G.F.TUCKER

Transactions

American Laryngological Association

SIXTY-SIXTH VOLUME

"DOCENDO DISCIMUS"

TRANSACTIONS

OF THE

SIXTY-SIXTH ANNUAL MEETING

OF THE

American Laryngological Association

1944

HELD AT THE HOTEL WALDORF-ASTORIA

NEW YORK, N. Y.

JUNE 7TH AND 8TH, 1944

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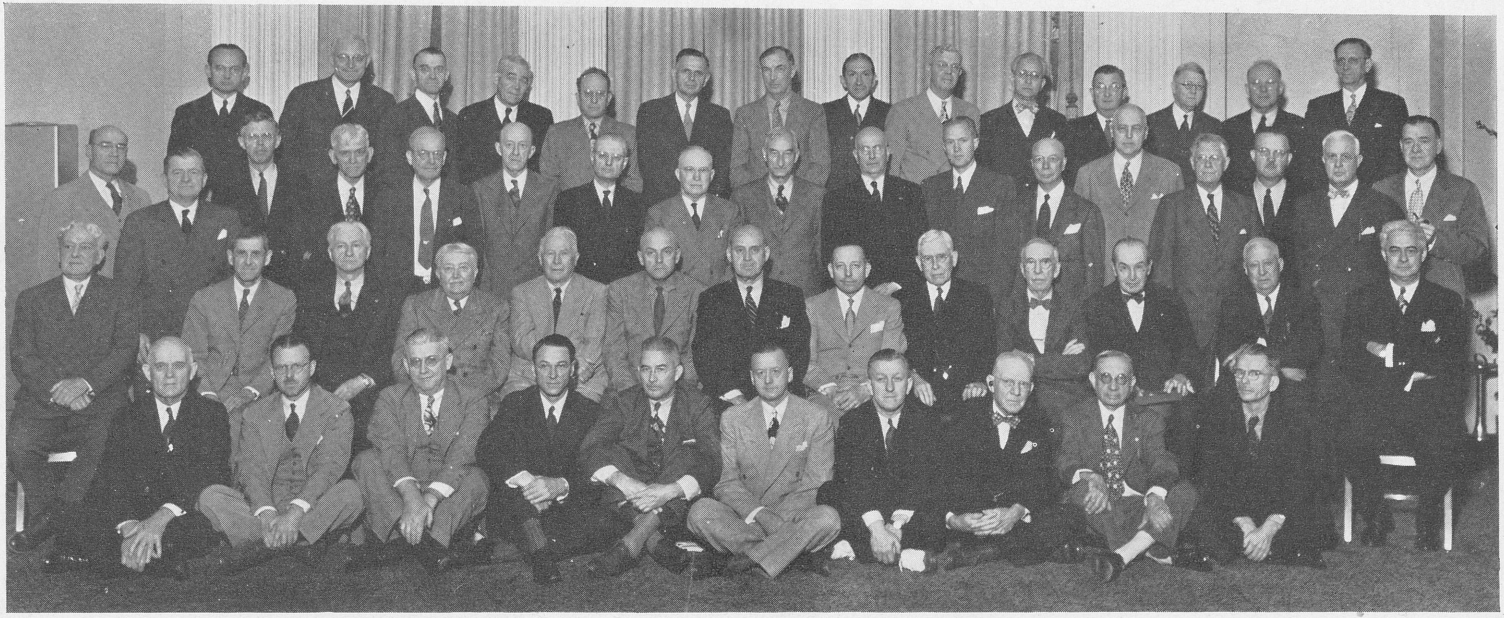

MEMBERS OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION WHO ATTENDED THE SIXTY-SIXTH ANNUAL MEETING HELD AT THE HOTEL WALDORF-ASTORIA, NEW YORK, N. Y., JUNE 7, 8, 1944

Top Row: Jackson, Jones, Clerf, Kelly, Beatty, Ballenger, Galloway, Weille, Grove, Salinger, Porter, Gatewood, Violé, Hourn.

Third Row: Berry, Bowers, Hill, Cody, Lyman, Taylor, Jarvis, Sauer, Brown, Fenton, Goodyear, Potts, Harkness, White, Schall, Orton, Cunning.

Second Row: Law, Coates, Carmody, Shurly, Ridpath, Lillie, Imperatori, Proetz, Halsted, Wood, Hurd, Mosher, McMahon.

Front Row: McCaskey, Heatly, Shea, Hilding, VanAlyea, Dixon, Boies, Foster, Iglauer, Simpson.

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TRANSACTIONS OF THE SIXTY-SIXTH ANNUAL MEETING

OF THE

AMERICAN LARYNGOLOGICAL ASSOCIATION

HELD AT THE

HOTEL WALDORF-ASTORIA, NEW YORK, N. Y.

JUNE 7TH AND 8TH, 1944

The Sixty-sixth Annual Meeting of the American Laryngological Association was called to order by the President, Dr. Charles J. Imperatori, at the Hotel Waldorf-Astoria, New York, N. Y., at 10 A. M. on June 7th, 1944.

He extended a cordial welcome to all the members and the guests present and declared the Scientific Session open.

REGISTRATION

The following Fellows were in attendance:

BALLENGER, HOWARD C.
BEATTY, HUGH G.
BERRY, GORDON
BLASSINGAME, CHARLES D.
BOIES, LAWRENCE R.
BOWERS, WESLEY C.
BROWN, JOHN MACKENZIE
CARMODY, THOMAS E.
CLERF, LOUIS H.
COATES, GEORGE M.
CODY, CLAUDE C.
CUNNING, DANIEL S.
DIXON, FRED W.
EGGSTON, ANDREW A.
FENTON, RALPH A.
FOSTER, JOHN H.
GATEWOOD, E. TRIBLE
KELLY, JOSEPH D.
LAW, FREDERICK M.
LIERLE, DEAN M.
LILLIE, HAROLD I.
LOOPER, EDWARD A.
LYMAN, HARRY W.
MACPHERSON, DUNCAN
MARTIN, ROBERT C.
MCCASKEY, CARL H.
MCCULLAGH, SAMUEL
MCMAHON, BERNARD J.
MCNALLY, WILLIAM J.
MORRISON, WILLIAM W.
MOSHER, HARRIS P.
MYERS, JOHN L.
MYERSON, MERVIN C.
NASH, C. STEWART

Goodale, Robert L.
Goodyear, Henry M.
Grove, William E.
Halsted, Thomas H.
Harkness, Gordon F.
Havens, Fred Z.
Heatly, Clyde A.
Hilding, Anderson C.
Hill, Frederick T.
Hourn, George E.
Houser, Karl M.
Hunt, Westley M.
Hurd, Lee M.
Iglauer, Samuel
Imperatori, Charles J.
Jackson, Chevalier L.
Jarvis, DeForest C.
Johnston, William H.
Jones, Marvin F.
Orton, Henry B.
Porter, Charles T.
Potts, John B.
Proetz, Arthur W.
Ridpath, Robert F.
Salinger, Samuel
Sauer, William E.
Schall, LeRoy A.
Shea, J. J.
Shurly, Burt R.
Simpson, W. Likely
Taylor, H. Marshall
Van Alyea, Oliver E.
Viole, Pierre P.
Weille, Francis L.
White, Francis W.
Wishart, D. E. Staunton
Wood, George B.
Woodward, Fletcher D.

