















Transactions  
American Laryngological Association

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Sixtieth Volume







TRANSACTIONS  
OF THE  
SIXTIETH ANNUAL MEETING  
OF THE  
American  
Laryngological Association

HELD AT ATLANTIC CITY, NEW JERSEY  
MAY 2, 3 AND 4, 1938

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TRANSACTIONS OF THE SIXTIETH ANNUAL  
CONGRESS  
OF THE  
AMERICAN LARYNGOLOGICAL ASSOCIATION

HELD AT

ATLANTIC CITY, NEW JERSEY, MAY 2, 3 AND 4, 1938

DR. JOHN F. BARNHILL, President of the American Laryngological Association, called the Sixtieth Annual Congress to order at The Marlborough-Blenheim, Atlantic City, New Jersey, at 10 A.M., May 2, 1938.

REGISTRATION

Registration for the meeting was as follows:

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TOBEY, GEORGE L., JR.  
TUCKER, GABRIEL  
VAIL, HARRIS H.  
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WATSON, ARTHUR W.  
WHERRY, WILLIAM P.  
WILSON, J. GORDON  
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WOODWARD, FLETCHER D.



## PRESIDENT'S ADDRESS

JOHN F. BARNHILL, M.D.

FOR the sixtieth time the president of the American Laryngological Association says "Thank You" to the Fellows who have honored him by electing him to its highest office.

Eighteen seventy-nine to nineteen thirty-eight—sixty years! The period just about covers the life of our specialty. Before 1879 those suffering from diseases of the upper air tract were treated by the general physician or surgeon into whose hands they fell.

Looking at the names of those who have presided over this society one sees specialists who have been among our ablest practitioners, writers and teachers. A study of the entire membership shows that it has at all times included many leaders. A commendable number have left a notable record.

Sixty years ago there were but few, even in our largest cities, who called themselves specialists in Otolaryngology. The number of specialists of that day has been multiplied by hundreds and scarcely any village is without one or more who boasts of skill in the management of diseases of the upper air tract.

Taken collectively in the United States and Canada, there are now about eight thousand physicians who limit their practice to treatment of affections of the ear, nose and throat. One American society has a selected membership approaching three thousand such men. Another is limited to five hundred members, while yet another, the section on Otolaryngology of the A.M.A., has no limitations whatsoever, except membership in the parent society. This association, the AMERICAN LARYNGOLOGICAL, has long been limited to a membership of one hundred! It is one of the oldest and best known. It is almost a father of them all and is presumed to be the leader of them all. It remains notably exclusive and some have wondered that with thousands from which to choose, only one hundred can be found who meet the requirements for admission. It has been asked, "Are there not now more than one hundred otolaryngologists in the United States and Canada who

have the supreme qualifications necessary to hold membership in this association, and, if so, would it not be of advantage to both the qualified surgeon and the society to admit a somewhat larger number, for, it is argued, there is a time in the career of every ambitious, forward-looking specialist when, if given such opportunity as this society affords, his talents will be developed and his accomplishments become more worthwhile; whereas, if isolated and compelled to await the death or retirement of a member and the creation of a vacancy, the former keen incentive will probably have become subdued or lost by uncertainty and passing years?"

Your president has seen this happen and he therefore holds the belief that a few more admissions each year would be wise. It would seem to him that the admission of five new members each year, over and above those necessary to keep up the present quota, until the maximum membership of one hundred and fifty be reached, would be helpful both to the individual and the Association.

*Relating to the Social Side of the Society.* It is the belief also that the social side of our association, especially as it relates to new members and those who must retire because of illness or age should receive most thoughtful attention. Many members have small opportunity of meeting the Fellows except at the annual meeting and then too briefly. This gathering should therefore offer as many opportunities as possible for close personal contact of members. Older members should go out of their way to meet and greet the newer men. It would prove delightful for both.

How well your president remembers a certain May morning, now exactly thirty years ago, when he came with trepidation, a youngster and somewhat of a stranger, into this association! How well and how gratefully he recalls the incident, when as he entered, Doctors Christian R. Holmes and Henry L. Swain came graciously forward to welcome and then to introduce him to others who were present. The passing of thirty years has in no sense lessened the feeling of supreme happiness he felt on that occasion.

Time passes with unbelievable swiftiness. Each year familiar faces are missing because of death, age or illness. Some one or more of our Fellows must remain at home unable to come but longing to be here. Any member who has missed a meeting be-

cause of illness will recall with pleasure that while thus prostrate, a telegram or other greeting may have come from this society. Such thoughtfulness on the part of the society carries a message that is a hundredfold more helpful than any medicine yet conceived. May this association never forget its absent members.

*Relating to Wives and Daughters.* Wives and daughters of members have been loyal to this society and have been a notable element in its progress. The association has likewise been loyal to its women, has welcomed their attendance and has assisted in the preparation of a program of social entertainment that would entice them to attend. On a careful study of this question it would seem, however, that the women would do well to organize among themselves, to elect their own officers and to arrange their own programs of entertainment. No suggestion is made here of a complete separation of the social activities of the Fellows and their wives, for indeed, there should always be a close understanding between them. The Society would always feel honored by such intimate union as would serve the best interests of both. All this is merely suggestive, the thought being that a larger percentage of women would probably attend if given the privilege of managing their own affairs.

*Relating to Better News Service.* It is a notable fact that the Fellows of this and other similar associations do not, at present, have sufficient news service in their special publications. It not infrequently happens that matters of much interest, even of great importance, happen to our Fellows and the same are unknown for many months. Our members are given chairs in Universities, receive medals, commendations, honors of many kinds in which every otolaryngologist is interested and about which all would be pleased to hear, but such information leaks out but slowly if at all, and then through channels that may or may not be reliable. Your President recommends, therefore, that the question of establishing a news column in each of our principal journals be referred to our council in the hope that more life and interest may be injected into a rapid and reliable dissemination of news relating to the members of our specialty. A news editor for this purpose is suggested.



*Relation of Climate to Otolaryngology.* On election to the presidency a year ago it was the intention of the speaker to devote this entire address to the problem of climate in relation to diseases of the upper respiratory tract. However, since more than one slip between the cup and oral cavity has occurred during this interim in your president's life, it has become impossible to carry out the wish. This is to be regretted since undoubtedly the effect of climate on the respiratory tract is one of the most important questions before the laryngologist, and, it is believed, the least understood. The writer who has no prejudice in the matter has felt somewhat qualified to discuss this subject because circumstances have compelled him to reside in several of the most noted climatic areas for periods long enough to observe conditions in each and to form a fairly trustworthy opinion concerning the health-giving value of each. In view of the frequent ignorance on this subject among medical men and the difficulty physicians have in obtaining reliable information as to particular locality best suited for those suffering from a known ailment, it seems wise to mention here, even if very briefly, a few points that may prove helpful to those most seriously concerned.

The first observation is, that the United States has within its boundaries wide areas wherein the climate is unexcelled. Nowhere is it perfect, since perfection in climate does not exist, but degrees of near approach may be found. While many parts claim to be health resorts at some period of the year, the most heralded all-year region is a broad belt extending across the entire southern portion of the United States. It will be observed that this area differs greatly in different places and this in a sense is fortunate. Some parts are very dry and dusty; others have an abundance of rain; some areas, chiefly mountainous, are comparatively cool; some have an almost equal temperature, both day and night; some have very marked changes from midday to midnight.

It is evident therefore, that a region may be selected to suit the nature of the disease requiring benefit. Patients having low physical resistance do best, as a rule, in places where the temperature is nearly the same throughout twenty-four hours. A sudden drop of thirty to forty degrees, regularly occurring in some regions, is disastrous to the progress of many ailments, unless the condition

is artificially met and overcome. Dry desert regions are usually dusty and sometimes dust storms occur. This dust is fine and blows through the smallest residential crevices. In some areas an otherwise perfect climate is reduced to a second or third class by dust so thick that one may write his name on any smooth surface after twenty-four hours. Dust is especially detrimental in all affections of the upper air tract. Nearly all the southwest is dry, which may vary in degrees but is marked in many places. Both skin and mucous membranes are affected. The former often is so dry as to become parched and cracked. A piece of fresh, soft bread exposed to the air for a few hours may be reduced to finest powder by rubbing between the thumb and fingers. These unfortunate conditions can and are being largely overcome by modernizing the homes.

All the southeastern portion of the country has a plentiful rainfall and in some places the temperature at midday and midnight does not greatly differ. The entire area lies but little above sea level. Patients who suffer greatly from marked climatic changes and from extremes of dryness, do best in this region.

That much ignorance exists among physicians as to the value of special climate, and especially climate most suitable to his patient, can scarcely be denied. The question may well be asked as to why this is and there are many explanations. Residents of any region that is considered good from the standpoint of health are as sensitive to criticism of their climate as they are to criticism of religion or politics. Their particular type of climate seems to them a panacea for all diseases and at all times. These persons dare not speak otherwise than boastfully, because in some places climate is almost their only asset and boasting is expected. Not to boast would be considered unpatriotic and inexcusable. Should the home doctor or the patient write to such a place for information, the answer would usually come from some commercial body or resident who has something to sell, and it is highly favorable in almost every instance.

Physicians have often written on climate after a too brief residence. A secretary of a commercial body would probably lose his position should he tell the whole truth about the climate of the region he represents. This does not mean that he wilfully deceives his correspondent, but as no climate is perfect, the whole

truth about even the best region in the world, would probably discourage many from seeking residence. An instance may be cited in which an invalid, on the advice of his home physician wrote to the commercial body of a well known health resort, only to receive a quoted report from a physician who had spent but a few days in the place during a most favorable weather period. Unfavorable portions of the article had been deleted and only the most favorable paragraphs quoted, representing a perfect climate. What resident physician, merchant, promoter or even common citizen of a health resort would be bold enough to tell the entire truth about its climate? He could scarcely remain a welcome resident.

How then, may the home physician who wishes to do the best for his patient, obtain reliable information regarding the question? It is almost impossible to obtain such information, but most dependable would be the honest, capable report from one who has resided long enough in the region to have noted all the indigenous freaks of climate. This informant must be free from all local financial interest and such a person is rare.

Unfortunately the home physician whose chief information is hearsay, often makes over-zealous statements regarding the benefits the patient may expect from change of residence. Patients have quoted their physicians as having said, "A few weeks in this climate will cure your chronic sinus suppuration, your long-standing cough, your arthritis." Even a short residence in a properly selected climate sometimes gives remarkable results, but as a rule long or permanent residence is essential and even then the care of a first-class physician is also necessary. It is seldom that climate actually cures the disease when serious and extensive pathologic conditions are once established in the individual. The most that should be expected is more or less complete relief which is often equivalent to cure so long as the patient remains in the favorable climate.

Treatment by climate is necessarily expensive. The patient is away from home, where if he is to receive benefit he must be well housed and fed, well clothed and entertained. There is no climate in the United States where the patient does not occasionally need artificial heat. There are occasional days even in the



best regions when the invalid suffers greatly without a good fire. He must also have clothing that is suitable to all types of weather. A good overcoat, even furs and woolen underwear, though seldom needed, are at times essential. One prolonged exposure to the severe chill of night common in some health resorts, may undo the gain of months. Sickness away from home in a health resort is too often only possible for the rich. All these questions must be considered sensibly before the patient is advised to change climate.

There can be little argument over the assertion that a strip of our country extending along the southern border from the Pacific to the Atlantic ocean is one of the best climatic regions of the world. This should be better known and the knowledge more frequently and scientifically used in the treatment of disease. The question arises, therefore, as to how the situation can be improved.

Since the value arising from climate, affects chiefly the upper air tract, there is no class of physicians who should be so much interested and well informed upon the subject as the otolaryngologist, and of the several societies interested in otolaryngology, none should have a greater interest than the American Laryngological Association. The American Academy of Ophthalmology and Otolaryngology, the American Otological Society and the American Laryngological, Rhinological and Otological Society has each some specific problem upon which it is laboring, but our Association has none and a proper study of climate is among the greatest of all problems. Undoubtedly an investigation of this great American asset would greatly benefit the people and would reflect great honor and credit upon the association making it. Your president would, therefore, call your attention to the question and would recommend that research be started to the end that a reliable treatise on climatic value be prepared, not only for otolaryngologists but for all who practise medicine, and would invoke nature's help in the maintenance and restoration of health. The suggestion is made that one or more members who have the time and are entirely free from local financial interest be appointed to prepare a volume which would contain facts about climate, obtained by prolonged residence and which would, of course, be strengthened still further by observations during the investigation

and publication. Such a volume would be expensive but if prepared as here suggested with full association approval, the pre-publication sale would probably pay the cost. It would be a much worth-while endeavor and it is logical for the American Laryngological Association to support it.

*Recommendations of the President.*

1. Increase of membership by the admission of five new members each year over a period of ten years and limiting the total membership to one hundred and fifty.
2. Encouraging the wives and daughters to organize their own society, wholly for social purposes, but functioning with the assistance of this association.
3. Appointment of one or more Fellows who are qualified according to the lines above set forth, to devote their time to the preparation of a volume on "American Climate in Relation to Diseases, especially of the Upper Air Tract."
4. The securing if possible of one or more pages in each of our American publications devoted to otolaryngology, to be used for the purpose of publishing social and other news relating to otolaryngologists.
5. To encourage reports of any serious accident or illness of a member and to direct the secretary to send a telegram or other message expressing the interest and sympathy of the Association in such event
6. The expression of the Association's position on the question of socialized medicine. With reverence for the past and with hopes for an increasingly useful future, the sixtieth session of the American Laryngological Association is now declared open.

## PLASTIC REPAIR FOLLOWING REMOVAL OF EXTENSIVE MALIGNANT TUMORS OF THE ANTRUM

FREDERICK A. FIGI, M.D.

MORE effective therapy for malignant tumors of the antrum has produced additional responsibility for the surgeon; that is, the correction of the deformity that at times follows their removal. Surgical treatment of these growths, as a rule, leaves no appreciable change in the facial contour provided the lesion has not extended beyond the sinus. However, when extrinsic involvement is present, therapy sufficiently radical to offer a possibility of curing the disease is likely to cause more or less marked deformity of the face. Although the patient may be freed from his malignant disease, he is often left so badly disfigured that he can neither mingle socially nor earn a livelihood. The rehabilitation of this group of patients accordingly is at times as important as curing them of their malignancy. Repair of these deformities often presents a major problem in plastic surgery.

Various forms of treatment have been used in dealing with malignancy of the maxillary sinus. The prognosis and the tendency to produce external deformity vary greatly with the different therapeutic measures. Resection of the superior maxilla and other cutting operations, which were commonly employed for the removal of these growths a few years ago, only infrequently effected eradication when the tumor was confined within the antrum, and very rarely did so when it had become extrinsic. Moreover, such procedures often caused considerable external deformity. Irradiation with radium alone, or with radium and the roentgen-rays combined, at times produces striking results in cases of highly malignant tumors in this situation and a minimal change in the facial contour is likely to follow. The combination of electrocoagulation or the actual cautery with irradiation has, however, proved most effective in the hands of the majority of surgeons interested in this field. This type of therapy has been used in dealing with these cases at The Mayo Clinic since 1917. The



antrum is opened into through the buccal fold, or, if the tumor is extrinsic, at the point elsewhere inside the mouth, where it approaches nearest the mucosal surface. Usually, the exposure is made by means of electrocoagulation, and destruction of the tumor is effected in the same manner. At the completion of the operation radium is packed into the antrum, especially into those portions of the cavity where thoroughness of the removal is questionable. With this type of treatment 40 per cent of five-year cures has been secured in the cases of primary tumors of the antrum and 53.4 per cent of five-year cures has been secured in the cases in which the involvement of the sinus was secondary to malignancy of the upper jaw.<sup>2</sup> In view of this relatively good prognosis it seems appropriate that serious consideration be given to the repair of deformities incident to the treatment of these tumors. The literature contains very few references to their correction. The inference would seem to be that the patient and his physician have been content with arrest of the malignant process, and have not taken cognizance of the functional and cosmetic aspects. The most detailed considerations on the repair of these deformities that I have been able to find have been that of Ohngren, in his excellent monograph on "Malignant Tumors of the Maxillo-ethmoidal Region," and that of Beck and Guttman.

Whether or not external deformity will result following removal of malignant tumors of the antrum in this manner depends very largely on the situation and extent of extrinsic involvement. If the growth is limited to the sinus, no appreciable change in the contour of the face is likely to develop postoperatively, even though the neoplasm completely fills the antral cavity and thorough cauterization of the entire bony wall is necessary. Maintenance of a large opening into the antrum is desirable in all cases in order to permit of inspection for possible recurrence, for at least a year following the operation. This is insured in the usual case by wide electrocoagulation of the area of approach on the anterior antral wall. If the tumor has eroded through the bony palate or is causing bulging of the alveolar process and is situated directly beneath the mucous membrane of the mouth, in which event it is exposed at one of these points, the perforation will be in the corresponding situation. While openings here are more bothersome to

the patient than those through the anterior wall of the antrum, they are readily closed with a special dental plate and accordingly are of no serious consequence. The most common external deformity in these cases is retraction and elevation of the upper lip and the angle of the mouth. This occurs not infrequently following removal of primary tumors of the antrum that are entirely intrinsic. It is encountered more frequently, however, in cases in which the growth has perforated the anterior antral wall or the alveolar process and in those cases in which the antrum is involved secondarily by a malignant neoplasm of the anterior part of the upper jaw. It is due to contraction of the scarring in the soft tissues overlying the anterior wall of the antrum and that resulting from loss of the mucous membrane in the labial sulcus and on the inner surface of the upper lip, together with loss of the underlying bony support. Perforation externally through the cheek into the antrum is one of the most conspicuous deformities occurring in this connection. It is likely to develop only in those cases in which the lesion has eroded through the anterior wall of the antrum and is infiltrating the skin or the subcutaneous tissues directly beneath it. At times this defect is very large and opens directly into the mouth and nose as well as into the antrum. In fact, it is often much larger than was anticipated in view of the very limited involvement of the skin and subcutaneous tissues, because of the tendency of the thinned-out skin to roll inward as healing occurs. When the tumor is already perforating the cheek, it is usually of decided advantage to carry out the electrocoagulation through this area, as an opening in the roof of the mouth may thereby be avoided while an external perforation is bound to occur. Not infrequently, when the neoplasm has eroded through the upper part of the antral wall or the floor of the orbit, sequestration of the lower orbital border or of a portion of the malar prominence may result. Complete loss of the eye is not a rare occurrence in these cases. The ocular globe is at times destroyed by the heat of the electrocoagulation or by the reaction from the radium used inside the antrum when perforation of the bone was not previously present. The patient should always be warned of this possibility. In some instances, even though there is loss of the entire lower portion of the bony orbit, the eyeball miraculously remains unharmed and

the vision in the eye unimpaired. However, considerable deformity and interference with function develop in these cases, since the eye recedes and sags markedly; it is supported only by a hammock of soft tissue, and diplopia, often of pronounced degree, results.

Displacement of the orifice of Stenson's duct occurs frequently as healing takes place within the antrum. This is due to traction of the scarring on the mobile mucous membrane lining of the cheek. In the usual case this altered position of the orifice is of no consequence whatsoever, and the patient is not even aware of it. Rarely, however, the opening of the parotid duct may be drawn sufficiently upward and anteriorly into the antrum so that saliva drains into the nasal fossa and thence from the nostril.

The optimum time for repair of the deformity in these cases is an important consideration. Opinions vary greatly in this regard. In the clinic of Professor Pichler in Vienna, which I visited recently, resection of the superior maxilla is carried out for the removal of these tumors, and if sacrifice of the full thickness of the cheek is necessary, an immediate plastic closure is done, even though this requires bringing up a flap from the neck and thorax. To the majority of us, as to the professor's own assistant, this procedure seems daring. On the other hand, some men prefer to delay proceeding with correction of the deformity for from two to four months. At The Mayo Clinic we have not felt justified in undertaking repair before the patient has been free from evidence of the malignant disease for approximately a year and we frequently wait much longer. This tides the patient over the period during which there is greatest likelihood of recurrence. I have, however, observed a return of the growth after approximately two years in several instances. This delay also permits the sequestrum to separate, the margins of the defect in the cheek and palate to heal over and the inflammatory reaction to subside. While a plastic closure can often be made much more readily immediately following excision of the tumor, as one does not then have the scarring and fixation of the tissues to contend with, there is considerable risk of covering over a small portion of the neoplasm that has not been completely removed. Should a recurrence develop beneath the grafted tissues, its recognition would be much longer delayed than it would have been if the defect had been left open. Sacrifice



of a varying amount of the graft together with a considerable portion of the adjacent normal tissue would then be necessary. Moreover, when immediate repair is planned, there is often a tendency for the operator to save questionable tissue whose sacrifice would increase the difficulty in closing the wound.

Procedures for the repair of the defects in the palate and cheek vary greatly, depending on the situation and size of the perforation, the training of the surgeon and whether or not the services of a competent dentist are available. Perforations in the palate and alveolar process may be dealt with surgically, but as a rule they are better closed with a special denture. This is made to furnish any teeth whose sacrifice has been necessary and it is held in place by means of clasps about the remaining upper teeth. In case the upper jaw is edentulous, spring clasps that overlap the margins of the defect or spring supports that are attached to the lower teeth or denture are required. There is rarely sufficient mucoperiosteum remaining on the palate to permit of surgical repair, and a skin flap is not in most cases satisfactory for this purpose. While a cutaneous flap effectually occludes the opening, it remains soft and flabby and is not sufficiently rigid to withstand the suction necessary to hold an artificial denture in place. The dental plate worn over such a skin flap accordingly requires special mechanical support, and in most instances it might better have been used for closure primarily.

When a perforation through the cheek is also present, the denture used to close the palatal opening may be made to support a prosthesis for closure of the former as well. Such prosthetic appliances are not infrequently employed for this purpose, although most patients much prefer to have a defect in this prominent situation repaired with their own tissues. We have occasionally used such mechanical means for closure temporarily while awaiting the time when it seemed safe to proceed with plastic repair. The prosthesis can be made sufficiently inconspicuous, so that the patient is able to resume his work during this period (Figs. 1 to 5).

In most instances, repair of a perforation through the cheek will require the bringing in of tissue from a distance by means of a pedicle flap, although if the opening is small a satisfactory closure can often be made by paring the margins and, after wide under-

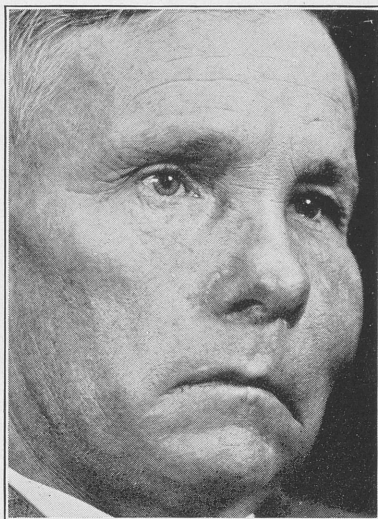


FIG. 1.—Extensive recurring squamous cell epithelioma (Grade 3) of the left antrum, upper jaw and cheek of a man, aged fifty-one years; tumor is bulging and about to perforate the cheek.

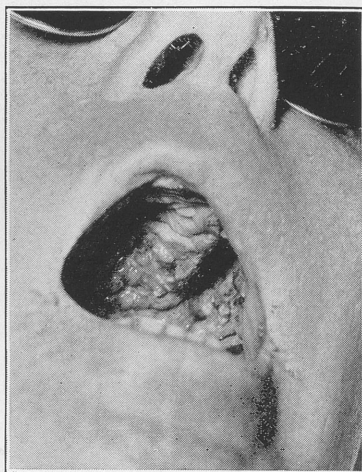


FIG. 2.—Fungating tumor mass presenting in the roof of the mouth of the patient shown in Fig. 1.

cutting, directly approximating them. This is the simplest type of closure; it requires a single operation and a minimum amount of time, usually not over a week. On the other hand, when pedicle flaps are necessary, multiple operations with several periods of hospitalization and a greater length of time are involved. Flaps for this purpose may be secured from the forehead, neck, arm, thorax, back or abdomen. The most useful sites are the forehead and the thorax. In women the temporofrontal or frontal region

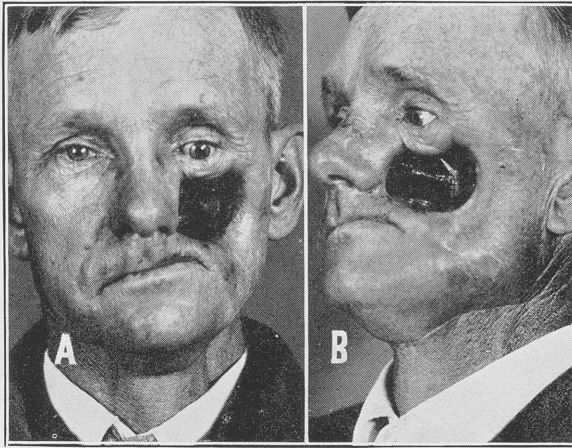


FIG. 3.—A, same patient as shown in Figs. 1 and 2; this photograph was made nine months after removal of the tumor with electrocoagulation and radium; in addition to the large perforation in the cheek there was destruction of the bony floor of the orbit and of the naso-antral wall; a large perforation was present in the roof of the mouth. B, same patient as shown in previous figures; metastasis into the cervical lymph nodes was present; the lymph nodes have been removed.

is frequently the site of selection, as the hairdress can be made to effectually conceal the skin graft and scar on the forehead. In men, the thorax is usually preferable, as the scarring in the frontal region is unduly prominent, unless the patient has a very thin skin. Forehead flaps possess the decided advantages of proximity and vascularity, which make it possible to transfer them more rapidly; the color and texture of the skin of the forehead also render the repair less conspicuous. If a flap is taken from this region it is possible to complete the repair in from four to six weeks. If a



flap is taken from the thorax for repair of a defect in the cheek, the flap must be tubed, which requires an additional surgical procedure. Moreover, the great length of the flap required for this purpose and the comparative avascularity of the tissues over the upper portion of the thorax, necessitate a delay of at least three months following the tubing, to permit a satisfactory blood supply to develop in the upper pedicle. The outstanding advantages of such flaps are that they permit the securing of an almost unlim-

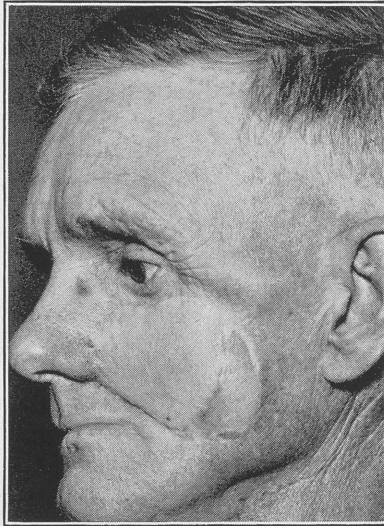


FIG. 4.—Same patient, four years following removal of malignant tumor of the left antrum; the perforation in the cheek and that in the roof of the mouth have been repaired with a tubed flap from the thorax.

ited amount of tissue for repair and the resultant scarring is on a covered portion of the body. Their chief disadvantages are the greater length of time required and the fact that the repair is more conspicuous, as the color and texture of the skin do not match that of the rest of the face (Figs. 6 and 7).

It is essential that the portion of the flap to be utilized in repairing the perforation be lined. This may be accomplished by turning under the distal end of the flap, but a full-thickness skin graft is usually preferable, as its application requires no more time than

the former and the part of the flap utilized in the actual repair remains thinner, less bulky and more pliable. The cosmetic result is also more satisfactory. At times a lining is secured by turning in a flap of skin from the margin of the defect, and covering it with the flap transplanted from the forehead or thorax. This procedure possesses the disadvantage of making it necessary to

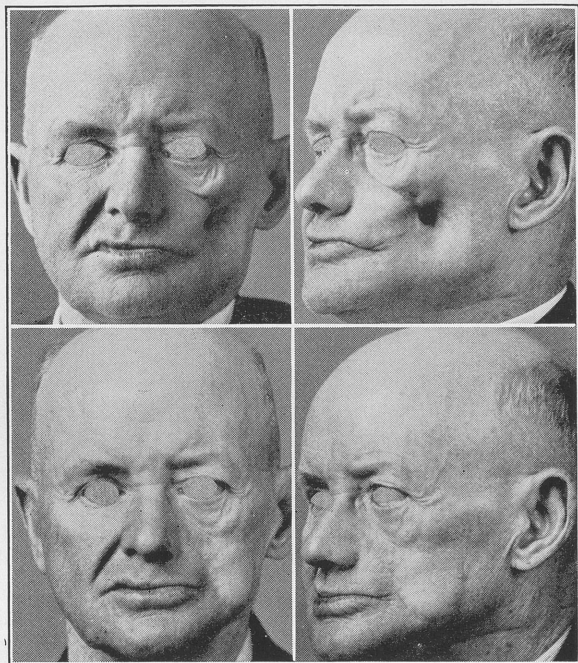
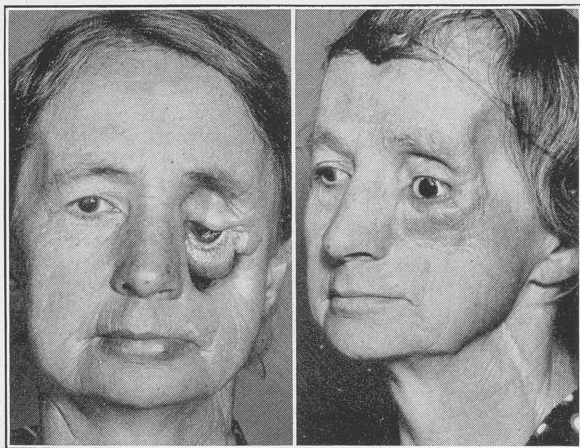


FIG. 5.—Above, postoperative perforation of the cheek following removal with electrocoagulation and radium of extensive recurring epithelioma (Grade 3) of the left upper jaw, antrum and cheek. Below, same patient following closure of the perforation with a tubed flap from the thorax.

graft a larger area on the face. When a free skin graft is used for a lining it must be applied to the flap approximately two weeks prior to transference in order to permit it to become firmly attached.

Retraction and elevation of the upper lip and angle of the mouth due to scarring and loss of the bony support of these structures, result in a very conspicuous deformity. For correction it is neces-

sary to free widely the scarred attachment of the lip and cheek to the superior maxilla, and to roll out the mucous membrane which has been drawn upward inside the lip. A Thiersch or shaved skin graft from the arm or thigh, which has been wrapped about dental compound with its deep surface outward, is then applied to the denuded inner surface of these structures in order to restore their lining. The graft is best taken from a surface that is free of hair



A

B

FIG. 6.—*A*, deformity following removal with diathermy and radium of an extensive recurring Grade 4, squamous-celled epithelioma of the left antrum, cheek and orbit of a woman, aged fifty-three years; the tumor had been explored previously through the cheek and was perforating the cheek; sequestration of the bony floor of the orbit and malar bone has occurred; the eye was destroyed by the reaction from the operation and irradiation. *B*, same patient two years following removal of the epithelioma; the perforation in the cheek has been closed with a flap from the forehead; a graft from the crest of the ilium has been inserted to restore the lower orbital border and malar prominence; the patient is wearing an artificial eye.

for, while it should be cut sufficiently thin to prevent inclusion of hair follicles, there is always a possibility of transplanting some of them into the mouth. The stent bearing the graft is best held in place by means of an appliance attached to the teeth. In the absence of teeth in the upper jaw the dental compound mold may be fused to the patient's upper denture, or silk sutures may be passed about the stent, through the cheek, and tied over a gauze pad on the outer surface. The jaws are immobilized when feasible



by means of interdental wiring so that the stent and graft will remain firmly in position. The stent is kept in place for ten days. Upon its removal the graft will almost invariably be found lining the restored buccal fold and firmly adherent. Providing immobilization can be maintained, thin grafts of this type seem to take practically as well within the contaminated oral cavity as on the surface of the body. Unless kept under tension for several months, however, they undergo marked shrinkage. It is accordingly neces-

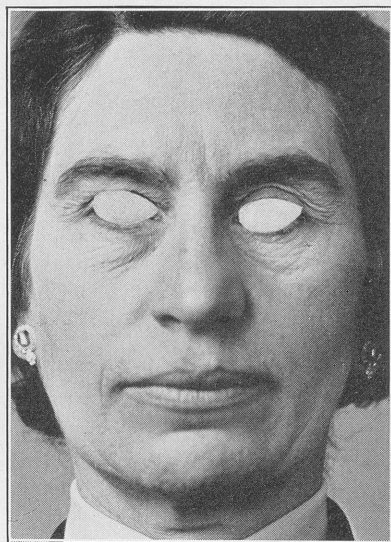


FIG. 7.—Patient three and a half years following electrocoagulation of adenocarcinoma that was filling the left antrum. Elevation of the upper lip has been corrected by relining the lip and labial sulcus with a skin graft. A prosthesis is being worn to support the lip and close the perforation which extends through the roof of the mouth into the antrum.

sary to replace the dental stent immediately with vulcanite. This must be worn continuously until the permanent prosthesis is ready. If it be left out for only a day at this time, the new buccal fold will be drawn down to such a marked extent that little will be accomplished by the operation. Once the lip and cheek have been mobilized by relining them in this manner, the symmetry of this portion of the face is readily restored with a proper prosthesis (Fig. 8).

Loss of the inferior orbital border requires either a bone or cartilage graft. While human cartilage that has been preserved in an aqueous solution of metaphen or that has been freshly

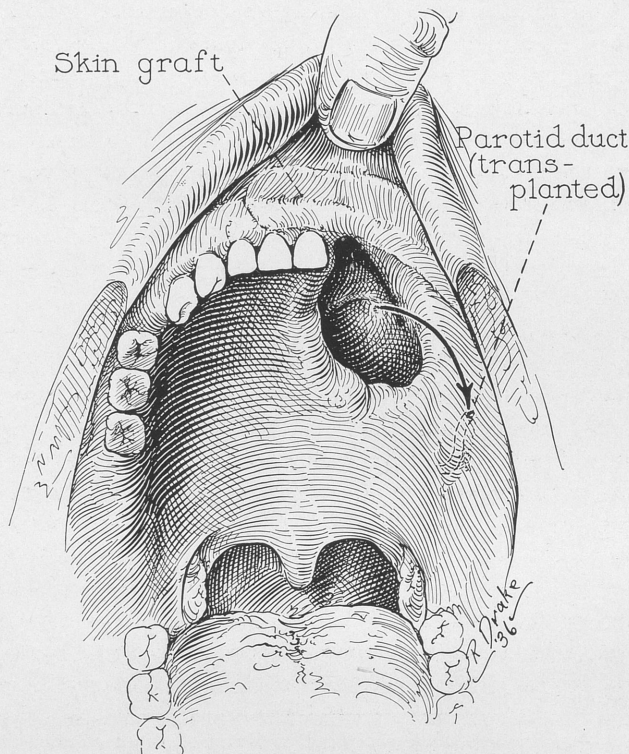


FIG. 8.—Drawing showing perforation through the roof of the mouth into the antrum, transplanted Stenson's duct and skin graft relining the labial sulcus (same patient as shown in Fig. 7). The meatus of Stenson's duct had been drawn up into the antrum by the scarring, and saliva was draining into the nose. The meatus of the duct was transplanted into approximately its normal position in the mouth.

removed from the patient's rib may be used for this purpose, it is, as a rule, less satisfactory than bone, because of its lack of rigidity. For restoration of the orbital floor and for elevation of the ocular globe, however, it is usually of decided advantage, as

it can be shaped more readily than bone. A bone graft from the iliac crest serves very well for restoring the inferior orbital border. It is easily shaped with bone forceps and is then inserted in a bed prepared by tunneling with scissors in the soft tissues of the upper part of the cheek, through a small lateral incision. With proper beveling of its ends it will at times remain in position without anchoring. When necessary to anchor it, I have found driving a short piece of a straight needle through it into the underlying bone serves the purpose well. The needle is inserted directly through the overlying skin and must be sufficiently short to keep its deep end from protruding into the nose or antral cavity. Its outer end is driven down flush with the surface of the graft. I have never seen this cause trouble later and it does immobilize the graft securely.

Displacement of the orifice of the parotid duct as a result of postoperative scarring is rarely sufficiently pronounced and in such a position as to require consideration. In one of the cases which I observed, the opening of the duct was drawn upward into the antrum and the resultant drainage of saliva from the nostril caused the patient much more annoyance than did the deformity of the distorted upper lip or the speech defect which resulted from perforation of the palate. It necessitated very largely foregoing the pleasure of social affairs, since anesthesia over the distribution of the infra-orbital nerve rendered it impossible for her to tell when salivary secretion was present in the nostril, until it dripped from the nose or ran down upon her lower lip. Inhalation of cold air tended to stimulate the secretion, probably because absence of the naso-antral wall and the large perforation through the floor of the antrum permitted the current of air to pass directly through the nostril into the mouth. Thus, the secretion was often bothersome while she was on the street. It was especially prone to drip from the tip of the nose as she sat with her head tipped slightly forward at the bridge or dinner table. Eating caused particularly active secretion. While it was apparent that the meatus of Stenson's duct had been drawn up into the antrum during the process of healing, careful search failed to reveal the site of this opening. In an attempt to correct the condition, the cheek and upper lip,



which were bound directly to the margin of the antral perforation by scar tissue, were freed up and the denuded surface covered with a skin graft. Although this restored the buccal fold and corrected the facial deformity satisfactorily, the drainage of saliva from the nostril continued as actively as ever.

Further attempts to locate the displaced outlet of Stenson's duct were unavailing, until secretion of the parotid gland was actively stimulated by having the patient suck a lemon. The orifice was then found on the anterior wall of the antrum just above the perforation in the palate. The obvious route of salivary drainage from this point, because of complete loss of the naso-antral wall, was directly into the vestibule of the nose and out through the nasal meatus.

The problem then was either to dry up or to divert the secretion of the parotid gland. It did not seem likely that drying up of secretion could be completely accomplished by means of irradiation or evulsion of the auriculotemporal nerve, and transplantation of Stenson's duct was decided upon, although no precedent for this procedure could be found in the literature.

Pentothal sodium was administered and the parotid duct was readily picked up through a small incision on the inner surface of the cheek, below and posterior to its opening in the antrum. After following the duct upward, it was cut across just at its antral opening, and the free end transplanted into a stab wound, in the approximate situation of the meatus of the normal duct. Primary healing occurred and the new meatus functioned normally. The patient has been fitted with a special denture which satisfactorily closes the perforation in the palate. Her symptoms have been relieved completely.

**SUMMARY.** Facial deformity usually does not occur following removal of malignant tumors of the antrum unless the growth is extrinsic. The most common disfigurements encountered in such cases are elevation and retraction of the upper lip and angle of the mouth, perforation of the cheek and nose, and loss of the malar prominence, inferior orbital border and the eye. Displacement of the meatus of the parotid duct very rarely is sufficiently marked to produce symptoms. Satisfactory plastic correction of these deformities is possible in most cases.

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## DISCUSSION

DR. ROBERT H. IVY, Philadelphia, Pa.:

I just stepped in here to listen to what Dr. Figi had to say and did not expect to be called upon to discuss his paper. The few cases of malignancy of the upper jaw and maxillary sinus that we have had, we have treated in about the same way as Dr. Figi, namely, by a combination of irradiation and surgery, although in our cases the irradiation is carried out by the radiologist. One of the most distressing features of these cases is the radiation necrosis, the patients many times enduring weeks of intense suffering and then an anxious time of waiting sometimes months for the sequestrum to separate. It requires a much longer time here for the dead bone to come away than in ordinary cases of suppurative osteomyelitis. Plastic repair cannot be begun until all of the dead bone has been removed. Dr. Figi is to be congratulated on the excellent results he has shown in the cases presented.

DR. CHARLES J. IMPERATORI, New York City:

I should like to ask Dr. Figi a few questions.

How long after the original operation does he do his plastic? My understanding of all these operations was that we produce this dehiscence in order to observe the interior of the antrum, to decide whether or not there was any recurrence before making a prosthesis or doing a plastic. Also, in his operation, I gather he does not ligate the common carotid.

Regarding the reference to Stenson's duct: there is a case, Dr. Figi, on record that was produced not by a malignant disease but by an extensive gingival and alveolar retraction within the maxillary sinus,

following a poorly done Caldwell-Luc operation. The same symptoms, as described, were present.

The condition was corrected by a plastic operation done by Dr. J. E. Sheehan of New York.

DR. JOSEPH C. BECK, Chicago, Ill.:

Our experience cannot match that of the Mayo Clinic although I have seen a number of cases. I would like to bring out some points that have been mentioned somewhat superficially regarding the prosthesis.

From the presentation, it would look as though there was no place for that when the cheek is destroyed. So far as I can see, the only benefit from the late war was the development of this prosthesis. I have watched Holmgren's work, whose methods I use much in the hospital.

I saw the artificial proposition which Dr. Salinger presented to our local society. I think this insertion of an artificial eye in the prosthesis is the most wonderful procedure in the case of a complete loss including the eye. From a casual glimpse in the street you could not realize that so much of the face had been lost. There is much to be said in favor of the prosthesis when it is properly made. If it is simply a mass of dental wax, it is of no value.

In the loss of the infraorbital margin, so beautifully shown here, a new method of fixation by a rim of bone from the ilium is suggested. If this does not come in contact with any of the mucosa and heals, it is a great advance, but the musculature of the orbicularis palpebrarum is lost in the lower lid. The lid is dropped and the cornea is constantly in danger and if these people do not lose the eye from disease or the operation, they are likely to lose the sight of it from a keratitis.

One procedure, the narrowing of the palpebral fissure, which simply scars the margins of the external one-third of the upper and lower edges still allows the patient to see perfectly well yet protects the cornea to a marked degree.

I am glad nothing was said about artificial implantations which are difficult on account of mucous membrane exposure but perhaps all of us have seen the ivory implantation last for a period of years. This is tolerated in some localities and may be at others.

DR. THOMAS E. CARMODY, Denver, Colo.:

I want to compliment Dr. Figi on this wonderful paper and his illustrations.



Of course, those of us who have smaller clinics or see fewer patients than they do at Rochester have very little in common with their work. However, we have seen a number of these cases in the last thirty years, and I have some observations to make.

I was very much interested in Dr. Figi's last case, and I was very much interested when he reported it some time ago. I have a case at the present time that is causing me trouble and requiring the same thing. The plan that he brings forward we sometimes have to use.

I had a case recently (not a malignant case) that had oversecretion of saliva in the same way, and recommended x-ray, because we felt that irradiation would stop the flow very much better. It has not entirely produced results up to the present time.

Formerly, we removed the whole maxilla, and as Dr. Figi has said in the first part of his paper, we found that our results were not so good, and very frequently we had recurrence, even after removing the whole of the maxillary bone. I have one case that has gone for something like fifteen years, after removal of the maxilla. The last twenty years, practically, we have used the method as described by Dr. Figi, and described originally by Dr. New, formerly with the soldering iron and later with the electric apparatus of Percy, and then later with the so-called radio knife.

In most of these cases, except the late cases, we can go through the mouth, and in those cases, of course, we can always replace with prosthesis. We have a number of these cases at present. Some of them have gone for a good many years.

Dr. Figi speaks of recurrence after one, two, three and four years. We had one that recurred even after ten years. We had one that recurred after seven, and one that recurred after nine. Some of those cases, of course, are very difficult to handle the second time, although one of these cases that I have had over fifteen years, recurred at eight years, and has not recurred since.

In those cases where they come late, the opening into the cheek, of course, requires plastic surgery, because, as Dr. Figi has said, prosthesis is not very satisfactory. One point that he brought out in the latter part of his paper I think is very important, and that is we do not have facial paralysis because we do not extend far enough back.

The Stenson's duct operation of Dr. Figi's I think is very interesting.

DR. FREDERICK A. FIGI (in closing):

Doctor Beck spoke of the usefulness of prosthetic appliances for the correction of deformities following the removal of malignant tumors of the antrum. Their use was mentioned in my paper but the paper

dealt primarily with plastic repair of such defects and these latter measures were accordingly stressed. There is no question but that prosthetic appliances are extremely useful in many of these cases.

Answering Dr. Imperatori's question, we like to wait until the patient has been free from any evidence of the malignant process for approximately a year before proceeding with plastic repair. However, we often wait much longer, at times for several years. In the case of a patient that is being taken care of at present, we waited for well over a year before undertaking reconstruction. A lined forehead flap was then turned down to close a perforation in the cheek and a bone graft was inserted subsequently in order to build up the infra-orbital border and the floor of the orbit and thus lessen the diplopia. Upon the patient's return for observation a few months later, and almost two years after removal of her neoplasm, she showed a recurrence of the malignant disease involving the mesial portion of the lower eyelid.

We do not routinely ligate the carotid vessels in these cases, either as a preliminary measure or at the time of the primary operation. It has been necessary to ligate the external carotid in a few cases where the tumor recurred and eroded into the vessels.

# A FULMINATING MONILIA OR ASPERGILLUS INFECTION OF THE NOSE

C. STEWART NASH, M.D.

## CASE REPORT

ON August 8, 1937, a woman, aged forty-three years, complained of a pain over her left eye. Although she admitted losing 28 pounds in weight during the previous four or five months, she insisted that up to this time she had had no sickness and that her present complaint, namely, a pain over the left eye, was unassociated with any other symptoms.

Four days later the upper left eyelid drooped and became swollen, the left eye protruded, and the vision had gone. She was nauseated and vomiting; had excessive thirst, frequent urination, difficult and rapid breathing; and for the first time called a physician, who immediately sent her to the hospital.

Within the next forty-eight hours she had a temperature of  $103^{\circ}$ ; the eye became proptosed from a massive non-fluctuating intra-orbital tumor; the left optic disc was markedly choked and soon became invisible because of an intervening exudate. The globe developed a stony hardness from increased intra-ocular pressure, and the left side of the nose became occluded by a scabby adherent mass. The right eye remained normal.

During the same time she became apathetic and drowsy. There developed a questionable left facial paralysis but a definite and complete right hemiplegia.

She remained acutely ill for two weeks, after which the fever and pain disappeared and she began to improve generally, although the left eye was completely destroyed and the right hemiplegia remained.

After thirty-three days of hospitalization she was removed to another part of the state and was not seen again by the author until March 2, 1938, or seven months following the onset of her symptoms.

**EXAMINATION.** *Negative Findings.* The abdomen, pelvis, heart and lungs were normal, the latter being confirmed by x-ray examination. The blood pressure remained at about 120/70. The blood studies were always normal, never showing any dyscrasia, but the white blood count of 8200 did not increase with the elevation of



temperature. The hemoglobin remained at 80 per cent. The Wassermann and Kahn tests were negative.

The anterior sinuses were definitely clear on transillumination, with the exception of the left frontal, where the light was obstructed by the superficial swelling. We note specifically that the left antrum was clear.

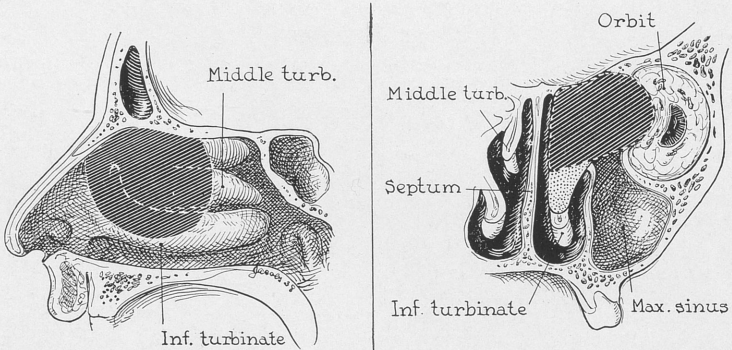


FIG. 1.—Sagittal and cross section of the area involved. The shaded area represents the extent of the process.

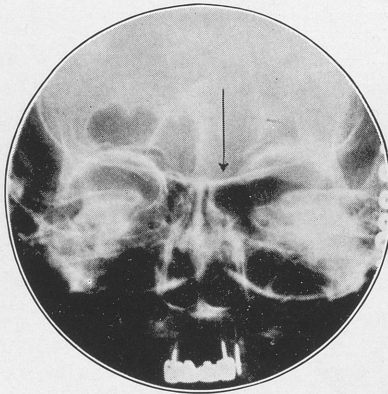


FIG. 2.—Area of bony absorption, indicated by the arrow.

*Positive Findings.* 1. The patient was admitted with a temperature of  $97^{\circ}$  which rapidly rose to remain at  $102^{\circ}$  to  $103^{\circ}$  for seven days, lowering to between  $99^{\circ}$  and  $100^{\circ}$  for the succeeding seven days, and

then coming down to normal for the remaining eighteen days of her hospitalization.

2. The urine showed sugar 4 plus, acetone 4 plus, diacetic acid 4 plus.

3. The blood sugar was 298 mg.

4. There was present within the left side of the nose, the left ethmoid and left orbit, a rapidly growing necrotic tumor which within forty-eight hours showed evidence of involving the meninges and left frontal cortex.

5. On the fifth day following the onset there was a complete motor paralysis of the right side of the body.

6. Biopsies were made of tissues taken from the left lateral wall of the nose and later from the intra-orbital mass. The laboratory reports concerning these specimens were as follows:

(A) Tissue from the nose:

"The specimens are formed of necrobiotic tissue in which are some glands, areas of abundant nuclear debris and perhaps some broken-down inflammatory cells. A fairly large number of septate and branching mycelia of a fungus are infiltrating almost every part of the specimen. The fungus cannot be definitely identified; it is probably a monilia or *Aspergillus*. Cultural study is necessary. There is no evidence of malignancy."

(B) Tissue from the orbit:

"The specimen is formed of a necrobiotic muscle, fat tissue and a few nerve bundles. Some small areas of inflammatory reaction can be recognized. There is no evidence of malignant tumor. The fungus described in the tissue from the nose is present and is infiltrating all tissue. It is quite numerous along the nerves. There is no doubt in my mind that this fungus is responsible for the necrobiotic and inflammatory swelling. Cultural study is necessary to identify the type."

7. The x-ray report was as follows: "There is a definite increased density in the left ethmoid region with a blurring and loss of cell outline; the remaining sinuses are clear. There is an increased density within the left orbit."

DISCUSSION. With these findings before us, we review the following points:

1. The patient was a severe diabetic.

2. She had lost 28 pounds within four months, with nothing to explain it except the diabetes.

3. She denied having had any illness until the onset of her present symptoms and specifically states that she had nothing wrong with her nose, sinuses, or eye.

4. In an apparently normal individual, then, only five days elapsed from the onset of her first symptom, namely, a pain over the left eye, until she had a non-suppurative, rapidly developing, fulminating process, characterized by the formation of a necrotic mass occluding the left side of the nose, the left ethmoid, the left orbit, and apparently the left side of the intracranial cavity.



FIG. 3.—Paralysis of the right arm and shoulder.

5. Within the same period of five days she had lost the left eye and developed a complete motor paralysis of the right side of the body.

6. Within twenty-one days all acute symptoms had subsided, leaving as their sequelæ a receding mass in the left nasal fossa, ethmoid and orbit, a complete loss of the left eye, and a total right-sided motor paralysis.



TREATMENT. 1. Insulin was given every day. Although 160 units were administered the first day, the average daily dosage was about 75 units.

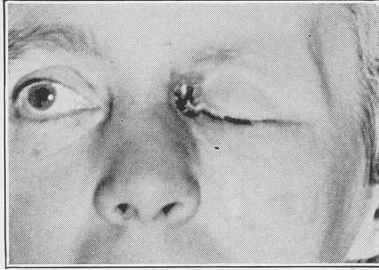


FIG. 4.—Sequestrum in situ.

2. Deep  $x$ -ray therapy directed anteriorly at the left orbit was given in doses of 210 R units on eight different occasions during the first two weeks.



FIG. 5.—Sequestrum in situ—the eyeball degenerated and retracted from the lamina papyracea.

3. Potassium iodide as a saturated solution was given in increasing dosage for seventeen days, after which a rash appeared and it was discontinued for eight days. Beginning again with minimum doses, she developed another rash after four days, and the treat-

ment was discontinued. The initial daily dosage was 2 grams, the maximum 9 grams.

4. Of the miscellaneous forms of therapy there were administered:

- (A) Antimony and potassium tartrate, 5 cc., on two occasions.
- (B) Fifty per cent glucose for insulin reaction.
- (C) Sedatives and other symptomatic treatment.



FIG. 6.—Sequestrum removed.

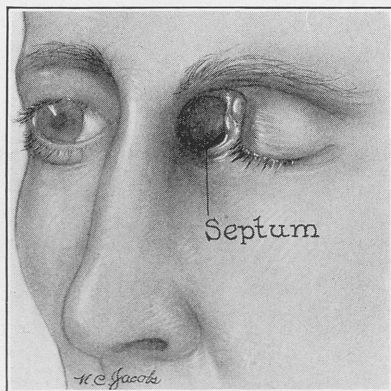


FIG. 7.—Pencil sketch of the patient made three weeks after the removal of the sequestrum.

*Present Condition.* Eight months following the onset, the condition of the patient is as follows:

She can walk about the room but is for the most part confined to an arm-chair. The left eye is collapsed, degenerated and shrunken

away from the nasal wall of the orbit. There is a perforation about  $\frac{1}{2}$  inch in diameter in the anterior portion of the nasal aspect of the

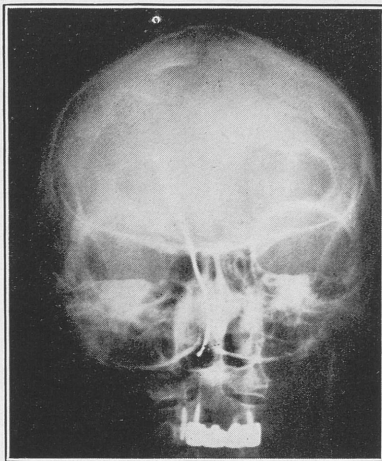


FIG. 8.—A probe inserted through the orbital perforation in the external naris.

orbit which communicates with the nose. This was partially occluded by a sequestrum but there were no signs of active inflammatory process

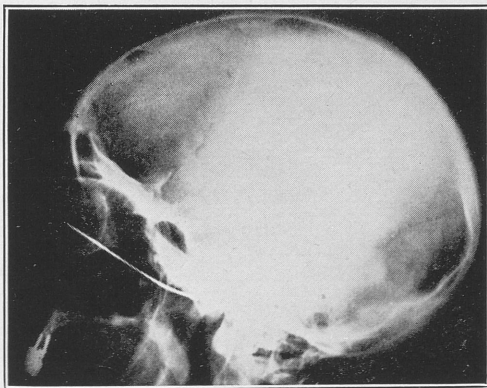


FIG. 9.—A probe inserted through the orbital perforation into the nasopharynx.

nor of malignancy. Inspection of the nose revealed the same sequestrum in the left middle and inferior meati.



A part of this degenerated bone was removed through the orbital perforation and seemed to be a cast of the remainder of the left ethmoidal labyrinth and left middle turbinate. Manipulation through the left side of the nose resulted in the removal of more sequestrum until the whole area was clear, and the underlying tissue appeared to be normal.

A light was passed through the orbital perforation into the nose and inspection under strong illumination was carried out.

The right arm and shoulder are still completely paralyzed. The right leg functions with a spastic gait but otherwise the right muscular function has returned. All right reflexes are increased. There are no sensory changes and no symptoms of intracranial pressure.

COMMENT. 1. We are assuming that this condition was a fulminating monilia or *Aspergillus* infection because the original bacteriological studies showed an abundance of typical microscopic forms and nothing else to account for the condition. On the other hand, reculture of this material on regular and special media showed no growth, consequently we lack this verification.

2. We do not know the exciting cause nor what part the diabetes played.

3. We do not know why the process was arrested. Certainly the treatment was empirical.

4. We do not know what happened intracranially, but the supposition is that there was a localized lesion in the meninges and the posterior aspect of the frontal lobe. In any case, the left motor area was involved, whereas the adjacent sensory and speech areas were not involved.

5. The prognosis so far has shifted three times.

(1) During the acute stage it was considered hopeless.

(2) During the convalescence from the acute stage it was considered fair for recovery from the acute condition but with the possibility of recurrence. The left eye was destroyed and there was a complete right hemiplegia.

(3) At the present time the possibility of an acute recurrence seems doubtful. There is a permanent loss of the left eye. In spite of the failure of a demonstrable motor function in the right arm and shoulder, it seems possible that all motor function might return.

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## DISCUSSION

DR. JOHN H. FOSTER, Houston, Texas:

There is nothing I can add to what Dr. Nash has said in discussing this case. Just to add to the record, I have recently had the misfortune to see a fatal case of monilia infection of the pharynx. The superficial type of mycotic infection, of course, is common in our portion of the country.

I have seen *Aspergillus niger* in the antrum, and other types of superficial infection. Recently, however, a general surgeon asked me to see a patient whom he had treated three weeks previously for peritonsillar abscess. He had incised it under general anesthesia and

evacuated a large amount of pus. In spite of that, the patient continued to suffer great pain over that side of the face and head, and the sinus continued to discharge. When I saw it, there was an opening through the superior pole of the tonsil leading up about an inch. This opening was enlarged, without any apparent effect on the condition. So I advised removal of the tonsil in order to secure better drainage, and did remove it. At that time I found a sinus leading upward to the base of the skull. I noticed then that the discharge was filled with flaky material, so I had this examined, and my technician reported that it was a fungous growth. This was later identified as a monilia. The patient was then put on heavy doses of iodide and given a vaccine which was advised by some for this condition, but in spite of that, the patient continued to suffer pain. Another sinus developed, going backward through the muscles up to the tip of the petrous portion of the temporal bone.

The patient gradually went down, developed intracranial symptoms and died of paralysis of respiration.

DR. HARRY WEBSTER LYMAN, St. Louis, Mo.:

I should like to ask Dr. Nash if the paralysis was of sudden onset, or did it come on gradually and progress?

DR. C. STEWART NASH (in closing):

The reason that this paper was presented was that I had never seen nor heard of just this particular condition. The librarians spent a considerable amount of time searching for any such thing in the literature, and they were unable to find it.

In regard to the paralysis, whether it was sudden or slow, I shall have to answer that in terms of time. There was a questionable facial paralysis on the opposite side which was either going to develop or to arrest, and that took place over a period of twenty-four hours. So far as the paralysis on the left side was concerned, that was about thirty-six hours in developing.



## CARCINOMA OF THE LARYNX AND TOTAL LARYNGECTOMY\*

S. J. CROWE, M.D.

AND

EDWIN N. BROYLES, M.D.

THERE are only two methods of treating a patient with carcinoma of the larynx. One is irradiation with  $x$ -rays or radium. This is a valuable aid in the treatment of malignant growths, but should be used only in inoperable cases or to supplement operative removal. At the present time the results of ray therapy are so erratic and unpredictable, that whenever the diagnosis of cancer has been established from the history, examination and biopsy, and the growth is still localized and can be safely removed, operation should not be postponed. The fact that the majority of carcinomas of the larynx are of the squamous cell, pearl forming variety, which is very resistant to radium treatment, adds force to this argument. We have all seen tumors of the tonsil or larynx permanently cured with irradiation therapy, but we have also seen tumors in the same location and with the same histologic appearance which were improved or even temporarily disappeared, only to recur locally or in the adjacent lymph glands. If this irregular response is due to a variation in the dosage, the method of application or the screens used, then irradiation treatment is still in the experimental stage. There are many patients with inoperable conditions, but until the results are more consistent and encouraging in these cases, I am opposed to limiting the treatment of an operable new growth in the larynx to  $x$ -ray or radium.

The other method is surgical removal. The general principles of the operation are the removal of the growth with an adequate margin to insure against recurrence and to preserve as far as possible the voice and normal channels for breathing and swallowing.

\* From the Department of Otolaryngology of the Johns Hopkins University and Hospital.

In carefully selected cases, laryngo-fissure is the operation of choice. By laryngo-fissure we mean removal, in one piece, of the affected vocal cord including its attachment to the arytenoid cartilage, the entire anterior commissure area and a large portion of the thyroid cartilage. It would perhaps be more accurate to describe this operation as excision of a growth in the larynx by the laryngo-fissure route. It differs from a hemi-laryngectomy, in that the posterior half of the cricoid is not removed. If the perichondrium is carefully preserved and, after the affected half of the larynx is removed, stitched over the exposed neck muscles, it forms a smooth lining, prevents granulations and increases the chances for a good voice. Removal by the laryngo-fissure route is contraindicated if the growth involves the anterior commissure, has fixed the true cord, or has spread to the ventricle or false cord. In all borderline cases where there is any doubt about the extent of the growth, the entire larynx should be removed.

As a result of our experience we have evolved the following one-stage operation for total laryngectomy which is simple, free from shock and other postoperative complications. The success of the operation depends on the most rigid adherence to the following rules:

1. The growth must be operable or intrinsic. The operation we propose is not suitable when the cartilages are invaded, the false cord is involved or the growth has spread to the muscles and glands of the neck.
2. The general physical condition must be such that the patient is a reasonably good operative risk.
3. Great emphasis is placed on preoperative elimination of infection in the sinuses, tonsils and particularly the teeth and gums, but we do not remove all teeth, good and bad, as they do in England.
4. The anesthesia is a combination of avertin, procaine and chloroform. Avertin relieves the patient of much anxiety and if not more than 75 mg. per kilo of patient's weight is given, it in no way interferes with the postoperative cough reflex. Very little chloroform is necessary and its use almost entirely prevents bronchial irritation and excessive mucus secretion after the operation. If the growth interferes with breathing, a tracheal tube is inserted

under local anesthesia before avertin is given; if not, the trachea is opened at a later stage in the operation.

5. The operative procedure is based on sound surgical principles. It is as simple as possible. Every effort is made to avoid traumatization with clamps, retractors or sutures; bleeding is controlled with ligatures instead of the high frequency cautery, and a closure is made that prevents leakage of mouth secretions into the wound or trachea. We avoid pockets or dead spaces; we use the smallest size chromic catgut, and the only drain left in the wound is a piece of rubber tissue smaller than a pencil, flattened and placed in the midline. A little of this drain is pulled out and cut off each day and removed entirely on the third day after operation. The wound is not probed, suctioned or irrigated because these procedures introduce skin organisms. Wound infection and a prolonged, uncomfortable convalescence often follow the use of a cutting or coagulating current.

6. Before the mucous membrane flap closing off the pharynx is sutured in place, a small, soft rubber catheter is passed through the nose and into the esophagus. This catheter extends only half-way down the esophagus. Fluids are given intravenously during the first twenty-four hours, but after this, fluids and a high-caloric diet are introduced into the catheter with a syringe. On the third day after operation, if there is no evidence of leakage or wound infection, the feeding tube is removed and the patient given a soft diet.

During the operation the foot of the table is elevated about 18 inches, and, in addition, suction is used to prevent aspiration of blood or mouth secretions. After the operation the patient is made as comfortable as possible in bed, with two or more pillows under his head. A special nurse is required to keep a moist cloth over the tracheotomy tube and with suction to aid him in expelling mucus from his trachea. During the postoperative period these patients need rest, and too much nursing and too many dressings are often detrimental.

The wound is tightly closed with the exception of one small drain in the midline but this is carefully watched, and at the first sign of infection, the skin sutures are removed and the superficial layers opened. This occurs with surprising infrequency.

If the operation is properly done there is no loss of blood; no shock; the patient usually sits up or gets out of bed on the second day; the feeding tube is removed on the third day and he is usually able to leave the hospital two weeks later.

The following illustrations show the operative technique. The incision is in the midline and extends from the hyoid bone to the lower border of the isthmus of the thyroid gland. (Fig. 1). The first step in the mobilization of the larynx is to remove the peri-

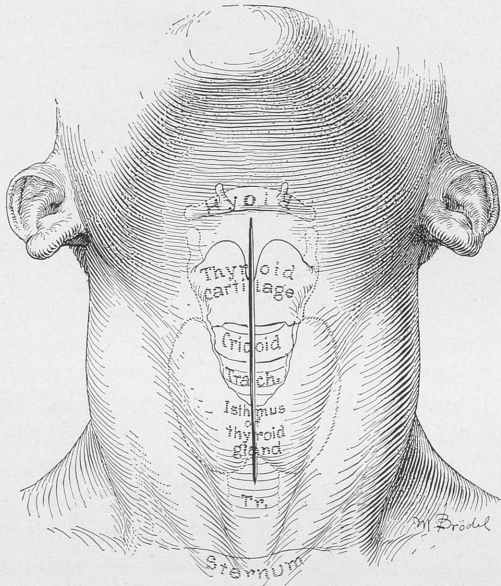


FIG. 1.—The incision is in the midline and extends from the hyoid to the lower border of the thyroid isthmus.

chondrium with as little trauma to surrounding tissues as possible. (Fig. 2.) The isthmus of the thyroid is then divided and sutured. (Fig. 3.) A tracheal tube is inserted through a transverse incision and far enough down to avoid a subglottic extension of the growth. The inner of the two tracheotomy tubes is used during the operation because it is smaller and interferes less with the subsequent operative procedure. The anatomical drawing shows the relation of the superior thyroid artery to the cornu of the thyroid cartilage.



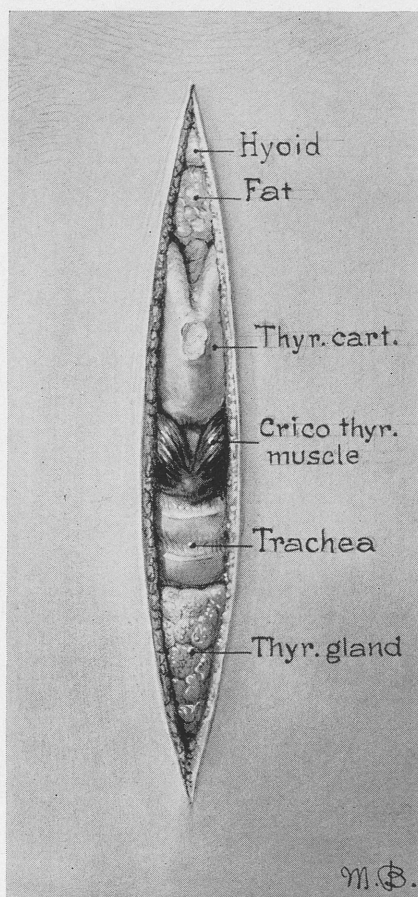


FIG. 2.—The sterno-hyoid and sterno-thyroid muscles are gently pushed aside exposing the thyroid cartilage, crico-thyroid muscles, trachea and thyroid gland.

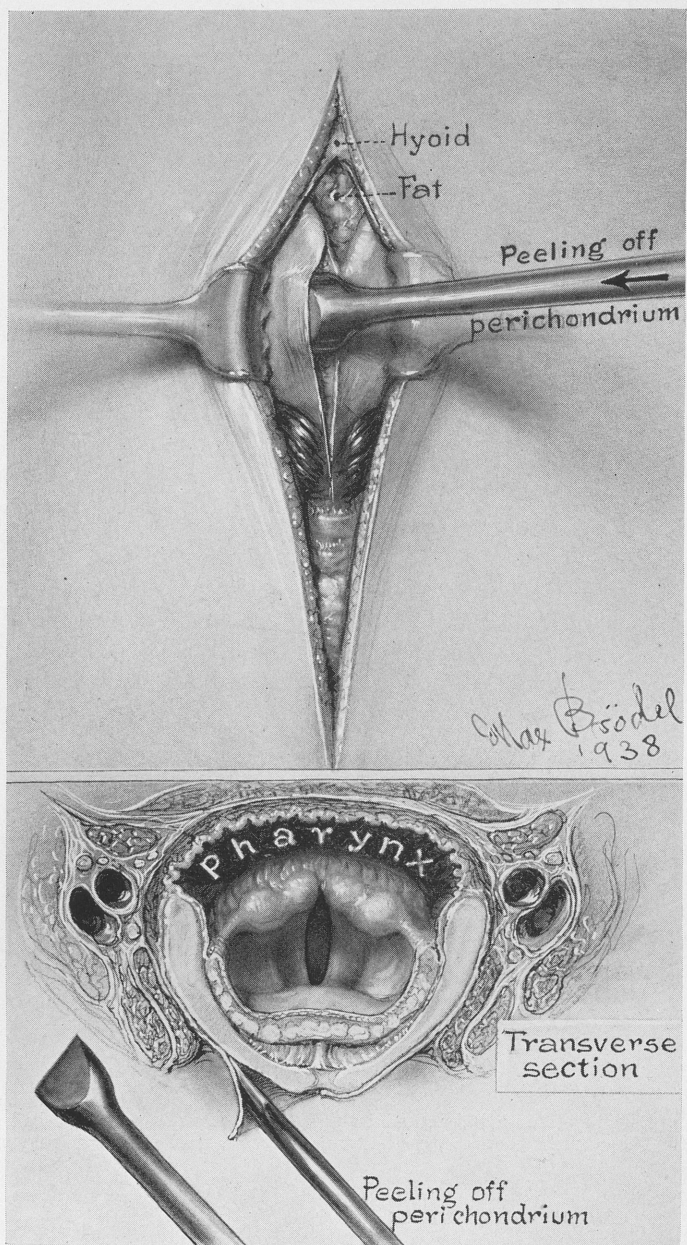


FIG. 3.—To avoid crushing or tearing the neighboring muscles and fascia the larynx is skeletonized by peeling off the perichondrium, and everywhere making the dissection as close to the trachea as possible.

(Fig. 4.) By making the dissection as close to the larynx as possible, injury to this artery and to the adjacent tissues deep in the neck is avoided. The larynx is gradually freed by incising the

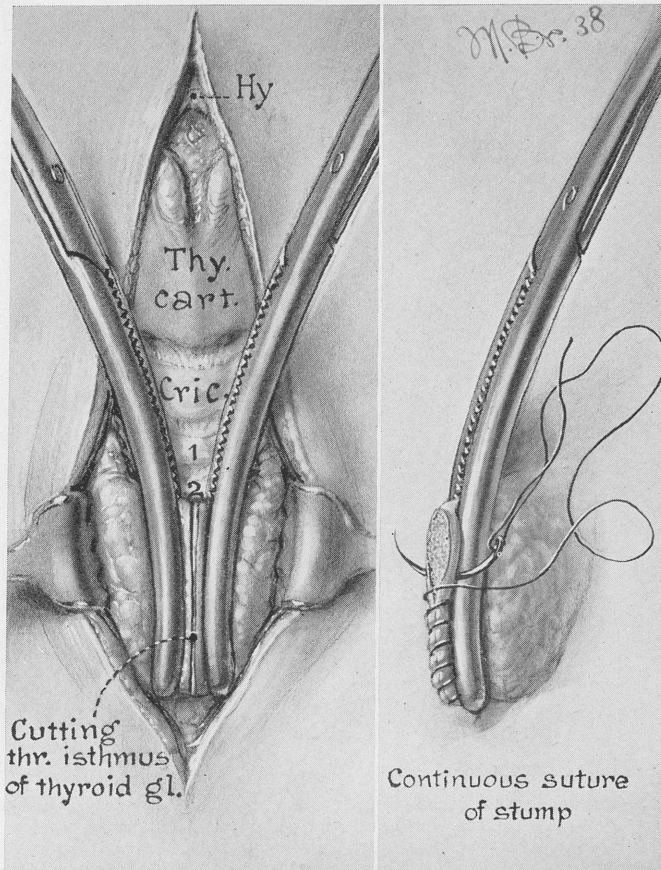


FIG. 4.—Method of dividing and ligating the isthmus of the thyroid gland.

thyro-hyoid membrane, packing the interior of the larynx and pharynx with gauze to prevent aspiration and finally dividing the cornu and ligating the superior laryngeal artery. (Fig. 5.)



The most important part of the operation is the closure of the pharyngeal defect. The mucous membrane on the posterior sur-

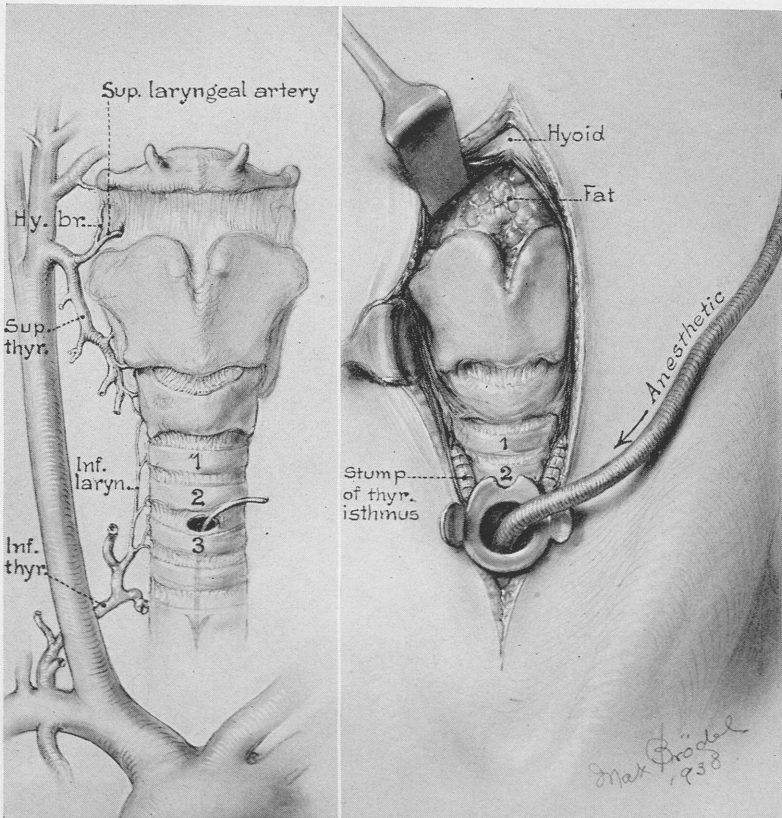


FIG. 5.—The anatomical drawing on the left shows the relation of the superior thyroid artery to the superior laryngeal and other vessels supplying the larynx; also the level of the transverse incision for the tracheal tube. On the right is shown the tracheal tube; the small rubber tube through which chloroform vapor is blown; the thyroid isthmus which has been divided and sutured, and the retractors placed for exposure of the cornu of the thyroid and ligation of the vessels lying deep in this angle.

face of the larynx is dissected off, as shown in Fig. 7. The larynx is then removed and the stump of the trachea anchored to the surrounding tissues. A series of interrupted sutures are placed to



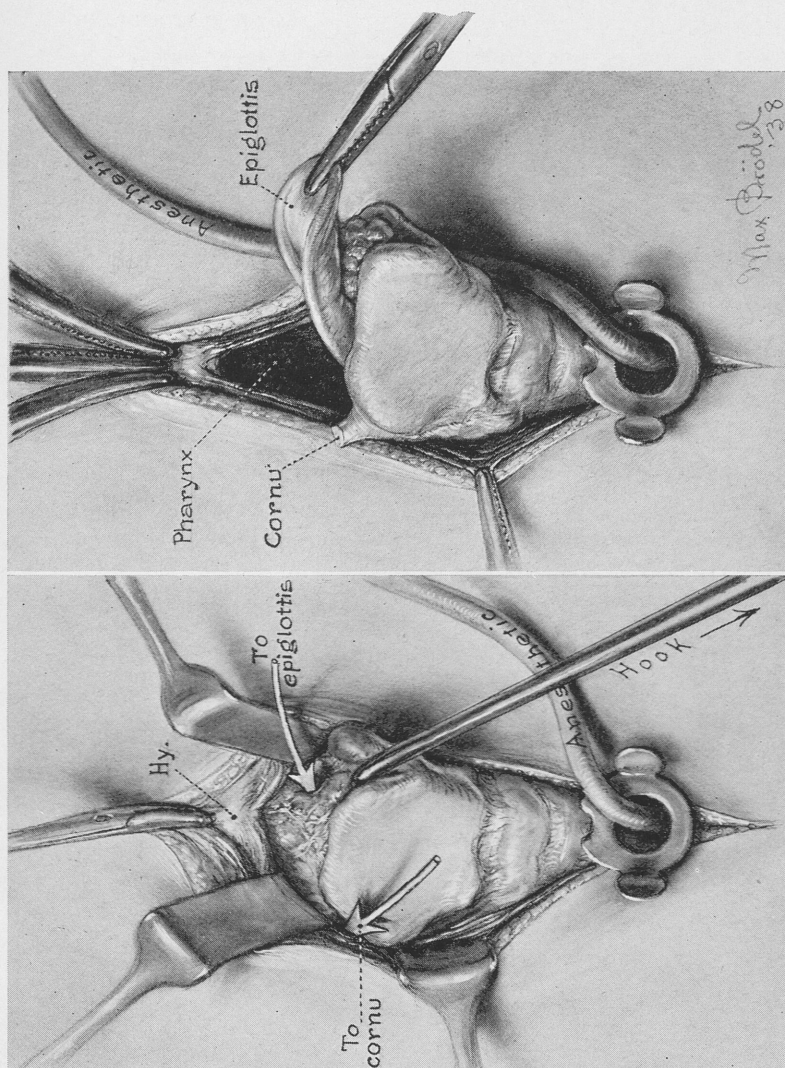


FIG. 6.—By rotating the larynx the cornu first on one side then the other is exposed and cut, the artery and vein ligated with chromic catgut (size 00), and the pharynx opened by dividing the thyro-hyoid membrane. The interior of the larynx is inspected for the subglottic extension of the growth, after which the pharynx and larynx are both packed with gauze to prevent aspiration of blood or mouth secretions.

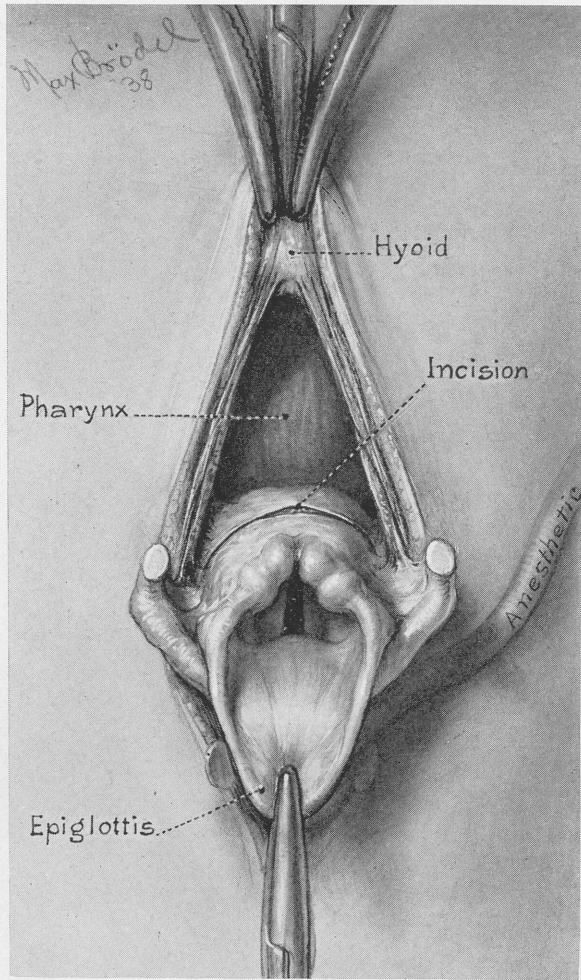


FIG. 7.—The mucous membrane on the posterior surface of the larynx and in the pyriform sinuses is carefully removed and used to close the pharyngeal defect.

