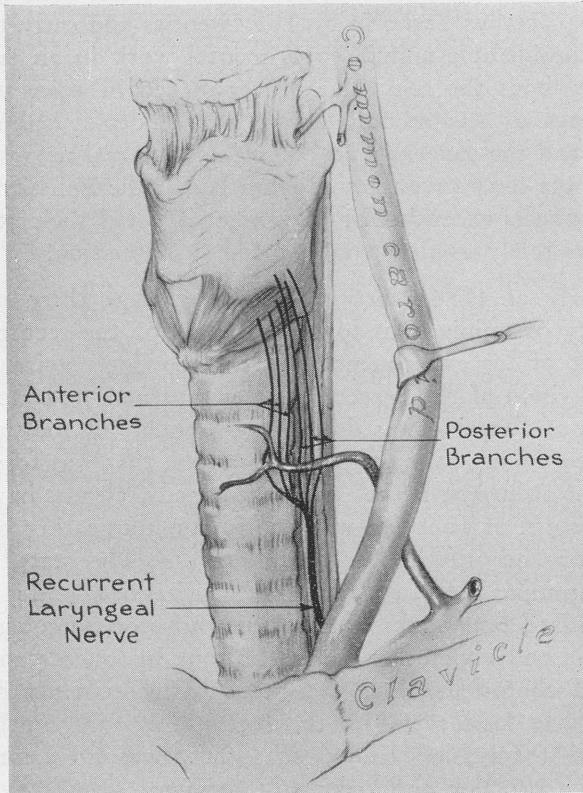


Fig. 4.—A six branched recurrent laryngeal nerve. This is an uncommon type of division in that usually the initial division into anterior (adductor) and posterior (abductor) trunks retain their integrity for longer distances before redividing. In this specimen all branching took place posterior to the inferior artery and at about the same level in the thyroid space.

failed to mention Rufus of Ephesus and Galen. The latter is accredited with being the first to record observation on aphonia and respiratory distress due to experimental paralyses produced by section or ligation of the recurrent laryngeal nerves. The next contributor of note was Vesalius⁴⁷ who in his late twenties and early thirties engaged in considerable animal experimental work in an endeavor to learn more about the role these nerves played in voice production. His drawings of the human larynx clearly depict the anatomical structures and the superior and recurrent laryngeal nerves. In general, all of the early investigators from Rufus the Ephesian to Reid¹⁸ with perhaps the exception of Magendie¹ directed their attention to these nerves solely as they were related to phonation.

As early as 1734 Martin¹⁹ mentioned that there prevailed a "contrariety of opinion" as to the function of the recurrent nerve. As a result of his experiments wherein the dog's voice was taken away by division of the nerves but later returned, he concluded that this return of function was due to the branches of the superior laryngeal nerve taking over the motor function that was formerly supplied by the recurrent nerve. He mentioned Galen as having stated that this return of function was due to communicating branches of the superior and inferior laryngeal nerves. The stated fact that such a communication or anastomosis exists has been and is still found in the standard text books of anatomy and otolaryngology, in spite of excellent and conclusive demonstrations by microscopic, macroscopic, and clinical evidence that negates the existence of such an anastomosis, at least as far as the motor fibers are concerned. As early as 1871 Luschka¹² stated that there was no communication between the superior and recurrent laryngeal nerves, even though they might share the same sheath for short distances. Recent experimental evidence and particularly the contributions of Lemere²⁵⁻²⁸ leave little to be wanted in the way of conclusive proof that an anastomosis of the superior and recurrent laryngeal nerve fibers does not exist. Heighon²⁰ in 1792 believed that the severed nerves reunited. The probability of spontaneous reunion of the severed ends of the nerves, seems somewhat improbable.

Magendie¹ in 1813 was the first to experimentally demonstrate that the cricothyroid muscle was innervated by the superior laryngeal nerve. He gave a good description of the distribution of the recurrent nerves and believed that the muscles they supplied opened the glottis, whereas the superior laryngeal nerves, in going to the

cricothyroid and transverse muscles had exactly the opposite function: that is, closed the glottis. This teaching was blindly followed by some writers for many years. In a lecture in 1836 he stated that the anatomists were unable to solve the problem of why the recurrent nerves took such a complicated course instead of coming off directly from the pneumogastric nerves.

A brief review of the literature will impress one as to the quality and caliber of the contributors. The controversies waxed hot and several of the proponents of the more or less accepted principles and theories were unrestrained in their criticism of their opponents. The criticisms were not solely confined to the type of experiment performed and the deductions but were sufficiently broad to encompass the veracity of the investigator and his qualifications to formulate or carry on acceptable experiments. There is evident attempt by some to confine their attention to pure anatomy and others to physiology and function. None were able to confine their work to a single stated phase and consequently there was much overlapping of ideas and conclusions. Should time permit and if you are interested in the fantastic products of a fertile imagination, read an essay by Hugh Ley²¹ wherein he presents his views and reasons for the upper movement of the cricothyroid cartilage during the acts of swallowing and coughing. Then read Hoover's²³ evaluation of this contribution.

One may sympathize with the student of laryngology of the early part of this century. His confusion in regard to the protean manifestations of laryngeal paralyses has been the result of the inexactness of the knowledge of the older texts such as Sajous,³⁷ Burnett,³⁵ Kyle,³⁶ and Bruck,³⁸ and feel comforted by the fact that progress of the knowledge of that period. One may smile smugly while reading some of the older texts such as Sajous,³⁷ Burnett,³⁵ Kyle,³⁶ and Bruck,³⁸ and feel comforted by the fact that progress has been made and that everyone should know that the internal branch of the superior laryngeal nerve is purely sensory and therefore would not be the nerve to supply all or even part of the motor filaments to innervate the arytenoid muscle. Or do you still believe that it does? In either event, one's patronizing attitude towards the older text is due for a severe jolt when one reads the later modern texts. Ballenger⁴¹ seventh edition, makes a positive statement that the interarytenoid muscle does receive some motor fibers from the internal branch of the superior laryngeal nerve. The com-

pletely revised 9th edition (1947) contains the same information except that it is no longer in the form of a positive statement. We now find such limitations as "possibly" and "it is thought to receive." Portmann⁴² chuses to ignore the minor details of the innervation of the larynx as such. In regard to the superior laryngeal nerve he makes the definite statement that this branch is exclusively sensory. On the same page, in discussing the recurrent laryngeal nerve, he states that one of the terminal branches of this nerve joins a lower branch of the upper laryngeal nerve to form Gallien's ansa. Later in the chapter, under the heading of "Paralysis" he states that total paralysis may be bilateral or unilateral: it may effect only a single muscle or all of the laryngeal muscles. We find under the caption "Paralysis of Isolated Muscles, in reality, this is more in the nature of a paresis, due to myopathies." Jackson and Jackson³⁹ in the 1946 edition and in those that precede it, is outstanding for obscurantism. On page 420 in regard to the internal branch of the superior laryngeal nerve we find the statement "This branch probably gives some supply to the arytenoid muscle." On page 424 we find the statement "On each side there is a superior and inferior laryngeal nerve. All four are branches of the vagi. Both pairs carry afferent and efferent impulses and all four nerves are connected by anastomotic or association fibers. In the distribution of these four nerves there is some overlapping as well as anastomoses and this is sometimes confusing in the diagnosis of paralysis." A further description of the superior laryngeal nerve: "The external (branch) chiefly motor, passes downward and is distributed to the cricothyroid muscle. A few stray neurons are traceable to an anastomosis with the inferior laryngeal nerve." On page 425 in description of the right inferior laryngeal nerve: ". . . as one nerve to the level of the upper border of the cricoid cartilage where it divides into two branches. The anterior branch is distributed to the cricothyroid, thyroarytenoid and the arytenoid muscles. The posterior branch is distributed to the right posterior cricoarytenoid muscle and to the arytenoid on both sides of the midline." In regard to the left inferior laryngeal nerve, it is stated that its position, branches and distribution are relatively the same as those of the right inferior laryngeal nerve. "In addition to the clearly defined motor and sensory innervation, the superior and inferior laryngeal nerves, there is a not so clearly understood, but definitely present, normal form of innervation known as tonus. The source of excitation impulses for this reflex tonus may be exerted through the superior laryngeal nerve

fibers or possibly, it may not be in the larynx—it may be vagal. All chains of neurons are not traceable.” Such descriptions should stimulate the duller student to a state of mild confusion and completely disorient the more brilliant ones. These specific sources of information were selected as typical of the group of accepted texts and not with the direct or ulterior motive of subjecting any of them to ridicule or undue criticism. The dissemination of an adequate explanation by King and Gregg⁴⁴ and confirmed in this paper should eliminate all opportunities for confusion.

The results of lethal injury to abductor fibers of both recurrent laryngeal nerves is well known and dramatic. Prior to the advent of thyroid surgery as an accepted procedure, there were few surgical reasons that afforded the opportunity or necessity to invade the areas of the neck through which the recurrent laryngeal nerves traverse. Associated with the popularization and vindication of the advantages of thyroidectomy in properly selected cases, came an increase in laryngeal paralyses. Involvement of the adductor fibers was and is relatively rare. The most unpleasant sequelae are aphonia or a markedly changed or impaired voice. Unilateral abductor paralysis is the most common of the postoperative complications and is seldom, if ever, annoying to either the patient or the surgeon. The quality and timbre of the voice may be unchanged and the diminution of available airway during inspiration is seldom cause for concern. The frequency of this condition is much greater than the reported figures would indicate. Goldman⁵⁰ reports on the incidence of laryngeal paresis and paralysis seen at the Thyroid Clinic of the University of California Hospital during the twelve and one half year period from July 1935 to January 1948. The results were obtained from medical appraisal and follow-up records of 1042 patients who attended the Thyroid Clinic and were operated upon for thyroid disease. From 80 to 90 percent of the operations were performed by resident staff members late in their training period, with or without operative supervision by a senior surgeon. He states “Limitation of motion or weakness of one vocal cord, indicating that the laryngeal nerve fibers were intact, was termed paresis; if, however, there was a complete abductor paralysis, the cord was said to be paralyzed. Examinations were conducted by the members of the Department of Otolaryngology.” The routine surgical procedure is anatomical thyroidectomy performed by an intracapsular excision with preservation of the lateral and posterior capsule and without

visualization of the recurrent laryngeal nerves. Generally between 3 and 4 grams of thyroid tissue is left behind.

Table No. 1 is an abstraction and a selected condensation of several tables in his report. In this manner attention is focused on the incidence of laryngeal paresis and paralysis of 5.7 percent. Unfortunately he does not mention or present data referable to the incidence of preoperative laryngeal paresis or paralysis in this group of patients.

TABLE I.

	NUMBER	PARESIS	PARALYSIS
Toxic diffuse goiter	379	3.3%	2.7%
Toxic nodular goiter	179	4.0%	3.1%
Non-toxic nodular goiter	389	2.3%	2.3%
Chronic thyroiditis	47	4.8%	3.8%
Carcinoma of thyroid	48	10.7%	8.0%
Total	1042	3.2%	2.5%

The patient with bilateral abductor paralysis presents a different picture. In the advanced cases, the difficulty with inspiration and the inspiratory howls during sleep leave no question as to the nature of their origin. In some patients these findings may be minimal and permit evading the correct diagnosis by substituting that of asthma. The amount of embarrassment to respiration extends from this minimal type to the most severe type wherein immediate tracheotomy is essential as a life saving measure.

It is evident why the surgeons doing the thyroidectomies were also avid for information in regard to the anatomic relations of the recurrent laryngeal nerves, an explanation for the varying types and extent of the paralyses, and for ways and means of avoiding these paralyses. The laryngologists were of no help. It was also evident that the mere size and consistency of the thyroid gland was not the answer. The association of excessive bleeding during surgery and the laryngeal complications was too common to be overlooked. The

next logical step was to critically review the anatomy of the area, obtain clear cut relationships between the inferior thyroid artery and the recurrent laryngeal nerve, and either devise changes in technique, which would eliminate and minimize the opportunity of injury to these nerves or present dicta which if followed would assist others to keep their percent of complications to the minimum. Woelfler¹⁴ stated that the nerve is always anterior to the artery or branch of the artery. Kocher¹⁵ who needs no introduction, in 1883 stated that the inferior thyroid artery always passes posterior to the nerve, comes forward on its inner side and then bends over it. Rotter¹⁶ reported on 15 examinations. He found that only one-third followed the pattern as presented by Kocher. Strecheisen¹⁷ presented data on the dissection of 56 bodies. His interest centered on whether or not the artery made a loop around the nerve. He made no mention as to the anterior or posterior position of the artery in relation to the nerve. Of more recent vintage are reports by Ziegelman³¹ who dissected 42 recurrent and 14 superior laryngeal nerves, and Berlin²⁴ who reports on the dissection of both nerves in 70 cadavers, and on 72 nerves during 70 thyroidectomies. Of this latter group, 68 nerves were not identified during the operative procedure and it is presumed that they were safely concealed deep in the tracheo-esophageal sulcus. These last two contributions will be discussed in some detail.

The time spent in recounting some of the findings of the surgeons and the surgeon-anatomist is deliberate and with the intent to emphasize certain points. There is no reason to question the quality and caliber of the authors. They were interested in the problem or they would not have reported their observations. One can assume that they were open minded, observant individuals who were familiar with the fundamental technique of the dissecting room. That although their major interests may have centered about some limited area, such as whether the artery looped around the nerve or was anterior or posterior to the artery. They could not obtain even this limited information without having exposed and isolated considerable portions of these structures. The existence of a divided nerve was established in 1832 by Meckel.⁴ Yet so few instances of divided trunk nerves appear in their literature that one must conclude that the divided nerves were not seen. This could be explained on the basis that the dissectors found one or the other of the branches of a divided nerve, considered it as being the entire nerve and deposited the remaining branch or branches in the waste basket—a common reposi-

tory for important anatomy. It would appear that in the earnest endeavor to prove or disprove some particular controversial point, the investigator lost interest in the surrounding and adjacent anatomy. This is understandable but unfortunate.

The article by Ziegelman³¹ reports on the dissection of 23 right recurrent laryngeal nerves, 19 left recurrent laryngeal nerves and 14 superior laryngeal nerves. The stated purpose of the dissections was to establish the relations of the nerves to the thyroid arteries, thyroid gland and the inferior cornu of the thyroid cartilage. In addition 7 larynges were dissected to demonstrate the distribution of the nerves to the muscles. The illustrations are photographs of drawings which are supposedly true representations of actual dissections. They show surprising detail in certain respects but are quite lacking in fundamentals. All the recurrent laryngeal nerves illustrated are single trunk nerves, all follow a surprisingly similar course and all go to the area of the cricothyroid articulation as single trunk nerves. The illustration of the distribution of the nerve fibers to the muscles in the 7 dissected larynges is not stated to be a composite picture, yet it is too finished a product to be a diagram and too diagrammatic to be a true picture. His conclusion, No. 8, states that the internal branch of the superior laryngeal nerve sends branches which communicate with the recurrent laryngeal nerve.