PRESIDENT'S ADDRESS

CHARLES J. IMPERATORI, M.D.

NEW YORK CITY

Fellows of the American Laryngological Association: welcome to New York!

In 1920, when I was elected a Fellow of this Association, the noble desire to become its President was a most remote one.

Having elected me to be your principal officer, it is my first duty to thank you for selecting and nominating me,—at least for a fleeting moment,—as your leader. I most sincerely assure you this high honor is duly appreciated.

It has been with high hope that throughout these two trying years, the affairs of the Association have been directed to your satisfaction. My further hope is that I have measured up to your choice and that your confidence remains as it was in June, 1942,—when you elected me.

In turn, I could not fail to thank each member of the Council for their support and guidance in the administration of the affairs of the Association. Also, I should fail all of you, did I not acquaint you with the zeal and ability with which our Secretary, Dr. Arthur W. Proetz, has taken over this office. He has published two Transactions of the Association, 1942 and 1943, under most difficult circumstances and carried on successfully his administrative duties with interruptions and unusual happenings. The thanks of the Association herewith, Dr. Proetz.

The finances of the Association are in a better state of stability as of today than ever before and for that, Dr. Hill, accept herewith the acknowledgment from the Fellows,—"Well done."

Re Annual Assembly, 1943.

In the early Fall of 1942 the question of the feasibility of holding the Annual Meeting of the Association in the Spring of 1943, was considered. This consideration was based mostly on the question

of transportation,—or really interference with it. After communication with the Office of Defense Transportation, it was concluded by your President that the proper course would be to advise the Council that the Annual Meeting for 1943 be omitted. These facts were presented to the Council. Previous consultation with the principal officers of the American Otological Society, the American Rhinological, Laryngological and Otological Society and the American Broncho-Esophagological Association, resulted in the unanimous opinion,—that because of prevailing conditions in all types of transportation,—no 1943 meeting should be held. These facts were presented to the Council and the majority of members of the Council present were in agreement that no meeting should be held during 1943.

Re Procedure in Electing New Fellows During 1943.

Within the past two years, your Council has carried on and by their actions have established several precedents, one of which in particular was the election of the recommended candidates by a postal ballot. In this Emergency, this was necessary even though it seemed to destroy the principle of the secret ballot. The Constitution was interpreted to provide for such a method. (Art. 7, Sec. 4, Con. & By-Laws, A. L. A. Revised 1938).

Insofar as your Officers-Members of the Council and their continuance in office were concerned, the interpretation by the Council was that there being no Annual Meeting of the Association, the Council would serve, as constituted, until their successors were elected. (Art. 7, Sec. 2, Con. & By-Laws, A. L. A. Revised 1938).

Re Selection of New Fellows.

A most important function of our organization is to perpetuate itself. The selection and election of a prospective candidate is a matter that must require careful consideration by every Active member of this organization. Maintenance of the idea that this is a senior organization is acceptable but only to that point, far this side, of where the individual members remain active and productive. The implication that one has reached the goal by attaining membership in this group is only just that and one that must be confirmed. The Association is a living fabric held together by the

Fig. 1.

connective tissue of tradition, the scientific accomplishments of each member, and good fellowship. Regardless of age, a prospective Fellow of this Association should be one whose accomplishments are such that the sponsors have every reason and hope to feel that this individual will stimulate the existing membership to better things in Laryngology, and by this association, improve himself. The spirit of good fellowship is most desirable but the sponsors must consider the professional and ethical qualifications as fundamentally above all others.

It would seem best to retain the present numerical strength of the Active Fellowship (100) for the time being, but careful consideration must be given in the future toward the expansion of the membership.

The location map which I have prepared shows that almost 6/7ths of the total membership are located East of a line from North to South including the Dakotas, Nebraska, Kansas, Oklahoma and Texas. (Fig. 1).

Out of a total membership (143), there are but 21 members West of this line and of these 15 are Active. In other words, practically half of the area of the United States is represented in our membership by approximately 15% of our total membership. Our organization should recognize more of the competent men of the West.

In searching through the membership list, a committee might reasonably find members among the Active Fellowship whose scientific or clinical activities have been very much curtailed. The voluntary request by an Active member to be transferred to the Emeritus Fellowship, is a most honorable and considerate act. It permits a waiting candidate to be recommended for Active Fellowship and embodying with it, all the hopes that are entertained for each new candidate. This, in no way interferes with any scientific activities of the individual fellow, excepting the privilege of holding office, voting and guiding the affairs of the Association. These are the prerogatives only of Active Fellows. The Emeritus or Honorary Fellow is considered in the most high regard and it has always been felt that his advice and counsel are most valuable. Fortunately, this organization possesses a high degree of good Fellowship; tenuous,—rather difficult to describe,—intangible to a degree,—never embodied in so many words,—but something that each Fellow becomes imbued with, that remains with him forever. The newly elected Fellow becomes transfused with it. The hearty welcome with which we greet him at his initial appearance, establishes and secures it.

Re Socialization of Medicine.

Socialization of medicine seems to be a trend of those in authority in our government. The "world does move" times are ever changing and the medical profession should prepare for this venture. As stated in a recent editorial of the Journal of the American Medical Association,—any compulsory health insurance plan cannot be but anchored to financial, administrative and political considerations and associated with it will be a tremendous volume of litigation, legislation and regulation. One must not forget that even today, as high as 20% of all medical men in practice are doing administration work. There is little doubt that in the New World

to come, either now or later, the people will subsidize the doctor and possibly for the betterment of many in the profession. Irrespective to what degree one who is a doctor of medicine feels,—that he is entitled to the freedoms promulgated by Jefferson, reiterated by Lincoln and redeveloped and intensified by Roosevelt,—the general political trend seems to be toward the socialization of medicine and a regimentation of all doctors. It would also seem that the public is ready for this experiment on themselves. Who will predict that such a trial will end in benefit or detriment to the public at large and to the benefit or detriment of the medical man. A decade or two can only test its adequacy or otherwise. A diploma and a license may be the "open sesame" to the practice of medicine. Beyond that, they are of small value and to the public, even less. Irrespective of what is in store for the medical man of tomorrow, he must recognize that the individual attention and study and desire to heal the sick, is the optimum toward which all his thoughts must be directed. Also, regardless of all bureaucratic and even totalitarian methods impressed on the individual, human endeavor has always been interlocked with individual initiative. There have always been and always will be outstanding individuals better equipped mentally and better able to co-ordinate their faculties than their fellows. These will be the superior medical men of the future and their reward will be accordingly.

Re Medical Education.