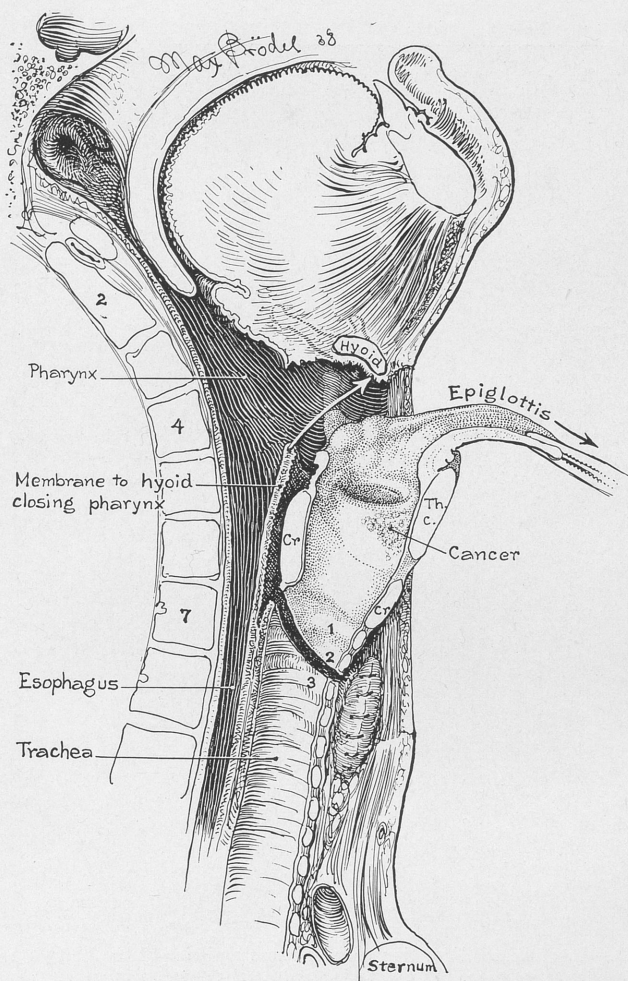


FIG. 8.—To show how much is removed (the epiglottis, the entire larynx, a part of the cornu of the thyroid cartilage and a part of the first and second tracheal rings) and how much is left in place. In making this dissection every effort is made to avoid injury to surrounding tissues and the formation of pockets.



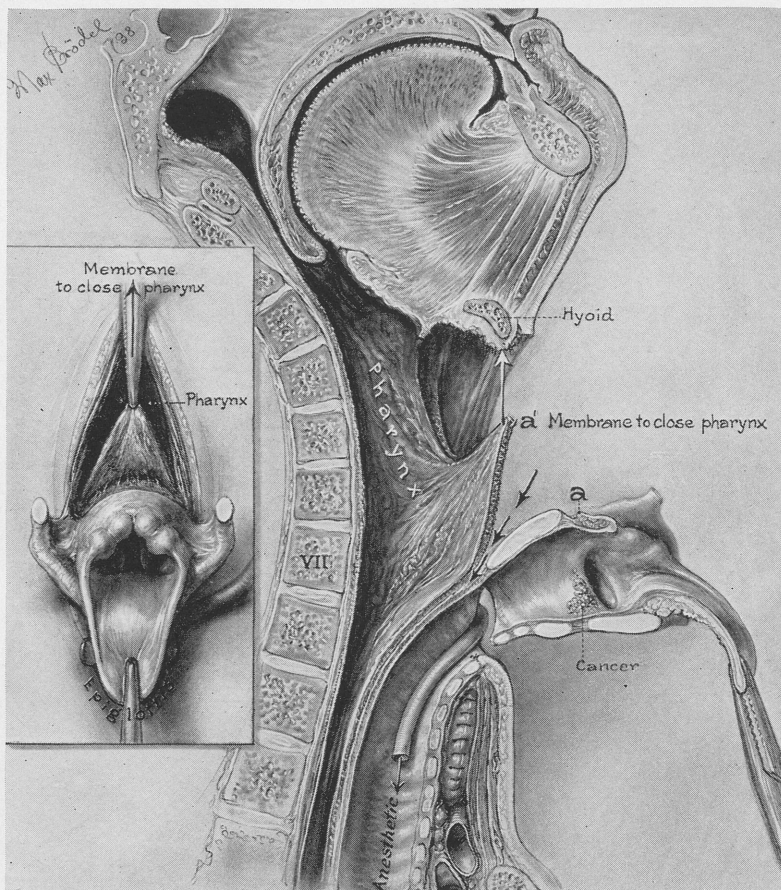


FIG. 9.—Inset shows dissection of membrane and size of the pharyngeal defect to be closed. Just before the larynx is cut free the trachea is pulled forward and anchored to the surrounding muscles as shown in Fig. 10. Chromic catgut (size 0) is used.



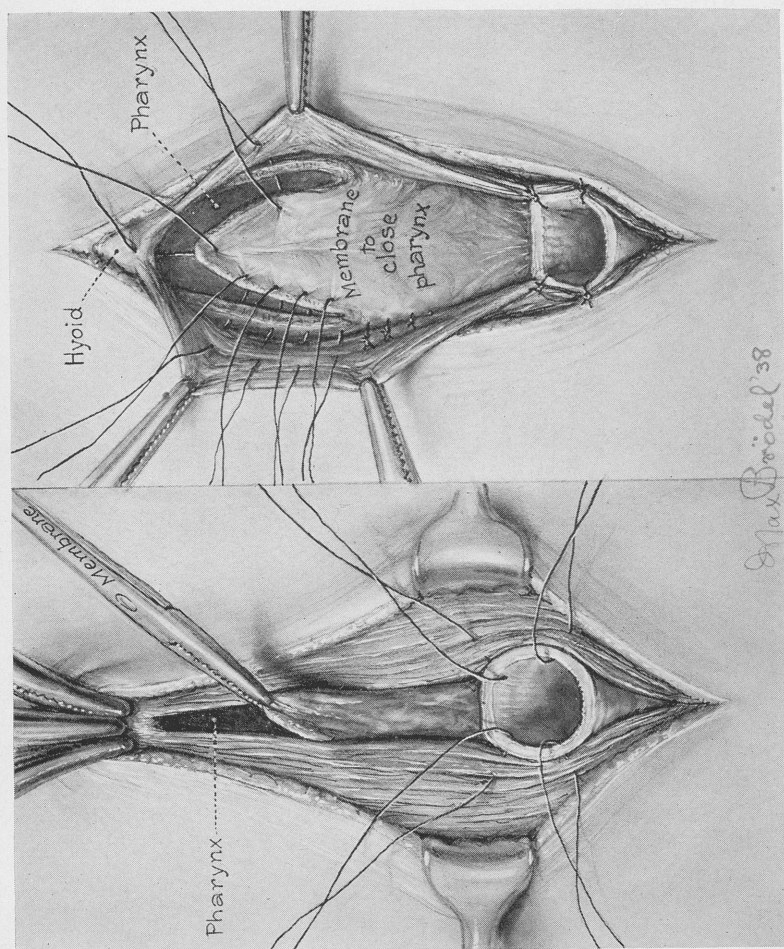


Fig. 10.—The most important feature of the operation is shown on the right: interrupted chromic catgut sutures (size 00) include the mucous membrane flap, the mucous membrane of the pharynx and a little of the undersurface of the sternohyoid muscle. When tied the muscle covers the line of incision and prevents leakage of saliva or food into the neck wound. To get this result, however, the defect must be closed without tension on any of the sutures.

include the mucous membrane of the pharynx, the mucous membrane flap and the under surface of the sterno-hyoid muscle. (Fig. 10.) When tied, the line of suture is everywhere covered with muscle. This is further reinforced by drawing the free surface of these muscles across the midline and suturing them in two

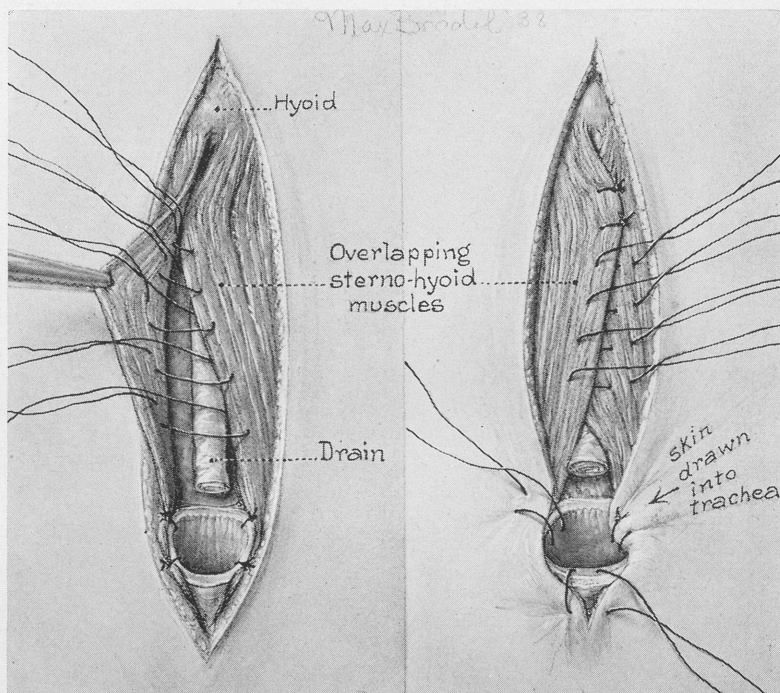


FIG. 11.—One small, flattened roll of rubber tissue is placed in the midline. This is the only drain necessary since no pockets or dead spaces have been left anywhere in the neck. The wound is then reinforced by overlapping the sterno-hyoid and the remains of the sterno-thyroid muscles. All subcutaneous fat is removed and the skin drawn into the trachea and loosely sutured. The tracheal tube, thickened with tape, as shown in Fig. 12, holds this skin in place.

layers. (Fig. 11.) The skin around the tracheal opening is freed from subcutaneous fat and stitched into the trachea where it is held in place by the tube. (Fig. 11.)

The incidence of postoperative complications, shock, pneumonia, cough and profuse mucopurulent tracheal discharge are in direct proportion to the care with which the above rules are followed.

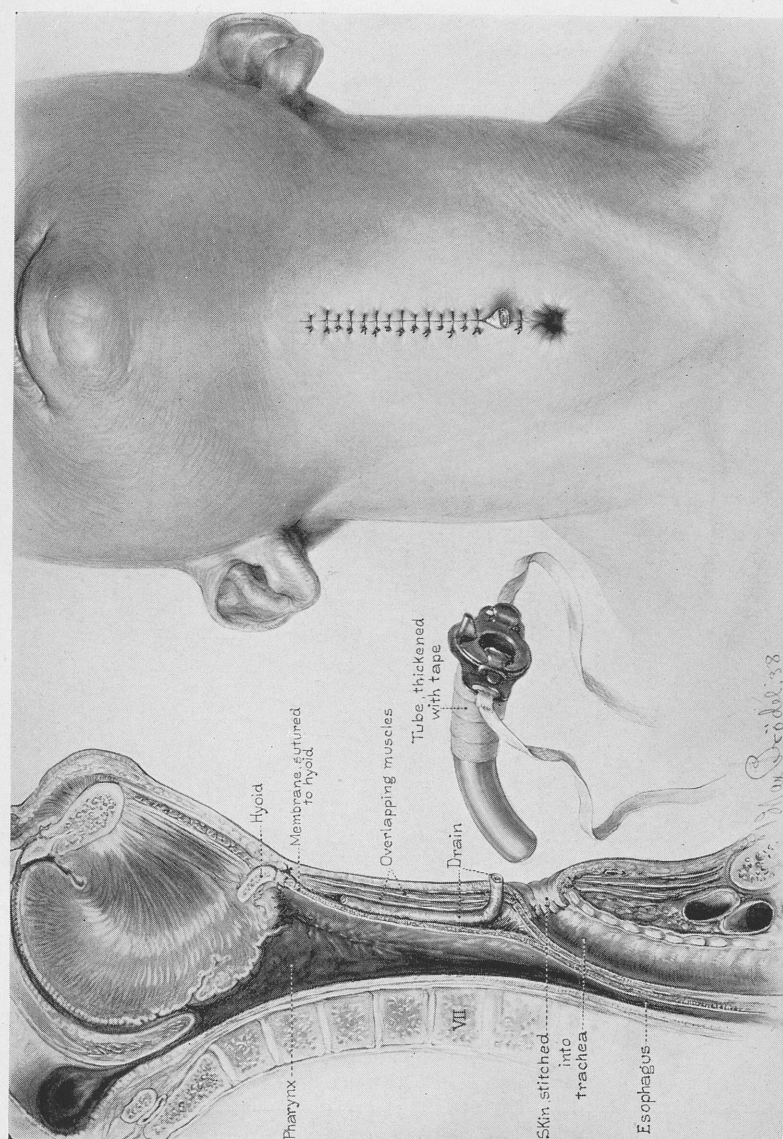


FIG. 12.—The wound is closed but the skin incision must be reopened at the first sign of infection. If there is no infection the rubber tissue drain is gradually withdrawn and removed entirely on the third day. If the tracheal opening ever becomes too small the skin around the lower half is elevated and the underlying anterior wall of the trachea removed with a high-frequency cutting current.



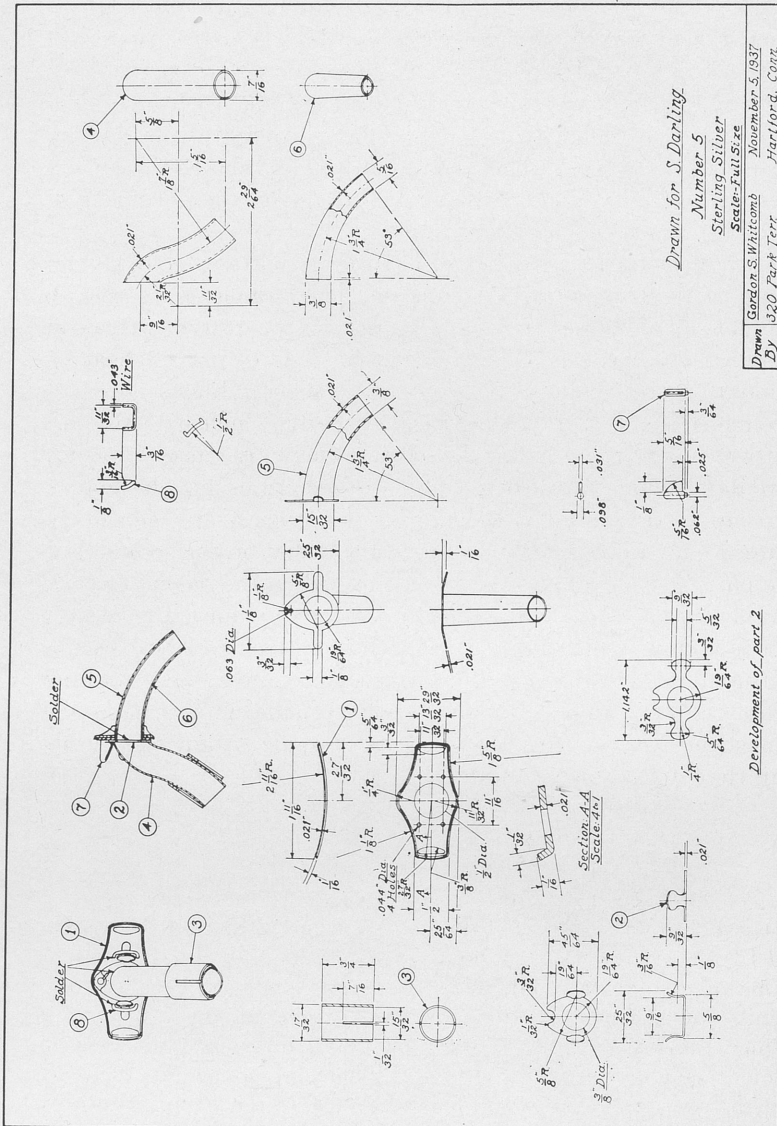


FIG. 13.—Details for the construction of a tracheal tube to be used with an artificial larynx. It was designed by a patient who finds it very comfortable and convenient.



SUMMARY. The cases for operation must be carefully selected. When the growth has invaded the cartilage, muscles, or glands we use irradiation therapy. All intrinsic malignant growths are removed by the laryngo-fissure route or total laryngectomy. The involvement of the anterior commissure or fixation of the cord are the deciding factors. The end-results of laryngo-fissure, which does not include the entire thyroid cartilage on the affected side, have been disappointing in our hands.

After the selection of the case the other important factors which insure a good result are: simple surgical principles as regards traumatism of tissues, hemostasis, and postoperative treatment; great care to close the pharynx tightly so as to prevent leakage of mouth secretions into the wound or trachea. Fluids are given through a small, soft rubber nasal catheter which extends only half-way down the esophagus. This catheter is removed on the third day if there is no leakage of mouth secretions into the wound. Our impression is that a feeding tube is not necessary, and in the future we intend to omit it and give a soft diet on the second day. The patient is encouraged to get out of bed on the second or third day; a special nurse is in constant attendance until the feeding tube is removed and longer if tracheal secretions are troublesome.

If used at all, x-ray or radium treatment is given after and not before the operation. The dissection is made with a knife and hemostasis secured with the smallest chromic catgut ties rather than the high frequency cautery.

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#### DISCUSSION

MR. VICTOR E. NEGUS, London, England:

It is kind of you, Mr. President, to ask me to discuss this paper, and I have great pleasure in doing so.

One of my greatest difficulties in recent years has been to form definite conclusions as to the correct indications for the treatment of laryngeal cancer. In my country, we have a very great variety of opinions and diversity of methods. There are some men who say that the use of radiation treatment for cancer of the larynx is entirely wrong and should never be employed. We have others who say that anybody who nowadays operates on a case of cancer of the larynx ought to be put in prison. It is very difficult to get a correct balance.

I have had the privilege of being associated with Sir St. Clair Thomson, and he is a man of open mind. Some years ago he was strongly prejudiced against radiation treatment, but not now, and we had to make some definite indications in reëditing his textbook. If I may, I will present them to you.

One point of interest to start with is, I think, the question of anesthesia. I was educated in Kings College Hospital, London, a hospital which was honored by the presence of Lord Lister as one of its surgeons. Lord Lister used no anesthetic except chloroform given on a mask by the drop method. I happened to be house surgeon to Sir Watson Cheyne, who himself was house surgeon and assistant to Lord Lister, and, therefore, the tradition is very close to me. Sir St. Clair Thomson was also house surgeon to Lord Lister, and naturally these methods, which were inculcated in early days, have had subsequent influence.

In operations on the larynx, whether by the laryngofissure route or total laryngectomy or lateral pharyngotomy, I have tried different means of anesthesia. One always thinks that possibly one may get into a groove. I have at times digressed slightly from the use of chloroform, using ether, given rectally in oil, and also avertin, and have come back again to what I was taught, that chloroform is best.

I had the opportunity of seeing Dr. Crowe operate on a patient who had been given some avertin beforehand, and the patient at the end of the operation seemed to be in a light state of anesthesia and to have the cough reflex present. We have been afraid of using any premedication because of the possibility of diminishing the natural defenses of the air tract, and that has also applied to the use of atropine, with its effect of drying up the bronchial secretions.

So much for the question of anesthesia. As to the indications for treatment, there are in my practice three methods of choice.

(Slide.) The first is excision by the laryngofissure route. I think the details of this operation vary slightly with different surgeons, but as I have seen it with Dr. Crowe, the area removed is identical. If a patient with a growth of this type came to me and I was satisfied that it was in Group 1 or 2 of Broder's classification—and this appears to me to be highly important in deciding on the right treatment—I should operate on that patient by the laryngofissure route and should remove a wide area of tissue, irrespective of the size of the growth. The contraindications for the operation would be that the growth was of the aplastic type in Group 3 or 4, or that it extended onto the arytenoid. There would unlikely be any metastasis in a growth of

this type. The fixation of the cord would not deter me from carrying out the operation.

(Slide.) This is a case of Sir St. Clair Thomson's which I show, where there is a very early type of growth and a patch of leukoplakia, as you see, with early malignant changes, but the area of infiltration by malignant disease is extremely small.

(Slide.) Most of that is a leukoplakial patch, but it shows the area removed, and in this operation, as we perform it now, the area would never be less. In this way the whole of the ventricular band and part of the aryepiglottic fold are removed, as far forward as the anterior commissure, sometimes taking the tip of the opposite cord; also going far down, if necessary taking away a small piece of the cricoid cartilage, and back sufficiently far to take the vocal process of the arytenoid with the growth; as we perform it now, we should not take out any less.

I had the privilege of seeing cases in Dr. Jackson's Clinic where the functional results were extraordinarily good, the glottis widely open, with good movement of the cord on the side of operation, excellent voice; and naturally, if one can obtain the desired result and can cure the patient by taking a slightly less extensive area away, that would be all to the good. But the influence of Sir St. Clair Thomson and of what I have seen myself has made me carry out these rather extensive operations in all cases.

(Slide.) This result was very good, even with wide excision, and one saw the patient later on, with an excellent voice and with a regenerated cord. There are many cases like this, so that the operation seems to be one that is satisfactory from a functional point of view, and satisfactory as regards the prevention of recurrence.

It is an operation that we hesitate to discard in favor of any other method, without some strong reason.

We remove the ala of the thyroid cartilage, so that the perichondrium shall form the inner face of the operative wound. One or two surgeons in England have advocated leaving the cartilage as a barrier against the possibility of recurrence, but, personally, I have removed it, as the healing process and vocal results seem to be better.

(Slide.) Here is a growth, where the carcinomatous area is extremely small, and anything so drastic as laryngectomy would appear to me to be entirely unnecessary.

(Slide.) Within a few sections taken away there is only commencing invasion, which can scarcely be diagnosed as malignant.

(Slide.) So we have a very definite indication for excision by the laryngofissure route. There are types of cases where the operation

would be quite unsuitable. Here is shown a man, sixty years of age, perfectly healthy, with a carcinoma extending along the whole of the cord, and reaching the posterior commissure. There are only two possibilities in his case. I do not think he would be suitable for the operation of Hautant and Ombredanne, and in my experience it is necessary to advise that he should have removal of his larynx or, alternatively, have treatment by radiation.

One indication which would decide in favor of irradiation treatment would be if that growth were of Type 3 or 4 (and we do see a certain number of Type 3 where the results of laryngectomy, in my experience, are bad) I should not hesitate to treat the patient by irradiation; but if it were Group 1 or 2, I should offer that possibility to the patient, and have carried out the treatment in some cases with success, as an alternative to laryngectomy.

This particular patient shown is very well, and I have a number who have remained well.

As to the exact method of treatment, I think that is rather less important. The method by which this man was treated was the interstitial implantation of radium needles.

(Slide.) That does not give quite a correct idea of the radium needles. They look to be out of place, but they are quite regularly arranged in reality. These needles were left in for a week.

(Slide.) Certain of the cases have been treated with telerradium, some with a 5-gram unit, and others with a 1-gram unit, and there have been some good results. Deep x-ray treatment also has been given by the protracted fractional method. But the interstitial use of radium, which has been particularly developed by Mr. Douglas Harmer in my country, is a satisfactory one, with little upset to the patient, and it has given some excellent results. I think it has a place, although it does not compete in my practice with operation, because the indications are different.

This is another case of an old man of eighty-two years who would have been quite unsuitable for laryngectomy. He was treated with interstitial radiation, with disappearance of the growth in a very short time, and with permanent freedom.

(Slide.) Here is a patient with a carcinoma at the front end of the cord, which was removed, and the result was quite good. The anterior end of the opposite cord had to be removed with a certain amount of narrowing; four years later the patient showed proliferation of the epithelium with hyperkeratosis and chronic inflammatory changes in the whole of the larynx. The probability was that malignant changes



were again starting, and as the disease was rather active it was treated with distance radiation, with good result. This form of treatment, I think, has its place.

(Slide.) Lastly, a word about total laryngectomy. If a patient came with a growth involving both cords, in Group 1 or 2, without obvious glands, he would be treated by total laryngectomy, and not by irradiation. If the growth were affecting both cords and was in Group 3 or 4, it would be treated by irradiation.

There is one slight difference in the technique. I have been in the habit, of late years, of using the incision of Sorensen of Gluck's Clinic in Berlin, making a flap with two lateral and one transverse incision, turned up so that the line of sutures in the pharynx does not correspond to the line of sutures in the skin flap. The operation is more extensive than that which Dr. Crowe has described, the perichondrium being left intact and the muscles left attached for fear of cutting through any infected areas, and particularly of touching the glands on the cricothyroid membrane. Dr. Crowe's operation is one of great beauty, I think, with excellent results, and if the cases were selected carefully, I can imagine that it has certain advantages.

Perhaps we have operated on rather more widely spread risks where this type of extensive operation appeared to be necessary.

Therefore, to sum up, we have got these three indications of excision by the laryngofissure route with a limited and differentiated growth, irradiation for the extensive growth of one cord or both cords, particularly if it is of the anaplastic type, and removal of the whole larynx for the growth of low-grade malignancy affecting both cords.

Thank you very much.

DR. CHEVALIER JACKSON, Philadelphia, Pa.:

From personal experience, I can corroborate all of the statements made by Dr. Crowe. It has been my good fortune to see two of the patients on whom he had operated by total laryngectomy, and the results were admirable in every way. His technique, as demonstrated this morning, I think is as nearly perfect as the technique of laryngectomy can be.

The objectives that he stated are ideal when one is to do a total laryngectomy. I think what he has said should be supplemented with a little further elaboration especially in the technique of laryngofissure. He did not have time, doubtless, to consider this in addition to going so thoroughly into his technique of laryngectomy. He stated that where the anterior commissure is involved laryngofissure is contra-indicated. My experience has been different in this class of cases.

Since 1913, I have had continual satisfaction from laryngofissure for malignant growth located in the anterior commissure, by modifying the technique. The incision through the skin and soft parts is made with great care and retraction is gentle, so as not to skeletonize the larynx, thus preserving all of the soft tissues in contact with the external perichondrium, so that the vitality will be conserved when the internal perichondrium is gone. Incision is then made through the perichondrium in the midline with a knife. The perichondrial incision is followed with the rotary saw, going through the ossified cartilage but not through the inner perichondrium. Then a subperichondrial dissection is done down on both sides a little farther on the more involved side without penetrating the inner perichondrium of the growth. Then excision is done with an abundant area of normal tissue around the tumor.

This modification of the operation of laryngofissure has yielded admirable results, and I think has saved a good many patients from the necessity of total laryngectomy. But please do not misunderstand me; I do not mean to cast any reflection whatever on the operation of total laryngectomy. Unquestionably and unfortunately it is indicated in most of the patients by the time we see them. There are only about 15 to 19 per cent of the patients who come in early enough for laryngofissure. The balance all require total laryngectomy or irradiation.

I feel, as Dr. Crowe does, rather adverse to irradiation in operable cases, but I am not at all sure that the last word has been said on that subject. Personally, I have had such good results from laryngofissure in early intrinsic cases that I hesitate in such a case to advise irradiation, but I am not at all sure that the day may not come when those very cases of early intrinsic disease will be treated by irradiation. That is not a forecast; I merely state it as a possibility, because I have seen clinically the work of others that leads me to question whether or not the future may see irradiation advisable in the class of cases now deemed suited for operation.

## PRESENTATION OF A COMBINED TRACHEOTOMY TUBE AND RETRACTOR

BERNARD J. MC MAHON, M.D.

I WISH to present a combined tracheotomy tube and retractor.\* This tube is used in post-tracheotomic stenosis following a high tracheotomy in which the prolonged wearing of a tube has caused an edema and hypertrophy of the subglottic tissues.

The patient for whom this tube was devised had had a tube in place, following a high tracheotomy, for six months. A low tracheotomy had been performed on two occasions, leaving a bridge between the two openings, but each time the tube returned to its original high position.

This tube was devised with three ideas in mind, primarily to retain the tracheotomy tube in a low position; second, to exert constant gentle pressure upon the hypertrophic subglottic tissue, for the purpose of hastening resorption and widening the airway; and third, to keep the wound sufficiently open so that the subglottic tissue would have maximum aëration and opportunity to return to normal, at the same time allowing constant observation of the progress of healing.

The mucosal reaction in the subglottic area, in the early stages, is hypertrophic in nature and capable of resorption, if the pressure exerted is not sufficiently great to cause a hyperplastic type of tissue to be formed. In the latter instance, scar tissue being established, the ensuing contracture is inevitable, the treatment of which necessitates many months of constant dilatation in order to permit the cannula of scar tissue to become established at a stage at which there is little danger of further contracture.

This mechanism of the process of repair of a post-tracheotomic stenosis is frequently overlooked in the treatments for the correction of this condition.

\* The technical work was done by Mr. William Fuchs in the Instrument Construction Laboratory of the Oscar Johnson Institute of the Washington University School of Medicine, St. Louis, Mo.

This tube which I am presenting is a modification of, or an addition to, the standard Jackson tracheotomy tube.

The modification consists of a monel metal plate, 3 cm. in length and 1.5 cm. in width, which is soldered to the face of the plate of the Jackson tube. There is a vertical slot, 2 cm. long by 0.5 cm. wide, in the middle of the plate, through which the inner tube may be inserted. At the top of the plate, immediately above the upper extremity of this slot, is placed a small threaded hole to receive a set screw.

Upon this plate there is superimposed a similar plate of the same dimensions, with small lips on the lateral edges along the entire length of the plate, to prevent rotation when slid upward or downward on the fixed plate. The metal which had formed the solid face of the movable plate along the extent of the slot for a distance of 2.5 cm. is turned inward at right angles at the upper extremity of the slot for 2 cm., the tip then being turned upward at right angles for 0.5 cm. When this plate is affixed to the attached plate, the retractor arm extends into the lumen of the trachea and hooks underneath the cricoid cartilage. The upturned tip of the retractor arm is inserted through a hole in the side of a conical rubber dropper bulb near its apex, which is directed upward. The conical rubber bulb, when in position, fits into and acts as a dilator upon the hypertrophic subglottic tissues.

The desired position of a movable plate is established and the plate held in this position by a set screw with a burled, wide-flanged head. Counterpressure is thus maintained between the tracheotomy tube in the inferior extremity of the incision and into the trachea, and the retractor dilator in the superior extremity acting upon the cricoid cartilage and the stenosed larynx.

The tube, removed daily for cleansing, can be reinserted without difficulty.

As the condition of the larynx improves and the tracheotomy tube tends to remain in the low position of its own accord, the extent of the dilatation is reduced, the retractor-dilator eventually removed and a standard tracheotomy tube inserted, decannulation being done at the opportune time.



## PRESENTATION OF A CASE OF RIEDEL'S DISEASE OF THE THYROID

DEFOREST C. JARVIS, M.D.

In 1928, Dr. Delavan, in his presidential address before this Association, stressed the need of further study of the significance of the behavior of lymphoid tissue in the upper respiratory tract. Since that time I have devoted considerable time to a study of this problem. After a number of varied approaches, I observed that the size of lymphoid tissue in the upper respiratory tract was related to the oxidation; so, using oxidizing catalysts of iodine and insulin, this approach was applied to lymphoid tissue and it was noted there was a decrease in size in from two days to a week.

This approach was also applied to cervical adenitis and it was noticed the cervical lymph glands decreased in size at a rapid rate. It was then applied to benign growths, like warts, and it was found that warts would disappear. It was later applied to a benign growth of the thyroid, a tumor, which I wish to present at this time as demonstrating the value of oxidizing catalysts.

**HISTORY:** Male, aged forty-five years. On Washington's birthday, 1937, while shaving himself, he discovered a small swelling on the front of his neck, just below the Adam's apple. It continued to increase in size. There has been no pain, no tenderness on palpation, and no redness of overlying skin. He has been able to shave over the growth each day.

Examination showed a tumor the size of a golf ball in the thyroid region in front of the neck. On palpation it was found to be very hard and free from tenderness. Mucous membrane of nasal septum +3 red; temperature, 98.6° F.; pulse, 87; respiration, 21; blood pressure, 120/64.

**TREATMENT:** Three drops of Amend's solution of iodine, which is one-seventh as strong as Lugol's solution, in a glass of water twenty minutes before evening meal each day. Three units of crystalline insulin subcutaneously on Wednesday and Saturday each week at 4 P.M.

(Slide.) This shows the growth taken on April 29, 1937.

(Slide.) This was taken on May 8, 1937. Decrease in size of the tissue above and below the growth makes it stand out more prominently.

(Slide.) Anterior view taken May 8, 1937.

(Slide.) Taken June 12, 1937. Lateral view.

(Slide.) Taken June 12, 1937. Just a little ring of tissue at lower border which disappeared. I checked up on him the week before coming to this meeting, and there has been no return of the growth.

DR. CARMODY: Was a basal metabolism taken?

DR. JARVIS: No, I do not take basal metabolism.

PRESIDENT BARNHILL: What was the nature of the tumor?

DR. JARVIS: Very hard and dense. A diagnosis of Riedel's struma was made. There was no biopsy.

PRIMARY CARCINOMA OF THE EPIGLOTTIS WITH  
TOTAL EXTIRPATION OF THE EPIGLOTTIS BY  
TRANSTHYRO-HYOID APPROACH: CASE  
REPORT

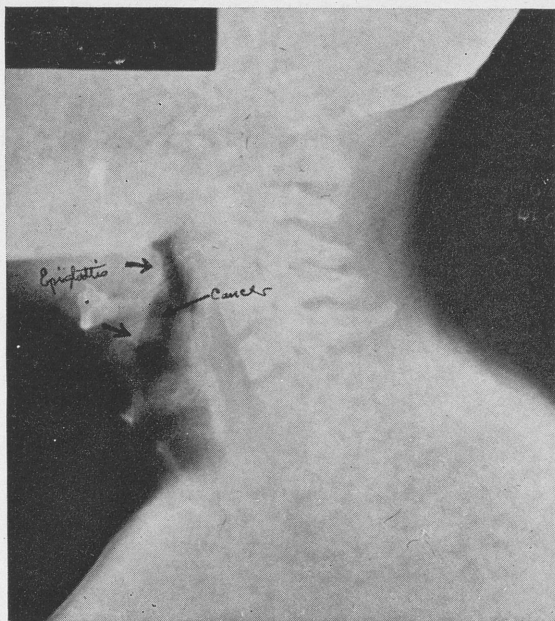
GABRIEL TUCKER, M.D.

It was my privilege to present at the Toronto meeting of this Association a paper on carcinoma of the epiglottis. In that paper a case of primary carcinoma of the epiglottis was reported, in which the under or laryngeal surface, of the epiglottis was involved, and treatment was carried out by total extirpation of the epiglottis by thyrotomy. I had no precedent for the method of procedure in this case and so far as I have been able to determine there is no record of removal of the epiglottis by this route in the literature. There has been no recurrence of the carcinoma after a period of over five years. I desire to report this follow-up observation and to add another case in which the primary lesion was in the same location on the epiglottis, and in which the same approach was used, modifying the operative procedure, however, by extending the incision upward and dividing the hyoid bone as well as the thyroid cartilage.

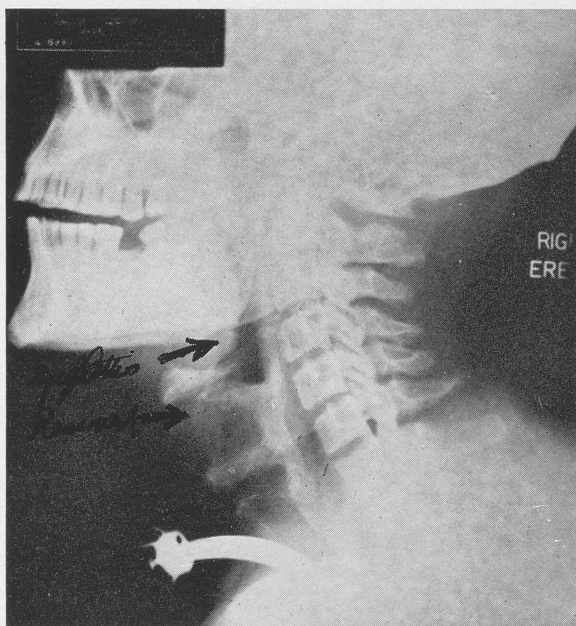
Carcinoma invading the epiglottis may occur as a primary lesion involving the tip of the epiglottis, the upper or lingual surface of the epiglottis, or the under or laryngeal surface. The most frequent involvement of the epiglottis by cancer is secondary. Secondary involvement may result from extension of the tumor from the base of the tongue or the pharynx from above; or the extension may occur from the ventricular band below. The treatment of primary cancer of the epiglottis by surgery is a much less serious problem than that of secondary involvement. Where carcinoma involves the tip of the epiglottis it may be treated by excision of the epiglottis through the mouth by means of direct laryngoscopy or suspension laryngoscopy, either with or without irradiation. The treatment of secondary involvement of the epiglottis, when it extends from the base of the tongue or pharynx

downward or from the interior of the larynx upward, becomes a very extensive surgical procedure if complete excision of the lesion is attempted. Where the lesion extends downward from the base of the tongue, trans-thyroid-pharyngotomy by the Trotter operation has been successfully performed in this country and abroad. Dr. Henry B. Orton, of this Association, has reported a number of cases of this type successfully treated. Secondary involvement of the epiglottis from the lesion within the larynx may be successfully treated by total laryngectomy. In my first case of primary cancer of the under surface of the epiglottis, the lesion was located low down, toward the base of the epiglottis; it did not seem suitable for operative removal through the mouth, and it was apparent that total laryngectomy would not more effectively remove the carcinoma than excision of the epiglottis by the laryngofissure route. In carrying out the operation through this approach I removed a portion of the ventricular bands and the entire base of the epiglottis with a wide margin of normal tissue surrounding the lesion, basing the procedure on the same principle as that of laryngofissure, where laryngofissure is used as the surgical procedure in carcinoma involving the true vocal cords. (Fig. 4A.) As the first case was successful, without recurrence for a five year period and with a perfect functional result—the patient possessing an excellent voice and having no difficulty in swallowing—I have now to report a second case in which the same procedure was successfully performed with a good result to the present time, about two months after operation. The advantage of removal of the epiglottis by the laryngofissure route is obvious. At first, slight difficulty in swallowing is experienced, but the larynx very soon compensates fully. There is no choking nor dysphagia. The remaining portion of the ventricular bands apparently fully compensates for the lack of epiglottis and assumes a protective function of the epiglottis in preventing the food from entering the larynx and trachea. The first step in the operative procedure is a preliminary tracheotomy, and it is advisable to await the healing of the wound for at least a period of ten days or two weeks. The second stage of this procedure is then carried out, extending the incision upward from just above the cricoid cartilage to well above the hyoid bone.



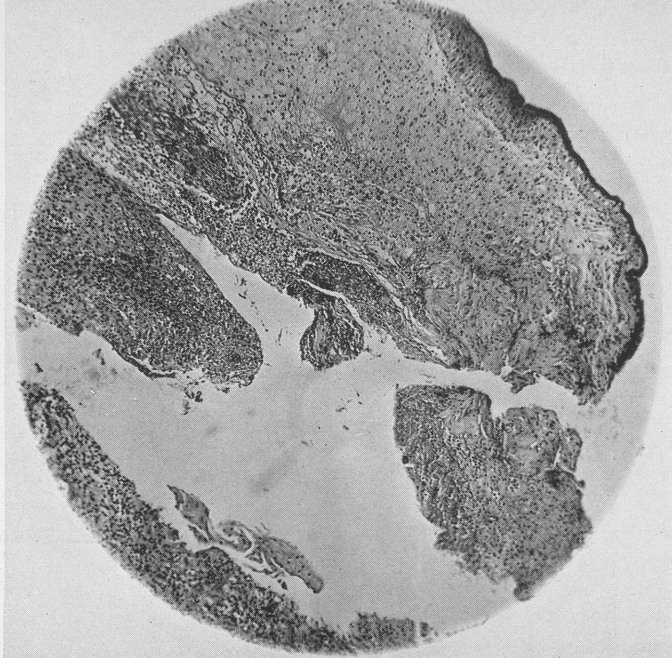


A



B

FIG. 1.—(A) Lateral roentgenogram of the larynx showing the lesion on the base of the epiglottis, and (B) lateral roentgenogram of the larynx and neck after the removal of the epiglottis, showing absence of the epiglottic shadow.



*A*



*B*

FIG. 2. *A*, Photomicrograph of the specimen of redundant tissue removed from the upper surface of the cord. Inflammatory tissue, no evidence of malignancy; Dr. Case. *B*, Photomicrograph of the tissue removed from the under surface of the epiglottis; squamous cell sarcoma, Grade 2; Dr. Case.

CASE REPORT. Cancer of the larynx, C. S. Case No. G316. Male, aged fifty-seven years; occupation, glazier. The patient complained of discomfort in his throat during the past year, slight dysphagia, and for the past six months increasing hoarseness. The family history was negative. The patient was a moderate user of tobacco and alcohol. Previous medical history was negative. General examination showed the patient to be well developed and nourished, overweight, skin very dark. He was very apprehensive that he might have "carcinoma of the throat." Examination of the throat showed general reddening of the mucous membrane of the pharynx. Tonsils were not actively diseased. Teeth showed some evidence of pyorrhea. The mucous membrane of the larynx was generally quite red. Examination of the glottis showed a pedunculated mass attached on either side to the upper surface of the true cords. On phonation the redundant tissue met in the midline. This lesion readily accounted for the patient's hoarseness. The under surface of the epiglottis could not be clearly visualized with the mirror, although a very careful examination was made, with cocaineization of the pharynx, on several occasions. Direct laryngoscopy was done for the purpose of removing the redundant tissue from the upper surface of the cords as well as for more satisfactory inspection of the under surface of the epiglottis. On direct laryngoscopic examination the redundant tissue was removed from the upper surface of the cords, and on inspection of the under surface of the epiglottis an ulcerated area was noted just above the attachment to the ventricular bands, in the midline of the epiglottis. There was distinct ulceration, with an infiltrated zone and a crater-like formation, the size of the lesion being almost that of the little finger nail. Tissue was removed from the infiltrated margin of the lesion for biopsy. The appearance on direct examination was very suggestive of carcinoma. The tissue specimens were submitted for microscopic examination. Redundant tissue from the true cords was reported as inflammatory, not neoplastic; no evidence of malignancy. (Fig. 2A.) The tissue from the lesion on the under surface of the epiglottis was reported as squamous cell carcinoma, grade two. (Fig. 2B.) General examination of the patient, including x-ray of the chest, and swallowing function and blood Wassermann, were negative. Following the direct laryngoscopic removal of redundant tissue from the true cords the patient's voice improved and we had considerable difficulty in convincing him that there was a more serious disease in his larynx which would require further operation. However, the referring physician, Dr. Luber, of Washington, D. C., came to our aid and the patient submitted to the operation for removal of the epiglottis.



*Operation.* The preliminary, or first stage of the procedure, consisted of tracheotomy under local anesthesia. An incision was made vertically in the midline below the cricoid cartilage. Sufficient opening was made to expose the upper trachea. A small section of the second tracheal ring was removed and a No. 6 tracheotomy tube was inserted.



FIG. 3.—Photograph of the patient showing the extent of the scar following the removal of the epiglottis. Patient is still wearing the tracheotomy tube, which is completely corked and the airway through the glottis is normal. Patient was successfully decannulated two months after operation.

Two weeks were allowed for the incision to granulate well around the tracheotomy tube; the tube being changed daily, and the second stage of the procedure was carried out under local 0.5 per cent novocaine anesthesia. A second vertical incision was made from above the hyoid bone to just above the upper border of the cricoid cartilage. The muscles were separated and the thyroid cartilage exposed; likewise the hyoid bone was exposed. The crico-thyroid membrane was incised and the thyroid cartilage was split in the midline with a turbinotome. The interior of the larynx was inspected, and it



was found that the lower limits of the carcinoma were well above the junction of the base of the epiglottis and the ventricular bands. In order to facilitate the removal of the epiglottis, the hyoid bone was divided in the mid line and, on retraction, the exposure of the epiglottis was very much improved. After freeing, the tip of the base of the epiglottis was grasped and retracted upward. The base of the epiglottis was readily freed from its attachment to the anterior

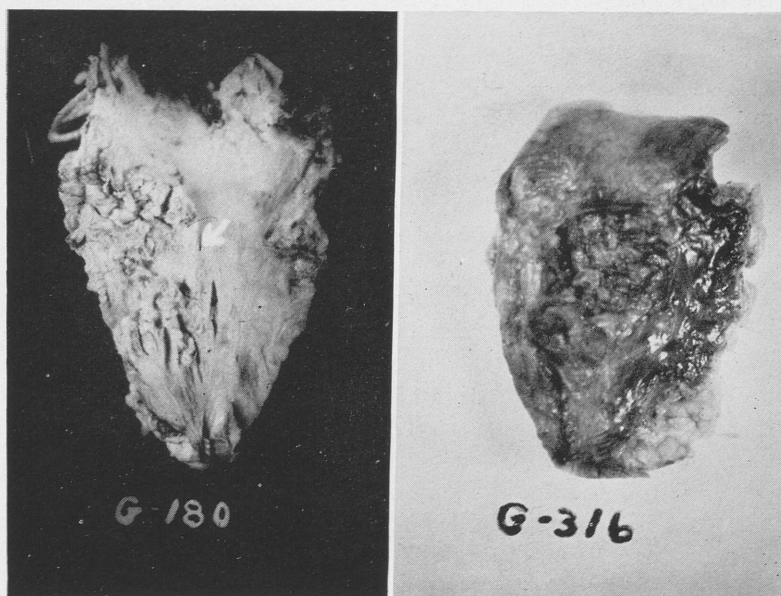


FIG. 4.—Photograph of the epiglottis in (A) Case No. C. S., G. 180, the arrow indicating the location of the squamous cell carcinoma, Grade 1, and (B) Case No. C. S., G. 316, the arrow indicating the location of the squamous cell carcinoma, Grade 2.

pharyngeal wall on the posterior surface of the hyoid. (Fig. 4B.) The mucous membrane at the lateral attachments of the epiglottis was closed by interrupted catgut sutures from within the pharynx. The outer portion of the wound was closed by interrupted sutures and a small rubber tissue drain was placed deeply in the wound. A small opening was left over the thyroid to facilitate drainage. The procedure was carried out under local anesthesia.

There was very little postoperative reaction in the wound. The drainage was removed on the third day and the wound healed promptly without fistula formation. A feeding tube was placed in the upper esophagus, and a second catheter was passed through the nose and nasopharynx into the pharynx and intermittent suction was used to remove the saliva from the pharynx and prevent its overflow into the larynx. The feeding tube was retained for a period of two weeks and by this time the patient was able to swallow without overflow into the larynx, and the feeding tube was removed. The tracheotomy tube, however, was left in position for a period of two months to avoid any possibility of accident. (Fig. 3.) It was reduced in size, and for several weeks a No. 3 tracheotomy tube was worn with a cork in it. There was no difficulty in swallowing. The patient had a clear voice, but considerable edema persisted in the arytenoids and ventricular bands, although the glottis showed an ample airway. There was no dyspnea when the patient was wearing the tube completely corked, which he was able to do both day and night.

COMMENT. 1. A second case of carcinoma of the under or laryngeal surface of the epiglottis is reported in which total extirpation of the epiglottis was done by the laryngofissure route. In the second case, the procedure, as I originally performed it, was modified by extending the incision through the hyoid bone. The first case, previously reported, shows no recurrence after five years and the patient has a normally functioning larynx.

2. Attention is again called to a type of primary cancer of the larynx which grows slowly and is late in extending beyond the epiglottis. A method of conservative surgical treatment by total extirpation of the epiglottis by the laryngofissure route has been successfully employed.

3. The earliest symptom in both reported cases of primary carcinoma of the epiglottis was local discomfort, increasing in severity until localized pain and dysphagia developed. The second patient's chief complaint, upon examination was, however, hoarseness, although local discomfort had been present for some time. The second patient had a multiple lesion. The hoarseness was due to an inflammatory lesion of the true vocal cords. The lesion on the under surface of the epiglottis was not visualized

until, on direct examination for removal of the redundant tissue from the cords, the ulcerated area was found. The occurrence of the two lesions in the same larynx, one producing local discomfort, the other hoarseness, is noteworthy. The carcinoma on the under surface of the epiglottis would have been overlooked had it not been that a direct examination was made. The conformation of the epiglottis was such that a satisfactory examination of its under surface could not be obtained with a mirror.

## TREATMENT OF CHRONIC STENOSIS OF THE LARYNX, WITH SPECIAL REFERENCE TO SKIN GRAFTING

V. E. NEGUS, M.S., F.R.C.S. (By Invitation)

LONDON

IN submitting this paper to so distinguished an assembly of laryngologists, I have more than one object in view. I wish to give a brief account of the various measures adopted by some of my colleagues and myself in dealing with these difficult cases, with some indication as to which methods appear to be the best. I also hope that the subject will be clarified by subsequent discussion.

The variety of methods of treatment is an indication of the difficulties met with by all. The type of case now under discussion must, of course, be limited to chronic conditions in which any active disease, such as syphilis, has been arrested. It seems best to subdivide the subject according to methods of treatment and not to consider together various types of stenosis, as certain cases, similar in causation, require different remedies.

There are so many references to the subject in the literature that it has been found impracticable to refer in detail to them here; I must apologize for this omission.

*Repeated Bouginage.* I have tried this form of treatment in certain instances with various types of dilators, but without much success; any improvement has been of temporary duration only. The use of electrically heated bougies is said by Dean and Johnston

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### LEGEND FOR FIGS. 1 AND 2.

FIGS. 1 and 2.—Permanent tracheostomy. The patient at the age of five was operated on for removal of tonsils. One week later she suffered from dyspnea with purulent bronchitis. Tracheotomy was performed, but subsequent attempts at removal of the cannula failed. Dilatation, followed by intubation, was tried. She was later referred to me with much swelling of the arytenoepiglottic fold on the right side. The tracheotomy tube was replaced at a lower level. There is now fixation of both arytenoid cartilages and attempted removal of the cannula has been followed by distress, which has necessitated replacement. The girl is now twelve years of age; she is well built, active physically and mentally. A larger tube than that seen in the illustration has now been fitted, enabling her to breathe freely and to speak easily with the help of an inspiratory valve. She can carry on all activities except bathing, which would be dangerous if the neck were submerged.





FIG. 1

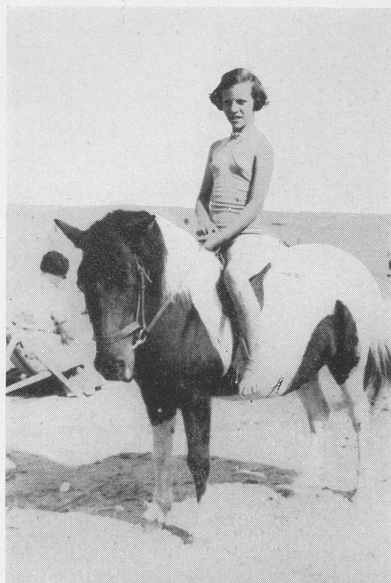


FIG. 2

to offer advantages, but I have had no personal experience in the method.

*Division of Webs With Cautery or Knife.* If adhesions form over a wide area it is useless merely to divide them, unless means are taken to keep the raw surfaces apart until healing is complete.

Rare cases are seen where a thin web obstructs only the anterior part of the glottis and this can be divided easily. Great care, however, must be taken to avoid the production of fresh adhesions, particularly if diathermy is used. One or two difficult cases have been referred to me where the condition was attributable to the previous use of diathermy in the area of the glottis.

*Temporary or Permanent Tracheostomy.* In many cases of severe stenosis, opening the trachea is first required and should not be postponed too long. The numerous conditions calling for the performance of this operation vary considerably in prognosis. The most suitable are chronic inflammatory cases of the slow, lupoid type, but it may be required for relief of active laryngeal tuberculosis. It may subsequently be possible to abandon the tube if satisfactory healing takes place. Healing with or without local cautery treatment, may, however, be followed by contraction of the scar tissue and glottic stenosis.

Tracheostomy may be called for in cases of stenosis due to double abductor paralysis and here, again, isolated cases of recovery may occur; I can recall one due to temporary peripheral neuritis, apparently of syphilitic nature, although previous specific treatment had been given without immediate effect.

Only too many cases are still met with where a high tracheostomy has been performed; the first necessity in such a case is to replace the tube at a lower level, well below the first tracheal ring and the cricoid cartilage. Unfortunately, in too many of these cases irreparable damage has been done by cicatricial stenosis, by inflammatory fixation of the arytenoid cartilages, or by injudicious local interference.

A subject of much debate must now be considered, and that is permanent tracheostomy. Owing to my good fortune in being the pupil and associate of Sir St. Clair Thomson, I have seen, in many and various conditions, the superior advantages of permanent tracheostomy compared with other treatments, and have been

much impressed by the wisdom of this means of lasting relief. Theoretically there are many objections to the formation of a permanent stoma in the neck, but in practice it is very surprising

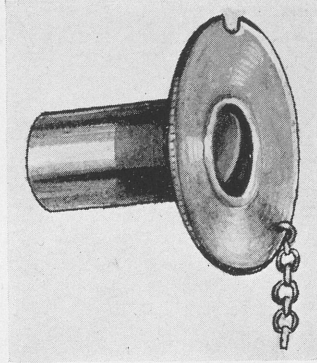
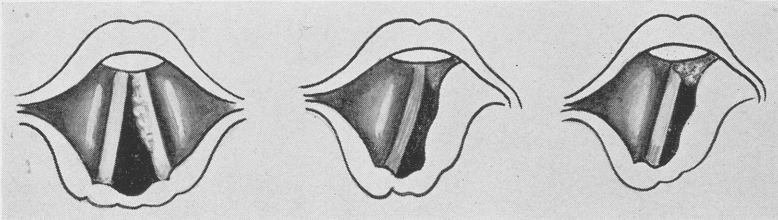


FIG. 3.—Valve to fit tracheotomy cannula. The hinged flap opens on inspiration and allows air to enter; it closes on expiration and allows the current of air to pass through the glottis for purposes of speech.



23-7-37

18-7-34

15-5-34

FIG. 4.—Carcinoma of the larynx followed by stenosis after laryngofissure. 15.5.34. Male patient aged fifty-three with a carcinoma limited to the anterior region of the left vocal cord. This was removed by the laryngofissure route. Microscopic examination showed the growth to belong to Broder's Group I. There was a satisfactory area of healthy tissue surrounding the growth. 18.7.34. Appearance after operation. The arytenoepiglottic fold on the operated side has been drawn inwards, while the arytenoid has moved somewhat forward. 23.7.37. Three years later the glottis had become considerably narrowed owing to cicatricial contraction in the operative scar; there was no recurrence of growth. A tracheostomy cannula was inserted and was fitted with a valve for purposes of speech. (Case referred to me by Sir St. Clair Thomson.)

to note what little disability is experienced, particularly when counterbalanced by the abundant airway and the preservation of voice. Children grow up healthy and well-developed (Fig. 1) and

adults lead a normal existence, able to enjoy active sports and life, while women bear healthy children. As with "tubed" horses the one and only great risk is getting the neck submerged (Fig. 2).

One proviso must be made in claiming success for permanent tracheostomy, namely, that some glottic space must be present for purposes of voice. The patient is provided with a hinged valve

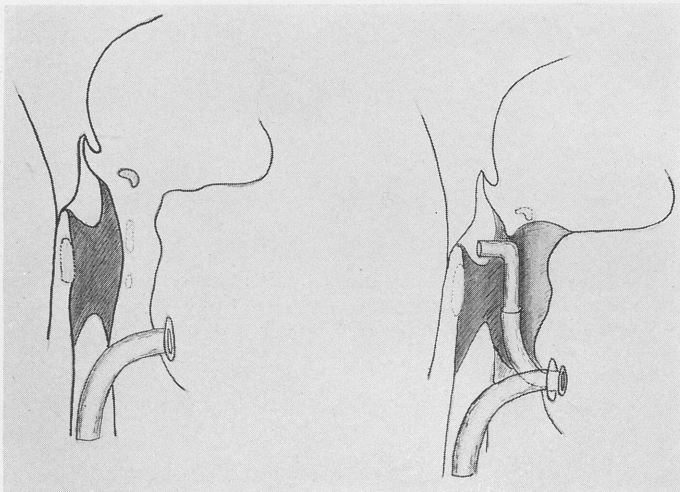


FIG. 5.—Complete stenosis of larynx. A woman aged thirty-one had complete stenosis of the larynx, caused by a self-inflicted wound. Perichondritis had led to sloughing of the thyroid and cricoid cartilage. A fistula was established between the remains of the laryngeal aperture and the exterior by means of skin-flaps. The central part of the hyoid bone was removed. A groove was made in the front of the neck, leading from the fistula down to the tracheotomy opening. A hollow extension was made to connect the tracheal cannula to the fistula, with restoration of satisfactory speech in a loud whisper. *A*, The dark area represents the original lumen of the larynx replaced by scar tissue. The position of the cartilage is indicated by dotted lines. *B*, The groove and fistula are shown, with the tube extension in place.

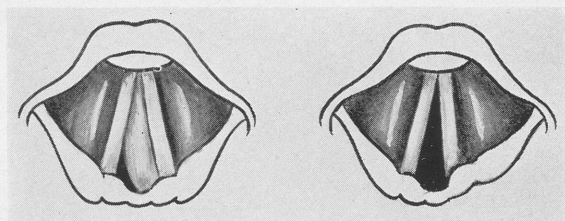
at the mouth of the tube, so designed that air can enter the lungs freely during inspiration, while part or all of it can escape through the glottis during expiration (Fig. 3). Patients dealt with in this manner include some who cannot separate their vocal cords because of fixation of the crico-arytenoid joints; others with permanent double abductor paralysis; others again with healed lupoid



or tuberculous laryngitis; and a certain number with cicatricial stenosis following surgical traumatism, sometimes after operations for cancer of the larynx by the laryngofissure route (Fig. 4), or by lateral pharyngotomy. Finally, a few have stenosis from traumatic or suicidal wounds of the larynx.

When dealing with healed malignant or tuberculous cases it would undoubtedly be unwise to attempt any active dilatation, for fear of promoting recurrence in a sensitive region.

*Operations to Shift the Position of the Vocal Cords.* Many attempts have been made, particularly in cases of double abductor paralysis, to restore the width of the glottis sufficiently to permit



5-1-32

14-12-32

FIG. 6.—Stenosis of larynx caused by a chondroma. The patient was an elderly lady with loss of voice, severe dyspnea and stridor. A large chondroma was blocking the larynx and also obstructing the esophagus. It was removed completely and, to repair the larynx, a flap of skin was turned in, leaving an open groove. Later the groove was converted into a tube and her voice and breathing powers were restored; the tracheostomy cannula, inserted as an emergency measure before the removal of the tumor, was no longer needed.

easy respiration. I have the impression that such efforts may only result in an inadequate airway and leave a more defective voice, inferior in both respects to the permanent tracheostomy fitted with an inspiratory valve.

Among such operations to be mentioned are those of Réthi and Wittmaack for displacement of the cord outwards or downwards; that of Chevalier Jackson for removal of the cord by ventriculo-cordectomy; and also Hoover's method of submucous resection of the cord.

*Nerve Anomosis.* In cases of double recurrent paralysis anatomically successful operations have been performed by Colledge, Ballance, and Frazier for anastomosing the phrenic, or some other

nerve, to the distal end of the injured recurrent laryngeal. The results do not appear to be satisfactory, possibly because of disordered coördination of the glottic movements.

*Continuous Dilatation.* Many methods of this type have been described; some surgeons have made prolonged use of metallic obturators or doubled-up rubber tubes, others have employed molds shaped to fit the larynx, while others again have inserted a hollow tube of vulcanite or rubber through which the patient might breathe. O'Dwyer's intubation tube may be employed.

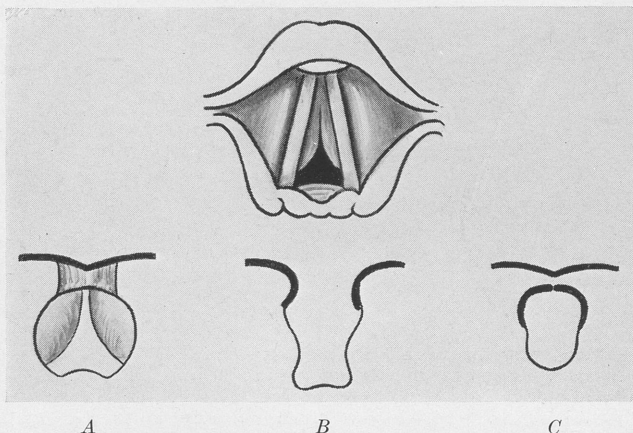


FIG. 7.—Stenosis of subglottic region. Female, aged sixty-two with history of huskiness for twenty years, getting worse, with recent stridor. A soft swelling of the posterior wall, causing obstruction below the glottis, was treated by a colleague with insertion of radium seeds, after the trachea had been opened. The swelling disappeared to a considerable extent, but stridor remained. The lumen of the trachea, just below the cricoid cartilage, was small and triangular in shape (A). The trachea was split in the midline and skin-flaps were turned in and united to the tracheal mucosa (B). The gutter so formed was later converted into a tube, with satisfactory restoration of the airway (C). The tracheotomy cannula was no longer required, and the voice was restored to normal.

Schmiegelow has described his method of holding a rubber tube in place by a transfixing peg of silver wire passed through the two alæ of the thyroid cartilage. In my practice such measures have given place to others to be described later.

*Laryngostomy With Prolonged Packing.* This method, employed in some clinics, is tedious and painful. It aims at relining the restored laryngeal airway with scar tissue and epithelium, which

spreads inwards from the muco-cutaneous margins. More rapid and effective methods appear to be available.

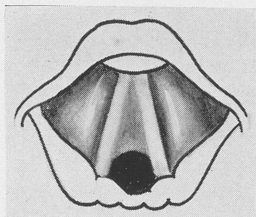
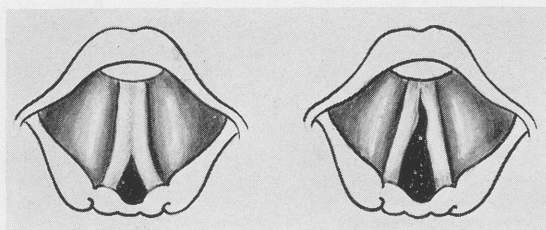


FIG. 8.—Stenosis of larynx treated by the skin-grafting method. The patient a girl, aged nineteen, was in a motor accident and had her lower jaw fractured. Subsequently she suffered from dyspnea and hoarseness and a web was seen between the vocal cords; the laryngeal cartilages were fractured and the whole organ was shortened. Division with the cautery was attempted elsewhere. After transference to my care, laryngofissure was performed and the cords were separated and kept apart by an oiled silk bag covered with a skin graft. The result was good and the voice was improved, stridor was eliminated and there was no dyspnea on gentle exertion.



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FIG. 9.—Stenosis of the larynx treated by the skin-grafting method. In 1933 the patient (a man, aged forty-nine) suffered from laryngeal stridor. A swelling was seen below the glottis and this was treated with diathermic coagulation. Tracheotomy was called for to relieve the respiratory distress. When referred to me there was marked obstruction caused by adherence of the vocal cords. In 1934, the larynx was opened by the laryngofissure route; the vocal cords were adherent along two-thirds of their length, there being only a small posterior gap. The opposed surfaces were separated and a skin graft was inserted with the aid of Mr. T. Pomfret Kilner. It was kept in place by an oil-silk bag filled with ribbon gauze. The retaining bag was removed after seven days, and the tracheotomy tube eleven days later. The voice was much improved and there was no stridor or dyspnea on gentle exertion. The cords remained separate and free along their whole length after one subsequent dilatation.

*Plastic Operations With the Use of Skin Flaps.* I have employed this method in a few cases with some success, either in restoring the power of speech or, in addition, the ability to breathe without

a tracheostomy. In one patient there had been extensive perichondritis of the laryngeal cartilages following a self-inflicted wound; the larynx as a tube no longer existed but was replaced by a mass of scar tissue (Fig. 54). The upper part of the epiglottis and the upper margins of the arytenoepiglottic folds remained, but there was no communication between the pharynx and the trachea and, consequently, no voice.

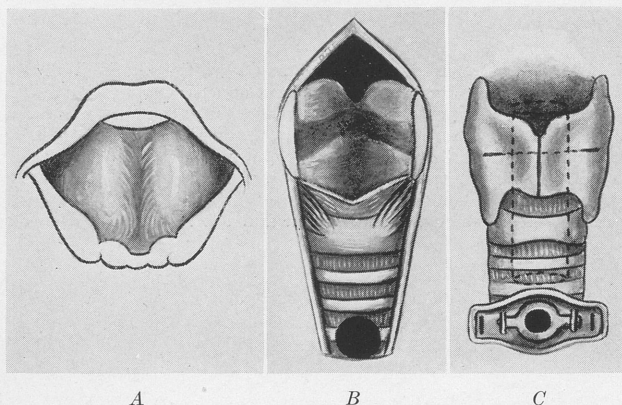


FIG. 10.—Complete stenosis of larynx. A boy, aged sixteen, fell off his bicycle and bruised the larynx. Stridor was noticed immediately, followed by cough and hemoptysis. Six days later respiratory distress necessitated tracheotomy. When first seen by me, two months later, there was complete stenosis. An interval was allowed to elapse before operation, to allow all inflammatory changes to subside. The larynx was then split through a median incision and the adherent cords were divided with a scalpel. Movement of the arytenoid cartilages was at once noticed. A skin graft was inserted wrapped round a rubber tube, held in place by Schmiegelow's method of transfixion with silver wire passed through both thyroid alæ (C). There was considerable reactionary swelling. The larynx was reopened and the tube removed twelve days later; the skin graft was in good position and firmly adherent. Subsequent progress was uneventful, with the return of a useful voice and a free airway. The tracheotomy cannula was corked fifteen days after the operation and removed three or four days later. The figures show the larynx viewed from above, the larynx opened, with adherent areas, and the retaining tube in position.

It was possible, after removal of the central part of the hyoid bone, to construct a fistula between the remains of the laryngeal aperture and the front of the neck by means of skin flaps turned in to unite with the mucosa. A gutter was then constructed, leading from the fistula to the tracheal opening. A hollow upward



extension of the tracheostomy tube was used to connect the fistula with the trachea and thus restore the power of speech, although a tracheostomy cannula was still required for inspiration (Fig. 5B).

Among other cases I have dealt with in the use of skin flaps is one of chondroma of the cricoid cartilage (Fig. 6) and another of cicatricial stenosis following irradiation of a subglottic neoplasm of uncertain nature (Fig. 7). In both of these a gutter was first formed, to be converted into a tube by dividing and turning in part of the skin flaps, with restoration of normal speech and respiration. It was possible to dispense entirely with any tube.

*Continuous Dilatation With Skin-grafting.* If the stenosis is in a region which may contract, the restored airway must be maintained for some weeks by the use of rubber sponges, Stent's material, rubber molds, rubber tubing, or a silver extension attached to a tracheostomy tube. Having canalized the previously obstructed endolarynx or subglottic region, the obturator is fitted. Instead of leaving the tract in which it lies to await the slow ingrowth of epithelium, the obturator is covered with a thin graft of skin, taken from the arm or leg. If rubber tubing is used it may be possible to dispense with a tracheostomy tube, but usually a cannula is retained until healing is complete.

This method has also been recommended by Arbuckle, Figi, and others, but its value needs emphasis, as it does not seem to have received the attention it deserves.

One girl, aged eighteen, operated on by myself, had not spoken for fifteen years because of complete subglottic stenosis, acquired at the age of three, consequent upon treatment for multiple laryngeal papillomata. The restoration of the subglottic lumen with skin-grafting, maintained in position by an extension upwards of the tracheal cannula, allowed her to speak without any real difficulty by the use of a flap valve. As no air had passed through the larynx all these years the organ was of infantile size and insufficient for respiration, so that she could not dispense with the cannula.

Chevalier Jackson has referred to growth of the larynx as an important factor in restoring the airway; but in 2 or 3 cases in my experience, prolonged obstruction appeared to have arrested growth.

*Skin-grafting With Temporary Fixation.* Three or 4 cases have come to me with partial or complete stenosis following falls on the larynx; in each of them the vocal cords adhered together over a wide extent. It was possible, by employing the laryngofissure route, to separate the cords accurately and to insert a thin skin graft. I have used for retention either a bag of oiled silk packed with ribbon gauze, an obturator of Stent's material, or a simple rubber tube and have found the latter to be the most efficient. The obturator or tube is left in place for a week or ten days.

One patient had considerable shortening of the glottis due to fracture of the thyroid and cricoid cartilages, but was enabled to speak well; she has no permanent tracheostomy, as the airway is adequate and she can exert herself, after this treatment (Fig. 8).

Another patient had only a small triangular gap at the posterior end of the glottis; in him the vocal cords were permanently freed and restored to their original length (Fig. 9). A boy, recently dealt with, was voiceless owing to complete stenosis at the level of the cords (Fig. 10). The results of skin grafting, after laryngofissure, appear to be perfect in his case. The retaining tube was removed after twelve days and the tracheostomy cannula two weeks later.

In cases limited to the endolarynx there is no marked tendency to contraction after operation and all goes well if the raw surfaces are prevented from reuniting. The application of a skin graft is so efficient a means of attaining this end that the general adoption of this method would appear to be desirable in suitable cases. The short period during which an obturator must be worn and the absence of prolonged after-treatment are advantages which should not be ignored.

This remark does not apply with equal force to stenosis of the trachea, or to laryngeal cases in which there has been much destruction of the cartilages.

The tendency to collapse of the re-formed tube necessitates the continuation of the dilatation by the wearing of an obturator for some months.

NOTE.—I wish to express my indebtedness to Sir St. Clair Thomson for his advice on the choice of methods and also for his kindness in reading and correcting this paper.

## AN EXPLANATION OF THE RESPIRATORY FAILURE SOMETIMES OCCURRING AFTER A SUCCESSFUL TRACHEOTOMY

V. E. NEGUS, M.S., F.R.C.S. (By invitation)

LONDON

SOME years ago Sir St. Clair Thomson directed my attention to the peculiar fact, observed by him, that some patients, after complete relief of laryngeal obstruction by tracheotomy, might cease breathing for no obvious reason, and even die. A tour he had made of all the Museums of the Medical Schools of London revealed a considerable number of specimens of laryngeal stenosis, from various causes, in which it was patent that this had been relieved by a correct and perfectly successful tracheotomy, and yet the clinical report recorded that the patient, with a completely restored airway, had succumbed to heart failure, with symptoms of embarrassed breathing.

His reference was, of course, to obstructions of some standing and not to acute conditions, such as diphtheria, where many factors interact to produce serious symptoms.

It appeared necessary to consult physiologists, and particularly those with especial knowledge of respiration and biochemistry. The answer to the problem was supplied after personal inquiries to Professor R. J. S. McDowall. Investigations on patients were made by Dr. R. D. Lawrence, who gave me the information sought for. The writings of Professor J. S. Haldane and Dr. Yandell Henderson were consulted, as therein is contained all the necessary information on gaseous exchange in the lungs. Dr. McCance has kindly criticized and corrected the conclusions reached and has also investigated certain of the patients.

The respiratory center depends normally upon the concentration of hydrogen ions in the blood reaching it and this in turn depends upon the functional activity of the lungs; for carbon dioxide regulates the pH of the blood and is removed *via* the lungs. The larynx normally takes an important part in regulating the respiratory exchange.

The blood is weakly alkaline in reaction, its pH being 7.4; any increase or decrease of the free hydrogen ions would alter this reaction, but in point of fact it is of remarkable constancy. (Fig. 1.) The reaction depends upon the ratio of  $\text{CO}_2$  present as carbonic acid, to that combined with sodium as bicarbonate.

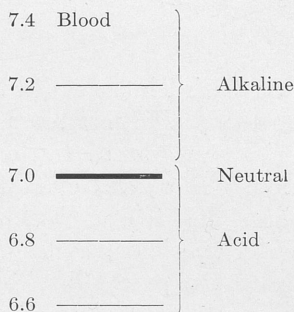


FIG. 1.—Reaction of the blood.

The percentage of  $\text{CO}_2$  in the arterial blood is constant in health and is normally 5.6; that in the venous blood is about 8 per cent. In diseased stages the percentages may be higher, as will be mentioned later. The  $\text{CO}_2$  in the arterial blood is in equilibrium with that in the alveoli of the lungs and it follows, therefore, that as the percentage in the blood must be kept constant, so also must that in the alveolar air remain at 5.6 per cent; the tissues generally, and the respiratory center in particular, are most intolerant of any change in the reaction of the fluid bathing them. In normal health any excess of acid is removed by increased lung ventilation; the individual breathes more deeply, rather than more rapidly, and washes out an increased quantity of  $\text{CO}_2$  from the alveoli, thus keeping the arterial content at 5.6 per cent, with maintenance of a normal acid base balance.

The larynx has a considerable regulating influence on these exchanges: the glottis widens on inspiration and becomes narrower on expiration, thus exercising some control over both inspiratory and expiratory currents of air. (Fig. 2.) These glottic movements affect the Hering Breuer reflex, which determines the rate of respiration; the slowing effect gives time for  $\text{CO}_2$  to be removed from



the blood and oxygen to be taken up. There is, at the same time, considerable influence on the rate of blood flow through the lungs,

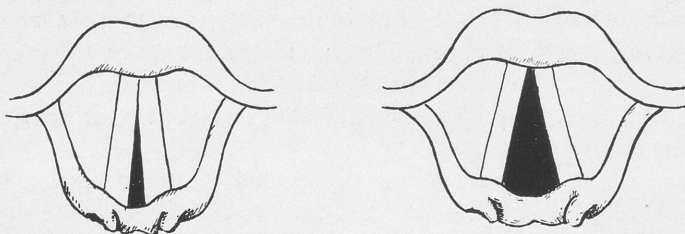


FIG. 2.—Exaggerated respiratory movements at the glottis.

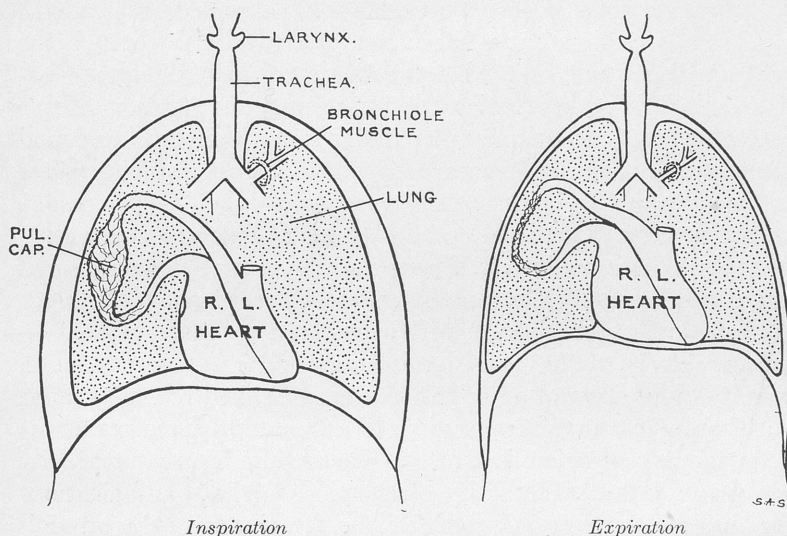


FIG. 3.—Respiratory changes in the pulmonary vessels.

At full *inspiration*, pressure in the thorax is at its lowest level. The glottis is well open, the bronchial muscle is relaxed and air enters the alveoli freely. Relief of pressure from the walls of the pulmonary capillaries causes them to dilate; blood is thus allowed to rush freely from the well-filled right side of the heart, through the lungs to the left ventricle, whence it is pumped out at high pressure.

During *expiration* the pressure rises and the pulmonary capillaries are compressed. Because of increased pressure on the great veins and right side of the heart, the flow of venous blood is impeded. Circulation through the lungs is slowed down and the left side of the heart receives less blood than during inspiration. Aortic pressure therefore falls. The bronchial musculature is in contraction and the glottis is less widely open than during inspiration. Escape of air from the lungs is thus obstructed.

dependent upon pump action, with dilatation of the pulmonary capillaries and suction action on the right auricle and great veins during inspiration. (Fig. 3.) The controlling mechanism is quite efficient in normal health and is extremely sensitive; a rise of 0.2 per cent of  $\text{CO}_2$  in the alveolar air produces an increase of 100 per cent in the lung ventilation. Alterations in the oxygen content of the blood, on the other hand, have but slight effect on the respiratory center.

Now, if the larynx becomes obstructed for some reason, this normal mechanism is brought into greater activity and the depth of respiration is increased; this entails considerable effort, as is required in laryngeal stenosis, and may be beyond the powers of the patients to sustain. Furthermore, the forced inspirations, against obstruction, cause the blood stream to rush through the lungs without sufficient time for the necessary respiratory exchanges. This occurs particularly in patients suffering from double abductor paralysis. Some other means must, therefore, be found to maintain the normal reaction of the blood. The changing factor is the alveolar  $\text{CO}_2$  content, and consequently the  $\text{CO}_2$  percentage in the arterial blood. In certain cases of laryngeal obstruction investigated for me by Dr. Lawrence, this rose to percentages of 6.2, 6.4, 6.6, 6.8, 7.0, 7.2, and 8.0; these figures show an enormous increase and yet they were well neutralized by some new mechanism. The latter is the withdrawal of sodium from combination with the proteins and other buffer substances and re-combination with carbonic acid to form sodium bicarbonate; this maintains the constancy of the acid alkali ratio. The sodium bicarbonate in the plasma may be estimated by biochemical methods and the findings can give a close indication of the alveolar  $\text{CO}_2$ ; the latter is difficult to estimate accurately in patients suffering from laryngeal obstruction. The increased output of alkali combines with the rising content of carbon dioxide and prevents any excessive increase in the percentage present as carbonic acid. The reaction of the arterial blood is thus maintained at a pH of 7.4, without any immediate exhausting call on the muscles of respiration. The respiratory center is now set at a higher level, and responds to blood containing more than the normal percentage of  $\text{CO}_2$ . Some of the increased acid is eliminated by the kidneys, chiefly as phos-

phates. (Fig. 4.) During these changes there may have been no anoxemia for the reason that there is, in normal conditions, a reservoir or excess of oxygen in the lungs; the patient is, therefore, not cyanosed if the obstruction has been of gradual onset, unless it reaches an extreme degree.

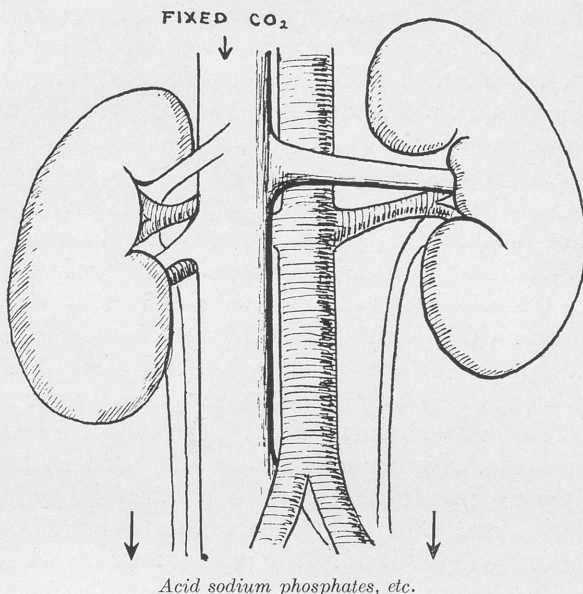


FIG. 4.—Removal of acid from the body. Diagram to illustrate elimination of  $\text{CO}_2$ , combined as acid sodium phosphate, etc.