The article by Berlin²⁴ is excellent. The dissection of both nerves of the 70 cadavers was done with special attention to the relation of the nerve to the thyroid gland and the thyroid adherent zone—that area just below the anterolateral aspect of the bar of the cricoid cartilage at the level of the 1st and 2nd tracheal rings. Illustration Fig. 3d. in his article is an excellent demonstration of a divided trunk nerve. The following quotation leaves no question as to how close he was to obtaining the long sought answer. "Twice the nerve was demonstrated branching into two terminal twigs about the level of the lower pole, the anterior branch crossing through the adherent zone and ultimately joining its posterior terminal companion beneath the inferior thyrocricoid joint. One such reasonably sized anterior branch was accidentally cut at operation on the left side, after first identifying the posterior twig which was thought to be the main stem of the nerve, with resultant partial temporary paralysis of the corresponding vocal cord." Since it was the anterior branch that was cut, the paralysis should have been of the adductor type. One wishes that a detailed description of the larynx was available.

One other anatomic relationship has been cause for considerable controversy. Are there occasions when the recurrent laryngeal nerve penetrates and courses through the thyroid gland substance? The answer to that question is unqualifiedly that the nerve does not transgress the gland parenchyma. In those instances wherein the thyroid gland is markedly enlarged and the posterior surface is nodular, the nerve, lying in one of the sulci may appear to transverse the gland substance. Careful dissection and microscopic sections will show that each has retained its integrity. Embryologically it is impossible for the nerve to do otherwise.

Discussions and arguments on several points of mutual interest with Dr. Brien T. King resulted in agreement that there must be a simple, logical and rational answer to the bizarre types of laryngeal paralyses. When the answer was available it should not consist of complicated arrangements of nerve fibers which violate the fundamentals of nerve fiber distribution or necessitate establishing intricate pathways for nerve impulses that do not exist in other parts of the body. It was evident that the explanation must be found in the areas adjacent to the larynx and trachea.

Late in 1947 King was convinced that there was a simple answer, that the nerve did divide extra-laryngeally into trunks of abductor and adductor fibers and that there was experimental evidence to prove this.²⁷ The few dissections he had done following the realization of the importance of divided trunk nerves showed the presence of such divisions in the danger area. King and Gregg⁴⁴ reported their results in 1948. More dissections were necessary in order to verify these findings.

Since the information to be obtained was of equal if not more practical value to the thyroid surgeons, I called on Dr. William H. Rustad (of the Division of Surgery of the University of California School of Medicine, San Francisco, California) for collaboration. It is only because of his dogged perseverance in pursuing available cadaver material, and in his complete disregard for the sacrifices of his time and efforts that this material is available. Many more than 100 cadavers were dissected. The courses of the recurrent nerve and inferior thyroid artery were charted on specially prepared forms. In this manner, the interrelationships of these two structures and the basic anatomy of the area were preserved in graphic form. These charts were supplemented, when necessary, with explanatory notes.

They are available for study or review at any time. In retrospect I would add one further step for completeness—a photograph of each dissection.

Sufficient dissections were done to permit the selection of 100 cadavers wherein dissections of both the right and the left nerves and inferior thyroid arteries was possible. This eliminates a preponderance of right or left sides, and permits comparison of the two sides of each specimen. Material, which for one reason or another, had had the nerve or artery distribution of one side destroyed or invaded was eliminated. The accompanying drawings and tables are self-explanatory. No attempt was made to follow the branches of the nerves to their terminal distributions. The average available cadaver material does not permit this to be done as a simple gross dissection. The nerve branches soon become minute threads that are indistinguishable from strands of connective tissue. With little effort, and a minimum of wishful thinking, one can produce a complicated network of pathways and anastomoses which would confuse anyone. Grossly, this artifact network appears real, but would not stand up under magnification by either the dissecting microscope or microscopic sections. A few attempts to accurately follow these intralaryngeal branches was sufficient to demonstrate the difficulties that would be encountered. A realization of these difficulties engendered a more understanding attitude toward those who, in all good faith, produced complex nerve anastomoses and reported what they saw as factual evidence.

COMMENT

It would be incomplete to offer this material without giving credit to Brien T. King, M.D. of Seattle, Washington, for his two contributions to laryngology. The number one contribution was the presentation⁴³ of the first satisfactory reconstruction operation for rehabilitation of individuals with bilateral abductor paralysis. The second contribution⁴⁴ was the recognition that the recurrent laryngeal nerve does divide into abductor and adductor trunks in the extralaryngeal space. His findings that these divisions into two branches in the extralaryngeal space are not an anatomical rarity but occur sufficiently often to constitute an accepted deviation from the routine text book description, namely, that the recurrent laryngeal nerve is a single trunk nerve. In this published article he presented

the evidence to account for the behavior of the adductor and abductor groups of muscles. This contribution nearly solved the problems that have been the basis for years of controversy.

The initial scope of the present work was essentially to confirm or deny the statement that the recurrent laryngeal nerve was found as a divided nerve trunk in the extralaryngeal areas in a appreciable percent; that is, sufficient to be of practical importance. In other words, the possibility of the nerve dividing in this area, prior to entering the larynx, was no discovery. It had been reported as a fact by Meckel⁴ and others, but was considered as an anomaly primarily confined to the right side of the neck. That there could be any relationship between this extralaryngeal division of the nerve, and the accepted knowledge that the recurrent laryngeal nerve normally divided into an abductor and adductor branch immediately after it passed behind the cricothyroid articulation, was completely ignored by both the anatomists and the more erudite clinicians. King and Gregg⁴⁴ pointed out the significance. If the recurrent laryngeal nerve did divide in any percentage approximating the 30% reported by Dr. King, it was essential that additional dissections be done in order that statistical variations or errors be kept to a minimum. The basis for his statistics (16 cadavers or 32 nerves) was not sufficient.

The results obtained from the reported 100 cadavers did confirm the fact that the recurrent laryngeal nerve does divide in the extralaryngeal space. The percent incidence was substantiated to a surprising degree. This confirmation of the figures is important. Of more importance was the finding of secondary and tertiary branches of either or both the abductor or adductor trunks in the extralaryngeal space. The area of these further divisions was confined to the superior thyroid and thyroid areas. The dissections, as they were developed, did not show secondary or tertiary branches lower than the thyroid area (Figs. 3 and 4). The demonstration of these divisions of the divided nerve trunks offers a complete explanation of the so-termed bizarre or controversial types of laryngeal paralysis. For the first time, one can readily understand the production of laryngeal paralysis that is confined to a single muscle or a small group of muscles. The explanation rests on the possibility of injuring or destroying one or more of these smaller branches. Likewise, we have for the first time a logical explanation of the

origin of changes in quality or timbre of voice or both, and of voice fatigue following surgical trauma. These changes are subjectively evident but are difficult if not impossible to demonstrate by direct or indirect laryngoscopy. Injury to one or some of these terminal branches is the logical answer. The demonstration of these smaller branches in the vulnerable area is sufficient to conclude that they can be injured. We are therefore equipped with sufficient anatomical material to explain any and all of the types of laryngeal paralysis and paresis that follow thyroid or other surgery in this area. The demonstration of the initial divisions of the recurrent laryngeal nerve into adductor and abductor trunks in the extralaryngeal area was a great contribution. However, it limited the explanation of the laryngeal paralyses to a total adductor or abductor type. It did not permit a logical explanation of partial adductor or abductor involvement or partial combinations of both nor could the simple separation of the two types of fibers explain a single muscle paralysis.

There are obvious reasons why there is little if any opportunity to carry out clinical experiments on the normal living human subject. There are reasons to question the validity of the findings produced by stimulation of the nerve branches or main adductor and abductor trunks or either of them in the deeply anesthetized patient or in the muscle responses in the larynx that is grossly infiltrated by malignant neoplasm. In a few personal experiences it was evident that a larynx that was sufficiently infiltrated with neoplasm to show fixation of the vocal cord on attempted voluntary motion showed no additional motion on artificial stimulation. The opportunities to obtain reliable results from stimulation of the major trunks or their branches in the living human subject are extremely limited.

Murtagh and Campbell⁴⁶ offer the thesis that there is a difference in size between nerve fibers of the adductor and those of the abductor nerves; the latter being predominantly the smaller and the less myelinated of the two. Now that attention has been directed to the fact that the recurrent laryngeal nerve is found as a divided trunk in the extralaryngeal space sufficiently often to eliminate this condition as being a rarity it should be an interesting and worthwhile project to obtain from fresh cadavers specimens of these branched nerves, and subject these divisions to microscopic examination. A rough protocol for correlating the microscopic and gross anatomic findings is intriguing. If the conclusive experimental and clinical

work demonstrating that the initial division of the recurrent laryngeal nerve separates the adductor and abductor fibers is substantiated by the microscopic sections, the cause for controversy will be settled for all time. If such an investigation is not under way, it should be started. The material for study of the normal nerve fiber distribution is easily available. The material from known cases of laryngeal paralysis of peripheral origin is limited. Even so, it should require only a few selected cases to complete a well rounded protocol.

In a preliminary report we⁴⁹ presented this material with attention directed to the fact that the finding of a divided recurrent laryngeal nerve was not an anatomical anomaly. It was emphasized that the surgeon invading the thyroid area should expect to find one or both nerves as divided structures in 43% of the patients. We emphasize this point to discourage the false sense of security that accompanies the exposure and isolation of a sizeable nerve in the danger area. It is no longer acceptable to consider any identified nerve in this area as being the entire recurrent laryngeal nerve unless one proves it to be so by following it to at least the level of the clavicle. This is not feasible as a routine procedure. The finding of secondary and tertiary branches in the extralaryngeal area and the ultimate and variable nerve-artery relationships were stressed to encourage the surgeon to bypass the unpleasant sequelae of laryngeal paralysis by avoiding mass clamping and ligature or suture to obtain hemostasis. The knowledge of the branched nerve and the inconstant relation of the nerve and its branches to surrounding anatomical landmarks, as well as the major branches of the inferior thyroid artery, leaves no opportunity for any single surgical approach to eliminate all the opportunities or probabilities of nerve damage. The knowledge of the variations in anatomical relationships in the complex nerve-artery associations does equip the surgeon to proceed in this area with the chances of nerve damage at the level of the irreducible minimum. These statements are made with the full realization that in spite of this knowledge and meticulous surgical technique, there will be instances wherein nerve damage cannot be avoided. Varying percentages of nerve damage following thyroid surgery are available.^{22, 50}

Frankly, the significance of the finding of secondary and tertiary branches in the extralaryngeal space was not realized until the total number of dissections was nearly complete. The initial objective of the dissections was to demonstrate the presence or absence of divided

nerve trunks and to note the area where the branching occurred in relation to the thyroid gland and the inferior thyroid artery. A review of the completed chart diagrams of the dissections to determine the relative positions of the nerve and the major branches of the inferior thyroid artery showed the presence of the secondary and tertiary divisions. Also evident was the opportunity to incorporate some one of the branches of a multi-branched nerve in a ligature or suture. The recognition of this fact supplied the missing information essential to satisfactorily explain the less common and less extensive types of laryngeal paralysis.

CONCLUSIONS

1. The dissection of 100 cadavers (200 recurrent nerves) shows conclusively that the division of the recurrent laryngeal nerve into two or more branches in the extralaryngeal area is not an anatomical rarity.

2. The variable and complex nerve-artery relationship establishes a potential nerve-damage-hazard associated with all surgery in the thyroid area.

3. One should expect to find divided trunks in 43% of adult humans.

4. Injury to one or the other trunks of a simple divided nerve will produce a pure adductor or abductor paralysis.

5. It was demonstrated that secondary and tertiary divisions of the recurrent laryngeal nerve occur in the extra laryngeal area.

6. The presence of these secondary and tertiary nerve divisions in this vulnerable area offers an adequate explanation for the more complex and heretofore controversial types of laryngeal paralysis.

7. Injury to one branch of a multi-branched nerve will produce paralysis only in the area supplied by that branch. In some instances, only a single muscle or a portion of a muscle may be supplied by this branch.

8. The present explanation of the various laryngeal paralysis following surgical trauma eliminates the necessity for fabrication of motor filaments in the otherwise purely sensory internal branch of the superior laryngeal nerve.

9. There is now available an adequate and logical explanation for all of the types of laryngeal paralyses found following surgery in the thyroid area. The explanation is based entirely on injury to the recurrent laryngeal nerve or its branches sustained during the surgical procedure.

TABLE II.

Bilateral Single Trunk Nerves	(70)	35%
Bilateral Divided Trunk Nerves	(48)	24%
Mixed Single and Divided Trunk Nerves	(82)	41%

TABLE III.

LOCATION OF DIVISIONS OF NERVES (86 NERVES)

Superior Thyroid Space	21	24.2%
Thyroid Space	47	54.7%
Inferior Thyroid Space	18	20.9%

TABLE IV

RELATION TO BRANCHES OF THE INFERIOR THYROID ARTERY
(200 NERVES)

	ANTERIOR	BETWEEN	POSTERIOR	PARALLEL
Right Single Trunk Nerves	19	9	18	10
Right Divided Trunk Nerves	17	17	20	8
Left Single Trunk Nerves	14	12	33	12
Left Divided Trunk Nerves	5	14	22	4
Total	55	52	93	34
Percent	27.5%	26.0%	46.5%	17.0%

TABLE V.
NUMBER OF DIVISIONS OF DIVIDED NERVES
(86 NERVES)

	2	3	4	5	6
Right	40	4	6	1	
Left	27	5	1	1	1
Total	67	9	7	2	1
Percent	77.91%	10.46%	8.14%	2.32%	1.16%

CHRONOLOGICAL DATA

- 100 A.D. Rufus The Ephesian. Greek anatomist. Treatise on Nomenclature on Parts of the Human Body. Aphonia and respiratory distress associated with injury of the recurrent laryngeal nerves.
- 160-200 A.D. Galen. The first recorded observations on experimental paralysis of the recurrent laryngeal nerves. Animals . . . aphonia and respiratory distress.
- 1514-1564. Vesalius.⁴⁷ Interested in the role the recurrent laryngeal nerves played in voice production. Experimental work and drawings of the human larynx showing the structures and the superior and recurrent laryngeal nerves.
1734. Martin.¹⁹ Animal experiments. Return of voice (dog) due to communicating branches of the superior and recurrent laryngeal nerves.
1792. Haighton.²⁰ Believed that the severed ends reunited.
1813. Magendie.¹ First to demonstrate experimentally that the cricothyroideus was innervated by the superior laryngeal nerve.
1823. Stedman.² Anomalous right recurrent nerve. Explanation was not adequate.
1826. Hart.³ Irregular origin of the right recurrent laryngeal nerve. Secret of the circuitous route was to be found in the natural law of embryonic development.
1828. Rainey.⁸ Anomalous right recurrent laryngeal nerve.
1832. Meckel.⁴ First to make mention of a divided recurrent laryngeal nerve. Rare but when it did occur it is always on the right side.
1836. Ley.²¹ Fertile imagination as to the function of the cricoid cartilage.
1836. Magendie.¹ Anatomists unable to explain why the recurrent laryngeal nerves took such a complicated course.