The preliminary education of the individual who would study medicine must be such that admittance can be gained to the highest rank medical school. Aside from various cults and pathies, there remain but few medical schools in the United States that do not qualify one to take the examinations of the Federal Examining Board. All of these types of lower standard medical schools should be eliminated. The enlightment of the public in this regard shall be such that they will understand that only the most complete curriculum is sufficient to qualify, that the Faculties of the medical schools are properly supervised and that eventually everyone entering a specialty is properly qualified by an Examining Board of that specialty. At this point, it would not be amiss to pay tribute to that Nestor of Otolaryngology,—Dr. Harris P. Mosher, who has been

President of the American Board of Otolaryngology and has guided it throughout its existence. This Association is proud that he and Dr. George M. Coates have served as representatives of this organization during these past years,—since 1925. The challenge to medicine in general and the specialities in particular, ever is that each decade shall have better qualified medical men.

If the medical student is instructed according to the curriculum of the standard medical school of today, if the student has the aptitude, if the instructors are individually interested in imparting their knowledge and there is not too much submersion into the socialization and regimentation of the individual medical man with the incidental diminution in initiative, I am of the opinion that the next generation of doctors taught here in the United States will be much better than any in the past, anywhere in the World.

Re Army and Navy Medical Men.

Improved sanitation, proper selection of troops, the various preventative measures and a realistic view of the venereal and alcohol problems, would seem to indicate that our sons are better military doctors than we were in the last war. The authority of the medical officer in the field and particularly so in tropical and jungle areas, does not seem to be sufficient. Flies, heat and humidity certainly make the digging of a latrine a high priority rather than a fifteen or seventeen.

The valor and efficiency of the soldier may be dissipated within a few hours by filth and bacteria, and one may postulate that the loss of front-line combat hours may be in proportion to the bacteria entering the intestinal tract. The use of the sulfonamides and the rapid evacuation of the sick and wounded, have been great life savers. These civilian medical men now serving in the Army and Navy Medical Corps, have adapted themselves to the duties to which they have been assigned with great éclat and valor. We salute them and also all those medical men of the regular service, whose years of devotion to these services have made these things possible. We only wished that we, ourselves, could have been permitted the honor to serve too.

Re The Outlook.

The outlook on the medical horizon is bright. The constant elimination of practices that were considered fundamental a short time ago and the initiation of new methods based on biological chemistry and physiology, confirms this statement. The brilliant results obtained by the use of the sulfa drugs in general and special use, both in civil and military practice, have been of such a character that what would have seemed incredible but a couple of years ago, has now become routine. Penicillin, Gramicidin and other antibiotics from fungi, are the newest anti-bacterial agents producing metabolic changes or inhibiting growth of micro-organisms in vivo. Further research in mycology will probably yield many new therapeutic agents.

Aviation medicine in the post war days will, undoubtedly, become a specialty in itself. Our country will assume an outstanding position in aviation transportation throughout the globe. Because of the various elements and conditions of travel, reactions from altitudes on the mucous membranes of the respiratory tract, eyesight, hearing, sensorium, etc., our specialty probably, with some modifications, will be the branch of medicine to perform this task. Submarine and subterranean studies must also be included in this group. Our present younger confreres will be the key men in the specialty. Rehabilitation of function and the various plastic procedures are such that special training is necessary. Of course, this is part of our work.

I cannot end this communication without sending greetings and godspeed to all those Fellows of our Association in the Armed Forces at home and overseas.

To our Country in the third year of this World War, a salute,

For we know that this is a fair land,

There are other fair lands, but ours is fairer.

The heritage of liberty and freedom that has been given to us by our fathers, is one that few people throughout all time have been granted.

Let us cherish this heritage with that ardor of those who fought and died,—that this Liberty and Freedom shall be preserved for us and our children.

Oh! that we may be worthy to continue to receive these precious gifts.

Again, it is with great pleasure that I welcome you to New York.

I now declare this 66th Assembly of the American Laryngological Association ready to transact whatever business that may be properly brought before it.

CHARLES J. IMPERATORI, M. D.

President, 1942-1944

108 East 38th Street,
New York.
June 7th, 1944.

MUCOCELE IN FRONTAL AND ETHMOIDAL SINUSES. SIMPLIFIED SURGICAL TREATMENT

HENRY M. GOODYEAR, M.D.

CINCINNATI, OHIO

A mucocele may occur in any of the nasal accessory sinuses but is most frequently found in the frontal and anterior ethmoid cells; occasionally in the antrum and rarely in the sphenoid sinus.

Etiology

Howarth[1] reported five cases with a history of injury in a series of fourteen cases. In one of my cases an external frontal, ethmoid operation had been performed 27 years before. In a second case, an antrum and ethmoid operation had been done fifteen years before.

St. Clair Thomson and V. E. Negus[2] state that a mucocele usually occurs with an "obstruction in the outlet of the cavity and it may be caused by blockage and cystic dilatation of a gland". It is reasonable to suppose that such an obstruction to a mucous gland might well occur secondary to injury or more probably following an obstruction produced by a chronic inflammatory process producing "a cystic dilatation of a mucous gland".

Lobell[3] feels that a degenerative process on a chronic inflammatory basis is very likely the major factor in the development of a mucocele.

Pathology

Logan Turner[4] states that the wall of a mucocele consists of epithelium, usually cuboidal in type, with occasional islands of cilia.

The mucocele usually contains a sterile thick gelatinous mucus, often of a yellow brown color, rarely cholesterol. If infected pus is present the lesion is called a pyrocele.

By a process of slow constant pressure there is a slow absorption of bone and often the floor of the frontal sinus and the lamina papy-

racea are destroyed, and this destruction advances with the increasing size of the mucocele.

Symptoms

From my cases and those observed in the literature, a slight disturbance of vision progressing to a diplopia often accompanied by a dull headache, which may be increased on movement of the eye, constitute the first symptoms of a mucocele. Thus, these patients usually come first to the attention of the ophthalmologist.

The onset is insidious and usually without pain. Three of my patients had had several changes of glasses before the usually globular elastic swelling appeared at the inner angle of the orbit.

In one of my patients, a soldier, severe headaches occurred and in the absence of ocular changes or swelling, malingering was suspected. A density of the frontal sinus, so slight as to be questionable, was the only available finding. After some weeks an external exploration revealed a mucocele completely filling a large frontal sinus.

Later the eye is displaced forward, downward and laterally. Diplopia, limited motion of the globe and a variable degree of impaired vision may be present. Ptosis of the lid, epiphora, and even atrophy of the optic nerve may occur.

There may be a "parchment-like" crackling on palpation, and if pulsation is present it means that the posterior wall of the frontal sinus or the floor of the ethmoid, or both, have been absorbed, and the mucocele is lying in contact with the dura mater.

If infection leads to a pyrocele, the swelling may be red and tender with temperature resembling an acute frontal sinus infection.

Intranasal Examination

Intranasal examination is usually marked by the absence of polypi or pus. In three of my cases I was impressed with the distinct expansion and lowering of the ethmoid bulla which was smooth.

Roentgenographic Findings

Early, there are no characteristic findings but with advancement, defects in the orbital and sinus wall occur. The frontal loses its scalloped appearance and the margined densities become smooth and regular.

Differential Diagnosis

Osteomas are equally insidious and painless in onset but roentgenograms show a sharp demarcated outline of dense bone, and in advanced cases there is no "parchment crackling" or pulsation on palpation.