But there comes a time when increasing obstruction prevents sufficient oxygen from reaching the alveoli, while the rapidly circulating blood has not had sufficient time to take up what oxygen is present. The distress of the patient then calls urgently for tracheotomy. Forewarning of the necessity for this operation could be provided by estimation of the alkali reserve, as follows from the observations made above.

The tracheotomy may proceed in a tranquil manner. The cannula inserted into the trachea at once gives free passage for entrance and exit of air. There has probably been no strain or coughing if

a small quantity of cocaine has been injected into the lumen of the trachea before opening it.

Breathing immediately becomes deeper and also somewhat faster, owing to removal of obstruction, and acceleration of the Hering Breuer reflex. Lung ventilation is suddenly and enormously increased and much  $\text{CO}_2$  is washed out of the alveoli and blood. In one case of mine, examined by Dr. Lawrence, the percentage of alveolar  $\text{CO}_2$ —and therefore of arterial  $\text{CO}_2$ —was brought back from 7.2 per cent to the normal 5.6 per cent after a few breaths and within as short a time as five minutes. But still, in spite of the abundant and clear airway, respiration may cease immediately, or after an interval of as much as one hour, because of lack of stimulation of the respiratory center. The condition is quite similar to that following voluntary overbreathing or what we can observe after forced artificial respiration in an experimental animal. For the time being the acid base ratio is upset; the percentage of free  $\text{CO}_2$  present as carbonic acid is suddenly lowered, while that fixed as bicarbonate remains stationary. The arterial blood has become so alkaline that the respiratory center no longer receives the necessary stimulation. Respiration then ceases because of acapnia. Oxygen lack is soon felt, but may have little effect on the respiratory center, which is bathed by such alkaline blood that it ceases to respond to even the most extreme stimulus of want of oxygen (Haldane). The patient may die without ever taking another breath. This time of danger, during which excess of alkalies is fixing too much of the available  $\text{CO}_2$ , may extend over some hours.

The practical deductions from all this are obvious and are probably utilized by many surgeons, although the rationale may not be thoroughly understood.

At the time of operation on any cases of laryngeal stenosis a cylinder of  $\text{CO}_2$  should always be at hand; and if breathing becomes shallow or of Cheyne-Stokes type, the patient should be made to inhale the gas. This respiratory stimulant should remain at the patient's bedside for some hours, by which time the balance will have been readjusted. Administration of ammonium chloride before operation could be adopted, but is less simple and less adjustable than the method described.



When the problem is considered it really becomes a simple one, with an obvious remedy, but neglect of the necessary precautions may lead to grave disaster.

One other practical point has already been referred to, *i. e.*, the advisability of estimating the alkali content in cases if there is doubt as to the degree of obstruction; but clinical care should be sufficient. Every patient with sufficient laryngeal or tracheal stenosis to cause any audible stridor on exertion should be advised that he is in a danger zone. If the stridor is noticeable when he is resting, the question of tracheotomy should be considered. If it is audible when he is asleep, the operation is urgently required. As we perform it nowadays a tracheotomy should be a straightforward performance without risk. A recognition of the possibility of this condition of acapnia and of the means of overcoming it should remove the possibility of anxiety in the after-care of such cases.

NOTE.—I am much indebted to Professor McDowall, Dr. Lawrence and Dr. McCance for their advice and practical help. I am also particularly grateful to Sir St. Clair Thomson, not only for initiating this inquiry, but also for reading, correcting and making additions to this paper. His opinion on the indications for tracheostomy has been of great value.

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#### DISCUSSION

DR. MERVIN C. MYERSON, New York City:

I wonder if Mr. Negus could tell us whether the stasis of the peripheral pulmonary circulation has something to do with this picture. Last year Dr. Kernan presented the results of a study on the value of helium in dyspneic individuals whose dyspnea was due to obstructive lesions of the larynx and trachea. As a result of his presentation it occurred to me it would be of value to have a carbon dioxide-oxygen apparatus and a closed type of inhalation mask in our special wards.

During the past year we have saved 3 patients by overdilating their lungs in the presence of their obstructive lesion before tracheotomy could be arranged for, or could be done, or before even an intubation tube could be inserted. In other words, these patients were brought back immediately and then a tracheotomy was done more or less deliberately.

MR. GEOFFREY BATEMAN, London, England:

I wish to discuss a point arising out of Mr. Negus' first paper. I have the privilege of working with Mr. Howarth in St. Thomas' Hospital. We had there a case of stenosis of the larynx which was rather a different problem from any mentioned by Mr. Negus. This was a boy who at the age of four years had had a high tracheotomy performed because of laryngeal diphtheria. At the age of seven years, he appeared at St. Thomas' Hospital where the first step in the treatment, a low tracheotomy, was performed. Later on a laryngoscopy was performed, and it was seen that he had a stricture at the level of the vocal cords which was completely obstructing the airway. Not the smallest bougie could be passed through. The associate plastic surgeon, Mr. Kilner, was asked to coöperate in this case. A laryngostomy was performed, the scar tissue was excised and under a dental stent mold a skin graft was put into the larynx through this laryngostomy, which was left open. After a week this stent mold was removed and replaced for another week. Then the stent mold was removed permanently.

During the course of the next fortnight, another stricture developed which almost completely obstructed the airway. At this stage I was asked if I would coöperate. The problem appeared to be, to keep the larynx dilated around the skin graft for a sufficiently long time to prevent any further fibrous tissue being formed beneath the skin graft. The laryngostomy was 25 mm. long and the stricture was at the upper end of this laryngostomy and extending upward about 12 mm. beyond the upper end of the opening. The aryepiglottic fold was about 17 mm. above the top of the laryngostomy. We had a tracheotomy tube made which had an upper extension, and then sliding upward from this extension was a second tube which was fixed by a screw on the front of this extension. The larynx was opened higher up. The mass of scar tissue was excised, and the tube fully extended with a skin graft wrapped around it was put into this laryngostomy. Then the larynx was partly closed again over this tube. At the end of a week, by letting the upper part of the tube slide down, the tube was removed and the site of operation cleaned up and then the tube was put back again and left, this time, for another fortnight, by which time everything was soundly healed and we were able then to put in a rubber tube with an upward extension. This was left in for approximately two months, after which time we assumed that there would be no further contraction and the tube was removed and the laryngostomy left open for another period of three months while we observed the case to make certain that there would

be no further contraction. There was no further contraction, and at this stage Mr. Kilner closed the laryngostomy completely by turning in a skin flap and bringing the skin together in an anterior vertical suture line. The patient now has a larynx without any vocal cords, a perfectly clear airway and a very strong whispered voice in which he apparently makes use of the aryepiglottic fold to get some noise attached to it.

It is now six months since the laryngostomy was closed, and the boy is breathing perfectly normally and is perfectly well.

DR. FREDERICK A. FIGI, Rochester, Minn.:

Mr. Negus' presentations have been extremely enlightening. I was particularly interested in his discussion of stenosis of the larynx, and his use of skin grafts in this connection.

I had the pleasure of hearing Prof. Schmiegelow deliver the Semon lecture which Mr. Negus referred to, and feel, as Mr. Negus does, that the combination of skin grafting together with India rubber tubes possesses definite advantages over the use of the rubber tubes alone as advised by Prof. Schmiegelow.

(Slide.) In this connection I would like to show some drawings made in a case of this type recently. This was the most severe case of laryngeal stenosis that I have attempted to take care of surgically. The patient, a young lady twenty-one years old, came to us originally as a child three years of age with multiple papillomas of the larynx. At that time we were using radium in the treatment of these cases. Her papillomas cleared up promptly, but after a few years there developed a perichondritis which resulted in complete closure of the laryngeal lumen except for a very minute tract. It was not until the patient was approximately twenty years of age that she returned for treatment of her laryngeal stenosis. An open operation was advised at that time.

Upon opening the larynx through laryngofissure a narrow tract that barely admitted a small probe was found. In addition to the thick, dense scarring inside the larynx there was marked thickening of the cricoid and thyroid cartilages. In fact, the stenosis was due more to the latter condition than to the scarring. Obviously bouginage could not possibly have accomplished a great deal. The scarring was dissected out, but as this produced only a small laryngeal lumen, it was necessary to core out the cartilage also, that is, the thickened inner portion of the cartilage was excised, leaving only a thin outer shell. This shell was so thin that it appeared questionable whether there would be ample support to maintain a satisfactory lumen even though the lining were restored. A thin shaved skin graft was

wrapped about a sea sponge and placed in the larynx. The sponge was compressed upon insertion in order to keep the skin graft applied to the denuded interior of the larynx under adequate pressure.

The sea sponge was removed at the end of ten days and the skin graft which had been applied directly to the cartilage was found to have taken perfectly. A soft rubber tube covered with a finger cot was then placed in the larynx, the cuff of the cot being drawn out through the tracheotomy opening and anchored firmly. This tube was worn by the patient continuously for seven months. Since its removal a few months ago the tracheal lumen has shown no tendency to contract and the external tracheal opening has been closed. In this connection a case treated by Prof. Nager is of interest. The patient, a boy about seven years of age, was treated for a severe cicatricial laryngeal stenosis. Following restoration of the laryngeal lumen no sphincter action remained in the larynx, so that when the patient drank fluids they ran directly down into the respiratory tract. I feared this possibility in the case just described, but there has been no difficulty whatsoever in this regard. There is, in fact, considerable movement of the arytenoids and the patient has developed a weak but very useful voice.

PRESIDENT BARNHILL:

Your President begs pardon of the Association for taking a moment of its time. He does so, hoping to call up discussion along a line similar to that which Mr. Negus has brought forward. He once had a patient who had alarming respiratory difficulties very like those described for which no satisfactory causal explanation could be determined. Sometime later he heard a lecture on the "Carotid Sinus" and its function in regulating respiration. That was of great interest and since then he has read several articles in which writers have referred to the carotid sinus in relation to respiratory troubles. Whether or not this "sinus" has anything to do with respiratory affections, or whether the influence of accumulated carbon dioxide acted through the sinus, he does not know. Therefore, should anyone in discussing the subject be informed concerning the carotid sinus, it would be of no small interest to hear from him, since it is probable that in the next few years we shall hear much about this sinus, its functions and its diseases. Would Mr. Negus kindly give his views?

MR. VICTOR E. NEGUS (in closing):

I am very glad, sir, that these papers have led to discussion, and I certainly will go away with information which will be of great help to me.



By the lack of comment of other Fellows of the Association, I take it the feeling would be rather in favor of these methods of skin grafting, which really give very little difficulty and considerable advantage. As to your remarks, sir, about the carotid sinus, I have no information.

Dr. Myerson speaks about stasis of the pulmonary circulation. In the mechanism that I was speaking about, of changes in  $\text{CO}_2$ , that would be secondary to failure of respiration, I think; actually, in these cases, even of an extreme degree when the patient is on the borderline of danger, there is no sign of stasis as evidenced by lack of cyanosis, but I expect it is a slightly different problem he is referring to. Helium, I know nothing about.

Mr. Bateman was speaking about a case that I had already heard of. I am not alone in England in using this method, because once a matter of this sort gets recognized, it spreads very quickly, and as we are lucky in having the coöperation of men who devote themselves to plastic surgery, as Mr. Bateman with Mr. Kilner, so have I sought their advice and coöperation.

I was extremely glad to see the diagrams and to hear the remarks of Dr. Figi, because I knew well that he had used this method for a considerable period of time; one thing I have learned is that one can extend the method to other types of cases. I have had one or two patients in whom I thought I could not restore the airway sufficiently for purposes of respiration, but only for purposes of voice. Dr. Figi said that in his case he thought the voice would come back. I certainly think that the voice is of greater importance to the patient than the power of breathing through the larynx, because it is easy for the patient to breathe through the tracheotomy tube; but if the patient is without voice, he is in an unhappy position. I have not the slightest doubt that the voice will be restored in the case he was referring to.

I have heard of one case of Sir Harold Gillies and Mr. Capps in St. Bartholomew's Hospital, where they had the problem of lack of sphincteric action to deal with. I rather fancy that when the muscle fibers in the aryepiglottic folds have been destroyed, so that the laryngeal aperture is unable to close, one could carry out a plastic operation of transplanting some of the inferior constrictor fibers to form a new sphincter. I can see no reason at all why, in the act of swallowing, during contraction of the inferior sphincter, it should not be made use of, to close the aperture of the larynx. That would require working out.

I think I am right in feeling that your Association would be generally in favor of the use of skin grafts in combination with these other methods.

FINAL RESULTS OF FIBROMA OF THE LARYNX IN A  
CHILD OF THREE YEARS. TRACHEOTOMY,  
REMOVAL BY LARYNGOFISSURE,  
PROLONGED INTUBATION

THOMAS J. HARRIS, M.D.

I FIRST saw the child in June, 1904, when she was admitted to the Manhattan Eye, Ear and Throat Hospital suffering from dyspnea which required an emergency tracheotomy. There was a history that she recently had been operated upon for removal of adenoids at the Post-Graduate Hospital. At this time there was no complaint of difficult breathing. Following the tracheotomy, examination under ether by the indirect method showed the cause of the stenosis to be a newgrowth blocking the larynx. A biopsy was performed and the laboratory reported the growth to be an angiomatous-fibroma. Removal by laryngofissure was decided upon. Operation showed that the growth was attached anteriorly to the thyroid cartilage. The tracheotomy incision was found to have been carried so high up as to involve the larynx. On that account, we were concerned about the behavior of the laryngeal wound. Our fears were realized. Due to the unhealthy condition of the tracheal wound, the incision in the cartilage became ulcerated and one wing was virtually destroyed. To prevent cicatricial stenosis an intubation tube was introduced.

The child was presented before the section of Laryngology of the New York Academy of Medicine, the following March (1905). At that time she was apparently well, there being no evidence of newgrowth in the throat. In the course of a few months, however, difficulty in breathing recurred and she was readmitted to the hospital. Examination showed the larynx to be filled with newgrowth. This was removed by Dr. John Rogers, who performed a second laryngofissure. The hospital laboratory reported this time that the growth was a papilloma. There was no recurrence of the growth, but long wearing of the intubation tube caused a virtually complete stenosis of the larynx. This was overcome by repeated dilatations by the use of urethral sounds introduced

through the tracheal opening. I have seen the patient from time to time in the intervening years. There has never been any return of the laryngeal symptoms from which she suffered as a child. An examination recently made (January 10, 1936) showed a decided improvement over a few years ago. The lumen of the larynx is diminished in size but the airway is free and the patient has no dyspnea. The right cord appears to be buried in a mass of fibrous tissue and the true and false cords seem to be fused together. I reported the case at the Annual Meeting of this Association in 1904 and again in 1905. At that time the child could not speak above a whisper. In the intervening years the quality of the voice has remarkably improved and in view of the laryngeal deformity is surprisingly good. If not raised or used too long it is clear and strong, although high-pitched and peculiar in character. It would appear to be produced by the false cords.

COMMENT. 1. This case is reported because of the opportunity it affords to contrast methods of treatment today with those in use a generation ago. It is further an excellent illustration of the value of follow-up reports over an interval of time. Often further study brings forth a revised or altered diagnosis or line of treatment.

Thirty-three years ago the *direct* method of laryngeal examination was little known. In the discussions which followed the reading of the paper, Dr. Payson Clark said he thought the case would be an excellent one for the use of the Kirstein autoscope, an instrument superseded and well-nigh forgotten today. The advances and achievements of direct laryngoscopy since that time are nothing less than marvelous and yet Dr. Jackson has never failed to emphasize the importance of the mirror examination, and innumerable have been the brilliant operations which have been performed by this method.

2. INTUBATION. Scarcely less noticeable is the lessened frequency of intubation today in a throat hospital, as compared with three decades ago—due undoubtedly in large part to the routine employment of Klebs-Loeffler antitoxin. It is noteworthy that at the time when the intubation was so popular, J. Solis-Cohen, one of the most eminent of the pioneers in laryngology in this country, said in discussion of the paper that he preferred tracheotomy to intubation.

*Laryngofissure.* Laryngofissure is today an accepted mode of procedure for the removal of newgrowths in the larynx but undoubtedly in this case the operation, if performed today, would have been by the direct method.

*Tracheotomy.* The case illustrates forcibly the importance in a quick tracheotomy of avoiding the carrying of the incision so high up as to cut the cricoid cartilage.

In this case, this undoubtedly accounted for the infection which followed the laryngofissure, with no well shut-off barrier existing between the tracheal wound and the larynx.

3. CHANGE IN THE NATURE OF THE GROWTH. Fibroma of the larynx in adults, according to Sir Felix Semon, is only second in frequency to papilloma, but is rare in children. Transformation into a papilloma is almost unheard of and at the original reporting, we were unable to find a similar case requiring tracheotomy.

*Dilatation.* In this day of advanced laryngeal technique, occasion for any other than the usual method would almost never be required. In our case, however, the retrograde approach through the tracheal wound proved of inestimable value, and led to a successful result.

*Voice.* Finally the case is a beautiful illustration of the recuperative power of the voice over a period of years. When first shown the child was hardly capable of a loud whisper—just what would be expected with a larynx so badly mutilated; but gradually year by year constant improvement has taken place until as reported, the young lady now has an excellent working voice.

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#### DISCUSSION

DR. HENRY L. SWAIN, New Haven, Conn.:

I do not recall whether this was the same case but I have had the honor of having seen just such a case for many years. After all these years I cannot be sure whether it was one of your cases or my own.

My case was a young girl, hospitalized, four years of age, upon whom a laryngofissure had been done to remove multiple papillomata which had become too numerous to manage in any other way. She then passed out of my observation and returned twenty-two years afterward, a healthy young woman. She whispered, "You don't



recognize me." Though I have forgotten the name now, I remembered it and she acknowledged it. I was very much interested in her. She was in excellent health, married and the mother of two children, but would not attempt to go without her tracheal tube in place.

What interested me particularly was to observe the effect in an adult who had not had the normal and customary use of her larynx, since four years of age. I expected to find a very much atrophied or poorly developed larynx, but it was not far different from one you would expect to find in a woman of her size and age, even under normal life conditions. The thyroid wings and surrounding structures seemed quite right and proper. Again I tried to induce her to go without the tube, as there seemed to be no real obstruction, although perhaps a slightly smaller larynx, but was unsuccessful.

She returned some years later with more courage and, aided by frequent dilatations, attained sufficient confidence to go without the tube.

I saw her five years ago. She appeared healthy and one could understand her speech fairly well.

DR. JOSEPH C. BECK, Chicago, Ill.:

Mr. Chairman: I want to speak of two types of pathological entities which are described in this paper, one, an angiomatous fibroma, and the second, an angiomatous fibroma becoming a papilloma. Of course in literature we more often find that a papilloma becomes malignant and an epithelial growth, but this transformation is not very common and having obtained examination reports from two different laboratories, find that the histologist-pathologist sometimes makes a mistake.

I would not be so definite in recognizing two different pathological entities. I think we are probably dealing with a papilloma or angio-fibroma.

## USE OF THE HYOID BONE AS A GRAFT IN LARYNGEAL STENOSIS

EDWARD A. LOOPER, M.D.

THE whole subject of laryngeal stenosis is a complicated one. To obtain a cure each case requires individual study and has particular problems in treatment. At best, these cases are tedious and difficult, many requiring treatment over a long period of time—from months to years. Even then, the final result may not be as good as desired.

For many years the greatest number of these patients have been children, secondary to improperly performed tracheotomies, a subject so often emphasized by Dr. Chevalier Jackson. Due to his efforts, surgeons have been gradually instructed in the proper method of performing a low tracheotomy, and such cases are fortunately becoming more rare.

Another factor which has contributed greatly to the decrease in these cases has been the advancement in treatment of laryngeal diphtheria. The brilliant results of prophylactic treatment of this disease with toxoid, the great interest shown by State Health Departments in the control of diphtheria, the establishment of modern quarantine hospitals, with staffs trained to administer large doses of antitoxin, intravenously when necessary in severe cases, have been one of the outstanding triumphs of modern preventive medicine. In such hospitals, when serious laryngeal involvement develops, a successful intubation can be quickly performed, obviating the necessity of a tracheotomy with its possible disastrous after-effects of laryngeal stenosis. So we can look to the future with assurance that we will have less and less of these complicated cases.

However, with the desire for fast transportation in airplanes, millions of automobiles, and other conveyances, accidents are proportionately increasing. In this group, injuries to the larynx are common. Lacerations are often deep, with resulting deformity and stenosis. Consequently, these cases have now become one of our most important problems in treatment.

In June, 1937, a patient was sent into the hospital with a history of having sustained a severe injury to his larynx following a railroad accident. On admission to the hospital, the patient was wearing a tracheotomy tube and was suffering with a badly lacerated larynx. In planning his treatment, it was obvious that some sort of graft would be helpful in this particular case. The thought occurred to me that it might be possible to make some use of the hyoid bone as a graft in such a condition. So after experimenting on a cadaver, the operation presently to be described, was tried with very good success. The patient's convalescence was greatly shortened and a satisfactory airway was established more quickly than had been expected.

Since that time this procedure has been used on other patients with results which have made me feel that it is an operation worthy of reporting, even at the risk of its being a new method of treatment in the preliminary stages of development.

This operation would seem to be best adapted to traumatic injuries of the larynx in adults. It has not been tried in children and in its present state is not suggested for these cases.

**OPERATION.** *Anesthesia.* After using tribromethanol anesthesia for a number of years, we have found it a most satisfactory basal anesthetic for laryngeal surgery. The patient goes to sleep quietly without excitement, his blood pressure is lowered, he breathes more slowly and sleeps for a longer period of time. Rectal administration of the drug keeps the anesthetist out of the way of the operation and there is no irritating action on the respiratory mucosa, so common with gaseous anesthetics.

It is to be preferred to local anesthesia, as with the patient unconscious, the operation can proceed more rapidly and there is little shock to the nervous system; a factor of importance in any surgical operation. So we now employ this method as a routine procedure in most of our cases.

*Technique of Operation.* The skin surface of the anterior part of the neck from the chin above, to the sternal notch below, is carefully shaved and cleansed with soap and water. This can be performed in the patient's room if desired. While going to sleep on the operating table, the neck surface is made aseptic by alcohol,

ether and iodine, or merthiolate, depending upon the choice of the operator. The patient is now draped in the usual manner.

As a previous tracheotomy has been necessary, the tracheotomy tube should be removed, any granulation tissue present trimmed away, and the raw edges cauterized with 10 per cent silver nitrate solution. A larger size tube is then inserted and tied with tape. The neck is extended and firmly fixed with pillows or small sandbags.

A vertical incision is now made in the skin, beginning above at a point on a line with the upper margin of the hyoid bone. (Fig. 1.)

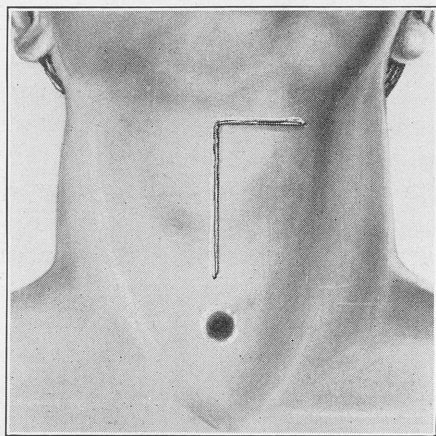


FIG. 1.—The incision in the skin extends from the lower margin of the thyroid cartilage upward to the center of the hyoid bone and then to its left tip.

This incision is carried downward about 1 inch to the left of the midline of the larynx to the lower end of the thyroid cartilage. By keeping the incision to the left you have a better chance of a good blood supply to the upper surface of the graft and it can be more securely covered over, than when the incised edges are directly in line with the graft. The incision should not be carried as far down as the tracheotomy opening, because it is important to retain an adherent unbroken bridge of tissue between the laryngeal opening above and the tracheotomy wound below. With this normal area left intact there is less possibility of contamination



and infection above. The tip of the graft will also be better protected and will have a more satisfactory blood supply.

From the upper point of the incision near the center of the hyoid bone, the incision is carried horizontally to the left on a line with the hyoid to its end. The subcutaneous tissue is separated, and the thyrohyoid muscle is cut across at its upper attachment. The anterior surface of the hyoid bone is now exposed throughout its entire left half. Beginning at the tip great care should be used

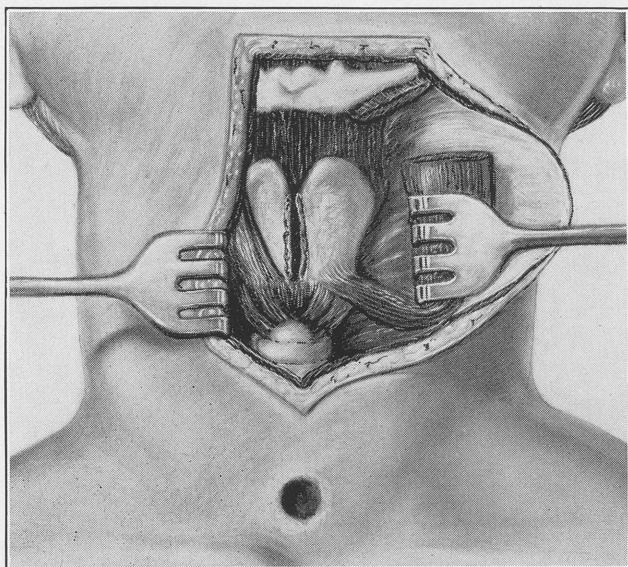


FIG. 2.—The hyoid bone is exposed, the thyroid cartilage incised and a bed made for the graft.

in freeing the bone from its posterior attachment; if a perforation occurs here, infection from the pharynx may develop with serious complications.

In dissecting out this end of the hyoid bone no effort should be made to skeletonize its upper, lower, and anterior surface as some adherent soft tissue is advantageous because it gives the graft better blood supply and protection. After being freed and elevated the end of the bone is now made ready to be swung into the desired position in the larynx. (Fig. 2.)

The bed for the graft requires particular preparation. A vertical incision is now carefully made in the thyroid cartilage down to the mucosal lining. The desired amount of mucous membrane is separated from the cartilage by submucous resection. Every effort is made to prevent injury or perforation to the interior of the larynx. Cartilaginous deformities, irregularities, and scar tissue formations, when found, are corrected if possible.

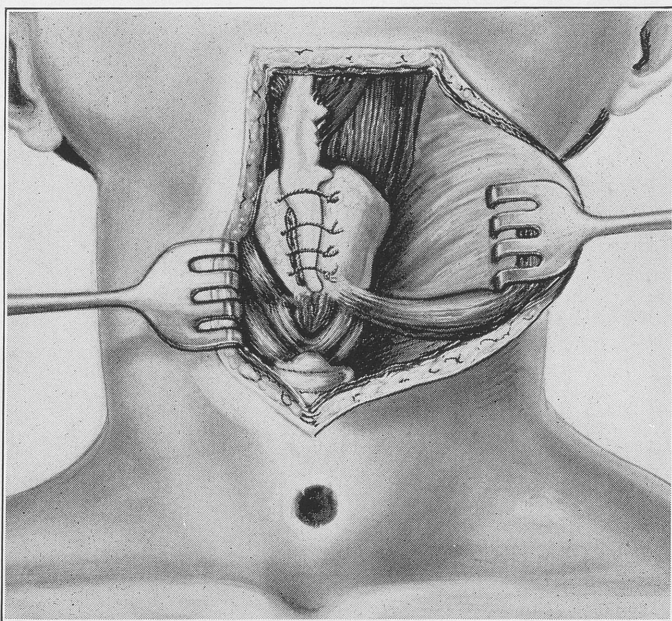


FIG. 3.—The end of the hyoid bone is rotated and placed between the separated thyroid cartilage.

The incised edges of the thyroid cartilage are now retracted, the end of the hyoid bone is placed between them and securely held in place with chromic catgut sutures. (Fig. 3.)

If perforation or infection is suspected in the laryngeal area, a small rubber tube drain should be inserted at the lower point of the wound for a few days. If perforation has occurred in the hyoid region, a small rubber tube drain should be placed in the left end of the wound.

The overlying structures are now replaced, bleeding vessels tied and the skin is closed with fine silk. The wound is covered with rubber tissue and vaseline gauze. (Fig. 4.)

A feeding tube should be inserted as in laryngectomy cases, to be retained for about ten days.

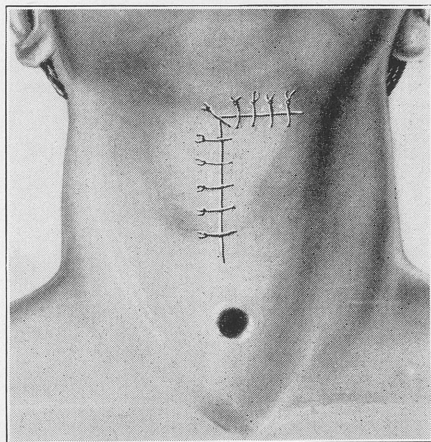


FIG. 4.—The wound in the skin is closed with fine silk.

*Postoperative Treatment.* These cases require the same careful after-treatment as laryngectomies. A suction machine should always be at the bedside of the patient. The resident should be impressed with the importance of aspirating bronchial exudate to prevent pulmonary complications. The patient's position should be changed frequently so as to lessen the possibility of bronchopneumonia. The wound should be frequently dressed and, if infection has developed, more drainage may be required.

If secondary hemorrhage occurs, the bleeding-point should be tied off locally if possible. Should bleeding be continuous and severe, ligation of the external carotid artery can always be performed with the assurance that the hemorrhage will be controlled.

*COMMENT.* This operation is proposed as a method of utilizing the hyoid bone as a graft in the treatment of certain cases of laryngeal stenosis.

The principle depends upon embedding the left end of the

attached hyoid bone, between the incised thyroid cartilage, to act as a wedge in enlarging contractures and deformities of the larynx and to permit a better airway. This firm bony graft acts as a splint to weakened and deformed cartilage. The ease with which the hyoid bone can be exposed, detached, and rotated makes it a practical procedure.

A living, attached, and accessible graft with the upper part of its blood supply undisturbed has advantages over "foreign" embedded grafts, such as cartilage from the ribs, ears, or other parts of the body.

The operation is an improvement in the treatment of certain cases of laryngeal stenosis in adults, resulting from injury. It is not proposed as a perfect and immediate "cure all" for every patient with laryngeal obstruction, and has not been tried upon children.

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#### DISCUSSION

DR. THOMAS E. CARMODY, Denver, Colo.:

I would like to ask a question. I understood in the first place that you cut this loose, but from the picture you still leave some attachment.

DR. LOOPER: Yes, sir.

DR. CARMODY: I have to operate soon, and I thought of using a rib instead, but I think this would be very much better if you can leave your graft attached. Does that interfere with deglutition at all?

DR. LOOPER: For a short time—a couple of weeks.

DR. MYERSON: I would like to ask, what is the exact anatomical condition within the larynx for which this operation is indicated?

DR. LOOPER: I think it is principally for those which are more or less external deformities and conditions where there has been a great deal of traumatism, irregularity, principally of the thyroid; certainly not indicated, in my opinion, in children or any post-tracheal conditions.



## POSTURAL TREATMENT OF ACUTE LARYNGO- TRACHEOBRONCHITIS

T. C. GALLOWAY, M.D.

RATHER clear pictures of acute infectious laryngotracheo-bronchitis have been given in the past twenty years by Baum,<sup>1</sup> Gittins,<sup>2</sup> Jackson,<sup>3</sup> Richards,<sup>4</sup> Smith<sup>5</sup> and others, so that little further discussion appears necessary. The mortality of the disease, however, remains high, averaging, according to Jackson,<sup>3</sup> about 70 per cent in cases requiring cannulation, although in Smith's<sup>5</sup> series it was only 33 per cent. This paper is presented to emphasize two factors that may tend to improve that rate.

The first point is that the secondary changes initiated by obstruction of the airway in the presence of infection may very soon become irreversible and progressive unless there is intervention which should be done earlier than it usually is done. The second is that postural drainage is a very valuable method of treatment but that it is frequently neglected or used indifferently or without full appreciation of the factors involved.

Certain things about this disease seem to have been rather definitely established. If not indeed a true clinical entity it has definite recurring characteristics determined by anatomical, physiological and bacterial factors. Its occurrence is not very rare and it may be reported more often when it is generally appreciated and recognized.

Symptoms are likely to include those of a mild upper respiratory infection followed by cough, hoarseness, increasing stridor, restlessness, dehydration, sepsis of varying degree and signs of cardiac and general exhaustion. Obstructive symptoms usually dominate the picture, though these may be slight if the obstruction is low in the bronchial tree or if sedatives have been given. Cyanosis is an alarming symptom, especially the pale cyanosis of exhaustion seen perhaps more often in low obstruction. The severity of symptoms is out of proportion to physical and roentgenologic findings.

Primary involvement may be chiefly of the larynx or subglottic areas, of the trachea, or of the bronchi. Usually the process appears to descend from the larynx and the whole respiratory tree is likely to suffer. Richards<sup>4</sup> and others have well described the end-state seen postmortem and the Jacksons<sup>3</sup> have given in detail the living pathological picture as related to the symptoms.

Diagnosis should not be difficult. Diphtheria, foreign body, croup, laryngismus stridulus and other causes of respiratory difficulty are to be ruled out, if necessary, by direct laryngoscopy or by bronchoscopy. The sudden onset of cyanosis with restlessness, especially if not explained by physical findings and particularly in the type of case considered an atypical bronchopneumonia should call into consideration this disease.

In order to visualize the processes of this condition, to understand the varying picture and to judge how soon it may be necessary to intervene to prevent irreparable damage, it may be useful to review some of the experimental work on obstruction of the airway as well as that on the pathogenesis of acute pulmonary infections.

Since 1844 many observers have shown that atelectasis and emphysema can be produced by bronchial obstruction. Corylos and Birnbaum<sup>6</sup> attempted to show that the important condition was prevention of entrance of air and absorption of that remaining. Jackson and Manges<sup>7</sup> demonstrated that foreign bodies, especially of the vegetal type, might have the same effect. Galbraith and Steinberg's experiments<sup>8</sup> seem to show that the essential factor is interference with respiratory movements due often to obstruction but often also to other causes which prevent inflation and deflation of the alveoli, leading to secondary vascular changes. Those changes which they considered most important were congestion, stasis, edema and trasudation.

Kernan and Barach<sup>9</sup> emphasized the importance of these circulatory effects and concluded, "As a result of the continued obstruction and the elevated negative pressure in the chest, blood enters the right chamber of the heart and lungs more freely, but is retarded in its departure from the lungs into the left heart as a result of the backward suction pressure on the left ventricle as it delivers blood into the extra-thoracic aorta. The elevated nega-

tive pressure within the chest exercises suction on the capillary walls, which is the important factor in the production of intra-alveolar exudate." They believe air is absorbed with production of atelectasis, and emphysema comes from increasing distention of the alveoli during inspiration, with rupture of blood-vessels and giving way of damaged alveolar wall. Increasingly greater respiratory effort keeps up a vicious circle.

For a clearer picture of what happens when infection is superimposed on these mechanical factors we may turn to recent work on the pathogenesis of pneumonia. Nungester and Jourdonais<sup>10</sup> were able to initiate pneumonia in dogs by introducing bacteria protected by mucin. Edema fluid carries from this focus multiplying bacteria. The spread from the primary infection, according to Loeschke<sup>11</sup> after many postmortem examinations of human material, is by gravity and by positive exudate pressure in a negative pressure lung. According to Gun, 12 spreading areas coalesce and if bronchi are not blocked, the exudate flows into open alveoli of lobe or lobule, there are fibrin deposits, leucocytic infiltration and the progressive changes of pneumonia.

If fibrinous plugs or infected exudate block a whole lobe we may have lobar pneumonia which Corylos and Birnbaum<sup>13</sup> considered really massive atelectasis with infection by the pneumococcus.

It seems also, in view of the experiments of Galbraith and Steinberg, that if there is much secretion, and yet not enough to block all of the bronchioles and so cut off the interlobular peripheral airways of Van Allen,<sup>14</sup> lobular pneumonia may result. If a plug blocks a bronchus or if so many of the bronchioles are blocked by thick secretion that the peripheral interlobular respiratory mechanism does not operate, one can think that air absorption and atelectasis might occur early before the open alveoli can fill with exudate, with the picture as it is in acute laryngotracheo-bronchitis.

The bacteria present, the reaction of the tissue to them, and especially the character of exudate or transudate produced may have much to do with the end-result, as already stated in regard to lobar pneumonia. Reports vary widely as to the organism most common in tracheobronchitis, the hemolytic streptococcus usually being given first place. Bradford and Leahy<sup>15</sup> believe the usual

cultural methods allow this organism to overgrow. By a careful plating technique they showed in a series of 36 cases that green-forming streptococci were predominant, and most cultures ran less than 5 per cent of hemolytic streptococci. The tissue reaction to this organism seems perhaps more compatible with the usual findings.

A rather narrow margin of chance may determine the end-result in certain cases of respiratory infection. It may be that when primary obstruction is late or incomplete, and exudate early invades open alveoli, the picture is one of lobar or lobular pneumonia. If there is primary obstruction with air absorption or if the primary process causes the outpouring of thick secretion or fibrinous plugs to block regional bronchioles before the alveoli are infected, atelectasis occurs, usually with emphysema. If the patient survives, the train of results may be as noted by Galbraith and Steinberg<sup>8</sup> in experimental animals: atelectasis, pneumonitis, focal necrosis, abscess formation, with later lung abscess or bronchiectasis.

That the picture may be a composite of these two types of processes is evidenced by many clinical observations. Holt<sup>16</sup> observed that emphysema occurred in the majority of children dying of lobular pneumonia. Cassas and Snow<sup>17</sup> observed roentgenologically, in many cases resembling bronchopneumonia, patchy atelectasis and emphysema with shifting roentgen and physical findings. They noted that the greatest involvement was in the dependent portions. It is an observation that cyanosis in lobular pneumonia, especially of the influenza type, may be of rather rapid onset and out of proportion to the physical findings.

This also would indicate that many of these cases might be amenable to the treatment to be outlined, although as Jackson,<sup>3</sup> Gittins,<sup>2</sup> and others emphasize, many cases considered bronchopneumonia, must be laryngotracheobronchitis. It is interesting to note that Kernan<sup>9</sup> did bronchoscopic aspiration in lobar pneumonia with reported benefit.

We may perhaps assume then, that the sequence of events in acute laryngotracheobronchitis is somewhat as follows: An infection involves the larynx and subglottic tissues, and congestion and edema lead to nearly enough complete obstruction to raise the



negative intrathoracic pressure, with added congestion, edema, exudate and capillary damage. Then thick secretion blocks bronchi or bronchioles, whose walls are already swollen. As spreading secretion blocks the regional bronchioles, the interlobular tracts are cut off, air in the alveoli becomes absorbed, the dependent portions become atelectatic, the overlying ones emphysematous. Infection and local circulatory changes add to the exudate, to air absorption, to atelectasis and emphysema and put out of commission more lung bed and further embarrass the heart. Cardiac and general exhaustion may here draw the final curtain.

The foregoing has been detailed to show that the problem is not one simply of blocked airway with anoxemia and perhaps sepsis and increased cardiac and respiratory effort and finally exhaustion, but that the process is much more complex. Grave secondary changes may at any time supervene, and then any treatment may be unsuccessful.

In other words, the patient is likely to be saved early or not at all. This means that if a trial of oxygen therapy and other measures do not give relief, tracheotomy is to be done without further delay. Cyanosis and rapid pulse are really late signs of imminent heart failure and must not be waited for.

In the condition herein discussed, bronchoscopic aspiration is often a necessary and life-saving procedure. As an adjunct, postural drainage seems to have been neglected, as there was found no emphasis on its use in tracheobronchitis, even where so frequently mention is made of the adverse effects of gravity on secretion. Postural treatment is widely used by chest surgeons and internists, but there is little about it in medical literature.

Nelson<sup>18</sup> showed that even in bronchiectasis where sputum is so viscid and tenacious, proper postural drainage was very effective and resulted at least in temporary cures. He insisted on the patient lying inverted with the hips over a double inclined plane, with the position maintained for days or even weeks with only very brief interruptions. Cures by it have been seen in early cases of bronchiectasis in children and it is usually part of the medical routine in lung abscess.

One pediatrician,<sup>19</sup> who sees many patients, states that for a

year he has treated influenza by postural drainage, and has not since then had one case of pneumonia develop.

Postural drainage must be such that a real gradient is maintained from the main lower bronchial area to the trachea and larynx. According to Morlock,<sup>20</sup> this should mean at least 15 degrees elevation of the foot of the bed; but better, as much as the patient will tolerate, which will increase with use. In children the prone position with the foot of the bed raised provides a much better gradient and it is usually accepted easily.

Having in mind the physiopathology of acute laryngotracheobronchitis it would seem that here postural drainage would be especially valuable. The usual protective mechanism fails, secretion not being moved by cough, tussive squeeze or ciliary action. Secretion as it is first formed, whether as mucus, transudate, exudate or hemorrhage poured out in trachea, bronchi or bronchioles, must first be fluid, and will flow some distance along the dependent wall before it forms plugs or crusts. At first at least, ciliary action probably helps, and Leonard Hill<sup>21</sup> showed that cilia from the horse's trachea moved material twice as fast in the horizontal as in the vertical position. Murphy,<sup>22</sup> injecting into the bronchi of cats material of the viscosity of secretions found in asphyxia neonatorum, demonstrated that gravity drainage would clear the tracheobronchial tree.

If secretion can flow to the areas of greater cough reflex, tussive squeeze and cough will be much more effective. If any cough or squeeze or ciliary action is left, the help of gravity will be important. If secretion stagnates, it should be as a spread dependent layer and not as an occluding mass lying in a bronchial well. If plugs or crusts form, they will be nearer the outlet, as we have observed bronchoscopically after instituting postural drainage, and will be more likely to be expelled by cough, or will be more accessible to removal by aspiration or bronchoscopy.

Lavage of the respiratory tract has been proven feasible by Stitt<sup>23</sup> and by Mintern,<sup>24</sup> the latter using as much as 400 cc. of fluid at a time in adults. Gittins,<sup>2</sup> in tracheobronchitis used epinephrin and normal saline in small amounts, aspirating it afterwards. Richards<sup>4</sup> showed that sodium bicarbonate solution tends to soften and dissolve plugs.

If one is to use fluid it would seem that, after it is instilled, the inverted posture should be used to guarantee its return and also to aid in the movement of secretion and plugs toward the tracheotomy opening. In recent cases, 4 to 15 cc. at a time of 3 to 5 per cent warm soda solution have been dropped into the tracheotomy tube with inspiration, usually with the gratifying result that it stirred up vigorous coughing so that it was presumably drawn back and forth over crusts and plugs. This was instilled with the head elevated and the child turned toward the side on which the stethoscope indicated most blocking. The head was then lowered and suction used by a catheter through the tracheotomy tube. Normal saline or Ringer's solution followed to wash out the soda. Intermittently, when there was much blocking, epinephrin solution 1 to 10,000 was used, and not the stronger solution which is known to produce undesirable secondary congestion in the nose. This treatment has proven of much value in recent cases although it cannot supplant entirely bronchoscopic aspiration and forceps removal of crusts. It proved especially valuable where even a 3.5 mm. bronchoscope could not be passed. A capable intern can be trusted to use postural irrigation, and save the laryngologist constant attendance and the patient tiring bronchoscopies.

It is possible that very early, when only the high laryngeal or tracheal obstruction is present, and before there is secretion or exudation, postural inversion might, by gravity flow of blood, increase the congestion and therefore the blocking. Later its value would seem to outweigh any disadvantage.

It is possible that it should be used in some conditions like post-laryngeal surgery where we now raise the patient's head or allow him to sit up. He is more comfortable with the larynx high, for one reason because secretions gravitate to the lower bronchial area, where he is less conscious of them and they do not make him cough. This very fact permits their collecting where it is least desirable to have them.

Treatment will be outlined only briefly. It includes:

1. Rest—physical and mental—so that minimum oxygen is required.
2. No opiates nor atropine.
3. Oxygen by tent, hood or catheter or through the tracheotomy

tube. It is better given with helium with moderate positive pressure according to the principles of Kernan and Barach.

4. Fluids by mouth and parenterally to relieve dehydration and increase fluidity of secretion.

5. Moisture-saturated air by croup tent.

6. Tracheotomy or rarely intubation, not delayed if above do not soon relieve.

7. Postural drainage and aspiration.

8. Postural irrigation if secretion, plugs or crusts block.

9. Bronchoscopic removal of crusts if necessary.

10. Expectorants(?).

Of the 10 cases requiring cannulation reported from my service at the Evanston Hospital, 2 died. The cases were favorable because they were usually referred early by alert pediatricians whose collaboration in the general case was invaluable. Some of these cases were seen every few hours, day and night, for four or five days, and no cases are included from a service of a larger charity hospital where the author could not be in frequent attendance. In all it appeared certain tracheotomy was inevitable. The 2 which died were seen only a few hours before death and 1 of these had had depressing doses of morphine. The series is too small of course for conclusions to be drawn, but it may indicate that with the measures here outlined the outlook in this disease is not too unfavorable.

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#### DISCUSSION

DR. LYMAN GILDER RICHARDS, Boston, Mass.:

Dr. Galloway has been very kind in mentioning my name in connection with this condition. Many of you know that I have been interested in it. He has certainly carried us along one step further in achieving success. His mortality of only 20 per cent even in this small series is the best figure that I have come in contact with.

We must recognize that in addition to the mechanical effect afforded by this infection we have always to deal with a generalized edema and thickening of the bronchial and bronchiolar walls. I have hoped since these are mostly streptococcic infections, that sulphanilamide might be a usual adjunct. In the cases where I have tried it, the results have been rather doubtful. I shall, however, continue to use it. I think also that immune blood transfusions, about which we now

hear considerably more than we used to, may likewise prove to be a useful adjunct.

I am glad Dr. Galloway mentioned this matter of certain solvents for encrusted material, particularly as aided by this gravity method. Of these, sodium perborate has been the most efficacious, but of late I have used a proprietary medication which purports to have almost the constituency of normal blood serum, and found it to be very useful in releasing this encrusted material. In conjunction with this postural drainage method these solvents will certainly help to maintain a patent airway.

I notice in one of the cases that the laryngeal pathology is described as that of edema and swelling. This, in general, has not been my experience. I have seldom seen any real supraglottic swelling, but rather an involvement of the cord edges with attachment of this encrusted secretion to them, or to the subglottic walls below that point. Pathologically, it seems to me to differ considerably from the ordinary form of acute edematous swelling which we associate with most streptococcic lesions.

It is something of a comfort to know that someone else wrestles with these patients in an effort to restore them to health. Their incidence seems to me to vary somewhat with different years. I noticed in the last statistics which I reviewed that there seemed to be fewer cases which required bronchoscopic removal of these secretions. The treatment was very much the same, but complications seemed to be relatively infrequent, and with tracheotomy, patients recovered in a short time. Yet when one observes pathological slides of fatal cases and sees the extraordinary amount of emphysema, and atelectasis which is produced either by these secretions blocked in the bronchi or else by swelling of the bronchial walls themselves and infiltration and cellular accumulations within those walls, one realizes how extremely virulent these infections can be, and if a method as easily applicable as postural drainage with irrigation will in any way decrease our mortality, it certainly is a very encouraging situation.

DR. WILLIAM B. CHAMBERLIN, Cleveland, Ohio:

Dr. Galloway has emphasized the introduction of the bronchoscope. Certainly there is a great advantage in its introduction before performing a tracheotomy. One is not at all hurried, but can perform the tracheotomy at his leisure.

I simply want to emphasize again the procedure of which I spoke at a recent meeting of the American Laryngological Association, whereby we use the Mosher life-saver instead of the bronchoscope

because it is much easier to introduce, and also because one can introduce it not by sight but by feeling, and perform the introduction of the tube much more quickly.

DR. BERNARD J. McMAHON, St. Louis, Mo.:

I would like to say just a word relative to the suggestion of using sulphanilamide in the treatment of acute laryngotracheobronchitis.

The patients in whom it was used were suffering from a mixed infection, *including* a streptococcic. As a consequence, the favorable reaction to sulphanilamide therapy would be expected to be in direct proportion to the preponderance of the streptococcus in the pathogenic bacterial flora.

While we know that a toxic reaction of sulphanilamide is hyperpnea, we do not know whether there is an essential increase in the respiratory rate, and consequently could not avail ourselves of this reaction with assurance.

DR. GALLOWAY: Sulphanilamide was used in all these patients. I am very enthusiastic about this drug because I have seen personally 9 cases of meningitis recover on its use since January, 1937, but I thought it had no effect in these particular infections.

Of course, fluids are very important as these patients are practically all dehydrated. Blood transfusions were not used.

Sodium perborate was mentioned. Dr. Beck pointed out years ago that hydrogen peroxide used in the throat was liable to produce a foam which might actually asphyxiate the patient. Dr. Richards, is there any danger of that?

DR. RICHARDS: I have seen none from that.

DR. CARMODY, Denver, Colo.:

I would like to ask Dr. Galloway about his catheter. Do you use the ordinary catheter with the blunt end for suction?

DR. GALLOWAY: I cut off the catheter.

DR. CARMODY: That is what I was going to speak of, cut it off at an angle and pass it through a flame.

DR. GALLOWAY: It will not pick up your mucosa, however.

DR. GALLOWAY (in closing):

I have a feeling about this disease that it is not a very septic condition as a rule, primarily, but that the sepsis is due to secondary infection, and that it is not the infection *per se* that makes patients so ill, but the mechanical factors, with secondary vascular changes and exhaustion.

The life-saver is valuable, but it is important in these cases to look into the trachea with a bronchoscope and it is very important to be able to carry oxygen in and to aspirate secretions even before tracheotomy.



## PERICHONDRITIS OF THE LARYNX, TRAUMATIC AND INFECTIVE

CHEVALIER JACKSON, M.D.

THE purpose of this paper is to contribute some observations on the difficulties and pitfalls that beset the path of the laryngologist in his diagnosis and treatment of this disease and of the diseases in which it is a complication. Though these observations will be grouped under the usual subheads a complete presentation of each subdivision of the subject will be avoided as unnecessarily iterative.

The subject of perichondritis is of utmost importance because failure to recognize it early, or the slightest error in its treatment, may condemn the patient to a loss of part of the cartilaginous framework of his larynx, a calamity that will permanently injure his voice and may entail many tedious months of treatment of stenosis of the airway. Moreover, its early recognition as a complication is of vital importance in the decision as to the best plan of treatment in cases of cancer and of most of the other serious diseases of the larynx.

INCIDENCE. The decrease in the incidence of both the infective and traumatic forms of perichondritis within the range of my fifty years of personal experience is remarkable.

It seems logical to attribute the lessened frequency of the infective form to the advances of medical science and especially preventive medicine. It is necessary only to cite a personal experience of 68 cases of perichondritis complicating typhoid fever some of which have been reported.<sup>4</sup> In my earlier days at the Old Western Pennsylvania Hospital in Pittsburgh I examined the larynges of 651 patients with typhoid fever during a period of little over a year. It is interesting to note that in 1937 the Western Pennsylvania Hospital had 3 cases of typhoid fever.<sup>4</sup> With the decrease in the incidence of typhoid fever has of course come a corresponding decrease of this form of perichondritis. This and other diseases have been mastered by the work of physicians who have

unselfishly devoted themselves to preventive medicine. The improved methods of treatment devised by medical men for diphtheria, tuberculosis, syphilis and practically all of the general diseases that are causative of laryngeal perichondritis have greatly reduced the incidence of this complication which formerly was so distressingly common in the experience of the laryngologist.

The decrease in the number of cases of *traumatic* perichondritis seen in civil practice is explainable by sociologic changes.

The remarkable diminution in incised wounds of the larynx is partly due to the gunman having replaced the cut-throat in criminal ambitions, and partly to the passing of the razor as a weapon and as an implement of suicide. In my early days the backed razor, a common weapon in the general region of Wylie Avenue in Pittsburgh, used to supply me abundantly with cases of traumatic perichondritis. A blow was struck with the razor held in the back position, and the blow was aimed at the neck. The safety razor does not lend itself to combat. The neck is a narrow target for the revolver; the modern murderer usually aims for the larger targets, the chest or abdomen, unless at close enough range to be sure of hitting the relatively smaller and more actively moving head. The safety razor does not appeal to the man bent on suicide. For self-destruction the revolver has been more often used. It is held to the head, practically never to the neck. The modern trend however, is toward a leap from a high window. This is in part due to the increase in the height of buildings, but largely to the enormous increase in the use of alcoholic beverages. A suicidal jump is becoming a common termination of the drinking party in the many-storied hotel. In striking the ground the cartilages of the extremities and the spine are badly damaged, but the laryngeal cartilages escape unless the neck has caught on a wire in the descent; the patient does not survive the general injury, so even in these cases there is no perichondritis.

Occasionally, a patient comes in with a larynx cut open by a broken windshield, but non-shatterable glass has diminished the number of these cases, and, considering the number of automobile accidents, traumatic perichondritis of the larynx therefrom seems relatively rare.

Notwithstanding the decreased incidence of typhoid fever and

diphtheria, the better management of other causative diseases, and the lessened frequency of trauma, perichondritis of the larynx remains a relatively common disease, though much less common than it was forty or fifty years ago.

ETIOLOGY AND PATHOLOGY. *Infection.* As with most other inflammatory conditions, infections, pyogenic or specific, are etiologically concerned to a greater or lesser extent. For the infective agent to reach and get implanted in the anatomically protected and highly resistant perichondrium requires the aid of contributory factors. These may be divided into two groups, namely excessive virulence, impaired defense.

Besides the theories and facts well known to bacteriologists as causes of increased virulence there is, in the case of the laryngeal cartilages, the additional fact of the proximity of the nasal accessory sinuses, the lymphoid ring and, especially important, peridental, prolific incubational areas. Clinical experience indicates that the edentulous are less susceptible to perichondritis of the larynx. Speaking broadly we may say the chief contributory factor in most cases is oral sepsis and neglect of proper oral hygiene. Except in the edentulous really good oral hygiene is impossible.

Of perichondritis complicating the specific infectious diseases we have seen instances of the following: syphilis, tuberculosis, lupus, typhoid fever, diphtheria, smallpox, scarlet fever, chickenpox, pneumonia, measles, typhus, Vincent's infection, anthrax, blastomycosis, actinomycosis, xanthoma, and various dermatoses.

Though not known to be specific infections, malignant, xanthomatous, herpetic and some other ulcerative diseases may be conveniently included in this group, as may also various acute virulent septic processes such as erysipelas, tonsillitis, and peritonsillar abscess.

The causes to be classed as impairment of defense include specific infections, hemic dyscrasias, avitaminosis, wasting diseases and trauma.

The factor of diminished powers of resistance was indicated by the fact that of 68 cases of perichondritis occurring in typhoid fever, all occurred late in the course of the disease when toxemia and asthenia were greatest and resistance presumably least.<sup>4</sup>

Of the conditions reducing resistance, avitaminosis and the

hemic dyscrasias are important but relatively infrequent. We have seen typical cases associated with scurvy, pernicious anemia, purpura, mononucleosis and agranulocytosis. Blood changes with purpuric spots in the laryngeal mucosa was noted in some of the cases of perichondritis complicating typhoid fever.<sup>4</sup>

Among local conditions diminishing resistance to invasion choked lymph channels and thrombosed capillaries are important, but the chief local cause is some form of trauma. Of this there are two classes, trauma to the epithelial, mucosal barrier and trauma that directly reaches the perichondrium and its underlying cartilage. The factor of mucosal trauma by cough, in admitting through the epithelial barrier the infections that start the ulceration that later reaches the perichondrium, rests largely on theoretic considerations. We have therefore only to consider direct trauma to the cartilage and the perichondrium.

Pathologically it makes a great difference whether causative trauma has exposed the perichondrium to infection or not. An incised wound reaching but not completely dividing the cartilage is usually followed by satisfactory granulation and healing. If the cartilage has been completely divided healing is much slower, granulations have a tendency to become exuberant, and more or less necrosis of cartilage usually occurs before healing ultimately is completed. The most familiar example of this is the usual healing process following laryngofissure. This is the chief but not the only form of surgical trauma. Misplaced tracheotomic incisions were exceedingly common causes among the perichondritic stenoses before the days when high tracheotomy was condemned by Chevalier Jackson.<sup>5</sup>

There are numerous kinds of external trauma. We have seen many cases from industrial and other accidents; a fall in which the neck strikes any hard edge, a step, an automobile coat rail; a blow with a fist, a ball, ball bat; the tramping of a cow, a horse, a football player. The protected position of the larynx between the projecting chin and the prominent thorax, together with the yielding suspension of the larynx are remarkably efficient in protecting it from trauma. The burning high-voltage electric wire found its way between these two projections in 3 cases. There is in my experience a great diminution in non-surgical incised



wounds of the laryngeal cartilages and their perichondrium as mentioned in connection with incidence.

The prominent feature of the pathology is the poor reparative power of cartilage, which is even worse in the laryngeal than in the other cartilages of the body. Repair when it does take place is by fibrous tissue. To build up this fibrous tissue requires a long period of granulation, and the granulations are prone to the exuberance that retards healing. New cartilage or cartilaginous repair rarely if ever occurs in the larynges of adults. In children the factor of growth manifests itself in the usual way.<sup>1,3,6</sup>

Perichondritis may be limited to only a small area of one cartilage or may involve the perichondrium of any or all of the laryngeal cartilages. In the days before antitoxin we saw cases in which a low tracheotomy would save life, but the diphtheritic chondral necrosis would leave only a shapeless mass of fibrous tissue with an island of cartilage having no landmark to indicate the child ever had a larynx. Such cases occurred also in the then prevalent severe form of typhoid fever in adults.<sup>4</sup>

If there is much calcification of the cartilage involved the pathologic processes may resemble those of an osteomyelitis.

The pathologic mechanism of the stenotic sequelæ of perichondritis is composed of two factors, the cicatricial contraction plus the necrotic loss of stiffening cartilaginous framework that normally maintains an open airway. Fortunate indeed is the patient whose perichondritis has stopped short of chondral necrosis.

A pathologically unique form of perichondritis is that associated with contact ulcer. It is due to the hammer-and-anvil trauma of the vocal processes in vocal abuse. It is associated with necrosis of the tip of the vocal process of the arytenoid; but it differs from all other perichondritic processes in that it practically never extends, no matter how long it persists; months, even years. Another unique feature of the pathology of contact ulcer is that healing, which ultimately takes place, leaves no serious sequelæ; there is only a small epithelized scar binding together, and down tightly to the cartilage, the new tissue that has replaced the ulcer.

**SYMPTOMATOLOGY.** *Acute Perichondritis.* In the cases unassociated with trauma the onset is insidious and symptomless. There may be chills, fever, sweats, dysphagia, odynphagia, hoarse-

ness and local pain, but these are rare. In the cases complicating typhoid fever, for example, the patient would sleep away and asphyxiate without a complaint or sign of fight for air. Dr. John Boyce and I discovered many such patients at the Old Western Pennsylvania Hospital in time to save their lives by tracheotomy. Dr. Joseph Barach, the internist, who worked with me on those cases saw a typhoid fever patient dying in one of the wards of the South Side Hospital; recognizing the similarity in appearance of the patient he did an emergency tracheotomy at the bedside and resuscitated the patient by artificial respiration. Subsequent mirror examination showed the larynx closed by the swelling of a perichondritis. In these cases the profound toxemia and the asthenic condition of the patient have something to do with the masking of the symptoms, but there are cases not due to typhoid fever in which the symptoms are relatively slight.

In the acute traumatic cases the symptoms of the perichondritis are usually masked by the general local symptoms of trauma. If the injury has penetrated the mucosa there may be spitting of bright or old blood or clots. Hoarseness soon supervenes, then may come dysphagia, odynphagia and dyspnea. Cough is usually present and may be painful.

*Chronic perichondritis* is so insidious in its onset and so masked by the disease to which it is usually secondary that it may be regarded as, in itself, symptomless. The general inclusive group of symptoms are cough, expectoration, pain, aching, hoarseness, dysphagia, odynphagia, and the patient himself may have discovered local tenderness.

**DIAGNOSIS.** In the gross form of perichondritis with chondral necrosis and abscess involving the thyroid and cricoid cartilages and their perichondria, the diagnosis is readily made. There are a large tender mass palpable externally, wheezing or severe dyspnea, and a mirror image of massive edema. There remains, however, in most of such cases the question as to the presence and nature of the underlying pathology. This usually requires direct laryngoscopy and often biopsy for definite determination.

When the disease, even though equally severe and advanced is limited to one or both arytenoid cartilages the diagnosis can be certainly made by direct laryngoscopy with exploration of the

hypopharyngeal face of the posterior laryngeal wall. This will reveal the posteriorly discharging or pointing abscess. If already discharging, a sequestrum of necrotic cartilage, invisible in the mirror, may be revealed. In an earlier stage the closed forceps used as a probe through the laryngoscope will reveal a boggy mass and a very significant absence of the characteristic free passive motility. In the earliest stage the crico-arytenoid joint is fixed; later the arytenoid eminence feels like a bag of pus. The color varies; early it may be red, but later it is pale, though rarely watery in appearance.

If the cricoid alone is involved, both the arytenoids are somewhat swollen and usually more or less fixed, but they are not so large, so rounded nor so boggy as when the arytenoidal perichondrium is involved.

*Palpation.* The most important of all means of diagnosis of laryngeal perichondritis is palpation of the *laryngeal cartilages*. Even the smallest area of thickening from a no longer tender perichondritis is readily detected by the laryngologist who palpates delicately and carefully every larynx he examines. It seems strange that this method of examination is not even mentioned in many of the textbooks on laryngeal diseases, and is not regularly taught as one of the most important means of routine laryngeal examination. Palpation of the neck for lymph nodes is, of course, usually done, and especially in cases of suspected tuberculous or malignant disease; but palpation of the laryngeal cartilages is quite another matter.

To get the maximum diagnostic aid the palpatory fingers must be always protected from blunting injury. Playing baseball, tennis and golf, playing the cello or guitar, striking the piano or typewriter keys, lessen tactile sensibility. The scrubbing of the fingers in surgical preparation is not injurious if there is no free alkali in the soap, but alcohol and mercuric bichloride solutions harden the epithelium. Fingers yellow-stained from handling hot cigarettes are impaired in tactile sense. Finding etching acid ruinous to tactile sensibility I used only the dry point.

There can be very little pathologic change in the cartilage or its perichondrium that is not revealed to the educated palpating fingers. This education can be acquired by palpatory visualiza-

tion of the entire laryngeal framework in thousands of patients. Unless this is carried out carefully and skilfully, full justice has not been done to any patient needing examination of the nose, throat and ear. Therefore abundant material for the acquirement of this palpatory education is available to everyone dealing clinically with these regions. Having carefully outlined the borders of the cartilages, the external surface of the thyroid wings is carefully felt for evidence of inflammatory thickening. The cricoid ring is felt almost in its entirety. The region of the crico-arytenoid joint requires especially particular palpation, and it is reachable from the corresponding side, using the examiner's right index finger for the left joint; his left index for the right. For best results it is essential that the examiner have a mental conception of the unseen cartilages in the neck, comparable to the mental conception of the sculptor looking at a block of marble or of Doctor Mosher looking at a block of wood, and a tactile sensibility comparable to the blind person reading his raised Braille letters. After obtaining all possible information by sense of touch the next thing to be determined is tenderness to touch. Whether or not the patient has evinced any evidence of sensitiveness during the palpatory examination, the whole procedure should be repeated, the patient being repeatedly asked if there is any tenderness. Speaking broadly, total absence of tenderness rules out active acute perichondritis; slight tenderness indicates chronic perichondritis; thickening totally free from tenderness indicates a previous perichondritis; boggiess with slight tenderness indicates a chronic perichondritis with fluid exudate; fluctuation is certain evidence of suppuration and this means chondral necrosis.

It cannot be too strongly emphasized that the foregoing refers only to palpation of the laryngeal cartilages; not to palpation of the neck. Having carefully palpated the cartilages seriatim, the next step is *palpation of the neck* including especial care in determining the condition of the lymphatics. This must be delicately, carefully and deeply done to avoid overlooking of slight and early involvement. The inflammatory lymph node associated with perichondritis is soft and tender, the metastatic lymph node is usually hard and, early, it is shotty and free from tenderness. After endolaryngeal ulceration starts the central hard metastatic node



can be definitely felt as covered with a softer, inflammatory, tender cortex. The nodes of syphilitic perichondritis develop rather rapidly, are large, soft and tender from the beginning of their involvement. The tuberculous nodes infected by the tonsillar route are harder, higher and further back as compared with the nodes that come secondarily to tuberculous laryngeal perichondritis. All these differences are distinctly elicited by the touch that has been trained in the careful palpation of the laryngeal cartilages, as well as of the entire neck in every patient examined. They do not alone establish a diagnosis but they go a long way toward the complete comprehension of all the details of the living pathology in the particular case, so essential to the getting of the best results in the treatment of the different cases.

In all acute infective diseases, but especially in typhoid fever, the possibility of laryngeal perichondritis should be borne in mind, and mirror laryngoscopy, palpation and, if necessary, direct laryngoscopy, should be used to establish an early diagnosis. It is essential also, as previously mentioned, to have always, in mind that a toxic patient may asphyxiate without a fight for air, and without any significant symptom to call attention to the larynx.

*Endoscopic Appearances.* In the non-traumatic cases the appearances are usually masked by the primary disease, especially in such conditions as diphtheria, syphilis, cancer and tuberculosis. In the cases complicating typhoid fever, if the larynx has been watched, the early appearance of mucosal inflammation will serve as a harbinger of perichondritis.

If the condition is well developed there is usually evidence of intense inflammatory changes with pallid edema of the aryepiglottic folds and massive swelling of the arytenoid eminences. Sometimes only one of these will be affected; then the other may be only slightly swollen. Pus may be seen discharging and there may be a sequestrum of cartilage visible. In some cases I have seen an entire arytenoid free as a foreign body in an abscess cavity.

*Differential Diagnosis.* Edema seen in the mirror may be evidence of mucosal and submucosal serous exudate, but if it is associated with tenderness on external pressure a diagnosis of perichondritis is fully supported. Deep palpation with the thumb on one side and two fingers on the other will reveal a lesion on either

arytenoid or on the posterior segment of the cricoid. It cannot be too emphatically stated, however, that in all cases with edema or swelling of one or both arytenoids the first and most important diagnostic possibility is cancer of the party wall or hypopharynx; the second is laryngeal tuberculosis; the third is syphilis. These all may coëxist with perichondritis, but differential diagnosis is of the utmost and most urgent importance.

Differential diagnosis presents a number of other problems, most of them easily solved by direct laryngoscopy and external palpation, but otherwise insoluble. A motionless cord may be fixation by the arytenoidal perichondritis, or paralysis from a complicating recurrent neuritis; or a paralysis in no way related to any laryngeal lesion. As pointed out many years ago<sup>8</sup> the differential diagnosis between paralysis and fixation is readily and certainly made by passive motion imparted by the closed forceps used through the direct laryngoscope.

TREATMENT. The first and most important indication is to stand by for tracheotomy and do it early rather than late, and always low. As a rule, the tracheal incision should be below the first ring. A high tracheotomy is certain to be followed by laryngeal stenosis.<sup>5</sup> Intubation is contraindicated when perichondritis is present, even in diphtheria.

Except for an urgently indicated tracheotomy, no attempt should be made to treat a perichondritis of the larynx until its cause has been certainly determined, and this should always be done without delay. Whether perichondritis is present or not we may be dealing with foreign body, syphilis, tuberculosis, cancer or other serious disease calling for widely different methods of treatment and often calling urgently. In syphilis there is not a moment to be lost in commencing systemic treatment.

If there is abscess formation, drainage is of course indicated, but it cannot be too strongly stated that this must be conservatively done, and beyond this, surgical measures are contraindicated until the perichondritis has entirely subsided. To clean out the diseased area, resecting necrotic cartilage and attempting to get an aseptic wound with prompt healing is absolutely contraindicated in perichondritis of the larynx.

When tracheotomy is needed there is always a temptation to go

in through the inflammatory area to drain it. This is a mistake. The tracheotomy should, if possible, be in the grossly normal area close to the level of the upper border of the sternum. The drainage of the perichondritic focus should be through a separate incision and this should be no larger than necessary. The classic advice "lay it freely open" does not apply. Any incision or other instrumentation that damages living cartilage will make the acute condition worse and the chances of sequellar stenosis greater.

Local applications are of little benefit, but neither patient nor physician will feel altogether satisfied if they are not used. Cold application, especially the ice-bag, is indicated in acute inflammatory stages, but strongly contraindicated after suppuration starts because cold lowers the vitality of the cartilage, which is poor enough at best; necrosis has already started, usually, when suppuration develops.

*SEQUELÆ. Stenosis.* The most frequent and the most serious sequel of perichondritis is stenosis. The seriousness is not because of danger to life; under proper care and supervision the mortality is very low. The patient who is dependent on a cannula because of laryngeal stenosis is partially disabled until the stenosis is cured, and he usually feels his handicap more than does the man who has lost a leg.

In the treatment of the sequential stenosis nothing equals the systematic use of core molds.<sup>1</sup>

*Paralysis* occurred in 4 cases of perichondritis complicating influenza in the epidemic of 1918. The author's passive motion test left no room for doubt as to the exclusion of fixation of the crico-arytenoid joint. In a preceding epidemic (1889), 3 similar cases were noted but, as only indirect methods were used, one or more of these may have been cases of fixation. Paralysis of the recurrent nerve in cases of perichondritis may be due to pressure on the nerve or to infective neuritis. Only 2 cases of recurrent paralysis as a sequel of perichondritis have been seen since. Both were enlisted men in 1918. It seems curious that this complication was seen four times, three times and twice, in three epidemics, respectively, and none was observed in any of the other epidemics.

*Fixation* may be due to cicatrices or to destruction of muscular

tissue. In almost all cases, however, fixation is due to *ankylosis of the crico-arytenoid joint*. Ankylosis occurs in almost all cases in which the crico-arytenoid joint is involved in the perichondritic process, and this means practically all cases of perichondritis of the cricoid cartilage. It may escape in those rare cases in which only the perichondrium of the anterior part of the cricoid ring is involved. Ankylosis is the rule following perichondritis of the arytenoid cartilage. The only exception is in cases of contact ulcer. In these the perichondritis and necrosis are limited to the vocal processes of the arytenoid cartilage.

PROGNOSIS. There is rarely any danger to life from perichondritis of the larynx, considered apart from the associated diseases of which it is usually a complication. Asphyxia from laryngeal stenosis is the greatest danger, and this can be avoided by timely tracheotomy.

The prognosis as to after-results is somewhat less favorable in syphilitic cases.

In cases of traumatic perichondritis, prognosis depends largely on whether or not the trauma has introduced infection. Non-infected traumatic perichondritis, as sometimes occurs with chondral fracture, usually heals in a few weeks without chondral necrosis. The perichondritis of a razor cut may be many months in healing, and stenotic sequelæ occur in most of the cases. These stenoses are, however, amenable to proper treatment. If the cut has not gone clear through the laryngeal cartilages the perichondritis is usually limited and the healing rapid. If, on the other hand, the cut has gone through into the interior of the larynx, the healing will be slow and complications probable. Hence the importance of the backed razor.

SUMMARY. 1. The incidence of laryngeal perichondritis has decreased at least 50 per cent in the author's experience of fifty years. The decrease is due to the development, by physicians, of the science of preventive medicine, a development that has practically eliminated typhoid fever, and has enormously decreased the incidence of diphtheria, scarlet fever and other diseases. Better control of syphilis, tuberculosis and diphtheria has been a large factor. Decrease in traumatic cases has been due to changing



times, especially the advent of the safety razor and of the ubiquitous motion picture that has developed in criminals an ambition to be a gunman rather than a cut-throat and has taught the suicidally inclined to use a revolver or to jump from a high place.

2. Very early diagnosis of small areas of perichondritis depends altogether upon keenly sensitive palpatory fingers. The delicate sense of touch must be preserved by avoiding injury to the fingers and should be developed by constant practice in examination of the normal. Palpatory visualization of the cartilages must be distinguished from ordinary palpatory search for lymph nodes in the neck.

3. Treatment should be conservative. The radical work of the surgeon dealing with cartilages elsewhere has no place in surgery of the larynx.

4. The best results in dealing with sequelar stenosis, in the author's experience, have been obtained with core molds.

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## DISCUSSION

DR. CHARLES J. IMPERATORI, New York City:

I had hoped that Dr. Jackson would go into detail regarding the treatment of these conditions, and particularly regarding the use of core molds, and inasmuch as yesterday the treatment of these atresias was suggested by the various authors and those who discussed it, by the use of the laryngostomy and implantation of skin flaps or the Thiersch graft.

There was a patient I had that was similar to that of Dr. Figi's. A young child obstructed with papillomata that was radiated, that is, radium was inserted within the larynx. This was some fifteen years ago. This child eventually developed a complete atresia of the larynx. When Prof. Schmiegelow was here, we presented this patient to him, and he operated by his method. Whether or not it was the after-treatment, that he did not carry out himself, which, of course, is very important, the patient was made much worse, so that atresia became a passageway of a few millimeters in diameter throughout the whole larynx.

Then a junior assistant of mine operated upon this same patient, repeating Prof. Schmiegelow's operation, with the result that the patient developed a complete atresia of the larynx. This boy gradually grew up with a tracheotomy tube in him. He developed a buccal voice, and in the course of time he came to a hospital where I was and applied for treatment. I used the Jackson core molds on this boy. It was necessary to perforate the cicatricial area and the core molds were introduced one after another, and after a period of nine or ten months, we had this boy so that he could go without his cannula.

He has remained well now for a year and a half, without a cannula or a tracheotomy tube.

The other case was a similar story of a woman who sustained an injury following an automobile accident, but she would not submit to the continuation of the core molds. She has now moved away from New York, and Dr. Hollander is taking care of her in Chicago.

The other case was that of a woman who swallowed a foreign body. It was inexpertly removed and the esophagus was perforated. An emphysema of the neck resulted, but an immediate mediastinotomy was done and also a tracheotomy; also inexpertly done. A complete atresia of her larynx due to chondroperichondritis resulted. This case also was treated similarly with the core molds. So it would seem to me that this method has a great deal of merit, and it may be that

we will combine this method, that is, of the soft rubber with the laryngostomy and the plastic operation and the insertion of the Thiersch graft. However, the results are so good, and I have absorbed so much conservatism from our English guests, that I think I will continue with this core mold treatment rather than attempt extensive plastic operations, the results of which seem to me, unless done very expertly, too frequently to be failures.

DR. GABRIEL TUCKER, Philadelphia, Pa.:

I should like to ask Dr. Jackson if he would say something about the diagnosis and treatment of perichondritis that sometimes follows operation on the larynx.

DR. MERVIN C. MYERSON, New York City:

I am very glad that Dr. Jackson mentioned, as he terms it, the flip test. In paralysis we note a certain resiliency and lack of resistance of the vocal cord during the attempt to pass a rigid instrument through the glottis, but in the presence of a fixation of the crico-arytenoid joint due to previous inflammation and healing, we have a fixation which is very resistant. It is worth mentioning because very frequently we are called upon to bronchoscope tuberculous patients who have a fixation of one or both cords, which narrows the glottis greatly.

In such cases it is very important that we use a bronchoscope of the size only which will pass the constricted glottis. If one attempts to use the usual 7-mm. bronchoscope in an adult larynx when it is so constricted, one may be forced to do a tracheotomy later.

DR. THOMAS CYRUS GALLOWAY, Evanston, Ill.:

The statement that Dr. Jackson makes that there should be no primary closure in cut-throat seems logical and proper, but on surveying our statistics at Cook County Hospital we were surprised to find that the cases in which there had been primary closure apparently did better than the cases in which this was done later. There is this to consider, that if one can make primary closure, the wound is not flooded by secretions and infection may thereby be lessened.

DR. JOSEPH C. BECK, Chicago, Ill.:

It takes a lot of courage to attempt a discussion of Dr. Jackson's paper. We both remember a physician who had an infective type of

perichondritis, a streptococcic sore throat that started with pharyngitis and then developed a severe perichondritis, in which an emergency external operation was necessary.

I would like to ask Dr. Jackson why he termed this perichondritis when he removes a sequestrum and we know there is always a chondritis there.

Meyer insists upon classifying this disease as a chondroperichondritis. I think that is really the case in these conditions.

I have enjoyed this paper, as you all have, but I would like to have that point cleared up.

DR. CHEVALIER JACKSON (in closing):

I have already taken too much time, but courtesy demands that I respond to the questions and points raised.

The point made by Dr. Beck is, of course, a good one. Unquestionably perichondritis means chondritis and necrosis, and it is the necrosis of the cartilage which makes perichondritis serious; the cartilaginous framework is destroyed as soon as the perichondritis has advanced to the stage at which the cartilage loses its nutrition and becomes a foreign body; and it is always a septic foreign body.

Doctor Imperatori has raised a number of interesting points. He discussed different methods of treatment. I am glad to know that his experience has been favorable in the use of core molds. They have revolutionized our handling of chronic stenosis due to perichondritis.

Time did not permit me to go into the matter of treatment, but in a general way it may be said that in the acute stage it should be conservative. In the cicatricial stage the core molds are ideal, whether there is any lumen or not. You can create a lumen and enlarge that lumen with core molds.

Postoperative chondritis discussed by Dr. Tucker is an enormous field in itself and a very important one; a field with which, of course, every laryngeal surgeon is fundamentally concerned, but we will not have time to take up its many phases.

I agree with Dr. Myerson perfectly in regard to care in the use of oversized bronchoscopes.

Dr. Galloway spoke about closing the wound in traumatic cases. This can be done, and very often it will be successful; the patient and surgeon are then very fortunate; but the mortality will be higher in the cases of infected wounds. That does not apply to clean operations where one cuts the cartilage; even though the airway is not sterile



our technique is sterile and our management of it is sterile. We close the laryngofissures now whereas we used to pack them open always; we believe this is an improvement in the aseptic technique. But the traumatic cases that come in cut by the windshield or in the committing of suicide or attempted murder, and in various accidental cases, one does not know what infection has been introduced. It is much safer to pack the wound open and keep it aërated until we find out what kind of infection is present.

Dr. Lois D. Greene told me a few moments ago that they see many of these suicidal cut-throat cases in China; and that she has found our method of open packing very satisfactory in handling them.

In two or three weeks healthy granulations will be formed and then the wound can be closed without danger. By that time one knows what infection has entered, and moreover a certain degree of immunity to it has been established.

# CANCER OF THE LARYNX—IMMEDIATE AND ULTIMATE RESULTS IN 102 OPERATIVE CASES

HENRY BOYLAN ORTON, M.D.

It is well at times to review one's work in a special field by an analytical survey, and to evaluate these findings. In the contents of this paper, comprising 102 operative cases, these findings are tabulated in two groups, one under laryngectomy and the other under laryngofissure.

ETIOLOGY. In analyzing this series of cases, I can find nothing that would enlighten one as to the origin of cancer.