- 1837. Hilton.⁹ Anomalous right recurrent laryngeal nerve.
- 1838. Reid.^{6, 18} One of the first to present experimental studies that approached completeness and were of scientific value.
- 1846. Hérard.⁵ Anomalous right recurrent nerve.
- 1847. Reid.⁶ Gave correct explanation of the origin of the anomalous recurrent nerves.
- 1848. DeMarquay.^{29-quoted} Anomalous recurrent laryngeal nerve.
- 1869. Philipeaux and Vulpian.¹¹ Anastomosing fibers when present come from the superior laryngeal nerve.
- 1871. Luschka.¹² No communication between the superior and recurrent laryngeal nerves even though they might share the same sheath for short distances.
- 1876. Habershon.¹⁰ Communications between the superior and recurrent laryngeal nerves.
- 1879. Woelfler.¹⁴ Dissections. Nerve always anterior to the artery.
- 1881. Semon.^{32-quoted} Formulated and presented his Law.
- 1883. Brenner.⁷ Anomalous right recurrent laryngeal nerve.
- 1883. Kocher.¹⁵ Dissections. Artery posterior to the nerve.
- 1884. Exner.¹³ Elaborate monograph.
- 1885. Rotter.¹⁶ Dissections (fifteen). Only one-third followed the pattern described by Kocher.
- 1886. Streckeisen.¹⁷ Dissections, 56. Mainly interested as to whether or not the artery formed a loop around the nerve.
- 1887. Hooper.⁴⁵ Excellent article . . . both historically and practically.
- 1892. Russell.³² Substantiated Semon.
- 1906. Grossmann.³³ In certain cases of paralysis the cords assume the cadaveric position due to the atrophy of disuse of the cricothyroid muscle. Work has not been confirmed.

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DISCUSSION

HARRIS P. MOSHER, M.D.: What is there to discuss in this paper? I don't see anything. I envy the speaker. His presentation of his paper is a thing I could never approach, and some of the men looking at me I don't think could approach it either. I believe it is the ideal way to present a medical paper.

JAMES M. ROBB, M.D.: I have always been interested in anatomy but have not seen demonstrated so clearly before the division of the recurrent laryngeal nerve.

I know the vocal cord must vibrate but occasionally I have seen peculiar movements which may be due to a paresis rather than to a paralysis of the cord. Is this possible?

DR. LOUIS H. CLERF: It is admittedly true what Dr. Mosher says, that it is difficult to discuss this paper, but we can talk about it. I want to congratulate Dr. Morrison for the magnificent bit of work he has done here. I do not think we should be too hard on Magendie because of his failure to interpret things correctly. All you need to do is take down the most recent textbook and when you get through reading about the innervation of the larynx if you are not thoroughly confused I would suggest you read it a second time because there still is much confusion coming down through the ages.

I have been interested a long time in the problem why an aneurysm of the arch of the aorta will produce a certain type of paralysis and why the surgeon who injured the recurrent nerve while doing a thyroidectomy has produced an entirely different picture in the larynx. Now, it is just as clear as injury to the extensor and flexor muscles of the arm or leg.

What interested me was finding in the literature that in 1938 Lahey and Hoover did some dissecting of the nerve and found it bifid. There are two branches or a division extralaryngeally in the occasional case. They concluded it was very unusual. In 1942, Weeks and Hinton dissected 58 nerves or half that number of bodies and found in 43, or 78 per cent, the nerve divided extralaryngeally and later Hinton working with Armstrong to further amplify this, secured 50 bodies, 100 nerves and they found 73 per cent divided extralaryngeally. They pointed out that commonly the division was near the inferior thyroid artery but they did not say anything particularly about additional branches being given off. That is important to explain bizarre paralyses that one sees occasionally.

In my experience one rarely sees a paralyzed vocal cord in an intermediate position following thyroid gland surgery. The common position is one of adduction, either unilaterally or bilaterally. I have seen only a few cases where the cord was in a position of abduction. Very recently I saw a patient who had a bilateral paralysis of the larynx. The cords were both well tensed and only slightly abducted, not in an intermediate position, but one might say, in a position between the midline and the intermediate position. That is the first time I have ever seen that. Too little time has elapsed to know what will happen but to me that is a strange paralysis and certain of Dr. Morrison's anatomical findings may afford an explanation.

LEWIS F. MORRISON, M.D. (closing): In reply to Dr. Robb's question, yes, we do have pareses, and they can be on two bases: one, that a portion of a muscle is involved and the rest of it is not involved, in which case there will be a fibrillation or true paresis. The other basis is, and you have to put a time factor on here, a certain number of injuries that go ahead to complete recovery later with or without evidence of a complete paralysis. Otherwise we would not be able to explain these people who have adductor involvement, who will be aphonic for a period of time and a month later or even as long as six months later regain function following psychic insult. In one instance at a New Year's Eve party a husband slapped his wife, she let out a yell and has talked well ever since.

DR. ROBB: Was that in California?

DR. MORRISON: Yes, sir. It can be readily explained on this rather simple, if direct, basis.

I am pleased that Dr. Clerf brought out the matter of confusion in the present literature. In the paper, as such, I did make mention of the fact that one should not smile too smugly about the peculiar statements in the older literature because if you really want to become confused just read the modern text books.

One other thing that I thought maybe someone might bring up is as to why after all these decades we should suddenly find that the recurrent laryngeal nerve divides outside of the larynx. Bodies have been dissected for as long as we know and only in a few instances is there mention of the fact that the nerve divides extralaryngeally. If it had only happened in five percent or three percent of the cases, I would say, well, maybe something is wrong; but to find as high a percentage as 24, or as Dr. Clerf mentions, 75 percent, somebody has been doing a lot of dissecting in the past and they have not been seeing what they have been looking at. That must be certain.

THE PLACE OF TRACHEOTOMY IN HEAD INJURIES

HOWARD McCART, M.D.

TORONTO, CANADA

Normal brain tissue is susceptible to damage from anoxia, and the brain injured, whether it be as a result of trauma, operation, inflammatory processes, impaired circulation, or space occupying lesion is more susceptible to anoxia.

It is felt that anoxia can be prevented in many cases of cerebral trauma by means of a tracheotomy where the simpler methods for relief have failed. In 1859 Marshall Hall stated in the *Lancet* that we must regard tracheotomy as a heroic remedy only appropriate to herculean forms of disease. Today in expert hands it is a dramatic but harmless procedure carrying no additional risk for any patient with secretory obstruction in the tracheo-bronchial tree.

Galloway¹ was one of the first to show the value of tracheotomy in combating cyanosis and flooding of the lower respiratory tract. The flooding of the lower respiratory tract in comatose patients with brain injury is due to the failure of the cough mechanism which normally clears the respiratory passages of accumulated secretions.

This cough mechanism consists of inspiration, closure of the glottis, increase in intrathoracic pressure, opening of the glottis and sudden expiration with release of the intrathoracic pressure. Any interruption in the physiology of this mechanism by coma or depression of the respiratory centre in the medulla will retard or abolish the cough reflex.

Respiratory obstruction causes an elevation of the intrathoracic venous pressure due to the laboured breathing necessary to overcome the partial obstruction which results in a reduction of the ventilating capacity of the lungs—any increased intrathoracic pressure is readily transmitted to the intracranial venous system producing venous congestion of the brain resulting in increased intracranial pressure which in turn produces anoxia.

From the Department of Otolaryngology, University of Toronto.

Practically every patient with a severe cerebral trauma is in danger of respiratory obstruction from salivary, pharyngeal and tracheo bronchial secretions. Any attempt to swallow may result in entry into the larynx of fluids and solids. If vomiting occurs there is a grave danger that the vomitus may be inhaled and respiratory infection follow. Many of these patients appear to salivate profusely, and the presence of a feeding tube often causes irritation which increases the accumulation of secretions in the pharynx. The respiratory passages are not cleared of the accumulated secretions due to the ineffectual cough so that stagnation of the secretions occurs necessitating constant medical attention to prevent atelectasis and infection. Other cases may become obstructed due to a relaxed pharyngeal musculature, laryngeal spasm, paralysis of the vocal chords or accumulation of dried exudate on the chords or subglottic area, and in children following prolonged endotracheal anaesthesia subglottic oedema is not infrequent. In critically injured brain cases decerebrate rigidity may be present resulting in the inability to clear the tracheobronchial secretions.

Partial respiratory obstruction also causes hypoventilation so that the ratio between the amount of air breathed in per minute and the amount of oxygen consumed per minutes falls; as hypoventilation becomes more severe carbon dioxide is retained in the body with an increase in the carbon dioxide content of the systemic and cerebral arterial blood which causes a marked dilatation of the cerebral vessels. Hypoventilation creates a fall in the oxygen content of the arterial blood bringing about an arterial anoxemia. When the cerebral vascular endothelium becomes damaged by anoxemia, it is permeable to fluids, resulting in edema, and haemorrhage is apt to occur. This arterial blood of low oxygen content is also a dilator of cerebral vessels.

The venous congestion and the arterial dilatation raises the intra-cranial pressure and bleeding from the cerebral vessels may occur. The intra-cranial pressure which in time depresses the medullary centre, accentuates the inadequate airway and decreases the aeration of the lungs thus establishing a vicious circle.

REPORT OF CASES

CASE 1.—S. H. A 70 year old female admitted to Toronto General Hospital in deep coma following an automobile accident. There

was no external sign of head injury, there was no response to painful stimuli and the respirations were 30-40 per minute. A few hours after admission extreme muscular effort became associated with breathing and a tracheotomy was performed.

There was considerable improvement in the ariway but the patient died three days later from midbrain and cerebral contusions. The temporary improvement indicated that the anoxia was partly responsible for the loss of consciousness.

CASE 2.—E. B. A 57 year old female admitted to hospital because of fatigue and inability to carry on her usual household duties. For the past two weeks previous to admission she complained of headache in the frontal region more particularly on the right side and which was associated with nausea and for the past two weeks was "befuddled" mentally and had occasional incontinence of bladder and bowels. Little to find on physical examination other than the patient was drowsy and would fall off to sleep during examination. There was an early papilledema. Pulse Slow 50-60 per minute. Ventriculogram showed a large space occupying lesion in right frontal lobe. A resection of the right frontal lobe was carried out removing a cystic astrocytoma.

The patient never regained consciousness following operation and twenty hours later developed acute dyspnea. A tracheotomy was performed but the patient died twenty hours later.

Autopsy was not obtained but in all likelihood the patient had involvement of the brain stem from uncal herniation at the time of operation.

CASE 3.—G. N. A 47 year old man admitted to hospital 45 minutes after having been struck by a car. He was well orientated as to time, place and person. Two hours later he gradually became confused and lapsed into deep coma with stertorous breathing. The left pupil was larger than the right and fixed, a spinal tap showed gross blood.

He showed decerebrate rigidity and the temperature rose to 105°. Following a removal of a subdural haematoma there was increased respiratory difficulty and more generalized muscular hyper-tonia. A tracheotomy was performed followed by marked improve-

ment in the patient's general condition. Subsequently the patient was at a low level unconsciousness and died six weeks following injury.

CASE 4.—B. U. A 17 year old boy sustained a compound fracture of the right frontal region, was hospitalized three weeks, returned to work two and a half months following his injury. Six months later was admitted to Toronto General Hospital because of headaches gradually becoming more severe and were frequently associated with nausea and vomiting. He also lacked ambition and energy and two weeks prior to admission began to have difficulty with his speech. On admission he lay in a position of opisthotonus, breathing rapidly, temperature 103.° He was confused and disorientated, early papilledema was present on the right side and left lower facial weakness was present. Ventriculogram revealed a space occupying lesion in the lower portion of the right frontal lobe. Operation revealed a large right frontal lobe abscess.

Following operation, cough was ineffectual and patient had accumulation of tracheobronchial secretions. Tracheotomy performed through which large amount of secretions were aspirated. The patient did not improve very much, though the spinal fluid cleared. He did not regain consciousness following operation, he became quadriplegic and paralysis of his respiratory muscles took place.

CASE 5.—H. P. An 18 year old boy struck by a car while riding a motor scooter and rendered immediately unconscious. On admission to hospital he was deeply comatose. Skull films did not show any gross evidence of fracture. Twenty-four hours after admission his temperature rose to 104° and respirations between 50 and 60 per minute—cough ineffectual. Tracheotomy performed and progressive improvement followed establishment of an adequate airway. Due to the depressed conditions there was an absence of cough reflex permitting accumulation of secretions. Patient discharged two weeks following admission.

CASE 6.—L. S. A 51 year old man was admitted to hospital giving history of good health until 10 days previous to admission when he became drowsy, mentally confused, some speech difficulty and unsteadiness in walking. Ventriculogram indicated tumour in the left frontal lobe. A left frontal lobectomy was performed for a

malignant tumour. Following operation the patient became cyanosed, had considerable difficulty with his breathing and anoxic, before a tracheotomy was carried out.

There was marked improvement in cyanosis and airway following tracheotomy but patient had a slow and unsatisfactory recovery. He was discharged from hospital six weeks after admission being aphasic though he could understand what was said to him. He did not have any motor paralysis.

If the tracheotomy had been more quickly performed it is unlikely this patient would have had any permanent damage to the central nervous system from the anoxia.

CASE 7.—L. H. A 56 year old man was admitted to hospital with a gross arterio venous malformation on the left side of the neck involving the internal maxillary and superficial temporal artery and also a small aneurysm of the right internal carotid. He also had moderate cardiac decompensation secondary to the arteriovenous aneurysm.

Two attempts to stab the right common carotid for an arteriogram failed and a cut down was done under general anaesthesia (pentothal). Following the closure of the wound the patient began to struggle and while he was being lifted on to a stretcher he coughed out his endotracheal tube. On the way back to his ward he became very cyanosed and stopped breathing. After artificial respiration and coramine his heart began beating and respirations resumed but were ineffectual. The endotracheal tube could not be re-inserted and a tracheotomy was carried out with the resumption of normal respirations and colour.