Malignant neoplasms are usually much more rapid in growth and in the cases of my experience have shown redundant, soft, free bleeding tissues in the ethmoid area.

Other lesions to be borne in mind are gummas, aneurysms, fibromas, angiomas, lymphoid tumors, meningoceles and chronic sinus infections.

Treatment

Doctor William Chamberlain[5] reported six cases of mucocele of the frontal sinus before this society at Washington in May, 1933. In discussing the paper, Doctor D. C. Smith of Boston, remarked, "I have obtained good results by simply leaving the lining of the mucocele in place". This sounded like an excellent idea to me, for a mucocele is lined with epithelium and we were at that time experimenting with skin grafts in an effort to obtain a patent opening into the nose following an external frontal sinus operation. In a mucocele the extension into the anterior ethmoid cells has already been lined by nature with epithelium for us. Why, then, should we destroy it only to have it replaced by contracting, hard, fibrous tissue? Should we remove most carefully only the floor of this extension into the ethmoid area we would have remaining the much sought for epithelial lined duct leading into the frontal sinus. The epithelium lining the mucocele in the frontal can certainly do no harm and pathologically there is no just reason for removing it.

I have used the following plan in operating upon these cases which I believe you will find, if you have not already used it, most satisfactory to the patient and gratifying to the surgeon.

The operation is performed under local anesthesia and a preliminary submucous resection of the nasal septum is done where there is need for better exposure of the middle turbinate.

Should the middle turbinate be wide or cystic in character, it is split with a narrow Bard-Parker knife on a long handle and the lateral one-half is removed with a clean cutting ethmoid forceps. This procedure will give a wide exposure of the ethmoid bulla which the mucocele has usually thinned and displaced downward.

The floor of the ethmoid bulla or anterior ethmoid cell is carefully opened, usually by a single bite or closure of an ethmoid forceps. If the lesion is a mucocele there is a flow of heavy mucoid material. The opening is enlarged by gently inserting a sphenoid biting forceps, removing as much of the floor of the mucocele as possible without injury to its lateral walls which are covered with epithelium and must be preserved.

The thumb is then placed in the upper inner angle of the orbit and the parchment-like bone is pressed back into a normal position. The diplopia is often immediately reduced and improves rapidly thereafter. Novocaine may be injected under the skin first, should there be undue pain on pressure.

No drain is placed in the nose. The patient is ready to leave the hospital in one or two days and the only after treatment is an occasional application of a two or five per cent solution of silver nitrate, should granulations occur.

556 Doctors' Building

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2. Thompson, St. C., and Negus, V. E.: Diseases of the Nose and Throat, ed. 4, N. Y. D. Appleton-Century Co., Inc., 1937, 9, 286.

3. Lobell, A.: Relationship between Mucoceles and Cysts; Report of Cyst of Maxillary Sinus, Arch. Otolaryng. 6:546 (Dec.) 1927.

4. Logan-Turner, A.: Mucocele of the Accessory Nasal Sinuses, Edinburgh M. J. 64:396, 1907

5. Chamberlain, W. B., and Barry, T. L.: Mucocele as Cause of Proptosis. Report of Six Cases, Arch. Otolaryng. 18:172-180 (Aug.) 1933.

DISCUSSION

Dr. Francis L. Weille: I would like to ask if you have seen a multi-locular mucocele.

Dr. Ralph A. Fenton: Mr. President, I should like to ask Dr. Goodyear whether in his judgment this relatively simple and extremely valuable procedure would be adequate to take care of the large mucoceles which we occasionally see that go clear out over the roof of the orbit and encroach upon the frontal sinus, sometimes occupying almost the whole of that cavity.

In such cases, in order to secure complete restoration of the orbital contents to the inner wall, it might be wiser perhaps to resort to operation through the typical external incision, which in our experience it has been possible to accomplish under local anesthesia, without local deformity.

Dr. George B. Wood: I would like to ask Dr. Goodyear his opinion as to the etiology of a mucocele. Does it develop as the result of the occlusion of the sinus ostium or does it begin as a mucoid cyst of the sinus mucosa? In other words, is it possible to have a mucocele inside a frontal sinus that does not completely fill up the sinus cavity?

Dr. Harris P. Mosher: I agree with the writer when he advises letting nature have a try at epithelization of the ethmoid. She will probably do a better job than any of us otolaryngologists have succeeded in during so far.

I was not familiar with the early effects of mucocele of the ethmoid on the eye. The only early sign of mucocele of the ethmoid that I knew was a little egg shell like tumor above the inner canthus of the eye. According to the books this can occur very early, even in children.

Lately at the Infirmary under Dr. Schall, there was an extreme case of exophthalmos due to mucocele of the ethmoid. Dr. Mac-

millan our roentgenologist, in answer to a question, said that some years ago there had been another similar case. The width of the ethmoid was at least twice its normal measurement. Its outer wall was pushed into the orbit, narrowing it markedly and pushing the eye down and out.

At the conclusion of the operation on the ethmoid the displaced wall of the ethmoid was pushed inward and the exophthalmos corrected by restoring the normal width of the orbit.

Dr. H. M. Goodyear (Closing): No doubt it is possible to have a multilocular mucocele, although I have not had one in my experience.

Dr. Fenton feels that the external approach might be preferable in advanced cases. It does take some courage when you have a large swelling about the eye to do so little in the nose toward its correction. If you must do an external operation you should at least have respect for the epithelium which has already been laid down for you, leading from this mucocele into the nose.

Dr. Wood asked a question, which probably is a little difficult to answer. My impression is that a mucocele of the frontal sinus does not occur because the frontal sinus duct is closed. I believe it occurs because the superior orbital cell or an anterior ethmoid cell undergoes inflammatory changes in its lining membrane and a mucous gland occludes and expands.

Dr. Mosher spoke of the x-ray findings, and I agree heartily with him. It is usually very easy to see how much of the bone has been destroyed and the medial orbital wall may be completely gone. Dr. Mosher asked how I feel about doing an external operation on these very large mucoceles. One can do no harm by trying the intranasal operation first.

THE LOCAL USE OF SULFADIAZINE SOLUTION, RADON, TYROTHRICIN AND PENICILLIN IN OTOLARYNGOLOGY

S. J. CROWE, M.D.

BALTIMORE, MARYLAND

Ear and sinus disorders are a frequent cause of disability in our armed forces, particularly among aviators, submarine crews and deep sea divers, who are subjected to frequent and severe changes in temperature and barometric pressure. It has been observed that many of our men in the tropics as well as those in colder and damper climates develop excessive hyperplasia of lymphoid tissue in their pharynx and nasopharynx. This condition in adults as in children predisposes to frequent colds, chronic nasal congestion and post-nasal discharge. In those subjected to great changes in pressure in the air or in the depths of the sea, the ear and sinus lesions may be purely mechanical. These lesions are designated as aero-otitis or aero-sinusitis in American literature, but a better descriptive word used by the British, is barotrauma. In the presence of an acute or chronic infection in the upper air passages these pressure changes often lead to suppuration, which necessitates hospitalization and often discharge from the service.