A history of heredity was found to exist in only 13 instances. Chronic irritation of some sort or other was noticed in 24 cases, or approximately 25 per cent.

Ninety-one cases were males and 11 were females, which gives a ratio of approximately 10 males to every female.

*Age.* Cancer is supposed to be rare under forty years of age, yet in this series we had 11 patients under forty, the youngest being twenty-five years and the oldest seventy-six years at the time of operation.

In the male group we noticed the largest number between the sixth and seventh decade of life. Two were of the colored race, 1 aged twenty-nine years at time of operation and the other twenty-eight years of age. The latter patient had a myxosarcoma of the larynx. (Fig. 2.) In the female group the largest number was noted to have occurred between the third and fourth decade of life.

## LARYNGECTOMIES—SHOWING SEX AND AGE AT TIME OF OPERATION

	Age: 25 to 30	31 to 40	41 to 50	51 to 60	61 to 70	71 and over
Male . . . . .	2	5	22	23	25	9
Female . . . . .	0	4	2	2	0	0

## LARYNGOFISSURE—SHOWING SEX AND AGE AT TIME OF OPERATION

	Age: 25 to 30	31 to 40	41 to 50	51 to 60	61 to 70	71 and over
Male . . . . .	2	0	0	1	1	1
Female . . . . .	0	0	2	1	0	0

In the etiology, excessive use of the voice played a part, in that we found that 30 patients had given a history of excessive use of the voice.

Considering the part that alcohol and tobacco played in the etiology of cancer, we found only 18 had used alcohol moderately or a little more, whereas 38 had used tobacco to excess. There has been no great increase of cancer from the promiscuous smoking of cigarettes either in males or females.

Syphilis as an etiological factor in preparing the soil for the growth of cancer, was observed in only 3 cases, 1 of which was in a colored man who had a myxosarcoma of the larynx. (Fig. 2.)

Papillomata of the larynx were found in 3 patients, who had been observed over a long period of time, with frequent removal of papillomata. In each instance, in the sixth decade of the patient's life the papillomata were found to have progressed to malignancy. One patient was observed in 1928 with papillomata of the larynx and was under observation for nine years before the character of the growth had changed to malignancy. This patient was operated on at the age of seventy-one years for total laryngectomy and is alive and well at the present time. Another patient, aged sixty-nine years, who had been observed over a period of fifteen years by very competent laryngologists, with removal of papillomata, was referred to me last year for operation. The third patient was seen by a very competent laryngologist in 1921 with chronic hoarseness and multiple papilloma of two years' duration. In this case the papillomata did not change to malignancy until 1932. It is interesting to note that all of these patients did use their voices to excess.

Pachydermia laryngis, which as Jackson and others have stated is a precancerous condition, was found in 2 cases. In one instance it was only after repeated biopsies were made that malignancy was diagnosed. This patient had a sinus from the larynx to the surface of the skin at the thyroid cartilage, and it was necessary to remove a large area of skin, fascia and muscle surrounding this sinus in doing the laryngectomy. This growth was of squamous cell type. The patient was operated on by total laryngectomy in 1927 and is alive and well at the present time with a very good pharyngeal voice.

Interesting facts as to irritating gases: we had 1 patient who had been in the World War and was severely gassed with mustard gas. Whether this was a coincidence or a reality it is difficult to determine as we had only one such patient. This patient was operated on in 1925 by laryngectomy; he is still living and has one of the best pharyngeal voices imaginable. (Fig. 1.)

**CLASSIFICATION.** In this series we have 62 intrinsic, 15 subglottic, 12 pyriform sinus, 2 epiglottic, 7 aryepiglottic fold, 2 arytenoid and 2 postcricoid: the last five are also known as extrinsic or epilaryngeal and hypopharyngeal.

*Intrinsic Cancer.* Seventy-five per cent of these cases, 77 in number, were intrinsic, originating in the interior of the larynx.

Intrinsic cancer in the inter-arytenoid area is rarely seen. Laryngeal cancer involving the ventricular band and ventricles is also rare, and is more dangerous than a true cordal cancer. A growth situated in this area causes early glandular involvement. The vocal cord is the favorite site of typical intrinsic laryngeal cancer. This usually spreads along the cord, may become subglottic and spread to the opposite cord, or cross at the anterior commissure.

In the intrinsic type, or cordal cancer, lymphatic drainage is almost negligible, and the operative results by laryngofissure are from 75 to 85 per cent cures, with practically no operative mortality. In this series of 8 laryngofissures, we have 6 patients alive and well at the present time, the period since operation ranging from ten to eighteen years.

The symptoms of intrinsic cancer depend upon the site of growth. A growth affecting the tension, vibration and approximation of the cords will produce hoarseness.

In the intrinsic true cordal cases, the cancer is located on the surface or edge of the vocal cord. These may, and oftentimes do, extend into the ventricle of the larynx and below the cord into the subglottic area.

**SUMMARY.** Intrinsic cancers are usually benign in the early stages. They grow slowly and if limited to the vocal cord do not metastasize to the glands. The type of malignancy here is usually squamous cell epithelioma. Occasionally we have a basal cell type. Adenocarcinoma is usually seen in the ventricles. Other forms are rare. We must differentiate this type from singer's



nodule, leukoplakia, innocent tumors, tuberculosis and syphilis. The course usually extends over a period of several years, depend-

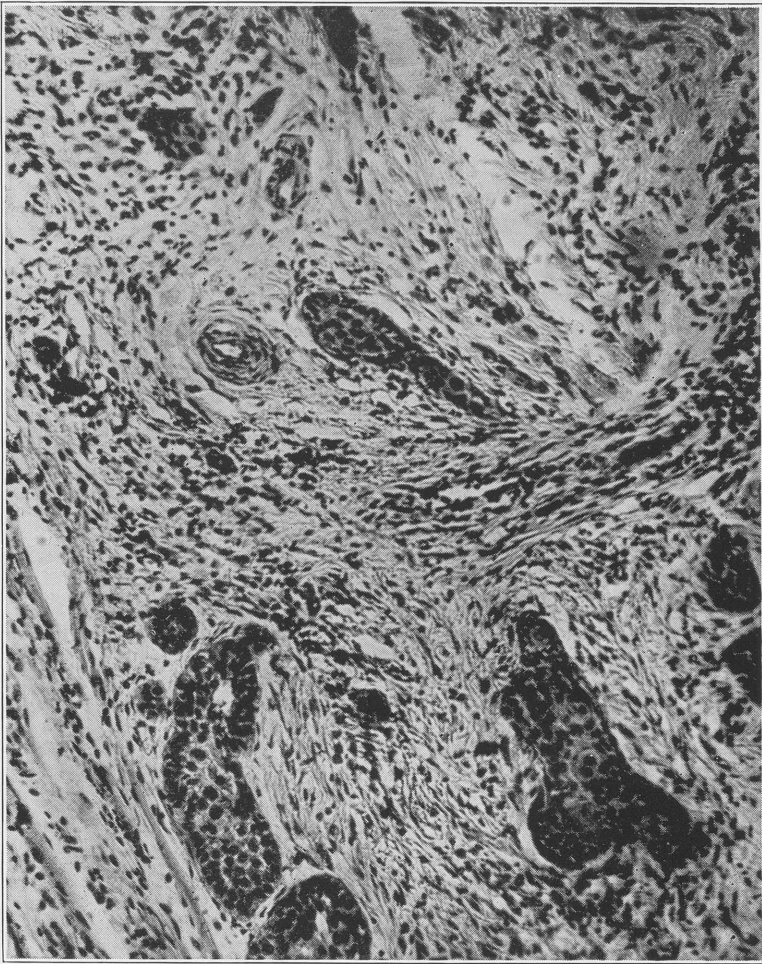


FIG. 1.—Basal-celled carcinoma. Shows an invading malignant growth of all the layers of the surface epithelium. Low power.

ing upon the location and grade of tumor. Even the most malignant forms usually last over two years.

This type of cancer is the most favorable for cure if diagnosis is made early.

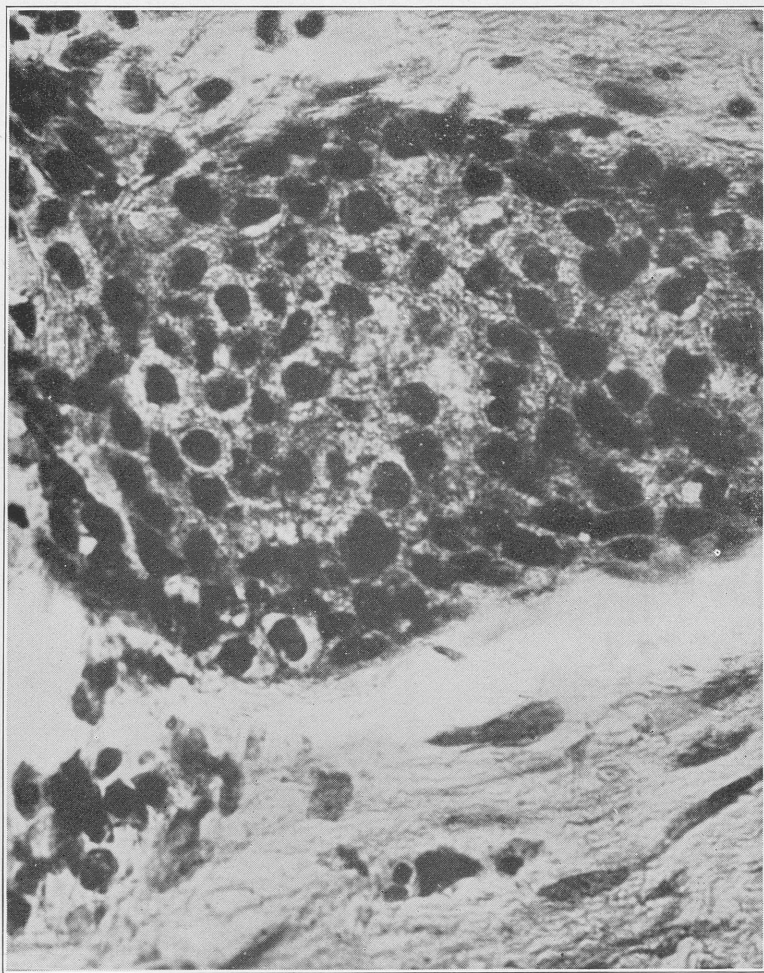


FIG. 1 *a*.—Same section shown in Fig. 1, high power.

*Subglottic.* Subglottic cancer originates on the inner or under surface of the vocal cords or subglottic area, chiefly in the anterior half of this region.

In the subglottic type, of which in this series we have 15, although well within the larynx, the growth has large lymphatic drainage with possible early metastasis along the party wall, as the esophagus and trachea. For this type I prefer a total laryngec-

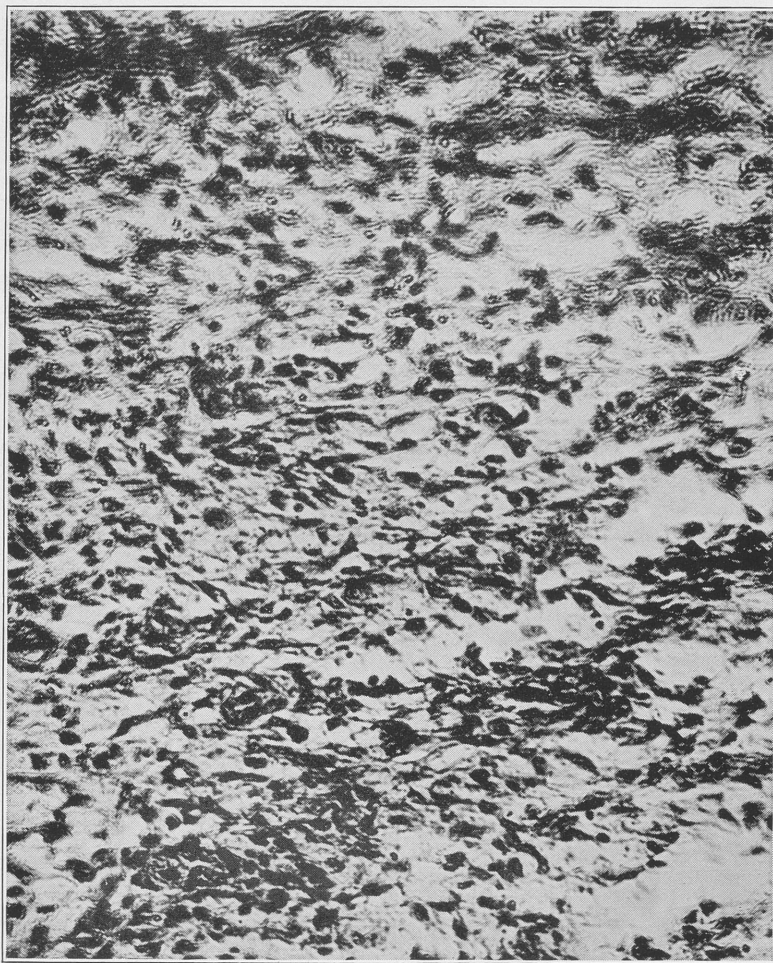


FIG. 2.—Myxosarcoma of larynx; low power. The tumor is quite vascular. It shows mucoid connective tissue areas, areas of large spindle cells, many showing mitotic figures



tomy to a laryngofissure, for in our series (Table 5) we had 7 cases in which the site of growth at operation was subglottic; and these 7 all had a recurrence in the glands of the neck, or metastases to the brain and lungs, and possibly 1 case to the prostate.

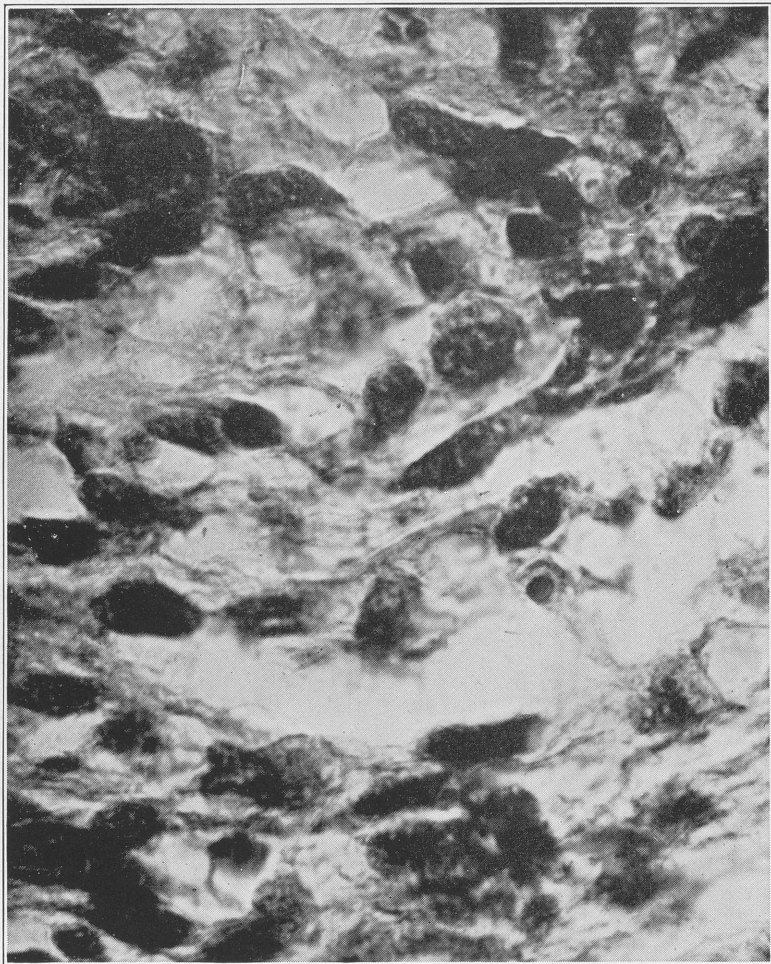


FIG. 2 *a*.—Same section shown in Fig. 2, high power.





FIG. 3.—Epidermoid carcinoma of larynx; low power. Grade I. Shows irregular papillæ covered by thickened stratified squamous epithelium, some dipping into the submucous tissue in the form of nests with central cornification.

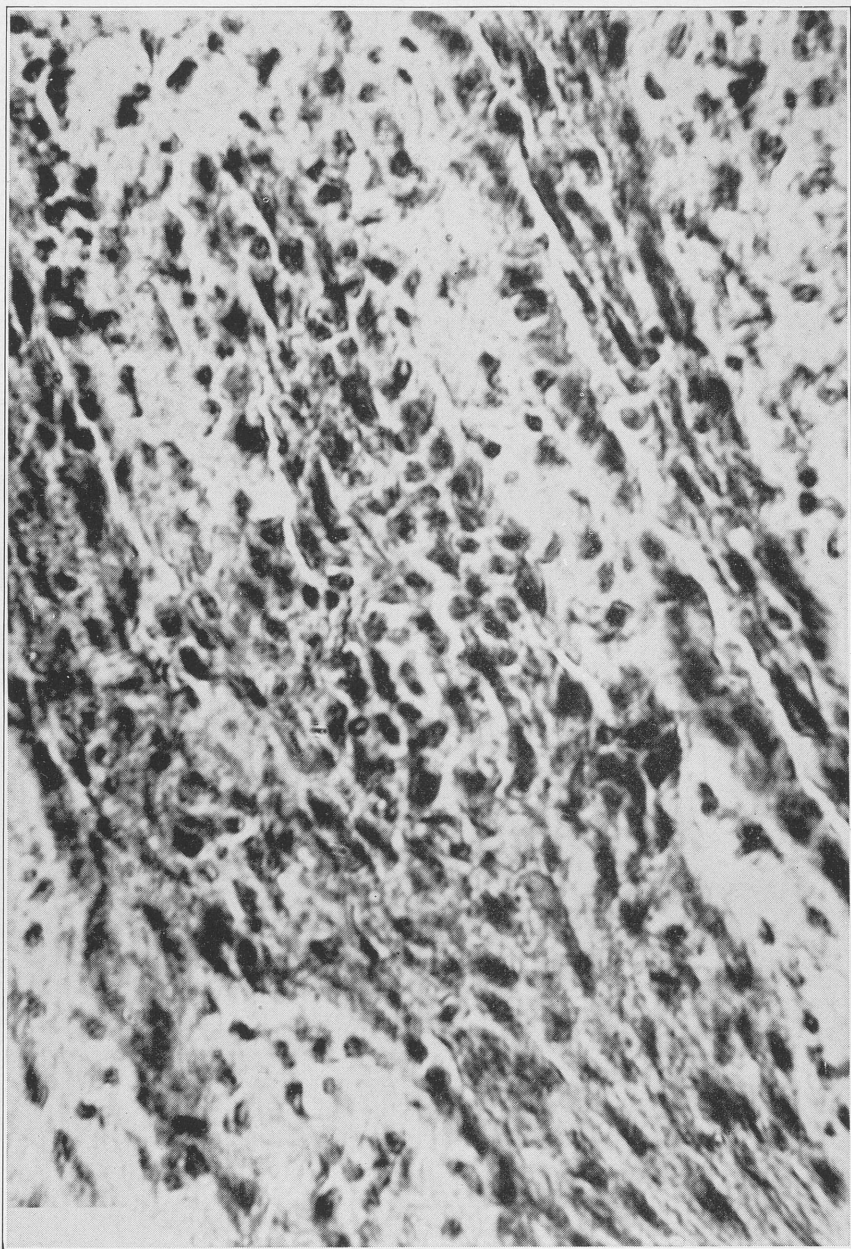


FIG. 3 *a*.—Same section shown in Fig. 3, high power.

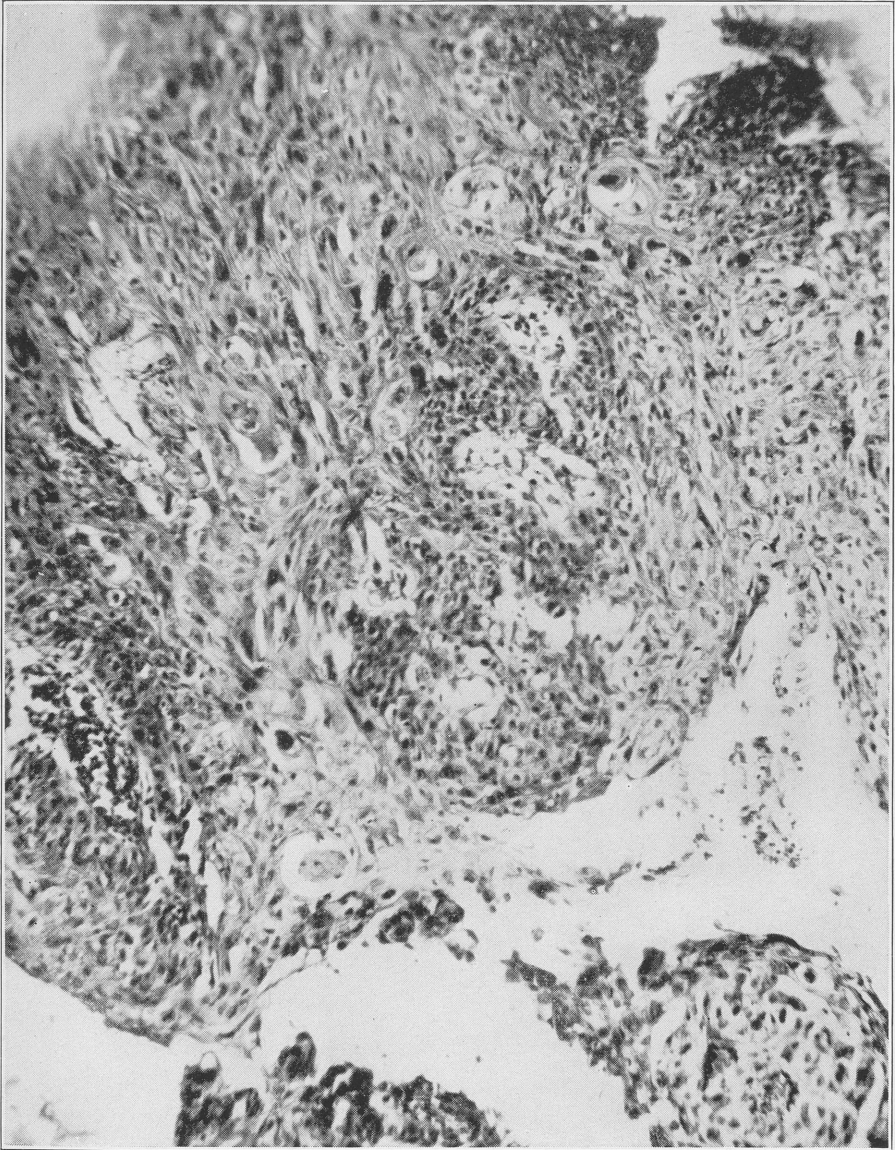


FIG. 4.—Epithelioma of larynx; low power. Grade II. The stroma is invaded by groups and strands of carcinomatous cells showing considerable hyperchromatism and a moderate number of mitotic forms. There is a moderate fibrous reaction in the stroma.



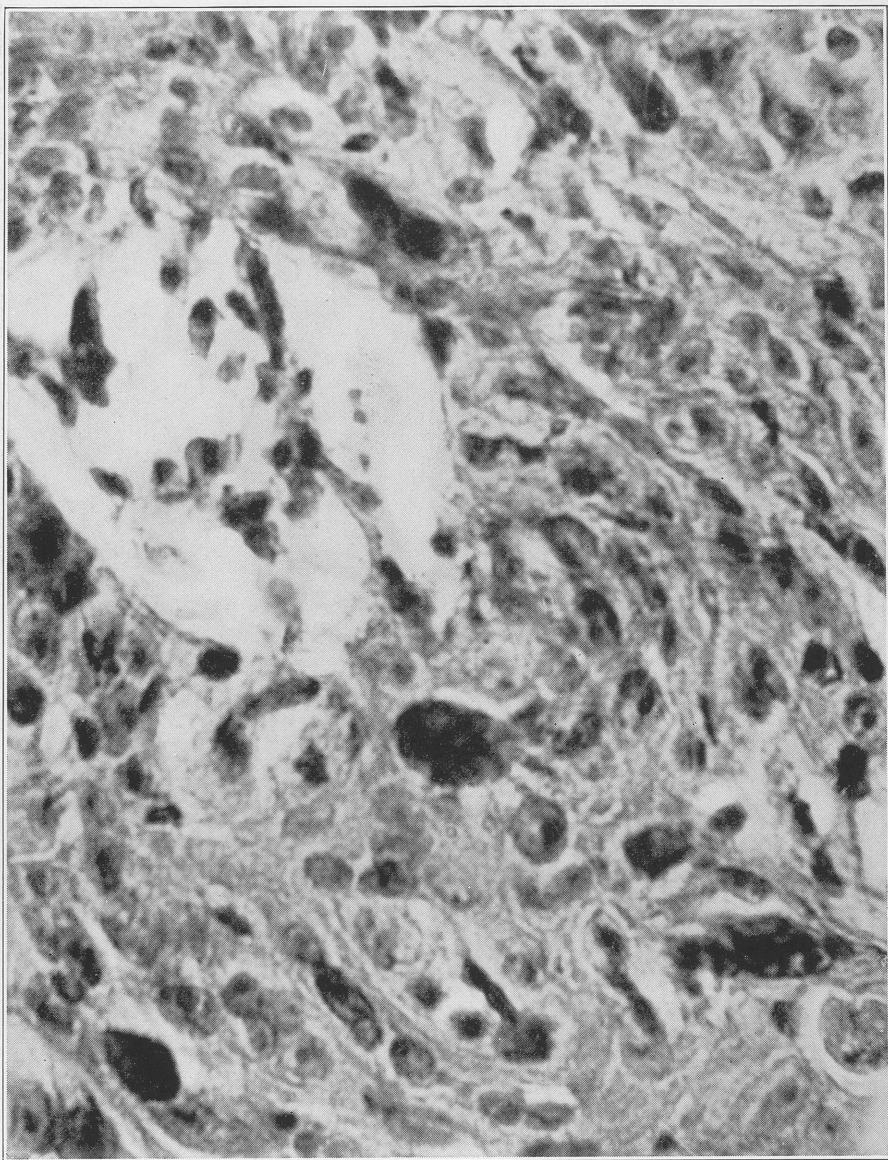


FIG. 4 *a*.—Same section shown in Fig. 4, high power.



It would seem inevitable that if we had a recurrence of these in total laryngectomy, we certainly would have had the same in laryngofissure. Therefore I reiterate that I prefer total laryngectomy to a laryngofissure in this type of cancer.

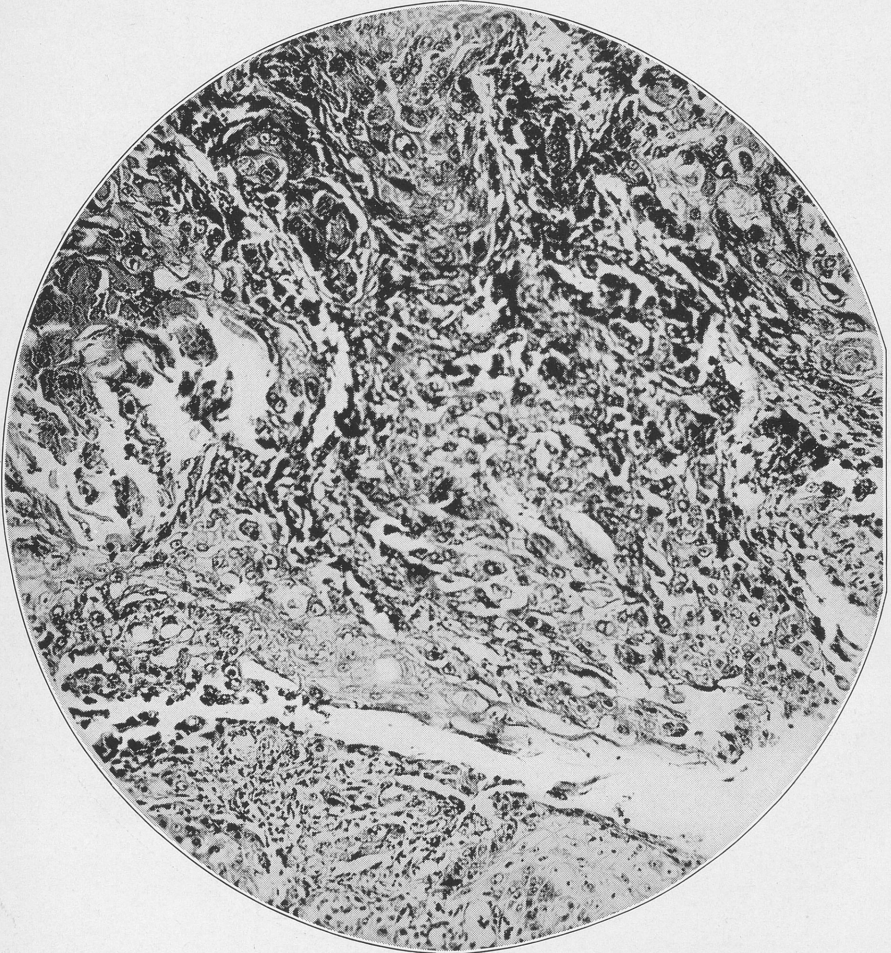


FIG. 5.—Epidermoid carcinoma of larynx; low power. Grade III. There is an invasive growth of groups of atypical pavement epithelial cells beneath the surface epithelium. The individual cells are larger than normal and show marked variation in staining. There are numerous epithelial pearls and a few mitotic figures present.

This subglottic type originates below the cords, usually in the anterior third and may involve the anterior commissure, spread to the opposite side and continue to spread upward to the vocal cords or downward to the cricoid cartilage.

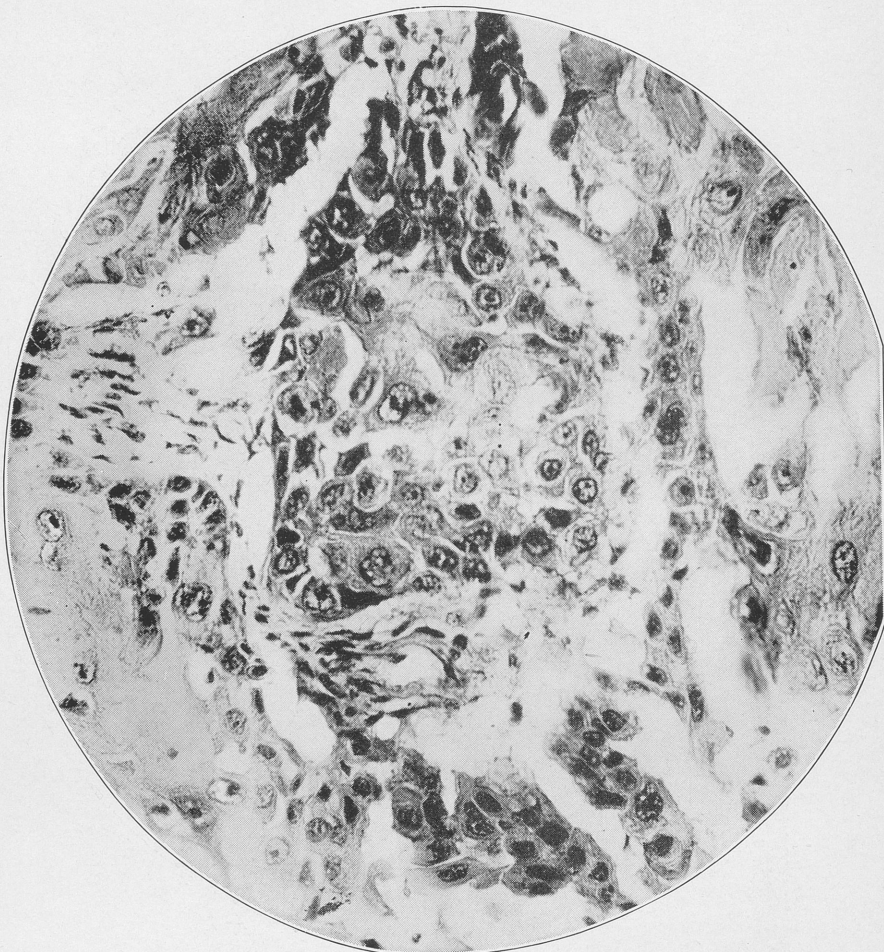


FIG. 5 *a*.—Same section shown in Fig. 5, high power.

Hoarseness is late in these cases, although sluggish movement of the cord was noticed. By direct and indirect examination of the larynx the lesion is usually demonstrated.

Dyspnea may be the first symptom, as in the case of one of my patients whose first and only symptom was dyspnea, for which he was referred to me. On laryngeal examination I could plainly

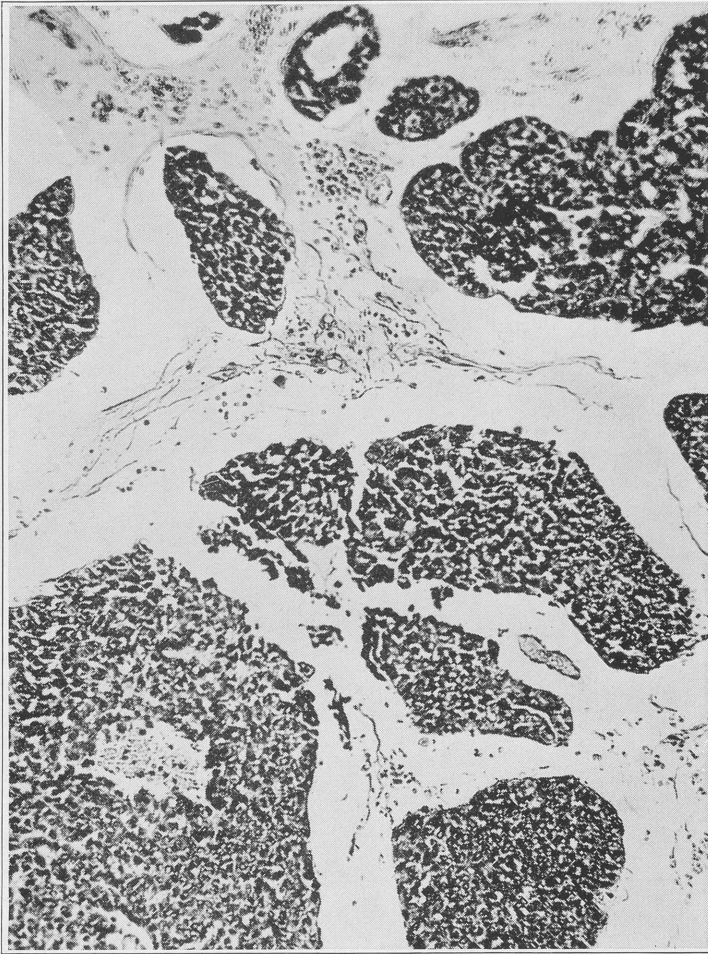


FIG. 6.—Undifferentiated carcinoma of larynx; low power. Grade IV. The surface epithelium is hypertrophic. In the fibrous stroma there is an extensive invasive growth of groups, cords and masses of undifferentiated epithelial cells which vary considerably in size, and there is some mitosis.



see a large growth protruding into the larynx, almost reaching the opposite side.

Dyspnea will remain the only symptom until such time as the growth has progressed to affect the mechanism of the larynx to

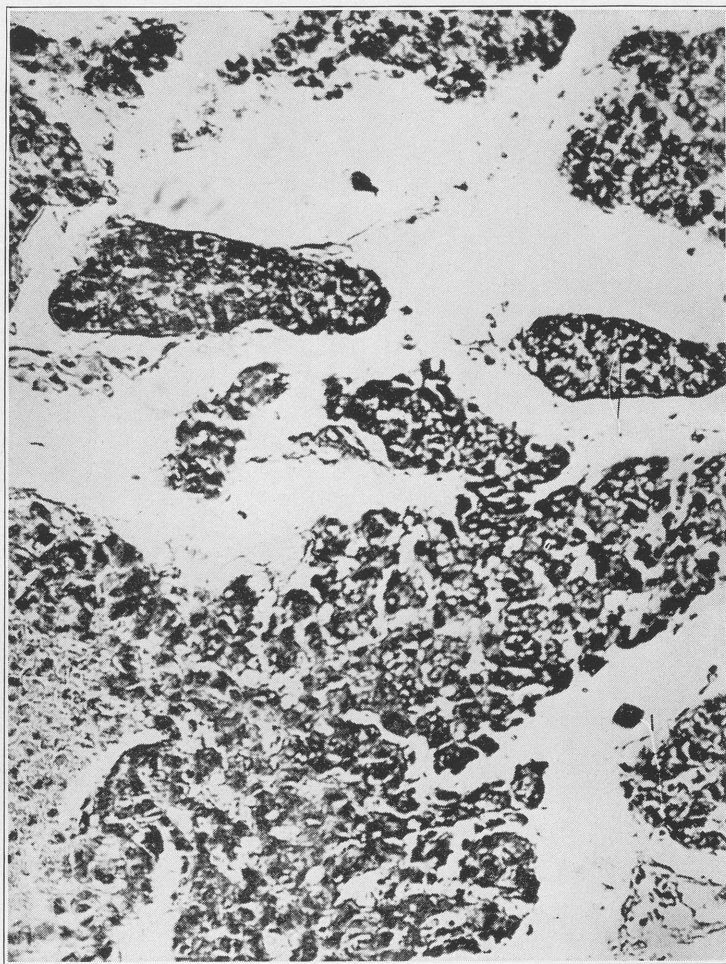


FIG. 6 a.—Same section shown in Fig. 6, high power.



produce hoarseness, or to the extent of causing stenosis necessitating a tracheotomy.

**SUMMARY.** Subglottic cancer may spread over a large area before recognition. Anteriorly they commonly pass through the crico-thyroid membrane and metastasize to the lymph nodes over this area. They often extend behind the isthmus of the thyroid gland and may extend down into the trachea. Posteriorly they may extend to the arytenoids involving the pharyngeal mucosa where rapid metastasis to the lymph nodes along the course of the internal jugular vein takes place. Superiorly they may extend into the supraglottic region invading the base of the epiglottis.

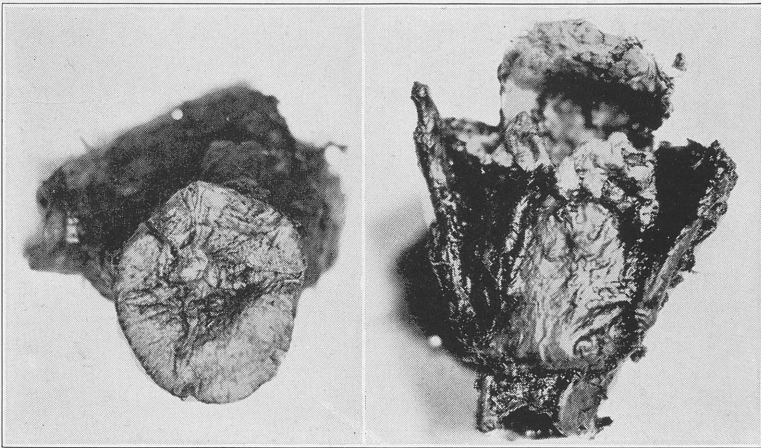


FIG. 7.—Epithelioma of larynx. Pachydermia and sinus extending to surface of skin. Laryngectomy, 1927. Patient still living.

Usually the lymph glands escape as long as the growth is confined to the interior of the larynx, or even to the epiglottis. This is due to poor lymphatic drainage and resistance of the cartilaginous walls.

Subglottic cancers are practically always of squamous cell type.

*Extrinsic Cancer.* The extrinsic cases are those which originate around the glottic opening or on its outer surfaces. These may again be subdivided into the epilaryngeal type, which includes neoplasms arising from the epiglottis, aryepiglottic folds, arytenoids

and pyriform sinus, of which we have 23 cases, and the hypopharyngeal type, which includes the postericoid cancer, of which we have 2 cases. These are more malignant than are the intrinsic and have early metastasis to the cervical glands.

Some pathologists state that this type is seldom arrested or cured by surgical procedure; although in this series we had 40 cases of extrinsic cancer, including the subglottic type, with 23 deaths, or approximately 42 per cent recoveries.

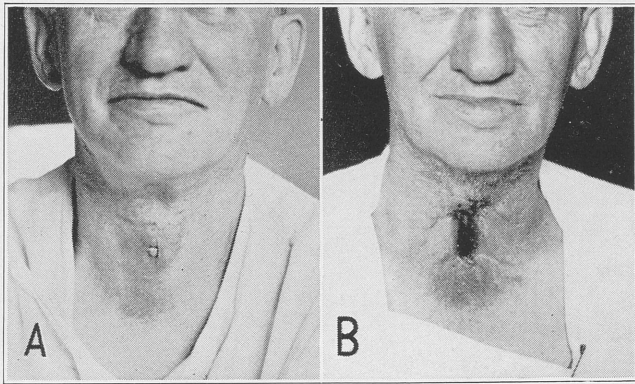


FIG. 8.—A. Picture of patient before operation. B. After operation in 1927. Patient alive and well at present time.

I believe that in the epilaryngeal and hypopharyngeal type of malignancy, if early diagnosis is made, very good results are obtained. In fact, too often when the patients present themselves for examination they are beyond any hope of a five year cure. All these types of cases should be diagnosed early for they are readily seen by direct or indirect examination.

The symptoms depend upon the site of the lesion, but with hoarseness, a tickling sensation, constant clearing of the throat, an abnormal sensation that is persistent in this region, we should awaken to the fact that we are dealing with a potential malignancy. Later, pain, localized or deferred, loss of appetite, difficulty in swallowing, increased salivation, and enlarged cervical glands usually denote a late and too often an inoperable state.

SUMMARY. These extrinsic cases are usually of a squamous cell type, although they may be papillary in character and occasionally we find a basal celled type.

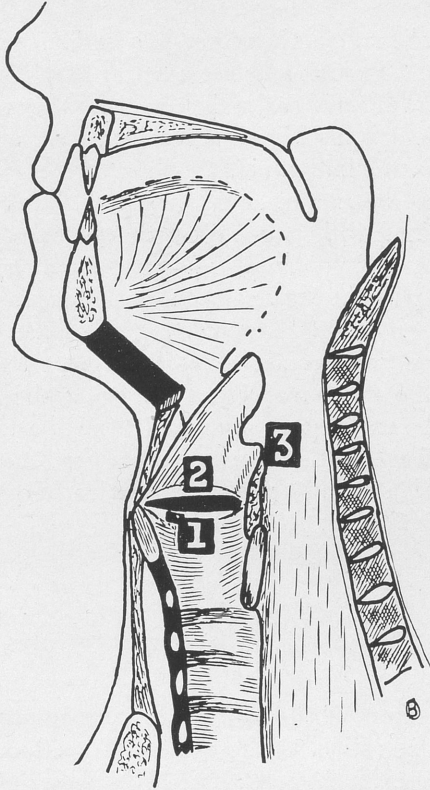


FIG. 9.—Intrinsic cancer. About 75 per cent of laryngeal cancer is intrinsic in origin. (1) Vocal cord favorite site. Intrinsic cancer usually cordal cancer. Spreads along cord, may become subglottic and spread to opposite cord or across commissure. (2) Ventricular band and ventricle. Ventricles—rare, more dangerous than cord; causes early glandular involvement. (3) Interarytenoid area rarely seen.

(1) Benign in early stages. Grows slowly. (2) If limited to vocal cords does not metastasize to glands. (3) Cancer usually squamous celled epithelioma. Occasionally basal-celled. Other forms rare. Adenocarcinoma seen in ventricles. (4) Must differentiate from singer's nodule, leukoplakia, innocent tumors, tuberculosis and syphilis. (5) Course usually several years, depending on location and grade of tumor. Even most malignant forms usually last over two years.

Epitheliomas originating on the epiglottis are relatively of a low grade malignancy until the growth has spread either to the base of the tongue or to the aryepiglottic fold. In other epilaryngeal situations, however, the lymphatic glands are invaded early and consistently.

Extrinsic malignancy, therefore, is a serious problem, and as Jackson stated, "To look and see is better than to wait and see."

The glands are affected early, the course is fairly rapid and unless prompt treatment is instituted, a year or a year and a half is the average span for the individual.

It is true that we cannot cure all cancers, but certainly if this region is examined with painstaking thoroughness, much can be accomplished by early diagnosis and radical surgery, for cosmetic surgery has no place in the treatment of malignancy.

**PATHOLOGY. Histology.** Included in this series we have 1 melanoma, 1 myxosarcoma, 4 basal cell type and 22 papillary epitheliomas; the most frequent type being the squamous cell carcinoma. The sarcomata originated from the right arytenoid.

Mackenty's Table in the *Transactions* of the American Laryngological Association in 1923 gives the relative frequency of cancer of the larynx as follows:

TABLE 1.		
Mackenty, per cent		This series, per cent
96	Squamous cell	75
2	Basal cell	3
1	Papillary carcinoma	22
1	Adenocarcinoma	2

Metastatic tumors of the larynx are exceedingly rare although inoculation metastasis in the respiratory tract has been reported by Hitz and Oesterlin in 1932. With this in mind we should be careful about disturbing a growth at the time of operation.

Turner, in 1924, described a case in which the first sign of a renal cancer was due to a metastasis in one vocal cord. In one of my cases the patient was operated on in 1937 for an intrinsic growth of a papillary type, Grade I, on his left vocal cord, dipping into the subglottic area. Within nine months the patient died from a very large inoperable cancer of the prostate. It is quite probable in this case that the original tumor might have been in his prostate with metastasis to his larynx.



We have in this series metastatic glands in the neck and to the tongue. These have usually been of the pyriform sinus type and also in the aryepiglottic folds. Also, metastasis in the esophagus



FIG. 10.—Subglottic cancer originates on inner or under surface of vocal cords or subglottic area, chiefly in anterior half of this region. (1) Intrinsic cancer of cord—becoming subglottic at anterior commissure. (2) Intrinsic cancer of cord—on inner or under surface but below free edge. Invasion of opposite cord by subglottic route or across commissure. (3) True subglottic cancer.

(1) May spread over large area before recognition. Anteriorly, commonly pass through crico-thyroid membrane and metastasize to lymph nodes; often extend behind isthmus of thyroid. May extend into trachea. (2) Posteriorly, may extend to arytenoids, invading pharyngeal mucosa; rapid metastasis to lymph nodes along course of internal jugular. (3) Superiorly, may extend into supraglottic region invading base of epiglottis. (4) Usually lymph glands escape if growth is confined to interior of larynx or epiglottis. This is due to poor lymphatic drainage and resistance of cartilaginous walls. (5) Practically always squamous celled epitheliomas.

through the party wall, has occurred in 1 patient. In another patient we had a metastasis to the brain and lung from an adenocarcinoma of the subglottic region two and a half years after operation.

Three patients had metastasis to the lung; 3 to the esophagus; 5 to the tongue; 15 to the glands of the neck.

The cases which had metastasis in the brain also had metastasis in the lung.

In the basal cell type there is apt to be very little tendency to recurrence. We had 4 basal cell type carcinomas. One man was seventy-two years of age at time of operation and he died of apoplexy twelve years and eleven months after operation. Another patient was operated on at the age of fifty years and he is still living twelve years and eight months following the operation.

In one of the cases that had adenocarcinoma the site of lesion was located in the subglottic region.

According to the classification of Broder, 73 cases reported by the pathologists are as follows: 27 cases Grade I; 26 cases Grade II; 16 cases Grade III; 4 cases Grade IV.

It is interesting to note that in the 4 cases of Grade IV, according to Broder's classification, 1 was an adenocarcinoma in which the patient died two and a half years later of metastasis in the brain and lung; another patient died of apoplexy two and a half years after operation without any recurrence (this growth was intrinsic, of a squamous cell type carcinoma); the third patient had squamous cell type epithelioma involving the right pyriform sinus, and from my records is still living; the fourth case was a squamous cell epithelioma (this patient died of a pneumonia six days following operation).

In other words, in Grade IV that is supposedly not amenable to surgery of the pyriform sinus type, we have 1 patient living six years after operation.

**SYMPTOMS.** The symptoms, of course, would vary according to the location of the lesion, whether it is intrinsic, epilaryngeal or hypopharyngeal.

In 89 cases we obtained a history of hoarseness from six weeks' to fifteen years' duration. Hoarseness is caused by some interference in the vibration, approximation, or tension of the vocal

cord. This would seem to make us view with suspicion any hoarseness in an adult extending over a period of a few weeks, and very careful examination of the larynx should be made to ascertain the cause.

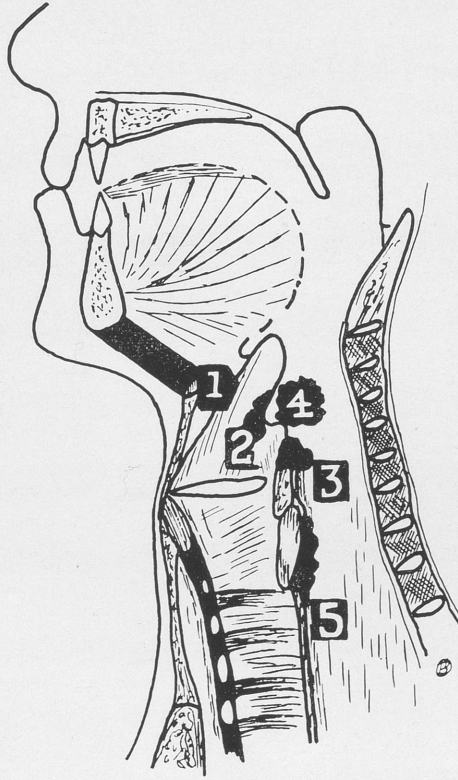


FIG. 11.—Extrinsic cancer. In this group are included neoplasms growing from the epiglottis, aryepiglottic folds, arytenoids, the pyriform sinuses and pharyngeal surface of the cricoid cartilage (post-cricoid cancer). (1) Margins of epiglottis. (2) Aryepiglottic folds. (3) Arytenoids. (4) Pyriform sinuses. (5) Pharyngeal surface of cricoid cartilage (post-cricoid cancer).

(1) Extrinsic cancers are usually squamous type, may be papillary in character. Occasionally we find basal-celled type. (2) Cancer of epiglottis relatively low grade, unless growth has spread to base of tongue or aryepiglottic fold. (3) In other situations lymphatic glands are invaded early. (4) "To look and see is better than to wait and see."

Local discomfort that might be characterized as a constant desire to clear the throat, and a tickling sensation, to the extent of

producing cough, was noted in 59 cases. This was noticed more in the epilaryngeal or hypopharyngeal type of malignancy, which was probably due to involvement of the superior laryngeal nerve, permitting saliva or food to trickle into the larynx, producing cough.

As to symptoms, pain was next in frequency. Forty-five patients complained of pain that was referred to the larynx, side of the neck, or to either ear.

Dyspnea, on moderate exertion, was complained of by 29 patients. Seventeen patients gave symptoms of stridor and wheezing. Difficulty in swallowing and dysphagia was complained of by 25 patients. Fetor, due to ulceration, was noted by 9 patients. Increased salivation was complained of by 15 patients; these were mostly of the epilaryngeal or hypopharyngeal type of lesion. Blood-streaked sputum or saliva was noticed by 5 of the patients.

An interesting observation was that 39 patients had lost weight. Usually loss of weight is considered a late sign, but in this series we have about one-third of the patients who had, on my first examination, stated that they had lost weight.

PHYSICAL SIGNS. Alteration of voice was observed in one way or other in 89 patients operated upon. It would seem, therefore, that any alteration of voice extending over a considerable length of time should be regarded seriously, and every effort made to determine the cause.

The movements of the vocal cords were noted and the following table shows the results:

TABLE 2			
Movements of cords:	Free	.	30
Cord sluggish:	Right	.	25
	Left	.	14
Cord fixed:	Right	.	19
	Left	.	14

This shows that the vocal cords do not need to be sluggish in their movement or even fixed for a larynx to harbor cancer; so if we wait for a cord to become sluggish we have lost valuable time for early diagnosis.

Why the right cord should be more affected than the left, in this series, I am not prepared to state.



Local discomfort in the larynx or throat for some time was noted in 59 cases.

Dyspnea was noticed in 27 cases, this ranging from slight dyspnea with the slightest exertion, to almost urgent dyspnea, necessitating a preoperative tracheotomy in 10 cases. These naturally occurred in the subglottic type. Stridor accompanied dyspnea in 17 cases; this being due to the narrowing of the glottic chink by the growth.

Choking with meals or on swallowing saliva was noted in 26 of the cases. This may be due to some involvement of the posterior branch of the inferior laryngeal or recurrent laryngeal nerve fila-

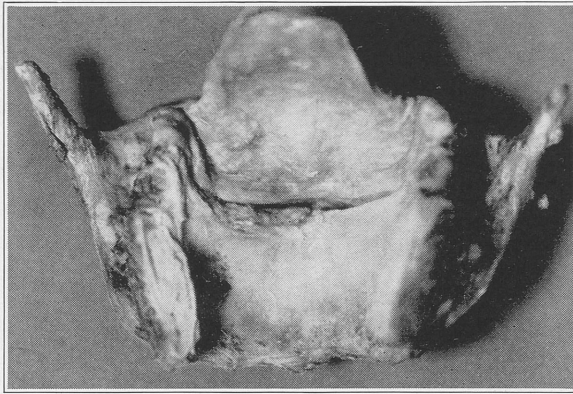


FIG. 12.—Epithelioma of larynx. Growth on left vocal cord crossing below anterior commissure and invading opposite cord. Laryngectomy.

ments causing a slight anesthesia of the larynx, or to food or saliva being directed to the larynx by deformity of its structure due to growth. This type was of course more noticeable in the extrinsic cases.

*Glands.* Cervical enlargement of the glands, either from infection or metastatic extension, was observed in 15 cases. This also was noticed in the more advanced cases; for in true cordal cancer lymphatic invasion is very rare. The same applies to mucus in the pyriform sinus which the patient cannot empty on swallowing, namely, that it accompanies extrinsic type of cancer. This was observed in 21 of the cases.

The sign of a cushion behind the larynx, preventing the feeling of the larynx grating on the cervical vertebræ, was noticed in

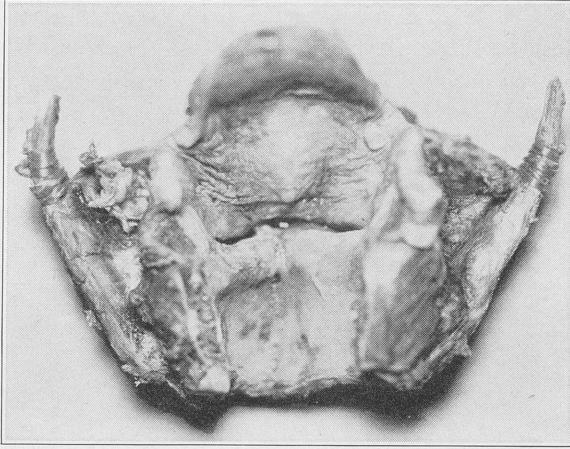


FIG. 13.—Epithelioma of larynx. Growth involving both cords. Laryngectomy.

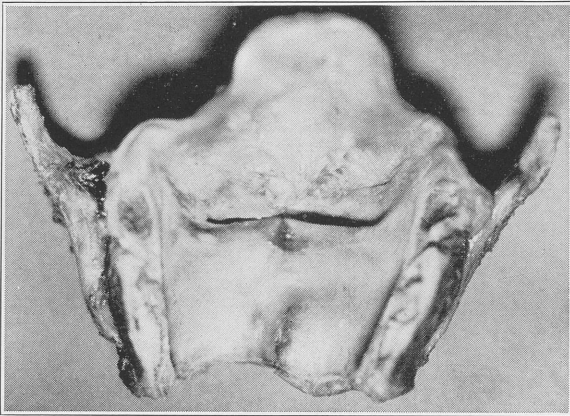


FIG. 14.—Epithelioma of larynx. Growth of ventricle spreading to epiglottis. Laryngectomy.

3 cases. This is usually the postcricoid type, but is also found in cancer of the posterior wall of the esophagus, which type is not included in this series.

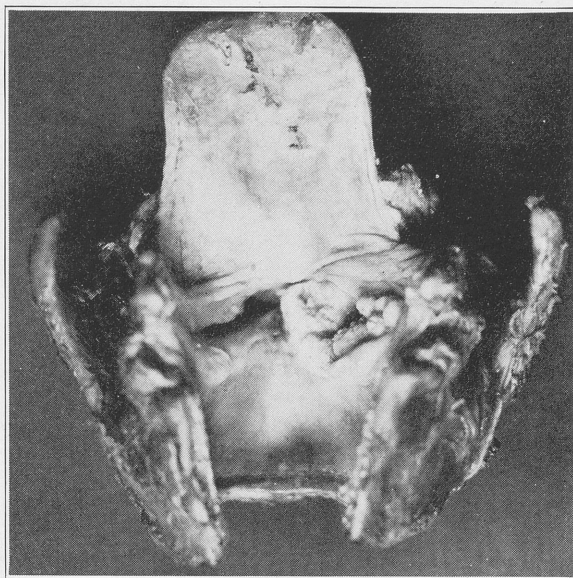


FIG. 15.—Epithelioma of larynx. Growth of right cord and ventricle. Laryngectomy.

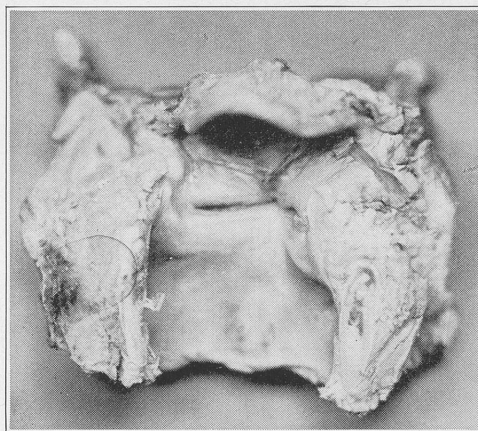


FIG. 16.—Myxosarcoma of larynx. Growth from right arytenoid invading pyriform sinus. Laryngectomy.



On examination we found 20 patients who gave the appearance of projecting papillomatous growth, these affecting the voice more than the superficial ulcer type, in which group there were 47.

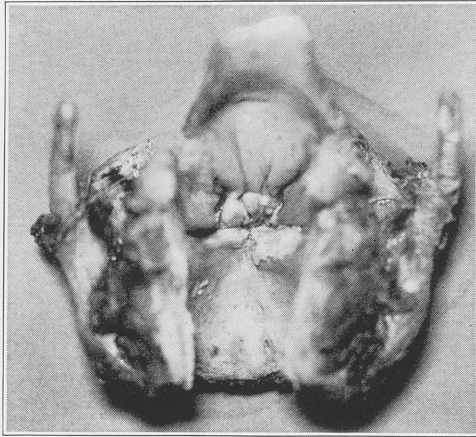


FIG. 17.—Epithelioma of larynx. Growth from right cord invading epiglottis Laryngectomy.

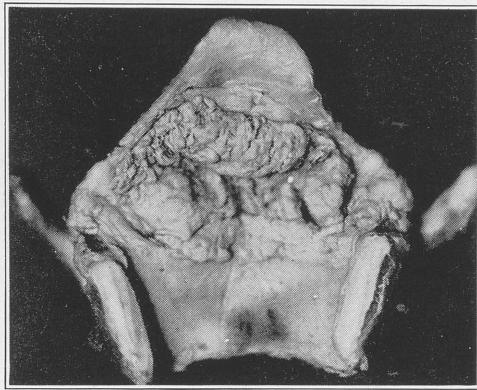


FIG. 18.—Epithelioma of larynx. Growth of the epiglottis, female. Laryngectomy.

The submucous or imbedded type of growth was noticed in 25 cases.



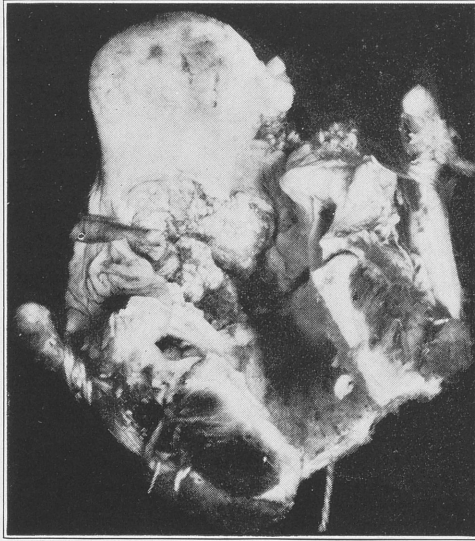


FIG. 19.—Epithelioma of larynx. Growth involving ventricle and epiglottis. Laryngectomy.

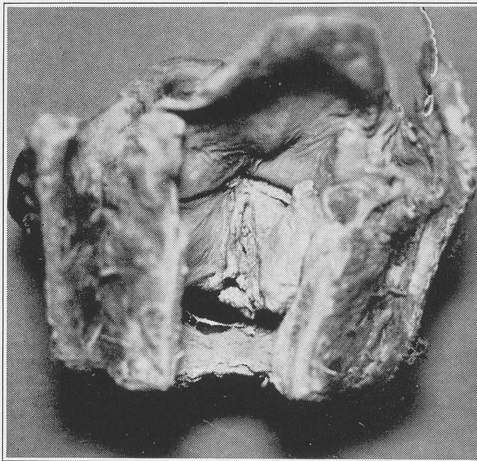


FIG. 20.—Epithelioma of larynx. Subglottic growth right side extending upwards. Laryngectomy.

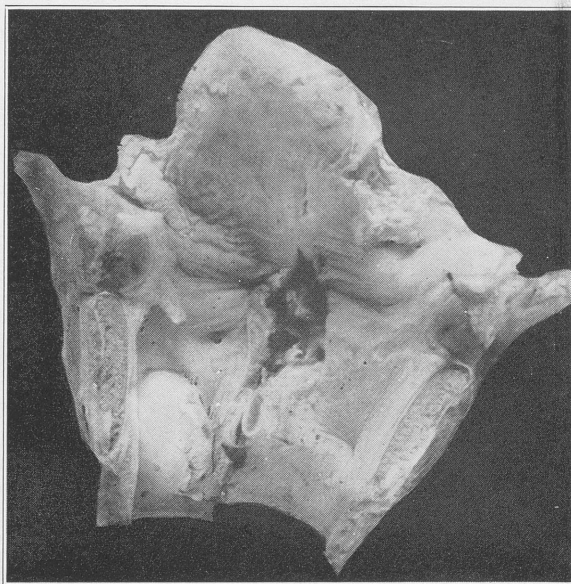


FIG. 21.—Epithelioma of larynx. True subglottic growth on left side. Laryngectomy. Metastasis to brain and lung two and one-half years later.

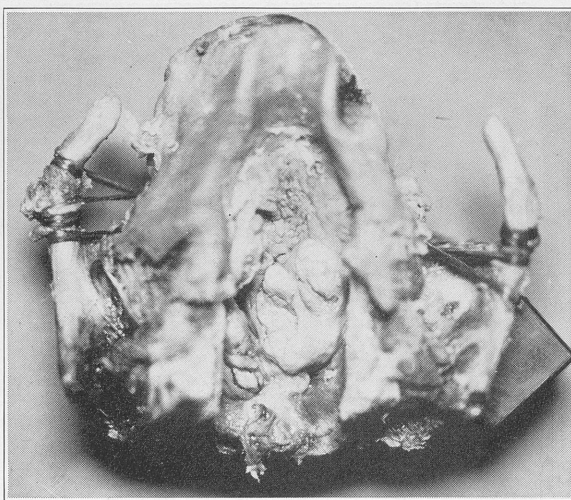


FIG. 22.—Epithelioma of larynx. Growth involving both cords and subglottic area. Laryngectomy.

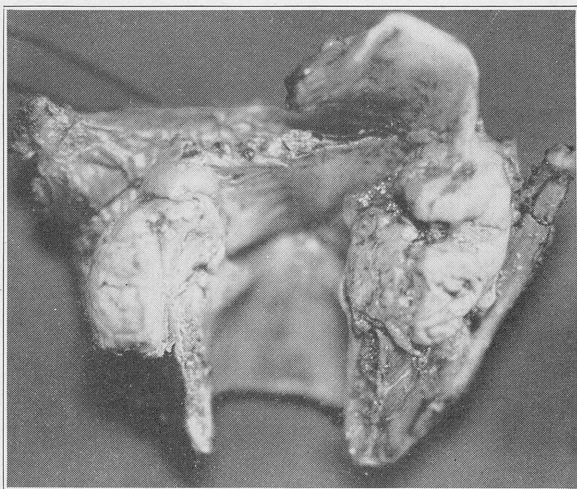


FIG. 23.—Epithelioma of larynx. Growth involving both arytenoids with infiltration of cords and right pyriform sinus. Grade III. Laryngectomy, 1934. Living.

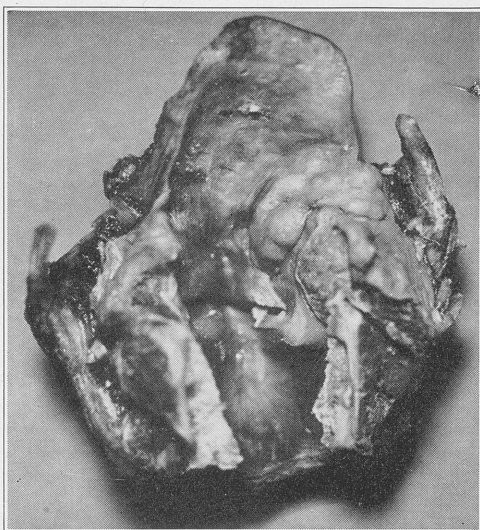


FIG. 24.—Epithelioma of larynx. Growth involving right aryepiglottic fold. Laryngectomy.



DIAGNOSIS AND PROGNOSIS. Diagnosis depends on an accurate history, physical signs already alluded to, proper laryngological examination, and biopsy with histological study. Consideration of these factors makes possible a correct diagnosis.

As this series shows, there were 8 patients out of 102 that presented themselves for examination early enough to do a laryngofissure operation according to my interpretation of a true cordal cancer. In the other 94 patients the disease had progressed to such an extent that in my judgment a total laryngectomy was in order.

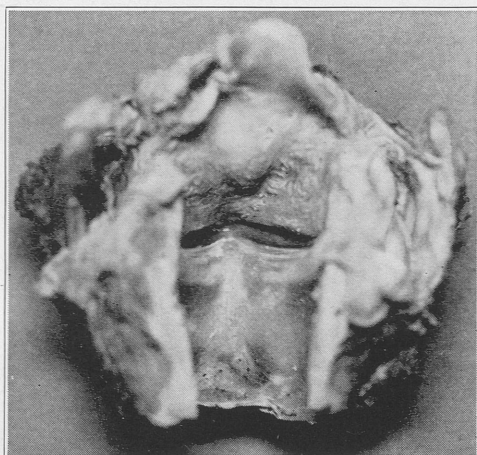


FIG. 25.—Epithelioma of larynx. Growth in right pyriform sinus showing ulcerating area. Laryngectomy.

Syphilis and tuberculosis are ruled out by proper tests and roentgen-ray studies of the chest, remembering however, that syphilis, tuberculosis and cancer may all be in the same larynx. Tuberculosis and cancer occurred in one of my cases. (Fig. 29.)

Biopsy was taken on all patients, and in so doing the histological examination rules out the various other innocent tumors, namely, laryngeal varix, fibromas, etc.

A patient with cancer talks with a strong rough voice, usually without pain. The opposite characteristics we note in tuberculosis.



Hoarseness varies from an intermittent to continuous type, usually better in the morning after resting.

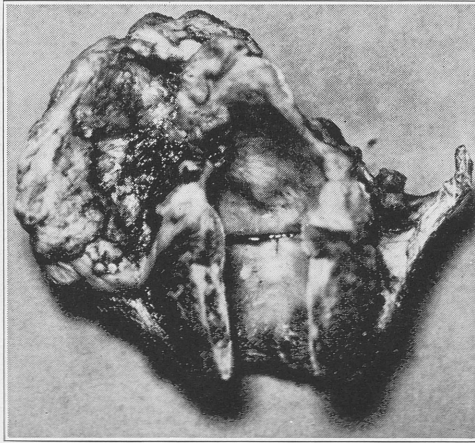


FIG. 26.—Epithelioma of larynx. Growth involving left aryepiglottic fold to base of tongue. Laryngo-pharyngectomy. No recurrence after two and one-half years.

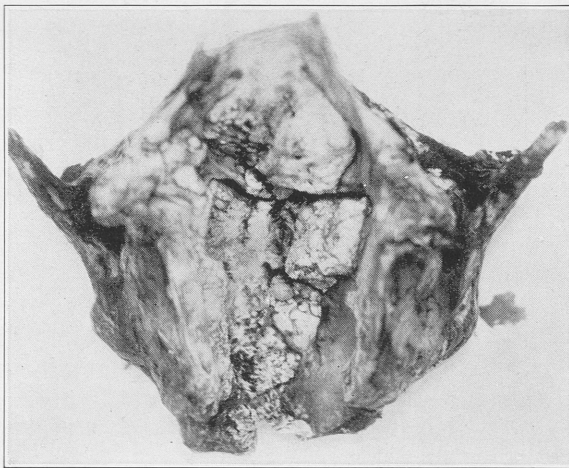


FIG. 27.—Epithelioma of larynx. Growth involving epiglottis mostly on right side, subglottic area on same side. Laryngectomy

Cough is also present, ranging from slight tickling sensation to constant irritating cough.

Difficulty in swallowing with or without food, dyspnea which has increased and which may be followed by stridor and wheezing, increased salivation, and mucus in the pyriform sinus which cannot be emptied, spell trouble, with additional signs, such as saliva blood-tinged up to a slight hemorrhage and loss of weight. Thirty-eight patients gave a history of loss of weight.

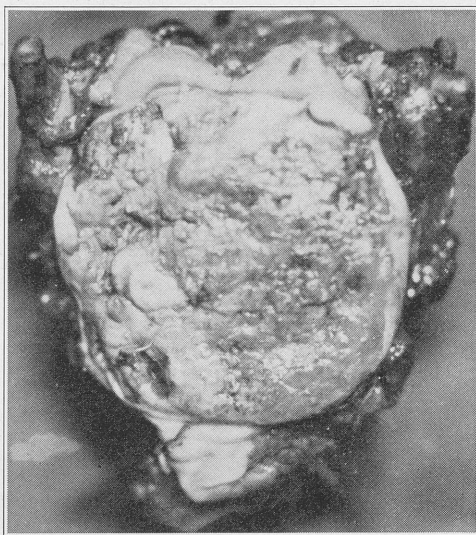


FIG. 28.—Epithelioma of larynx. Post-cricoid or hypopharyngeal. Laryngopharyngectomy. Recurrence two years later.

Palpation of the neck and finding unilateral or bilateral glands, plus the laryngological examination with proper illumination, ascertaining the type of growth, as whether it is projecting into lumen of the larynx, or a superficial ulcer, an imbedded tumor or a combination of these, usually indicates unilateral involvement, although if seen late this may have become bilateral.

The mobility of the cords is noted, as to whether they are free or sluggish in action or fixed, and with the physical signs already mentioned as dependent upon the site of lesion. Last, but not least,

a direct laryngoscopy is done and a small piece of tissue removed by means of biting forceps. Then with histological examination of the specimen removed, we will know the type and grade of malignancy, also whether it is radio-resistant or radio-sensitive.

*Prognosis.* The prognosis varies according to the location of the lesion.

In true cordal cancer with operation, 80 to 85 per cent cures are obtainable.

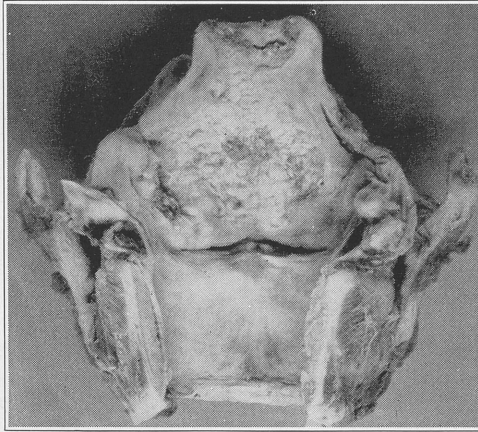


FIG. 29.—Epithelioma of larynx. Growth on right cord with tuberculosis of the epiglottis.

In the subglottic type of intrinsic cancer, the results of possible cure are lessened according to the distance of the growth from the edge of the vocal cords. In other words, the cords are like timber-line, nothing taking root above, but as you come into the valley, vegetation is abundant; so the farther away from the cords the greater is the growth and lymphatic extension.

In the epilaryngeal type of cancer, which comprises cancer of the epiglottis, aryepiglottic fold, arytenoid, pyriform sinus and hypopharyngeal or postericoid, the prognosis is best in the order named.

**TREATMENT.** In this series of cases of true cordal cancer, the treatment consisted of laryngofissure. (See chart of laryngofissures.)

In the subglottic type, as well as all extrinsic types, the treatment was by total laryngectomy, laryngo-pharyngectomy and one lateral trans-thyroid pharyngotomy.

*Preoperative Preparation.* The patient is admitted to the hospital four or five days prior to the date of operation. During this time metabolic rate, blood chemistry and roentgenograms are made. The internist examines the patient as to his cardio-vascular system and his opinion is given as to operative risk.

1. Careful attention is given to the cleansing of the mouth with tooth-brush and 25 per cent alcohol mouth-wash before and after feeding.

2. The colon is cleansed by a laxative and a high enema of pure bicarbonate of soda, drams 1, to 2 quarts of water; this is followed by colonic irrigations of salt solution through a large colon tube, using 2 or 3 gallons.

3. Every other day a modified irrigation using 1 gallon of salt solution is given.

4. Bicarbonate of soda, drams 3, in twenty-four hours, is given by mouth, between meals, for three days preceding operation.

5. The diet is nutritious, of a low-protein content; no eggs, milk or beef. Well cooked cereals with cream, chicken, thick soups, spaghetti, rice, bread and butter, vegetables, fruits and juices are given.

6. Twelve hours starvation; water up to within three hours of operation.

7. Three hours before operation, the neck is shaved well back, also the upper part of the chest, using green soap on cotton (no brush), sterile water, then alcohol. No dressing applied.

8. Bladder is emptied at the last moment.

*Operation of Laryngectomy.* Avertin-ether anesthesia is used. A "T" incision is made. The transverse incision is on a level with the hyoid bone, extending from one anterior border of the sternocleido-mastoid muscle to the other. The vertical incision is made from the center of the hyoid bone to the suprasternal notch. These flaps are dissected backward far enough to afford good exposure, the requisite procedure in attacking all cancers. All superficial veins are ligated. Then the superficial cervical fascia is divided in the median line. The sternohyoid muscles are



separated on both sides, severed from the hyoid bone, and reflected laterally. The thyrohyoid and sternothyroid muscles are then removed. At this point if the isthmus of the thyroid gland is high, it is divided and ligated, and the gland separated from the trachea at its upper margin. In doing this the cricothyroid arteries are ligated. The superior laryngeal nerve, artery and vein are exposed and ligated with three ligatures, dividing the vessels between two of them so that the upper ligated end of the vessels have two liga-



FIG. 30.—Differential diagnosis—acute edema of larynx. Postmortem specimen.

tures. The larynx is then rotated so as to bring the posterior border of the thyroid cartilage into view. The constrictor muscles are cut along this border. The same procedure is carried out on the opposite side.

The larynx is now free, excepting the posterior wall, where it is in relation to the esophagus.

The trachea is severed just below the cricoid cartilage. A small gauze pack is inserted into the larynx to prevent leakage from the

mouth into the wound. The tissue above the severed trachea is packed with gauze, preventing blood from entering the trachea. An anchor suture is placed in the trachea for future use. The Tucker anesthesia tube is placed in the trachea and surrounded

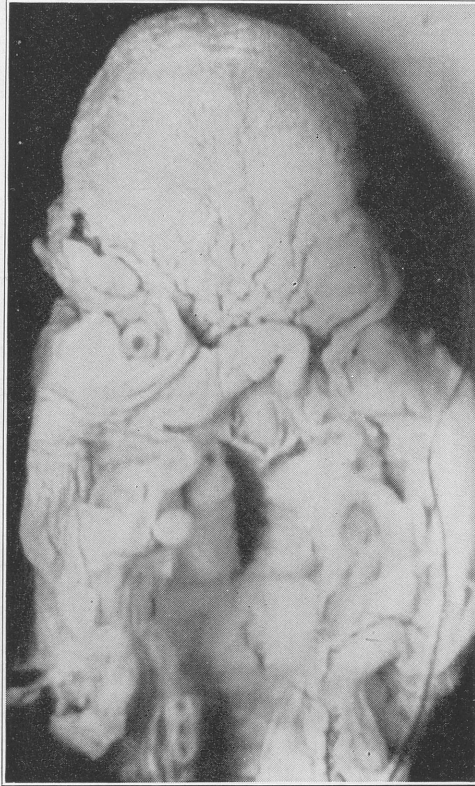


FIG. 31.—Syphilis of larynx. Differential diagnosis; showing contractions due to ulcerations. Postmortem specimen.

firmly with gauze packing, and the anesthesia is then continued through the trachea. The larynx is dissected from the pharynx. The pharynx is then opened and a large pack is inserted into the pharynx which is withdrawn through the mouth after operation. The larynx is removed; the pharyngeal opening is closed with

interrupted thread sutures, and just before the last suture is tied, the packing is removed from the mouth. Nasal feeding tube is passed through one nostril and guided down the esophagus. The last suture is then tied. Interrupted catgut sutures are placed in the esophagus. The third layer of sutures is in the constrictor

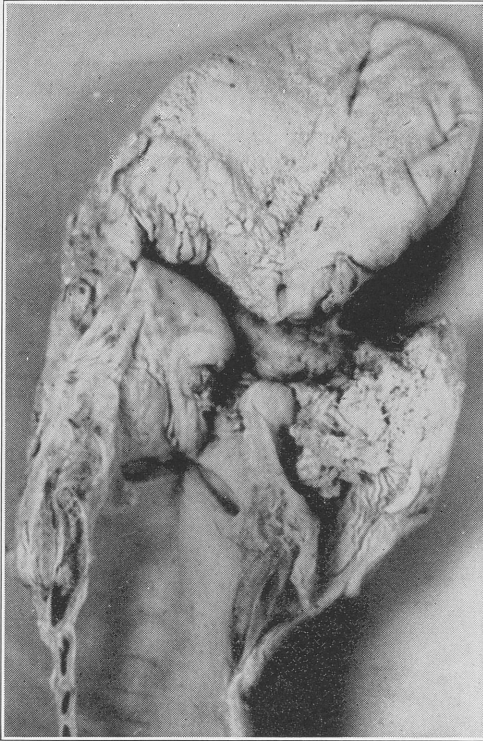


FIG. 32.—Postmortem specimen. Cancer involving right pyriform sinus, epiglottis, base of tongue.

muscle of the pharynx. Attention is then directed to the hyoid bone, which is removed by careful dissection; this allows a better closure. The trachea is anchored to the skin by means of silkworm gut suture to relieve tension. Black silk thread is used to suture skin to the trachea. The sternohyoid muscle is now brought to



the area behind the trachea so that we have four lines of sutures over the pharyngeal openings. Counter drains are placed on both sides of the neck and across the neck at the top of the "T." The skin is brought together with skin clips.