LARYNGOSPASM FOLLOWING PENTOTHAL AS AN ANESTHETIC

CASE 8.—D. N. A 14 year old boy fell 30 feet from a tree to the ground becoming immediately unconscious. Eight days later admitted to Toronto General Hospital semi comatose, more or less restless, was never conscious enough to talk. The right pupil was dilated and fixed, the right ear drum perforated. Complete right hemi-paresis. Temperature 104.° Following upper mid temporal burr holes for subdural hematoma, the patient developed respiratory obstruction due to accumulated secretions so that a tracheotomy was

performed. The airway was greatly improved and the tracheotomy tube was removed in three weeks. After remaining semi-comatose for nearly two months the patient gradually began to respond and on discharge from hospital, was alert, attentive, interested in his surroundings and orientated in all spheres.

CASE 9.—R. S. A 23 year old man admitted to Toronto General Hospital eight hours after having been struck by a car while riding a bicycle. He was a known epileptic from early childhood. On admission he was deeply comatose, with blood and brain tissue escaping from left ear. X-rays revealed multiple skull fractures, short rapid respirations suggested some brain stem damage, all four limbs were hypertonic. Following a second temporal decompression the patient had some respiratory difficulty with dramatic improvement following tracheotomy.

This patient was discharged from hospital 5 months after admission having slowly but gradually improved in regard to his level of unconsciousness. On discharge from hospital he was reasonably well orientated though attention was poorly sustained. It was felt that tracheotomy saved this young man's life.

CASE 10.—M. S. A 14 year old girl was thrown from a horse, eight hours later was admitted to Toronto General Hospital deeply comatose and showed signs of rigidity. Following removal of a subdural hematoma, her respirations became more labored and she was becoming continually plugged with mucus. A tracheotomy was performed with marked improvement of her respiratory problem. Her state of unconsciousness did not change and she was sent home with a nurse five weeks after admission with the tracheotomy tube in place.

CASE 11.—A. T. A 68 year old man, eight hours before hospital admission complained of sudden severe headache in vertex and within a few moments was unconscious. A large subdural hematoma removed. Four weeks following operation, the patient's condition was precarious and due to ineffectual cough he had an accumulation of tracheobronchial secretions. A tracheotomy was carried out with marked respiratory improvement. Discharged from hospital 6 weeks after admission with almost full bladder control and active movement of right upper and lower limbs which were almost equal to those of the left.

It was felt that tracheotomy saved this patient's life when the general condition was very precarious.

COMMENT

Eleven cases are reported in tracheotomies well performed after severe brain injuries. Four of these patients died most likely because of midbrain and cerebral contusions or the presence of extensive damage to the medullary respiratory centre which may explain failure of a tracheotomy to restore normal breathing. In some cases it may be difficult to interpret whether or not the pulmonary oedema is of cardiac origin in which case tracheotomy is of no avail. The average age of the group that died was 47 years and that of the group which survived was 35 years. It was felt that this later group of patients would have died if tracheotomy had not been carried out.

Many of the usual procedures such as postural drainage, hypoventilation by means of blow bottles or carbon dioxide inhalations and bronchoscopy are impractical or dangerous to patients with brain injuries. Aspiration through an endotracheal tube should only be carried out as an emergency measure to maintain a patent airway during the performance of a tracheotomy, as it may cause trauma and further laryngeal surgery will be necessary if left for any length of time. Bronchoscopic suction is ideal for removal of isolated plugs of mucus or in mild cases of secretory obstruction, but in many cases it is contraindicated especially in head injury cases complicated by fractures of the cervical vertebrae.

CONCLUSION

It should be stressed, where the simpler procedures for the removal of the tracheo bronchial secretions have failed, that a tracheotomy should be performed in all patients with cerebral injury who are comatose or semi-comatose and show signs of respiratory distress or cyanosis.

The indications for tracheotomy in brain injury vary in individual cases, and the indications are not necessarily those of the classical picture of dyspnea due to laryngeal obstruction. Other indications for the procedure of tracheotomy are patients with relaxed pharyngeal musculature, loss of swallowing and gag reflexes, crusting in the pharynx and larynx which leads to exhaustion of the patient due to the muscular effort associated with the labored breathing

required to overcome the partial respiratory obstruction. One should not forget that the presence of a feeding tube increases the amount of pharyngeal secretions and may cause laryngeal inflammation or perichondritis. Finally unconscious patients are often very sensitive to slight degrees of oxygen want and even minor degrees of cyanosis are important as it may not be recognized due to loss of blood or shock.

The author expresses appreciation to Dr. K. G. McKenzie and Dr. H. Botterell, Department of Neuro Surgery for the histories reviewed in this report.

170 ST. GEORGE STREET.

REFERENCE

1. Galloway, Thomas C.: Tracheotomy in Bulbar Poliomyelitis, J. A. M. A. 123:1096-1097 (Dec. 25) 1943.

DISCUSSION

THOMAS C. GALLOWAY, M.D.: The things that Dr. McCart has told us are so obvious and so true it is hard to realize that it is only a few years ago that there was so much confusion and doubt about this subject, and that there is still in some quarters.

His message should be gotten across to every practitioner in medicine, surgery, or pediatrics.

We have stressed the urgency of anoxia. Eighty seconds may make permanent changes. Three minutes is serious. Five minutes is likely to leave a very permanent and severe damage to the brain and eight minutes is likely to result in death from the anoxia.

Reviewing some experiments of Coryllos on drowning, I have been struck by the importance of spasm and the similarity to the state in which these patients are to drowning. Coryllos showed in submersion experiments in dogs, drowning takes about four to five minutes. There are four stages, a preliminary stage of apnea, the first and second stages associated with carbon dioxide accumulation with increased blood pressure which in itself in an anoxic patient is an indication for tracheotomy; and in the third stage in which the heart begins to fail there is a period of only 10 or 15 seconds in which resuscitation is possible.

These patients with brain injury, are depressed by respiratory obstruction or other causes and are very susceptible to further anoxia and then may get a laryngeal spasm.

One of our patients was getting along well late in the course of polio, received a few chunks of ice, and died in spasm.

The experiments are substantiated by the work of Motley and in physiology show that in drowning the patients may die not of flooding of the airway or of the pulmonary respiratory area but they die of spasm. In very many of these

cases there is no fluid in the lungs. The airway may however be flooded in the stage of terminal asphyxia.

A man may sit stupidly by and watch a patient die from respiratory obstruction who would plunge without hesitation into the surf to rescue a drowning patient. Yet the emergency is just as great with the patient who has respiratory obstruction with developing spasm.

Just a question about technique of tracheotomy. The operation is still semantically damned by being called radical. Some of the younger men do not know how to do tracheotomies and I think that the reason that they do not know is that the operation is considered so simple that some of us do not take the trouble to teach men how to do it. I have just a few points of technique which I insist on, especially if a respirator may be used. There is no undermining of fascia. The only transverse incision I make is to cut transversely through thyroid fascia to free the isthmus. There should be exact orientation in the midline. A high tracheotomy is permissible if a respirator may be needed. Hemostasis must be meticulous. To prevent emphysema there should be an open airway over which the tracheotomy is done. I like an endotracheal tube about as well as a bronchoscope if I have a cooperating anesthesiologist. Dr. McCart is so right in saying one must not depend upon the endotracheal tube for long. In cerebral injuries if a patient is likely to require help longer than 20 hours, one should not use an endotracheal tube but the tracheotomy tube.

JAMES M. ROBB, M.D.: I would like to support this matter of tracheotomy. You heard me mention it yesterday.

And I shall keep saying this as often as I can hoping that the entire profession may become aware of the fact that a tracheotomy is an extremely important procedure, to be done early rather than late.

O'Dwyer observed between 1867 and 1881 in the New York Foundling Home that not a single child who had been tracheotomized lived. He sought a remedy and the intubation tube resulted. Now, because of suction, chemotherapy, and antibiotics, tracheotomy alone is the method employed, with no fear of tragic consequence.

I am so glad that both Dr. McCart and Dr. Galloway have discussed tracheotomy, impressing upon the profession that it is a life-saving effort which must be done early.

WILLIAM J. McNALLY, M.D.: I would like to discuss Dr. McCart's paper in order to reemphasize what has just been said. At the neurological institute in Montreal there are many of these cases. As Dr. Robb says, the patient, may be under anesthesia with a intratracheal tube in position for a long while and frequently after operation the intratracheal tube is allowed to remain in. When it is removed after some hours the patient may have difficulty and the laryngologist is called upon. In a recent case I was called in, the patient had had an intratracheal tube in position for 20 hours. A cerebral arterio-venous aneurysm was suspected. The patient was unconscious. When I saw the patient, he was breathing with a little expiratory difficulty. There was a suspicion of an intrapulmonary condition, so tracheotomy was postponed to have a chest x-ray taken to confirm the physical findings, within an hour an emergency had arisen. We decided upon immediate tracheotomy but the heart stopped while the anesthetist was introducing an intratracheal tube. Immediate opening of the chest and cardiac massage failed to revive the patient. We had lost the opportunity by not doing the

tracheotomy the first time we saw the patient. Many times you cannot delay, even for what seems to be a wise but not vital additional examination.

HOWARD W. D. McCART, M.D. (closing): I think Dr. Robb was very timely in what he said about waiting for the patient to die. That was my first recollection of a tracheotomy in 1924 and 1925 when I was house surgeon. You waited till the patient's pulse was 150, 160 before the tracheotomy was done and he frequently died on the table, and I have mentioned and others have mentioned, these people should get a lot of watching and should be as carefully handled as in polio cases, otherwise the patient will die.

As far as the endotracheal tube is concerned, I don't know whether it caused the granuloma or the tube was left in intracheal cases too long. If difficult, it is easy enough to clip it on the bronchoscope. I did ask Dr. Ireland to do this on a private case. He would not go. I put the bronchoscope in and did the tracheotomy at my leisure, no fuss, but my recollection of tracheotomies in 1924 was blood all over and everybody was excited.

Executive Sessions

1952

FIRST EXECUTIVE SESSION

MAY 23, 1952

The First Executive Session of the Seventy-Third Annual Meeting of the American Laryngological Association held at the Royal York Hotel, Toronton, Ontario convened on May 23, 1952 at 12:15 P. M., following the First Scientific Session. Dr. H. Marshall Taylor, President, presided.

The following were elected to *Active Fellowship* in the Association:

DR. JOHN ERICH
DR. PERCY E. IRELAND
DR. MERCER LYNCH
DR. JULIUS W. McCALL
DR. ALDEN H. MILLER

To *Corresponding Fellowship*:

DR. RICARDO TAPIA ACUNA, Mexico City, Mexico

The Newcomb Award was given to Dr. William J. McNally of Montreal.

The following Fellows were elected to serve as a Nominating Committee:

DR. RALPH FENTON
DR. ARTHUR W. PROETZ
DR. CHARLES BLASSIGAME
DR. DANIEL CUNNING
DR. LEWIS MORRISON

CITATION FOR THE NEWCOMB AWARD

The distinguished Fellow who is being cited for the Newcomb Award for 1952 was born in Bryson, Quebec 55 years ago. He received his early education in the Provinces of Quebec and Nova Scotia and was awarded the Degree of Bachelor of Arts by Dalhousie University in 1921. He was granted the Degree of Doctor of Medicine and Master of Surgery by Dalhousie University in 1922 and has been awarded a number of Honorary Degrees as follows:

Master of Science, McGill University in 1925.

Doctor of Science, McGill University in 1934.

Doctor of Laws, St. Francis Xavier University in 1951.

He early manifested interest in research problems and received the Cooper Research Scholarship in Experimental Physiology at McGill University in 1924 and worked under the direction of Professor John Tait. The following year was spent at Rijks University, Utrecht, Holland as the Travers Allan Travelling Fellow from the Royal Victoria Hospital of Montreal.

Outstanding achievement in the field of Otolaryngology has brought many honorary distinctions. The Dalby Memorial Prize offered by the Royal Society of Medicine of London was awarded to him in 1938. He was appointed to the Honor Section of the American Academy of Ophthalmology and Otolaryngology in 1944. He has achieved honorary membership in the Scottish Otolaryngological and Laryngological Society, has been President of the Canadian Otolaryngological Society and is an Honorary Member of the Section of Otology of the Royal Society of Medicine of London. In addition, he is a Diplomat of Laryngology and Otology, Royal College of Physicians and Surgeons, of England as well as the American Board of Otolaryngology and a Fellow of the Royal College of Surgeons of Canada.

His contributions to medical literature are numerous and many of them have received favorable comment by foreign as well as American Journals.

He presently is Professor and Chairman of the Department of Otolaryngology, McGill University, Montreal.

Although devoted to his profession which occupies a great part of his time he has a few hobbies. He likes to play golf and also spends some time each summer "roughing it" in his garden getting close to nature.

The James E. Newcomb Award for 1952 is presented DR. WILLIAM JAMES McNALLY in recognition for his outstanding contributions in the field of Otolaryngology.

Signed,

NEWCOMB AWARD COMMITTEE.

ANNUAL BUSINESS MEETING

MAY 24, 1952

The meeting convened at 9:35 A. M., on May 24, 1952. The President, Dr. H. Marshall Taylor presided.

The report of the Secretary was accepted as circularized.

The combined reports of the Treasurer, Auditor were received and accepted with thanks.

The Nominating Committee recommended for election as Councillors the following:

LOUIS H. CLERF

JOHN MACKENZIE BROWN

EDWIN N. BROYLES

HARRY P. SCHENCK

GORDON F. HARKNESS

BERNARD J. McMAHON

FREDERICK T. HILL

RALPH A. FENTON

GORDON B. NEW

H. MARSHALL TAYLOR

There were no nominations from the floor and the Committee's recommendations were approved unanimously.

The report of the Librarian and Historian was presented and accepted unanimously.

Dr. Frederick T. Hill reporting for the American Board of Otolaryngology stated that the Board examined 96 candidates at the Chicago meeting in October and 78 certified, 14 conditioned and 4 failed. At the May meeting in Toronto 98 candidates were examined; 78 were certified, 16 were conditioned and 3 failed. The report was accepted.

Dr. Arthur W. Proetz reported for the Laryngological Research Committee. He indicated that there had been so little time that no

meeting of the Committee was held. The following proposals were submitted:

First, to make a survey of persons in this country with definite problems and the mental capacity and research training to investigate effectively, and this should be done in a fairly personal manner and not by means of routine questionnaire.

Second, to make a survey of the places of institutions themselves where the work can be carried out.

Third, to make a survey of the ability of persons or corporations in various communities able to supply funds for research.

It would be the approach of the committee to go to such individuals or corporations with a definite problem, a particular investigator and a laboratory lined up. It is felt that perhaps funds would be more easily obtained from individuals in the community where the work was to be done or from corporations particularly interested in nature of research.