From years of experience in treating children with somewhat similar conditions, we believe these men can be greatly helped by two simple and practical methods of treatment, local application of a sulfadiazine solution in the nose and nasopharynx to discourage the growth of bacteria, and beta and gamma irradiation of the pharynx and nasopharynx to remove hyperplastic tissue. The combination of sulfadiazine and irradiation is often better than either of them alone.

The solution we use for the treatment of an acute upper respiratory infection is 2.5 per cent sulfadiazine in ethanolamines (Pickrell's solution). The patient is instructed to put one-half medicine

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From the Department of Otolaryngology, Johns Hopkins University and Hospital.

Read in Dr. Crowe's absence by Dr. Donald F. Proctor, Baltimore.

dropper of the solution into each nostril and allow it to flow backward along the floor of the nose into the nasopharynx and pharynx. This should be repeated ten to twelve times a day for three days and four to six times a day for an additional week. Most of the drug administered in this way is swallowed and is of little value, since the blood level rarely exceeds 1 mg. per hundred cubic centimeters after a full course of this treatment. With each application however, some of the sulfadiazine solution is absorbed by lymphoid tissue, and judging from clinical and bacteriologic studies previously reported[1-2] the concentration of the drug in nasopharyngeal lymphoid tissue soon reaches a level that inhibits the growth of pyogenic bacteria.

We have used this method of treatment for prevention of ear and sinus complications of acute colds in approximately 1500 patients during the past three years, with results far better than we have obtained with any other form of treatment. Until the concentration in nasopharyngeal lymphoid tissue has reached a level that inhibits the growth of bacteria, the treatment is of no value. Therefore it is necessary to begin the local application of this drug as soon as possible after the first symptoms of a cold and use it in large amounts and at frequent intervals for at least a week. This treatment, continued for a month instead of a week, is often very helpful in chronic nasopharyngitis if the infecting organism is sensitive to the sulfonamides. Local irritation of the skin around the external nares may be avoided by applying cold cream. In a few patients with sensitive, allergic mucous membranes the sulfadiazine solution causes an increased feeling of rawness, burning and congestion. If this happens the drug should be discontinued, but in our experience less than 5 percent have any symptoms of allergic rhinitis or any evidence of sensitivity to the drug.

The sulfadiazine treatment gives only temporary relief. The real problem is to get rid of hyperplastic lymphoid tissue, which often extends into the posterior nares, grows up on the anterior wall of the sphenoid, on the lateral walls of the nasopharynx, around and in the orifice of the eustachian tubes and in other locations that are inaccessible for surgical removal.[3] It so happens that next to the sex cells, lymphocytes are the most sensitive cells in the body to beta

and gamma irradiation. Therefore, in treating these patients the dosage employed is so small that there is no danger of a burn or a dry nasopharynx in which crusts form.

The action of the irradiation is to inhibit mitosis in the germinal centers and thus stop the formation of new lymphocytes. Observation of hyperplastic lymphoid tissue under this treatment leads us to believe that lymphocytes, like the skin cells, have a brief life cycle, probably not more than two weeks. Under irradiation treatment no new lymphocytes are formed to replace those discarded, and the mass gradually shrinks and finally disappears, leaving the nasopharynx covered with smooth mucous membrane not unlike that on the nasal septum. If the dosage is just right the result is perfect and there is never any recurrence. If the dosage is too great ecchymoses appear in the nasophayngeal mucous membrane, but there is no excuse for ever getting a burn.

At the Johns Hopkins Hospital we use from 800 to 1000 millicuries of radon[4] with a brass filter 1 mm. in thickness, but a more practical applicator for use in the army and in civilian hospitals and office practice contains a radium salt. The applicator is made of monel metal. The handle is 15 cm. in length and the radium containing chamber 15 mm. in length, 1.5 mm. in diameter and 0.3 mm. in thickness. This allows the passage of more beta rays than the 1 mm. of brass in the radon applicator, but has been used with success in the Hagerstown Clinic for the Prevention of Deafness in Children, which is sponsored by the Maryland State Board of Health and the Childrens' Bureau in Washington. The results[5] in 259 patients have been as good as those obtained with radon, and the time required for each treatment is only 6.6 minutes. Of the entire group under treatment in Hagerstown the hearing has improved as much as 40 db. in 39 ears and 10 db. or more for several frequencies in 127. Fifteen chronically discharging ears are now dry and fifteen patients with a history of repeated attacks of acute otitis media have been well since the irradiation treatment was finished. Thirty-two patients treated for severe recurrent upper respiratory infections have had very minor colds during the past winter Ten patients with infected adenoids had severe asthmatic bronchitis with each cold; five of these have had no attacks during the past

winter and the other five are greatly improved, a better result than we have ever obtained from operative removal of tonsils and adenoids in children.

Irradiation treatments should never be given until there has been a thorough inspection with a nasopharyngoscope of the nasal passages, the region of the orifice of the accessory nasal sinuses and particularly the nasopharynx and orifice of the eustachian tubes.

The teeth, pharynx, tonsils and base of the tongue must also be examined. Palpation with the finger or mirror examination of the nasopharynx are not satisfactory, especially in children. It is not the size, but the location of lymphoid nodules that is important. Irradiation treatments should be spaced at intervals of four to five weeks, and the nasopharynx must be examined with the nasopharyngoscope before each treatment. Greatly enlarged adenoids are better treated by operative removal, supplemented by irradiation at the conclusion of the operation. Often only one treatment is necessary, but the nasopharynx should always be examined again six months later and the irradiation repeated if there are any symptoms or visual evidence of recurrence.

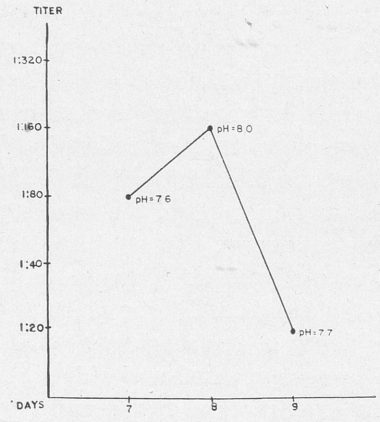
At the Johns Hopkins Hospital we treat approximately 125 patients each month in the outpatient department with beta and gamma irradiation of the nasopharynx. This leaves our beds free for seriously ill patients and at the same time we obtain most satisfactory results in a group of patients, who in pre-irradiation days would have been admitted for operative removal of tonsils and adenoids. For 15 years it has been our practice to irradiate the nasopharynx only, since treatment of the tonsils and pharynx would subject the doctor to too much exposure. If the tonsils are chronically infected they should be removed at operation, but it is our impression from observation on several thousand patients that the nasopharynx is usually the primary focus from which infection spreads to the sinuses, ears and tonsils. If the nasopharynx is freed of lymphoid tissue, recurrent colds become less frequent and severe, and infections in the sinuses, ears and tonsils often heal spontaneously.