The laryngectomy tube is made to fit tightly in the trachea by winding narrow tape around the upper end. Dressings are applied, the patient is removed to bed, and the postoperative treatment instituted.

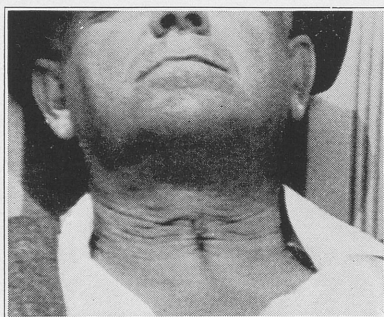


FIG. 33.—Patient was operated on by laryngofissure, June 24, 1927, and is the only one in our series of laryngofissures who gave a history of heredity. Patient is alive and well at the present time, eleven years after operation.

*Postoperative Care.* The gown is changed before leaving the operating room.

The bed has been warmed and the hot-water bag removed before patient is put into bed; 500 cc. 5 per cent glucose, are given intravenously.

A bed suction pump is installed alongside of patient's bed and kept there.

A necessary laryngectomy tray with duplicate tube is always in readiness.

Patient's position is changed frequently. Saliva from the mouth is removed by suction. Twenty-five per cent alcohol mouth-wash is used. Outer dressings are changed as often as soiled.

No morphine is given. Patient is out of bed the next day. Diet is of a high-caloric value.

*Special nursing.*

*Complications.* For the immediate complications following operation see Table 3, in which it is noted that 3 of these patients were among the first 11 operated upon.



TABLE 3.—DEATH FROM ANY CAUSE WITHIN THREE WEEKS OF OPERATION

Case No.	Age at operation, years	No. of days following operation	Cause of death
3 . . . . .	70	2	Diabetes; chronic nephritis
6 . . . . .	54	25	Abscess and gangrene of lung
11 . . . . .	75	5	Pneumonia
49 . . . . .	64	6	Pneumonia; abscess of lung
77 . . . . .	28	21	Meningitis; septic pneumonia, 4 plus

TABLE 4.—DEATH FROM OTHER CAUSES

Case No.	Age at operation, years	Age at death, years	Period after operation	Cause of death
4	72	85	12 yrs., 11 mos.	Apoplexy
17	48	53	5 yrs.	Struck by train
23	63	65	2 yrs., 6 mos.	Apoplexy
26	38	40	2 yrs., 3 mos.	Sudden death by accident
33	76	82	6 yrs.	Coronary thrombosis
41	60	60	2 mos.	Tuberculosis
45	62	66	4 yrs., 2 mos.	Apoplexy
48	49	49	4 mos.	Hemorrhage
86	71	71	1 mo.	Gastric ulcer perforation

TABLE 5.—DEATHS FROM MALIGNANCY ELSEWHERE THAN LARYNX

Case	Age at operation, yrs.	Time of onset after operation	Site of growth at operation	Age at death, yrs.	Site of recurrence
1	50	6 mos.	Subglottic	50	Glands
2	63	13 mos.	Pyriform sinus	64	Glands
8	66	8 mos.	Pyriform sinus	66	Glands
9	50	1 yr.	Epiglottis	51	Tongue
10	60	1 yr., 6 mos.	Arytenoid, aryepiglottic fold	62	Glands
13	60	2 yrs.	Ventricle	62	Esophagus
14	55	2 yrs.	Posterioricoid	57	Esophagus
16	74	9 mos.	Pyriform sinus	75	Esophagus
20	37	2 yrs., 6 mos.	Trachea	40	Brain and lung
29	63	1 yr.	Pyriform sinus	64	Neck
37	60	5 mos.	Aryepiglottic fold	60	Glands
38	67	8 mos.	Pyriform sinus	68	Glands
40	69	2 yrs., 6 mos.	Cord—ventricle	72	Glands
42	73	1 yr.	Epiglottis, ventricle, pyriform sinus	74	Glands
43	48	1 yr.	Pyriform sinus	49	Glands
51	44	1 yr., 5 mos.	Pyriform sinus	45	Glands
52	45	2 yrs.	Esophagus	47	Glands
53	65	2 yrs., 6 mos.	Subglottic	68	Tongue and glands
54	73	1 yr., 4 mos.	Subglottic	74	Lung
56	69	1 yr.	Subglottic	70	Brain and lung
70	53	9 mos.	Aryepiglottic fold, pyriform sinus	54	Tongue
78	40	1 yr., 3 mos.	Subglottic	41	Glands
87	49	6 mos.	Left cord, extending to subglottic	49	Prostate?

Later complications: Hiccough became a very distressing complication in 3 cases. Medication did not seem to have any effect in controlling the spasm. The only beneficial result was from partially withdrawing the feeding tube from the esophagus and then reinserting it to half the distance. In this series we have had very little postoperative vomiting, but in these 3 cases vomiting started early. Therefore, the presumption is that possibly the lower end of the feeding tube became curved upon itself in the esophagus.



Fig. 34.—Photograph of laryngectomized patients attending speech class, taken two weeks ago.

Hemorrhage: Two of the patients had severe hemorrhage. One, a male patient, three weeks following the operation, while sitting in a chair playing cards with his nurse, suddenly had a severe hemorrhage. The other patient, a female, with a hypertension also, had a severe hemorrhage two weeks following the operation. In both instances bleeding was from the superior thyroid artery. The value of specially trained laryngectomy nurses is hereby emphasized, for they were able to control the hemorrhage by pressure until the wound was opened and bleeding-points ligated. Both of these patients are alive and well at the present time.

Erysipelas: One patient developed erysipelas two weeks following the operation, from which he made an uneventful recovery only to succumb five months later from a recurrence of the malignancy.

TABLE 6.—SHOWING PATIENTS ALIVE AND WELL

Case No.	Age at time of operation, yrs.	Sex.	Present age, yrs.	Period since operation
5	50	Male	63	12 yrs., 8 mos.
7	55	"	67	12 yrs., 3 mos.
12	50	"	61	10 yrs., 7 mos.
15	48	"	58	10 yrs.
18	66	"	76	9 yrs., 6 mos.
19	49	"	59	9 yrs., 7 mos.
21	43	"	51	8 yrs., 3 mos.
22	70	"	78	8 yrs., 5 mos.
24	41	"	49	8 yrs., 3 mos.
25	54	"	62	8 yrs., 3 mos.
27	54	"	62	7 yrs., 11 mos.
28	67	"	75	7 yrs., 10 mos.
30	60	"	67	6 yrs., 8 mos.
31	49	"	57	7 yrs., 5 mos.
32	52	"	60	8 yrs.
34	60	"	67	6 yrs., 6 mos.
35	46	"	53	6 yrs., 4 mos.
36	63	"	69	6 yrs., 3 mos.
39	75	"	81	5 yrs., 9 mos.
44	55	"	60	5 yrs., 3 mos.
46	49	"	54	5 yrs.
47	40	"	45	5 yrs.
50	47	"	52	4 yrs., 10 mos.
55	60	"	65	4 yrs., 3 mos.
57	52	"	56	4 yrs., 2 mos.
58	40	"	44	4 yrs.
59	58	"	62	3 yrs., 11 mos.
60	46	"	50	3 yrs., 10 mos.
61	29	"	33	3 yrs., 9 mos.
62	63	"	67	3 yrs., 6 mos.
63	36	Female	40	3 yrs., 6 mos.
64	45	Male	49	3 yrs., 5 mos.
65	37	Female	41	3 yrs., 5 mos.
66	52	Male	56	3 yrs., 5 mos.
67	48	"	51	3 yrs., 1 mo.
68	52	"	55	2 yrs., 8 mos.
69	56	Female	59	2 yrs., 8 mos.
71	61	Male	64	2 yrs., 6 mos.
72	63	"	65	2 yrs., 4 mos.
73	68	"	70	2 yrs., 3 mos.
74	49	Female	51	2 yrs., 3 mos.
75	53	Male	55	2 yrs., 1 mo.
76	39	"	41	2 yrs., 1 mo.
79	42	Female	43	1 yr., 7 mos.
80	67	Male	69	1 yr., 6 mos.
81	56	"	57	1 yr., 5 mos.
82	51	"	52	1 yr., 4 mos.
83	59	"	60	1 yr., 4 mos.
84	61	"	62	1 yr., 4 mos.
85	46	"	47	1 yr., 1 mo.
88	62	"	63	9 mos.
89	71	"	71	5 mos.
90	69	"	69	5 mos.
91	40	Female	40	4 mos.
92	61	Male	61	4 mos.
93	63	"	63	1 mo.
94	55	"	58	2 yrs., 8 mos.

LARYNGOFISSURE. The indication for which the operation of laryngofissure was done was where the growth was limited to one cord, freely movable, and with a history of short duration. In most of these cases the growth was so small that it was almost entirely removed by biopsy. These patients were operated on by laryngofissure with very gratifying results, as the tables under laryngofissure show.

It hardly seems necessary to describe in detail the operation of laryngofissure before this Association; all are familiar with the technique described by Sir St. Clair Thomson and Chevalier Jackson, which has been carried out in this series.

Figure 33: This patient, a minister, was operated on by laryngofissure eleven years ago and had carried on his activities until retirement last year. I saw the patient two weeks ago and he is looking remarkably well with no recurrence.

TABLE 7.—CASES OPERATED ON—8

Sex	No. of patients	Private patients	Hospital patients	Age
Male . . . . .	5	2	3	26-79
Female . . . . .	3	3	0	41-54

TABLE 8.—SHOWING AGE AT TIME OF OPERATION

	Age: 25 to 30	31 to 40	41 to 50	51 to 60	61 to 70	71 and over
Male . . . . .	2	0	0	1	1	1
Female . . . . .	0	0	2	1	0	0

TABLE 9.—RECURRENCE OF MALIGNANCY

Case No.	Age at operation	Time of onset after operation	Age at death	Site of recurrence
6 . . . . .	61 yrs.	10 mos.	62 yrs.	Larynx

TABLE 10.—DEATHS FROM ANY CAUSE WITHIN THREE WEEKS OF OPERATION

Case No.	Age at operation	No. of days following operation	Cause of death
5 . . . . .	79 yrs.	6	Angina pectoris

TABLE 11.—SHOWING PATIENTS ALIVE AND WELL

Case No.	Age at operation, yrs.	Sex	Present age, yrs.	Period since operation
1 . . . . .	48	Female	66	18 yrs., 5 mos.
2 . . . . .	41	Female	54	12 yrs., 8 mos.
3 . . . . .	59	Male	70	10 yrs., 10 mos.
4 . . . . .	54	Female	64	10 yrs., 1 mo.
7 . . . . .	26	Male	26	8 mos.
8 . . . . .	26	Male	26	6 mos.



SPEECH AFTER LARYNGECTOMY AND THE PSYCHOLOGY OF THE PATIENT. I am convinced that all patients can acquire a pharyngeal voice if they wish to. Some of our patients begin with the Mackenty artificial larynx for practice in forming the words, but later all discard the instrument.

In order to assist these patients in the rehabilitation of speech, my office nurse, Miss Lumbreyer, and my secretary, Mrs. Weiss, with my sanction instituted a speech class. It is indeed a revelation to see how quickly these patients acquire the pharyngeal voice. Being grouped together, this close association is a great incentive for one to try and surpass the other. Figure 34: A recent picture of some of the class patients. This class averages between 15 and 20 patients at each lesson.

Specially trained nurses are in attendance for all operations and after-care. It is essential in these cases that the nurse should have a cheerful disposition, for this is a most important factor in tiding the patient over the usual period of depression which comes on about the third day. Visitors are limited to the immediate family.

The psychology of getting the patient out of bed the day following operation is most valuable, in that it raises the morale materially. After such an operation the patient naturally assumes that he will be bed-ridden for some time, and he is very much surprised when permitted to be out of bed for fifteen minutes morning and afternoon, increasing the time daily. Despite the handicap, not one of the patients have taken the attitude that life is not worth living.

Consider for a moment the case of an Italian patient who could neither read nor write English but who could speak it—and we removed his larynx. Certainly this man had every reason to be most despondent, but strange as it may seem he was able to talk before he left the hospital and at the present time has one of the best pharyngeal voices. This is but one example of many that could be cited.

With all this in mind I cannot agree with those who stated that a laryngectomized patient scarcely finds life worth living.

IRRADIATION. Sixteen patients had postoperative irradiation with varying success. In some cases it was given as a precautionary measure; in others after a gland resection, if the glands were low in the neck. Preoperative irradiation was given to 2 patients.

## RECAPITULATION

*Laryngectomies* (including 1 lateral pharyngotomy)—94 Patients

Alive and well . . . . .	57
Died within three weeks of operation . . . . .	5
Had Recurrence . . . . .	23
Died from other causes . . . . .	9
Alive and well . . . . .	57
Died from other causes . . . . .	9
<hr/>	<hr/>
Successful . . . . .	66
Alive and well under one year . . . . .	5
Alive and well one to five years . . . . .	30
Alive and well five to ten years . . . . .	18
Alive and well ten years and over . . . . .	4
<hr/>	<hr/>
	57

*Laryngofissure*—8 Patients

Alive and well . . . . .	6
Died within three weeks of operation of coronary disease . . . . .	1
Had recurrence . . . . .	1
<hr/>	<hr/>
	8
Alive and well ten years or over . . . . .	4
(Ten yrs., 1; eleven yrs., 1; twelve yrs., 1; eighteen yrs., 1)	
Alive and well under one year . . . . .	2

*Combined Results*—102 Patients

Alive and well . . . . .	63
Died within three weeks of operation . . . . .	6
Had recurrence . . . . .	24
Died from other causes . . . . .	9
Alive and well under one year . . . . .	7
Alive and well one to five years . . . . .	30
Alive and well five to ten years . . . . .	18
Alive and well ten to eighteen years . . . . .	8
<hr/>	<hr/>
	63

$$63 + 9 = 72 \text{ successful cases}$$

CONCLUSIONS. That early recognition and diagnosis of cancer of the larynx offers its cure by surgery, either by laryngofissure, laryngectomy or by lateral trans-thyroid pharyngotomy.

That with 50 per cent recurrences from subglottic cancer, I believe a total laryngectomy is preferable to a laryngofissure in this type.

I cannot agree with those who say that the operation of laryngectomy is a mutilating one.

In my experience I have found that laryngectomized patients are not despondent; they are a happy lot and getting a great deal out of life.

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## DISCUSSION

DR. GABRIEL TUCKER, Philadelphia, Pa.:

I consider it an honor to open the discussion on Dr. Orton's paper. My first work in laryngectomy was done with Dr. Orton. Dr. Orton's skill is amazing. I may say that Dr. Orton does this procedure in about an hour and I think that to those of us who are doing laryngectomy that means a great deal. The beautiful moving picture demonstration that he has given us on the technique of laryngectomy might have been a photograph of any one of a number of laryngectomies that has been my privilege to see him perform.

I want to compliment Dr. Orton on his presentation and by way of emphasis, on the value of surgical treatment of cancer of the larynx I should like to refer to a series of my own cases. At the present time irradiation therapy is being urged as a substitute for surgery, particularly in early cases of carcinoma of the true vocal cords. My own series of cases is one of a large number of groups in which there have been obtained five-year cures in over 80 per cent of the cases by the procedure of laryngofissure. I think we should hesitate to refer early intrinsic cases of cancer of the larynx to the roentgenologist for treatment when surgery offers so much in the way of restoration of function and non-recurrence of the disease. The consecutive series of cases which I should like to mention includes a follow-up on 200 cases previously reported from the bronchoscopic clinics of the University of Penn-

sylvania. Of 315 cases in this series, 154 were considered suitable for surgical treatment, 144 intrinsic and 10 extrinsic involvement, because of the location and extent of the lesion. The extrinsic cases were all within the boundaries of the aryepiglottic folds, the tip of the epiglottis and the arytenoids posteriorly. Two of these extrinsic cases have been previously mentioned at this meeting and were treated by total extirpation of the epiglottis with cure. The surgical procedures employed were total laryngectomy, partial laryngectomy using Sir St. Clair Thomson's thyrochondrotomy technique with wide intralaryngeal excision of the growth, including the cartilage. In many of this group of cases postoperative irradiation was used either by intralaryngeal application of radium or external roentgen therapy. The group of early cases that I particularly wish to mention were those where the involvement was limited to the anterior two-thirds of the cords and the anterior commissure in which laryngofissure was done. Two methods of laryngofissure were used, the usual operative procedure after Jackson in which the involved cord is removed, including the internal perichondrium, and laryngofissure by the anterior commissure technique of Jackson used in those cases where the anterior commissure was involved. In the series there were 92 patients in which involvement was such that the procedure of laryngofissure was carried out. In this group there were 12 recurrences that have resulted in cancer deaths. The percentage of cure in the group shows 13+ per cent cancer deaths and 86+ per cent cures to date. All the recurrences after laryngofissure recurred before the four-year period had elapsed. There were 55 patients that showed cures over a period of from four to fifteen years. There remained under the four-year period without recurrences 25 patients, 6 three-year cures, 11 two-year cures, 8 one-year cures. Total number of patients without recurrence in the series, 80. There was one postoperative death in a patient who had an extensive bronchiectasis. The patient developed a pneumonia in spite of the fact that a tracheotomy had been done and frequent aspiration of the secretion from the lung carried out. There were no other fatal postoperative complications in the series.

The functional results after laryngofissure are excellent. In no case has a permanent tracheotomy tube been required. The functional results after laryngectomy have also been excellent. In the series an unusual coincident occurred. Two physicians associated in surgical practice developed Grade 2 squamous-cell carcinoma of the right ventricular band in each case. Both were treated by total



laryngectomy, one five years ago and the other one and a half years ago. Each physician has developed an excellent voice by regurgitating air from the esophagus and both are very successfully carrying on their practice in surgery without the use of an artificial larynx.

All cases considered unsuited for surgery were referred for radium or deep irradiation therapy. In the surgical treatment of carcinoma of the larynx irradiation was used where there was recurrence. In all cases where the lesion was not suitable for surgical removal, irradiation was used. I have seen a number of cures after the use of irradiation, but the percentage of cures does not compare to that of surgery. I have observed that recurrence in irradiated cases is frequently at the site of the primary lesion, indicating that the recurrence and metastatic lesion is probably more susceptible to irradiation than the primary lesion itself. This has been borne out by the observation on patients where recurrences have taken place postoperatively; irradiation therapy has produced a very remarkable disappearance of the growth.

MR. VICTOR E. NEGUS, London, England:

It has been a great pleasure, sir, and an education to hear the paper of Dr. Orton, and also to listen to Dr. Tucker's remarks. The results that Dr. Orton has obtained seem to be extraordinarily good, and I envy him his success.

There are one or two points to which I should like to refer. One is the small number of cases of postericoid carcinoma he seems to have met with. We seem to get quite a large number, comparatively; in the last few years, about 22 cases, although not necessarily operable.

His remark about the right cord has a point of interest. It has been discussed by two or three people. A laryngologist connected with the Conservatoire of Music in Moscow, I think, reported that the average individual does not use both cords equally, but that one cord vibrates more than the other; I have seen that myself with the stroboscope, in a case where one cord was stationary and the other moving freely, and it is possible that the right cord in the majority of people may move more than the left. That is merely a suggestion.

Diabetes, of course, is usually a contraindication. I have removed the larynx in a patient with severe diabetes, and the result was quite good. Some years afterward the man got a recurrence in glands on one side of the neck, which I had to remove by block dissection, and he is perfectly well now after twelve years.

As to speech, we have a speech therapy department in my hospital in London where these people are educated, and the attempt is usually

made to get them to learn the esophageal method of speech as being simpler and more natural than speech with an artificial larynx. Many of them get on very well indeed.

There is one question I should like to ask Dr. Orton, which is one of doubt in my mind, and that is about teeth; whether he has all the teeth removed before operation, or only any teeth that may be carious. Mr. Trotter, who has had a great influence upon us in England, has laid down an axiom that before such operations of pharyngotomy, or operations of that type, including pharyngeal diverticulum, the teeth should be removed to avoid sepsis in the wound and infection of the lungs.

The removal of the hyoid bone seems to be a very useful modification, because that is a point I think where one has the greatest difficulty of healing, but I had not known before this that it was carried out habitually.

One has generally used the method of removing the larynx from above down, but it does not seem to make very much difference, and in certain cases where there has been obstruction, one has employed Mackenty's method—at the beginning of the operation skeletonizing the larynx, inserting a big rubber tube, and then continuing the operation from above down. It comes to very much the same method as Dr. Orton's, I think, where he opens the trachea before finishing removal.

Another little point is cutting the trachea obliquely so that the resulting opening is bigger than if it is cut transversely. We use a tapered tube with a double flange. It also keeps the dressings from falling over the opening of the tube.

The series of cases reported by Dr. Tucker is marvelous, I think. It is extremely encouraging, and confirms one's opinion that it would be quite wrong to abandon an operation of that type with such wonderfully good results as regards the absence of recurrence, and also—as I have had the opportunity while I have been here of seeing for myself—with extraordinarily good functional results.

DR. FLETCHER D. WOODWARD, Charlottesville, Va.:

Our problem is what to do for the many cases of advanced extrinsic cancer of the larynx that we see. Cases suitable for laryngofissures are very rare.

After trying many types of procedure, we have come to the point whereby in these cases of advanced cancer we do a laryngectomy, disregarding the extension of the tumor into the neck, suture the tracheal

stump to the skin, close the wound, and follow with fractional dose x-ray therapy after the method advocated by Dr. Coutard. By doing that, I believe we have gotten some results. We have two cases which have been apparently cured for four years, and there are several others which have not been that long but are still free of cancer so far as we know.

I should like to have Dr. Orton say a few words as to what he does with these apparently hopeless cases that come in.

DR. JOSEPH C. BECK, Chicago, Ill.:

May I ask a question? I think I did not hear anything about the cases that had been previously x-rayed or where radium therapeutics were used, the method of radium application and the therapeutic experience in those cases.

Another question. In the introduction of the feeding tube as presented, does he permit immediate swallowing?

DR. HENRY B. ORTON (in closing):

I wish to take this opportunity to thank the various gentlemen for discussing this paper.

Replying to Dr. Tucker: Since in my series I have performed more laryngectomies than laryngofissure, it does not mean that I am trying to discredit the operation of laryngofissure. In the patients on whom I perform a laryngofissure, the growth is located on the surface or edge of the vocal cord at the junction of the anterior and middle third, and does not extend to the anterior or posterior extremities of the vocal cord, nor dip down into the subglottic area. All my laryngofissure cases have been of this type and the results are in keeping with the high percentage of cures extending as far back as eighteen years ago. Another reason for my preference of laryngectomy to laryngofissure, is that we had 7 cases in which the site of growth at operation was subglottic, and these 7 had all a recurrence in the glands of the neck with metastasis to the brain and lung. It would seem inevitable that if we had a recurrence of these in total laryngectomy we certainly would have had the same in laryngofissure.

Replying to Mr. Negus: We do see quite a number of the post-cricoid type, but unfortunately by the time I see them they have a mass of glands on both sides of the neck that are fixed, and I certainly do not think these are suitable cases for surgery.

Regarding the question of dental hygiene: if the patient to be operated upon has carious teeth, as most of them do, the very worst

teeth are removed and the remaining ones are cleaned up. We do not make it a rule to remove all of the teeth.

Replying to Dr. Beck: In regard to the effect of *x*-ray and radium on the larynx before operation. I have given a few patients a pre-operative *x*-ray and they have healed perfectly well, but I will not operate on a larynx after massive radiation has been used.

Replying to Dr. Woodward as to what is done with the indigent hopeless cases. This is a problem I have had on my hands for many years; being chairman of the Cancer Control for the State of New Jersey I am still trying to find a place for this type of patient who is beyond all possible hope of operation or even a cure by radiation.



## AN ANALYSIS OF 75 CASES OF BRONCHIECTASIS FROM THE VIEWPOINT OF SINUS INFECTION

ROBERT L. GOODALE, M.D.

THE purpose of this paper is to present the clinical findings with special reference to the rôle of sinus infection obtained from a study of 75 cases of bronchiectasis at the Pulmonary Clinic of the Massachusetts General Hospital. Three years ago it was decided to assign a laryngologist to this clinic in order to carry on investigations regarding the etiological rôle of sinus infection, and to arrive at a more definite understanding of the importance of sinus infection when present in cases of bronchiectasis. Through the kindness of Dr. Mosher this assignment was offered to me and here at the beginning of this paper I wish to thank him for his help throughout this investigation.

Before the cases are referred for inspection of the nose and throat they have been given a complete general examination including an *x*-ray of the chest after injection with lipiodol. I have not included in this study any cases where there is any doubt in regard to the presence of bronchiectasis. The nose and throat investigation consists of the routine procedure followed in this hospital. It includes a careful history and an examination of the nasal passages, postnasal space, pharynx and larynx, and transillumination and *x*-ray of the sinuses. Lipiodol is not used in the *x*-ray of the sinuses, as it has been found that this procedure in our clinic adds little in the way of diagnosis to what can be discovered by Dr. MacMillan's usual *x*-ray technique. In approaching this problem, the following questions presented themselves.

1. To what disease does the patient attribute the onset of his pulmonary infection?

2. Do the cases show any tendency to arrange themselves in corresponding etiological groups?

3. What is the incidence of sinusitis for each such etiological group?

4. Does the incidence of sinusitis in each such etiological group vary with the ascribed etiology?

5. Does the sinus infection bear any relationship to the extent of the bronchiectasis?

6. Whether or not any sinus infection was present at the onset, does it play any part in the subsequent course of the pulmonary disease?

To simplify the data in a study which is essentially statistical, I am going to present it in four sections. The first section is intended to give the general picture of this series and is mainly a matter of vital statistics. The second section gives the etiology as obtained in the histories and reasons for considering the several diseases as distinct etiological groups. The third section correlates the incidence of sinus disease with the etiology and the fourth section correlates the sinusitis with the severity and extent of the bronchiectasis.

I

In the first two charts I will show you the material on which the study is based.

CHART 1.—NUMBER OF CASES—75

	Cases	Per cent
Male . . . . .	34	45 $\frac{1}{2}$
Female . . . . .	41	54 $\frac{1}{2}$

*Age by Decade at Onset of Bronchiectasis*

	1st	2d	3d	4th	5th	6th
No. of cases . . . . .	24	20	10	7	5	3

The incidence of bronchiectasis is slightly higher for women. It is interesting to note also that bronchiectasis is much more likely to occur in the first two decades of life.

CHART 2.—DURATION OF THE BRONCHIECTASIS AT THE TIME OF EXAMINATION

	Cases
1 year or less . . . . .	5
2 years . . . . .	7
3 years . . . . .	8
4 years . . . . .	4
5 to 10 years . . . . .	22
11 to 20 years . . . . .	16
21 to 30 years . . . . .	6
Cases Undetermined . . . . .	7

In this series 65 per cent of the cases at the time of examination have had their bronchiectasis for more than five years. Only 5 cases have had it for a year or less.

By the time these patients come to us for a nose and throat examination, the bronchiectasis is frequently of several years' duration, and sinusitis, if present at all at the onset of the pulmonary disease, therefore has had plenty of time to become established as a chronic infection. The greater number of cases, 65 per cent, have had their pulmonary disease for more than five years as is shown in Chart 2. With these few vital statistics in mind, I shall now discuss the diseases to which the patients attribute their bronchiectasis.

## II

CHART 3

Disease at onset	No. of cases
Upper respiratory infections:	
Head colds . . . . .	21
Sinusitis . . . . .	6
Head colds with pneumonia . . . . .	5
Total . . . . .	32
Pneumonia . . . . .	21
Influenza . . . . .	5
Asthma . . . . .	2
Bronchitis . . . . .	1
Whooping cough . . . . .	5
Tb. contact . . . . .	1
T. and A. . . . .	2
Unknown . . . . .	6
Total . . . . .	75

The classification of upper respiratory infection, "sinusitis" and "colds with pneumonia" can be grouped together and account for 42.6 per cent of this series. Pneumonia alone is the next most frequent etiological disease.

By "upper respiratory infections" is indicated the group of patients that give "head colds" but not "sinusitis" in the history. It may be reasoned that the classification of upper respiratory infections as distinct from sinusitis is an unimportant one here. However, it does indicate degrees of severity. I have put it down here in a separate group intentionally to emphasize the fact that only 6 of these cases could state positively that their bronchiectasis followed a sinus infection.

There is also another classification which should be explained, that of "colds with pneumonia." I realize that some observers believe that all cases of pneumonia have an onset preceded by an

upper respiratory infection. But these 21 cases of "pneumonia" could not recall any preceding upper respiratory infection. Only 5 cases were able to remember a head cold occurring at the time of their pneumonia. I am inclined to place these "colds with pneumonia" cases with the sinusitis group, as without exception, as I will show later, they all have a chronic sinusitis. Also, in order to have a clear-cut differentiation of etiology I wish to keep the 21 cases of "pneumonia alone" as a distinct group.

So far, I have considered the disease occurring at the onset of the bronchiectasis, the age at which the bronchiectasis began, and the relative proportion of the two sexes. On checking these three factors against each other, I found some rather striking relationships.

CHART 4.—AGE AT ONSET BY DECADES

Disease at onset	No. of cases						
	1st	2d	3d	4th	5th	6th	?
Upper respiratory infections:							
Head colds . . . . .	2	8	4	2	2	2	2
Sinusitis . . . . .	2	3	..	1			
Colds and pneumonia . . . .	1	1	1	2			
Total . . . . .	5	12	5	5	2	2	2
Pneumonia . . . . .	11	4	4	2			
Influenza . . . . .	2	3					
Asthma . . . . .	..	..	1	1			
Bronchitis . . . . .	1						
Whooping cough . . . . .	3	1	..	..	..	..	1
Tb. contact . . . . .	..	1					
T. and A. . . . .	..	1	1				
Unknown . . . . .	4	2					

Upper respiratory disease is the etiological factor more often in the 2d decade, but continues later in life than any other factor. Pneumonia is much more frequently the cause of bronchiectasis before the age of ten years.

In considering the age at onset, in relationship to the disease at onset, the cases which gave an upper respiratory etiology, seemed to occur more frequently between the ages of ten and twenty years. Also upper respiratory disease tends to continue as the causative factor, later in life than any other disease. In contrast to this, pneumonia has its highest incidence in the first decade of life.

The next relationship, that of sex to etiology, shows another striking fact. The group of upper respiratory cases have a higher



proportion of men than women, and conversely the cases of pneumonia alone, whooping cough, bronchitis, influenza, all seem to have a higher proportion of women. This is important as it is another point of differentiation in etiology.

CHART 5.—ETIOLOGY AND SEX

Disease	Male	Female
Upper respiratory infections		
Head colds		
Sinusitis		
Head colds and pneumonia . . . . .	20	12
Pneumonia . . . . .	8	13
Influenza . . . . .	1	4
Asthma . . . . .	1	1
Bronchitis . . . . .	0	1
Whooping cough . . . . .	1	4
T. and A. . . . .	0	2
Unknown . . . . .	4	2

Upper respiratory infection is more frequently the etiology among males. Pneumonia, influenza and whooping cough are more frequently the etiology among females.

When we see that the upper respiratory group has age at onset and sex incidence characteristics that are different from those of the pulmonary group, it lends support to the belief that there is no one single cause of bronchiectasis. There seem to be several diseases that have etiological significance. As the cases with upper respiratory etiology tend to occur later in life than the pneumonia cases, I suggest that these form a group where repeated upper respiratory infections, either head colds or sinusitis, over a considerable period of time, have led to pulmonary infection. This would be a type of slow onset, where the mechanisms of inhalation, chronic cough or lymphatic drainage play their part.

The pneumonia group represents a type in which the bronchiectasis has occurred as the result of one acute infection. At the risk of repetition I would like to sum up the data so far presented. We have here a group of cases who have had bronchiectasis for a considerable period of time for the most part. They have acquired the pulmonary disease in the majority of cases before the age of twenty years. In fact, nearly a third of them developed the disease before the age of ten years. They ascribe their bronchiectasis to several diseases. The greater number acquired it following an upper respiratory infection. However, 21 cases had pneumonia

at the onset which was not preceded, so far as they know, by an upper respiratory infection. The upper respiratory group and the pneumonia group have special points of differentiation, namely, age at onset and sex which, to my mind, confirm the statements of the patients and indicate that bronchiectasis can occur without a preceding upper respiratory infection.

### III

I will now take up the relationship of the etiology as given by the patients and the presence of sinusitis as shown by x-ray.

CHART 6.—RELATION OF DISEASE AT ONSET TO SINUSITIS (BY X-RAY)

Disease at onset	Cases	Incidence of sinusitis	
		Sinusitis	Negative
Upper respiratory infections:			
Head colds . . . . .	21	11	10
Sinusitis . . . . .	6	6	0
Colds with pneumonia . . . . .	5	5	0
Total . . . . .	32	22	10
Pneumonia . . . . .	21	12	9
Influenza . . . . .	5	2	3
Asthma . . . . .	2	2	0
Bronchitis . . . . .	1	0	1
Whooping cough . . . . .	5	4	1
Tb. contact . . . . .	1	0	1
T. and A. . . . .	2	1	1
Unknown . . . . .	6	3	3
Total . . . . .	75	46	29

In the upper respiratory group the incidence of sinusitis is a little less than 69 per cent. As one would expect, it is greater than in the pneumonia group, but note that in this latter group 57 per cent have sinus infection.

It is surprising to note here the relatively high incidence of sinusitis as found in the group of 21 cases of pneumonia. Twelve have now a demonstrable sinus infection. Nine have a negative sinus x-ray. As one would expect, the group of "colds," "sinusitis," and "colds and pneumonia" have a much higher incidence of sinus infection. Twenty-two have sinusitis and 10 have no evidence of sinusitis. How can we explain the sinus infection in cases which do not give upper respiratory infection or sinusitis as an etiological factor?

It is striking to observe the high incidence of frequent upper

respiratory infections, subsequent to the onset of bronchiectasis. Chart 7 correlates the frequency of upper respiratory infection with *x*-ray evidence of sinusitis. The *x*-ray picture corresponds to the history of upper respiratory infections occurring subsequent to the onset of bronchiectasis rather than to the ascribed etiology.

CHART 7.—RELATIONSHIP OF SUBSEQUENT UPPER RESPIRATORY INFECTIONS AND SINUSITIS

	Sinusitis Cases	Negative Cases
A. Group as a whole:		
Upper respiratory infection since onset:		
Slight . . . . .	9	17
Frequent or severe . . . . .	37	12
	<hr/> 46	<hr/> 29
B. Comparison of pneumonia and upper respiratory groups:		
1. Cases of pneumonia:		
Upper respiratory infection since onset:		
Slight . . . . .	2	7
Frequent or severe . . . . .	10	2
	<hr/> 12	<hr/> 9
2. Cases with head colds or sinusitis at onset:		
Upper respiratory infection since onset:		
Slight . . . . .	2	3
Frequent or severe . . . . .	20	7
	<hr/> 22	<hr/> 10

The incidence of sinusitis closely parallels the frequency or severity of upper respiratory infections occurring since the onset of the bronchiectasis.

Naturally it can be stated that there are grades of chronic sinusitis, and that there are cases with a negative sinus *x*-ray who have had repeated attacks of acute sinusitis with recovery. In other words, can we rely entirely on the negative *x*-ray evidence of sinusitis to rule out sinus infection etiologically? Or can bronchiectasis occur in a case with a negative sinus history and absolutely negative sinus examination?

In this series of 75 cases, 46 now have chronic sinus infection and 29 have negative sinuses according to the *x*-ray evidence. Of these 29 negative cases, 18 have a history or clinical evidence suggestive of sinusitis, either a recurrent acute sinusitis or a mild chronic sinusitis.

Eleven cases have absolutely no evidence of past or present sinusitis. In these negative cases the disease at onset is as follows:

Pneumonia, 6 cases.

Upper respiratory infection, 1 case. Here the infection was more pharyngeal than nasal.

Influenza, 1 case.

Whooping cough, 1 case.

Tb. contact, 1 case.

T. and A., 1 case.

In this small group comprising  $14\frac{1}{2}$  per cent of this series, sinusitis seems to have played no part in the onset or subsequent course of their bronchiectasis.

In considering the group who give "head colds" as the etiological factor, it is evident that a negative sinus x-ray is not conclusive. In the pulmonary group we have another very important thing to consider and that is the undeniable fact that these people had a severe pulmonary infection whether or not they had a nasal infection at the same time.

To sum up the facts so far presented I will give very briefly the conclusions which I have reached at this point. There seem to be definitely, several different diseases which can cause bronchiectasis. The largest group is that of upper respiratory infection. Pneumonia alone accounts for a considerable number, 21 cases in this series. This statement, however, does not give a clear picture of the situation when we see the patient in later years. As you have seen, these cases show a marked susceptibility to severe upper respiratory infection. As time goes on these repeated upper respiratory infections have produced the picture of sinus disease which is greater than the etiology would indicate. This point may seem quite obvious but I have a feeling that when we see a case of bronchiectasis with sinus infection, there is a tendency to consider the sinusitis as the original cause of the bronchiectasis without considering the possibility that the sinusitis may have developed subsequently and independently of the pulmonary disease.

#### IV

The next problem to be considered is the relationship of sinusitis to the pulmonary infection. Whether or not the sinus infection was an important factor at the onset of bronchiectasis, once the patient has acquired a sinus infection, does it predispose him to



further pulmonary damage? Also, is there any correlation between the degree of sinusitis and the extent of bronchiectasis? The practical question arises, when considering the therapeutic management of one of these cases, whether we should consider a radical sinus operation. So far as the bronchiectatic cavity is concerned, we do not expect to eradicate it by sinus surgery. But the importance of removing the sinus infection is shown in the next chart in which you may observe a high percentage of sinusitis in cases who have had repeated pulmonary infection.

CHART 8.—RELATIONSHIP OF SINUSITIS TO REPEATED PULMONARY INFECTION.

Case No.	Pulmonary infection	Original etiology	X-ray
1	Empyema	Head cold	Both ethmoids chronic with active infection; left frontal thickened
2	Pneumonia	Sinusitis	Fluid both antra
3	Pleurisy	Sinusitis	Chronic thickening both antra and ethmoids
4	Pneumonia	Sinusitis	Both antra and ethmoids thickened
5	Pneumonia	Head colds	Neg. (clinically recurrent acute sinusitis)
6	Pneumonia 3 times	Head colds	Left pansinusitis
7	Pneumonia	Pneumonia	Neg.
8	Pleurisy	Pneumonia	Both ethmoids and antra pus
9	Pneumonia 2 times	Pneumonia	Neg.
10	Pneumonia 3 times	Pneumonia	Neg.
11	Pneumonia	Pneumonia	Both ethmoids and antra secretion
12	Pneumonia	Pneumonia	Both ethmoids and antra thickened
13	Pneumonia	Pneumonia	Bilateral pansinusitis
14	Pneumonia	Asthma	Both ethmoids and antra thickened; pus
15	Pneumonia	Wh. cough	Both ethmoids and antra thickened; pus
16	Pneumonia and empyema	Wh. cough	Both ethmoids and antra thickened; no pus

These 16 cases have had severe pulmonary infection subsequent to the onset of bronchiectasis. Four of these cases have negative sinus x-rays. The remaining 12 have a marked degree of sinusitis.

In Chart 8 I have collected 16 cases who had a subsequent pulmonary infection. Two of these cases, as you see, have had 3 attacks of pneumonia and 1 has had two attacks of pneumonia occurring after the onset of bronchiectasis. Six of these cases had an onset attributed to head colds or sinusitis. In this group there is evidence of definite sinus infection. There are 7 cases who attribute the onset to pneumonia. Three of these have no evidence of sinusitis. Four have a marked degree of sinus infection. The

case of asthma and the 2 cases of whooping cough also have marked sinus infection. It seems that in these cases of recurrent pulmonary infection there is an extremely high incidence of severe sinusitis which constitutes a possible source of reinfection for the lungs.

CHART 9.—LOCATION OF SINUSITIS

Sinuses involved	No. of cases
Right antrum only . . . . .	0
Left antrum only . . . . .	4
Both antra . . . . .	3
Left ethmoid only . . . . .	3
Both ethmoids only . . . . .	3
Right ethmoid only . . . . .	0
Right frontal only . . . . .	1
Left frontal only . . . . .	0
Both ethmoids and antra . . . . .	18
Both ethmoids and frontals . . . . .	2
Both ethmoids and right frontal . . . . .	1
Right pansinusitis and left antrum . . . . .	1
Right pansinusitis and left antrum and ethmoid . . . . .	1
Left pansinusitis . . . . .	1
Left pansinusitis and right antrum and ethmoid . . . . .	3
Both pansinusitis . . . . .	5
	<hr/> 46

The ethmoids and antra are about equally subject to infection. In 18 cases there was bilateral infection of the ethmoids and antra.

Chart 9 gives the location of the sinus pathology. I cannot explain why no cases show infection of the right antrum or right ethmoid alone. In those cases where more than one sinus is involved, we find infection of the right antrum and ethmoid frequently. Does this bear any relationship to the fact that the left lower lobe of the lung is more often the site of bronchiectasis than any other one lobe? Does it suggest a homolateral factor?

In another chart, which I will show you presently, 20 cases of left lower lobe bronchiectasis are given, 9 of whom had *x*-ray evidence of sinusitis, and of these, 2 cases had right-sided sinusitis alone. Of 6 cases with right lower lobe bronchiectasis, 2 have sinusitis and both have more involvement of the left side than the right.

But it is suggestive in examining the 4 cases of left antrum infection to find that they all have infection of the left lower lobe, 2 of them with a left lower lobe only. Three of these cases give a history of upper respiratory infection, and 1 of whooping cough

at the onset of the bronchiectasis. One case was complicated by a tumor in the mediastinum and this case had an involvement of both lower lobes and right middle lobe. The pathology in the sinuses of these 4 cases is very slight, as follows:

Case 1. Small amount of cystic membrane left antrum.

Case 2. Left antrum very slightly thickened.

Case 3. Cyst in left antrum.

Case 4. Left antrum chronic; small amount of fluid.

Case 4 is the patient with mediastinal tumor and extensive bronchiectasis.

Without giving each case in detail, we may summarize them as a whole in regard to the *x*-ray diagnosis. Seventeen cases showed pus or retained secretion in addition to changes in the mucous membrane in the sinuses affected. Three cases had cysts only. Six cases had very slight thickening of the lining membrane of the sinuses. Ten cases had moderate thickening, and the remaining 9 cases had a marked thickening of the membrane. Generally speaking, those cases that had the greater number of sinuses involved, also had a more severe pathological change in the membrane.

I should like to call your attention to the high incidence of infection of both ethmoids and antra in this series. In fact, it is striking that the ethmoid was infected quite as frequently as the antrum. This is very important from the operative point of view. In those cases which have been operated on where the ethmoids and the antra were infected, the best results were obtained, as you would expect, where a thorough exenteration of the ethmoids was done, in addition to a radical antrum operation. In fact, no matter how bad the antrum seems to be, if the ethmoid is also infected, unsatisfactory results will be obtained if the ethmoid is only partly cleaned out.

In the next 2 charts I will show the relationship of the extent of bronchiectasis to the sinusitis. In the first chart I have gone into the question of the location of the sinusitis in cases of one lobe bronchiectasis. In the second chart is given the relationship of sinusitis to the location of bronchiectasis.

There is no evidence here that there is any connection between the side of the chest which is affected and the location of the sinus-

itis. The question of the incidence of sinusitis in cases involving one lobe is important to the thoracic surgeon as these are the cases which lend themselves more favorably to lobectomy. It is important to note that the incidence of sinusitis is much lower among these cases than it is where more than one lobe is infected.

CHART 10.—SINUSITIS IN CASES OF UNILATERAL BRONCHIECTASIS

Lobe involved	Cases	Incidence	Cases	Sinuses involved	Cases
L.L.L.	20	Sinuses neg.	11	L. antrum	1
		Sinusitis	9	R. fron.	1
				B. eth. and antra	5
				B. eth. and fron.	1
				R. fron. and R. eth.	1
R.L.L.	6	Sinuses neg.	4	L. eth.	1
		Sinusitis	2	L. fron. and eth.	1
				B. antra	1
R.L.L. and R.M.L.	5	Sinuses neg.	2	B. eth.	2
		Sinusitis	3	B. pan.	1
L.L.L. and L.U.L.	5	Sinuses neg.	2	B. eth. and antra	2
		Sinusitis	3	L. eth.	1

The side on which the sinusitis occurs does not correspond to the side of the chest which is affected. Cases in which one lobe is infected have a lower incidence of sinusitis.

CHART 11.—RELATIONSHIP OF LOCATION OF BRONCHIECTASIS TO INCIDENCE OF SINUSITIS

	Sinusitis (per cent)
1. L.L.L.	45
2. L.L.L. and L.U.L.	60
3. R.L.L.	33
4. R.L.L. and R.M.L.	60
5. R.U.L., 1 case	Neg.
6. B.L.L.	67
7. L.L.L. and R.M.L.	73
8. R.L.L. and L.U.L. <i>Situs inversus</i> , 1 case	Sinusitis
9. B.L.L. and R.M.L.	67
10. B.L.L. and L.U.L., 1 case	Neg.
11. Pan bronchiectasis	75

Cases of bronchiectasis where one lobe only is affected have a lower incidence of sinusitis than those cases where more than one lobe is affected.

Chart 11 gives the relationship of infection of the various lobes of the lung to the incidence of sinusitis. Cases in which one lobe only is involved have a lower percentage of chronic sinusitis than those in which 2 or more lobes are diseased. In other words, the degree of sinus involvement parallels the extent of the bronchiectasis. Reduced to percentages, the sinuses are infected in 40.7 per cent of those cases in which one lobe only is involved, and in those with more than one lobe involved, in 73 per cent of the cases.



CONCLUSIONS. 1. I do not think that we can attribute all cases of bronchiectasis to a preceding upper respiratory infection, although the greater number of cases certainly have had a high incidence of head colds at the onset.

2. Other diseases, pneumonia being the most important, can cause bronchiectasis.

3. When we see the patient in later years, we find a very high incidence of sinus infection. This is higher than can be accounted for by the history of the disease at onset. It bears a closer relationship to the history of subsequent upper respiratory infection.

4. The sinusitis corresponds to the extent of the bronchiectasis.

5. Once the patient has acquired a chronic sinusitis, the chance of further damage to the lungs is increased because these patients are more susceptible to repeated respiratory infections.

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#### DISCUSSION

DR. HENRY M. GOODYEAR, Cincinnati, Ohio:

I was particularly interested in the operative treatment of these cases, especially children. I do not believe we ever have an active antrum infection without some ethmoid infection at the same time.

If opaque oil is placed in an antrum, and the patient permitted to bend forward, or lie down, before making roentgenograms, often oil will have made its way to the ethmoid area, or even into the frontal sinus of the same side.

A child, lying with an antrum filled with pus, must infect the ethmoid labyrinth.

I do not believe we are justified in opening the ethmoid cells at the primary operation in these cases. A window resection under the inferior turbinate will drain the antrum, and incidentally give the ethmoid cells an opportunity to recover.

The prevention of a bronchiectasis is so important that prolonged coughs and sinus infections in children demand every attention until recovered.

To permit a permanent dilation of the bronchial tubes by indifferent treatment is unforgivable, and, as a last resort, I would sacrifice my practice to go to some dry climate should a child of mine be threatened with such a life handicap.

I wish to ask Dr. Goodale if the staphylococcus is predominant in the secretions of his cases.

DR. THOMAS C. GALLOWAY, Evanston, Ill.:

What was the type of pneumonia in these cases? According to the work of Galbraith and Hubbard, and perhaps some clinical observations of Holinger which he has recently recorded, the lung infections which produced bronchiectasis are likely to be of the type of infection caused by tracheobronchitis with atelectasis. I saw such a case autopsied recently in which if the patient had lived, one could have assumed, I think, that there probably would have been bronchiectasis. The interesting factor was that this case was associated with an asthma, and I wonder if Dr. Goodale has made any particular observation in these cases as to the presence of allergy, and whether or not the associated sinus and lung condition could have a common etiology in allergy.

DR. HARRIS P. MOSHER, Boston, Mass.:

The way this paper came about was this: Dr. Churchill, of the Thoracic Clinic, at the Massachusetts General Hospital, said, "I do not believe that all cases of bronchiectasis hark back to sinus disease. Do you know of anybody who can answer this question for me?"

He put it in such a challenging way that there was nothing for me to do but to produce a man to work on the question, and this man was Dr. Goodale, and you have heard the result of his investigations. He has proved to my mind that the old-fashioned idea that bronchiectasis was practically always caused by sinus disease is a fallacy. It has been partly proved for a long time that there was a direct anatomical connection between sinuses, the pharynx and the lungs, but it remained for LaSalle last year to prove that there was a direct bacteriological invasion from the sinuses to the pharynx, to the lungs, then to the heart, and from there on. I feel that Dr. Goodale has changed our ideas about the incidence of bronchiectasis and sinus disease very materially.

DR. ROBERT L. GOODALE (in closing):

Thank you very much for the interesting discussion.

Dr. Mosher has told you the history of the study. It is a great privilege for me to have the opportunity to pursue it.

In regard to Dr. Goodyear's remarks as to the ethmoid operation in children, I entirely agree. I mentioned that this was a group largely of adults. I feel that in an adult you have a different problem from what you have in a child.

My remark in regard to the operative procedure was based largely

on cases which I have operated on myself and have seen operated at the hospital. They were all adults.

Dr. Galloway asked what type of pneumonia occurred at the onset. It was not possible to get that from the history. Many of these cases came into the hospital for the first time, years after they had acquired their bronchiectasis. They didn't know what type of pneumonia they had.

So far as the allergic picture is concerned, very few of these cases were allergic. I think, as I showed on the chart, only 2 cases were asthmatic. In the others I found no evidence of allergy.

In regard to the possibility of an upper respiratory infection, not sinusitis as the cause of bronchiectasis, a great many of these cases, as I have shown, had acute upper respiratory infections of a severe type, and yet did not have sinusitis, and I believe it is possible that that might be an etiological factor. Certainly all these bronchiectatic cases are extremely susceptible to infections.

## THE ANAPHYLAXIS REACTION: TISSUE CHANGES AS OBSERVED IN LIVING TISSUES IN THE RABBIT

HARRY P. SCHENCK, M.D.

AND

RICHARD G. ABELL, PH.D.

THE inclusion of this paper in the program of the Sixtieth Annual Congress of the American Laryngological Association has undoubtedly appeared to be the injection of an irrelevant subject into an otherwise perfect program. Anaphylaxis would seem to be a subject quite foreign to the problems we, as laryngologists, are concerned with. Allergic reactions in our patients, however, are frequently the cause of concern and allergy continues to offer many problems which require solution. In order to understand more fully the mechanism of allergy in man we turned to the study of the mechanism of anaphylaxis in laboratory animals. We were able to see and record certain changes in the living tissues of the rabbit during the anaphylaxis reaction. More recently, we have been able to make similar observations upon the living blood-vessels of man during the allergic response and to compare these reactions with those occurring in the rabbit during the anaphylaxis reaction. We hope to present our observations in man at a subsequent meeting of the Association.

That rabbits become hypersensitive to foreign proteins, when these are injected in the proper amount, is well known. The reinjection of the particular protein in question, a sufficient length of time after the original injection, produces anaphylactic shock. If sensitization is adequate, and if the amount of protein injected is sufficient, anaphylactic shock is fatal.

It has been shown that death in the rabbit in anaphylaxis is a cardiac death, caused by obstruction of the pulmonary circulation. Such obstruction, it is generally believed, is produced by contraction of the pulmonary arterioles, and the arterioles, in anaphylaxis, are thought to contract not only in the lungs but also elsewhere in the body.



This report is concerned with direct observations of the behavior of arterioles, capillaries, and venules of the rabbit during the reaction of anaphylaxis. These observations were made with the high powers of the microscope upon living blood-vessels in transparent moat chambers introduced into the rabbit's ear.

Such a moat chamber contains a very thin, inclosed space, called the *bay*, which has a glass bottom and a mica top. Following the introduction of the chamber into the ear of a rabbit, blood-vessels grow into the bay through two entrance holes at the proximal end. The vessels then grow across the bay toward the distal end, which opens into a much larger enclosed space, called the *moat*. Solutions may be introduced into, and subsequently removed from, the moat by way of two access cannulæ, one sealed into each end.

Since the moat opens directly into the bay, solutions introduced into the moat pass by diffusion into the bay, where they come in contact with the vessels growing there. The reaction of the vessels can be clearly seen with the high powers of the microscope because the bay has a glass bottom and a mica top and is only 50 to 100  $\mu$  deep.

*Fig. 1, a*, shows the vessels in the bay of a chamber in the ear of a rabbit sensitized to horse serum, and this is the *normal* appearance of the arterioles, capillaries, and venules of the sensitized rabbit. The proximal end of the bay is at the bottom of the photomicrograph; the distal end, which communicates with the moat, at the top. The arrows pointing upward are placed next arterioles; those pointing downward, next venules. *A1* and *A2* are large arterioles. *C* indicates two peripheral capillaries, *V* a venule, and *L* a lymphatic. The white letter *a* is placed over a segment of one entrance hole. The rectangle, on the right side of the photograph is the portion of the bay shown in *b*, *c*, and *d* of this figure. Enlargement,  $\times 27.4$ .

*Fig. 1, b*, shows the vessels six and a half minutes after the intravenous injection of 5 cc. of horse serum into this rabbit, which, as you remember, is sensitized to horse serum. The injection caused the arterioles to contract, *obliterating their lumina*, and *stopping the circulation*. No contraction of capillaries or small venules occurred, and no precipitate appeared in the blood plasma. Branch *X* of

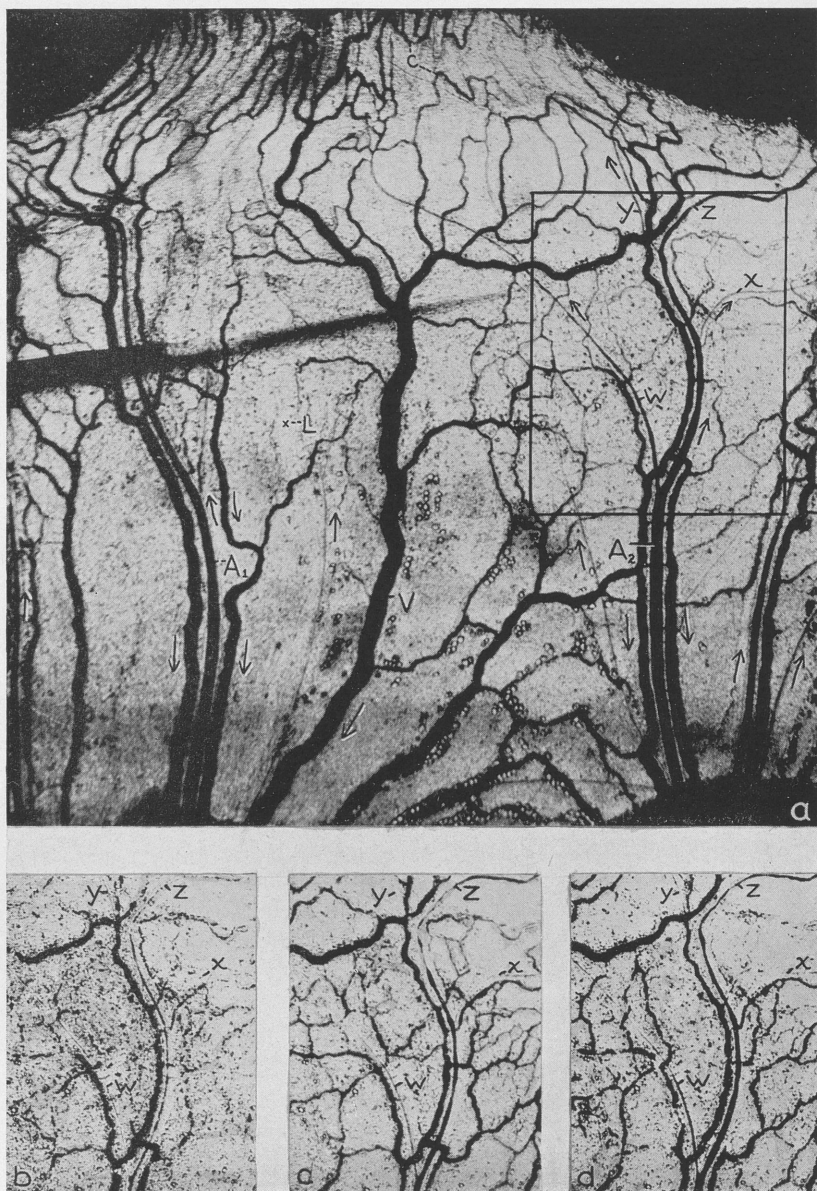


FIG. 1.

arteriole *A2* was completely contracted when this photomicrograph was taken; branches *W*, *Y*, and *Z* had begun to relax.

*Fig. 1, c*, is a photomicrograph of the same region shown in *b*, taken seven minutes later. Branch *Y* was completely contracted but branch *Z* had relaxed to some extent. Most of the vessels shown completely lacked circulation.

*Fig. 1, d*, is the same region shown in the preceding photomicrographs. This photomicrograph was taken ten minutes later than *c*. Arteriolar branches *W* and *Z* were more relaxed and a sluggish circulation was passing through *Z*. There was practically no circulation in the majority of capillaries and venules.

It will thus be seen that after the initial arteriolar contraction, if anaphylactic shock is not fatal—and it was not in these experiments—the arterioles gradually relax and the circulation begins again.

This injection did not desensitize the rabbit, and the experiment just described was repeated three more times at four day intervals, with similar results.

*Fig. 2* is a photomicrograph of the vessels in the bay of a chamber in the ear of the same rabbit as shown in *Fig. 1*, taken two minutes and forty seconds after the beginning of intravenous injection of 5 cc. of horse serum. This was the third intravenous injection of horse serum into this sensitized rabbit. The arterioles have completely contracted, thus stopping the circulation. No change in diameter of the capillaries or of small venules occurred, and no precipitation could be seen within the vessels, such as forms *in vitro* when horse serum is added to blood from a rabbit sensitized to horse serum. Enlargement,  $\times 27.4$ .

*Fig. 3* shows photomicrographs of that portion of the bay of the chamber shown in the rectangle in the upper right hand corner of *Fig. 2*. *Y* and *Z* are branches of arteriole *A2* designated by the same letters in *Figs. 1* and *2*. Enlargement,  $\times 27.4$ .

*a*. Photomicrograph taken twenty-seven days after that shown in *Fig. 2*, and one hour before the instillation of horse serum within the moat. The vessels have recovered from the previous intravenous injections and represent the normal appearance of vessels in the sensitized rabbit.

*b*. Photomicrograph taken twenty-five minutes following the



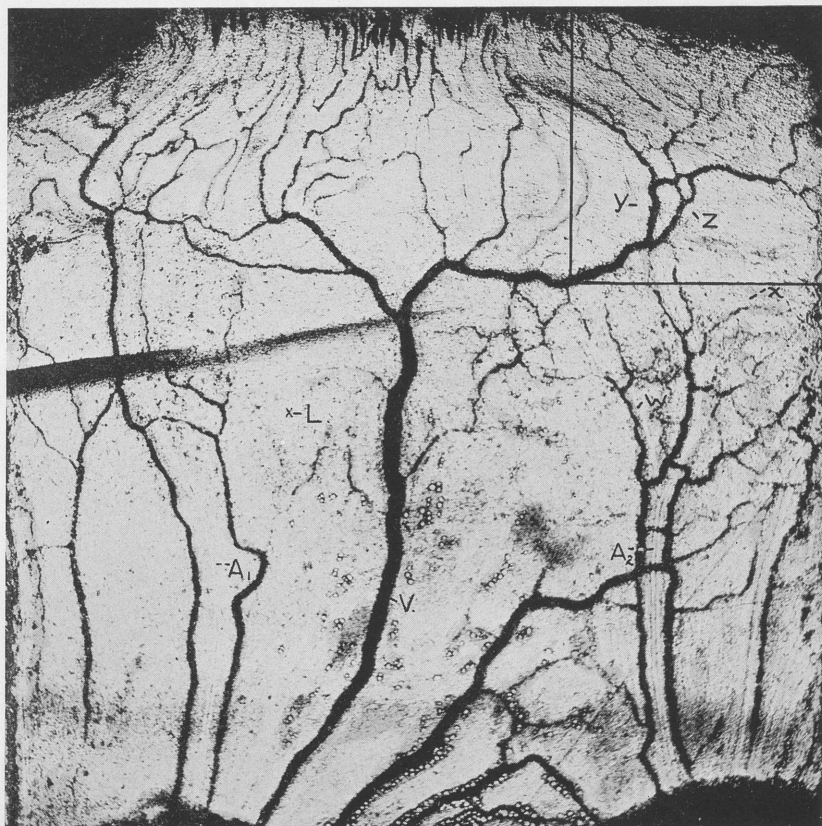


Fig. 2

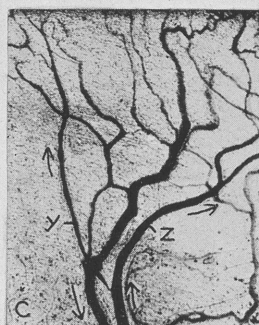
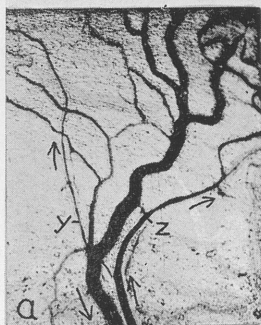


Fig. 3



instillation of horse serum within the moat. In this case, the reaction is not a general one as in the experiments just described. On the contrary, it appears to be purely local in character. There has been contraction of those parts of the arteriole closest to the moat, that is, closest to the source from which the serum is diffusing. This contraction stopped the circulation in all of the vessels shown in photograph. No contraction of the capillaries or venules occurred. The horse serum was still in the moat when this photomicrograph was taken.

*c.* Photomicrograph taken three days after that shown in *b* of this figure. The arterioles are no longer contracted but actually wider than before the serum was introduced into the moat. The circulation was full and continuous. Horse serum was still present in the moat.

Besides causing arteriolar contraction, the intravenous injection of horse serum into the sensitized rabbit, or the introduction of serum into the moat of a chamber in the ear of a sensitized rabbit, causes leukocytes in the vessels to adhere to the endothelium of the capillaries and venules and emigrate in large numbers through the walls of these vessels. The leukocytes frequently adhere to the capillaries and venules in such large numbers that they block these vessels. An example of a venule blocked by a mass of leukocytes is shown in Fig. 4, *A*. This plugging occurred following the intravenous injection of 5 cc. of horse serum into a sensitized rabbit. *A* is a drawing ocular record with an enlargement  $\times 335$ .

Fig. 4, *B* (drawing ocular record, enlargement,  $\times 335$ ) shows a second effect produced by horse serum when introduced into the sensitized rabbit. This effect is upon the leukocytes themselves. They become sticky toward each other, and adhere to each other, forming clumps which frequently plug up the capillaries and small venules. Such a clump of leukocytes is shown in a large vein at *M*.

A third effect produced by the introduction of serum into the moat of a chamber in the ear of a sensitized rabbit is emigration of leukocytes from the capillaries. Fig. 4, *C* (drawing ocular record, enlargement,  $\times 670$ ) shows leukocytes that have emigrated from the capillaries following the instillation of serum for the seventh time within the moat of a chamber of a rabbit sensitized to this antigen. The number of leukocytes that emigrated from the ves-

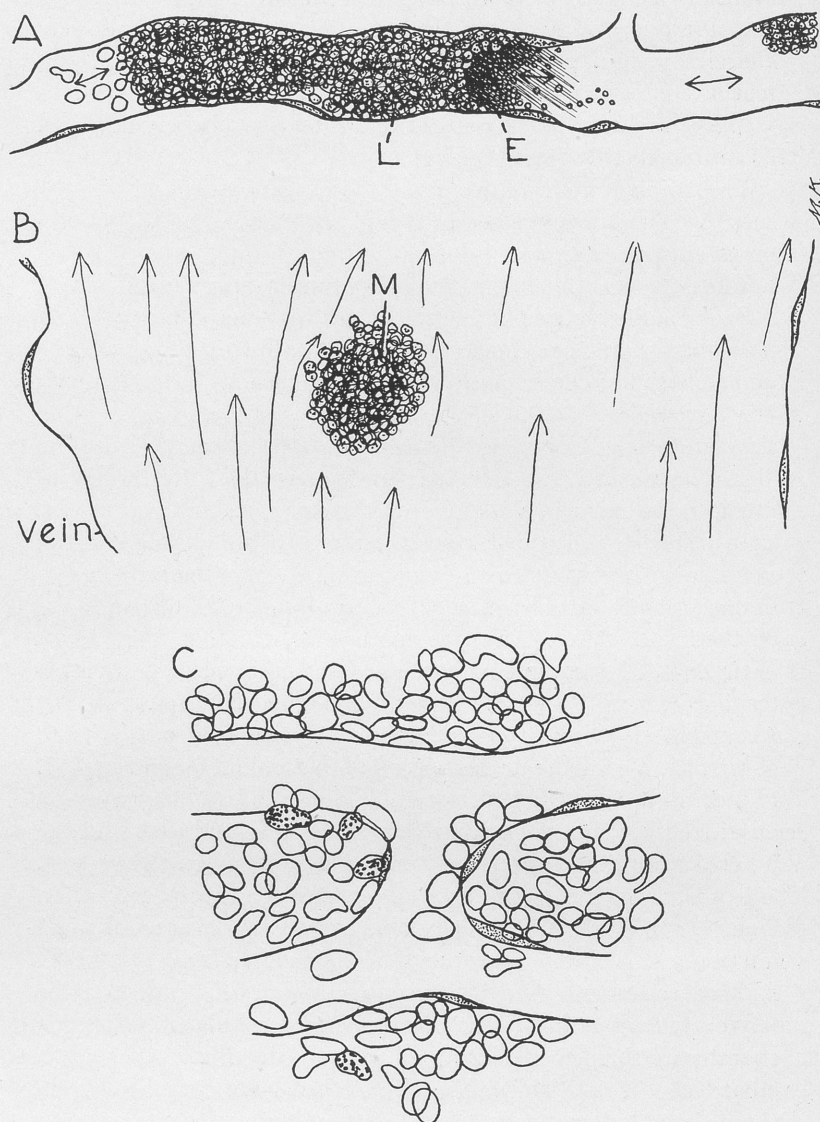


FIG. 4.

sels shown in this record was approximately 10 times as great as the number that emigrated from similarly placed capillaries in the chamber in the ear of a normal rabbit, following the initial instillation of serum within the moat.

We concluded that the injection of horse serum into the rabbit sensitized to this antigen causes:

1. Arteriolar contraction, which blocks the circulation;
2. A change in consistency of the endothelium, such that leukocytes adhere to the walls of the capillaries and venules in large numbers, and emigrate into the surrounding tissue;
3. A change in the leukocytes so that they become *sticky toward each other*, frequently occluding the capillaries and small venules, and sometimes forming leukocytic emboli;
4. No contraction of blood capillaries or of small venules occurs, and
5. The blood-vessels do not become occluded by any specific precipitates.

In order to study the reaction to a sensitizing agent which is more constant in its effects in rabbits, we have carried out preliminary experiments with fowl blood and can at this time report on the local reaction obtained in one animal.

In none of our rabbits did we produce a fatal anaphylactic response by means of horse serum alone. In 12 rabbits, receiving 1 cc. of citrated fowl blood intravenously, and four days later 2 cc. of citrated fowl blood subcutaneously, the subcutaneous injection of 2 cc. of fowl blood four days or longer after the last sensitizing dose, resulted in a fatal anaphylactic reaction in every instance. Twelve other rabbits were similarly sensitized to citrated fowl blood and were subsequently subjected to shocking doses ranging from 2 cc. to 0.1 cc. The rabbits receiving 0.25 cc. or less of citrated fowl blood as a shock dose survived; the others died promptly in an anaphylactic shock.

We then introduced citrated fowl blood into the moat of a chamber in the ear of a rabbit sensitized to fowl blood. Since the moat has a capacity of only 0.15 cc., the total amount of blood in the moat was less than the quantity necessary to produce a fatal anaphylactic response.

No change in the vessels was observed during the first half hour

following the instillation of the fowl blood within the moat. At the end of this time there had been a slight increase in adherence of leukocytes to the walls of the capillaries and venules closest to the moat. This sticking gradually increased, but at the end of one hour had only reached phase 3 of the scale proposed by Clark and Clark (1935). Six minutes later it was seen that the leukocytes had become sticky toward each other, but no large clumps of leukocytes had yet been observed. Many fewer blood cells were passing from the peripheral capillaries into the venules at this time, there being some variation in the rate of blood flow.

One hour and sixteen minutes following the introduction of the fowl blood into the moat, concentration of blood corpuscles was noted in a few of the capillaries closest to the moat. By this time there had been a decided increase in adherence of leukocytes to the walls of the peripheral capillaries and venules. Three minutes later two venules were observed to be completely occluded by leukocyte clumps.

One hour and forty-seven minutes following introduction of fowl corpuscles into the moat, a narrowing of one of the peripheral arterioles was seen.

Three hours and fifty-seven minutes after the introduction of the fowl corpuscles, adherence of leukocytes to the walls of the capillaries and venules in the entire distal one-quarter of the bay was massively phase 4. Extensive emigration was in progress, and in places large numbers of leukocytes could be seen in the tissue outside of the vessels. At this time the circulation through many of the peripheral capillaries and venules had ceased.

Five hours and twelve minutes following the instillation of the fowl corpuscles within the moat, blood corpuscles were so concentrated in several more of the capillary loops closest to the moat that the outlines of the individual cells could not be seen. Such concentration of cells was present at this time not only in several of the peripheral capillary loops, but also in two peripheral arterioles, the circulation through which had consequently ceased.

The next morning, twenty hours following the instillation of the fowl blood within the moat, concentration of blood in the peripheral vessels was extensive. Continuing proximally along the same blood-vessels that showed concentration at the periphery, pulsation



of corpuscles in non-circulating plasma could be seen. Farther proximally, that is, at a greater distance from the moat, the blood still continued to circulate through the vessels, in the most advanced of which leukocyte sticking was phase 4. Spontaneous contraction of the more peripheral of the circulating arterioles was observed, and large numbers of recently emigrated leukocytes were seen in the surrounding tissue.

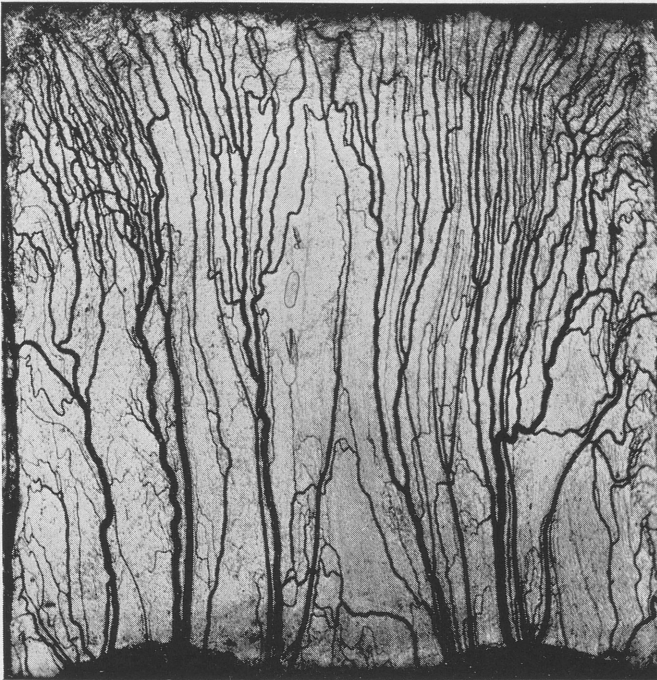


FIG. 5 A.

The concentration of blood in the peripheral vessels, following introduction of fowl blood into the moat of a chamber in the ear of a rabbit sensitized to fowl blood, evidently indicates that the permeability of such vessels has been increased. In such instances, the plasma passes through the vessel walls, leaving the corpuscles concentrated within the vessels. Numerous experiments have

shown that the introduction of Ringer-Locke solution into the moat causes no such increase in permeability in any of the vessels in the bay. Consequently the increase in permeability described in this experiment is due to the effect of the fowl blood upon the vessels.

It appears likely that the rather slow rate at which the vessels in the bay reacted to the fowl blood in the moat is due to failure



FIG. 5 B.

of substance from the fowl blood to reach the vessels more rapidly in appreciable amounts.

This experiment with fowl blood is obviously preliminary and not yet completely controlled. While sodium citrate alone has been placed in the moat and it produces no such reaction as the citrated fowl blood, it remains to test the effect of chicken blood

plasma and of laked chicken blood corpuscles in sensitized rabbits, before the question of apparent slowness of the reaction can be discussed intelligently. Furthermore citrated chicken blood has not yet been placed in the moat in non-sensitized rabbits.

*Fig. 5, a*, shows the normal appearance of blood-vessels of the rabbit sensitized to fowl blood, taken shortly before the introduction of fowl blood into the moat. *Fig. 5, b*, shows the same vessels twenty hours following the instillation of fowl blood within the moat. The fowl blood was still in the moat when this photomicrograph was taken.

(Motion picture of tissue reactions in the chamber when fowl blood was instilled in the moat of an animal sensitized to this antigen. Photography by Dr. E. A. Swenson, Department of Anatomy, University of Pennsylvania School of Medicine.)

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#### DISCUSSION

DR. HENRY M. GOODYEAR, Cincinnati, Ohio:

I should like to ask the Doctor if he has tried histaminase (histamine detoxicant) experimentally in these cases. From some of the experimental work which has been done, apparently small doses of histaminase will protect rabbits or dogs against anaphylactic reactions. I have tried it in allergic patients but it seemed to make them worse. However, I have one patient now with vasomotor disturbance, swelling of the face and arms intermittently, having been afflicted for many years, who gets perfect relief with small doses of histamine detoxicant.

I have another patient who is apparently getting relief from two 5-grain tablets a day at the beginning of an attack of migraine. The patient has had migraine for many years, and was accustomed to going to bed two or three days with nausea, vomiting and severe hemicrania.

DR. HARRY P. SCHENCK (in closing):

We have not tried histamine or histaminase. This probably looks very simple as we present it, but is a result of twelve years work on these chambers by a large group. The work was in part made possible by a grant from the Rockefeller Foundation under which Dr. Abell held a Fellowship.

We have been looking for fundamental things. This clumping of leukocytes, for instance, is something which I certainly never suspected in either anaphylaxis or allergy. It does occur in man, as we will show later, and we have tried no drugs for this reason, because we are still too ignorant of what this mechanism is and what really occurs in anaphylaxis and in allergy.

Of course we will try to solve these other problems in time, but what would be still better would be the adoption of this technique by others so that the research could be speeded up. It is not a one-man job.

To show you how handicapped we are, we are still not sure which of these cells are eosinophils, and everyone asks us right off, whether these leukocytes which emigrate out into the tissue are eosinophils. We do not know, and thus far we cannot prove that they are. We are fairly certain that most of them are, because of the size of granules in them, but we are still unable to prove it. We have not even used adrenalin in any of these experiments.



## SULPHANILAMIDE: CHEMICAL AND MICROSCOPIC STUDIES OF ITS INFLUENCE UPON INFECTED SINUSES OF RABBITS\*

BERNARD J. MC MAHON, M.D.

ACCORDING to the numerous reports which have appeared in the literature during the past year, sulphanilamide (para-aminobenzene-sulfonamide) therapy has continued to hold a prominent place in the successful treatment of pneumococcal,<sup>1</sup> meningococcal<sup>2</sup>, gonococcal<sup>3</sup> and other infections of a much more remote etiology, as well as those caused by the *Streptococcus hemolyticus*.<sup>4</sup>

The fact that the immediate toxic effects have been relatively few has been an added impetus to its more extensive administration. Some toxic reactions have been easily compensated for by the withdrawal of the drug, while others of a more serious nature such as acute hemolytic anemia<sup>5</sup> and agranulocytosis<sup>6</sup> are probably attributable to idiosyncrasies, according to Marshall and his co-workers.<sup>7</sup>

Many theories have been advanced to explain its mode of action. Domagk,<sup>8</sup> in the treatment of experimental streptococcal infections in mice, reported that the phagocytosis of the streptococci by the leucocytes was the important factor in removing the organisms from the tissues. Levaditi and Vaisman<sup>9</sup> stated that sulphanilamide interfered with capsule formation and thus rendered the streptococci more susceptible to phagocytosis. Colebrook and his co-workers<sup>10</sup> asserted that the serums of patients treated with certain of the sulphanilamide derivatives were capable of inhibiting the growth of the hemolytic streptococcus *in vitro*, and that the blood and serum of such patients also exerted a bactericidal, as well as bacteriostatic, action upon the hemolytic streptococcus.

On the other hand, Gross, Cooper and Peebles<sup>11</sup> do not believe that the phagocytic activity of the polymorphonuclear leukocytes and the monocytes is important in controlling experimental

\* From the Department of Otolaryngology, Oscar Johnson Institute, Washington University School of Medicine.

streptococcal infections in mice. Bliss and Long<sup>12</sup> state that its effect in experimental streptococcal infections in mice leads them to believe that its action is primarily one of slowing down of the rate of multiplication of the streptococci, thus permitting them to be phagocytosed by the white blood cells. They have likewise demonstrated that the leucocytes are essential, if recovery from the streptococcal infection is to be expected.

Osgood and Brownlee,<sup>13</sup> after a series of experiments in the culturing of human marrow, conclude that the major action of sulphanilamide on the beta-hemolytic streptococci seems to be a neutralization of the toxins. By decreasing the rate of cell division, while not killing the organism, sulphanilamide thus favors the more extensive phagocytic actions of the leucocytes, though it has no direct effect upon phagocytosis.

It is thus obvious from these and other excellent contributions, too numerous to mention, that the trend of thought of most investigators, relative to the action of sulphanilamide is inclined along similar channels.

Nevertheless, the empirical status of sulphanilamide has not been changed.

Marshall, Emerson and Cutting<sup>14</sup> have found that after administration of sulphanilamide to dogs, it was recovered from practically all the organs and tissues of the body in amounts varying from 3 mg. per cent in fat, to 20.7 mg. per cent in liver, with a blood sulphanilamide range of 10.2 to 19.8 mg. per cent, respectively.

No mention was made of its occurrence in nasal or sinus tissues.

*Purpose.* Our purpose in undertaking these experiments was, first, to ascertain whether or not sulphanilamide was deposited in nasal and sinus tissues of rabbits in sufficiently large amounts to theoretically affect a streptococcal infection in these tissues; secondly, to study its effect upon some tissues in which infection was not present, and upon others in which infection had been induced by the instillation of a virulent culture of hemolytic streptococcus into the paranasal sinuses.

*Method.* Healthy rabbits, weighing between 3 and 4 pounds, were selected and observed for seven to ten days for freedom from infection.

*Administration of Sulphanilamide.* Sulphanilamide was administered to each rabbit on the basis of 1.5 grams per kilogram (10 grains per pound)<sup>15</sup> for each twenty-four hours, the entire dosage being given at the same time each day.

The tablets were placed individually in the hypopharynx and crushed, after which several droppers of water were introduced into the rabbit's mouth. This method of administration was used in the sulphanilamide control group only.