In the discussion the Fellowship expressed the opinion that efforts should be made to secure or offer to help young men, well recommended, who need a small sum of money to carry out certain problems and further recommended that the Secretary in his post-session letter inform the Fellowship that the Committee is interested in young men who have projects that should be investigated. The report was accepted.

On the recommendation of Council two propositions regarding an increase in dues were submitted. The first was to increase the annual dues from \$35.00 to \$40.00, this to include the registration for the Annual Meeting, or to increase the annual dues to \$50.00, this to include the registration fee as well as the cost of the dinner and reception. The Fellowship unanimously voted that the dues be increased from \$35.00 to \$40.00 annually, and this would include the registration fee for the Annual Meeting.

There being no further business the meeting adjourned at 10 A. M.

Respectfully submitted,

LOUIS H. CLERF,

Secretary.

REPORT OF THE SECRETARY
AMERICAN LARYNGOLOGICAL ASSOCIATION

MAY 24, 1952

The Seventy-second Annual Meeting of the American Laryngological Association was held on May 9th and 10th, 1951 at the Claridge Hotel, Atlantic City under the Presidency of Dr. Gordon B. New.

There were 77 Fellows and 151 Guests in attendance.

Thirteen scientific papers were presented and were well received.

The following were elected:

Emeritus Fellowship:

DR. HUGH G. BEATTY, Columbus, Ohio

DR. T. ROY GITTENS, Sioux City, Iowa

DR. DEFOREST C. JARVIS, Barre, Vt.

DR. FLETCHER D. WOODWARD, Charlottesville, Va.

Active Fellowship:

DR. GUY L. BOYDEN, Portland, Oregon

DR. FRANCIS W. DAVISON, Danville, Pa.

DR. HOWARD W. D. McCART, Toronto, Ontario

DR. LAWRENCE C. McHENRY, Oklahoma City, Okla.

DR. ROBERT E. PRIEST, Minneapolis, Minn.

DR. F. JOHNSON PUTNEY, Philadelphia, Pa.

DR. AUBREY G. RAWLINS, San Francisco, Calif.

DR. DEGRAAF WOODMAN, New York, New York

The deRoaldes Award was presented to Dr. Arthur W. Proetz.

The Newcomb Award was given to Dr. John D. Kernan.

The following Officers were elected:

Dr. H. Marshall Taylor.....President

Dr. William J. McNally.....First Vice-President

Dr. Thomas C. Galloway	Second Vice-President
Dr. Louis H. Clerf	Secretary
Dr. Gordon F. Harkness	Treasurer
Dr. Harry P. Schenck	Librarian and Historian
Dr. Arthur W. Proetz	First Councillor
Dr. Frederick T. Hill	Second Councillor
Dr. Ralph A. Fenton	Third Councillor
Dr. Gordon B. New	Fourth Councillor

There were no deaths or resignations of Fellows during the past year.

The Council at its Annual Meeting held in New York during January 1952 recommended:

For Active Fellowship:

JOHN B. ERICH, M.D., Rochester, Minn.

PERCY E. IRELAND, M.D., Toronto, Ontario

MERCER G. LYNCH, M.D., New Orleans, La.

JULIUS W. MCCALL, M.D., Cleveland, Ohio

ALDEN H. MILLER, M.D., Los Angeles, Calif.

For Corresponding Fellow:

RICARDO TAPIA ACUNA, Mexico City, Mexico

The Council completed the organization of the laryngological Research Fund Committee. The following were elected to membership:

For three year period:

ARTHUR W. PROETZ, M.D., *Chairman*

HARRY P. SCHENCK, M.D.

ALBERT C. FURSTENBERG, M.D.

For six year period:

LAWRENCE R. BOIES, M.D.

ANDERSON C. HILDING, M.D.

LEWIS F. MORRISON, M.D.

Dr. Samuel Salinger was reappointed Abstract Editor.

The present membership of the Association is:

Active	94
Honorary	5
Corresponding	11
Emeritus	19
	<hr/>
	129

Respectfully submitted,

LOUIS H. CLERF,

Secretary.

EXHIBIT "A"

REPORT OF THE TREASURER AND AUDITORS
AMERICAN LARYNGOLOGICAL ASSOCIATION

BALANCE SHEETS

April 1, 1952

General Fund:

Assets:

Checking Account \$ 6,197.90

Liabilities and Equity:

Liabilities:

Due Trust Fund 6,338.73

Equity:

Deficit Balance—April 2, 1951 \$ (529.79)

Add: Excess of Income Over Expense—Year

Ended April 1, 1952 (Exhibit "B") 388.96

Deficit Balance—April 1, 1952 (140.83)

Total Liabilities and Equity \$ 6,197.90

Trust Fund:

Assets:

Savings Account \$ 2,341.17

Due From General Fund 6,338.73

Bonds:

Par

Cost

U. S. Defense 2½—G—1953 \$ 3,000.00 \$ 3,000.00

U. S. Defense 2½—G—1954 1,000.00 1,000.00

U. S. Treasury 2¼—1956 /59 1,000.00 1,003.50

U. S. Defense 2½—G—1956 5,000.00 5,000.00

U. S. Defense 2½—G—1957 1,000.00 1,000.00

U. S. Defense 2½—G—1958 2,000.00 2,000.00

U. S. Treasury 2¾—1958/63 1,000.00 1,101.20

U. S. Treasury 2½—1964/69 3,000.00 3,000.00

U. S. Treasury 2½—1967/72 1,000.00 1,000.00

U. S. Savings 2½—G—1959 5,000.00 5,000.00

U. S. Savings 2½—G—1960 1,000.00 1,000.00

U. S. Savings 2½—G—1961 1,000.00 1,000.00

Total Bonds \$25,000.00 25,104.70

Total Trust Fund Assets \$33,784.60

Equity:

Fund	Principal	Unused Income April 1, 1951	Expended 1951-52	Income 1951-52	Unused Income April 1, 1952	Principal and Unused Income April 1, 1952
Casselberry	\$ 5,000.00	\$ 622.85	\$	\$ 110.99	\$ 733.84	\$ 5,733.84
Johnston	5,000.00	150.69	101.67	252.36	5,252.36
de Roaldes	3,000.00	387.38	164.00	159.48	382.86	3,382.86
Newcomb Trust	2,000.00	310.83	7.24	45.47	349.06	2,349.06
Newcomb Prize	500.00	12.56	4.00	10.04	18.60	518.60
Hourne Scholarship	20.23	.8842	1.30	21.23
Research	14,414.19	1,792.25	319.91	2,112.16	16,526.35
Totals	\$29,934.42	\$3,277.44	\$175.24	\$747.98	\$3,850.18	
Total Equity						\$33,784.60

EXHIBIT "B"

AMERICAN LARYNGOLOGICAL ASSOCIATION

STATEMENT OF INCOME AND EXPENSE

YEAR ENDED APRIL 1, 1952

General Fund:

Income:

Dues:

1950—1 Member @ \$35.00.....\$ 35.00

1951—85 Members @ \$35.00.....2,975.00

1951—8 New Members @ \$100.00.....800.00

\$3,810.00

Registration Fees1,360.00

Sales of "Transactions"532.80

Total Income\$5,702.80

Expense:

Annual Meeting:

Banquet\$ 950.00

Programs198.65

Projection Equipment Rental and
Operators51.00

Reporting Service132.91

\$1,332.56

Council Meeting:

Council Dinner	\$ 64.43	
Travel Expense	943.13	
		<u>1,007.56</u>
Clerical Hire	500.00	
Cost of "Transactions"	2,019.13	
Stationery and Printing	178.15	
Treasurer's Bond	75.00	
Auditing	95.00	
Mimeograph Service	12.44	
Transfer to de Roaldes Fund	94.00	
		<u>5,313.84</u>
Total Expense		5,313.84
Excess of General Fund Fund Income Over Expense (Exhibit "A")		\$ 388.96

Trust Fund:

Income:

Interest—U. S. Bonds	\$ 625.00	
Interest—Savings Account	28.98	
Transfer From General Fund	94.00	
		<u>747.98</u>
Total Income (Exhibit "A")		\$ 747.98

Expense:

Newcomb Trust Fund—Book Transportation	\$ 7.24	
de Roaldes Fund—Gold Medal Award	160.00	
de Roaldes Fund—Engrossing Award	4.00	
Newcomb Prize Fund—Engrossing Award	4.00	
		<u>175.24</u>
Total Expense (Exhibit "A")		175.24
Excess of Trust Fund Income Over Expense		\$ 572.74

EXHIBIT "C"

AMERICAN LARYNGOLOGICAL ASSOCIATION
STATEMENT OF CASH RECEIPTS AND DISBURSEMENTS
YEAR ENDED APRIL 1, 1952

Cash Balance—April 1, 1951:

Checking Account	\$3,315.18	
Savings Account	2,312.19	
		<u>\$5,627.37</u>

Cash Receipts:

Dues	\$3,810.00
Registration Fees	1,360.00
Donations to Research Fund	1,950.00
Sale of "Transactions"	532.80
Trust Fund Interest Received	653.98

Total Cash Receipts \$8,306.78

Cash Disbursements:

Annual Meeting	\$1,332.56
Council Meeting	1,007.56
Clerical Hire	500.00
Cost of "Transactions"	2,019.13
Stationery and Printing	178.15
Treasurer's Bond	75.00
Auditing	95.00
Book Transportation	7.24
Mimeograph Service	12.44
Embossing de Roaldes and Newcomb Awards ..	8.00
de Roaldes Gold Medal	160.00

Total Cash Disbursements 5,395.08

Excess of Receipts Over Disbursements 2,911.70

Cash Balance—April 1, 1952 \$8,539.07

Consisting of:

Checking Account (Exhibit "A")	\$6,197.90
Savings Account (Exhibit "A")	2,341.17

Total Cash (As Above) \$8,539.07

EXHIBIT "D"

AMERICAN LARYNGOLOGICAL ASSOCIATION

SECURITY AND FUND TRANSACTIONS

YEAR ENDED APRIL 1, 1952

Security Transactions:

None

Fund Transactions:

Principal of Trust and Scholarship Funds, April 1, 1951 \$27,984.42

Additions:

Research Fund Contributions:

Lester Sunderland Foundation.....	\$1,500.00
Walter H. Theobald, M.D.....	100.00
H. Marshall Taylor, M.D.....	100.00
W. H. Johnston, M.D.....	100.00
Henry B. Orton, M.D.....	150.00

Total Additions 1,950.00

Principal of Trust and Scholarship Funds, April 1, 1952

(Exhibit "A") \$29,934.42

As Treasurer, I wish to call the attention of the members to the following pertinent facts:

1. One year ago, even with \$250.00 withdrawn from the Johnston Fund, we had an excess of General Fund expense over income of \$1,858.95. This was largely due to the necessity of paying for the "Transactions" for two years out of the General Fund covering one year. This year the General Fund income exceeds expenses by \$388.96.

2. Our Liabilities (various Fund Accounts with accrued income) exceed our Assets by \$140.83.

3. One year ago our Cash Disbursements exceeded our Cash Receipts by \$1,150.92. This past year our Cash Receipts exceeded our Cash Disbursements by \$2,911.70. Two years ago at the annual meeting we had no banquet nor registration fee, yet our expenses for this meeting were high. A registration fee with present dues is practically a necessity as all income from dues and the sale of "Transactions" is needed to cover the operating expense of the Association.

4. Relative to our Research Fund, donations to the same have been \$1,950.00 as against \$250.00 for the previous year. Changing the name was a good idea but, I believe, the Research Fund Committee will do well to study the problem from the standpoint of something more positive. A goal must be reached financially so that the income will be sufficient to support a worthwhile project. And, in order to interest philanthropy from without the organization, I believe a more definite purpose is necessary for the success of the Fund.

Respectfully submitted,

GORDON F. HARKNESS, M.D., *Treasurer*,

American Laryngological Association.

ACCOUNTANT'S REPORT

We have examined the balance sheet of the American Laryngological Association as of April 1, 1952, and the related statements of income and expense and cash receipts and disbursements for the year then added. Our examination was made in accordance with generally accepted auditing standards, and accordingly included such tests of the accounting records and such other auditing procedures as we considered necessary in the circumstances, with the exception that the allocation of equities to the various funds was accepted as shown by the books without verification by us.

ACCOUNTANT'S OPINION

In our opinion, except that as noted above the allocation of fund equities was accepted by us without verification, the accompanying balance sheet and related statements of income and expense and cash and disbursements fairly present the financial position of American Laryngological Association at April 1, 1952, and the results of its operations for the year then ended, in conformity with generally accepted accounting principles applied on a basis consistent with that of the preceding year.

McGladrey, Hansen, Dunn & Co.,
Certified Public Accountants.

FINAL EXECUTIVE SESSION

The Final Executive Session was called to order by President Taylor at 12 noon immediately following the Scientific Session.

The Association unanimously elected as Councillors the Fellows recommended by the Nominating Committee in their report. A short recess was held while the new Councillors retired and elected the following Officers:

President.....	Louis H. Clerf
First Vice-President.....	J. Mackenzie Brown
Second Vice-President.....	Edwin N. Broyles
Secretary.....	Harry P. Schenck
Treasurer.....	Gordon F. Harkness
Librarian and Historian.....	Bernard J. McMahon
First Councillor.....	Frederick T. Hill
Second Councillor.....	Ralph A. Fenton
Third Councillor.....	Gordon B. New
Fourth Councillor.....	H. Marshall Taylor

President-elect Clerf accepted the gavel from the Presiding Officer, expressed his thanks to the Fellowship for their aid during his years as Secretary and his gratitude for this great honor that has been conferred upon him.

The meeting was adjourned at 12:15 P. M.

Respectfully submitted,

LOUIS H. CLERF,

Secretary.

List of Fellows

1952

LIST OF FELLOWS

Date Indicates Year Admitted to Active Fellowship

Attendance at meetings is shown by figures following each name.

Asterisk (*) indicates year paper was read.