In giving irradiation treatments the best protection for the operator is a distance of 30 feet from the applicator. Two pairs of thin

rubber gloves will protect the hands from the beta rays while handling the applicator, but the use of a lead containing apron or gloves, as used in x-ray work, are of no value in protection against gamma rays. When not in use the applicator should be kept in a 7/16 inch hole in the center of a lead cylinder which is from four to six inches in diameter, depending on the amount of radium or radon in the applicator. This hole contains a glass tube filled with alcohol. Before inserting the applicator into the patient's nose, the excess alcohol should be washed off and the applicator dipped into boroglycerine. This prevents nasal secretions from sticking to the applicator and is important, because the hands must never be used to wipe the applicator. The first evidence of too much exposure of the hands shows in the nails, which become cracked and ridged. It is also wise to have a complete blood examination at intervals, but with care there is no danger, as evidenced by the fact that many of us have given these treatments several times a week since 1928.

Tyrothricin

Tyrothricin is in many ways superior to either penicillin or the sulfonamide powders for local application. When it is brought into contact with a sensitive organism it kills almost instantaneously. It is truly bactericidal, while penicillin and the sulfonamides are bacteriostatic and must be in contact with the bacteria for several hours in order to produce a lasting effect. For this reason it is better to use tyrothricin to irrigate an acutely infected antrum, to fill the canal following paracentesis and to wash the wound and surrounding soft parts after every sinus and mastoid operation. If suppuration continues it means the bacteria have not been reached by the drug, or they are resistant to the action of the drug. Both tyrothricin and penicillin when used locally either kill or inhibit most strains of staphylococci, streptococci (both aerobic and anaerobic) and pneumococci, but have no effect on Escherichia coli, Proteus, Pseudomonas aeruginosa (pyocyaneus) and Friedlander's bacillus. Tyrothricin is insoluble in water and does not penetrate tissues when applied locally. It is not only useless, but extremely toxic when injected into tissues or intravenously. Penicillin on the other hand penetrates tissues well and for this reason is of value in the local treatment of chronic infections, provided the concentration in the



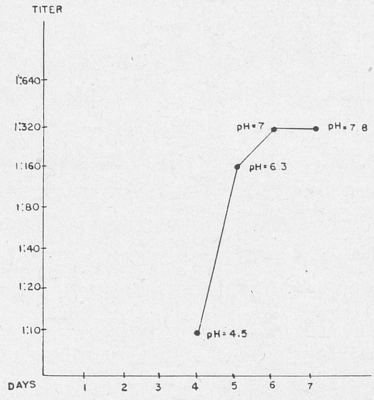


FIG. 1.—Changes in bacterial titer and pH during growth of penicillium. (From Fisher).

FIG. 2.—Shows the daily bactericidal titer of penicillin during growth, correlated with changes in pH. (From Fisher).

wound can be kept at the proper level over a sufficient length of time.

PENICILLIN

Crude penicillin[6] can be made easily and inexpensively by inoculating suitable liquid culture media with spores from stock cultures of penicillium notatum, and keeping the inoculated flasks at room temperature (22° to 25° C.) until pH and titration tests indicate the antibacterial activity has reached its peak. The pH of the culture medium which starts at 3.5 to 4.5 is unchanged for three or four days, then rises rapidly. When the antibacterial activity also reaches its peak the pH is usually between 7 and 7.5, although this figure may vary with each lot. If the alkalinity is allowed to go much above this point the antibacterial activity of the medium is rapidly lost. Figure 1 shows the changes in bacterial titer and pH during growth of the mold penicillium. On the 8th day after inoculation the pH of the fluid under the layer of growing mold is 8.0. The titer, (determined by adding this fluid to a series of broth

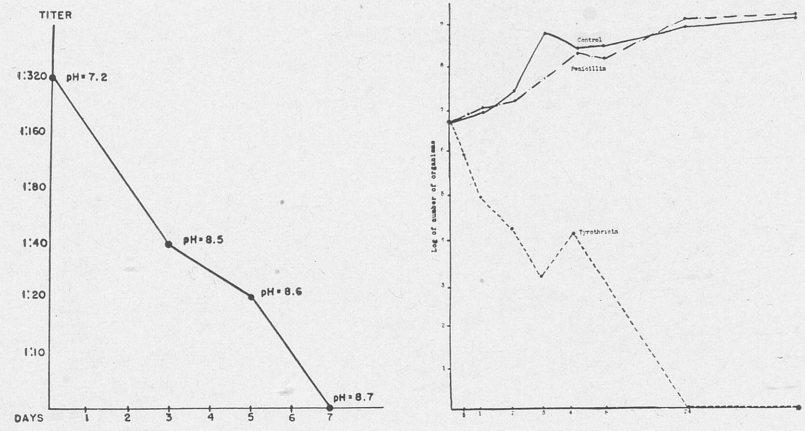


FIG. 3.—Shows the rapid loss of antibacterial activity when the filtrate is kept at room temperature. (From Fisher).

FIG. 4.—Shows how completely resistant a strain of staphylococcus aureus may be to penicillin, and how the same strain is sensitive to tyrothricin.

tubes in the proportion of 1:20, to 1:320 and inoculationg with a stock culture of Staphylococcus) was 1:80 on the 7th day, 1:160 on the 8th day, but suddenly and rapidly fell to 1:20 on the 9th day. Contaminating organisms introduced into the flask at the time of inoculation with the mold, or when testing for antibacterial activity, will also inhibit the growth of the mold or destroy the activity of the penicillin that has been produced. To obtain the most potent penicillin the contents of the flask must be passed through a Seitz filter at the right moment. Figure 2 shows the daily bacterial titer correlated with changes in pH.

Both crude penicillin and the purified sodium or calcium salt of penicillin are unstable. To maintain its potency crude penicillin is best kept in a -30° C. refrigerator. Figure 3 shows the rapid loss of antibacterial activity when the filtrate is kept at room temperature. When needed for a treatment or operation, the required amount is thawed by placing the flask of frozen penicillin in warm water. The stock solution may be melted and refrozen repeatedly

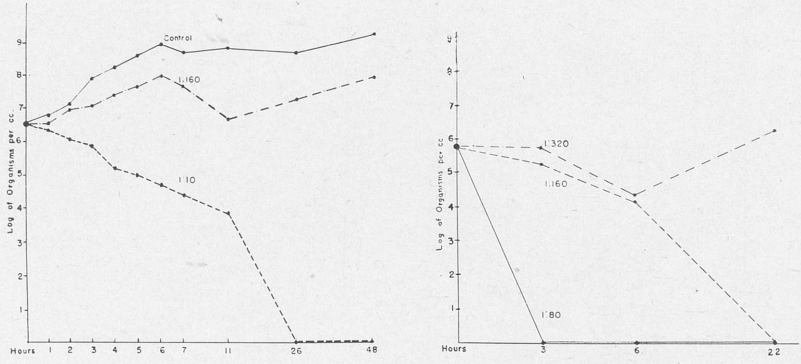


FIG. 5.—Both the concentration of the antibacterial agent and the time factor are important, as is shown in Figs. 5 to 8. Fig. 5 shows the effect of two dilutions of penicillin on Staphylococcus aureus, compared with the normal growth curve.