In the Group II rabbits, the sulphanilamide tablets were ground in a mortar with 10 cc. of warm water, and this suspension administered through a stomach tube, followed by 5 to 10 cc. of warm water to clear the tube.

*Inoculation.* All of the Group II rabbits were infected with a twenty-four-hour broth culture of a virulent strain of *Streptococcus hemolyticus*, obtained from the heart blood of patients who had died of a *Streptococcus hemolyticus* septicemia. Five-tenths cubic centimeter of the culture was injected through the lateral membranous window directly into the sinuses on the right side, and four to nine days were allowed to elapse in order to be certain that infection was well established in these sinuses before the administration of sulphanilamide. (Charts 2, 3 and 4).

On the day on which the sulphanilamide administrations were begun, one rabbit out of each group of 6 was killed as a control to determine the extent to which the sinus infection had progressed.

The daily administration of sulphanilamide was instituted for a period of one to four days in 4 rabbits. At the end of this time, the 4 rabbits were killed, as also was one which had originally been infected, but had not received sulphanilamide, in order to obtain an idea of the uninterrupted progress of the infection in the sinuses over the entire period of the respective series.

Immediately before the rabbits which had been given sulphanilamide were killed, 10 cc. of heart blood was taken, and quantitative studies made, the modified technique of Marshall, Emerson and Cutting being followed.<sup>16</sup>

All rabbits were killed by injecting air into the hearts.

The nose was severed immediately anterior to the orbits and divided in midline along the septum. The right side, the infected side, was placed in 10 per cent formalin for twenty-four hours,

decalcified, sectioned and stained with hemotoxylin and eosin. The turbinates and sinus walls of the left side of the nose, the uninfected side, were thoroughly exenterated and weighed, this tissue was then placed in 10 cc. of alcohol in a mortar and ground with a small amount of sand until thoroughly disintegrated, after which the solution was placed in a large centrifuge tube and quantitative studies made according to the technique of Marshall, Emerson and Cutting,<sup>14</sup> as follows: "After standing overnight, the alcoholic tissue suspension was centrifuged, the alcohol poured off, and the tissue residue extracted four times with alcohol. The final volume of the alcoholic extract of the tissue was adjusted so that a total of 20 cc. of alcohol was used for each gram of tissue. Sulphanilamide was determined in the alcoholic extract by the following modification of our original alcohol method for blood.

"Ten cubic centimeters of the alcoholic extract were measured into a small flask; 5 cc. water; 2 cc. of 0.3 N HCl and 1 cc. of 0.1 per cent sodium nitrite were successively added. After three minutes 1.5 cc. of the alcoholic solution of dimethyl  $\alpha$  naphthylamine (1 to 100 cc. alcohol) were added. After five minutes, the solution was filtered and the filtrate compared in a colorimeter with a standard solution of sulphanilamide in alcohol, which had been treated in the same way as the alcoholic extract."

RESULTS. The purpose of the control group was twofold, viz.: to determine the length of time which could be permitted to elapse between doses, before the blood would be found to be free of sulphanilamide, and to ascertain whether or not there were any cytological changes in the mucosa of the nose and paranasal sinuses caused by sulphanilamide alone, in healthy uninfected rabbits, before proceeding with the administration of sulphanilamide to the infected group.

The sulphanilamide was given by mouth in this group (Group I) in order to simulate the manner of administration in humans, since we know from the reports of Marshall and his co-workers<sup>17</sup> that absorption from the intestinal tract takes place rapidly and completely.

Of the 6 rabbits, 3 died, consequently the immediate ante-mortem blood determinations could not be done. The cause of death was not determined, unless it could be attributed to the



repeated cardiac punctures and exsanguination, as 10 cc. of blood was aspirated every few days for sulphanilamide determinations. We did not feel that the deaths were caused by the sulphanilamide, inasmuch as there were no symptoms of toxicity in these, or in any of the rabbits in this group.

CHART 1.—GROUP I—SULPHANILAMIDE CONTROLS

Rabbit	Sulphanilamide administration		Elapsed time, days	Last dose before death	Sulphanilamide determinations*	
	Dosage, gr.	Total, gr.			Blood, mg. per cent	Tissue, mg. per cent
1	35	205	19	5 days Died	0.8— 19.0	0.0
2	15	75	6	24 hrs. Killed	3.7— 13.7	27.0
3	20	110	8	48 hrs. Died	2.4	8.0
4	35	130	8	24 hrs. Died	Neg.	6.0
5	35	170	15	5 hrs. Killed	0.5 36.0 42.0	27.0
6	35	165	15	5 hrs. Killed	1.6 6.0 49.0	29.0

\* The sulphanilamide determinations were done by Miss Jane Erganian, of the Washington University School of Medicine, through the courtesy of the Department of Medicine.

The wide variations in the amount of sulphanilamide found in the blood, as shown in Chart 1, were the result of two factors: the diminution of the daily dosage too far below the pro-rated weight dosage, and the permitting of more than twenty-four hours to elapse between doses. This variation in amount occurred irrespective of the total dosage over the entire period. This is consistent with the findings of Marshall and others,<sup>17</sup> viz., that the drug is rapidly eliminated, and sustained dosage must be adhered to in order to maintain an optimum blood concentration of 11 to 16 mg. per cent in humans.

A similar variation was found in the sulphanilamide content of the nasal tissue, the amount being found to be more or less, according to the elapsed time since the last dose before death.

In the Group II rabbits, the number of inoculations, the dosage and elapsed time were arbitrary. The desideratum was a well-established infection of the sinuses on the right side. This was

considered to be present when there occurred, and persisted, a definite swelling over the sinus window, with or without the presence of secretion in the right nostril. In several of the rabbits the sinuses were aspirated and the original organism recovered in pure culture.

CHART 2.—GROUP II—SERIES 1

Rabbit	Hem. strep. inoculations		Sulphanilamide administration			Day killed	Sulphanilamide determinations		Toxic signs
	No.	Days	Days	Dosage, gr.	Total, gr.		Blood, mg. per cent	Tissue, mg. per cent	
8	2	12	4	40	160	14th	38	23	Yes
11	2	12	4	40	160	14th	47	31	Yes
12	2	7	4	40	160	14th	27	9	No
13	3	4	2	40	80	6th	19	17	No
14*	2	2	Average . . . . .				30		
15†	2	7	Average . . . . .					17	

\* Early control.

† Late control.

Because of the variation in the sulphanilamide blood content in Group I, we decided that the administration of a sulphanilamide suspension with a stomach tube would be the more accurate method.

A No. 16 catheter was therefore passed, without difficulty, into the stomach and a 10 cc. syringe used to instill the proper dosage, prepared as indicated above.

CHART 3.—GROUP II—SERIES 2

Rabbit	Hem. strep. inoculations		Sulphanilamide administration			Day killed	Sulphanilamide determinations		Toxic signs
	No.	Days	Days	Dosage, gr.	Total, gr.		Blood, mg. per cent	Tissue, mg. per cent	
16	3	9	4	{40-35 30-20}	125	14th	31	17	Yes
18	3	9	1	35	35	13th	32	17	Yes
19	3	9	4	{40-35 30-20}	125	14th	17	14	Yes
21	3	9	4	{40-35 30-20}	125	14th	17	Died	No
20*	2	6	Average . . . . .				24		
17†	3	9	Average . . . . .					16	

\* Early control.

† Late control.

Having found in the control group that daily dosage was necessary in order to maintain the blood and tissue sulphanilamide

above a certain level, we administered the desired dosage to each of the Group II rabbits daily, for four successive days. Our clinical experience had suggested that the optimum initial clinical response is obtained within ninety-six hours, and consequently the cytological response should be expected within the same length of time. It was not our intention to attempt to eradicate the infection entirely, since sufficient time was not allowed, but to determine the cytological response while infection was still active, in the presence of the sulphanilamide in the tissues.

The sulphanilamide dosage was reduced 5 grains a day in Rabbits 16, 19 and 21, Series 2, because of the toxic reactions, but maintained in Series 3, despite the toxic reactions.

The total dosage in this group, as in Group I, did not directly influence the blood or tissue sulphanilamide. This is very evident in Rabbit 18, Series 2, which had received the smallest dose, but showed the highest blood and tissue concentration in the series. Evidently a maximum initial dosage supplies a comparatively large amount of sulphanilamide to the blood and tissues within twenty-four hours, in certain animals.

The sulphanilamide concentration in both blood and tissue showed a wide variation: from the lowest in the blood of 17 mg. per cent to the highest of 76 mg. per cent; and the lowest in the tissue of 9 mg. per cent to the highest of 47 mg. per cent. The tissue sulphanilamide ranged from 11 to 67 per cent lower than the blood sulphanilamide. This is probably explainable on the basis of individual susceptibilities, or a disproportion between the rate of absorption and the rate of excretion of the sulphanilamide.

CHART 4.—GROUP II—SERIES 3

Rabbit	Hem. strep. inoculations		Sulphanilamide administration			Day killed	Sulphanilamide determinations		Toxic signs
							Blood,	Tissue,	
	No.	Days	Days	Dosage, gr.	Total, gr.		mg. per cent	mg. per cent	
24	3	9	4	45	180	14th	32	18	Yes
25	3	9	4	40	160	14th	76	47	Yes
26	3	9	4	40	160	14th	74	44	Yes
27	3	9	4	40	160	14th	22	13	Yes
22*	3	9	Average		.	.	51		
23†	3	9	Average		.	.		30	

\* Early control.

† Late control.

## MICROSCOPIC FINDINGS

GROUP I. SULPHANILAMIDE CONTROL RABBITS. The epithelium and subepithelial tissue appeared normal. The glands showed no pathognomonic changes, other than the slight distention of hyperactivity. There was no exudate in the sinuses or nose.

GROUP II. INFECTED CONTROL RABBITS. The early controls showed the usual picture of an acute hemorrhagic exudate in the sinus cavity. This exudate consisted principally of red blood cells among which were interspersed moderately large numbers of monocytes, polymorphonuclear leucocytes in small numbers in some slides, in larger numbers in others, and sloughed epithelial cells. The monocytes and the epithelial cells appeared to be more actively phagocytic than the polymorphonuclear leucocytes, especially the former. The late controls, as a rule, showed a marked predominance of monocytes, epithelial cells and many histiocytes containing smaller monocytes and lymphocytes, but very few polymorphonuclear leucocytes. Large numbers of streptococci were seen in the large monocytes only and not in the polymorphonuclear leucocytes.

*Epithelium.* The epithelium varied in thickness according to the reaction, the cells were either regular in outline but engorged, with the cilia apparently intact, or greatly thickened due to vacuolization, edema or protoplasmic disintegration. In other areas it showed various degrees of fragmentation, from the extrusion of individual cells into the sinus cavity, to patchy or generalized sloughing of large areas, leaving in some instances an intact layer of basal cells, in others the unprotected subepithelial tissue.

In the late controls the epithelium showed a progression or exaggeration of the early condition, the intact epithelium being thickened by a reduplication of the epithelial cells, by monocytic infiltration, or by extreme edema.

*Subepithelial Tissue.* The most marked reaction was at the site of injection of the sinus, extending from the immediate subepithelial tissue into the deeper tissue, and consisting of edema, a moderate infiltration in some instances, and a marked invasion in others, of plasma cells, monocytes, epithelioid cells, young connective tissue cells and small numbers of polymorphonuclear leucocytes.



In the glandular areas the infiltration consisted chiefly of monocytes and plasma cells. The glands were uniformly greatly distended throughout, and in places disintegrating. In the later controls this same picture persisted, except that young and older connective tissue cells were more numerous, and some lymphocytes were present. The polymorphonuclear leucocytes were practically absent.

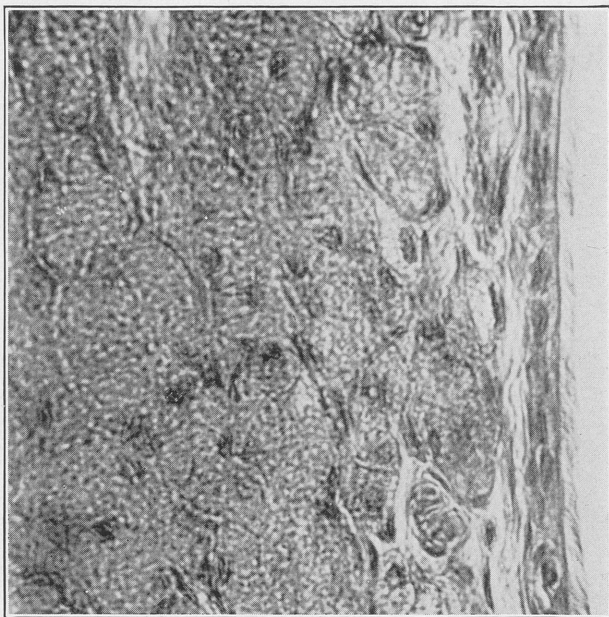


FIG. 1.—Typical picture found in normal and in sulphanilamide-treated control rabbits (Group 1). The sinus mucosa contains low columnar ciliated epithelium. There is no cellular infiltration or disintegration of the glands of the subepithelial tissue. (High power.)

*Infected Sulphanilamide Rabbits.* The tissue reactions in the infected rabbits which had been given sulphanilamide were very similar to those found in the control rabbits. The behavior of the epithelium was practically identical, and the same types and relative numbers of cells were found in the subepithelial tissue, except that there were larger numbers of free glandular cells, from ruptured glands, and of polymorphonuclear leucocytes.

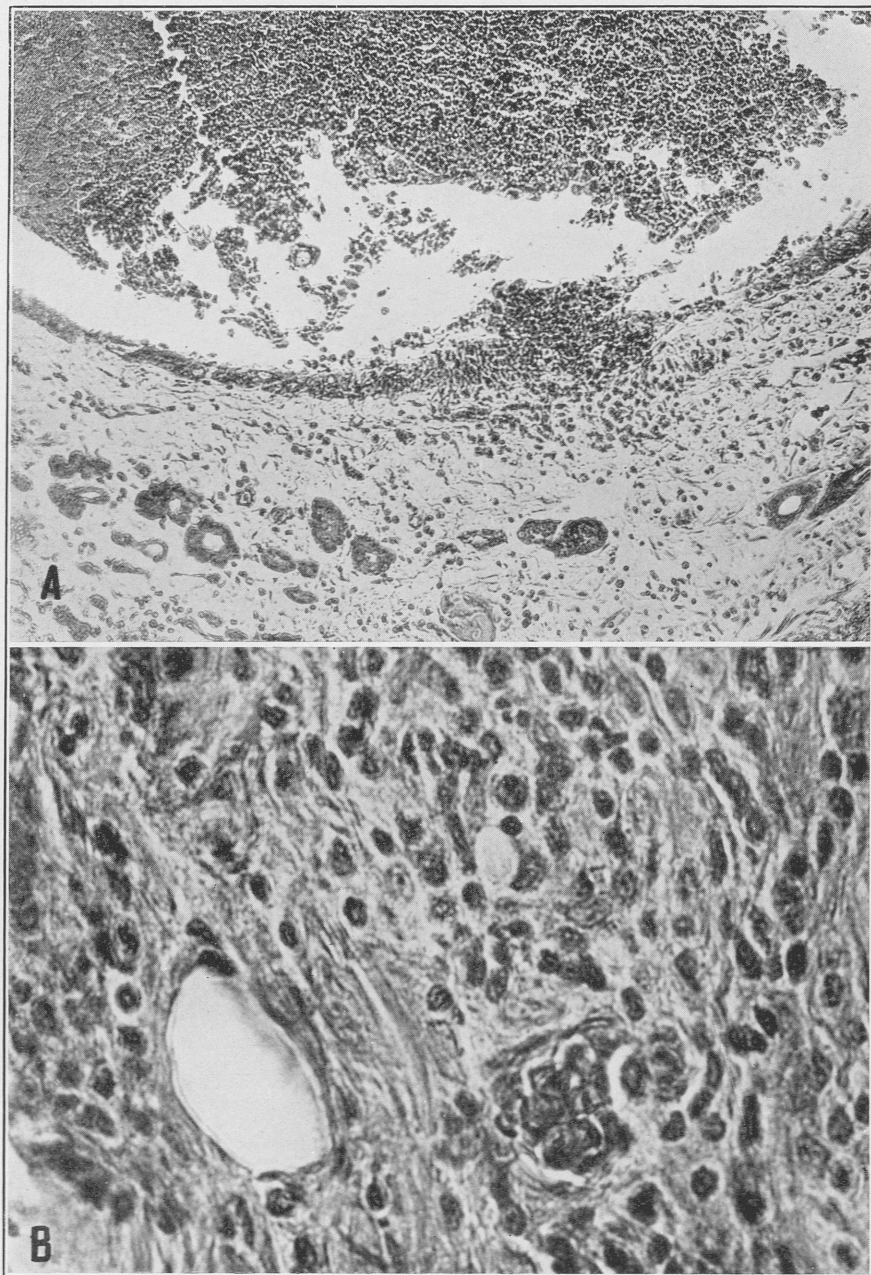


FIG. 2.

*Toxic Reactions.* The presence of toxic reactions was general, occurring in 9 of the 12, or 75 per cent of the Group II rabbits.

The following symptoms were found to be constant as to the time of onset, the character and the duration. Rapid, labored respiration, apathy, early spastic clonus of all legs, with a partial or complete paralyses of hind legs, in the Series 3 rabbits especially, with inability to stand, accompanied by straddling of forelegs. In some there was a spasm of the abdominal muscles with arching of the back. Vertigo was also present, manifested by a weaving of the head and body and falling to one side or the other, usually toward the side on which the legs were involved, especially on attempt to overthrow. There was no nystagmus.

The onset of these symptoms occurred in from two to four hours after the administration of the sulphanilamide, the duration being for about six hours, before all symptoms subsided. The subsidence was in the reverse order of the incidence.

The amount of sulphanilamide given was undoubtedly a significant factor, since the advanced symptoms occurred on the third or fourth days of administration, though the rapid respiration was frequently present after eighteen hours. Nevertheless one rabbit No. 18, Group II, Series 2, had received only 35 grains, and showed toxic signs within two hours, which lasted for six and one-half hours, strongly suggesting an idiosyncrasy, since the 3 which did not show toxic signs received three to five times as much sulphanilamide.

There were no deaths in the rabbits in which toxic signs had been noted, though 7 were killed in five hours while the symptoms were moderating, but still present.

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LEGEND FOR FIG. 2.

FIG. 2.—A, typical picture found in rabbits of Group II. The sinus mucosa is markedly edematous and infiltrated with monocytes and plasma cells and a few polymorphonuclear leucocytes; some glands are disintegrating. The sinus cavity contains an exudate composed of red blood cells, monocytes, polymorphonuclear leucocytes and sloughed cells of the ruptured epithelium of the sinus wall, in which the exudate may be seen. (Low power.) B, typical cytologic reaction found in all rabbits of Group II. The sinus mucosa contains subepithelial tissue heavily infiltrated with monocytes, plasma cells, epithelioid cells and a few polymorphonuclear leucocytes. (High power.)



The more severe and prolonged toxic reactions were present in those rabbits showing the largest amount of tissue sulphanilamide, from which we may conclude that there was an equivalent amount in the tissues of the central nervous system. This may account for the symptoms, which were definite manifestations of a marked irritation, and probably a degeneration, of certain parts of the central nervous system.

**SUMMARY.** In reviewing the findings that have presented themselves in the course of this study, we are confronted with several aspects of the influence of sulphanilamide upon rabbits, aside from our preconceived plan of study of the cytological changes in the nasal and sinus tissues, as a result of its administration.

The fact that the absorption, the dissemination and the elimination of sulphanilamide occurs with great rapidity, is borne out by the variability of the amounts recovered from the blood and nasal tissues, irrespective of the total dosage. This suggests two deductions: that sulphanilamide is not likely to give rise to chronic toxicosis, and that its field of usefulness is limited to therapeutic, and not prophylactic, administration.

The frequent occurrence and type of toxic manifestations was an interesting phenomenon. These toxic signs, which were more apparent in the rabbits which had been given the sulphanilamide at twenty-four hour intervals, did not fully develop until the third day, indicating that there was sufficient accumulation of the drug in nerve tissue to cause the paralyses. The hyperpnea was probably due to an acidosis, as described by Marshall.<sup>7</sup>

It was not possible to estimate the threshold dosage which might have caused the toxic signs, since the total dosage for four days ranged from 35 to 180 grains, with the blood sulphanilamide from 17 to 76 mg. per cent, and tissue sulphanilamide from 14 to 47 mg. per cent. This wide variation in the amount of sulphanilamide retained by the blood and tissues even for a short time when full doses were given, strongly suggests a predilection for the drug in certain animals, and probably in humans. This possibility should be borne in mind in prescribing maximum doses.

After six to nineteen days' administration of moderate doses, sulphanilamide did not cause tissue changes in any of the unaffected rabbits, the microscopic picture of the nasal and sinus tissues



resembling, in every respect, that of normal rabbits. So that, in interpreting the changes which occurred in our Group II rabbits, we were certain that they were caused by infection, alone or in combination with sulphanilamide.

The cytological reaction in the tissues of the infected control rabbits was very pronounced, showing evidence of a complete mobilization of the defensive cells, viz.: monocytes, plasma cells, polymorphonuclear leucocytes, young connective tissue cells and epithelioid cells, at the point of ingress of the infection and in the sinus and nasal cavities. There was an identical cytological response in the rabbits which had been given sulphanilamide and in which the tissue contained large amounts of the drug. We were unable to determine the presence of streptococci in these tissues, either extracellularly or intracellularly, consequently no criteria could be established relative to specific phagocytosis here.

The cells of the exudate in the sinus and nasal cavities, however, showed active phagocytosis of the streptococci. There were large numbers of organisms present in the early controls, both extracellularly and within the polymorphonuclear leucocytes to some extent, but chiefly in the monocytes. While in the late controls, there were fewer streptococci, and those practically entirely within the monocytes. In the sulphanilamide rabbits there were even smaller numbers of streptococci found. Whether this was due to the actual absence of the organisms or the presence of unrecognizable involution forms, is a matter for conjecture, as is the thought that the absence of the organisms may have been induced by the large amount of sulphanilamide in the tissues.

CONCLUSIONS. 1. Sulphanilamide administered to 6 uninfected rabbits, and 12 others whose sinuses had been infected with hemolytic streptococci, was recovered in substantial amounts from the tissues of the nose and paranasal sinuses and from the blood of practically all rabbits.

2. The reaction to sulphanilamide in the tissues of the nose and paranasal sinuses of these rabbits was not cytological.

3. The beneficial effects derived from the use of sulphanilamide in such infections must be the result of its influence upon the organism itself, or upon its toxins, in permitting more active phagocytosis by the polymorphonuclear leucocytes and monocytes.

4. Sulphanilamide should be equally as effective in the treatment of streptococcal sinusitis as of streptococcal infections elsewhere in the body.

5. The signs of sulphanilamide toxicity which occurred were, hyperpnea, apathy, vertigo and spastic and flaccid paralyses.

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## DISCUSSION

DR. BURT R. SHURLY, Detroit, Mich.:

Mr. President and Gentlemen: This problem of the therapeutic action of sulphanilamide I believe is one of the most important that has come before us in the last thirty years since antitoxin and salvarsan with its derivatives were brought out.

We have extensively used it in various hospitals in Detroit and a considerable amount of investigation has been carried on under Dr. Gordon Myers, Professor of Medicine in the Medical School at Wayne University, and Dr. E. L. Bullock of the Shurly Hospital.

I should like to show some slides and I hope those who have already seen them will be willing to look at them again.

One reason that I want to present this subject is because the Associated Press obtained my discussion in some way, and whereas I had said that 100 cases of pneumonia were treated with 6 deaths, they quoted that I had reported 1700 cases of pneumonia with 6 deaths.

(Slide.) Absorption from the gastro-intestinal tract is complete in three to five hours in dogs. It is rapid in humans. In the body it is freely diffusible in the tissues, excreted through the kidneys, partly clear and partly modified in form. Rate of clearance 22.5 cc. per minute.

(Slide.) Prerequisites for sulphanilamide administration. There are certain very important comments upon this. Sulphanilamide is being extensively sold over the drug counters throughout the country and this practice should certainly be condemned. It cannot be used like aspirin. General infection is amenable to sulphanilamide. There should be daily clinical observation for toxic symptoms and signs. A frequent hemoglobin and white blood count should be made. That is of the greatest importance. These cases should be under direct observation because the action of the drug is very rapid. Its elimination is also rapid and the toxic symptoms can occur very quickly.

It is desirable procedure that the blood sulphanilamide level should be taken at the end of twenty-four and forty-eight hours with a frequent red blood count and differential count.

(Slide.) Serious toxic manifestations are acute hemolytic anemia with jaundice or hemoglobinuria, agranulocytosis, toxic psychosis, optic neuritis and exfoliative dermatitis. We had some very extensive involvement in this group of conditions.

Toxic symptoms are lassitude, malaise, headaches, dizziness, drowsiness, anorexia, nausea, vomiting, cyanosis, acidosis, fever not attributable to infection, skin eruption, abrupt rise in white blood corpuscles, and these should be watched and guarded very carefully.

(Slide.) Contraindications: Renal insufficiency, severe anemia.

Medication contraindicated: Acetanilid, phenacetin—in fact we give no other drugs when we are giving this except bicarbonate of soda.

(Slide.) Infections in which sulphanilamide is of little or no value: *Streptococcus viridans*, non-hemolytic streptococcus, staphylococcus, syphilis, virus infections, tuberculosis.

(Slide.) Infections in which it has been reported to be of value: Typhoid fever, undulant fever, actinomycosis, certain dermatological conditions.

(Slide.) Infections in which it is of possible or probable value: Malaria, pneumococcus, pneumonia, bacteremia, empyema.

(Slide.) Use in eye, ear, nose and throat cases: Otitis media due to *Streptococcus hemolyticus* or *pneumococcus*, mastoiditis, meningitis, septicemia, brain abscess, erysipelas, nose, throat, and sinus conditions, tonsillitis, pharyngitis, due to the *Bacillus hemolyticus*, and gonorrheal ophthalmia.

We have had a number of cases with operative indications in the hospital under immediate observation, watched from hour to hour and with blood counts taken. It was possible to see these clear up in twenty-four to forty-eight hours. One of them, a very definite case of petrositis, made a perfect recovery under sulphanilamide, although we were ready to operate and an operation had already been ordered at another hospital for the day before.

(Slide.) Infections in which it is of proven value: *Bacillus hemolytic streptococcus*, meningitis, erysipelas, cellulitis, puerperal fever, septicemia, otitis media, tonsillitis, meningococcus, gonococcus, *Bacillus coli*, aerobacteria.

(Slide.) Other routes of administration: In our own cases we only used the subcutaneous and oral method. It may be given by intraspinal, intrapleural, intraperitoneal and intraarticular route.

The use of sodium bicarbonate is 30 to 60 grains with an initial massive dose: 10 to 15 grains with each subsequent dose, and that adds to the value of it and modifies the acidosis.

(Slide.) Oral dosage is of the greatest importance because in these desperate cases we want to give the maximum doses as quickly as possible, and that chart is one that has been worked out on the basis of 15 grains to 20 pounds for a patient; 90 grains initial dose; subsequently 15 grains 6 times daily. We continue the use of it throughout the night.

(Slide.) Action against the *bacillus hemolytic streptococcus*, bacteriostasis, alteration in morphology of microorganisms, increased phagocytosis by polymorphonuclear and macrophages due to reduction in repelling substances produced by bacteria, as also brought out by Dr. McMahon.

(Slide.) We have on the market the four preparations—prontysil, a proprietary preparation, soluble prontysil, also marketed under various names, sulphanilamide, and the benzyl-sulphanilamide.



(Slide.) This is a chart of one of these pneumonia cases showing the remarkable effect which this often has upon temperature. In 100 cases of pneumonia of various types, we used diathermy, sulphanilamide in full dosage, and serum as the type indicated, there were 6 deaths, taking them in routine.

It seems to me that we should have more and more thorough scientific investigation to understand the exact use of this remedy, because I believe it has a tremendous value. The dosage must be heroic for many of the cases because before one can be perfectly sure of the bacterial examination, the damage may be done. Of course the danger is that we do not guard against these toxic symptoms, that we do not use it scientifically, that it is used generally by the public and it is not given according to the proper indications. I think we have much more to learn about it, but already it is the grandest specific for certain cases that we have had for a long time.

## THE TREATMENT OF ACUTE NASAL ACCESSORY SINUS DISEASE

A. C. FURSTENBERG, M.D.

How do you treat acute infections of the nasal accessory sinuses?

Such is the question frequently asked by our students of post-graduate otolaryngology. It arises from the mass of confusion regarding this problem; first, that there is little unanimity of opinion among the members of our profession; secondly, that treatments advocated today are changed tomorrow; thirdly, that the proponents of specific therapeutic measures do not always practice what they recommend; and finally, that there is an appalling lack of sustained conviction on the subject of sinus disease in general.

Those who have expounded their own opinions have often been looked upon contemptuously by the students of different thought, so that it requires a little courage to pose one's personal views on this abstruse subject. Particularly is this true when we realize that pervasive methods of treatment, advocated by authorities on the subject, often differ to an extreme degree in the physiological principles which govern the care of acute infections.

The only generalization, therefore, which seems accurate about the treatment of acute nasal accessory sinus disease is that an inordinate variety of methods are in practice and that no specific cure, either medicinal, surgical or biological, has yet been discovered.

Perhaps I may be able to shed a little light on this subject by dealing particularly with some things that should not be done in the treatment of acute infections of the sinuses. It is true that to dwell on the negative phases of a subject is to admit one's lack of positive knowledge; nevertheless, there is much in some of our present therapeutic measures that is reprehensible, and the contention that there are things that should not be done can be supported by both clinical and laboratory experimentation.

And now let us answer the question: How do you treat acute infections of the nasal accessory sinuses? and to the answer subjoin

the clinical observations and laboratory data which, it is hoped, will lend validity to our claims.

For the past thirty years, in the Department of Otolaryngology in the University of Michigan, nothing other than most conservative measures have been employed. In the early days, Dr. R. Bishop Canfield opposed incisively the use of any operative procedures in acute inflammatory diseases of the nasal accessory sinuses, and we have not discovered any good reason for deviating from this principle. "Don't do bone work in the presence of an acute infection in the sinuses," has been a surgical axiom of long standing in our department. It is an immutable law enforced even to the extent of discouraging the practice of antrum lavage, either by needle puncture or through a cannula passed into the normal ostium in the middle meatus. The former is certain to traumatize bone, the latter probably accomplishes the same effect in 25 to 50 per cent of cases where even the skilled operator may encounter difficulty in introducing the cannula through the normal passage and occasionally perforates the thin osseous wall of the middle meatus. The avoidance of surgical measures of even the most minor proportions is a practice to which we give sedulous adherence.

But, apart from our concern about the dangers of injuring bone in the presence of an acute inflammatory disease, every effort is made to avoid traumatizing the sensitive and acutely inflamed mucous membranes. The use of irritating solutions such as the silver salts, colloidal preparations, mercurochrome, etc., is condemned and the application of a thick cotton or gauze tampon, which may do mechanical injury to the mucous membranes, is not considered an acceptable method of procedure in our clinic. No chemical agent is used which may irritate the Schneiderian membrane and no mechanical methods such as suction, tamponade or surgical interventions which may injure the linings of the nose or osseous structures are employed. Our practice, which has been in operation for many years is of such a conservative nature that we are not warranted in speaking of it as a specific method. It embraces only those orthodox principles which are more or less applicable to all forms of infections, notably: rest, promotion of

drainage, relief of pain, hydration and nutritional support for the patient.

The sinus sufferer is put to bed in order that he may avoid fatigue and exposure to rapid changes of temperature. Morphine or codeine is administered freely and whenever necessary to allay pain. There need be no apprehension about the liberal use of adequate doses of narcotics, since the illness is evanescent and sedation for the relief of pain will be required only for a period of several days. The question is frequently asked how are we going to relieve the antrum sufferer unless we irrigate his sinus? To this question we can only reply that morphine in large enough doses, that is, quantities which give the desired pharmacological action, is our best available antidote for pain. It serves its purpose well in other excruciatingly painful diseases such as angina pectoris, stone in the common duct and Dietl's crisis and it will give equally good effects in tempering the pain of an acute infection of the maxillary sinus. Moreover, it has been a frequent experience that patients with an acute nasal accessory sinus disease usually present a pansinusitis and are victims of the pain produced by retained secretions in several of the other sinuses, at the same time suffering the general reactive effects common to septic processes no matter where they may occur in the body. Obviously a local attack upon the antrum under such conditions, even if it accomplishes the purpose for which it is designed, can be expected to influence but slightly the pain and general suffering of such a widespread inflammatory process.

Heat or cold applied to the sinus regions occasionally exert a beneficial effect in relieving pain, although it is doubtful that either extreme of temperature possesses any potent values of a therapeutic nature in this disease. Steam inhalations (unmedicated) are frequently employed and ephedrine sulphate, 3 per cent solution in water (not the solutions in oil), is gently sprayed into the nose or dropped into the nasal cavities with the patient's head in the Proetz position. The latter measures are the only ones used by us for their shrinking effects upon the nasal mucous membranes.

The irritating influences of menthol, camphor or other medicinal agents in the steam and the volatile oils frequently used in com-



bination with ephedrine, are avoided for reasons which will be presented in a discussion of the experimental work offered in support of the measures above advocated. Careful attention is given to the nutritional requirements of the patient and by the administration of copious quantities of fluids: every effort is made to keep the sinus sufferer well hydrated throughout the course of his illness.

The point of the foregoing is that most conservative means of combating an acute infection in the nasal sinuses are recommended; that nothing of a specific character is advocated, other than the assiduous avoidance of any factor, chemical or mechanical, that might be harmful to mucous membrane or bone which is already taxed to the limit of its natural powers in the fight against an acute infection.

You are not content, I am sure, to accept unchallenged, all of the above assertions. It therefore becomes necessary to present evidence in support of these claims. As previously stated, the basis for our convictions are twofold: first, clinical observations, and secondly laboratory experimentation. Let us look into the former.

During the past twenty-five years many complications of acute nasal accessory sinus disease have come to our attention. They have ranged from more or less local processes such as osteomyelitis of the bones of the face and skull, to systemic conditions of varying degrees of severity exerting their influence upon the heart, kidneys or other remote structures of the body. For example, a boy, aged twelve years, came to us suffering from a diffuse osteomyelitis of the skull which became manifest ten days following an intranasal drainage of the left antrum. A doctor entered the clinic with a streptococcus septicemia which had developed two days following the needling and lavage of an acute maxillary sinus. A nurse consulted us because of symptoms of a brain abscess, which followed an attempt to enlarge the fronto-nasal duct for drainage of an acute frontal sinusitis. Another patient sought advice on account of an orbital cellulitis which promptly supervened, following a partial exenteration of an ethmoidal labyrinth which harbored an acute inflammatory process.

At first these experiences did not elicit any particular concern

on our part. They were regarded merely as the "mine-run" of complications that were prone to appear in any clinic where large numbers of upper respiratory infections are treated. However, a more careful inquiry into this situation revealed an appallingly large group of similar cases. Out of 58 cases of osteomyelitis of the skull, 42 presented a history of some previous surgical attack upon an acute nasal accessory sinus disease or an acute exacerbation of a chronic one; our records demonstrated many instances of orbital cellulitis developing subsequent to operations upon the ethmoidal labyrinth, and there appeared an amazing number of blood stream infections in patients who had been treated surgically for acute inflammatory diseases of the sinuses. But what was of greater significance to us was the observation that these complications did not occur in our hands. Without exception they were seen in individuals referred to the clinic on account of the complications from which they were suffering. It was an extraordinary fact that patients hospitalized by us in the early stages of an acute sinus disease, and subjected to our routine measures, did not number among the group which developed the serious sequelæ of acute nasal sinusitis.

To perpend this question which arose as a result of the above observations, namely, Why did not these serious complications occur in our experience? I undertook to make a brief clinical study to which I have already alluded, by which it is hoped that some of the claims for conservative treatment will be substantiated. A series of 300 cases of acute nasal accessory sinus disease was studied in the University Health Service and in the Hospital clinic. Each patient was subjected to a careful history, a clinical examination and a radiographic study of the sinuses. When the diagnosis was established as implicitly as these means will permit, the patient was hospitalized and treated in the conservative manner above described. In not a single instance was any surgical measure employed; not even needle puncture and lavage of the antrum nor irrigation of the sinus through the normal ostium. The average period of hospitalization was one week, the first five days of which the patient was strictly confined to bed. All of these patients were returned to us for check-up examinations six months to two years following their discharge from our institution. Of

these individuals, 296 were asymptomatic; apparently in perfect health and clinically and radiographically free from any evidence of chronic nasal accessory sinus disease. Four individuals of this series, ranging between the ages of eighteen and twenty-three years, presented unmistakable evidence of a chronic maxillary sinusitis. Twenty-six of the group had experienced one or more episodes of sinus disease following the attack which first brought them to our attention, 12 of whom had been treated by lavage of the antrum through a needle introduced beneath the inferior turbinate. The point that strikes most significantly is that the 4 cases of chronic maxillary sinusitis occurring in this group were found among those 12 who had been treated surgically during an attack of acute nasal accessory sinus disease subsequent to their experience in our Health Service. From this statistical study at least one fact is derived, namely, that out of 300 consecutive cases of acute nasal accessory sinus disease, 4 individuals appeared later with a chronic infection of the antrum. In each instance this observation was made in those persons who had been subjected to an admittedly minor surgical procedure.

To be sure, we must acknowledge the fallibility of statistical studies. We are open to derision if we speak succinctly of facts derived from a compilation of this character. Perhaps it is coincidence that has furnished the results of this brief study. It must be pointed out, too, that in this series we were dealing with young, healthy students ranging between the ages of seventeen and twenty-three years, and that while under our jurisdiction we were able to put them to bed and keep them confined, until complete resolution of the infective process had occurred. It would be invidious, too, not to point out that the 4 cases of chronic maxillary sinusitis occurred in individuals who had had one or more acute attacks following their first consultation with us. However, after taking into account the possibilities of error in diagnosis, the vagaries and fallacies of statistical computations, not to mention the whims and prejudices of observers, we are still forced to the conclusion that utmost conservatism as the treatment of acute nasal accessory sinusitis is worthy of avowed recommendation by the members of our profession.

In medical circles, one frequently exposes himself to ridicule

and places himself in an ignominious position by adhering rigidly to stereotyped rules. It is not my wish to leave the impression that modifications of the above therapeutic measures are not occasionally in order. There can be no doubt of the value of antrum lavage in some cases of subacute maxillary sinusitis. In using this term subacute, I refer particularly to those sinuses that continue to drain a thick, purulent discharge for a period from three to six weeks following the subsidence of the acute fulminating infection. All of us have observed the satisfactory results derived from one or more irrigations of such a diseased sinus; but here again it is to be emphasized that the process to which reference is now made involves a stage intermediary to the acute and chronic disease. I need not point out again that my text deals specifically with the early acute phases of accessory sinusitis in which we encounter pain, discharge, sepsis and an edematous, inflamed mucous membrane. For this stage of the process, we adhere sedulously to conservative principles of treatment.

Reference has already been made to the laboratory data which may, in a measure, support our claims and furnish a newer concept in the treatment of infections involving the mucous membrane tracts of the body. We speak rather vaguely of the resistance of the patient and the virulence of the infection, but little success has been attained in the study and definition of these terms. Recently, however, some inspiring work has been conducted in our Laboratory of Bacteriology under the leadership of Dr. W. J. Nungester on the effects of mucin on the virulence of pathogenic bacteria. He has produced unassailable proof that when bacteria are suspended in sterilized mucin and injected intraperitoneally, subcutaneously or into the lower air passages, they are so injurious to the animal host that it will frequently die from small sublethal doses. In other words, he has produced convincing and indisputable evidence that bacteria in the presence of mucin increase to an appreciable degree the virulence of the infection.

It would be superfluous to cite his numerous scientific observations. Suffice it to say that, in concluding one of his recent publications concerning the effect of mucin on bacteria, he makes the following comments: "The mechanism of the action is not yet understood. Mucin does not interfere with phagocytosis but does



inhibit the bactericidal properties of phagocytic cells. It enables bacteria to survive in the body of the host for longer periods without diminution in number, or it may allow the organisms to increase in number and so result in the death of the animal. The viscosity and cohesive properties of the mucin appear to be important indices to the effectiveness of mucin on bacterial infections."

In connection with this work we have undertaken to study the effect of certain chemical and mechanical stimuli upon the production of mucin in the mucous membranes of the nose. Such preparations as argyrol and mercurochrome when dropped or sprayed into the nose cause a rapid and profuse secretion of mucin. Likewise, the slightest trauma of the nasal mucous membranes, even the gentle application of a cotton tampon, dry or saturated with sterile water, will activate the secretion of mucin; and the mere needle prick of the mucous membrane is a potent stimulus in activating, perhaps in a reflex manner, the mucous glands of the Schneiderian membrane. It was interesting to observe that any kind of irritant, chemical or mechanical, excited the secretion of mucin, the quantity of which was in direct proportion to the intensity of the stimulating factor.

From our recent studies which have been in progress for a period of only sixty days, we have observed that physiological salt solution irrigated through the nasal cavities is a potent method of dispelling the mucin. The latter is freely soluble in the salt solution and is disposed most effectively by the saline irrigation. If we are willing to accept the view that mucin, in the presence of bacteria, increases the virulence of the infection, and this claim has the fiat of leaders in the field of bacteriology, then the careful employment of saline nasal irrigations in coöperative adult patients may be a therapeutic suggestion of plausible value. This recommendation should be posed, however, solely on the assumption that the physician knows how the irrigation should be given and is fully aware of its mechanical dangers when injudiciously performed.

CONCLUSIONS AND COMMENTS. 1. Our experience with acute nasal accessory sinus disease in the University Health Service, where many such conditions are seen each year, gives credence to the conviction that conservative measures suffice in the vast

majority of cases to bring about a successful resolution of the infective process.

2. The hazards of surgical attacks upon the walls of acutely infected sinuses, and risks of serious complications attending them, are herein emphasized.

3. A critical analysis of 300 consecutive cases of acute sinusitis would seem to indicate that surgical measures, even of a most conservative nature, are rarely indicated.

4. The effect of mucin upon the virulence of bacteria opens up a new and fascinating field of study. Our leaders of bacteriology have given authoritative support to the claims that when bacteria are suspended in a menstruum of mucin, they become most destructive invaders of the animal host. From these scientific observations there are, perhaps, lessons to be gained for the physician in the practice of rhinology.

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#### DISCUSSION

DR. H. I. LILLIE, Rochester, Minn.:

I also was trained by Dr. Canfield, had these same principles instilled into my philosophy of practice and I never have had occasion to deviate from them. At the clinic we, too, have had patients with serious complications come to us after having undergone some of these minor surgical procedures for relief of their symptoms.

Our method of taking care of acute paranasal sinusitis is similar to that outlined except that we do not use nasal irrigation.

We have learned that in acute rhinitis, in which apparently the paranasal sinuses are not involved, the infection, left to itself, goes through three stages: (1) the irritative, congestive or dry stage, in which sneezing and dryness are complained of; (2) the stage of marked serous secretion; and (3) the mucopurulent stage. When the mucopurulent stage is reached, marked relief of all subjective and objective signs occurs. It has been my observation that resolution takes place very rapidly after an infection of this kind reaches the mucopurulent stage. The patient should be confined to bed. Hot, wet compresses over the face and eyes, with the patient in the upright position, not lying down, semireclining at most, afford a marked degree of relief. Sufficient sedation for relief of pain is indicated. Lavage or irrigation of a sinus never is undertaken until the subacute

stage of the infection has been reached. At this stage the application of astringents to the nasal membrane, followed by moderate general suction, is beneficial. If the secretions come very rapidly, they are easily removed by the house officer with a cannula attached to the suction apparatus. General suction, producing hyperemia, is not used to clear the nostrils of accumulated secretions.

There is only one condition, I think, wherein surgical interference is justifiable and that is in one of the acute fulminating infections of the frontal or maxillary sinus, with threatening signs of serious complications. I deprecate any effort to approach through the region of the nasal frontal duct. It is my opinion that external trephination is much less hazardous in such a condition than an intranasal operation because approach through the vascular and glandular system near the cribriform plate is avoided. External trephination has been employed on several occasions and in my experience it has given quick relief without complication. In that type of paranasal sinus disease that follows swimming the relief is great and the danger much reduced.

DR. GEORGE B. WOOD, Philadelphia, Pa.:

It has been a great pleasure to listen to Dr. Furstenberg's paper this afternoon. My own experience leads me to agree with him absolutely in almost everything he has said though there is a certain type of case in which conservative treatment is a very difficult program to carry out, and I believe that in those cases of acute fulminating streptococcic sinusitis, especially frontal, operation is at times forced upon us. However, even in these cases I am not positive in my own mind what is the best procedure.

For instance, a nurse at the Howard Hospital in Philadelphia, developed a frontal sinusitis following bathing in a city pool. I saw her three days after the attack began when her eye was swollen shut with edema extending onto the forehead. She was treated conservatively for four days, her temperature ranging up to 104 with increasing edema and marked proptosis of the eye. The frontal sinus was entered through the external wall, and we found the bone of this wall already sweating pus, with pus under pressure in the sinus. She developed osteomyelitis of the skull and died several months later of a brain abscess.

Another nurse at the Graduate Hospital with a rapidly progressing orbital cellulitis, edema of the skin of the forehead, severe pain, and rising temperature again developed osteomyelitis and finally died of brain abscess a year or so later. In this case, after consultation with

Dr. Coates and other members of the staff, the sinus was drained by a small opening through the floor and the insertion of a rubber drainage tube.

You can understand that I am still in a quandary as to what to do with this type of case and I wish someone would enlighten me.

DR. ARTHUR W. PROETZ, St. Louis, Mo.:

I am in complete accord with everything Dr. Furstenberg has said but I should like to ask him a question. Will he elucidate a little further the experiments with bacteria suspended in mucin? I understood him to say that bacteria penetrate the nasal mucosa more easily when they are suspended in mucin, the inference being that mucin is favorable to infection rather than otherwise. It seems a little incongruous that nasal mucus should be rich in a substance which is inimical to defense against bacteria.

Further, washing the nose does not rid it of mucin. It is only a matter of minutes before the glands have supplied a fresh coat. The mucus coat is necessary for the activity of the cilia and serves as a conveyor belt to transport foreign material to the pharynx. The mucin content, normally about 3 per cent, has a direct and important influence on the viscosity of the secretion, which directly determines the effectiveness of the cilia. Too great a viscosity increases the load; too little interrupts the continuity and the tenacity. In either case we have interfered with that machinery which Nature has set up to keep the nose clean.

I should like particularly to know about the experiments regarding the effect of mucin on penetrability.

DR. HENRY L. SWAIN, New Haven, Conn.:

The two previous speakers have taken some of my fire.

In the first place, after much discussion in the past two or three days of gross surgical procedure, including major removal of the larynx, it seems good to have someone, with the courage of his convictions, come before this distinguished body and present the conservative attitude which Dr. Furstenberg has. I am particularly delighted that he did it.

However, those of us who were fortunate enough at the time of the Detroit meeting to visit Ann Arbor and see the delightful environment in which his work lies, cannot help wondering whether or not his conservatism may be governed by such life. While I do not mean this in any narrow sense, we must bear in mind that his is a University



setting, in a delightful village, moulded after New England ideals, and it is vastly different to treat sinus diseases in a group of people there, than in one from the stockyards of Chicago or the dock hands of Boston and New York. However, the general principles involved are tremendously important and particularly true.

I wish all physicians could see the living ciliated epithelium working, as Dr. Proetz has presented in one of his demonstrations—that beautiful soft wave of ciliated action seen in the trachea of the rabbit or cat, whichever it may be. This would be a wonderful help in mapping out the treatment.

It is really painful to see some clinical work upon the nose with cotton applicators roughly forced through the nose and each time knocking off ciliated epithelium. The protection of the ciliated epithelium and beautiful layer of mucous membrane is lost and all such rough procedures traumatize our most vital protection.

It is very gratifying to have these milder procedures again brought to our attention because we easily forget. It is often necessary to do something besides using simple salt solution, a favorite method of mine though not used as an irrigation. My experience in that, confirms the opinion of Dr. Furstenberg.

We are not speaking of this from pride of performance but we note as did he the greater number of complications among those who are most radical in their treatment in the nose, and some of us perhaps from heredity, dislike unnecessarily heavy-handed methods.

My practice is in the little town called New Haven. We have large University groups with three or four thousand students scattered over the city and the irresponsibility of such a group of young men, Dr. Furstenberg shares at Ann Arbor. Unless his group occasionally gets over into the big city of Detroit and meets harmful city influence, they live a quiet, protected, comfortable life, full of natural vigor. In my little pilgrimage through life, I have had official connection with a school near New Haven, where the students live together and live a uniform life. For these, one can prescribe differently than for the more wandering group of a large University. Then there is the rougher type that comes up the Sound in canal boats, and they present a different problem, which we try to meet.

While all these things must be taken into consideration, it is delightful to have Dr. Furstenberg speak to us in a kindly, sweet way, like a cooling spring in a parched and thirsty land, and we are delighted that he did so.

DR. ROBERT F. RIDPATH, Philadelphia, Pa.:

I was very much pleased with Dr. Furstenberg's presentation. I feel that the subject of acute sinusitis, however, must be differentiated from the subacute and, of course, the chronic. When we mention acute sinus infections, are we speaking of an acute sinus infection *per se*, or are we speaking of the ordinary coryza, cold, etc.? All colds, as we understand them, are potential sinus infections, because the mucous membrane of the sinus is going through the same pathological phenomena caused by infection that we have in the nasal membrane. First, we have a shock to the system; second, a congestion. Dr. Furstenberg has brought out a very good point in regard to this second stage in which we have our congestion to avoid any operative measures. Why? Because in the congestive stage the membrane is so swollen, in the majority of cases, that the sinus is entirely obliterated, and of course if you operate at this period, you are certainly doing the patient a decided injury.

The third stage, of course, is the outpouring of the secretion.

First, the shock; second, the congestion; third, the outpouring of the secretion, and fourth, of course, the resolution or the membrane—coming back to its normal stage.

I should like to ask Dr. Furstenberg the question, is *x*-ray or deep *x*-ray therapy of any value in the acute stage of a sinus infection? Second, does he use infra-red light, either to the face, the sinuses, at this time, or does he use it to the back of the neck? Third, were not our grandmothers and our mothers very, very wise in trying to bring congestion from the upper respiratory tract or the head by keeping heat to the feet or to the extremities of the body in all of these acute congestions?

DR. BURT R. SHURLY, Detroit, Mich.:

I wanted to add just a word lest the memory of such departed ones as Dr. Stucky should be forgotten. When we deal with acute inflammation in the nose and throat, I trust that the younger generation will not forget the great value of a good dose of calomel and salts to the lymphatic system.

DR. THOMAS E. CARMODY, Denver, Colo.:

Mr. Chairman and Friends: It seems that Dr. Furstenberg and Dr. Lillie have not had the results that some of the rest of us have had in order to teach them. They had a good teacher. Some of the rest of us have opened up some of these sinuses with dire results.

I remember a case that I opened up about twenty years ago. I did an antrum lavage in a streptococcic case and the patient died. It taught me a very good lesson. In talking with a number of my friends, I found they had had the same results. We also know that the general surgeon has had that result in operating on acute tubes and in acute infections generally. We should leave them alone.

The treatments that Dr. Furstenberg and Dr. Lillie outline are all very good. I do not use ephedrine; I use epinine instead, 1 to 800 dilution, and I feel that it is very much better. I do not use the Proetz method, although I use a modification of it, having the patient lean forward, because they can do it very much easier. If they lean forward far enough so that the top of their head is parallel to the floor, they can drop the solution in and contract down the tissues.

I do not believe in packing with argyrol and a lot of those things, because I believe that you excoriate the membrane much more than is necessary. I do not use the saline irrigation that Dr. Furstenberg has recommended, although I think it can be used under the circumstances that he speaks of, but it is too likely to be used by people who do not understand it, and sometimes used in children.

I have for many years used only the steam inhalation, not using anything with it. A great many people, of course, like to add benzoin or something else, and sometimes there is no objection to adding something to it because it only gives the odor in the air. The main thing is the great amount of moisture in the air and giving these patients a great deal of water, one way or another, and sometimes of course giving them saline by the vein in order to have a large amount of secretion.

I am not familiar with the experiment Dr. Furstenberg has outlined, but I think there is no question but that it may be of help to us.

I have always been against probing the nasal frontal duct because I have seen some bad results from this. I have had 3 or 4 cases like Dr. Lillie has spoken of, one following swimming, and opening just simply by trephining has relieved these. We have known, of course, of many cases of ethmoidal infection that have died following operation, but if we simply make a small trephine opening in order to relieve the pressure, and prevent it going the other way, we may help some of these cases.

DR. A. C. FURSTENBERG (in closing):

I merely rise, ladies and gentlemen, to thank you very kindly for this splendid discussion. Dr. Lillie has spoken about the nasal irrigation, and I remember at one time he questioned its value in upper

respiratory infections. I must add, too, that up to the present time I have not advocated nasal irrigations. I have merely mentioned the possible effect of salt solution upon an accumulation of mucin in the nose and suggested that it may have some value in the treatment of sinus disease.

Dr. Wood has spoken about the fulminating type of frontal sinusitis. By that term, I visualize a frontal sinus infection associated with congestion and swelling of soft tissue over the frontal bone and in and about the orbit.

When such a condition occurs, is it not due to a perforation of the osseous wall of the frontal sinus (frequently the frontal plate of the frontal bone) and the production of an inflammatory process in soft tissue? If it is the result of such a pathological change, is it not possible to drain the abscess, merely by an incision through the soft tissue? Avoid bone work if it is possible to do so. Avoid anything that will stir up a green infection in bone. It has been my experience that these patients have gotten along satisfactorily after simple drainage of the soft tissue. Wait several weeks before doing bone work and, much to your surprise and gratification, many of these patients will get well spontaneously. Others, of course, will require more radical procedures later.

Dr. Proetz has asked about the experimental work with mucin. I have seen much of this in progress, but the one experiment that impressed me more than any other was as follows: Rats were placed in a box containing normal air and pneumococci sprayed into the container. Fourteen animals were employed in this experiment but none of them developed pneumonia. When, however, the pneumococci were sprayed into the box in the presence of mucin, all of the rats developed pneumonia. This experiment has been done repeatedly and I believe it convinces one that mucin has something to do with increasing the virulence of infection. Whether it is cohesive action, phagocytic action or what not, we are not prepared to say, although many bacteriologists will agree at the present time that mucin does have something to do by way of increasing the virulence of infection.

Dr. Ridpath asked about the value of *x*-ray in the treatment of acute nasal sinus disease. Our meager experience over a period of about two years has resulted in many disappointments. None of our observations to date offer any scientific evidence that this treatment is of real service. I have serious doubts that *x*-ray therapy has any great value in the treatment of acute nasal accessory sinusitis.



## CHORDOMA: WITH REPORT OF TWO CASES

ROBERT F. RIDPATH, M.D.

CHORDOMA is a rare and usually fatal tumor which arises from the fetal notochord. This structure is a cord of cells around which the vertebral column develops, regularly persisting in infants in the centers of the intervertebral disks. Portions of chorda tissue commonly continue to be present at the base of the skull and in the coccyx, this tissue gives origin to certain small tumors which Virchow described as "ecchordosis spheeno-occipitalis."

As the cells multiply they become vacuolated and contain a mucin-like substance. The walls of these cells break and a syncytium forms in a gelatinous matrix. As the vertebræ develop, the notochord becomes segmented. The intravertebral portion disappears except for rare "chorde reste," and the intervertebral portion remains as the nucleus pulposus of the intervertebral disks. In spite of the fact that notochordal remnants are present in the nucleus pulposus of all intervertebral disks throughout life, very few chordomas have been found between the base of the skull and the sacrum.

Small masses of soft, jelly-like tissue arising from the clivus blumenbachii (the sloping surface between the sella and foramen magnum composed of the sphenoid and occipital bones) were described by Luschka in 1856. Virchow, elaborating upon the subject, attributed these to degeneration of the cartilage and gave them the name "ecchondrosis physaliphora." H. Muller was the first to ascribe their origin to notochordal tissue. He showed that the notochord extended to the spheeno-occipital synchondrosis and that "nests" of tissue could be demonstrated in the basilar cartilage. These were similar in structure to the nucleus pulposus of the intervertebral disks. In 1894, Ribbert gave the tumor the name chordoma. He found chorda remnants in the bony tissue beneath the dura mater of the clivus and held that they produced tumors only when misplaced and lying outside the bony encasement.

Recently, several excellent reviews have served to direct atten-

tion to this interesting group of tumors. A chordoma is a neoplasm of notochordal origin. It arises from the embryonic remains of the chorda dorsalis, the primitive axial skeleton of a vertebrate, which is derived from the entoderm. Such cells may escape encasement by cartilage during development or conceivably as a result of trauma. Theoretically a chordoma may be produced at any point along the axial skeleton where notochordal cells are found.

The chordomas have been classified as (1) cranial, (2) vertebral and (3) sacro-coccygeal. They have been reported as occurring in various locations in the following order of frequency: in the sacro-coccygeal region, in 91 cases; in the spheno-occipital region in 52 cases; in the cervical portion of the spine, in 12 cases; and in other locations rarely.

Of the 52 reported cases of spheno-occipital chordoma, 10 of those being naso-pharyngeal, only 12 have been reported in the American literature. However, that is probably due to the laxity on the part of we Americans, as I know of 5 in Philadelphia which have not been reported. In each of the locations mentioned the tumor presents a fairly constant symptomatology.

As we are considering these tumors primarily from the nose and throat standpoint, we will go a little more into the detail of that aspect, taking up first the embryology.

The developing notochord extends as far forward as the bucco-pharyngeal membrane, and temporarily remains attached to its caudal wall as the hypophyseal pocket forms. The chorda makes a single large sigmoid curve in the midsagittal plane of the basilar plate of the skull, lying near the dorsal surface of the cartilage at three points: in the hypophyseal fossa, a short distance caudad to the fossa and anterior to the foramen magnum. It is near the ventral surface midway between the hypophyseal fossa and the foramen magnum. As the base of the skull is molded, the posterior end of the cranial part of the chorda is forced backward and dorsad, so that it lies on the occipital plate in a dorsal groove anterior to the foramen magnum. The anterior extremity is forced forward so as to lie between the cartilage and perichondrium of the dorsum sellæ. The middle part comes to lie between the cartilaginous occipital plate and the dorsal wall of the pharynx. This middle

part often presents a number of kinks, branches and irregularities. Frequently it is embedded in the retropharyngeal tissues, and its cells may intermingle with the epithelial cells of the pharynx.

As far as incidence of these tumors is concerned, the majority caused the first symptoms in the third, fourth and fifth decades of life. The average age was thirty-six years. The youngest patient was one day, and the oldest eighty-two years. The tumor was found more frequently in males than in females, in the ratio of

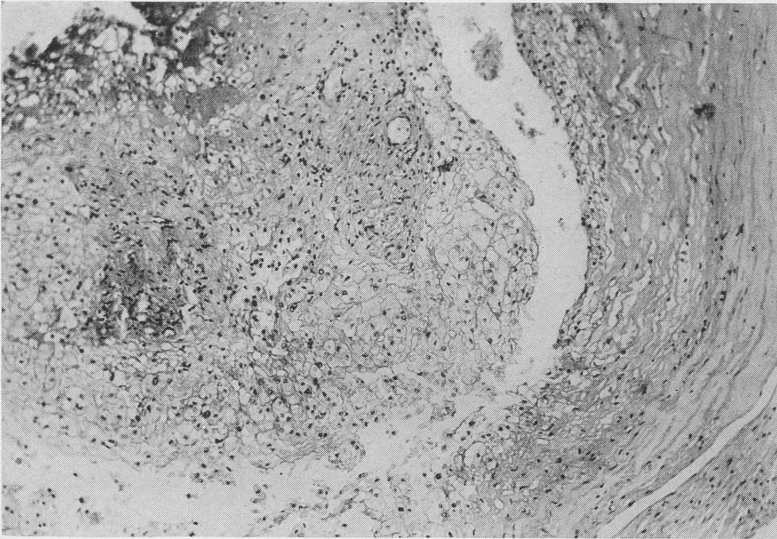


FIG. 1.—Spheno-occipital chordoma, low power, 100 diameters. Note just to the right of the center, large vacuolated cells resembling cartilage cells. They differ from cartilage in the absence of inter-cellular substance.

3 to 2. The factors which excited neoplastic proliferation were unknown, but the frequency of the history of trauma deserved emphasis.

The segregation of benign chordomas from malignant chordomas as a rule is not difficult. In typical instances the benign tumors are attached to a small median exostosis of the clivus by a narrow stalk which perforates the dura. The stalk is not always found, and in these instances the tumor lies in the subdural space or nestles beneath the dura against the bone of the clivus. These benign

tumors are relatively common, being present in 2 per cent of autopsies, according to European authors. The malignant tumors vary in size from small (2 to 3 cm.) to massive neoplasms, the largest of which was 11 by 7 by 6 cm. In form they are pedunculate, mushroom-like, globular or diffuse. On the clivus they are generally covered by dura and in the nasopharynx are limited by the mucosa. The mucous membrane of the nasal fossa, paranasal sinuses and pharynx is seen elevated in a characteristic bulging manner by the slowly infiltrative, expanding growth.

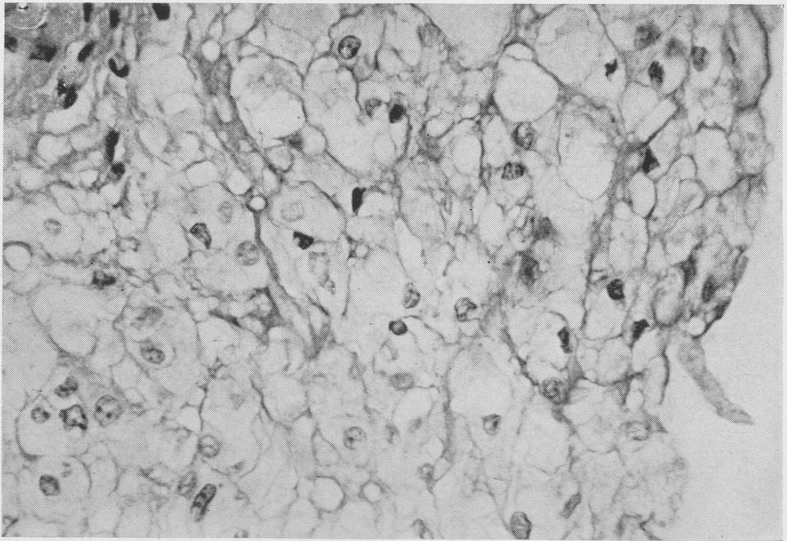


FIG. 2.—Spheno-occipital chordoma, magnified 450 diameters. Area referred to in Fig. 1. Note large vacuolated cells containing mucinous material.

The majority of cranial chorda masses arise from the clivus in the region of the spheno-occipital synchondrosis. The typical ventral extension is in the direction of the nasopharynx. It is common for the basilar plate to be infiltrated and partially destroyed. Somewhat less frequently a smooth, round mound of tumor elevates the mucosa in the midline of the posterior epipharyngeal wall. These growths extend for variable distances anteriorly and caudally, although, as a rule, are restricted to the vault of the epi-



pharynx. In exceptional instances tumor tissue presses through the posterior choana into the nasal fossæ and even into the maxillary antra. Contrary to expectations the volume or extent of the nasopharyngeal growths does not parallel the size of the intracranial neoplasm. The regional petrous portions of the temporal bones, the ethmoid sinuses as far forward as the crista galli, and the osseous walls of the orbital cavities are occasionally involved.

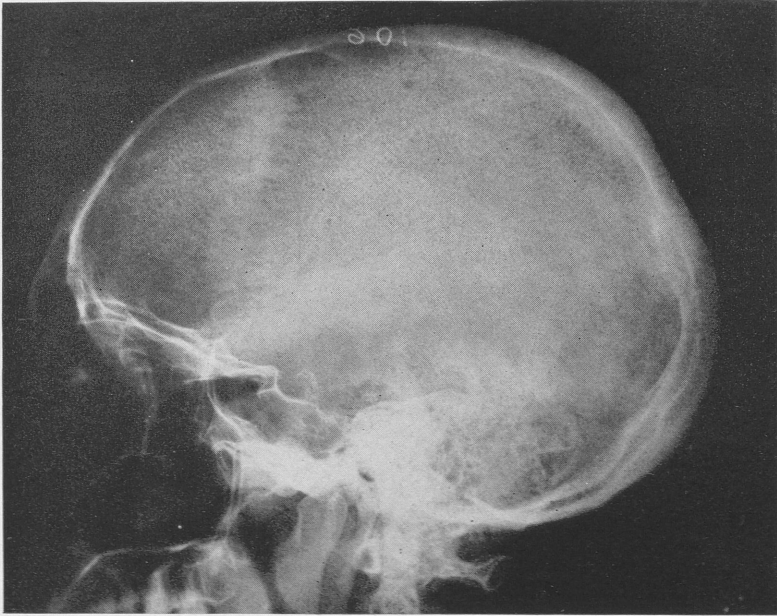


FIG. 3.—“There is a very large tumor in the nasopharynx. This is continuous with a soft tissue mass which fills about 80 per cent of the sphenoidal sinuses. There is a definite defect in the basilar process of the occipital bone in the neighborhood of the clivus.” (*X-ray report of Dr. L. J.*)

It is fairly characteristic of this widespread anterior growth to be associated by ventral invasion into the walls of the nasal fossa and into the superior maxillary bones, with the tumor encroaching on the nasal cavities, the maxillary antra and the soft tissues of the face.

The histologic picture is characteristic, but chordoma must be differentiated from two common types of tumor, myxochondroma

and colloid carcinoma. Pathologically variable pictures are presented in the different phases of chordoma. The early structure resembles hyaline cartilage, but without intercellular substance. The cells are large, rounded, vacuolated, and hyperchromatic, and may even resemble epithelium. An alveolar structure is seen in more active cases. Much mucinous material may distend the cells and infiltrate the invaded tissues. Syncytial masses of vacuolated cytoplasm may be prominent.

The symptomatology from the rhinolaryngological viewpoint is fairly constant. The nasopharyngeal symptoms, although they accompany or follow the onset of headaches and visual disturbances may be the first evidence of the disease. As a result of the obstruction of the nasopharyngeal passages by the tumor, difficulty in nasal breathing, nasal voice and mucoid discharge from the nose develop—this with nasal bleeding. Involvement of the sinuses and loss of weight are constant factors.

In the differential diagnosis we rely almost solely on the aid of roentgen examination and biopsy. However, in certain cases, a proper analysis of the signs and symptoms should lead to a surprising number of accurate diagnoses. In recapitulation, the salient clinical features may be summarized as follows: a gradual onset of nasopharyngeal and neurologic symptoms in middle-aged patients; the presence of an obstructing, midline, nasopharyngeal tumor; progressive bilateral palsies of the cranial nerves, which may be quite widespread with little or no evidence of intracranial hypertension; sensory as well as motor manifestations, and symptoms arising from compression of the brain stem or the upper cervical portion of the spinal cord.

It would seem wise to enumerate the common mistakes and to dwell briefly on a few of the more important points of differential diagnosis. There are many good reasons for confusing a chordoma with a craniopharyngioma (tumor of Rathke's cleft and hypophyseal stalk). The manifestations referable to the cranial nerves, especially the defects in the visual fields and the roentgenographic changes may be identical. There may even be supracellar calcification, such as is common in a craniopharyngioma. The adenomas and carcinomas of the hypophysis may destroy a considerable amount of bone, and in order to complete the imitation of a chor-

doma they may grow into the nasopharynx. Therefore, one distinct point of difference between the anterior group of chordomas and the hypophyseal tumors deserves emphasis. In the former there is little or no evidence of hypophyseal dysfunction. As craniopharyngiomas usually occur in children, chordomas are less frequently confused with these tumors. Parasellar meningiomas, although they rarely extend into the nasopharynx, should be considered in the differential diagnosis.

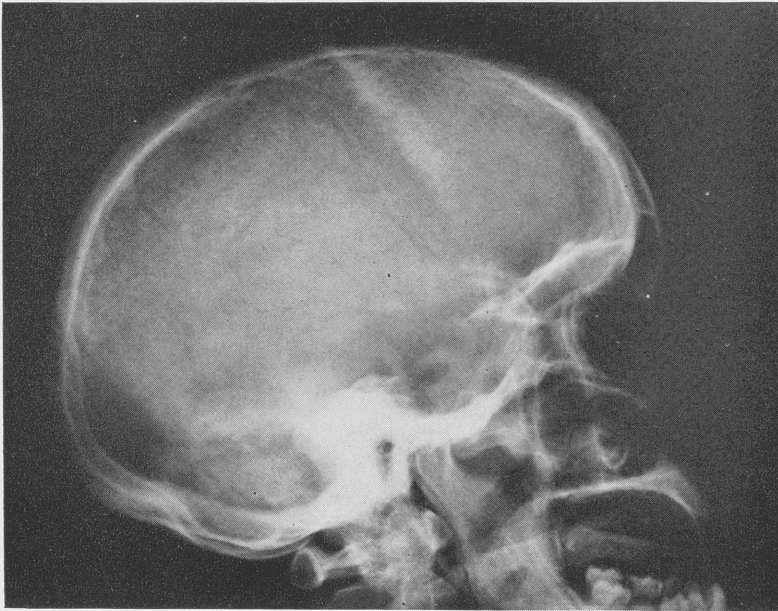


FIG. 4.—“The sella turcica has been practically destroyed. The body of the sphenoid bone and the entire basilar process of the occipital bone have been completely destroyed. Ventrally the destruction involves the ethmoid mass. The floors of the middle fossæ are destroyed mesially. There is absolutely no suggestion of a sphenoid sinus.” (*X-ray report of Mrs. A. B.*)

Chordomas which involve the nasopharyngeal passages almost invariably are overlooked or are considered as one of the more common nasopharyngeal tumors. The rare juvenile nasopharyngeal fibroma may arise in the same situations and may grow in much the same manner as a chordoma. It may extend into the

cranial cavity and cause various neurologic symptoms. However, these tumors develop before the twenty-fifth year. There is a tendency for the tumor to disappear spontaneously, chiefly toward the end of the second decade of life. Atypical chondromas, chondrosarcomas and myxosarcomas in this region may offer diagnostic difficulties not only to the clinician but also to the roentgenologist and pathologist. Other local tumors such as pharyngeal dermoid cysts and teratomas usually occur in infants and children. Although a primary carcinoma of the nasopharynx may invade the base of the skull and involve the cranial nerves, metastases are almost invariably found in the cervical lymph glands.

As regards prognosis and treatment, little offer of hope can be promised; chordomas invariably prove fatal in all instances in which the patients are under observation for a long period. The prognosis of so-called nasopharyngeal tumors is variable, depending largely on the presence or absence of cranial involvement, or involvement of the spinal cord. One patient, in the literature, had a history of difficult breathing through his nose for seven years. A tumor projecting from the posterior wall of the nasopharynx was removed. It recurred a few months later. A second operation was performed four and a half years after the first. Five months later the patient was well. In a similar instance the patient returned with a recurrence four and a half years later.

It seems that operation, as a rule, offers only temporary amelioration of symptoms. Yet it is possible that pedunculated, encapsulated, non-invasive tumors may be completely eradicated by excision. Supplementary roentgen and radium treatment are of no established benefit.

The first case I wish to report is:

Dr. L. J., aged thirty-eight years, white, a Haitian, who entered the Temple University Hospital November 16, 1936. His chief complaints were: occipital headaches for the past three years; nasopharyngeal catarrh with muco-purulent and bloody discharge, and more or less nasal respiratory insufficiency. There was some dyspnea at times. Coryza two months ago. He had lost 12 pounds in the past two months. The family history was negative. The past medical history reveals that under local anesthesia, on September 16, 1936, he was operated upon for fibrous adenoids. There is also a history of



a "flying piece of iron" causing a considerable bruise and cut in the scalp some years previously. The present medical history dates to about three years ago when he noticed some impairment of hearing on the right side and consulted a physician, who found nothing. Three months ago severe headaches radiating to the back of the head over the occipital region became very pronounced. There was also a bloody postnasal discharge. The headaches were of such severity that he was unable to eat or sleep and at this time became cognizant of beginning loss of weight.

He returned to his physician, who found by posterior rhinoscopy a large hard tumor which did not bleed on palpation. He entered the hospital and this growth was removed and he returned to his practice after a period of four days.

Three weeks later the bloody discharge and frequent epistaxis returned, and examination revealed a hard tumor mass in the same region with considerable ulceration in the upper portion. No enlargement of the glands was found. On October 16th another operation under local anesthesia was performed and for fifteen days the patient was free from symptoms.

However, after a short period the severe headaches and bloody discharges were more severe than before. In consultation with his physician he decided to consult Dr. Jackson at the Temple University Hospital. It is remarkable that up to this time no *x-ray* or laboratory studies were made. Dr. Jackson kindly referred the patient to me, and my examination on November 18, 1936, was as follows:

"There is quite a large tumefied mass in the meso- and epipharyngeal space, more on the right side and toward the midline than on the left. Postrhinoscopic view shows that this mass extends to the tip of the choana and is associated with the base of the sphenoidal sinus, and is hard and immobile. Nasal examination shows an infection of the sphenoidal cavity with some bloody, tenacious pus coming from the ostium. The mucous membrane in this area is decidedly inflamed and granular. The particular kind of growth I am unable to designate, but am inclined to think of chordoma, as it appears to start from the base of the sphenoidal sinus, or at least from near this area, and probably extends downward and upward into the sphenoidal cavity." A biopsy taken at this time revealed a typical picture of nasopharyngeal chordoma.

Dr. Chamberlin's *x-ray* report was as follows: "There is a very large tumor in the nasopharynx, as seen clinically, and this appears to be continuous with a soft tissue mass which fills about 80 per cent of the sphenoidal sinuses, leaving only a little air in the ventral and

cephalic corner of the sphenoid sinuses. The other sinuses present a normal x-ray appearance. There is a definite defect in the basilar process of the occipital bone in the neighborhood of the clivus.

The blood examination showed: red blood cells, 4,000,000; white blood cells, 11,400; hemoglobin, 76 per cent.

The urine examination was negative with the exception of a slight trace of albumin.

The Wassermann was negative.

The second case, Mrs. A. B., white, forty-two years old, was admitted to the Temple University Hospital on December 30, 1934. Her chief complaints were headache and loss of vision. The family history is negative. Past medical history is irrelevant except for the fact that twenty years ago in an accident she was caught under a pile of dirt and rocks. At that time she began to have headaches, which were sudden in onset and were localized over the vertex. These frequently occurred twice a day.

The present medical history begins about one year ago when she began to have constant and severe headaches along with failing vision in the left eye. This progressed slowly, and about six months ago vision in the right eye began to be impaired. For the past few days she has been totally blind. The pain in the head begins at the base of the skull in the occipital region and radiates fan-shape, upwards over the skull.

There has been no tinnitus at any time, but vertigo and dizziness have always been present. Incontinence of urine, mental deterioration, as well as gastro-intestinal disturbances, have been constant factors. The mental deterioration is quite obvious in attempting to take the history, as she has forgotten many details regarding her past and present history which were obtained from her sister. There is a complete loss of vision. Sensation of the face is normal. There is a definite right facial weakness of the peripheral type. The jaw and tongue deviate to the left; there is no atrophy or tremor. Hearing is impaired. The voice is masculine. The hands are slightly enlarged; the feet are slightly enlarged. Heart sounds are rather faint. Reflexes of the upper extremities are equal, 3. Reflexes of the upper patella are equal, 2. Achilles reflex left, 2; right, 1. There is a suggestive Babinski on the right but not on the left. Heel to knee test and finger to nose test are well done. Muscle strength is normal. Patient is unable to sit up with arms folded across chest.

The patient is a very large, obese female, about forty-two years of age. The hair is scant over forehead, but normal in texture. The skin

of the face is heavy, thick and wrinkled, especially around the forehead. There is proptosis of both eyes, with thick discoloration of the skin around the eyes. The pupils are not equal; the right is about 2 mm. larger than the left. Corneal reflexes are acute in both eyes—not as much so in the right. Palpebral fissure on the right is larger than on the left. On moving the eyes to the right there is a definite nystagmus; on looking to the left the nystagmus is not so great, but the external ocular movements of the left eye are poor. Upper rotation is not possible.

The roentgenographic report is as follows:

"The sella turcica has been practically destroyed. What appear to be intact anterior clinoids are found to be approximately twice as far apart as in the normal. The two middle fossæ exhibit thin floors which are not divided from each other by any suggestion of a pituitary fossa, and likewise the body of the sphenoid bone and the entire basilar process of the occipital bone have been completely destroyed. The foramen magnum has lost its ventral rim completely. The mesial ends of the petrous portions of the temporal bones have been destroyed and bone has been destroyed down to the very margin of the jugular foramen on each side, but especially on the left. Ventrally the destruction involves the ethmoid mass and has completely destroyed the bone through which the optic foramina normally pass. The floors of the middle fossæ are destroyed mesially and a soft tissue mass in the nasopharynx, in the position ordinarily assumed by adenoid tissue, appears to connect directly with the tumor, or at least with the cranial contents. There is absolutely no suggestion of a sphenoid sinus."

Clinical examination postrhinoscopically reveals a large soft tissue mass in the epipharynx, practically occluding this whole area. By the use of the pharyngoscope this mass is seen filling the entire choana and extending into and involving the sphenoid region.

Histological report from autopsy specimen—"malignant chordoma."

It is a significant fact that in no case of speno-occipital chordoma reported has there ever been metastasis.

The patient died January 3, 1935.

The temperature varied from 97.2° to 99.3°.

The pulse was more or less steady from 90 to 100.

Respirations, 20.

Blood examination, December 31, 1934, showed: red blood cells, 4,320,000; white blood cells, 6000; color index, 9; Type II.

The Wassermann was negative.

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## DISCUSSION

MR. GAVIN LIVINGSTONE, London, England:

Three years ago I was reporting a case of chordoma which was under the care of Mr. F. J. Cleminson at the Middlesex Hospital. I wish I could have heard Dr. Ridpath's concise summary of this interesting condition instead of reading through the literature on the subject.

I have a slide which is taken in part from Sir Arthur Keith's book on Embryology and demonstrates the vestigial notochordal remnants. The diagram is schematic and shows the most common site just below the base of the sphenoid.

Chordoma may be divided into two types: (a) a malignant type in which the mucin-producing cells predominate, and there are many hemorrhages in all stages of development. These tumors invade but do not metastasize; and (b) an encapsulated type with lipoids and fat globules which do not recur after removal.

In the case I reported, the main tumor, which was of the former variety, was in the postnasal space and was not ulcerated. With radium needles the main mass rapidly disappeared and a year later at autopsy there was no recurrence in the nasopharynx, but in the cranial cavity was an extension the size of a golf ball. The x-ray picture showed tumor invasion of the base of the sphenoid only during the last two months.