Active Fellows

1942. ASHLEY, REA E., 384 Post Street, San Francisco 8, California, 1946, '47, '48, '49, '50, '51*.
1936. BALLENGER, HOWARD C., 723 Elm Street, Winnetka, Illinois, 1936, '37, '38*, '40, '41, '46*, '47, '48, '51, '52*.
1949. BECK, AUGUST LEO, 42 Huguenot Street, New Rochelle, N. Y., 1949, '50, '51, '52*.
1938. BLASSINGAME, CHARLES DECATUR, 414 Physicians and Surgeons Bldg., Memphis 3, Tennessee, 1938, '39, '41, '42, '44, '44, '46*, '47, '50, '51, '52*.
1944. BOIES, LAWRENCE R., 1631 Medical Arts Building, Minneapolis 2, Minnesota, 1944, '46, '47, '48*, '49, '50*, '51*.
1941. BOWERS, WESLEY C., 17 East 61st Street, New York 21, N. Y., 1941, '42, '44, '49.
1951. BOYDEN, GUY LEE, 1735 N. Wheeler St., Portland 12, Ore., 1952*.
1932. BROWN, JOHN MACKENZIE, 1136 W. 6th Street, Los Angeles, 14, California, 1935*, '37, '40, '41, '42, '44, '47*, '49, '50, '51, '52.
1940. BROYLES, EDWIN N., 1100 North Charles Street, Baltimore, Maryland, 1940, '41*, '42, '46, '48*, '49, '50, '51*, '52.
1930. CAMPBELL, EDWARD HASTINGS, 133 S. 36th St., Philadelphia 4, Pennsylvania, 1930, '31*, '32*, '33, '35, '38, '39*, '40, '41, '47, '48, '49, '51, '52.
1945. CAMPBELL, PAUL A., School of Aviation Medicine, Randolph Field, Texas, 1946, '47, '49, '51.
1942. CANFIELD, NORTON, 175 St. Ronan St., New Haven 11, Connecticut, 1942, '46, '47, '48, '49*, '50, '51.

1925. CLERF, LOUIS H., 1530 Locust Street, Philadelphia 2, Pennsylvania, 1925, '27*, '28, '29, '30*, '32, '33, '34, '35, '36*, '37, '38, '39, '40, '41*, '42*, '44*, '46, '47, '48, '49, '50, '51, '52.
1939. CODY, CLAUDE CARR, 1304 Walker Avenue, Houston, Texas 1939, '41, '42, '44, '46*, '47, '48*, '49, '50, '51, '52.
1941. CUNNING, DANIEL S., 118 East 53rd Street, New York 22, N. Y., 1941, '42*, '44, '46, '47*, '48, '49, '50*, '51, '52.
1951. DAVISON, FRANCIS W., Geisinger Memorial Hospital, Danville, Pa., 1951, '52.
1941. DIXON, FRED W., 1027 Rose Building, Cleveland, Ohio, 1941, '42*, '44, '46, '47*, '49*, '50, '51, '52.
1947. EAGLE, WATT WEEMS, Duke Hospital, Durham, N. C., 1948*, '49, '51.
1947. ENGLER, CLARENCE W., 2323 Prospect Avenue, Cleveland, Ohio, 1947, '48*, '49, '50, '51.
1952. ERICH, JOHN B., Mayo Clinic, 102 Second Avenue, Rochester, Minn.
1923. FENTON, RALPH ALBERT, 1020 S.W. Taylor Street, Portland 5, Oregon, 1923, '25*, '26, '28, '30, '32, '34, '35, '37*, '39, '40*, '42, '44, '46, '47*, '49, '50*, '52.
1930. FIGI, FREDERICK A., Mayo Clinic, Rochester, Minnesota, 1930, '32*, '34, '35, '38*, '42*, '47*, '48, '50*.
1932. FURSTENBERG, ALBERT CARL, 201 South Main Street, Ann Arbor, Michigan, 1933*, '36*, '38*, '46, '48*, '50, '51.
1936. GALLOWAY, THOMAS CYRUS, 636 Church Street, Evanston, Illinois, 1936, '37, '38*, '39, '40, '41, '42*, '46*, '47, '48, '50, '51, '52.
1940. GATEWOOD, E. TRIBLE, Professional Building, Richmond, Virginia, 1940, '41, '42*, '44, '47.
1940. GILL, WILLIAM D., 705 East Houston Street, San Antonio, Texas, 1940, '41*, '46.
1940. GOODALE, ROBERT L., 330 Dartmouth Street, Boston, Massachusetts, 1940, '41, '42*, '44*, '46, '47, '49, '50*.

1928. GOODYEAR, HENRY M., 556 Doctors' Building, Cincinnati 2, Ohio, 1928, '29, '30*, '31, '32, '33, '34*, '35, '36, '37, '38, '39, '40, '42, '44*, '46, '47, '48, '49*, '51.
1950. HALL, COLBY, 1136 West Sixth Street, Los Angeles, Calif., 1950.
1940. HANSEL, FRENCH K., 634 North Grand Avenue, St. Louis 3, Missouri, 1940, '42, '46, '47, '51.
1933. HARKNESS, GORDON FOLLETTE, 215 Main Street, Davenport, Iowa, 1934, '35, '36*, '40, '41, '44, '46, '47, '48, '49, '50, '51, '52.
1944. HAVENS, FRED Z., 102 Second Avenue S-W., Rochester, Minnesota, 1944, '46, '49, '51.
1942. HEATLEY, CLYDE A., 11 North Goodman Street, Rochester, N. Y., 1942, '46*, '49, '50, '51, '52.
1945. HICKEY, HAROLD L., 1612 Tremont Street, Denver 2, Colorado, 1946, '47, '48, '49, '50, '52.
1943. HILDING, ANDERSON C., 324 West Superior Street, Duluth, Minnesota, 1944*, '46, '47, '48*, '49, '50, '51.
1928. HILL, FREDERICK THAYER, Professional Building, Waterville, Maine, 1928, '29*, '30*, '31, '32, '33, '34, '35, '36, '37*, '38*, '39, '40, '41*, '42, '44*, '46, '47, '48, '49*, '50, '51, '52*.
1945. HOLINGER, PAUL H., 700 North Michigan Ave., Chicago 11, Illinois, 1946, '47, '49, '50*, '51*, '52.
1944. HOOPLE, GORDON D., 1100 East Genesee St., Syracuse, New York, 1946, '47, '48, '49, '50, '51, '52.
1930. HOUSER, KARL MUSSER, 2035 Delancey Street, Philadelphia 3, Pennsylvania, 1930, '32*, '33, '34*, '37, '38, '39, '40, '41, '44, '48.
1952. IRELAND, P. E., Room 82, Banting Institute, University of Toronto, Toronto, Ontario, 1952.
1938. JACKSON, CHEVALIER LAWRENCE, 1901 Walnut, Philadelphia 3, Pa., 1938, '39, '40, '41*, '42, '44*, '46, '47*, '48, '49, '51.

1939. JOHNSTON, WILLIAM HOWARD, 1515 State Street, Santa Barbara 7, California, 1939, '40, '41, '42, '44, '46, '47, '49, '50, '52.
1942. KELLY, JOSEPH D., 140 East Fifty-fourth Street, New York 22, N. Y., 1942, '44*, '47, '48, '49, '51.
1921. KERNAN, JOHN, D., JR., 103 East Seventy-eighth Street, New York 21, N. Y., 1921*, '22, '25, '26, '27, '29, '31, '34*, '35, '36, '37*, '39, '40*, '42*, '47, '48, '51.
1942. KING, EDWARD D., 10220 Riverside Drive, North Hollywood, California, 1942, '46, '47, '49*, '50.
1950. KLINE, ORAM R., 514 Cooper Street, Camden, N. J., 1950, '51, '52.
1936. LEJEUNE, FRANCIS ERNEST, Ochsner Clinic Building, Prytania and Aline Streets, New Orleans 15, Louisiana, 1937, '46*, '47, '50, '51, '52.
1933. LIERLE, DEAN M., University Hospital, Iowa City, Iowa, 1933, '34, '35*, '37, '38, '39, '40*, '42*, '44*, '46, '47*, '48, '49, '50*, '51, '52.
1922. LILLIE, HAROLD I., Mayo Clinic, Rochester, Minnesota, 1923*, '24*, '25, '26*, '28, '31*, '33, '36*, '37, '38, '40, '44, '46*, '47, '48*, '49*.
1949. LINDSAY, JOHN R., 950 E. 59th Street, Chicago 37, Illinois, 1949, '50, '51*.
1935. LOOPER, EDWARD A., 104 West Madison Avenue, Baltimore 1, Maryland, 1935, '36, '37, '38*, '39, '41, '42, '44, '47, '48*, '49, '51.
1927. LUKENS, ROBERT M., 1308 Hunting Park Avenue, Philadelphia 40, Pennsylvania, 1927, '28, '31*, '32, '35, '36, '38, '39, '40*, '42, '46, '48*, '49, '51.
1939. LUPTON, IRVING MARTIN, 806 Medical Arts Building, Portland, Oregon, 1946, '50.
1952. LYNCH, MERCER G., Ochsner Clinic, Prytania and Aline Sts., New Orleans 15, La.
1928. MACPHERSON, DUNCAN, 920 Fifth Avenue, New York City 21, N. Y., 1928, '29, '30, '31, '33, '34*, '39, '40, '41, '44, '49, '51.

1941. MARTIN, ROBERT C., 384 Post Street, San Francisco 8, California, 1941, '44, '47, '48, '50, '51.
1948. MAXWELL, JAMES H., 201 S. Main Street, Ann Arbor, Michigan, 1948, '49, '50*, '51*, '52.
1952. MCCALL, JULIUS W., 610 Rose Building, Cleveland 15, Ohio, 1952.
1951. MCCART, HOWARD W. D., Medical Arts Bldg., Toronto, Canada, 1951, '52*.
1939. MCCASKEY, CARL HEBER, 608 Guaranty Building, Indianapolis 4, Indiana, 1939, '40, '41, '42*, '44, '46*, '47, '48, '49, '50, '51, '52.
1951. MCHENRY, LAWRENCE C., 117 North Broadway, Oklahoma City 2, Okla.
1945. MCLAURIN, JOHN G., 1322 Medical Arts Building, Dallas 1, Texas, 1946, 49.
1933. MCMAHON, BERNARD J., Missouri Theatre Building, St. Louis 3, Missouri, 1934*, '35, '36, '38*, '41, '44, '46*, '47, '48, '49, '50*, '51, '52.
1931. McNALLY, WILLIAM JAMES, 1509 Sherbrooke Street West, Montreal, Canada, 1931, '33, '34*, '39, '44, '47, '49, '49, '52.
1952. MILLER, ALDEN H., 500 South Lucas Ave., Los Angeles 17, Calif., 1952.
1950. MORRISON, LEWIS F., 3536 Jackson Street, San Francisco 18, California, 1950, '51, '52*.
1940. MORRISON, WILLIAM W., 140 E. 54th Street, New York 22, N. Y., 1940, '41*, '44, '47, '49*, '50, '52.
1927. MYERSON, MERVIN C., 416 North Bedford Drive, Beverly Hills, California, 1927 '28*, '29, '30, '31*, '32*, '33*, '34*, '35, '36, '37, '38, '39*, '40, '41*, '42, '44, '50*.
1937. NASH, C. STEWART, Medical Arts Building, Rochester 7, New York, 1937, '38*, '40, '42, '44, '46, '49, '50, '51, '52.

1922. NEW, GORDON B., 102 Second Avenue, Southwest, Rochester, Minnesota 1922, '24, '25*, '26*, '28*, '31*, '33*, '35*, '37*, '39*, '41*, '46*, '48*, '49*, '51*.
1927. ORTON, HENRY BOYLAN, 224 Delavan Avenue, Newark 4, New Jersey, 1927, '28, '29, '30*, '31, '32, '33*, '35, '36, '37, '38*, '40, '41, '42, '44*, '46, '47*, '49, '51*, '52.
1948. PHELPS, KENNETH A., 1137 Medical Arts Building, Minneapolis 2, Minnesota, 1948, '50.
1951. PRIEST, ROBERT E., 79, S. Ninth St., Minneapolis 2, Minn., 1951.
1924. PROETZ, ARTHUR WALTER, 1010 Beaumont Building, St. Louis 8, Missouri, 1924, '25*, '26*, '27, '28, '29, '30, '31, '32*, '33*, '34, '35*, '36, '37, '38*, '39*, '40, '41, '42*, '44, '46, '47*, '48*, '49*, '50, '51*, '52.
1951. PUTNEY, F. JOHNSON, 255 South 17th Street, Philadelphia 3, Pa., 1951, '52.
1951. RAWLINS, AUBREY G., 384 Post Street, San Francisco, Calif., 1951.
1934. RICHARDS, LYMAN GILDER, 1101 Beacon Street, Brookline 46, Massachusetts, 1934, '35, '36*, '37, '38, '39, '47, '49, '51*.
1945. ROBB, JAMES M., 641 David Whitney Building, Detroit, Michigan, 1946, '48, '49*, '51, '52.
1938. SALINGER, SAMUEL, 25 E. Washington Street, Chicago, Illinois, 1938, '39, '40*, '42, '44*, '47, '48, '49, '50, '51, '52*.
1921. SAUER, WILLIAM E., 3720 Washington Boulevard, St. Louis 8, Missouri, 1921, '23, '25, '28, '31, '32, '35, '37, '39*, '41, '44, '47, '50.
1932. SCHALL, LEROY ALLEN, 243 Charles Street, Boston 14, Massachusetts, 1934*, '35, '38, '40*, '41, '42, '44*, '46, '47, '48*, '49, '50*, '51.
1934. SCHENCK, HARRY P., 326 South 19th Street, Philadelphia 3, Pennsylvania, 1934, '35, '36*, '37, '38, '39, '40, '41*, '44*, '46, '47, '49, '50, '51, '52.

1930. SEYDELL, ERNEST M., First National Bank Building, Wichita, Kansas, 1930, '31*, '33*, '34, '35, '37*, '38, '39, '40*, '41, '42, '46, '47, '48, '51.
1928. SHEA, JOHN JOSEPH, 1018 Madison Avenue, Memphis, Tennessee, 1928, '29, '30*, '31, '32*, '33, '34, '35, '36, '37, '38, '40, '41*, '42, '46, '47*, '48, '49, '50, '51*, '52.
1939. SIMPSON, W. LIKELY, 512 Physicians' and Surgeons' Building, Memphis 3, Tennessee, 1939, '41, '44*, '46, '47*, '48, '50, '51*, '52.
1950. SMITH, AUSTIN T., 1830 Spruce St., Philadelphia 3, Pa., 1950, '51, '52.
1924. TAYLOR, HERMON MARSHALL, 111 West Adams Street, Jacksonville 5, Florida, 1924, '25*, '26, '27, '28, '31, '34, '35*, '36, '37, '38, '39*, '40, '41, '42, '44*, '46, '47, '48, '49, '50*, '51, '52*.
1947. THEOBALD, WALTER H., 307 N. Michigan Avenue, Chicago, Illinois, 1947, '48*, '49, '50, '51, '52.
1927. TOBEY, HAROLD G., 403 Commonwealth Avenue, Boston 15, Massachusetts, 1927, '29*, '30*, '31, '32, '35, '37, '39, '41, '46, '48, '49.
1950. TREMBLE, G. EDWARD, 1390 Sherbrooke St. W., Montreal, Quebec, Canada, 1950, '51, '52.
1925. TUCKER, GABRIEL, 250 South Eighteenth Street, Philadelphia 3, Pennsylvania, 1925, '26*, '27*, '29, '30, '31, '32, '33, '34, '35*, '36, '37, '38, '39, '40*, '41*, '42, '46, '47, '48, '49, '50, '51.
1943. VAN ALYEA, OLIVER E., 135 South LaSalle Street, Chicago, Illinois, 1944, '46*, '47, '48, '49, '50, '51, '52*.
1941. VIOLÉ, PIERRE P., 1930 Wilshire Boulevard, Los Angeles 5, California, 1944*, '46, '47, '50, '52.
1940. WEILLE, FRANCIS L., 247 Commonwealth Avenue, Boston 16, Massachusetts, 1940, '41, '42, '44, '46*, '47, '48, '49, '50, '51, '52.
1948. WHALEN, EDWARD J., 750 Main Street, Hartford, Connecticut, 1948, '50, '51, '52.