FIG. 6.—Shows the bactericidal and inhibiting action of three dilutions of penicillin on beta hemolytic streptococcus (group A). (From Fisher).

for as long as two months without loss of potency, provided it does not become contaminated. The most important points for maintaining the potency of penicillin are; adjust its pH close to neutral, keep it frozen and at all times completely free from contaminating organisms.

Many new and possibly better drugs are sure to be found through the intensive studies being made of the chemical composition of penicillin and similar substances, just as the various sulfonamides were developed following the demonstration of the antibacterial properties of sulfanilimide. Treatment with these drugs is of little value however, if the organism happens to be a resistant strain. Careful and detailed bacteriologic studies are essential if the therapy of bacterial infections is to be placed on a rational rather than an empirical basis. The mere identification of organisms is not sufficient, especially if we are dealing with a Staphylococcus aureus infection. Penicillin is useful in the treatment of Staphylococcus infections, but of 90 strains of Staphylococcus (aureus and albus)

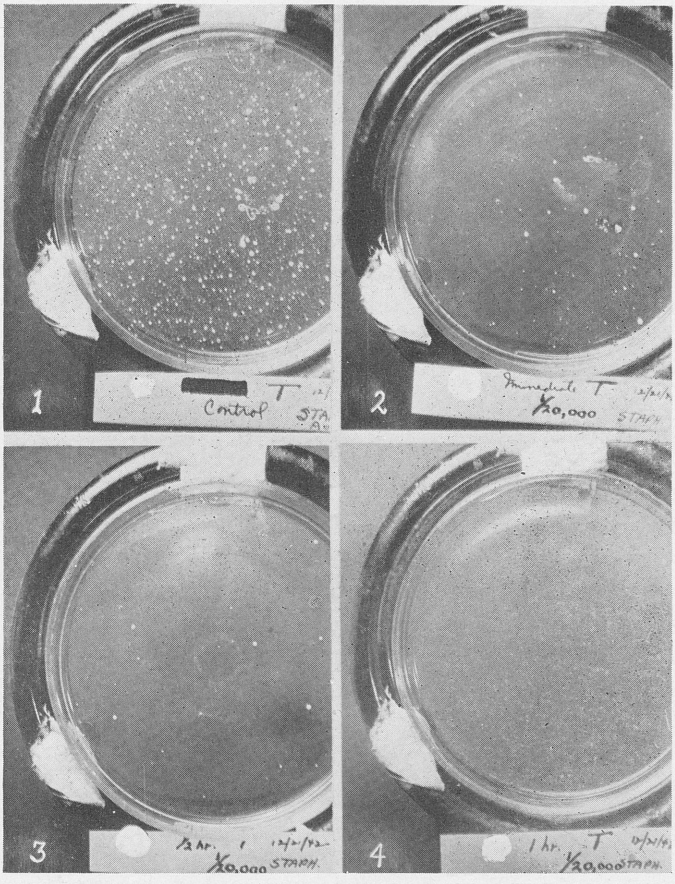


FIG. 7.—These photographs (1 to 4) show the rapidity with which tyrothricin kills staphylococcus, in contrast to the slower action of penicillin, shown in Fig. 8. The plate poured immediately, (i. e. within 2 or 3 minutes after inoculating with one drop of 24-hour staphycoccus culture a 5 cc. broth tube containing tyrothricin in the proportion of 1:20,000) shows a marked reduction in the number of organisms (2). The plate poured at the end of an hour is sterile (4).

Fisher[6] found by titration tests 12 that were resistant to penicillin. In addition, some strains of Staphylococci in local infections acquire resistance to penicillin during the course of treatment.[7]

The organisms found most commonly in acute infections of the upper air passages are beta hemolytic streptococci, pneumococci, anaerobic or micro-aerophilic streptococci and H. influenzae. All of these are sensitive to penicillin and tyrothricin except H. influenzae. In addition to one or more of these sensitive organisms, resistant bacteria such as Escherichia coli, proteus, pseudomonas aeruginosa (pyocyaneus), Friedlander's bacillus and certain strains of staphylococcus aureus frequently are found in chronic sinus and ear infections. Of this latter group Staphylococcus is the only organism that may be sensitive to penicillin, tyrothricin AND the sulfonamides, while Escherichia coli is affected only by the sulfonamides.

When any chemotherapeutic agent is to be employed in the treatment of an infected patient it is important to identify as quickly and as accurately as possible the organism or organisms we have to deal with, and by titration tests determine their sensitivity to the various drugs at our disposal. After two years experience we believe these test tube determinations of the sensitivity or resistence to penicillin, tyrothricin or the sulfonamides are a reliable indication of the result we may expect in the treatment of clinical infection with these drugs. Figure 4 shows how completely resistant a strain of staphylococcus aureus may be to penicillin. If this happens in the test tube where the penicillin is in direct contact with the bacteria, it is evident that this drug could be of no value in the treatment of infection with this particular strain of staphylococcus.

One essential then for the successful treatment of infection with LOCAL chemotherapy is identification of the organisms and determination of their sensitivity. At this stage in the development of local chemotherapy this should be a part of the routine pre-operative examination of every patient. A second essential is to administer the antibacterial agent in such a way that it comes into ACTUAL contact with the infecting bacteria in EFFECTIVE concentration and

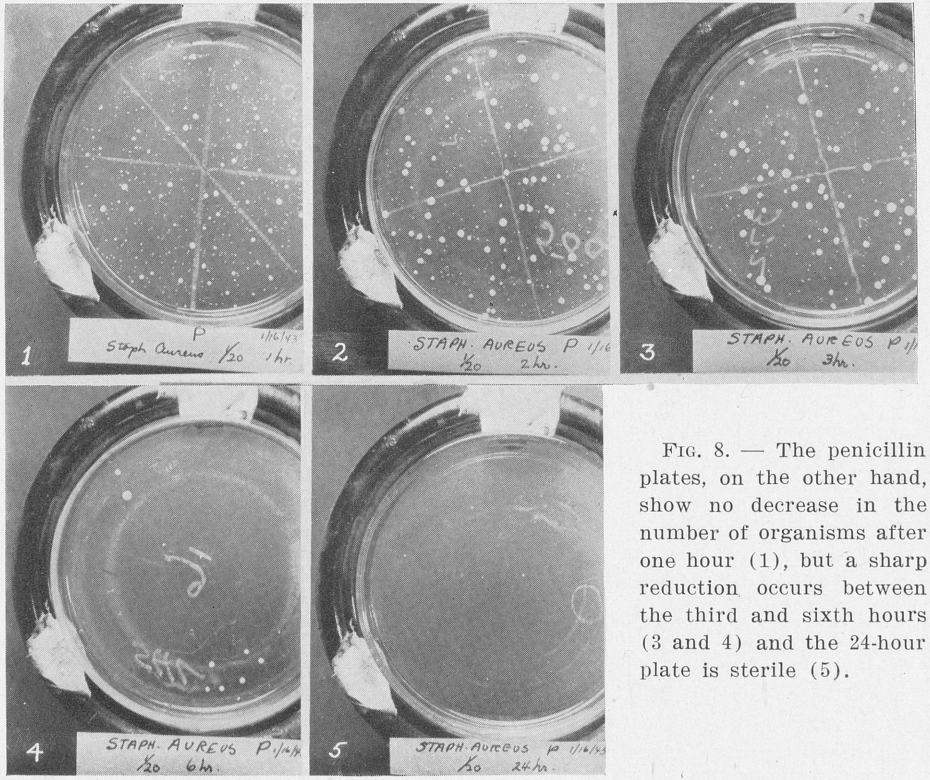


FIG. 8. — The penicillin plates, on the other hand, show no decrease in the number of organisms after one hour (1), but a sharp reduction occurs between the third and sixth hours (3 and 4) and the 24-hour plate is sterile (5).

for an ADEQUATE period of time. Both the concentration and the time factor are important as shown in Figures 5 to 8.