DR. ROBERT RIDPATH, (in closing):

I really have finished the paper in the time allotted. The other part of the paper was the report of the 2 cases; this will be printed in full in the *Transactions*. The one thing that Mr. Livingstone mentioned in his discussion is very characteristic of chordoma; this I tried to point out—that we do not have metastases. Glandular involvement is *nil* in all chordomas. We have extension, proliferation and destruction of the tissues, but the glands are not involved and in chordoma these are the diagnostic differences from any of the other tumor masses that we have in this particular locality.

## OSTEOMYELITIS OF THE FRONTAL BONE: A REPORT ON EIGHT CASES

RICHMOND MC KINNEY, M.D.

FOR several years an increasing amount of attention has been given to osteomyelitis originating in the frontal sinuses and extending to the frontal bone, with a growing recognition of the clinical importance of this condition.

The following notes and conclusions are deducted from observations made in a series of cases coming to the charity service of the John Gaston Hospital in Memphis, with the surgical work largely being done by my associate, Dr. Charles K. Lewis, of the Department of Otolaryngology, and the pathological study by Dr. Leo Harris, of the laboratory of the University of Tennessee College of Medicine.

Although one writer, Woodward, offers as a premise that osteomyelitis as a complication of frontal sinusitis is comparatively rare, and that no one has reported more than 3 cases occurring under his personal observation,<sup>1</sup> my observation has been that the majority of these cases have the frontal sinuses as a source of origin.

In our service at the John Gaston Hospital, there have occurred during the past two years not less than 12 cases which symptomatically may be regarded as osteomyelitis of the frontal bone, of which I give the histories of 8, and in several of these the frontal sinuses definitely were the point of origin. Frequently, traumatism of a sinus is an active factor in producing the disease.

These cases ranged in ages from thirteen to forty years, thus bearing out the assertion that since this is a condition involving the diploë, the more marked development of these in adolescence and young adult life is largely instrumental in causing the greater incidence of osteomyelitis in the period before middle life.

The pathology of this condition has been so thoroughly presented by Mosher and Judd,<sup>2</sup> and various others, especially by Mosher, that it scarcely is necessary to go into this in the present report of

cases. Suffice it to say that the infection found in most of our cases was predominantly staphylococic in character, although other bacterial organisms may have been present. This infection, as is well-known, extends by the diploëtic veins, with localized abscesses in the bone marrow.

The symptoms are clearly defined, for usually the edema and, frequently, redness extend from the orbital ridge to the hairline rather quickly, and there is a low-grade septic temperature rise, although these cases may be free from fever. In only 2 of our cases, however, was there a definite leucocytosis.

Of the cases that we have seen reported, a number of them have been diagnosed from clinical symptoms alone, without giving the microscopic pathology from bone sections, but doubtless the bone pathology was present, when the clinical findings were so definite.

I am of the opinion that this condition occurs much more frequently than would be judged from the number of cases reported, and that some cases reported as merely chronic frontal sinusitis are in reality complicated by osteomyelitis.

This is a very insidious disease, and unless of the fulminating type, in which development of the process is rapid, the amount of bone involvement found on operation may be surprising. One of our cases was subjected to 3 operations before we were convinced that all of the diseased bone was removed.

While very extensive removal of the frontal bone may be necessary, including the inner table, the dura rapidly becomes covered by granulations, and the soft tissues thicken and afford ample protection to the brain. We have not observed our operated cases long enough to note the bone regeneration seen by Furstenberg,<sup>3</sup> but doubtless the younger cases definitely do show a certain amount of bone regeneration during the course of years.

In operating on these cases we have not followed any definite procedure, but mainly have sought to remove all of the diseased bone, which can be done only by thoroughly reflecting the soft tissues covering the frontal bone, and then biting and chiseling the cheesy bone away until what is judged to be normal bone is encountered. The amount of bone to be removed at times is appalling, but without thorough removal, operation is useless. X-ray films are useful as guides, but occasionally are misleading,

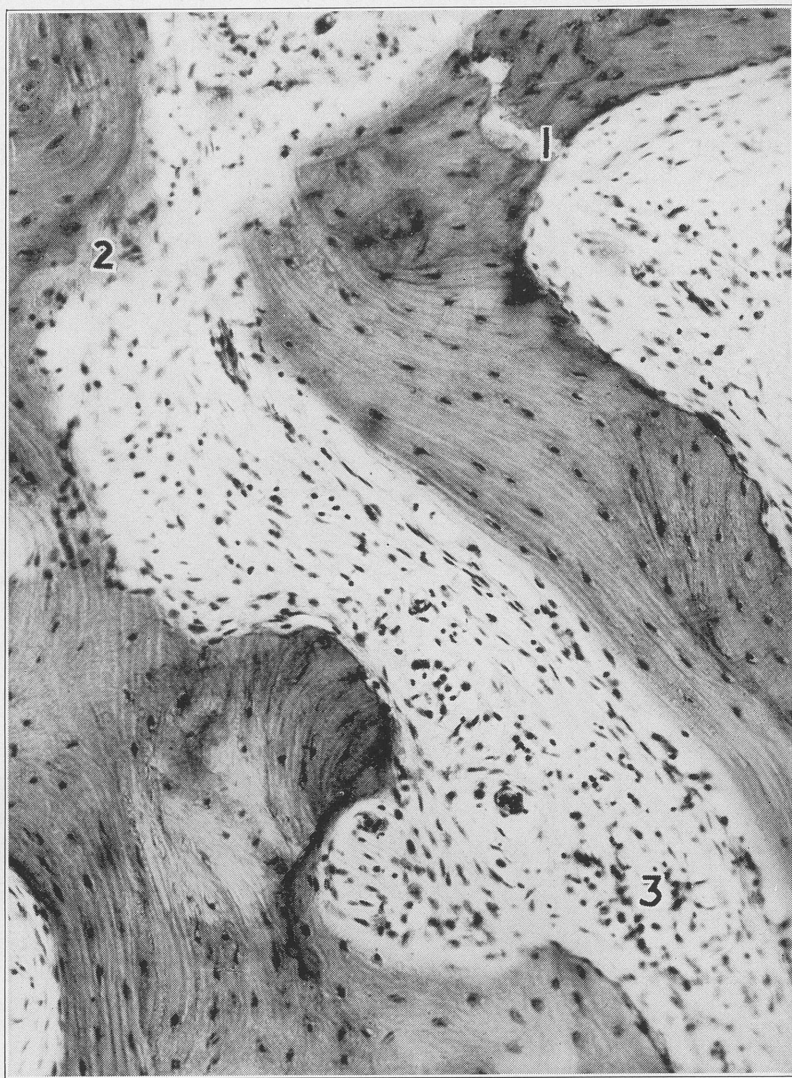


FIG. 1.—Photomicrograph showing spaces of bony absorption, 1, in which degenerated bone cells are visible; also irregular haziness of osteoid tissue, denoting a destructive process. Osteoblastic activity in the margins of bone is indicated by 2 and chronic inflammatory cell infiltration by 3.



as bone, macroscopically apparently in good condition, is sometimes undergoing osteomyelitic changes.

The mortality rate in our series was in about the same proportion as in reports made by others, 3 of the 8 cases reported dying; one of these of meningitis, another from brain abscess, and another from a concurrent bronchopneumonia. Lewis<sup>4</sup> found the mortality rate much higher in cases following a sinus operation, with which conclusion I must concur.

The treatment is essentially surgical, prompt and thorough, in order to avoid a possible, yes, probable leptomeningitis, brain abscess or thrombophlebitis.

With these brief introductory remarks I shall report, more or less in abstract, owing to their large number, the cases comprising this series. Three of the most interesting are given in detail, with stereopticon views bearing upon clinical aspects and bone pathology. Complete records, with *x*-ray films, blood chemistry, and other details are in the hospital files, but could not be given other than synoptically in this place.

W. K., a boy, thirteen years of age, white, was admitted to the Isolation Hospital on November 2, 1936, with a diagnosis of erysipelas. This provisional diagnosis was made owing to the fact that the child had a redness and swelling of one side of the face, extending from the supra-orbital arch to the hair roots, which had been present for several days prior to admission. *X*-ray films showed extensive involvement of the ethmoidal cells with, also, involvement of the frontal sinus on that side. Immediately, the ethmoidal cells of this side were drained under general anesthesia, but in a short time a pansinusitis developed, and the frontal and ethmoidal sinuses were then drained. The frontal bone was found to be soft and bluish, typically osteomyelitic in appearance. The child, being diabetic, had very little resistance, and three days later developed meningitis, which brought death on December 7th. The white blood count taken just before the first operation, was 8520.

COMMENT. This was undoubtedly a case of fulminating osteomyelitis beginning in the ethmoidal cells. Owing to the child's general condition, I doubt very much indeed if treatment, however radical and early, would have been of any value in preventing the fatal termination.

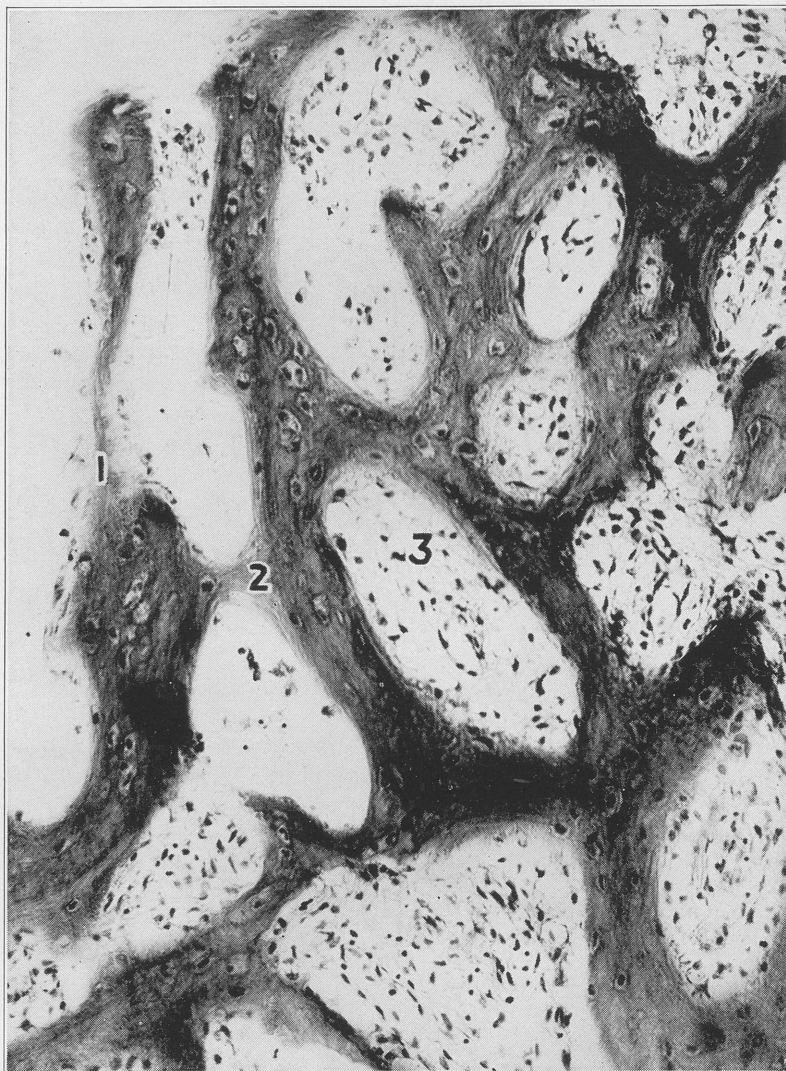


FIG. 2—Photomicrograph showing osteoblastic activity in the margins of bone, 2, with regeneration of bone. Spaces of bony absorption are indicated by 1 and chronic inflammatory cell infiltration by 3.

B. L., a male negro, aged forty years, was admitted to the hospital December 29, 1936, with the history that he was struck over the glabella with cotton scales six weeks before admission. He was running a temperature averaging around  $101^{\circ}$  when admitted, was poorly developed and poorly nourished. Films showed apparently an osteomyelitis of the frontal bone, nasal bone, and ethmoid cells. He had a rather low white blood count, this being 3650.

December 31, 1936, the man was given ether, and an incision made along the right eyebrow down to the bone. The periosteum of the right frontal sinus was elevated, and a piece of necrotic bone removed, pus and granulation tissue being curetted from the frontal sinus down to and including the nasal bone and the nasal process of the maxilla. This was removed, and the wound packed with iodoform gauze.

January 7, 1937: As there was apparently more involvement of the bone, the previous incision was reopened under novocaine anesthesia, and the discoloration of the bone found to extend further toward the floor of the frontal sinus and over the bridge of the nose. This discolored bone was removed and a quantity of granulations and necrotic bone cleaned out of the ethmoidal capsule. January 19, 1937: The wound had not healed as satisfactorily as was expected; the man this time was given intratracheal anesthesia, the wound reopened, and the edges of the bone previously exposed found to be in good condition with the exception of a small area over the bridge of the nose. This was removed, and from that time on the man made an uninterrupted convalescence.

The pathological laboratory reported that the sections of bone showed chronic osteomyelitis.

COMMENT. This case is interesting in that it showed that failure to remove even the smallest portion of diseased bone, where there are osteomyelitic changes, may continue to give rise to unfavorable symptoms. Further, it might be of some interest to note that three different kinds of anesthesia were used for the three different operations, for no especial reason, however.

W. H., a male negro, twenty-three years of age, was admitted to the hospital with the history of having developed swelling and inflammation in the right eye from three to four days before admission, which he thought was due to a head cold. He had a cellulitis in the right eye, had a temperature of  $101^{\circ}$  to  $102^{\circ}$ , a leucocyte count of 13,600, and appeared quite toxic. There was decided tenderness over the right frontal bone. X-ray films showed the right antrum



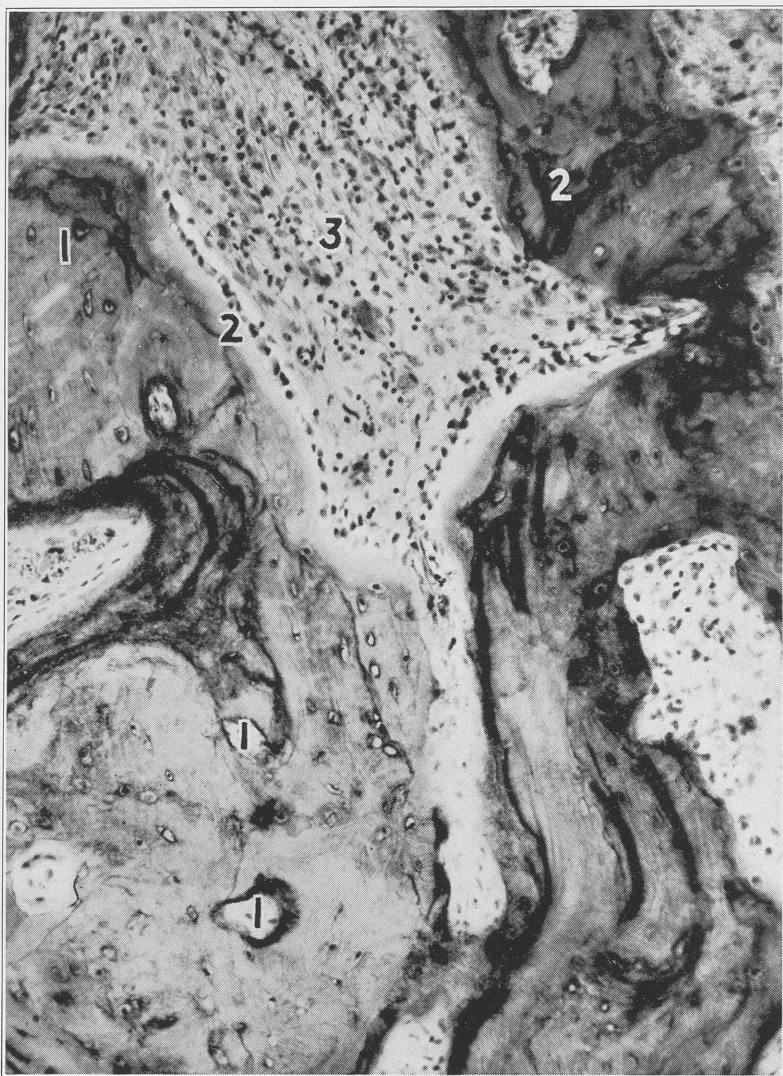


FIG. 3.—Photomicrograph showing chronic inflammatory cell infiltration, 3, characterized by a few polymorphonuclear leukocytes, plasma cells and many lymphocytes. In addition, a desmoplastic reaction is noted. Spaces of bony absorption are indicated by 1 and osteoblastic activity in the margins of bone by 2.



and right ethmoidal cells with increased density, apparently due to the presence of fluid.

The next day, under nitrous oxide-ether, the bone over the right frontal sinus was removed, and a quantity of foul-smelling pus liberated. The ethmoids were opened and the bone found to be necrotic. Bluish, soft bone above the right orbit was extensively removed. The man was discharged later to the out-patient department, and in a short time was discharged as cured.

The bone removed in this case was not examined by the pathological laboratory, but had the typical appearance of osteomyelitic bone.

T. M., a negro male, aged twenty-two years, was admitted to the hospital February, 1937. He stated that he had been struck over the left eye seven months before, following which there was a swelling, which ruptured, leaving a fistula discharging thick, creamy pus. There was no fever. His leucocyte count was 9450. An x-ray examination gave the diagnosis of chronic, unilateral pansinusitis, with activity present.

Under colonic anesthesia, the right frontal sinus was opened, and the bone surrounding the fistula found to be necrotic. This was removed, and the sinus cleaned of pus and granulations. The pus showed a growth of hemolytic *Staphylococcus aureus*. The laboratory reported that the specimens of bone removed revealed a chronic osteomyelitis.

The wound continued to drain, and the man returned to the hospital for reoperation on two occasions. The last time, in October, 1937, films showed some cloudiness of the frontal sinus, but no extension of the osteomyelitis. Intratracheal anesthesia was used this time, the old incision reopened, and the disease of the bone found to be progressing not only along the cut edges of the bone, but over the right frontal sinus, and well up on the frontal bone in the midline. The bone showed the characteristic discoloration and moth-eaten appearance of osteomyelitis.

All bone that appeared to be diseased was removed, the resection being carried back into the bone around this area in an effort to remove all traces of osteomyelitis. The dura was covered by granulation tissue. The denuded field was painted with iodine, the dura covered with a greased iodoform pack, and the scalp closed, as was usually done in cases of this character that came to operation. Skin clips were used on the edges of the scalp to control bleeding.

Postoperative treatment, besides the usual saline irrigations and sedatives, consisted of large doses of sulphanilamide and two blood

transfusions. The man was discharged a month after admission, with only a small gap between the eyebrows still open. When seen over three months afterward, he was free from any further symptoms of osteomyelitis. At this time he was cautioned to come into the out-patient department every day for the purpose of dressing. This he failed to do, and after some three months, presented himself at the office in very bad condition. He had gone down into Mississippi, where he was working, had permitted the wound to close too quickly and had developed an epidural accumulation of pus. He was immediately sent into the hospital, where an effort to save him by establishing drainage was made, but in a short while he died from toxemia.

H. M., a male negro, aged nineteen years, was admitted to the hospital in September, 1936, with a complaint of purulent discharge from the right eye with swelling of the lid of two days duration. X-ray films made on admission showed marked cloudiness of the right ethmoid cells, with only moderate involvement of the other corresponding accessory sinuses. Through an external opening the ethmoid cells were drained of a thick, white pus. Shortly after he was discharged, apparently much improved, and instructed to return to the out-patient department where he could be kept under observation pending subsidence of the acute symptoms. Two months later he was readmitted to the hospital, the incision not having healed, there being a small fistula from which pus was draining. There was no pain or fever, and his general condition was good.

Under colonic ether anesthesia, the old incision was reopened and enlarged, and the bone covering the right frontal sinus and ethmoid cells was found to be necrotic, with pus in the frontal bone. Resection of this bone was carried up to what was apparently healthy tissue, and the man was discharged March 9, 1937. Since then, so far as we know, there has been no recurrence. The laboratory diagnosis was chronic osteomyelitis.

M. H., a male negro, aged twenty-five years, was admitted to the hospital on September 25, 1937, with a pansinusitis of the left side and spontaneous rupture of the corresponding frontal sinus. The man also had a bronchopneumonia. This condition began some two weeks previous, with a headache and pain in the left eye, which later began swelling, became inflamed and discharged a purulent material. He had some fever and light rigors, all increasing in severity to the day of his admission. There was a leucocyte count of 13,200.

An external incision was made, and the frontal bone exposed. This

was found quite necrotic, bluish and cheesy, and thick pus was evacuated from the frontal sinus and ethmoid cells of this side. The man's condition would not admit of further operative treatment, and he was returned to the ward for observation. Continuing to run a septic temperature course, a blood culture on March 10th was reported positive for *Streptococcus hemolyticus*. This septic condition, together with a bronchopneumonia, which he had on admission, was responsible for his death on March 16, 1937. The bone removed showed chronic osteomyelitis.

M. H., a female negro, aged thirty-nine years, entered the hospital complaining of pain and tenderness over the eye and forehead of one year's duration, this increasing in severity until it was almost unbearable. She ascribed the origin of this trouble to an acute head cold.

Edema and tenderness were found over both supra-orbital regions, extending upward to the hairline. X-rays of the bone underlying this area showed it to have a moth-eaten appearance. She had a temperature of  $100^{\circ}$ , with a total white cell count of 9400.

Avertin and novocaine anesthesia were used, and an incision was made supra-orbitally. This was bisected by a vertical incision extending up beyond the edematous area, forming two triangular flaps. When the flaps were retracted, the bone was found to be dark purple and necrotic in appearance. A triangular section of the bone was removed, and with it a fragment of dura about the size of a 5-cent piece. The dura was discolored and bled freely, but there was no indication of abscess formation. Because of loss of blood and the uncertainty of blood donors for a transfusion, it was decided to postpone the completion of the operation until a later date. The laboratory reported that the bone showed chronic osteomyelitis.

After a week's interval, during which the patient received 550 cc. of blood intravenously, avertin was administered, and the incision reopened. The remainder of the diseased bone was removed with enough of the normal to insure a reasonable margin of safety. Iodine was painted over the dura and bone, a greased iodoform pack placed over the dura, and drains inserted. The edges of the scalp were then pulled loosely together to prevent retraction. The postoperative treatment consisted of large doses of sulphanilamide, irrigation of the wound with normal saline, and another transfusion. Convalescence was uneventful, and four months after operation there was found no sign of active pathology.

M. R., a colored female, aged twenty-two years, was admitted to the hospital complaining of a severe pain over the right antrum and

eye, with headache, fever and a regular chilly sensation for the past three weeks. There was periorbital edema of the right eye and cheek, with extension of the edema toward the hairline of the corresponding side. There was a white blood count of 17,900. The x-ray report showed that there was a chronic pansinusitis with fluid in the right antrum, and the right frontal bone looked osteomyelitic.

An external frontal resection was done, with removal of the bone high on this side. She was discharged from the hospital some days later, but returned in about a week with extension of the inflammation upward from the right frontal sinus.

Under intratracheal anesthesia, the right frontal, ethmoids and antrum were again cleaned out, and cheesy bone removed some distance above the right frontal sinus. This was found to be osteomyelitic. When seen some months later, the woman was free from any symptoms whatsoever and appeared to be entirely well.

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#### DISCUSSION

MR. VICTOR E. NEGUS, London, England:

I am glad to have the opportunity of speaking on this subject, Mr. President, not because I have anything to say different from what has been said already, but rather to add what evidence I can to the conclusions already reached, not only in the paper this morning on established osteomyelitis, but also on the paper yesterday on the treatment of acute sinusitis.

At the beginning of the time when I was working at Kings College Hospital, where I spent my medical life, we had rather violent and drastic methods of dealing with these cases, thinking that we had to get ahead of the disease in cases of acute involvement of the frontal sinus, and the results were really disastrous.

In a very short time the principles of delay were established, as they had been in acute mastoiditis as well, in our practice. We have now come to conclusions similar to those expressed yesterday and today, the conclusions being that in acute sinusitis there should be



the greatest conservatism, but when there is established osteomyelitis, the treatment should be drastic.

(Slide.) We have had a considerable number of cases of acute involvement of the frontal sinuses, and if the patient comes to us with such a condition, with swelling, and even with the eye completely closed, the treatment is conservative.

Mr. Bateman collected cases seen during the last ten years; we have a series of cases where there is not only involvement of the maxillary sinus but also an involvement of the frontal sinus, and many like cases with a collection of pus, shown as a fluid level.

In England it is still necessary to point this out because, unfortunately, there are a few surgeons who would feel it their duty to operate on the frontal sinus in an acute case.

In my practice the frontal sinus would never be touched and the principles are similar to those expressed yesterday.

The question of lavage of the maxillary sinus is one that need not be gone into, but the frontal sinus itself will not be touched, and my experience has been that as a result, spread into the bone, with complications of osteomyelitis, complications in the frontal lobe, with abscess, meningitis, and sinus thrombosis, have almost disappeared. The cases that are on record have been ones following operation.

Now that line of treatment has, I think, almost eliminated these complications and I feel firmly convinced, both from reports and from my own experience, that the complication of osteomyelitis is usually secondary to precipitate operation on the frontal sinus. I have recently, in the last few months, seen 2 unfortunate cases where osteomyelitis has followed operations on the antrum. They were not cases of my own, but I was asked to see them in consultation, and, so far as one could gather, there had been no reason for disaster.

An intranasal antrotomy had been performed, and in 1 case an operation on the ethmoid cells, but subsequently a puffy swelling appeared over the frontal region and it proved to be due to involvement of the cancellous bone which spread in the manner described today. The process was practically afebrile in its course but the creeping osteomyelitis progressed and even the most extensive operation, to get beyond the area of involved bone, failed to prevent secondary infection of the frontal lobe, and both of those cases died from brain abscess.

I do not know whether this is a common experience. I merely mention these cases as being the only 2 I can recall in many years, and I would reiterate that our principles are identical, I think, with those

expressed, the greatest conservatism being adopted in acute cases and only when there is established osteomyelitis, has extensive removal of bone been carried out; but I have not been so fortunate as the reports I have heard today indicate, in the results of these cases.

DR. GEORGE M. COATES, Philadelphia, Pa.:

My experience does not coincide with the previous speaker's or entirely with Dr. Furstenberg's talk of yesterday. In the last three or four years I have seen quite a good many of these cases of osteomyelitis, and I think Dr. Samuel R. Skillern has been associated with me in the care of all of them. I would appreciate it if the chairman would give him the privilege of the floor after I have said a few words.

In all the cases that I have seen in the last ten years or more, not more than 2 or 3 have followed any surgical trauma. Osteomyelitis, two or three years ago, followed a needle puncture of the antrum in an acute swimming case, and after some days the anterior end of the middle turbinate was removed. Practically all the others have not followed any surgical procedure on the nose or sinus, though 1 mild case did follow drainage of the frontal sinus through the floor.

I have seen the frontal sinus drained externally in acute cases and in chronic cases over a long period of years, and I have never seen, with that one exception, osteomyelitis follow surgical intervention on the frontal sinus.

Do not misunderstand me. I am not advocating radical treatment of these acute frontal sinus cases, but when you have an osteitis of the frontal plate of the sinus, with swelling, and closing of the eye, and exophthalmos, I believe we are justified in doing an external operation.

In the cases I have seen, particularly those recently, where conservatism has been followed, and we have finally had to open the frontal sinus because osteomyelitis had developed, we have in a large proportion of the cases found a frontal lobe abscess already established and we have had to drain this, and in 1 case it was discovered at autopsy, when we had failed to reach the abscess although the diagnosis had been made.

As to the treatment of cases of osteomyelitis after they have developed, I have felt for a long time, following the work of Mosher and Judd, that wide, ruthless eradication of the diseased bone is absolutely necessary. I have seen conservative treatment applied and I have possibly seen 1 or 2 of these cases get well by sequestration, the sequestrum being removed later, but I have seen so many of these cases

develop intracranial complications, meaning a frontal lobe abscess, particularly, that I am no longer in favor of conservative treatment once the osteomyelitis has developed.

I furthermore believe that the method of removing this should be from good bone down and not from bad bone up, making a wide, ruthless removal of the bony plate, preferably in one piece, if possible.

There is a difference between an osteomyelitis and an osteitis. We do not always have diploic bone in the anterior wall of the frontal sinus. If you do not have diploic bone, your pathological condition, as I understand it, is going to be an osteitis and this, if not removed by surgical intervention, in time will spread up to the juncture of the two tables, internal and external walls, where your diploic bone is more likely to commence, and, once it gets there, you have your developing osteomyelitis of the frontal bone, and I think that the only safe method is to remove it and remove widely.

DR. SAMUEL R. SKILLERN, Philadelphia, Pa.:

Since last September, Dr. Coates and I have operated on 5 acute or chronic frontal sinuses complicated by osteomyelitis. As I recall, they all had some intracranial collection of pus, those with multiple brain abscesses having passed on "To the happy hunting grounds." Dr. Coates has just told you that we follow the technique of Furstenberg and Mosher (*i. e.*, wide extirpation), but he did not mention our method of trying to avoid disfigurement and scar.

We keep our skin incision within the upper confines of the hair of the eyebrow, and join the inner edges by a straight line above the root of the nose.

To avoid a sudden depression of the soft tissues, we bevel the edges of the cut bone around the sinus cavity; this allows the periosteum of the anterior wall to cover the curetted bone of the posterior wall and, as it becomes adherent, to completely obliterate the sinus cavity, with very little depression of the soft tissues.

If after cleansing the frontal and fronto-ethmoidal cells, we find the posterior plate involved (softened bone or a fistula), we attack this plate. We have found in all of our early cases of osteomyelitis, that the first manifestations of this disease of the bone was at the junction of the anterior and posterior plates of the sinus cavity. In 2 cases that I recall, we followed the infection upward simply by removing the outer table over the congested and bleeding blood channels until we reached the clot or embolus that was obstructing the return flow. Cultures from these clots gave the same growth as

that taken from the sinus cavity. We were fortunate that the infection had not passed through the inner table, otherwise these patients might not have made their uneventful recoveries. If we find the posterior plate involved, we try to remove all bone for at least 2 inches beyond this infection.

To avoid the median scar and to obtain greater exposure, we start our incision at the outer end of the brow opening, carry it upward and backward just anterior to the temporal fossa into the fronto-temporal hair line. If a longer incision is necessary, it can be carried backward without fear of cutting any large vessels or nerves, and with the assurance that it will be hidden as soon as the hair regains its normal length.

The bone incision is outlined with the Killian chisel. Then the bone is incised to expose the dura. The half circle is completed by cutting the posterior wall of the frontal sinus just above the superciliary ridge.

Nature had anteceded us in several of our cases. We found the posterior plate lying free, as a sequestrum.

With clean instruments, for the upper bone incision and removal of the amputated frontal bone, our bone flap is separated from above downward. Care must be exercised in digging the superior longitudinal sinus out of its channel, otherwise the bone is easily elevated from the dura.

It is much easier to remove the bone piece-meal by first separating it for an inch or so from the dura, then biting away the bone, but as we wished to obtain a complete section to show the disease from its destruction of bone to its outer limitations, we removed this section in one piece.

To deviate from our subject, it is essential to speak of an important factor in the operation, *i. e.*, bleeding. The ascending lateral nasal veins as well as the veins of the scalp are very troublesome, as they bleed freely and are difficult to pick up and ligate. We have found that electrocoagulation is far more satisfactory than ligation.

May I briefly speak of one case? A boy was sent to us with a discharging surgical fistula in the super-orbital wall, with the diagnosis of acute frontal sinus disease following swimming. Our clinical findings as well as our roentgenograms confirmed this diagnosis. There was no evidence of osteomyelitis, but there was osteonecrosis of the floor of the frontal sinuses.

As we removed the anterior wall of the right frontal sinus, we noted a grayish-white substance filling the sinus cavity. I said "pus." Dr. Coates replied, "Hold on! Is that pus? It may be brain tissue."



After the anterior plate was removed we found his opinion was correct, as the sinus cavity was filled with herniated brain tissue.

We drained the brain abscess with rubber tubing and gauze, covered the dura with rubber tissue and gauze which was kept saturated with Staphylococcus Bacterial Antigen (Lilly), with the cut edges of the soft tissue approximated to prevent retraction.

We removed the stitches in seventy-two hours to find a clean granulating wound.

Knowing the regenerative powers of bone dust, I suggested chipping off thin layers of bone from the healthy edges of the cut skull. These were scattered over the dura, then covered by the periosteum of the elevated flap. I am happy to report that these bone chips grew, coalesced, and formed a new anterior plate over the greater majority of the area.

The old saying that "one swallow does not make a summer" may be quite true, but if regeneration of bone occurred in this case, there is a possibility that it will occur in the majority of cases, therefore, why not try these "implants" of bone? If they fail to grow, no harm has been done. If regeneration does occur, we have the normal bony protection for the brain.

I wish to thank you, Mr. President, for calling upon me to discuss a paper presented before this distinguished society.

DR. A. C. FURSTENBERG, Ann Arbor, Mich.:

Mr. Chairman and Members: I have listened with great interest to Dr. McKinney's presentation and can concur wholeheartedly in what he has said about osteomyelitis of the skull. It has been a rather interesting observation that among our 58 cases of osteomyelitis of the skull, 42 of them followed some surgical interference with nasal accessory sinus disease. In 4 instances we observed a devastating attack upon the calvarium following the simple intranasal drainage of an acute maxillary sinusitis. Consequently we often wonder whether this dreadful surgical tragedy is due to some error of omission or commission on the part of the otolaryngologist.

I am particularly interested in the method of spread of infection in osteomyelitis of the skull because it has much to do with our principles of treatment. In many instances the extension is by continuity of tissue; that is, the infection passes directly from the frontal sinus into the diploë of the skull between the external and internal tables. In other cases, however, the spread is chargeable to an intracranial yet extradural infection. In this connection one may demonstrate

with ease that in many of these cases we are dealing with a thrombophlebitis of the vessels of the mucous membranes of the frontal sinus, which in turn communicate with the meningeal vessels. The infection extends through these veins into those of the dura where there develops an exudate between the inner table of the skull and the meninges. This may subsequently spread over the dura, causing destruction of the inner table of the calvarium and eventually the diploë and external plate. In many of our cases the internal table has been the first to be destroyed; later the diploë and finally the external table.

When such a condition exists, it would seem necessary to remove radically, the full thickness of calvarium if we entertain the hope of curing the patient with an extensive osteomyelitis of the skull.

Then too, I have been interested for some time in the reparative processes in osteomyelitis of the skull. In all of my cases there has been extensive regeneration of bone. In two cases we have removed the entire calvarium and, much to our surprise, it has been restored through the medium of regenerated bone within periods ranging from six months to two years.

It can be demonstrated that bone will regenerate from any fibrous connective tissue background which contains bone and which is irritated by a little infection. If periosteum is stripped free from bone it will not regenerate bone, but if bone dust is added to periosteum and then irritated with a mild infection, regeneration will rapidly follow. To this discussion I should like to add that the dura mater is a very active osteogenetic membrane. The external layer of the dura is a periosteal element and if a little bone dust is sprinkled upon it and irritated with a mild infection, regeneration becomes active.

In my series of 58 cases, most extensive reparative processes in osteomyelitic defects of the skull are much in evidence. Consequently we need have no apprehension in removing wide areas of bone when dealing with this disease. Our only hope of successful treatment lies in the radical removal of all the infected bone, including full thickness of the calvarium.

DR. FREDERICK T. HILL, Waterville, Me.:

I think after hearing from such an outstanding series as Dr. Furstenberg has presented, anything I could add would be rather ludicrous. There is one point I think we could keep in mind, however, from a study of Dr. Mosher's series of cases—that is, the importance of the diploic veins, as center of infection and the fact that x-ray studies,

if carefully done to show these veins, may give us information of some prognostic value as to the probable occurrence of an osteomyelitic process. The veins are much more common and larger than we have realized and they can be demonstrated in the incipient stage of the case, so some idea is given of the possibilities that may impend later with a case of osteomyelitis.

DR. FLETCHER D. WOODWARD, Charlottesville, Va.:

This is a subject in which I have been interested for a long time, and it seems to me that the problem, so far as etiology, pathology and operative treatment are concerned, is pretty well settled. During this time there has been one thing which has been overlooked, which to me has been the most important of all in the consideration of osteomyelitis: that is the bacteriological side of it.

It is the staphylococcus that causes osteomyelitis, and its characteristics should be studied. Operative intervention does not have a great deal to do with its causation. It is primarily a question of the organism itself, so what are we going to do if we have this organism present?

Unfortunately, there isn't a great deal we can do along the line of specific therapy, but there are some things that have come out in the last few years which are helpful. One is, that practically all of them have a very low blood titer to the staphylococcus toxin. This examination is readily carried out in any laboratory. If they have a low blood titer, it can be raised by giving toxoid and, if you can raise the titer, you have done something to help.

Another thing—you should anticipate this condition by getting an early culture, and if we are dealing with a staphylococcus infection, we immediately start giving staphylococcus toxoid to increase the patient's resistance as best we can.

If we have an established case of osteomyelitis, we use the antitoxic serum for the purposes of neutralizing the toxemia and for a certain amount of bactericidal effect which the antitoxic serum apparently possesses, as well as toxoid.

Finally, repeated small transfusions are important. But have blood titers made on your available donors and choose that blood which has the highest titer.

MR. VICTOR E. NEGUS, London, England:

If I might be allowed to make one correction to avoid any misapprehension, I mentioned 2 cases of osteomyelitis of the frontal bone following operations on the maxillary sinus performed by two

colleagues, and I should like to make it clear they had the same views as those I have mentioned. The operations were carried out on cases of chronic sinusitis and not on cases of acute sinusitis.

DR. JOHN F. BARNHILL, Miami Beach, Fla.:

Just a few words about the operation for osteomyelitis. Practically all cases should be operated. The exact time for performing the operation and the extent of the procedure will depend much upon the judgment of the surgeon who does the work, or upon him and his consultants.

I agree with those who remove wide areas of the diseased bone and by wide areas is meant those that include all bone that is evidently diseased and all that is strongly believed to be affected. It is of small value to take away any part of the diseased bone unless all is taken away. To be thus thorough may mean that an extensive operation must be done. But, as Dr. Furstenberg has pointed out, to remove a rather wide area of the skull is not as serious as one might fancy since it has no function except to protect the intracranial contents; and so the patient may live on in good health provided protection to the brain is assured in some other way.

During an operation for the removal of affected bone how may the operator know when all the osteomyelitic structure has been removed? The answer is difficult except to experienced operators. X-ray pictures are not wholly reliable and appearances are sometimes deceptive. As the line between healthy and diseased bone is approached the difficulties increase, and it is just here that greatest care is needed.

Dr. Jackson has spoken of teaching the fingers. It is equally necessary to teach the eyes and ears, especially in bone surgery. The fingers may be taught to recognize diseased bone when passed over it; the eyes may be trained to catch the slightest change in color, as, for example, from the slightly pinkish normal to the beginning ashen gray, and the ears may be attuned to analyze and evaluate the various sounds that are made when a gauge is driven through, or the rongeur takes away a bite of bone. There should also be added to these trained senses the important sense that appreciates and appraises the resistance of bone to the action of instruments. There is an unmistakable difference between the *feel* of the instruments when biting away softened or partly softened bone, and the hard, unyielding bone of a table of the skull; and there can be no mistaking by the trained ear the difference in sound when biting away healthy bone, and bone that is but slightly diseased. And so the trained operator continues the



removal when operating for osteomyelitis, until the bone *feels* right, *looks* right and *sounds* right when bitten through.

After healthy bone has been reached the edges of the osseous wound are smoothed and beveled, ample drainage is provided for and reinfection is provided against. This is important; and it is also important to give the patient a balanced diet; and, when possible, to keep him in the open air and sunshine of a warm climate. Cure may be expected in a large percentage of cases.

DR. JOSEPH C. BECK, Chicago, Ill.:

I missed any reference made to operative complications in extensive removal of bone. I happened to have had one very extensively operated case in my group, who had developed epilepsy, and in this connection I was prompted to ask Dr. Furstenberg whether he had ever re-operated any of his cases to see whether he really had bone changes.

In this case our Chairman said that the skull is only for the purpose of protection. Well, it seems to me that it is also there for the purpose of making trouble as in some of the cases of epilepsy and I think the reader of the paper, who has had quite an experience, should say something about that.

My own case was referred to a brain surgeon who very cleverly transplanted fascia after breaking up adhesions in the denuded portion. I should like to hear something regarding that.

DR. A. C. FURSTENBERG, Ann Arbor, Mich.:

I can merely quote more statistics by saying that in our series of cases of osteomyelitis of the skull we have had but one patient return to us with epilepsy. This was a young boy, aged sixteen years, who had had a frontal lobe abscess associated with an osteomyelitis of the frontal bone. The abscess had been drained successfully and the patient made an uneventful recovery, only to return to us two years later with a Jacksonian epilepsy involving one side of the body.

Our brain surgeon opened the wound, removed a plaque of regenerated bone, and excised some scar tissue from the brain. The patient has remained in good health, free from convulsive seizures, since this secondary operation about two years ago.

DR. RICHMOND MCKINNEY, (in closing):

I am more than gratified to have offered a subject which has brought out such uniform discussion, with a great deal of unanimity of opinion.

The only question seems to be as to whether this condition follows operations on the sinuses or not, and that seems to be fifty-fifty in opinion.

Dr. Skillern spoke of the incision. He describes an incision that would leave a very small scar. The class of patients we have had for the past two years is not the kind where we consider the cosmetic effect. They are rather proud of the scars and like to show them, and we endeavor to get the soft tissues off the best way we can in order to remove the diseased bone thoroughly.

Once I had a case of gastrostomy I had done following a lye stricture of the esophagus, and even after the lumen of the esophagus was restored, the woman was so proud of the gastrostomy, she did not want to have it closed, she wanted to show it to her friends. These patients like to show their scars. In private cases perhaps we would use a little different method with a view to cosmetic results, but I have never had a private case of osteomyelitis.

Dr. Furstenberg is always interesting in his discussions, and I am sorry that we do not get to see these patients afterward so that we can observe the bone regeneration. It is hard to get them back after they have once apparently recovered. We cannot get to see whether there has been bone regeneration or not, but we have noticed this beginning in some cases, and I do not doubt, especially in view of his vast confirmatory experience, that there is complete regeneration.

Dr. Beck brought up a question which I cannot answer. I have not had any experience with that. Only in the past two years have we been paying much attention to this subject of osteomyelitis in my service at the John Gaston Hospital, a large charity hospital, and as is altogether natural with us in the South, a large portion of the patients are colored and difficult to follow after they leave the hospital. In my service in the Department of Otolaryngology of the University of Tennessee, we have only recently been paying much attention to the subject, and I am following the cases more closely now, and having the young men on the service look after them. We probably will have a more complete study in the future.

My few cases reported dwindle into insignificance beside Dr. Furstenberg's 52, but I hope that some of these days we will have a much larger number to report.

I want to thank the different ones who discussed the paper, and I appreciate it very much—they were even gentle in their discussion!

## OBJECTIVE EVIDENCE OF THROAT IRRITATION FROM THE HYGROSCOPIC AGENT IN CIGARETTE SMOKE\*

HOWARD C. BALLENGER, M.D.

THE laboratory experiments of Mulinos and Osborne<sup>1,2</sup> in the rabbit's eye and the clinical investigation of Flinn<sup>3</sup> led to their conclusion that diethylene glycol is definitely less irritating to the eye of the rabbit and the respiratory mucosa than is glycerine when used as the hygroscopic agent in cigarettes. Haag,<sup>4</sup> at the Medical College of Virginia, in duplicating the work of Mulinos and Osborne, failed to find any difference in irritation of the conjunctival sac of rabbits as determined by the appearance of hyperemia, edema, blepharospasm and the objection of the animal. Holck and Carlson,<sup>5</sup> at the University of Chicago, failed to find difference in salivation in humans subjected to the smoke of the two types of cigarettes. From a clinical standpoint, Sharlit<sup>6</sup> was unable to find any difference between the irritating properties of the glycerine- and diethylene glycol-treated cigarettes. The writer,<sup>7</sup> in a clinical investigation in 1936, came to the same conclusion.

The clinical investigation reported here was planned along the general lines of the one which formed the basis of Flinn's<sup>3</sup> paper. The results are not in agreement with Flinn's report, and taken in conjunction with the reported work of Haag, and Holck and Carlson as well as the writer's own earlier clinical investigation, indicate that the current status of scientific knowledge of the relative irritating effects of diethylene glycol and glycerine when used in cigarettes does not justify the conclusion that there is any important difference in this specific property of these two hygroscopic agents.

The present investigation is a continuation of that done in 1936. As in the first investigation three lots of cigarettes were prepared (Lots 1, 2 and 3), one of which contained diethylene glycol (Lot 1) as the hygroscopic agent (2.68 per cent). Another lot (Lot 3) was

\* From the Department of Otolaryngology, Northwestern University School of Medicine, Chicago.

moistened with glycerine (2.69 per cent) and the third lot which were the control cigarettes (Lot 2) had no hygroscopic agent of any kind. Otherwise the cigarettes were identical. All were made by one tobacco manufacturer out of the same batch of tobacco and differed only in the hygroscopic agent used. The presence of diethylene glycol in the cigarettes of Lot 1, and of glycerine in the cigarettes of Lot 3 was established by the isolation and qualitative identification of these compounds.

Ten well known otolaryngologists were invited to select a group of cigarette smokers, the majority of whom (74 per cent) seemed to have symptoms from smoking.

The identity of the various types of cigarettes was not known to any of the examiners connected with the tests or the subjects until after the completion of the investigation and the final reports had been made.

The 102 subjects (Chart 1) upon whom the present tests were conducted varied in age from seventeen to sixty-two years (average about thirty years). They had been smoking an average of about 20 cigarettes a day for an average number of twelve years.

*Method of Study.* The tests were conducted in two stages. The first lasted for a period of three weeks in which the various lots of cigarettes were issued for a period of one week to each subject. At the end of each week he reported to the examiner for examination and report of symptoms, if any, when a new lot was issued. The cigarettes were rotated in such a manner that at any given time one-third of the subjects were smoking Lot 1 cigarettes, another third were smoking Lot 2 and the final third were smoking Lot 3. This was done to equalize any climatic changes or epidemics of colds. Any objective signs or subjective symptoms of irritation were noted.

The second phase of the test was purely subjective, that is, each smoker was given all three lots of cigarettes for a period of a week. He smoked them in any manner he chose in an effort to determine any difference in irritation or symptoms from the three types. The conclusions from both tests were noted and tabulated.

*Symptoms from Smoking.* The various symptoms attributed to smoking which the subjects presented prior to the tests (Chart 1) and listed in the order of their frequency of occurrence were: a dry



CHART 1

Case No.	Age	No. smoked per day	No. of years smoked	Brand	Symptoms attributed to smoking
1	32	18	12	Tareyton	
2	40	20	10	Pall Mall	Soreness of throat
3	48	10	10	Chest.	
4	20	18	5	Phil. Mor.	
5	28	20	7	Chest.	Dry nose and throat
6	25	20	5	Camels	Dryness, bad taste
7	30	15	10	Phil. Mor.	
8	44	15	15	Cam. and Chest.	Raw burning throat, cough
9	23	10	4	Camels	
10	32	15	10	Chest.	
11	39	15	18	Kools	Bad taste
12	45	40	20	Kools	Cough
13	31	25	15	Lucky	Dry throat and dry cough
14	34	15	14	Lucky	Cough
15	30	15	10	Lucky	Irritated throat
16	27	15	10	Lucky	Sneezing, cough
17	40	12	15	Phil. Mor.	Hoarseness
18	35	15	13	Lucky	Irritated throat, eye fatigue
19	38	30	20	Lucky	Dry throat, cough
20	43	20	20	Tareyton	Dry nose, cough
21	26	10	3	Kools	Cough
22	29	20	10	Lucky	Stuffy nose, dry throat, cough
23	31	15	15	Vir. Rounds	Cough
24	29	8	10	Marlbor.	
25	27	27	20	Phil. Mor.	Stuffy nose, dry throat, cough
26	35	20	15	Old Gold	
27	30	20	15	Lucky	Dry throat, cough, postnasal drip
28	31	15	12	Camels	Dry throat, cough
29	28	6	10	Phil. Mor.	
30	36	15	15	Phil. Mor.	Stuffy nose, dry throat, cough
31	24	10	4	Lucky	Dry throat, cough
32	25	10	11	Lucky	Hoarse, cough, mucus in throat
33	30	10	12	Camels	Dry throat
34	..	20	10	Lucky	
35	28	15	12	Chest.	Cough
36	24	5	6	20 Grand	
37	24	15	8	20 Grand	Cough
38	32	15	10	Lucky	
39	27	6	6	Camels	
40	..	15	6	Camels	Loss of voice (occ.)
41	62	30	20	Lucky	Dry throat, cough
42	40	20	..	Chest.	Dry throat, cough
43	37	20	..	Chest.	Dry throat, mucus throat
44	46	20	..	Camels	Cough
45	..	..	..	Camels	Irritated throat
46	30	..	..	Phil. Mor.	Irritated throat
47	48	..	..	Camels	Cough
48	28	20	..	Camels	
49	..	20	..	Phil. Mor.	Irritated throat, postnasal drip
50					

CHART 1—*Continued*

Case No.	Age	No. smoked per day	No. of years smoked	Brand	Symptoms attributed to smoking
51	26	20	9	Chest.	Husky voice
52	32	18	14	Lucky	Cough, occasional
53	33	20	13	Camels	Dry throat, cough, irr. voice
54	24	15	7	Lucky	
55	29	30	12	Raleigh	
56	38	9	15	Raleigh	
57	29	20	12	Chest.	Cough, occasional
58	28	20	12	Raleigh	Raw throat
59	17	23	2	Camels	
60	29	15	9	Marvels	
61	37	20	16	Chest.	Dry throat
62	53	25	38	Old Gold	Dry throat, husky voice, cough
63	40	35	20	Camels	Cough (chronic)
64	28	25	10	Camels	
65	25	10	8	Camels	Postnasal drip
66	31	25	15	Phil. Mor.	Dry cough, postnasal drip
67	30	20	10	Chest.	Nervousness
68	28	10	6	Lucky	Rough voice, postnasal drip
69	30	15	15	Tareyton	Dry throat, cough, perspir. hands
70	31	25	15	Camels	Mucus in throat, cough (wet?)
71	31	40	20	Camels	Dry throat and nose, cough
72	32	20	15	Phil. Mor.	Irritated throat, felt "dopy"
73	30	15	20	John Cott.	Hoarseness
74	35	15	15	Lucky	Hoarseness, loss of appetite
75	37	20	14	Camels	Dopiness
76	39	20	18	Chest.	Secretion in throat
77	41	20	18	Old Gold	
78	38	15	20	Raleigh	Cough
79	34	12	6	Camels	Burning tongue
80	30	10	15	Camels	Dry throat, migraine
81	29	10	9	Chest.	Irritated throat, husky voice
82	24	7	4	Kools	Irritated throat
83	30	20	10	Camels	Cough
84	27	15	5	Camels	
85	30	35	10	Lucky	
86	56	20	40	Fatima	Cough
87	48	10	30	Camels	Cough
88	26	25	8	Phil. Mor.	
89	25	30	4	Lucky	Cough
90	23	20	4	Camels	
91	26	25	8	Phil. Mor.	
92	45	30	30	Camels	Tickly throat, husky, cough
93	29	20	5	Phil. Mor.	Cough
94	25	20	20	Phil. Mor.	Dry throat
95	28	25	10	Pall Mall	Dry throat
96	30	20	18	Camels	Irritated throat
97	29	12	7	Phil. Mor.	
98	25	8	3	Camels	Occasional cough
99	24	15	7	Phil. Mor.	
100	25	10	10	Chest.	Irritated throat, cough, husky
101	22	10	7	Chest.	
102	22	10	3	Phil. Mor.	Occasional cough

or irritated throat (36); coughing, usually dry and worse in the morning (34); husky or hoarse voice (15); excess secretion, usually mucus (13). Symptoms referable to the nose such as stuffiness, dryness, or sneezing were mentioned in a few (10) instances. Various complaints mentioned by only 1 or 2 individuals in each instance were tickling of the throat, burning of the tongue, "dopy" feeling, burning of eyes, eye fatigue, bad taste, nervousness and loss of appetite.

The amount of smoking and the presence of symptoms seems to have some relationship, especially the morning cough, when more or less dark colored mucus is raised (Chart 3). The total number of symptoms attributed to smoking is higher in the heavy smokers than in the light smokers. However, the secretion of mucus in the nose or pharynx, stuffy or irritated feeling in the nose, dry or irritated sensation in the mouth or pharynx and husky or hoarse voice do not seem to have much relationship to the number of cigarettes smoked per day.

*Objective Examination.* An effort was made in each case by each examiner to determine by careful objective examination the amount of irritation produced during the test periods. This examination consisted of inspection of the mucosa of the nose, throat, and larynx. Notations were made of changes in the secretions, dryness, granulations, thickness and color. The color was charted after comparison with and in accordance to the Tallqvist scale.

Five examiners stated that no difference between the two lots could be ascertained. Three examiners found, in the few instances in which changes were noted, that they were so evenly divided as to be without significance. The two examiners who reported differences based on objective examination were not in agreement as to which cigarette produced the more irritation. One found more irritation from the diethylene glycol cigarettes and the other found more irritation from the glycerine cigarettes. Of the 102 subjects included in these tests, 75 were found to have no unfavorable changes in the mucosa of the nose, throat or larynx as indicated by the regular periodic examinations. In 10 cases the most unfavorable results followed the use of the untreated cigarettes. In 9 subjects the most adverse reactions followed the use of the diethylene glycol treated cigarettes. In 7 instances the most

unfavorable results followed the use of the glycerine treated cigarettes. It would seem that these results indicate clearly that no objective evidence of irritation occurred in this investigation which could be attributed to the effect of either of the hygroscopic agents under investigation.

*Subjective Reports.* In connection with this phase of the investigation, if consideration is given only to the subjects' reports of adverse symptoms believed by them to have resulted in irritation from the smoking of the various lots of cigarettes, and indefinite answers concerning likes or dislikes, flavor, good or bad, etc., are eliminated, we find the reports are so evenly divided as to be without significance (23 each). However, all subjective reactions of any type are reported in Chart 2.

*The Local Effects of Cigarette Smoke.* The local effects of cigarette smoke on the mouth, throat and nose are debatable. The writer is unable to pick out a "smoker's throat." It is questionable whether redness of the mucosa indicates irritation from smoking. Excessive serous secretion or salivation of short duration may be an indication. The subjective sensation of dryness or burning does not seem to have an associated objective manifestation. The cough, especially in the morning, appears to be due to the mucus secreted by the lung and bronchial mucosa in an effort to remove the non-absorbable constituents of the inspired smoke. The hygroscopic agent would seem to have little or no effect on this.

The possibility of a relationship existing between the amount of smoking and the lymphatic hyperplasia of the pharynx present in some individuals, was considered. A total of 224 subjects were examined with this object in view. Varying amounts of hyperplasia of the lymphatic follicles or bands of the pharynx were observed in 44 per cent of all cases. As shown in Chart 4, in the group of 41 subjects who smoked from 5 to 14 cigarettes per day, enlargement of the lymphatic tissue was found in 37 per cent, slightly lower than the average for the group as a whole. In the group of 132 subjects who smoked from 15 to 24 cigarettes per day this enlargement was observed in 48 per cent, somewhat higher than the average. However, in the group of 51 subjects who smoked from 25 to 40 cigarettes per day, the incidence of lymphatic



CHART 2.—RESULTS OF SMOKING (THREE WEEKS' PERIOD)

Order of preference*						
Case No.	3 weeks period	1 week period	Diethylene glycol 1	Glycerine 3	Controls 2	Objective signs
1	...	...	.....	Did not like	Better than 1	None
2	2-3 1	...	Throat discomfort	Throat discomfort	Cough, hoarse	None
3	1 2-3	...	Not as harsh	Strong taste	No symptoms	None
4	...	1 2 3	Mild	Stronger	Better than 1	None
5	1 2-3	...	Better than 3	Dry throat	Burning tongue	None
6	3 2 1	3 2 1	Dry throat, strong	Mild, better taste	Milder than Camels	None
7	3 1 2	3 1 2	.....	.....	.....	None
8	...	...	.....	.....	.....	None
9	1-3 2	1 3 2	.....	.....	.....	None
10	...	...	.....	.....	.....	None
11	...	2 1 3	Dry throat, cough	Irritated (cold)	Dry throat	None
12	3-2 1	...	Irrit. throat, cough	.....	Less irritation	None
13	...	2 3 1	.....	.....	.....	None
14	1 2 3	1 2 3	Milder than 2	Irrit. throat (cold)	Less irritation	None
15	1 2-3	1 2 3	.....	Slight irritation	Dry throat	None
16	...	...	.....	.....	.....	None
17	3 2 1	...	Irritated throat	.....	Slight, dry nose	None
18	3 2 1	1 3 2	.....	Least irritation	Bad taste	None
19	3 1 2	1 3 2	.....	Mildest	Cough	None
20	1-3 2	1 3 2	.....	.....	Slight sting	None
21	1 3 2	1 2 3	.....	Less dryness	Dry throat, secret.	None
22	3 2-1	1 3 2	Cold, secretion, red.	Less irritating	Less secretion	None
23	1-3 2	1 2 3	.....	.....	Dry nose	None
24	1-3 2	1 3 2	Slight cough	.....	Distasteful	None
25	...	1 2 3	.....	Bad taste	Dry throat (one day)	None
26	...	1 2 3	Nasal secretion	.....	.....	None
27	3 1-2	3-1 2	Coughing	Less irrit. than 2	Throat better	None
28	...	1 3 2	Increased secret.	Burning	.....	None
29	1 2-3	1 2-3	.....	Occ. throat irrit.	Dry throat	None
30	1-3 2	1 3 2	.....	.....	Bad taste	None
31	...	...	.....	.....	.....	None
32	1-2 3	1-2 3	.....	Most irritating	.....	None
33	1-3 2	1-3 2	.....	.....	Most irritating	None
34	2-3 1	2-3 1	Cough, hoarse	Clearing throat	Better, same as Lucky	None
35	1 2 3	1 2 3	Least irritating	Most irritating	.....	None
36	...	...	Stronger	.....	.....	None
37	3 1-2	3 1-2	.....	Least irritating	.....	None
38	...	...	.....	.....	.....	None
39	2 1-3	2 1-3	Husky voice	Clearing throat	Less clear, bad flavor	None
40	3 2 1	3 2 1	.....	Least irritating	.....	None
41	2 1-3	2 1-3	Dryness	Dry tongue	.....	None
42	1-2 3	2 1 3	.....	More irrit. than 2	.....	None
43	1-3 2	1-3 2	.....	.....	Taste terrible	None
44	1-2 3	2 1 3	.....	More irrit. than 2 (cold)	Less irrit. than Camels	None
45	1-2 3	1-2 3	.....	Dry throat	.....	None
46	...	...	.....	Increased secret.	.....	None
47	...	2 1-3	.....	.....	.....	None
48	...	...	.....	.....	.....	None
49	1-3 2	1-3 2	.....	.....	Stinging	None
50	...	2 1-3	.....	.....	.....	None
51	1-2 3	2 1 3	.....	Heartburn	.....	None

\* Bar between numbers indicates no preference.

CHART 2.—RESULTS OF SMOKING (THREE WEEKS' PERIOD)—*Continued*  
Order of preference\*

Case No.	3 weeks period	1 week period	Diethylene glycol 1	Glycerine 3	Controls 2	Objective signs
52	1-2 3	2 1 3	Dryness	Dry throat	Singing	None
53	...	1 3 2	.....	.....	Dry throat	2 most
54	2 1-3	2 1-3	Cough, rough throat	Cough, rough throat	Cough, rough throat	2 least
55	2-1 3	2-1 3	.....	Sl. throat irrit.	.....	None
56	...	...	.....	.....	.....	None
57	2-3 1	2-3 1	Raw throat, cough	.....	.....	1 most
58	...	1-2 3	Irrit. throat	Slight hoarseness	Irrit. throat	3 most
59	1-2 3	1-2 3	.....	Irrit. throat, cough	.....	3 most
60	1-3 2	1-3 2	.....	.....	Clearing throat, sting	3 most
61	...	1-3 2	.....	.....	.....	2 most
62	1 3 2	1 3 2	.....	.....	Cough, tickling	None
63	3 2 1	3 2 1	Irrit. dry nose	.....	Irritating	None
64	3 2 1	3 2 1	Throat dry	Mild and best	Less irrit. than 1	2 most
65	1-3 2	...	Irrit. throat	Dry throat	Dry throat	None
66	3 2 1	3 2-1	Irritation	Less irritation	Milder than 1	None
67	1 3 2	1 3 2	Least irrit., headache	Less irrit., mucus	Dry nose, headache	None
68	...	3 1-2	Less dryness	Dry throat	Less dryness	1 least
69	1-3 2	1 3 2	Less dryness	Less dryness	Most irritating	None
70	...	...	.....	.....	.....	None
71	3 1 2	3 1 2	Dry throat	.....	Dry throat	None
72	1-3 2	1 3 2	.....	.....	More irritating	None
73	1 2 3	1 2 3	Least irritating	Most irritating	Slight irritating	2 least
74	1 2-3	1 2-3	Least irritating	Hoarse (cold)	Nose irrit.	3 most
75	...	1 2-3	.....	.....	.....	3 most
76	1 2 3	1 2 3	Less irritating	.....	Less than 3	3 most
77	1-2 3	1-2 3	.....	More irrit.	Most irrit.	2 most
78	1-2 3	2 1 3	.....	Harsh	.....	2 most
79	2 1-3	1 2 3	Irrit. tongue	Irrit. tongue	Least irrit.	3 most
80	2 3 1	2 1 3	More irrit. than 2	Slight dryness	Less irritating	2 most
81	...	1 2 3	.....	.....	.....	1 most
82	...	1 2 3	.....	.....	.....	1 most
83	2 1 3	2 1 3	Irritation	Most irritation	Less irritation	1 most
84	1 3 2	1-3 2	.....	.....	.....	None
85	3-2 1	3 2 1	No taste	Same as Lucky	Milder than Lucky	None
86	1-2 3	2 1 3	Unsatisfactory	Tongue sore	Better than 1	None
87	1-3 2	1-3 2	Good	Good	Irritating	None
88	1-3 2	1-3 2	.....	Better than 2	Dry throat	1 most
89	3 1 2	1-3 2	Tastes better than 2	Least irrit.	Irritating	None
90	1-3 2	1 3 2	.....	.....	Dry throat	None
91	1 3 2	1 3 2	.....	.....	Irrit. throat	None
92	1 2-3	1 2 3	Less irritating	More irritating	Cough (cold)	3 least
93	3 2 1	3 2 1	Strong and irrit.	Mild, less irrit.	Better than 1 (cough)	None
94	2 3 1	2 3 1	Harsh, impossible	Slight irrit.	Better than 3	1 most
95	2 3 1	2 3 1	Harsh, strong	Slight irrit.	.....	2 most
96	3 1-2	3 2 1	Bites	.....	Bites	2 most
97	3 2 1	3 2 1	.....	.....	Sl. burning tongue	None
98	1-3 2	3 2 1	.....	.....	Irritating	2 most
99	2 3 1	2 3 1	Hyperemia of throat	Slight irrit.	.....	1 most
100	3 1-2	3 2 1	.....	Cough	Irritating	None
101	3 2 1	3 2 1	.....	Best	Better than 1	None
102	1 2-3	1 2 3	.....	Irrit. tongue	Slight irrit.	None

\* Bar between numbers indicates no preference.

hyperplasia was 42 per cent, slightly smaller than the average. As a result it is concluded from the tabulation of this group that no definite relationship exists between the enlargement of the lymphatic tissue of the pharynx and the number of cigarettes smoked per day.

CHART 3.—SYMPTOMS ATTRIBUTED TO SMOKING (198 CASES)

	No. of cases	Without symptoms (%)	Cough, (usually morning) (%)	Husky voice (%)	Excess mucus (%)	Dry or irritated throat (%)	Dry or cong. nose (%)
5-14 cig. per day	38	28	21	7	6	20	0
15-24 cig. per day	119	24	25	13	8	27	5
25-40 cig. per day	41	18	32	9	7	21	5

As in the preceding investigation,<sup>7</sup> the controls or untreated cigarettes were favored less than the other two types. This latter finding may be accounted for by the increased burning rate of dry tobacco with a higher temperature of the smoke. This is in accord with Titkemeyer,<sup>8</sup> who believes the deleterious effects on the buccal mucous membrane to be due partly to the raising of the mouth temperature. He believes this factor is more important than the chemical constituents of the tobacco. This investigation would seem to support this view.

CHART 4.—LYMPHOID HYPERPLASIA OF PHARYNX AND NUMBER OF CIGARETTES SMOKED DAILY

	No. of cases	Per cent with lymphoid hyperplasia
5-14 cigarettes per day . . . . .	41	37
15-24 cigarettes per day . . . . .	132	48
25-40 cigarettes per day . . . . .	48	42
Average for group . . . . .	224	44

CONCLUSIONS. Careful objective examination failed to show any significant difference in irritation of the nasal or throat mucosa from cigarettes moistened with glycerine, diethylene glycol or in cigarettes with no hygroscopic agent.

The subjective symptoms or sensations of irritations, when present, were not marked enough in respect to distribution, character or degree to justify definite conclusions.

The lymphoid hyperplasia of the pharynx does not appear to have any relationship to the number of cigarettes smoked.

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## DISCUSSION

DR. ARTHUR W. PROETZ, St. Louis, Mo.:

I have been looking forward with a great deal of anticipation to this interesting paper of Dr. Ballenger's, because, as Dr. McMahon said, several of us have been working on the effects of cigarette smoke for the past year.

I do not at all want to talk about the relative merits of diethylene-glycol and glycerin. I should like to confine my remarks chiefly to methods.

You will probably become more and more conscious of work with cigarette smoke as times goes on. There have been more pseudo-scientific announcements made in regard to cigarettes than almost any other one thing, so that when anyone finally does submit a scientific statement it will have to be based on the most indisputable evidence that can be collected.

We found that there were so many variables in the production of cigarette smoke that the problem seemed almost hopeless. Irritability depends on many things: how fast tobacco is smoked; how moist it is; in what position it is held; how it is being smoked—that is, puffed quickly or puffed slowly; how long one holds the smoke in the mouth or lungs; and many other things.

Just to show you how these vary, I quickly mounted a few little tests that we made. These represent the sedimentation of cigarette smoke on a piece of moistened gelatinized paper. I will pass them around to you. They show the variety of tar deposit one obtains if



the smoke from one puff is allowed to settle upon the paper five minutes, seven and a half minutes or ten minutes. There is also a difference between the last puff of a cigarette allowed to burn without previous puffing and one smoked in the ordinary way. It is important also whether one bases his estimation on the first puff, middle puff, last puff, or all of them.

Some people smoke a cigarette halfway and then throw it away; some people smoke it to their fingers, and it makes all the difference. I mention these things chiefly to show what tremendous errors can creep into observations, and what a necessity exists for standardization of methods.

Here is a photograph of a machine which we built. It is about  $3\frac{1}{2}$  feet long, weighs 85 pounds and grinds and clicks and bangs and puffs and fumes. All it is good for is to smoke a cigarette. But the point is that it smokes cigarettes all exactly alike at a uniform rate; frequency, interval and dilution so that conclusions may be comparable—all in the interests of standardization.

When we came to subjective and objective examinations in the clinic, we were in still more trouble. So far we have made no comparative clinical experiments with smoke so that what I say is not directly referable to what Dr. Ballenger says in regard to his cigarettes. As a background for our own investigations, we have made some 2000 tests, of which, unfortunately, I was able to tabulate only 800 or 900 before I came, trying to determine how nearly accurate individual observation of throats in general may be, how nearly subjective symptoms corresponded with objective symptoms in the clinic; trying, in other words, to find some basis on which to build any kind of statistics based upon visual information.

I will not go into great detail because the time is short, but, roughly, throats were graded as follows:

No. 1 was a perfectly negative throat;

No. 2 was merely red enough so that it could not be regarded as strictly negative;

No. 3 was a definitely red throat; and

No. 4 was an acutely inflamed throat; the type of throat we encountered only once, I think.

There were eight observers, all trained in the same clinic. All observers agreed 28 times or only 15 per cent (of these 853 investigations): Two opinions, 112 times, or 62 per cent; Three opinions, 26 times, or 14 per cent of the time; and four opinions once, or 0.5 per cent of the time. Thirteen of these records were marked incomplete. Temperature and humidity were watched as well as general weather conditions.

I may say the examiners had no idea what the other examiners were doing. The record was kept by an assistant.

In regard to the subjective sensation of irritation as compared with the objective findings of people who were subjectively normal; in 41 instances they were marked No. 2, and in 39 instances "very red throat," No. 3. In one instance the patient was marked "extremely red throat," No. 4.

These subjects consisted of a group of senior medical students and stenographers, assistants and workers in the clinic. They were not people who came there with any disease, but they were normal individuals and unprejudiced, as they had no idea what it was all about.

The whole point I wish to make is that we need a much more definite means of determining these things, if we are going to arrive at any tenable conclusions.

Dr. Ballenger's presentation goes into great detail in trying to eliminate sources of error. I hope he will carry it even farther. I was interested in his mention of the Tallqvist scale. It occurred to me to develop a colorimeter and once more trouble began, because all parts of the throat are not the same color. If one could develop a colorimeter to measure the least affected areas, the deepest red, supplemented by over-all readings, one might develop a formula to express a standard change in the throat; but even aside from that, we are going to have to determine just what it is in a throat that constitutes irritation.

I did not get up to confuse the issue, as I seem to have done, but the whole problem is so confusing that I simply want to call your attention to the diversity of effects and to the great care that must be exercised in reaching any conclusions.

May I congratulate Dr. Ballenger on his very fine beginning?

DR. JOHN F. BARNHILL, Miami Beach, Fla.:

Newspapers report that the rate of smoking in the United States has increased eight billion cigarettes per year for the past several years. Is this enormous increase a serious menace to the well being and health of our people?

Dr. Ballenger did not mention the bad effects, if any, of constant cigarette smoking on the individual. There are many smokers who smoke one cigarette after another. It would be of interest to have Dr. Ballenger's views of the ultimate effect on such an addict.

DR. WILLIAM B. CHAMBERLIN, Cleveland, Ohio:

It probably is not pertinent to this discussion, which is relative to the effect of cigarette smoking upon the upper air passages, but the

reports that have been rather prevalent of late of so-called tobacco angina are interesting. In this connection I happened to see, not so many years ago, a reference to 300 cases of gangrene of the leg. These cases of gangrene were reported, by the way, from Mt. Sinai Hospital, New York, as occurring almost exclusively in Bohemian Jews, who were inveterate cigarette smokers.

DR. D. C. JARVIS, Barre, Vt.:

Nearly ten years ago the Department of Industrial Hygiene of the U. S. Public Health Service came to Barre to study silica dust inhalation, and their personnel included a full-term medical man who stayed with us two years and a half, and a dust-count engineer who stayed with us over a year. In due course of time the dust-count engineer came to the office complaining of cough, which he explained had appeared since coming to Barre. I thought his cough was due to the dusty environment of the granite sheds where he worked, and told him when he adapted his respiratory tract to this environment, the cough would disappear, as it generally does with apprentices who enter the industry; but he told me his time in the shed included only the gathering of dust samples, and all the rest of his work was done outside.

I asked him if he had done any work or heard of any work done on cigarette dust count. He said he did not know of any work of that kind and later, because he was an inveterate smoker, I asked him if he would do this. He returned after a few days and informed me that he had done some work on himself and his friends at varied puffs, and he could safely say each time we inhale from a cigarette, we take in 120,000 particles of dust, 10 microns or under in size; so apparently we were dealing with a mild dust problem which might overload the lung lymphatics and bring about cough.

We decided to apply the wet filter principle and, after discussion, this was applied by moistening the mouth end of the cigarette with saliva for about  $\frac{1}{4}$  inch, before lighting and starting to smoke it. Dust counts made subsequently showed he controlled the greater part of the dust in this way and developed a cool smoke, and his cigarette cough disappeared within two weeks. Since that time when individuals I know to be smokers of cigarettes, have presented the symptom of cough, I have suggested that they moisten the cigarette for  $\frac{1}{4}$  inch to cut down the dust phase and produce a cooler smoke, and in the majority this results in the disappearance of a cough within two weeks. If the cough does not markedly disappear in two weeks' time, one looks elsewhere for the source of the cough.

DR. HARRIS VAIL, Cincinnati, Ohio:

I believe Dr. Ballenger has made a real contribution to medicine. Personally, I dislike very much the advertising that one brand of cigarettes is making, but that is neither here nor there. I think one thing has not been mentioned so far in the discussions, and that is the fact that the health of the posterior sinuses of the nose may be a very important factor in the symptoms which the smokers have. I believe that if you would study a similar series of non-smokers with the same amount of health in the sinuses, you would find about the same results as are found in the case of the smokers.

There is no doubt that many of the symptoms of postnasal irritation and pharyngeal irritation come from posterior sinusitis. I should like to ask Dr. Ballenger if he has examined carefully the sinuses of the cases he was studying in his experiments.

DR. SAMUEL SALINGER, Chicago, Ill.:

I am a cigar smoker and, as such, have assumed a more or less tolerant attitude toward the finely chopped-up spinach which has to be treated with hygroscopic agents and then wrapped in paper, and which we commonly call cigarettes; however, I am not prejudiced on that account.

I had the pleasure of participating in this test with Dr. Ballenger, and one of my first conclusions was the absolute worthlessness of subjective sensations. That was proven to me very conclusively by one fact. Two of my subjects were physicians, who had been in the habit of smoking one particular brand of cigarette which I do not care to mention.

Their subjective reactions to Nos. 1, 2 and 3 were that they were vile, viler, and vilest, and they could scarcely wait for the day when they could go back to their favorite cigarette. They resumed this favorite in the fifth week and, after smoking it for several days, had to give it up because it did not taste right and then adopt some other cigarette, proving very conclusively how much psychology enters into the enjoyment of a cigarette.

Objectively my findings were practically those that Dr. Ballenger detailed to you. I could not find the slightest difference in the local reaction between 1 and 2, or 2 and 3. It has always been my impression that such local findings as I was able to determine were based more on the quantity of tobacco consumed than on the relative quality of it.



I differ slightly with Dr. Ballenger when he makes the statement he has never been able to detect a smoker's throat. I have had occasion many a time to have a patient under examination who displayed such a violent redness of the soft palate and fossæ, that I was prompted to ask whether he had a sore throat and, much to my surprise, the patient would reply, "No," adding that he was not conscious of any subjective symptoms.

DR. M. G. MULINOS, New York, N. Y.:

It is presumptuous on my part to discuss a clinical paper. I am a pharmacologist, but, since I was quoted and partly misquoted, I think I should straighten out some of the points made.

First of all we at Columbia worked on rabbits' eyes, not the nose and throat. The smoke was not passed over the eye but made into a solution. The smoker was invented for the purpose and we measured edema, not redness, due to the fact that in the mucous membrane of the eye, at least, as soon as we obtained what we called a one-plus inflammation, the blood-vessels would be shut by the edema and the mucous membrane would be paler than normal, and, consequently redness as an index of inflammation was worthless.

As far as the irritation being due to temperature changes is concerned, I know that one cigarette company claims that the smoke from their cigarettes, due to the menthol it contains, is actually cooler than any other. We\* have made over 1000 observations on cigarette smoke emerging from the mouth end of the cigarette, and found that until the burning cigarette actually touched the thermocouple, there was absolutely no difference in the temperature of the smoke or air emerging from the mouth end of the cigarette, the mouth end in this case, of course, being the pipe holder; and, therefore, there is probably nothing to the contention that a cigarette smoke is warmer or cooler than any other smoke.

One other point was that our work at Columbia was substantiated by a double number of experiments performed by Dr. Wallace at New York University,† a paper that was not quoted—the work of Sharlit, that was quoted, was not based upon anything but a personal opinion, without any figures. The work of Dr. A. J. Carlson, at Chicago, was based upon the amount of saliva secreted, and that was based on an unfair assumption, due to the fact that all I have to do is think of cigarette smoke, and I salivate, probably protectively, and

\* Mulinos and Cockrill: *Arch. internat. de pharmacod. et de ther.*, **58**, 200, 1938.

† *Arch. Otolaryngol.*, **23**, 306, 1936.

I suppose that most of you who smoke much, salivate in anticipation—consequently, that is out.

The only paper that remains in comparison to the objective experiments of ourselves is that of Haag, who performed only a small number of experiments and may or may not have been able to repeat our experiments. We are willing to grant his experiments were against ours. The point remains only his paper was against the fact that when you add hygroscopic agents to a cigarette, it depends on the chemical nature of the hygroscopic agent whether the smoke is more or less irritating. The point was that both cigarettes, or all three cigarettes—two containing hygroscopic agents, and one not—were all very irritating and, due to the fact that both Wallace and ourselves performed hundreds of experiments and treated the data statistically, we were able to find a slight difference between the three.

Whether the patient smoking for years, not a week or two, can accumulate this irritation, to show up clinically by evidence such as shown by the second speaker, which is objectively immeasurable, is a question for you as clinicians to settle, not for us in the laboratory.

DR. H. C. BALLENGER (in closing):

I fully agree with Dr. Proetz in his statement that the clinical investigations perhaps are not of great value. Certainly any that have been done to date, including my own, would not justify definite conclusions. I hope Dr. Proetz (and I am sure he will) will develop a more precise method of making the clinical examination. The laboratory work is perhaps of more value; but that is of necessity on animals. However, the reports which have made the greater impression are the clinical reports, even though they are of less value.

Undoubtedly Dr. Vail's comment that the postnasal discharge is from a sinus rather than smoking, is true, and I fully agree with him in that respect; however, we put down the symptoms as the patients gave them to us and in most of them they attributed the mucus secretion to the smoking. Roentgenographic studies of the sinuses were not done.

Dr. Mulinos is quoted correctly in the paper. Due to hasty delivery, his work was grouped with that of Flynn. The technique and results of both men are reported in some detail in a previous paper.

## Memorials



DUNBAR ROY



## DUNBAR ROY

1866-1937

CHARLES DUNBAR ROY departed this life on July 5, 1937, at his home in Atlanta, Georgia, after a short illness, although he had been in failing health for a number of years. Always a loyal Fellow, he was a member of the Council at the time of his death, but was unable to attend either of the last two meetings on account of illness during the current year. It was rare, indeed, for him to miss an annual meeting, this having occurred only five times since 1920, during which period he presented four papers before the Association. For many years he maintained his office for the practice of Ophthalmology and Otolaryngology in the Grand Opera House Building in Atlanta.

Dr. Roy was born at Atlanta, Georgia, November 21, 1866. His father, Gustavus Garnett Roy, a practicing physician, was a Virginian of Scotch ancestry, and his mother was Flora Dillard, of English extraction, born in Greensboro, Alabama. His early life was spent in the countryside of Virginia along the Rappahannock River, but he attended the public schools in Atlanta until 1880, when he entered Richmond College, at Richmond, Va., graduating with the degree of A.B. in 1884. He then took up the study of medicine at the University of Virginia, under the preceptorship of his father, as was customary at that time, obtaining his medical degree in 1889. He served a rotating internship for eighteen months in the City Hospital of New York City, where he also began the study of Laryngology, as well as in the New York Polyclinic, followed by a year's study of Ophthalmology and Otolaryngology in Europe. He returned to Atlanta in 1893 and at once became associated with the outdoor clinic of the Atlanta Medical College. Later on in his medical career he filled the position of Otolaryngologist to the Grady Hospital, where he eventually became Consulting Otolaryngologist, as well as to the Georgia Baptist Hospital, St. Joseph's Infirmary and the Atlanta Hospital. In several of these institutions, he was also Ophthalmologist.