1948. WILLIAMS, HENRY L., 2nd, Mayo Clinic, Rochester, Minnesota, 1948, '49, '50*, '51, '52*.
1942. WISHART, D. E. STAUNTON, 170 St. George Street, Toronto, Canada, 1942, '44, '46, '47*, '49, '51, '52*.
1951. WOODMAN, DEGRAAF, 156 E. 37th Street, New York 16, N. Y., 1951.

Honorary Fellows

1949. (1917) COATES, GEORGE M., 1721 Pine Street, Philadelphia 3, Pa., 1949*, '51, '52.
1939. (1907) JACKSON, CHEVALIER, Schwenksville, Pennsylvania, 1939*, '40*.
1943. (1918) LEWIS, FIELDING O., Providence Road, Media, Pennsylvania.
1947. (1904) MOSHER, HARRIS P., 127 Front Street, Marblehead, Mass., 1947, '48, '49, '50*, '51, '52.
1937. NAGER, F. B., Freiestrasse 3, Zurich, Switzerland.

Corresponding Fellows

1946. ALONSO, JUSTO M., Mercedes 1233, Montevideo, Uruguay.
1942. BATSON, OSCAR V., 133 South Thirty-sixth Street, Philadelphia 4, Pennsylvania, 1942*.
1938. BLAIR, VILRAY P., 508, North Grand Avenue, St. Louis 3, Missouri.
1943. EGGSTON, ANDREW O., 653 Park Avenue, New York, N. Y., 1944, '49.
1950. LARSELL, OLOF, University of Oregon Medical School, Portland, Oregon, 1950*.
1921. LE MAITRE, FERNAND, Avenue Victor Hugo, 120 (XVI), Paris, France.
1930. NEGUS, VICTOR E., 149 Harley Street, London, W 1, England, 1938*.
1950. NEIL, JAMES HARDIE, 64 Symonds Street, Auckland, New Zealand.

1937. PORTMANN, GEORGES, 25 bis Cours de Verdun, Bordeaux, France, 1947.
1924. SCHAEFER, J. PARSONS, 4634 Spruce Street, Philadelphia 3, Pennsylvania, 1934.
1952. TAPIA ACUNA, RICARDO, Bucareli 82, Mexico 6, D. F.
1936. VIALLE, JACQUES, 27 Rue de France, Nice (Alpes Maritimes), France.

Emeritus Fellows

1941. (1923) BARLOW, ROY A., Charles Town, West Virginia.
1937. (1915) BARNES, HARRY ALDRICH, Kingston, Mass., R.F.D. No. 2.
1951. (1944) BEATTY, HUGH G., 150 E. Broad St., Columbus 15, Ohio, 1951, '52.
1950. (1921) BERRY, GORDON, 36 Pleasant Street, Worcester, Mass., 1951.
1944. (1913) BUTLER, RALPH, 1930 Chestnut Street, Philadelphia, Pennsylvania.
1950. (1937) CROWE, SAMUEL J., The Johns Hopkins Hospital, Baltimore 5, Md.
1946. (1933) FOSTER, JOHN H., Eagle Lake, Texas, 1948, '49, '50, '51, '52.
1951. (1928) GITTINS, THOMAS R., 408 Davidson Bldg., Sioux City, Iowa.
1936. (1898) GOODALE, JOSEPH L., Argilla Road, Ipswich, Massachusetts.
1943. (1932) GRAHAM, HARRINGTON B., Bohemian Club, San Francisco 2, California.
1933. (1905) HALSTED, THOMAS H., 40 Trinity Place, Boston 16, Mass., 1949.
1951. (1928) JARVIS, DEFOREST C., Quarry Bank Bldg., Barre, Vt., 1952.

1950. (1928) LYMAN, HARRY WEBSTER, 906 Carleton Bldg., St. Louis 1, Mo., 1950, '51, '52.
1948. (1939) MYERS, JOHN LLEWELLYN, 1115 Grand Avenue, Kansas City, Missouri.
1940. (1923) SEWALL, EDWARD C., 430 El Escarpado, Stanford University, California.
1948. (1923) SPENCER, FRANK R., 429 Pine St., Boulder, Colorado.
1950. (1922) WHITE, FRANCIS W., 471 Park Avenue, New York 22, N. Y.
1946. (1905) WOOD, GEORGE B., 329 Hathaway Lane, Wynnewood, Pennsylvania.
1951. (1935) WOODWARD, FLETCHER D., 104 E. Market St., Charlottesville, Va.

BENEFACTORS

DR. ARTHUR W. DEROALDES

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DR. JOHN F. BARNHILL
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DR. LOUIS H. CLERF
MR. LESTER T. SUNDERLAND
DR. WILLIAM E. GROVE



Obverse of the deRoaldes medal

RECIPIENTS OF THE DEROALDES AWARD

- 1928. DR. CHEVALIER JACKSON
- 1931. DR. D. BRYSON DELAVAN
- 1934. DR. HARRIS P. MOSHER
- 1937. DR. LEE WALLACE DEAN
- 1943. DR. RALPH A. FENTON
- 1949. DR. GEORGE M. COATES
- 1951. DR. ARTHUR W. PROETZ

RECIPIENTS OF THE CASSELBERRY AWARD

1923. DR. GEORGE FETTEROLF AND DR. HERBERT FOX
1928. DR. RALPH A. FENTON AND DR. O. LARSELL
1929. DR. RICHARD A. KERN AND DR. HARRY P. SCHENCK
1929. DR. EDWARD H. CAMPBELL
1931. DR. ARTHUR W. PROETZ
1934. DR. ANDERSON C. HILDING
1936. DR. FRANCIS E. LEJEUNE: DR. JOEL J. PRESSMAN
1939. DR. H. MARSHALL TAYLOR: DR. BRIEN T. KING
1940. DR. FRENCH K. HANSEL
1941. DR. NOAH D. FABRICANT
1946. DR. PAUL H. HOLINGER
1949. DR. HENRY B. ORTON

RECIPIENTS OF THE NEWCOMB AWARD

- 1941. DR. BURT R. SHURLY
- 1942. DR. FRANCIS R. PACKARD
- 1943. DR. GEORGE M. COATES
- 1944. DR. CHARLES J. IMPERATORI
- 1947. DR. HARRIS P. MOSHER
- 1948. DR. GORDON BERRY
- 1949. DR. GORDON B. NEW
- 1950. DR. H. MARSHALL TAYLOR
- 1951. DR. JOHN D. KERNAN
- 1952. DR. WILLIAM J. McNALLY

PAST OFFICERS

Presidents

(Year of Election)

1879.	Louis Elsberg	1909.	J. E. Logan
1880.	J. Solis-Cohen	1910.	D. Braden Kyle
1881.	F. I. Knight	1911.	James E. Newcomb
1882.	G. M. Lefferts	1912.	George A. Leland
1883.	F. H. Bosworth	1913.	Thomas Hubbard
1884.	E. L. Shurly	1914.	Alexander W. MacCoy
1885.	Harrison Allen	1915.	G. Hudson Makuen
1886.	E. Fletcher Ingals	1916.	Joseph L. Goodale
1887.	R. P. Lincoln	1917.	Thomas H. Halstead
1888.	E. C. Morgan	1918.	Cornelius G. Coakley
1889.	J. N. Mackenzie	1919.	Norval H. Pierce
1890.	W. C. Glasgow	1920.	Harris P. Mosher
1891.	S. W. Langmaid	1921.	Harmon Smith
1892.	M. J. Asch	1922.	Emil Mayer
1893.	D. Bryson Delavan	1923.	J. Payson Clark
1894.	J. O. Roe	1924.	Lee Wallace Dean
1895.	W. H. Daly	1925.	Greenfield Sluder
1896.	C. H. Knight	1926.	Chevalier Jackson
1897.	T. R. French	1927.	D. Bryson Delavan
1898.	W. E. Casselberry	1928.	Charles W. Richardson
1899.	Samuel Johnston	1929.	Lewis A. Coffin
1900.	H. L. Swain	1930.	Francis R. Packard
1901.	J. W. Farlow	1931.	George E. Shambaugh
1902.	J. H. Bryan	1932.	George Fetterolf
1903.	J. H. Hartman	1933.	George M. Coates
1904.	C. C. Rice	1934.	Dunbar Roy
1905.	J. W. Gleitsman	1935.	Burt R. Shurly
1906.	A. W. deRoaldes	1936.	William B. Chamberlain
1907.	H. S. Birkett	1937.	John F. Barnhill
1908.	A. Coolidge, Jr.	1938.	George B. Wood

- | | |
|-------------------------------|--------------------------|
| 1939. James A. Babbitt | 1944-5. Harold I. Lillie |
| 1940. Gordon Berry | 1946. Frank R. Spencer |
| 1941. Thomas E. Carmody | 1947. Arthur W. Proetz |
| 1942-3. Charles J. Imperatori | 1948. Frederick T. Hill |
| 1949. Ralph A. Fenton | 1950. Gordon B. New |
| | 1951. H. Marshall Taylor |

Vice-Presidents

- | | |
|------------------------|--------------------|
| 1879. F. H. Davis | J. O. Roe |
| 1880. W. C. Glasgow | W. Porter |
| 1881. E. L. Shurly | E. F. Ingals |
| 1882. C. Seiler | S. Johnston |
| 1883. S. W. Langmaid | W. H. Daly |
| 1884. J. H. Hartman | G. W. Major |
| 1885. H. A. Johnson | J. N. Mackenzie |
| 1886. E. C. Morgan | S. W. Langmaid |
| 1887. J. N. Mackenzie | C. E. DeM. Sajous |
| 1888. W. C. Glasgow | C. E. Bean |
| 1889. E. Holden | J. H. Hartman |
| 1890. J. O. Roe | S. Johnston |
| 1891. M. J. Asch | J. C. Mulhall |
| 1892. S. Johnston | W. E. Casselberry |
| 1893. J. C. Mulhall | S. H. Chapman |
| 1894. C. C. Rice | A. W. deRoaldes |
| 1895. J. Wright | D. N. Rankin |
| 1896. T. M. Murray | H. S. Birkett |
| 1897. A. W. MacCoy | F. W. Hinkel |
| 1898. J. W. Farlow | M. R. Brown |
| 1899. T. A. DeBlois | A. A. Bliss |
| 1900. H. L. Wagner | D. Braden Kyle |
| 1901. J. W. Gleitsmann | T. Melville Hardie |
| 1902. G. A. Leland | W. Peyre Porcher |
| 1903. J. H. Lowman | W. J. Freeman |
| 1904. Thomas Hubbard | C. W. Richardson |
| 1905. J. L. Goodale | |

1906. G. H. Makuen	A. B. Thrasher
1907. J. P. Clark	J. E. Rhodes
1908. E. Mayer	F. R. Packard
1909. C. G. Coakley	H. P. Mosher
1910. Robert C. Myles	J. M. Ingersoll
1911. F. C. Cobb	B. R. Shurly
1912. A. W. Watson	W. Scott Renner
1913. F. E. Hopkins	Geo. F. Shambaugh
1914. Clement T. Theisen	Lewis A. Coffin
1915. J. Gordon Wilson	Christian R. Holmes
1916. Thomas H. Halsted	Greenfield Sluder
1917. John Edwin Rhodes	D. Crosby Greene
1918. G. E. Shambaugh	John R. Winslow
1919. Francis R. Packard	Harmon Smith
1920. Harmon Smith	W. B. Chamberlin
1921. Dunbar Roy	Robert C. Lynch
1922. George Fetterolf	Lorenzo B. Lockard
1923. Hubert Arrowsmith	Joseph B. Greene
1924. Ross H. Skillern	Gordon Berry
1925. John E. Mackenty	Robert Levy
1926. Lewis A. Coffin	William V. Mullin
1927. Charles W. Richardson	Hill Hastings
1928. Robert Clyde Lynch	Francis P. Emerson
1929. William B. Chamberlin	Ralph Albert Fenton
1930. Harris P. Mosher	James A. Babbitt
1931. Joseph B. Greene	E. Ross Faulkner
1932. Gordon Berry	Frank A. Spencer
1933. E. Ross Faulkner	Thomas S. Carmody
1934. Gordon B. New	Samuel McCullagh
1935. Edward C. Sewall	H. Marshall Taylor
1936. William P. Wherry	Harold I. Lillie
1937. Frank R. Spencer	Bernard J. McMahon
1938. Ralph A. Fenton	Frederick T. Hill
1939. John H. Foster	Thomas R. Gittins
1940. Charles H. Porter	Gordon F. Harkness

1941. Arthur W. Proetz	Henry B. Orton
1942-3. Harold I. Lillie	Dean M. Lierle
1944-5. John J. Shea	Thomas C. Galloway
1946. H. Marshall Taylor	C. Stewart Nash
1947. John J. Shea	Frederick A. Figi
1948. Henry B. Orton	Anderson C. Hilding
1949. LeRoy A. Schall	Fletcher D. Woodward
1950. W. Likely Simpson	Lyman G. Richards
1951. Wm. J. McNally	Thomas C. Galloway

Secretaries and Treasurers

1879. G. M. Lefferts	1895. H. L. Swain
1882 D. Bryson Delavan	1900. P. E. Newcomb
1889. C. H. Knight	1911. Harmon Smith

Secretaries

1912. Harmon Smith	1935. James A. Babbitt
1918. D. Bryson Delavan	1939. Charles J. Imperatori
1919. J. M. Ingersoll	1942. Arthur W. Proetz
1920. George M. Coates	1947. Louis H. Clerf
1933. William V. Mullin	1952. Harry P. Schenck

Treasurers

1912. J. Payson Clark	1933. James A. Babbitt
1923. George Fetterolf	1935. Charles J. Imperatori
1932. William V. Mullin	1939. Frederick T. Hill
1948. Gordon F. Harkness	

Librarians

1789. F. H. Bosworth	1930. John F. Barnhill
1883. T. R. French	1934. Burt R. Shurly
1903. J. H. Bryan	1935. George M. Coates

Librarian and Historian

1936. George M. Coates	1947. Harry P. Schenck
1944. Louis H. Clerf	1952. Bernard J. McMahon

PAST FELLOWS

Dates indicate original election to the Association

**Deceased*

Honorary Fellows

- *1908. Barnhill, John F., Miami Beach, Florida.
- *1893. Birkett, Herbert S., Montreal, Canada.
- *1878. Bosworth, Francke H., New York City.
- *1918. Dean, Lee Wallace, St. Louis, Mo.
- *1881. Delavan, D. Bryson, New York City.
- *1893. deRoaldes, Arthur W., New Orleans, La.
- *1890. De La Sota Y. Lastra, Ramon, Seville, Spain.
- *1879. French, Thomas R., Brooklyn, N. Y.
- *1880. Garcia, Senior Manuel, London, England.
- *1903. Harris, Thos. J., New York City.
- *1878. Johnston, Samuel, Baltimore, Md.
- *1878. Lefferts, George Morewood, Katonah, N. Y.
- *1914. Levy, Robert, Denver, Colo.
- *1883. Mackenzie, John N., Baltimore, Md.
- *1881. Mackenzie, Sir Morel, London, England.
- *1910. Masser, Ferdinand, Naples, Italy.
- \$1910. Moure, J. J. E., Bordeaux, France.
- *1918. Oliver, H. K., Boston, Mass.
- *1906. Pierce, Norval Harvey, San Diego, Calif.
- *1909. Semon, Sir Felix, Great Missenden, England.
- *1878. Solis-Cohen, J., Philadelphia, Pa.
- *1889. Swain, Henry L., New Haven, Conn.
- *1914. Thomson, Sir St. Clair, London, Eng.
- *1903. Tilley, Herbert, London, England.
- *1914. Wagner, Clinton, New York City.
- *1890. Wright, Jonathan, Pleasantville, N. Y.