Dr. Roy's teaching career began when, soon after his return to Atlanta, he was appointed Professor of Diseases of the Eye, Ear, Nose and Throat in the Southern Medical College, and later Clinical Professor of Ophthalmology and Otolaryngology in Atlanta Medical College, which institution later became the medical department of Emory University. He became Emeritus Professor in 1926. That he retained his loyalty to his Alma Mater is shown by the fact that he was President of the Atlanta Association of the University of Virginia Alumni from 1923 to 1926.

Dunbar Roy became a Fellow of the American Laryngological Association in 1913, was first Vice-President in 1921, President in 1935 and after that a member of the Council until his death. He was Chairman of the Section on Otology, Rhinology and Laryngology of the American Medical Association in 1910, Vice-President and Chairman of the Southern Section of the American Laryngological, Rhinological and Otological Society in 1903, and President in 1923. He was a Fellow of the American Ophthalmological Society and obtained his certificate from the American Board of Otolaryngology in 1925. During the World War, in 1917 and 1918, he was a First Lieutenant, M. C., in charge of the Southeastern Aviation Unit, U. S. A. He was also a delegate from our Association to the International Otolaryngological Congress which met in Copenhagen, Denmark, in 1928.

In 1924, the University of Virginia elected him to membership in the Phi Beta Kappa Honorary Fraternity. In addition to the above-mentioned societies, he was a Fellow of the American Otological Society, Honorary Fellow of the Southeastern Surgical Congress and formerly Chairman of the Eye, Ear, Nose and Throat Section of the Southern Medical Association.

Dr. Roy was, of course, a life-long Democrat in politics and his religious affiliation was with the Protestant Episcopal Church. He belonged to the Beta Theta Pi College fraternity. He contributed many scientific papers, mostly of a practical nature, the following having appeared in the *Transactions* of the Association: "Oto-laryngological Diseases Among Negroes," 1915. "Epithelioma of Posterior Pharyngeal Wall Cured by Electro-cautery," 1916. "Foreign Body (Carpet Tack) in Bronchial Tube," 1918.

"Intranasal Ethmoid Exenteration," 1921. "Some Clinical Observations on Nasal Hemorrhage," 1933. President's Address, 1935. "The Frequency and Effect Upon the Eustachian Tube of Residual Lymphoid Tissue in the Nasopharynx," 1936.

He was married to Carrie Ellett, of Richmond, Va., who survives him. Always popular, companionable and affable, youthful in appearance considering his age, a very loyal Fellow of the Association and a regular attendant; his presence surely will be missed at our Annual Meetings.



LORENZO BRUCE LOCKARD



## LORENZO BRUCE LOCKARD

1872-1937

DR. LORENZO B. LOCKARD was born in Toledo, Ohio, November 12, 1872, and died in Denver, Colorado, July 3, 1937. His father was Lorenzo B., and his mother, Phoebe M. (Archibald) Lockard.

Dr. Lockard received his early education in the schools of Toledo and Syracuse University, and his medical education at the University of Pennsylvania, from which he graduated in 1894. He took post-graduate work in Europe, mostly in Vienna, where he was first assistant for one year to Dr. M. Hajek (later Professor Hajek of the University of Vienna). After his graduate work abroad, he returned to Toledo, Ohio, to practice Laryngology, Rhinology and Otology, and on January 31, 1900, he was married to Lois C. Lytle, of Toledo, Ohio.

On account of failure in health he moved to Pasadena, California, where he remained but one year, moving to Denver, in 1901. He immediately drew to him a large clientele and became an assistant in Otolaryngology in the Denver College of Medicine. Because of his tremendous energy and large practice, his health again failed in 1904, and he was compelled to take two years rest. During these two years he wrote his book, "Tuberculosis of the Nose and Throat," which is a standard even today.

The writer had the good fortune to be associated with Dr. Lockard during this period and for nine years thereafter and felt honored to be consulted and to share his material in the compiling of this book. It is his belief that no one else except Mrs. Lockard knew the amount of energy expended in writing this monograph.

Dr. Lockard was compelled to work and rest intermittently for a number of years, but he always remained courageous and in good spirits, in spite of this handicap.

The American Laryngological Association elected him a member in 1912 and in the same year he was elected a member of the American Laryngological, Rhinological, and Otological Society.

He had been a member of the American Academy of Ophthalmology and Otolaryngology since 1900, being a first Vice-President in 1909 and 1910. He was an outstanding member of his local as well as the national medical societies, and he possessed a host of friends both among the profession and the laity.

His work in medicine is acknowledged both at home and abroad and we have lost a wonderful physician and a true friend.

T. E. C.





JOHN WOODFORD FARLOW



## JOHN WOODFORD FARLOW

1853-1937

JOHN WOODFORD FARLOW, a distinguished Laryngologist of Boston, and during the latter part of his life Librarian of the Boston Medical Library, died at his summer home in Manchester, on the 23d of last September, at the age of eighty-four. His residence in Boston was 127 Bay State Road. He was born in the city of Boston, Mass., August 28, 1853.

His father, John Smith Farlow, a merchant and later a railroad president, was born in Ireland and was of English and Irish descent. His mother, Nancy Wight Farlow (Blanchard) was born in Boston and of American parentage.

The early life of Dr. Farlow was spent in Boston and in the nearby town of Newton, Mass., where he attended public schools until June, 1870. Then he entered Harvard College, from which he graduated with the degree of A.B. in 1874, and received his M.D. from the Harvard Medical School in June, 1877. He was interne in the Boston Lying-in Hospital for a term of three months and later began the study of Laryngology at the Harvard Medical School under our distinguished Fellow Member, Dr. Frederick Irving Knight. He then spent two years in Vienna, Strasburg and Paris, and, upon his return in 1880, opened an office for general practice in Boston. Soon after he became connected with the Boston Dispensary, the Staniford Street Dispensary, and the Free Dispensary for Diseases of Women. He was later connected with the Carney Hospital, South Boston, (where he founded a department of Diseases of the Nose and Throat), with the Boston City Hospital and the Free Home for Consumptives in Boston; in the latter part of his life he was Consulting Physician for Diseases of the Throat at the Boston Dispensary. He was Clinical Instructor of Laryngology at the Harvard Medical School, and was on its Faculty from 1892 to 1906. He was admitted to Fellowship in the American Laryngological Association in 1892, where he held the offices of Vice-President in 1898 and of President in 1901.

Other societies of which he was a member were the Massachusetts Medical Society, the American Medical Association, the Boston Society for Medical Improvement, the New England Oto-Laryngological Association and the Boston Medical Library. He retired from active practice in 1916, and became the Librarian in charge of the Boston Medical Library. He was a former President of the Massachusetts Library Association, and of the Medical Historical Club of Boston.

Dr. Farlow is survived by his wife, the former Annie Hardon Burr, whom he married in 1877, a daughter, Mrs. William R. Castle, wife of the former under-Secretary of State and a son, John Smith Farlow of Boston. Dr. Farlow's principal diversions were European travel and motor trips. He was a Protestant and a liberal Republican.

Dr. Farlow was a man of particularly broad education. His original training in Laryngology under Dr. Frederick Irving Knight and his close friendship with that master of Otology, Dr. Clarence J. Blake, were especially fortunate; and the high position which he attained among the foremost specialists in Boston and elsewhere was ample proof of his superiority. He was indeed a cultivated gentleman of the best type and a scholar of unusual attainment, accurate alike in his judgment of men and their published statements. Of a quiet nature, he was genial, generous and thoroughly appreciative of the good in others. That he was respected as a Laryngologist is well shown by the writings which he has left. When his professional activities became too burdensome, he was appointed with unanimous approval custodian of the Boston Medical Library. To this position he brought the dignity of his own personality and learning, in addition to his peculiar fitness as a representative of the best in Medicine. He was beloved everywhere, and the Library prospered under his management until the end of his career.

D. B. D.

PAPERS PUBLISHED IN THE TRANSACTIONS OF THE AMERICAN LARYNGOLOGICAL  
ASSOCIATION

"A Case of Partial Laryngectomy," 1892.

"Two Cases of Nasal Tuberculosis," 1893.

"A Case of Polypoid Lipoma of Larynx," 1895.

- "A Case of Myxoedema of the Throat," 1896.
- "A Case of Subglottic Fibroma," 1897.
- "A Case of Disease of Accessory Sinuses," 1898
- "Is the So-called American Voice Due to Catarrhal or Other Pathological Conditions of the Nose?" 1899.
- "Ozaena of Sphenoidal Origin," 1900.
- "Cases of Paralysis of the Right Vocal Cord," 1901.
- "President's Address," 1902.
- "Facial Erysipelas From Erosion of the Nasal Septum," 1903.
- "Two Cases of Chronic Pemphigus of the Mouth," 1904.
- "Ethmoid Disease," 1905.
- "Case of Spasm of the Oesophagus," 1907.

SOME FURTHER PUBLICATIONS NOT INCLUDED IN THE TRANSACTIONS OF THE  
AMERICAN LARYNGOLOGICAL ASSOCIATION

- An article on "Manuel Garcia," published in the Boston Med. and Surg. Jour., April 20, 1905.
- Address on the "Medical Library and Its Contents" for the Kings County, N. Y., Medical Society, published in the Aesculapian, October, 1908.
- Address on the "Importance of the Medical Library to the Medical Profession," before the Hartford, Conn., Medical Society, February 19, 1912, published in the Yale Medical Journal, March, 1912.
- Dr. Farlow also wrote the Reports of the Librarian for the Annual Meetings of the Boston Medical Library for twelve years.
- "Five Cases of Large Visible Pulsating Artery on the Posterior Wall of the Pharynx," Boston Med. and Surg. Jour., 1887. Also in *Annals de l'Oreille*, Paris, 1887.
- "On Examination of the Throat With Especial Reference to the Manner of Using the Tongue Depressor," 1887.
- "Hypertrophy of the Glandular Tissue at the Base of the Tongue," Boston Med. and Surg. Jour., 1888.
- "Use of Resorcin in Whooping Cough," Boston Med. and Surg. Jour., 1890.
- "A Case of Alleged Naphtha Poisoning in a Rubber Factory" (with R. H. Chittenden), 1892.
- "Is Not Too Much Importance Attached to the Condition of the Tonsil?" Boston Med. and Surg. Jour., 1893.
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- "Treatment of Prolongation Forward of the Nasal Septum," Boston City Hosp. Med. and Surg. Rep., 1898.
- "On Some Forms of Adenoid Disease, Which Are Often Overlooked, and on Conditions Which May Simulate Adenoid Disease," Boston Med. and Surg. Jour., 1898.
- Chapter on "Methods of Examination and Diagnosis of Diseases of the Nose and Throat," in *American Text Book of Diseases of Eye, Ear, Nose and Throat*, Philadelphia, 1899.



JOHN PRICE BROWN



## JOHN PRICE BROWN

1844-1938

DR. JOHN PRICE BROWN, of 29 Edgewood Crescent, Rosedale, near Toronto, Canada, was born in Manchester, England, in 1844, and died at his home on April 3, 1938, in his ninety-fifth year. His father, Joshua Brown, Esq., who was a farmer, lived to the great age of nearly one hundred years, while his mother, Sarah Price, almost equalled him in her longevity, dying at the age of ninety-five years. Joshua Brown was the author of a "Treatise on the Laws of Matter and Motion." He was a cousin of the poetess Felicia Dorothea Hemans. The family came to Canada in 1850, settling on a farm in the town of Walsingham, Norfolk County, Ontario. During his boyhood, Dr. Brown worked upon the farm. His early education was received at home and later he studied at the University of Toronto in the Department of Arts, and in 1868 graduated from its Department of Medicine, on which occasion he won the Star and the University gold medals, the first graduate in Medicine at this University to so distinguish himself.

Following graduation in Medicine, he entered upon a general practice in the village of Hollin, later removing to the town of Galt, where he remained for eighteen years, attaining the position of Coroner and winning the confidence and affection of the community. Abandoning general practice, he devoted himself to the study of Laryngology under the late Dr. Ernest L. Shurly of Detroit, and then went to Edinburgh, where he secured the degree of F.R.C.P., and returned to the University of Toronto the next year to graduate as a specialist. He then established himself in Toronto, where he gradually developed a successful practice and an increased proficiency in his specialty. In the year 1901 he was elected a Fellow of the American Laryngological Association, through the sponsorship of his friend, Dr. Shurly. He became Emeritus Fellow in 1914.

During his professional career in that city, he was a founder of the Toronto Western Hospital for which he drew the plans, and served as Chairman of the Building Committee for the original structure, and as a Governor for many years, while continuing a large private practice.

While not an important contributor to Medical Literature, he published a textbook on Laryngology which was used at the Toronto University for several years. In 1910 the difficulty which eventually destroyed his eyesight appeared and within a few years

he was obliged to retire from practice. Since then, when total blindness had overtaken him, he turned to his typewriter for occupation and relief of mind, and with his own hands wrote many highly characteristic and interesting letters to his various friends. In his student days he had developed a decided interest in the writing of short stories which he sold to local publications. Several years ago he again turned to fiction and published a number of historical novels, in which he was particularly versed. They were of distinct value. The first of them was "Hickory of the Lakes," after which came "How Hartman Won," "The Builders," "In the Van," "The Macs of '37" and "Laura the Undaunted." All of these appeared under the nom-de-plume, "Eric Bohn."

During the eighteen years that he spent at Galt, he met and married Agnes, daughter of William Graham, Esq., who died eleven years later. His second wife was Florence Ellen, daughter of Col. G. S. Jennings of the United States Army.

The record of his original scientific work will be found in the papers published by him in the *Transactions* of the American Laryngological Association.

He was a collaborator on the *Journal of Laryngology*, of London. He was a Fellow of the American Laryngological, Rhinological and Otological Society.

Dr. Brown was a member of the Anglican Church but became a Presbyterian when he married his first wife. He was for many years one of the Board of Managers of Old St. Andrew's Church, Jarvis Street, Toronto. His favorite pursuits, in his later years, were novel writing and chess playing. He had no interest whatever in outdoor sports. In politics, he was a lifelong Conservative.

Dr. Brown is survived by two sisters, Miss Helen S. Brown and Mrs. Charles Thompson, both residing in California, two sons, Newton Harcourt Brown, of the Great West Life Assurance Company, and Frank Erichsen Brown, K. C. '00 C, both of Toronto; ten grandchildren and seven great-grandchildren.

PAPERS PUBLISHED IN THE TRANSACTIONS OF THE AMERICAN  
LARYNGOLOGICAL ASSOCIATION

"Rubber Splints in Treatment of Septal Curvature," 1902.

"Sarcoma of Nose," 1903, 1905.

"Operation for Septal Deflection," 1906.

"Two Unusual Frontal Sinus Cases," 1908.

"Recent Cases of Nasal Sarcoma," 1909.

"Operations on Malignant Growths of Upper Air Tract," 1910

"Spindle Cell Sarcoma of Larynx," 1911.

"Malignant Diseases of Upper Air Passages; 2 Cases of Epithelioma," 1912.

**Executive Sessions**  
**1938**





## FIRST EXECUTIVE SESSION

MONDAY, MAY 2, 1938

THE First Executive Session of the Sixtieth Annual Congress was called to order at The Marlborough-Blenheim, Atlantic City, New Jersey, by Dr. John F. Barnhill, President of the Association, immediately following the first scientific session.

On recommendation of the Council, Dr. Vilray P. Blair, of St. Louis, was elected to Corresponding Fellowship; Dr. Henry Hall Forbes of New York City and Dr. Elmer L. Kenyon of Chicago were elected to Emeritus Fellowship; and the following were elected to Active Fellowship: Dr. Charles D. Blassingame, Memphis, Tenn.; Dr. George E. Hourn, St. Louis, Mo.; Dr. Chevalier L. Jackson, Philadelphia, Pa.; Dr. Samuel Salinger, Chicago, Ill.; and Dr. Harris H. Vail of Cincinnati, Ohio. The membership now stands as follows: 5 Honorary Fellows; 20 Corresponding Fellows; 16 Emeritus Fellows and 100 Active Fellows.

The President appointed as Auditing Committee, Dr. Ernest M. Seydell of Wichita, Chairman; Dr. Gordon Berry of Worcester, and Dr. C. Stewart Nash of Rochester.

The election of a Nominating Committee by the Association was as follows: Dr. Harris P. Mosher, Chairman, Dr. William B. Chamberlin, Dr. George M. Coates, Dr. Chevalier Jackson and Dr. Burt R. Shurly.

As there was no further business, the meeting adjourned.

## ANNUAL BUSINESS MEETING

TUESDAY, MAY 3, 1938

The Annual Business Meeting of the Association was held at The Marlborough-Blenheim, Atlantic City, New Jersey, at 10 A.M., on May 3, 1938, President John F. Barnhill presiding.

The report of the Secretary, Dr. James A. Babbitt, with the recommendations from the Council, was then read.

### REPORT OF THE SECRETARY

The Fifty-ninth Annual Congress of the Association was held at The Marlborough-Blenheim, Atlantic City, New Jersey, on May 30, June 1 and 2, 1937, under the Presidency of Dr. William B. Chamberlin.

There were sixty-six Fellows in attendance. All scheduled papers were read as listed on the annual program and there was ample discussion.

The following deaths of Fellows have been reported to the Council since the last Annual Congress:

DR. DUNBAR ROY, Atlanta Ga., Active Fellow, July 5, 1937

DR. LORENZO B. LOCKARD, Denver, Colo., Active Fellow, July 31, 1937

DR. JOHN W. FARLOW, Boston, Mass., Emeritus Fellow, September 23, 1937

DR. J. PRICE BROWN, Toronto, Ont., Emeritus Fellow, April 3, 1938

An informal preliminary meeting of five members of the Council was held in Chicago on October 12th in connection with the meeting of the American Academy of Ophthalmology and Otolaryngology. Matters of interest connected with the program, the location and official business of the annual meeting, and general items of interest were informally discussed as an aid to the President, who was temporarily confined by illness.

The Annual Meeting of the Council was held at the University

Club, in Philadelphia, Pa., on January 8, 1938, at 3 o'clock, with the following in attendance: Doctors Spencer, McMahon, Babbitt, Imperatori, Coates, Newhart and Shurly. Doctors Barnhill and Chamberlin were absent on account of illness.

The Council decided that the Sixtieth Annual Congress should be held at The Marlborough-Blenheim, Atlantic City, New Jersey, May 2, 3 and 4, 1938, in conjunction with the meeting of the Congress of American Physicians and Surgeons.

Dr. Charles J. Imperatori was reappointed Abstract Editor for the year.

Dr. James A. Babbitt was nominated as representative on the Board of Governors of the American College of Surgeons.

The Council at this meeting made the following nominations: To Emeritus Fellowship: Dr. Henry Hall Forbes of New York City, and Dr. Elmer L. Kenyon of Chicago; for Corresponding Fellowship: Dr. Vilray P. Blair of St. Louis; and for Active Fellowship:

DR. CHARLES D. BLASSINGAME, 414 P. & S. Bldg., Memphis, Tenn.

DR. GEORGE E. HOURN, University Club Bldg., St. Louis, Mo.

DR. CHEVALIER L. JACKSON, 3701 N. Broad St., Philadelphia, Pa.

DR. SAMUEL SALINGER, 25 E. Washington St., Chicago, Ill.

DR. HARRIS H. VAIL, 802 Carew Tower, Cincinnati, Ohio.

The resignation of Drs. Harry L. Baum and Samuel R. Skillern, Jr., and the withdrawal of Dr. Porter Stiles, from the Associate Fellowship, were accepted.

The suggestion of Dr. Imperatori that a space in the annual business meeting program be given for the introduction of newly elected Fellows of the Association with somewhat more formality, was approved.

The Council approved having invitations sent to Dr. Goodale of Boston and Mr. Negus of London, to present papers at this annual meeting; and at the suggestion of Sir St. Clair Thomson invitations were to be sent to Mr. Gavin Livingstone and Mr. Geoffrey Bateman of London to attend the annual meeting without presentation of papers. At the suggestion of the President of the American Laryngological, Rhinological and Otological Society similar invitations were to be sent to Dr. Holmgren of Stockholm and Dr. Coutard of Paris.

The Treasurer in his annual report to the Council presented a careful study of the depression of values in the Casselberry and Johnston Funds. Upon his motion, the Council voted to place one-half of the balance then in the Treasury, amounting to \$505.00, in the Johnston Fund.

Motion was made that the Secretary should furnish the Treasurer with full information from the minutes and records of the Association on the provisions of the Casselberry, Johnston and deRoaldes Funds. This has been carefully prepared and submitted to the Treasurer.

The Treasurer also presented a very careful analysis of the budget expenses of the Association and offered certain suggestions. It was decided, however, not to increase the dues for the present.

Dr. Imperatori automatically retired from the Memorial Fund Committee and Dr. Burt R. Shurly was appointed for a five-year rotating period.

The Council voted to continue the same Candidates' Nominating Committee under the Chairmanship of Dr. Fenton.

A motion was passed by the Council that the resolution of the Illinois State Medical Society against regimentation and federalization of the Medical Profession of the Nation as indicated in the Senate Joint Resolution No. 188 be approved for adoption by the Association at the annual meeting.

After dinner, the Council went into session as a Committee on the Whole for consideration of the proposed new Constitution and By-Laws. These were referred back to the committee for presentation at the Second Council meeting and possible adoption by the Association at its annual business meeting.

The second Council meeting was held at The Marlborough-Blenheim Hotel, Atlantic City, May 1, 1938, President Barnhill presiding.

The following Members of the Council were present: Drs. Barnhill, Spencer, McMahon, Babbitt, Imperatori, Coates, Newhart, Shurly and Chamberlin.

The Secretary announced the death of Emeritus Fellow Dr. J. Price Brown, Toronto, Canada, since the last Council meeting. The Secretary and Treasurer added brief supplementary reports to those given at the January meeting.



Upon the Treasurer's recommendation, the Council approved the investment of approximately \$1000 of the Casselberry Accrued Fund in a bond approved by the Financial Committee, Drs. Shurly, Babbitt and Imperatori; also approved offering \$250 in Casselberry prize next year, and urged special advertising of this for original research work. The Committee announced that no Casselberry award would be made this year.

The Council recommended a special contribution this year of \$50 to the Army Medical Museum for encouragement of their pathological and graduate work.

Motion was passed that the American Laryngological Association approve the attitude of the American Medical Association in relation to Socialized Medicine, and directed the Secretary to notify the American Medical Association and the Illinois State Society of their action after approval by the Association at this Annual Meeting.

The Secretary was directed to send letters of appreciation to Arthur Gidden, Esq., and Joseph G. Myerson, Esq., for their courteous legal advice in reference to constitutional changes and Memorial Fund bequests, respectively.

The Secretary was directed to send a congratulatory telegram to Dr. Delavan on his eighty-eighth birthday today and letters of Association greeting to Drs. Large, Sonnenschein, Forbes, Lewis, Hubbard, Pierce and Ingersoll.

Dr. Imperatori, of the Committee on Revision of the Constitution, was requested to prepare a draft of the minor and clarification changes to be presented at the Annual Business Meeting for adoption. After presentation of President Barnhill's social plans for this meeting, the Council adjourned.

At a brief special meeting following the Monday afternoon Scientific Session, the Council voted to recommend for election to Honorary Fellowship the name of Dr. D. Bryson Delavan at the Annual Business Meeting, May 3d.

Respectfully submitted,  
JAMES A. BABBITT,  
*Secretary.*

The recommendation of the Council that Dr. D. Bryson Delavan be elected to Honorary Fellowship was approved by a rising vote and the Secretary was instructed to so inform Dr. Delavan. The report of the Secretary was then approved and placed on file.

#### REPORT OF THE AUDITING COMMITTEE.

The Chairman of the Auditing Committee, Dr. Seydell, reported that the Committee had examined the Treasurer's books, checks and vouchers and found everything in perfect condition and in balance, and they wished to commend him for the excellence of his system.

Report accepted and placed on file.

## TREASURER'S REPORT

The submitted report is for the period from May 31, 1937, to April 30, 1938.

The balance as of April 30, 1938, deposited with the New York Trust Company, is \$1,976.67.

### FINANCIAL STATEMENT, THE AMERICAN LARYNGOLOGICAL ASSOCIATION, APRIL 25, 1938

#### GENERAL FUND

<i>Assets</i>	
Cash in bank . . . . .	\$1,976.67
Accounts Receivable: Unpaid Dues, 1938 Active Fellows (6 members) . . . . .	120.00
Total Assets, General Fund . . . . .	\$2,096.67

#### SPECIAL FUNDS

##### *Casselberry Fund*

Securities (at market value) . . . . .	\$2,860.00
Cash in Savings Account, Fidelity-Philadelphia Trust Company . . . . .	1,303.73
Total Assets Casselberry Fund . . . . .	\$4,163.73

### REPORT OF THE FIDELITY-PHILADELPHIA TRUST COMPANY FISCAL AGENT FOR

#### THE AMERICAN LARYNGOLOGICAL ASSOCIATION, INC.

##### *Casselberry Fund, No. 30,664*

		<i>Stocks</i>	Market as of 4/8/1938
Par value			
10 shares	1500 Walnut St. Corp. V. T. C. for \$3 Cuml. Preferred Par \$1.00 . . . . .		(No value)
			With Bonds
4 shares	1500 Walnut St. Corp. V. T. C. for Common Par \$1.00 . . . . .		(No value)

*Bonds*

\$3,000	Brandt, Wm. K., premises, 1708 Chancellor St., Phila., 1st Mtge. Series	(\$450.00)
	"A" Gold Reg. 3s due 9/1/1942	15
1,500	1500 Walnut St. Corp. 15 yr. Income	(\$330.00)
	1st Mtge. Reg. 6s, due 4/1/1950	@ 22 as a unit with com. and pref. stocks
1,000	United States of America Treasury Bond 3 1/8s due 6/15/1949-46	(\$1,060.00)
		106 2/32
1,000	United States of America Treasury Bond 2 3/4s due 3/15/1951-48	(\$1,020.00)
		102 4/32
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\$6,500	Total Bonds	

4/12/1938. Verified as to Securities,  
(signed) W. A. NIRAUG, Auditor

*Johnston Fund*

Securities (at market value)	\$1,885.00
Cash in Savings Account Fidelity-Philadelphia Trust Company	954.00

Total Assets Johnston Fund . . . . . \$2,839.00

REPORT OF THE FIDELITY-PHILADELPHIA TRUST COMPANY  
FISCAL AGENT FOR  
THE AMERICAN LARYNGOLOGICAL ASSOCIATION, INC.

*Johnston Fund, No. 30,664.1**Stocks*

Par value		Market as of 4/8/1938
25 shares	1500 Walnut St. Corp. V. T. C. for \$3	
	Cuml. Preferred Par \$1.00	(No value)
		With Bonds
10 shares	1500 Walnut St. Corp. V. T. C. for	
	Common Par \$1.00	(No value)

*Bonds*

\$3,750	1500 Walnut St. Corp. 15-yr. Income	(\$825.00)
	1st Mtge. Reg. 6s, due 4/1/1950	@ 22 quoted as a unit with Preferred and common stocks
1,000	United States of America Treasury Bonds, 3 1/8s due 6/15/1949-46	(\$1,060.00)
		106 2/32
<hr/>		
\$4,750	Total Bonds	

4/12/1938. Verified as to Securities,  
(Signed) W. A. NIRAUG, Auditor



*deRoaldes Fund*

Cash in Savings Account in Girard Trust Company, Philadelphia . . . . .	\$626.38
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TOTAL ASSETS (market values), April 25, 1938 . . . . .	<u>\$9,725.78</u>
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*Liabilities*

Unpaid Bills . . . . .	None
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## ACCRUED INTEREST

*Special Funds*

## JOHNSTON FUND

Cash in Fidelity-Philadelphia Savings Fund Account, as of May 31, 1937 . . . . .	\$467.42
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*Receipts*

1937

July 2	6 mos. int. \$1000 U. S. A. Treas. Bond, 3 1/8% . . . . .	\$15.63	
	Less Commission on above at 4% . . . . .	.63	
		<u>\$15.00</u>	
	Less semi-annual charge for safe keeping of securities . . . . .	2.50	
			12.50

Dec. 28 1938	Interest on Savings Fund Account . . . . .		9.10
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Jan. 8	6 mos. int. \$1000 U. S. A. Treas. Bond, 3 1/8% . . . . .	\$15.63	
	Less Commission on above at 4% . . . . .	.63	
		<u>\$15.00</u>	
	Less Commission to cover overdraft . . . . .	.04	
			14.96

Feb. 2	Amount deposited, as agreed upon at Council Meeting in Philadelphia on January 8, 1938, <i>i. e.</i> , one-half of amount in checking account as of December 31, 1937 . . . . .	505.00	
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	<u>\$1,008.98</u>
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*Disbursements*

1937 Sept. 14	Payment made to Dr. Stetson, honorarium, expenses	\$54.98
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	<u>\$954.00</u>
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## DEROALDES FUND

Cash in Girard Trust Company Savings Fund, as of May 31,		
1937		\$755.09
1937	<i>Receipts</i>	
Nov. 30	Interest on Savings Fund	13.89
		<u>\$768.98</u>
1937	<i>Disbursements</i>	
July 9	Tiffany & Co. for medal engraving, etc. (Dr. Dean)	142.60
		<u>\$626.38</u>

## CASSELBERRY FUND

Cash in Fidelity-Philadelphia Savings Fund Account, as of		
May 31, 1937		\$1,107.52
1937	<i>Receipts</i>	
June 2	6 mos. int. \$1000 U.S.A. Treas. Bonds 3 1/8%	\$15.63
	Less Commission on above at 4%	.63
		<u>\$15.00</u>
	Less semi-annual fee for safe keeping of securities	1.00
		<u>14.00</u>
Oct. 2	6 mos. int. \$1000 U.S.A. Treas. Bonds 2 3/4%	\$13.75
	Less commission on above at 4%	.55
		<u>13.20</u>
Nov. 3	Transferred from Principal to Income	\$32.19
	Less commission for transferring	.04
		<u>32.15</u>
Dec. 28	Interest on Savings Fund Account	22.26
1938		
Jan. 4	6 mos. int. \$1000 U.S.A. Treas. Bonds	\$15.63
	Less commission on above at 4%	.63
		<u>15.00</u>
March 1	6 mos. int. \$3000 1708 Chancellor St. 1st, 3s	\$45.00
	Less commission on above at 4%	1.80
		<u>43.20</u>
March 2	6 mos. int. 1708 Chancellor St. (Brandt)	\$45.00
	6 mos. int. \$1000 U.S.A. Treas. Bonds	13.75
		<u>\$58.75</u>
	Less commission on above at 4%	2.35
		<u>56.40</u>
	Total	<u>\$1,303.73</u>
<i>No Disbursements</i>		

The total value of the securities of the Association, with accrued interest, is, as of April 30, 1938:

Casselberry Fund . . . . .	\$4,163.73
Johnston Fund . . . . .	2,839.00
deRoaldes Fund . . . . .	626.38
Total . . . . .	<u>\$7,629.11</u>

## GENERAL FUND

*Receipts*

Cash in bank, May 31, 1937, New York Trust Company . .	\$1,699.50
Dues, 1937 (paid after May 31, 1937) . . . . .	\$180.00
Assessments, 1937 . . . . .	980.15
Dues, 1938: 91 Active members at \$20 . . . . .	1,820.03
2 Associate members at \$15 . . . . .	30.00
Sale of <i>Transactions</i> . . . . .	500.03
Refund, when Treasurer's Bond was reduced . . . .	5.79
Cash received from members at Banquet at Atlantic City . . . . .	177.00
	<u>3,693.00</u>
Gross Receipts, General Fund . . . . .	\$5,392.50

*Expenditures*

Jas. C. Muir, Lantern at Annual Meeting . . . . .	\$10.00
Jas. C. Muir, Running Lantern, Atlantic City . . . .	20.00
Marlborough-Blenheim, Banquet . . . . .	256.90
V. E. Speciale, Music at Banquet . . . . .	25.00
Sundries, Attendant, Convention Hall . . . . .	3.00
Telegram, Dr. Delavan . . . . .	.50
Waiters and waitresses . . . . .	20.00
Dr. C. G. Heyd, Expenses, as Guest . . . . .	25.25
T. D. Taussig, Bonding Treasurer . . . . .	37.50
Dr. J. A. Babbitt, Postage . . . . .	17.03
Dr. J. A. Babbitt, Honorarium . . . . .	300.00
S. A. Wilson Company, Multigraphing letters . . . .	3.25
K. McCord, Registrar fee . . . . .	10.00
S. A. Wilson Company, 500 envelopes . . . . .	5.25
Master Reporting Company, Report Annual Meeting . . . . .	164.26
M. J. Callahan, Flowers for Dr. Dunbar Roy . . . .	11.13

Dr. J. A. Babbitt, Postage . . . . .	\$35.00
Dr. C. J. Imperatori, Treas. and Abstr. Editor Honorarium . . . . .	200.00
R. H. Stetson, Honorarium (expenses) . . . . .	54.98
Dr. C. L. Jackson, Refund, charged assessment in error . . . . .	10.00
Dr. G. E. Hourn, Refund, same as above . . . . .	10.00
S. A. Wilson Company, 1000 letterheads . . . . .	10.25
C. Wm. Miller, Printing 150 Information ballots . . . . .	44.50
S. A. Wilson Company, Stationery . . . . .	11.00
Dr. J. A. Babbitt, Postage . . . . .	15.03
Wm. J. Dornan, Printer, 300 <i>Transactions</i> . . . . .	1,100.00
Dr. B. R. Shurly, R.R. fare, etc., to attend Council Meeting, Philadelphia . . . . .	49.25
Dr. C. J. Imperatori, Postage . . . . .	6.00
Dr. F. R. Spencer, R.R. fare, etc., to attend Council Meeting, Philadelphia . . . . .	121.60
Emlen & Co., Services in connection with investiga- tion of Walnut and Chancellor St. properties . . . . .	10.00
Dr. H. Newhart, R.R. fare, etc., to attend Council Meeting, Philadelphia . . . . .	79.25
Wm. J. Dornan, Printer, stationery . . . . .	67.00
Dr. B. J. McMahon, R.R. fare, etc., to attend Coun- cil Meeting, Philadelphia . . . . .	71.60
Amer. Laryng. Assn., deposit one-half of check book balance as of December 31, 1937 . . . . .	505.00
Dr. F. R. Spencer, Express charges . . . . .	.93
S. A. Wilson Company, Stationery . . . . .	3.83
S. A. Wilson Company, Mimeograph letters . . . . .	4.34
Dr. J. A. Babbitt, Stamps, expressage, etc. . . . .	18.08
Dr. C. J. Imperatori, Postage . . . . .	5.00
Wm. J. Dornan, Printer, stationery . . . . .	73.50
Exchange on Candian checks . . . . .	.62
Gross Expenditures . . . . .	\$3,415.83
Net Balance . . . . .	\$1,976.67

The sources of income of the Association are from the annual dues and the sale of *Transactions*.

Budget of expenditures approximate \$3000.

For a number of years, the heaviest expense, that of the *Transactions*, was not paid until after the collection of the annual dues



in January. In other words, 50 per cent of the last year's expenses were paid for by dues of the succeeding year. The assessment of last year, \$980, has corrected this.

The Council having favorably voted on the recommendation of the Treasurer, \$505 was deposited in the Johnston Fund, this amount being one-half of the balance of money in the treasury as of December 31, 1937.

The Treasurer sincerely hopes that this practice will be continued, and that the other Funds may be similarly added to.

The Treasurer's Bond has been reduced from \$15,000 to \$9000 because of the depreciation of the securities.

Respectfully submitted,

C. J. IMPERATORI,

*Treasurer.*

APRIL 30, 1938.

The report of the Treasurer was accepted and placed on file.

The Secretary recalled to the notice of the Association that in last year's Minutes the Treasurer was required by vote of the Association to prepare mimeographed copies of the itemized Treasurer's report for general distribution. The Council rescinded this, believing it was unnecessary and an arduous piece of work which was not particularly justified, as there would be a full report at this meeting.

The Secretary again called attention to the effort which was being made to dispose of old volumes of *Transactions* by sale, explaining that a special price would be made for any considerable number of volumes.

Report was accepted and placed on file.

#### INFORMAL REPORT OF THE LIBRARIAN

It is hardly worth while for the Librarian to come up to the platform before you to say that he cannot report progress. He will simply have to follow in the footsteps of his distinguished predecessors in this office, Dr. Barnhill and Dr. Shurly, and say that every effort has been made during a good many years past to establish the Library on some sort of foundation, but that all efforts have been fruitless up to the present time.

As I have reported before, and as my predecessors have reported, the effort has been made for many years to get the Army Surgeon-General's Library to take charge of our library, such as it is, and as we might hope to build it up, but owing to present conditions in Washington and in the Army and their lack of proper building and storage facilities, although they are willing to undertake this when the time comes, no progress has been made up to this time.

Respectfully submitted,

GEORGE M. COATES,  
*Librarian.*

This report was accepted.

Dr. Beck reported that he had recently had luncheon with the Surgeon-General, and there appears to be now a very definite plan for the new building in Washington, in which event the Surgeon-General will have the proper facilities and will coöperate with this Association in housing our library. This statement was made with the approval of the Surgeon-General. Dr. Coates corroborated this and after some discussion, the meeting continued.

#### REPORT OF ABSTRACT EDITOR

The Abstract Editor has the honor to report that the Scientific Proceedings of the Fifty-ninth Congress of the American Laryngological Association were abstracted and sent to the Archives of Otolaryngology, so that they were published in the October, 1937, number.

The Abstracts were published in book form and distributed to the members of the Association two weeks in advance of the Annual Meeting.

The honorarium was received from the Treasurer, and the necessary expenses paid.

Respectfully submitted,

C. J. IMPERATORI,  
*Abstract Editor.*

## REPORT OF THE NOMINATING COMMITTEE

The report of the Nominating Committee was made by Dr. Mosher, Chairman of the Committee. The following men were nominated for the Board of Directors of the Association for the year 1938-39:

- Dr. George B. Wood, Philadelphia, Pennsylvania.
- Dr. Ralph A. Fenton, Portland, Oregon.
- Dr. Frederick T. Hill, Waterville, Maine.
- Dr. James A. Babbitt, Philadelphia, Pennsylvania.
- Dr. Charles J. Imperatori, New York City.
- Dr. George M. Coates, Philadelphia, Pennsylvania.
- Dr. John D. Kernan, New York City.
- Dr. Burt R. Shurly, Detroit, Michigan.
- Dr. William B. Chamberlin, Cleveland, Ohio.
- Dr. John F. Barnhill, Miami Beach, Florida.

This report was accepted.

In the absence of Dr. Delavan, the report of the Committee on the Control of Cancer was read by the Secretary.

ACTIVITIES OF THE AMERICAN SOCIETY FOR THE CONTROL OF  
CANCER FOR THE YEAR 1937

The Women's Field Army of the American Society for the Control of Cancer enrolled approximately 100,000 women in forty-one states of the Union in its first drive for memberships in April, 1937, making available \$30,000 for expenditures for cancer education by the National Society and \$70,000 for further educational and promotional work in the several states by the State Women's Field Army Executive Committees.

An even more intensive educational program has been carried on in forty-four states during the past year and the second annual drive for enlistments has been under way since April, 1938. In 1937 approximately one and a half million pamphlets or circulars on cancer were distributed by the Society to the laity: in the drive this year about two and a half million pieces of literature have been sent out.

This educational drive is beginning to show results, for the physicians and the cancer clinics at the hospitals are reporting

more people coming for examination of lesions suspicious of cancer. A number of clinics are reporting a marked increase in the number of individuals coming in the *earlier* stages as compared with several years past.

The present Congress of the United States passed a joint resolution providing that the President issue annually a proclamation setting aside the month of April each year as Cancer Control Month and the first proclamation was made by the President on March 28th just past.

This past Congress also appropriated \$1,500,000 to the United States Public Health Service to carry on work in the cancer field. One-half of this is being spent for a building just outside of Washington, D. C.; the rest is being spent for research and other activities. A National Cancer Advisory Council has been appointed to assist the Surgeon-General of the United States Public Health Service in this program.

The American Society for the Control of Cancer celebrated its twenty-fifth Anniversary in March, 1938, with a dinner in New York City, a nation-wide radio broadcast and an exhibit on cancer in the International Building in Rockefeller Center for a week, attended by approximately 8000 visitors.

D. BRYSON DELAVAN,  
*Chairman, Committee on Cancer.*

This report was accepted and placed on file.

In the absence of Dr. Fenton, its Chairman, the report of the Candidates' Nominating Committee was read by Dr. Shea, accepted and placed on file.

#### REPORT OF CANDIDATES' NOMINATING COMMITTEE

Dr. Fenton has asked your secretary to prepare a report for the Annual Meeting from the communications he has sent to the Council.

On December 29, 1937, Chairman Dr. Ralph A. Fenton informed the Council that it was not found possible to assemble the entire Candidates' Nominating Committee at Chicago in the Fall of 1937 but from the composite suggestions of the various members of the Committee he submitted a list of 24 possible candidates



and asked further suggestions from the Council respecting any of these candidates and other names that might occur to them.

Since that time individual members of the Committee have stimulated the proposal of certain names, of which three formal nominations are in hand and three more will soon arrive. As there are already two remaining on the Candidates' list not yet acted upon, and no immediate openings for election, it is probably not necessary to stimulate further activity at the moment.

Respectfully submitted,

GEORGE M. COATES,

FRANK R. SPENCER,

JOHN J. SHEA,

JAMES A. BABBITT,

RALPH A. FENTON, *Chairman.*

JAMES A. BABBITT,  
*Secretary.*

At this point, in accordance with the recommendation of the Council at its January meeting, the five new Fellows elected to the Association, Doctors Blassingame, Hourn, C. L. Jackson, Salinger and Vail, were called to the platform and formally presented to the Association by President Barnhill, who opened his remarks with this sentence: "Gentlemen, you have just been elected to what the oldest member in the Association has called the greatest Society in the world, and we who are members of it and have been members for a number of years quite agree with him."

#### REPORT OF THE AMERICAN BOARD OF OTOLARYNGOLOGY

Herewith is the report on examinations given by the American Board of Otolaryngology at examinations held during 1937.

<i>City</i>	<i>Total exam.</i>	<i>Passed</i>	<i>Formal</i>	<i>Cer- tified</i>	<i>Condi- tioned</i>	<i>Pend- ing</i>
Philadelphia . . . . .	97	67	7	74	23	
Chicago . . . . .	103	68	12	80	22	1

Total Otolaryngologists certified to date, 2668.

Respectfully submitted,

W. P. WHERRY, M.D.,  
*Secretary-Treasurer.*

HARRIS P. MOSHER,  
*Chairman*

Dr. Mosher read the report and then said: "I think it will interest you and please you, as it has pleased the members of the Examining Board, to know that of the men conditioned, the majority come up and eventually work off their conditions. They get advice from the Secretary as to what to do, and they accept it very willingly, profit by it. It is a very great pleasure for the Examining Board to find that at the next examination, or perhaps the one after that, the majority of these conditioned men are passed."

Dr. Mosher's report was accepted and placed on file.

#### REPORT OF THE MEMORIAL FUND COMMITTEE

At the annual meeting one year ago it was urged that more information be sought relative to the effect of gift and estate taxes on bequests to our Memorial Fund.

In its investigations, your committee has been favored by the interest of the brother of our Fellow, Dr. M. C. Myerson. Joseph G. Myerson, Esq., is an Assistant General Attorney of the Commercial Investment Trust Corporation of New York City and is an authority on these matters. After careful review he informs us that though in a few instances it might be necessary to find a specific parallel case on which a judicial ruling has been passed, in order to make sure that "scientific research" is included under the exemptions listed, "in many states the amount of a bequest may be deducted in fixing the value of the estate for tax purposes." He recommends that we plan for these bequests and investigate the law in each case, where such is indicated. He generously offers to advise our Association without cost to us "as to any inquiry in a particular state and upon a stated set of facts." This is gracious of him and the Committee hopes our membership will give us an opportunity to present to him such proposed gifts and bequests for his analytical review.

In general it may be stated that:

1. Such a bequest would be deductible from the estate when fixing the value of the estate for transfer tax purposes. Also such deduction would lessen the tax rate on the entire estate if such deduction brings the estate total down into a lower bracket.

Example: in Massachusetts up to \$25,000, tax rate is 1 per cent; \$25,000-\$50,000, tax rate is 2 per cent.

2. If the gift is made before death, in installments or in one sum (up to a total 15 per cent of the income), it would be deductible from the income tax, as well as lessen the ultimate total that is subject to taxation at death.

3. Gifts up to \$5000 are exempt from the gift tax.

4. If given in the form of a stock that has appreciated greatly since its purchase, this gain would be taxable if the sale be made by the individual, not taxable if given to the Association and sold by it.

The Memorial Fund Committee invites each Fellow: to give serious consideration to arranging for some gift or bequest; and to permit the Committee to investigate the application of these tax laws to his individual case. It calls the attention of the membership to the continuing and permanent nature of this Fund, controlled by you and succeeding Fellows through your Council, the income of which is to be administered by your carefully chosen rotating committee of five. It suggests that each Fellow's loyalty to our Association, and his natural desire to project his scientific activities beyond his allotted life span, will combine to find this Fund an ideal agency for his need.

JAMES A. BABBITT (1938),  
GEORGE M. COATES (1939),  
GORDON BERRY (1940), *Chairman*,  
WILLIAM B. CHAMBERLIN (1941),  
BURT R. SHURLY (1942).

This report was accepted and placed on file.

#### REPORT OF THE BIOGRAPHER AND HISTORIAN

The Secretary in his report has recorded the deaths of two of our Active Fellows, Dr. Dunbar Roy of Atlanta, Georgia, and Dr. Lorenzo B. Lockard of Denver, Colorado, and two Emeritus Fellows, Dr. John W. Farlow of Boston, Massachusetts, and Dr. J. Price Brown of Toronto, Canada. The biographic histories of these Fellows with adequate photographs will be prepared and published in the *Transactions*.

GEORGE M. COATES,  
*Historian.*

Under miscellaneous business a brief report was made by Dr. Imperatori for the Committee on Revision of the Constitution and By-Laws. As announced in the "Call for the Annual Meeting" of March 31, 1938, these changes were simply ones of minor adjustment, clarifying the program of the annual meeting, outlining the organization of special committees and rewording or correcting any paragraphs necessary. Discussion of these minor changes followed, and Dr. Imperatori was directed, with the help of the other members of the Committee and the Secretary, to prepare this for publication.

At the close of the formal business meeting, Dr. Richmond McKinney of Memphis, Tennessee, rose and extended a very warm invitation to the Association to come to the Memphis Country Club for its next annual meeting. Upon motion, the Association expressed its appreciation for this generous invitation and directed that the matter be placed before the Council for its consideration.

As there was no further miscellaneous business to come before the Association, the Business Session adjourned at 11.15 o'clock.



## FINAL EXECUTIVE SESSION

WEDNESDAY, MAY 4, 1938

The final Executive Session was called to order by President Barnhill at 12.15 o'clock on Wednesday afternoon, May 4, 1938.

Motion was made and seconded that the report of the Nominating Committee be accepted. President Barnhill then requested the duly elected candidates to retire and elect their officers for the ensuing year. After a brief recess, the Secretary reported the following officers:

Dr. George B. Wood, *President*.

Dr. Ralph A. Fenton, *First Vice-President*.

Dr. Frederick T. Hill, *Second Vice-President*.

Dr. James A. Babbitt, *Secretary*.

Dr. Charles J. Imperatori, *Treasurer*.

Dr. George M. Coates, *Librarian and Historian*.

Dr. John D. Kernan, Jr., *First Councillor*.

Dr. Burt R. Shurly, *Second Councillor*.

Dr. William B. Chamberlin, *Third Councillor*.

Dr. John F. Barnhill, *Fourth Councillor*.

Dr. Barnhill then courteously presented the gavel to President-Elect Wood, who was deeply moved and responded with words of sincere appreciation.

Motion was made by Dr. Spencer that appreciation be extended to Dr. Barnhill for his reception and entertainment of the Association. This was enthusiastically carried by rising vote. Dr. Barnhill responded by expressing his personal thanks to all the officers of the Association for their coöperation during the past year.

On motion, President Wood adjourned the meeting until next year.



List of Fellows  
1938

### MEMORIAL FUND

As approved by the Council, it is earnestly requested that the Fellows of the Association give thoughtful consideration of the mention in their wills of some special bequest to this important fund for research. Such attention would be deeply appreciated.

MEMORIAL FUND COMMITTEE



## LIST OF FELLOWS

1938

Attendance at meetings is shown by the figures following each name.  
Asterisk (\*) indicates year paper was read.

### *Active Fellows*

1923. BABBITT, JAMES A., 1912 Spruce Street, Philadelphia, Pennsylvania, 1923, '24\*, '25, '26, '27, '28, '29\*, '30, '31, '32, '33\*, '34, '35, '36, '37, '38.
1936. BALLENGER, HOWARD C., 25 East Washington St., Chicago, Ill., 1936, '37, '38.
1923. BARLOW, ROY ALEXANDER, 277 Alexander Street, Rochester, New York, 1923, '24\*, '28, '29\*, '30\*, '37.
1908. BARNHILL, JOHN F., 5369 Pine Tree Drive, Miami Beach, Florida, 1922, '24, '27, '28, '29, '30, '31, '33, '35, '37, '38.
1921. BERRY, GORDON, 36 Pleasant Street, Worcester, Massachusetts, 1921\*, '22, '23, '24, '25, '26, '27, '28\*, '29\*, '30, '31, '32, '33, '34, '35, '37, '38.
1893. BIRKETT, HERBERT S., No. 71 The Linton, 1509 Sherbrooke St. West, Montreal, Canada, 1920, '21, '22\*, '23, '25, '26, '27, '28\*, '29, '30, '31, '32, '34, '35, '36, '38.
1938. BLASSINGAME, CHARLES DECATUR, 414 P. and S. Building, Memphis, Tennessee, 1938.
1932. BROWN, JOHN MACKENZIE, 1136 W. 6th Street, Los Angeles, California, 1935\*, '37.
1933. BUCKLEY, ROBERT E., 50 East 63rd Street, New York City, 1935, '36\*, '37.
1913. BUTLER, RALPH, 1930 Chestnut Street, Philadelphia, Pennsylvania, 1920\*, '21, '23, '24, '27, '29, '31, '32, '37, '38.
1930. CAMPBELL, EDWARD HASTINGS, 1904 Spruce Street, Philadelphia, Pennsylvania, 1930, '31\*, '32\*, '33, '35, '38.
1924. CARMODY, THOMAS E., 806 Metropolitan Building, Denver, Colorado, 1924, '26\*, '27, '28, '30, '32\*, '34, '35, '37\*, '38.
1917. CHAMBERLIN, WILLIAM B., 10515 Carnegie Avenue, Cleveland, Ohio, 1920, '21\*, '22, '26, '27\*, '28, '29, '30\*, '31, '32, '33\*, '34, '35\*, '36\*, '37\*, '38.
1925. CLERF, LOUIS H., 1530 Locust Street, Philadelphia, Pennsylvania, 1925, '27\*, '28, '29\*, '30\*, '32, '33, '34, '35, '36\*, '37, '38.

1917. COATES, GEORGE MORRISON, 1721 Pine Street, Philadelphia, Pennsylvania, 1920, '21, '22, '23, '24, '25, '26, '27, '28, '29, '30\*, '31, '32, '33, '34, '35, '36, '37\*, '38.
1937. CROWE, SAMUEL JAMES, 4332 North Charles Street, Baltimore, Maryland, 1937, '38\*.
1918. DEAN, LEE WALLACE, School of Medicine, Washington University, St. Louis, Missouri, 1920\*, '21\*, '22, '23, '24, '25\*, '26, '27\*, '28, '29\*, '31, '32, '34, '35, '36\*, '37, '38.
1935. EVES, CURTIS C., 1910 Spruce Street, Philadelphia, Pennsylvania, 1935, '37, '38.
1919. FAULKNER, E. ROSS, 101 East Fifty-eighth Street, New York City, 1920, '21, '22, '23\*, '25, '27, '28, '30, '32, '33, '34, '35, '37, '38.
1923. FENTON, RALPH ALBERT, 806 Medical Arts Building, Tenth and Taylor Streets, Portland, Oregon, 1923, '25\*, '26, '28, '30, '32\*, '34, '35, '37\*.
1930. FIGI, FREDERICK A., Mayo Clinic, Rochester, Minnesota, 1930, '32\*, '34, '35, '38\*.
1933. FOSTER, JOHN H., 1304 Walker Avenue, Houston, Texas, 1934, '35, '37, '38.
1932. FURSTENBERG, ALBETT CARL, 201 South Main Street, Ann Arbor, Michigan, 1933\*, '36\*, '38\*.
1936. GALLOWAY, THOMAS CYRUS, 635 Church Street, Evanston, Illinois, 1936, '37, '38\*.
1928. GITINS, THOMAS ROY, 408 Davidson Building, Sioux City, Iowa, 1928, '31, '32\*, '34, '36.
1922. GOLDSMITH, PERRY G., 513 Medical Arts Building, Toronto, Ontario, 1922\*, '23\*, '24, '26, '28, '35, '36.
1913. GOLDSTEIN, MAX A., 4574 West Papin Street, St. Louis, Missouri, 1920, '21, '22, '25\*, '26, '27, '28, '29, '30, '32\*, '34, '36, '38.
1928. GOODYEAR, HENRY M., 556 Doctors' Building, Cincinnati, Ohio, 1928, '29, '30\*, '31, '32, '33, '34\*, '35, '36, '37, '38.
1932. GRAHAM, HARRINGTON B., 490 Post Street, San Francisco, California, 1932, '35.
1905. GRAYSON, CHARLES P., 262 South Fifteenth Street, Philadelphia, Pennsylvania, 1921.
1906. GREENE, D. CROSBY, JR., 23 Bay State Road, Boston, Massachusetts, 1920\*, '21\*, '22, '23, '24\*, '25, '26, '27, '34, '35.
1917. GREENE, JOSEPH B., 331 Haywood Building, Asheville, North Carolina, 1921, '22, '23\*, '24, '26, '27, '28, '29\*, '31, '32, '33, '35, '36, '37\*.

1934. GROVE, WILLIAM E., 324 Wisconsin Avenue, Milwaukee, Wisconsin, 1934, '35, '36\*, '37, '38.
1933. HARKNESS, GORDON FOLLETTE, 2410 River Drive, Davenport, Iowa, 1934, '35, '36\*.
1903. HARRIS, THOMAS J., 104 East Fortieth Street, New York City, 1921, '22, '24\*, '25, '26, '28, '29, '30, '35, '36, '38\*.
1915. HASTINGS, HILL, 306 Medical Office Building, 1136 West Sixth Street, Los Angeles, California, 1921\*, '24, '27\*, '28, '30\*, '35.
1928. HILL, FREDERICK THAYER, Professional Building, Waterville, Maine, 1928, '29\*, '30\*, '31, '32, '33, '34, '35, '36, '37\*, '38.
1938. HOURN, GEORGE E., University Club Building, St. Louis, Missouri, 1938.
1930. HOUSER, KARL MUSSER, 2010 Spruce Street, Philadelphia, Pennsylvania, 1930, '32\*, '33, '34\*, '37\*, '38.
1919. HURD, LEE MAIDMENT, 39 East Fiftieth Street, New York City, 1921, '23, '25, '26\*, '28, '32\*, '34, '37, '38.
1925. IGLAUER, SAMUEL, 707 Race Street, Cincinnati, Ohio, 1926, '27, '30, '32, '33\*, '35\*, '36, '38.
1920. IMPERATORI, CHARLES JOHNSTONE, 108 East Thirty-eighth Street, New York City, 1921\*, '22, '24\*, '25\*, '26\*, '27\*, '28, '31, '32, '33\*, '35\*, '36, '37\*, '38.
1907. JACKSON, CHEVALIER, 3701 North Broad Street, Philadelphia, Pennsylvania, 1921\*, '22\*, '23, '24\*, '25\*, '27\*, '28, '29, '30, '31, '32, '33, '34, '35, '36, '37\*, '38\*.
1938. JACKSON, CHEVALIER LAWRENCE, 3701 North Broad Street, Philadelphia, Pennsylvania, 1938.
1928. JARVIS, DEFOREST CLINTON, Room 7, Quarry Bank Building, Barre, Vermont, 1928, '30, '32\*, '33, '34\*, '35, '37\*, '38.
1921. KERNAN, JOHN D., JR., 120 East Seventy-fifth Street, New York City, 1921\*, '22, '25, '26, '27, '29, '31, '34\*, '35\*, '36, '37\*.
1929. KISTNER, FRANK B., 600 Mayer Building, Portland, Oregon, 1930, '35, '37.
1923. LARGE, SECORD HERBERT, Hanna Building, Cleveland, Ohio, 1924\*, '25, '26, '31, '34.
1936. LEJEUNE, FRANCIS ERNEST, 632 Maison Blanche, New Orleans, Louisiana, 1937.
1918. LEWIS, FIELDING OTIS, Providence Road, Media, Pennsylvania, 1921\*, '22\*, '23, '25\*, '28, '29, '31, '32, '33, '34.

1933. LIERLE, DEAN M., University Hospital, Iowa City, Iowa, 1933, '34, '35\*, '37, '38.
1922. LILLIE, HAROLD I., Mayo Clinic, Rochester, Minnesota, 1923\*, '24\*, '25, '26\*, '28, '31\*, '35, '36\*, '37, '38.
1935. LOOPER, EDWARD A., 104 West Madison Avenue, Baltimore, Maryland, 1935, '36, '37, '38\*.
1927. LUKENS, ROBERT M., 1923 Spruce Street, Philadelphia, Pennsylvania, 1927, '28, '31\*, '32, '35, '36, '38.
1929. LYMAN, HARRY WEBSTER, 906 Carleton Building, St. Louis, Missouri, 1929, '31, '34, '35\*, '36, '38.
1928. MACPHERSON, DUNCAN, 140 East Fifty-fourth Street, New York City, 1928, '29, '30, '31, '33, '34\*.
1921. McCULLAGH, SAMUEL, 2 East Fifty-fourth Street, New York City, 1921\*, '22, '23\*, '25, '26\*, '27, '28, '31, '34, '35, '37, '38.
1936. MCGREGOR, GREGOR, 170 St. George Street, Toronto, Canada, 1936, '37\*.
1923. MCKINNEY, RICHMOND, 899 Madison Avenue, Memphis, Tennessee, 1923, '24\*, '26\*, '27, '29, '31, '32, '33, '36, '37, '38\*.
1933. McMAHON, BERNARD J., Missouri Theatre Bldg., St. Louis, Missouri, 1934\*, '35, '36, '38\*.
1931. McNALLY, WILLIAM JAMES, 1509 Sherbrooke Street West, Montreal, Canada, 1931, '33, '34.
1904. MOSHER, HARRIS P., 828 Beacon Street, Boston, Massachusetts, 1920, '21\*, '22, '23\*, '24, '25, '26, '27, '28\*, '29, '30, '31\*, '32\*, '33, '34, '36, '37, '38.
1927. MYERSON, MERVIN C., 198 Lincoln Place, Brooklyn, New York, 1927, '28\*, '29, '30, '31\*, '32\*, '33\*, '34\*, '35, '36, '37, '38.
1937. NASH, CARLTON STEWART, Medical Arts Building, Rochester, New York, 1937, '38\*.
1922. NEW, GORDON B., Mayo Clinic, Rochester, Minnesota, 1922, '24, '25\*, '26\*, '28\*, '31\*, '33\*, '35\*, '37\*.
1923. NEWHART, HORACE, 527 Medical Arts Building, Minneapolis, Minnesota, 1923, '25\*, '26, '28, '30, '33, '34, '35, '36, '38.
1927. ORTON, HENRY BOYLAN, 24 Commerce Street, Newark, New Jersey, 1927, '28, '29, '30\*, '31, '32, '33\*, '35, '36, '37, '38\*.
1903. PACKARD, FRANCIS R., 302 South Nineteenth Street, Philadelphia, Pennsylvania, 1920, '21, '22, '23, '24, '25, '26, '29, '30, '31\*, '32, '33, '34, '35, '36, '38.
1906. PIERCE, NORVAL H., Del Mar, San Diego Co., California, 1920\*, '21, '22, '23, '24, '25, '26\*, '27\*, '28, '30, '33.



1927. PORTER, CHARLES TERRELL, 520 Commonwealth Avenue, Boston, Massachusetts, 1927, '28, '30, '31\*, '32, '35, '37, '38.
1931. POTTS, JOHN B., 1620 Medical Arts Building, Omaha, Nebraska, 1931, '33\*, '38.
1924. PROETZ, ARTHUR WALTER, 1010 Beaumont Building, St. Louis, Missouri, 1924, '25\*, '26\*, '27, '28, '29, '30\*, '31, '32\*, '33\*, '34, '35\*, '36, '37, '38\*.
1934. RICHARDS, LYMAN GILDER, 319 Longwood Avenue, Boston, Massachusetts, 1934, '35, '36\*, '37, '38.
1930. RIDPATH, ROBERT F., Central Medical Building, Eighteenth and Chestnut Streets, Philadelphia, Pennsylvania, 1930, '31, '33, '37, '38\*.
1938. SALINGER, SAMUEL, 25 E. Washington Street, Chicago, Illinois, 1938.
1921. SAUER, WILLIAM E., 3720 Washington Boulevard, St. Louis, Missouri, 1921, '23, '25, '28, '31, '32, '35, '37.
1932. SCHALL, LEROY ALLEN, 270 Commonwealth Avenue, Boston, Massachusetts, 1934\*, '35, '38.
1934. SCHENCK, HARRY P., 1912 Spruce Street, Philadelphia, Pennsylvania, 1934, '35, '36\*, '37, '38\*.
1923. SEWALL, EDWARD CECIL, Stanford University, Clay and Webster Streets, San Francisco, California, 1924, '26\*, '28\*, '35\*, '36\*.
1930. SEYDELL, ERNEST M., First National Bank Building, Wichita, Kansas, 1930, '31\*, '33\*, '34, '35, '37\*, '38.
1928. SHEA, JOHN JOSEPH, 1018 Madison Avenue, Memphis, Tennessee, 1928, '29, '30\*, '31, '32\*, '33, '34, '35, '36, '37, '38.
1909. SHURLY, BURT R., 62 Adams Avenue W., Detroit, Michigan, 1920, '21\*, '22, '24, '25\*, '26, '27\*, '28, '29\*, '30, '31, '33, '34, '35\*, '36, '37, '38.
1928. SONNENSCHN, ROBERT, 4518 Woodlawn Avenue, Chicago, Illinois, 1928, '29\*, '30, '31, '32, '33, '34, '35, '36.
1923. SPENCER, FRANK ROBERT, Physicians' Building, Boulder, Colorado, 1923, '24\*, '26, '27\*, '28, '30, '32\*, '34, '35, '37, '38.
1934. STEVENSON, WALTER, 904 W. C. U. Building, Quincy, Illinois, 1934, '36.
1889. SWAIN, HENRY L., Colonial Building, 195 Church Street, New Haven, Connecticut, 1920, '21\*, '22, '23, '24, '25, '26, '27, '28, '29, '30\*, '31, '32, '33, '34, '36, '38.
1924. TAYLOR, HERMON MARSHALL, 111 West Adams Street, Jacksonville, Florida, 1924, '25\*, '26, '27, '28, '31, '34, '35\*, '36, '37, '38.

1937. TOBEY, GEORGE L., JR., 270 Commonwealth Avenue, Boston, Massachusetts, 1937, '38.
1927. TOBEY, HAROLD G., 270 Commonwealth Avenue, Boston, Massachusetts, 1927, '29\*, '30\*, '31, '32, '35, '37.
1925. TUCKER, GABRIEL, 269 South Nineteenth Street, Philadelphia, Pennsylvania, 1925, '26\*, '27\*, '29, '30, '31, '32, '33, '34, '35\*, '36, '37, '38.
1938. VAIL, HARRIS H., 802 Carew Tower, Cincinnati, Ohio, 1938.
1936. VOISLAWSKY, ANTONIE P., 33 East Sixty-eighth Street, New York City, 1936, '37, '38.
1933. WANAMAKER, ALLISON T., 817 Summit Avenue, Seattle, Washington, 1933, '37.
1892. WATSON, ARTHUR W., 2013 Locust Street, Philadelphia, Pennsylvania, 1921, '23, '25, '27, '31, '32, '38.
1924. WHERRY, WILLIAM P., 1500 Medical Arts Building, Omaha, Nebraska, 1924, '25\*, '26, '27, '28, '29, '30, '31, '33, '34, '35, '36, '37, '38.
1922. WHITE, FRANCIS W., 471 Park Avenue, New York City, 1922, '23\*, '25, '26, '27, '28, '29, '31, '32, '37.
1908. WILSON, J. GORDON, 5755 Kenwood Avenue, Chicago, Illinois, 1920, '21, '22, '24, '25, '26, '28, '29, '30, '31\*, '32, '33, '35.
1905. WOOD, GEORGE B., Suite 504, Physicians' Building, Twentieth and Chestnut Streets, Philadelphia, Pennsylvania, 1921, '22\*, '23\*, '25\*, '28, '29, '31\*, '32, '33, '34, '37\*, '38.
1935. WOODWARD, FLETCHER D., 104 East Market Street, Charlottesville, Virginia, 1935, '36\*, '37, '38.

*Honorary Fellows*

1938. DELAVAN, D. BRYSON, 30 East Sixtieth Street, New York City.
1910. MOURE, E. J., 25 bis Cours de Verdun, Bordeaux, France.
1914. THOMSON, SIR ST. CLAIR, 64 Wimpole Street, W., London, England.
1936. TILLEY, HERBERT, 72 Harley Street, Cavendish Square, London, W., England.
1937. NAGER, F. R., Freiestrasse 30, Zurich, Switzerland.

*Corresponding Fellows*

1938. BLAIR, VILRAY P., 508 North Grand Avenue, St. Louis, Missouri.

1893. DESVERNINE, CARLOS M., 52 Cuba Street, Havana, Cuba.  
 1930. EMERSON, FRANCIS P., Franklin, Massachusetts.  
 1901. GRANT, SIR JAMES DUNDAS, K. B. E., 32 Lexham Gardens, London, W. 8, England.  
 1919. KELLY, ADAM BROWN, Blyth's House, Helensburg, Dunbartonshire, England.  
 1931. LASAGNA, PROFESSOR FRANCESCO, Royal University of Parma, Parma, Italy.  
 1926. LAW, FREDERICK M., 140 East Fifty-fourth Street, New York City, 1934, '35, '36, '37, '38.  
 1921. LE MAITRE, FERNAND, Avenue Victor Hugo, 120 (XVI), Paris, France.  
 1903. MCBRIDE, P., 3 St. Peter's Grove, York, England.  
 1919. MCKERNON, JAMES F., New Canaan, Connecticut.  
 1896. MACDONALD, GREVILLE, Wildwood, Haslemere, England.  
 1930. NEGUS, VICTOR E., 149 Harley Street, London, W 1, England, 1938\*.  
 1919. PATERSON, DONALD ROSE, 15 St. Andrew's Crescent, Cardiff, Wales.  
 1937. PORTMANN, GEORGES, 25 bis Cours de Verdun, Bordeaux, France.  
 1919. ROGERS, JOHN, JR., 177 East Seventy-first Street, New York City.  
 1924. SCHAEFFER, J. PARSONS, Jefferson Medical College, Philadelphia, Pennsylvania, 1934.  
 1896. SCHMIEGELOW, ERNST, 18 Norregade, Copenhagen, Denmark.  
 1913. TURNER, LOGAN, 27 Walker Street, Edinburgh, Scotland.  
 1901. WINGRAVE, WYATT, Lyme Regis, Dorset, England.  
 1936. VIALLE, JACQUES, 27 Rue de France, Nice (Alpes Maritime), France.

*Emeritus Fellows*

1937. (1915) BARNES, HARRY ALDRICH, 61 Church Street, Dedham, Massachusetts.  
 1935. (1928) BECK, JOSEPH C., 185 North Wabash Avenue, Chicago, Illinois, 1936, 1937, 1938.  
 1934. (1897) CLARK, J. PAYSON, 71 Marlborough Street, Boston, Massachusetts.  
 1928. (1893) COOLIDGE, ALGERNON, 198 Beacon Street, Boston, Massachusetts.  
 1934. (1913) DABNEY, VIRGINIUS, 1633 Connecticut Avenue, Washington, D. C.

1938. (1922) FORBES, HENRY HALL, 1192 Park Avenue, New York City.
1936. (1898) GOODALE, JOSEPH L., 258 Beacon Street, Boston, Massachusetts.
1933. (1905) HALSTED, THOMAS H., Lyndon Road, Fayetteville, New York.
1927. (1896) HARDIE, T. MELVILLE, 30 N. Michigan Boulevard, Chicago, Illinois.
1918. (1888) HINKEL, F. WHITEHILL, 28 Norwood Avenue, Buffalo, New York.
1926. (1895) HOPKINS, FREDERICK E., 146 Chestnut Street, Springfield, Massachusetts.
1927. (1895) HUBBARD, THOMAS, Lock Box 245, Ashtabula, Ohio.
1933. (1904) INGERSOLL, JOHN M., 1700 S. Bayshore Lane, Miami, Florida.
1938. (1918) KENYON, ELMER L., 104 South Michigan Boulevard, Chicago, Illinois.
1934. (1914) LEVY, ROBERT, Metropolitan Building, Denver, Colorado.
1933. (1907) SHAMBAUGH, GEORGE E., 122 South Michigan Avenue, Chicago, Illinois.



*Recipients of the deRoaldes Award*

1928. DR. CHEVALIER JACKSON.  
1931. DR. D. BRYSON DELAVAN.  
1934. DR. HARRIS P. MOSHER.  
1937. DR. LEE WALLACE DEAN.

*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 HILDING.  
1936. DR. FRANCIS E. LEJEUNE. DR. JOEL J. PRESSMAN.

*Benefactors*

- DR. ARTHUR W. DEROALDES.  
DR. WILLIAM E. CASSELBERRY.  
DR. SAMUEL JOHNSTON.

## 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. Halsted
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. Gleitsmann	1935. Burt R. Shurly
1906. A. W. deRoaldes	1936. William B. Chamberlin
1907. H. S. Birkett	1937. John F. Barnhill
1908. A. Coolidge, Jr.	

*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	

1885.	H. A. Johnson	G. W. Major
1886.	E. C. Morgan	J. N. Mackenzie
1887.	J. N. Mackenzie	S. W. Langmaid
1888.	W. C. Glasgow	C. E. DeM. Sajous
1889.	E. Holden	C. E. Bean
1890.	J. O. Roe	J. H. Hartman
1891.	M. J. Asch	S. Johnston
1892.	S. Johnston	J. C. Mulhall
1893.	J. C. Mulhall	W. E. Casselberry
1894.	C. C. Rice	S. H. Chapman
1895.	J. Wright	A. W. de Roaldes
1896.	T. M. Murray	D. N. Rankin
1897.	A. W. MacCoy	H. S. Birkett
1898.	J. W. Farlow	F. W. Hinkel
1899.	T. A. De Blois	M. R. Brown
1900.	H. L. Wagner	A. A. Bliss
1901.	J. W. Gleitsmann	D. Braden Kyle
1902.	G. A. Leland	T. Melville Hardie
1903.	J. H. Lowman	W. Peyre Porcher
1904.	Thomas Hubbard	W. J. Freeman
1905.	J. L. Goodale	C. W. Richardson
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. E. Shambaugh
1914.	Clement F. Thiesen	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 R. Spencer
1933.	E. Ross Faulkner	Thomas E. 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

*Secretaries and Treasurers*

1879.	G. M. Lefferts	1895.	H. L. Swain
1882.	D. Bryson Delavan	1900.	J. E. Newcomb
1889.	C. H. Knight	1911.	Harmon Smith

*Secretaries*

1912.	Harmon Smith	1920.	George M. Coates
1918.	D. Bryson Delavan	1933.	William V. Mullin
1919.	J. M. Ingersoll	1935.	James A. Babbitt

*Treasurers*

1912.	J. Payson Clark	1932.	William V. Mullin
1923.	George Fetterolf	1933.	James A. Babbitt
	1935.	Charles J. Imperatori	

*Librarians*

1879.	F. H. Bosworth	1930.	John F. Barnhill
1883.	T. R. French	1934.	Burt R. Shurly
1903.	J. H. Bryan	1935.	George M. Coates



## PAST MEMBERS

Dates correspond with election to respective Fellowships.

*\*Deceased**Honorary Fellows*

- \*1915. Bosworth, Francke H., New York City.
- \*1914. Cohen, J. Solis-, Philadelphia, Pa.
- \*1915. deRoaldes, Arthur W., New Orleans, La.
- \*1914. De La Sota Y Lastra, Ramon, Seville, Spain.
- \*1880. Garcia, Senor Manuel, London, England.
- \*1928. French, Thomas R., Brooklyn, N. Y.
- \*1924. Johnston, Samuel, Baltimore, Md.
- \*1914. Lefferts, George Morewood, Katona, N. Y.
- \*1922. Mackenzie, John N., Baltimore, Md.
- \*1881. Mackenzie, Sir Morell, London, England.
- \*1910. Massei, Ferdinand, Naples, Italy.
- \*1918. Oliver, H. K., Boston, Mass.
- \*1918. Semon, Sir Felix, Great Missenden, England.
- \*1914. Wagner, Clinton, New York City.
- \*1921. Wright, Jonathan, Pleasantville, N. Y.

*Corresponding Fellows*

- \*1892. Browne, Lennox, London, England.
- \*1901. Collier, Mayo, Kearsney Abbey, Kent, England.
- \*1936. Fraser, John S., Edinburgh, Scotland.
- \*1887. Gouguenheim, A., Paris, France.
- \*1894. Holden, Edgar, Newark, N. J.
- \*1881. Labus, Carlo, Milan, Italy.
- \*1902. Lermoyez, Marcel, Paris, France.
- \*1897. Luc, H., Paris, France.
- \*1894. MacIntyre, John, Glasgow, Scotland.
- \*1920. McKenzie, Dan, 9 Weymouth Street, Portland Place,  
London, W. 1, England.
- \*1880. Meyer, Wilhelm, Copenhagen, Denmark.
- \*1896. Mygind, Holger, Copenhagen, Denmark.
- \*1894. Sajous, C. E. DeM., Philadelphia, Pa.
- \*1881. Thornton, Pugin, London, England.
- \*1880. Whistler, W. McNeil, London, England.
- \*1894. Wolfenden, R. Norris, Kent, England.

*Emeritus Fellows*

- \*1914. Brown, J. Price, Toronto, Canada.
- \*1934. Bryan, Joseph H., Washington, D. C.
- \*1922. Cobb, Frederick C., Bradenton, Florida.
- \*1931. Coffin, Lewis A., New York City.
- \*1914. De Blois, Thomas Amory, Boston, Mass.

- \*1918. Farlow, John W., Boston, Mass.
- \*1924. Freer, Otto T., Chicago, Ill.
- \*1914. Gleitsmann, Joseph W., New York City.
- \*1912. Knight, Charles H., New York City.
- \*1923. Leland, George A., Boston, Mass.
- \*1912. Lincoln, William R., Cleveland, Ohio.
- \*1922. MacCoy, Alexander W., Philadelphia, Pa.
- \*1931. Mayer, Emil, New York City.
- \*1931. Myles, Robert C., New York City.
- \*1914. Porter, William, Ocean Springs, Miss.
- \*1934. Renner, W. Scott, Buffalo, N. Y.
- \*1924. Rhodes, John Edwin, Chicago, Ill.
- \*1935. Rice, Clarence C., 52 Park Avenue, New York City.
- \*1932. Richards, George L., South Yarmouth, Mass.
- \*1914. Robinson, Beverley, New York City.
- \*1934. Smith, Harmon, New York City.
- \*1936. Wagner, Henry L., San Francisco, Calif.

*Active Fellows*

- \*1880. Allen, Harrison, Philadelphia, Pa.
- \*1917. Arrowsmith, Hubert, Brooklyn, N. Y.
- \*1879. Asch, Morris J., New York City.
- \*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.
- \*1915. Canfield, R. Bishop, Ann Arbor, Mich.
- \*1934. Carmack, John Walter, Indianapolis, Ind.
- \*1889. Casselberry, William E., Chicago, Ill.
- \*1883. Chamberlain, C. W., Hartford, Conn.
- \*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, Gerard 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.
- 1926. Dennis, Frank Lowndes, Colorado Springs, Col.
- \*1901. Dickerman, E. T., Chicago, Ill.
- \*1878. Donaldson, Frank, Baltimore, Md.

- \*1914. Fetterolf, George, Philadelphia, Pa.
- \*1901. Freeman, Walter J., Philadelphia, Pa.
- \*1917. Friedberg, Stanton A., Chicago, Ill.
- \*1897. Frothingham, Richard, New York City.
- 1909. Getchell, Albert C., Worcester, Mass.
- \*1907. Gibb, Joseph S., Philadelphia, Pa.
- \*1878. Glasgow, William Carr, St. Louis, Mo.
- \*1878. Hartman, Jacob H., Baltimore, Md.
- \*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.
- \*1901. Hyatt, Frank, Washington, D. C.
- \*1882. Ives, Frank L., New York City.
- \*1878. Ingals, E. Fletcher, Chicago, Ill.
- \*1880. Jarvis, William C., New York City.
- \*1878. Johnson, Hosmer A., Chicago, Ill.
- \*1879. Johnson, Woolsey, 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.
- \*1878. 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.
- 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.
- \*1908. Randall, B. Alexander, Philadelphia, Pa.
- \*1882. Rankin, D. N., Allegheny, Pa.
- \*1902. Richardson, Charles W., Washington, D. C.
- \*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, Va.
- \*1878. Shurley, 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.
- 1924. Stein, Otto J., Chicago, Ill.
- \*1911. Stout, 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.
- \*1888. Van der Poel, S. O., New York City.
- 1896. Ward, Marshall R., Pittsburgh, Pa.
- \*1879. Ward, Whitfield, New York City.
- \*1886. Westbrook, Benjamin F., Brooklyn, N. Y.
- \*1924. White, Leon E., Boston, Mass.
- \*1922. Wishart, David J. G., Toronto, Ontario.
- \*1896. Woolen, Green V., Indianapolis, Ind.

*Associate Fellows*

- 1929. Alden, Arthur Maxwell, St. Louis, Mo.
- 1929. Arbuckle, Millard F., St. Louis, Mo.
- \*1932. Atkins, Richard Travis, New York City.
- 1932. Baum, Harry Lester, Denver, Colo.
- 1930. Hansel, French K., St. Louis, Mo.
- 1934. Hasty, Frederick E., Nashville, Tenn.
- \*1925. King, James Joseph, New York City.
- 1922. Monroe, Otho Lee, New York City.
- 1934. Skillern, Samuel R., Jr., Philadelphia, Pa.
- 1933. Stiles, Porter, Birmingham, Ala.

