Corresponding Fellows

- *1892. Browne, Lennox, London, England.
- *1940. Colledge, Lionel, London, England.
- *1901. Collier, Mayo, Kearsney Abbey, Kent, England.
- *1893. Desvernine, Carlos M., Havana, Cuba.
- *1930. Emerson, Francis P., Franklin, Mass.

- *1936. Fraser, John S., Edinburgh, Scotland.
- *1901. Grant, Sir James Dundas, London, England.
- *1887. Cougenheim, A., Paris, France.
- *1894. Holden, Edgar, Newark, N. J.
- *1919. Kelly, Adam Brown, Helensburgh, Scotland.
- *1881. Labus, Carlo, Milan, Italy.
- *1931. LasSagna, Francesco, Parma, Italy.
- *1926. Law, Frederick M., New York, N. Y.
- *1902. Lermoyez, Marcel, Paris, France.
- *1897. Luc, H., Paris, France.
- *1896. MacDonald, Greville, Haslemere, Surrey, England.
- *1894. MacIntyre, John, Glasgow, Scotland.
- *1903. McBride, P., York, England.
- *1920. McKenzie, Dan, London, England.
- *1919. McKernon, James F., New Canaan, Conn.
- *1880. Meyer, Wilhelm, Copenhagen, Denmark.
- *1896. Mygind, Holger, Copenhagen, Denmark.
- *1919. Paterson, Donald Rose, Cardiff, Wales.
- *1941. Patterson, Norman, Herts, England.
- *1919. Rogers, John, Jr., New York City.
- *1894. Sajous, C. E. DeM., Philadelphia, Pa.
- *1896. Schmiegelow, Ernst, Copenhagen, Denmark.
- *1946. Segura, Eliseo, Buenos Aires, Argentina.
- *1940. Soto, E. Fernandez, Havana, Cuba.
- *1881. Thornton, Pugin, London, England.
- *1913. Turner, A. Logan, Edinburgh, Scotland.
- *1880. Whistler, W. McNeil, London, England.
- *1901. Wingrave, Wyatt, Lyme Regis, Dorset, England.
- *1894. Wolfenden, R. Norris, Kent, England.

Emeritus Fellows

- *1928. Beck, Joseph C., Chicago, Illinois.
- *1901. Brown, J. Price, Toronton, Canada.
- *1891. Bryan, Joseph H., Washington, D. C.
- *1897. Clark, J. Payson, Boston, Mass.
- *1899. Cobb, Frederick C., Bradenton, Florida.
- *1905. Coffin, Lewis A., New York City.
- *1893. Coolidge, Algernon, Boston, Mass.
- *1913. Dabney, Virginius, Washington, D. C.
- *1882. De Blois, Thomas Amory, Boston, Mass.

- *1892. Farlow, John W., Boston, Mass.
- *1922. Forbes, Henry H., New York City.
- *1905. Freer, Otto T., Chicago, Ill.
- *1880. Gleitsmann, Joseph W., New York City.
- *1922. Goldsmith, Perry G., Toronto, Canada.
- *1906. Greene, D. Crosby, Jr., Boston, Mass.
- *1917. Greene, Joseph B., Asheville, N. C.
- *1896. Hardie, Thomas Melville, Chicago, Ill.
- *1915. Hastings, Hill, Los Angeles, Calif.
- *1888. Hinkel, Frank Whitehill, Buffalo, New York.
- *1895. Hopkins, Frederick E., Springfield, Mas.
- *1927. Hubbard, Thomas, Toledo, Ohio.
- *1919. Hurd, Lee Maidment, Rowayton, Conn.
- *1920. Imperatori, Charles J., Essex, N. Y.
- *1904. Ingersoll, John Marvin, Miami, Fla.
- *1918. Kenyon, Elmer L., Chicago, Ill.
- *1929. Kistner, Frank B., Portland, Oregon.
- *1885. Knight, Charles H., New York City.
- *1939. Large, Secord H., Cleveland, Ohio.
- *1894. Leland, George A., Boston, Mass.
- *1943. Lincoln, William R., Cleveland, Ohio.
- *1886. MacCoy, Alexander W., Philadelphia, Pa.
- *1896. Mayer, Emil, New York City.
- *1943. McCullagh, Samuel, New York, N. Y.
- *1923. McKinney, Richmond, Memphis, Tenn.
- *1878. Porter, William, Ocean Springs, Miss.
- *1901. Myles, Robert C., New York City.
- *1923. Newhart, Horace, Minneapolis, Minn.
- *1903. Packard, Francis R., Philadelphia, Pa.
- *1942. Potts, John B., Omaha, Nebr.
- *1903. Renner, W. Scott, Buffalo, N. Y.
- *1897. Rhodes, John Edwin, Chicago, Ill.
- *1884. Rice, Clarence C., New York City.
- *1905. Richards, George L., South Yarmouth, Mass.
- *1878. Robinson, Beverly, New York City.
- *1907. Shambaugh, George E., Chicago, Ill.
- *1908. Smith, Harmon, New York City.
- *1892. Wagner, Henry L., San Francisco, Calif.
- *1892. Watson, Arthur W., Philadelphia, Pa.
- *1939. Wilson, J. Gordon, Old Bennington, Vt.

Active Fellows

- *1880. Allen, Harrison, Philadelphia, Pa.
- *1917. Arrowsmith, Hubert, Brooklyn, N. Y.
- *1879. Asch, Morris J., New York City.
- *1923. Babbitt, James A., Philadelphia, Pa.
- *1906. Ballenger, William L., Chicago, Ill.
- *1880. Bean, C. E., St. Paul, Minn.
- *1904. Berens, T. Passmore, New York City.
- *1924. Bigelow, Nolton, Providence, R. I.
- *1893. Bliss, Arthur Ames, Philadelphia, Pa.
- *1895. Boylan, J. E., Cincinnati, Ohio.
- *1892. Brown, Moreau R., Chicago, Ill.
- *1933. Buckley, Robert E., New York City.
- *1915. Canfield, R. Bishop, Ann Arbor, Mich.
- *1934. Carmack, John Walter, Indianapolis, Ind.
- *1924. Carmody, Thomas E., Denver, Colo.
- *1889. Casselberry, William E., Chicago, Ill.
- *1883. Chamberlain, C. W., Hartford, Conn.
- *1917. Chamberlin, William B., Cleveland, Ohio.
- *1896. Chappell, W. F., New York City.
- *1882. Chapman, S. Hartwell, New Haven, Conn.
- *1902. Coakley, Cornelius G., New York City.
- *1913. Coffin, Rockwell C., Boston, Mass.
- 1918. Cox, Gerald H., New York City.
- *1880. Cushing, E. W., Boston, Mass.
- *1878. Cutter, Ephraim, West Falmouth, Mass.
- *1880. Daly, W. H., Pittsburgh, Pa.
- *1878. Davis, F. H., Chicago, Ill.
- *1941. Davis, Warren B., Philadelphia, Pa.
- 1926. Dennis, Frank Lownes, Colorado Springs, Col.
- *1901. Dickerman, E. T., Chicago, Ill.
- *1878. Donaldson, Frank, Baltimore, Md.
- *1935. Eves, Curtis C., Philadelphia, Pa.
- *1919. Faulkner, E. Ross, New York City.
- *1914. Fetterolf, George, Philadelphia, Pa.
- *1901. Freeman, Walter J., Philadelphia, Pa.
- *1917. Friedberg, Stanton A., Chicago, Ill.
- 1940. Fuchs, Valentine H., New Orleans, La.
- 1909. Getchell, Albert C., Worcester, Mass.
- *1897. Frothingham, Richard, New York City.

- *1907. Gibb, Joseph S., Philadelphia, Pa.
- *1878. Glasgow, William Carr, St. Louis, Mo.
- *1913. Goldstein, Max A., St. Louis, Mo.
- *1905. Grayson, Charles P., Philadelphia, Pa.
- *1934. Grove, William E., Milwaukee, Wisconsin.
- *1879. Holden, Edgar, Newark, N. J.
- *1907. Holmes, Christian R., Cincinnati, Ohio.
- *1882. Hooper, Franklin H., Boston, Mass.
- *1893. Hope, George B., New York City.
- *1938. Hourn, George E., St. Louis, Mo.
- *1939. Hunt, Westley Marshall, New York, N. Y.
- *1901. Hyatt, Frank, Washington, D. C.
- *1925. Iglaue, Samuel, Cincinnati, Ohio.
- *1878. Ingals, E. Fletcher, Chicago, Ill.
- *1882. Ives, Frank L., New York City.
- *1880. Jarvis, William C., New York City.
- *1878. Johnson, Hosmer A., Chicago, Ill.
- *1879. Johnson, Woolsey, New York City.
- *1944. Jones, Marvin F., New York City.
- *1879. Kealhofer, R. H., St. Louis, Mo.
- *1901. King, Gordon, New Orleans, La.
- *1885. Knight, Charles H., New York City.
- *1878. Knight, Frederick Irving, Boston, Mass.
- *1898. Kyle, D. Braden, Philadelphia, Pa.
- *1880. Langmaid, Samuel W., Boston, Mass.
- 61878. Lincoln, Rufus P., New York City.
- *1911. Lockard, Lorenzo B., Denver, Colo.
- *1913. Loeb, Hanau W., St. Louis, Mo.
- *1897. Logan, James E., Kansas City, Mo.
- *1888. Lowman, John H., Cleveland, Ohio.
- *1919. Lynah, Henry L., New York City.
- *1915. Lynch, Robert Clyde, New Orleans, La.
- *1879. McBurney, Charles, New York City.
- *1927. McGinnis, Edwin, Chicago, Ill.
- 1936. McGregor, Gregor, Toronto, Canada.
- *1913. McKimmie, O. A., Washington, D. C.
- *1885. McSherry, H. Clinton, Baltimore, Md.
- *1914. Mackenty, John E., New York City.
- *1898. Makuen, G. Hudson, Philadelphia, Pa.
- *1881. Major, G. W., Montreal, Canada.
- *1881. Morgan, E. C., Washington, D. C.

- *1886. Mulhall, J. C., St. Louis, Mo.
- *1925. Mullin, William V., Cleveland, Ohio.
- *1914. Munger, Carl E., Waterbury, Conn.
- *1892. Murray, T. Morris, Washington, D. C.
- *1881. Mynter, H., Buffalo, N. Y.
- *1893. Newcomb, James E., New York City.
- *1895. Nichols, J. E. H., New York City.
- *1894. Park, William H., New York City.
- *1892. Porcher, W. Peyre, Charleston, S. C.
- *1927. Porter, Charles T., Boston, Mass.
- *1908. Randall, B. Alexander, Philadelphia, Pa.
- *1882. Rankin, D. N., Allegheny, Pa.
- *1902. Richardson, Charles W., Washington, D. C.
- *1930. Ridpath, Robert E., Philadelphia, Pa.
- *1879. Roe, John O., Rochester, N. Y.
- *1881. Robertson, J. M., Detroit, Mich.
- *1913. Roy, Dunbar, Atlanta, Ga.
- *1878. Rumbold, T. F., St. Louis, Mo.
- *1879. Seiler, Carl, Philadelphia, Pa.
- *1893. Shields, Charles M., Richmond, Pa.
- *1909. Shurly, Burt R., Detroit 1, Mich.
- *1878. Shurly, E. L., Detroit, Mich.
- *1892. Simpson, William Kelly, New York City.
- *1919. Skillern, Ross H., Philadelphia, Pa.
- *1909. Sluder, Greenfield, St. Louis, Mo.
- *1879. Smith, Andrew H., Geneva, N. Y.
- *1932. Smyth, Duncan Campbell, Boston, Mass.
- *1928. Sonnenschein, Robert, Chicago, Ill.
- 1924. Stein, Otto J., Chicago, Ill.
- 1934. Stevenson, Walter, Quincy, Ill.
- *1911. Staut, George C., Philadelphia, Pa.
- *1879. Tauber, Bernhard, Cincinnati, Ohio.
- *1903. Theisen, Clement F., Albany, N. Y.
- *1899. Thorner, Max, Cincinnati, Ohio.
- *1892. Thrasher, Allen B., Cincinnati, Ohio.
- *1937. Tobey, George L., Jr., Boston, Mass.
- *1888. Van der Poel, S. O., New York City.
- *1938. Vail, Harris H., Cincinnati, Ohio.
- *1936. Voislowsky, Antonie P., New York City.
- *1933. Wanamaker, Allison T., Seattle, Wash.
- 1896. Ward, Marshall R., Pittsburgh, Pa.

- *1879. Ward, Whitfield, New York City.
- *1886. Westbrook, Benjamin F., Brooklyn, N. Y.
- *1924. Wherry, William P., Omaha, Neb.
- *1924. White, Leon E., Boston, Mass.
- *1939. Williams, Horace J., Philadelphia, Pa.
- *1922. Wishart, David J. G., Toronto, Ontario.
- *1940. Wood, V. Visscher, St. Louis, Mo.
- *1896. Wollen, Green V., Indianapolis, Ind.