Clinical experience shows that ALL staphylococcus infections do NOT respond promptly or satisfactorily to penicillin, even when given intravenously and in adequate amounts. Acute spreading infections are cured more easily than chronic infections with thick fibrous walls, a poor blood supply and sluggish drainage. This is equally true when penicillin, tyrothricin or the sulfonamides are applied LOCALLY in the treatment of infection. The rapid cure that often follows one irrigation of an acute frontal or maxillary sinus infection with crude penicillin or tyrothricin, suggests that during the acute stage the organisms are superficial and the drug comes into contact with them, while in chronic infections the bacteria are located deep in the tissues. The reason irrigations are less effective in chronic infection is the difficulty of bringing the drug into direct contact with bacteria deep in the tissues. This difficulty can be obviated in chronic infections of the sinuses or mastoid, by combining surgical operation for the removal of infected tissue and local chemotherapy to prevent wound infection. At the conclusion of the operation the bacteria are on the surface of the exposed bone and surrounding soft tissues, and are just as susceptible to the action of penicillin, tyrothricin or a sulfonamide powder as they would be in a test tube.

If we know what organisms we are dealing with and use the drug or combination of drugs that will kill them without injuring the tissues or causing toxic symptoms, often it is possible so nearly to sterilize a wound that post-operative fever, swelling, pain and suppuration are entirely absent. This means much for the comfort of the patient, shortens the period of hospitalization and saves time and energy for the surgeon.

Sensitive bacteria are killed within an hour by tyrothricin, but the action of penicillin (1:20) is at least six times as long. For this reason tyrothricin is preferable in an operation where it is important to reduce wound infection and its consequence to a minimum. If the infecting organism is sensitive to both tyrothricin and penicillin, the best results are obtained by using tyrothricin at the opera-

tion and crude penicillin, diluted with an equal amount of normal saline, to saturate the packs and for any post-operative irrigations that may be necessary. All antibacterial substances, including the sulfonamides, must reach the bacteria in adequate concentration in order to be effective. Tyrothricin is insoluble in water and kills only the bacteria on the surface of the wound, while penicillin penetrates and reaches bacteria in the tissues.

The absence of wound infection greatly reduces pain, swelling, suppuration and the frequency of post-operative treatments. Most important of all, prevention of wound infection reduces post-operative polyp and granulation-tissue formation and thus makes it possible to cure, without a deforming operation, selected cases with a chronic frontal sinus infection. The frontal sinus may be entered through an external incision in the eyebrow not more than 2 or 3 cm. in length. All infected mucous membrane together with the entire floor of the frontal sinus and all orbital extensions of the ethmoidal cells are removed and a large opening made into the nose. The wound and surrounding soft parts are thoroughly washed with 1:1000 tyrothricin and the frontal sinus packed with gauze saturated with penicillin. The end of the pack is brought out through the nose and the external incision closed. There is rarely any post-operative edema of the eyelid and the incision heals per primam. The pack is removed under light vinethene anesthesia 24 to 48 hours later.

The frontal sinus is rendered so nearly sterile by this procedure that excessive growth of granulations is prevented and therefore the opening into the nose does not close, PROVIDED the operation has been thorough and the sinus is irrigated daily for ten days with either tyrothricin or penicillin. The choice of the best drug to use is determined by titration tests. After the first ten days, even though no gross pus has been seen in the irrigating fluid at any time since operation, the frontal sinus, in these non-deforming operations, should be irrigated at intervals with penicillin or tyrothricin for another two weeks. The object of these prolonged irrigations is to prevent the multiplication of any bacteria in the wound that may still be viable.

The following case history is of interest from the point of view of both the general and the local use of chemotherapy:

A child (J. D.—history number 317321) was admitted to the hospital with a septic temperature and a tender swelling under the arch of the zygoma and the temporal muscle. There was no discharge from the ear, no swelling or tenderness over the mastoid and his hearing was good. The illness began with an acute nasopharyngitis two weeks previously. The family physician had given him enough sulfathiazole by mouth to mask the symptoms of otitis media and mastoiditis, but not enough to kill the organisms. On admission he was toxic, dehydrated, and with the exception of the swelling in his temporal fossa his symptoms might have been due to drug fever. A prominent internist recently stated that in approximately half of the patients he had seen in consultation during the past two years, the symptoms were due to toxic manifestations of sulfonamide therapy. A wise rule is to redouble the search for signs and symptoms of sulfonamide poisoning whenever therapy has been continued for longer than ten days.

In this child, however, both the location of the swelling and the mastoid films suggested a rupture of infected zygomatic cells and an abscess under the temporal muscle. At operation extensive destruction of the deeper cells in the mastoid process and an osteomyelitis of the zygoma was found. A simple mastoid operation was supplemented with thorough flushing of the wound and abscess cavity with tyrothricin 1:1000 in distilled water.

The wound was then lightly packed with gauze saturated with crude penicillin, diluted 1:1 with saline. The skin incision was closed with the exception of the lower half inch, through which the end of the gauze was brought out.

The culture at operation showed a heavy growth of beta hemolytic streptococcus. Culture of a piece of the gauze packing removed from the mastoid cavity at the first dressing four days later showed that beta hemolytic streptococci were still present. The penicillin and tyrothricin applied locally at the operation had NOT sterilized the wound bacteriologically, but clinically the organisms had lost their virulence, since there was no pus, no swelling, redness, tenderness or other evidence of wound infection. No further packing or irrigation was necessary. If at the first dressing or any subsequent dressing the patient's general condition is good and there

is no visual evidence of suppuration, the wound should be inspected, but never touched. There is no better way to infect a clean wound with skin organisms than by probing or inserting packs.

This child would have recovered following the operation if local chemotherapy had not been used, but the immediate bactericidal action of tyrothricin plus the prolonged bacteriostatic effect of penicillin, with which the packing was saturated, so nearly sterilized the wound that the skin incision healed per primam, and so far as dressings or irrigations were concerned the patient was well on the fourth day after the operation. Contrast this with the wound left open to granulate and the weeks of dressings that would have been necessary due to wound infection, and you get a clear idea of the value of these drugs in otolaryngology.

SUMMARY

The cardinal principles of successful chemotherapy are: all infecting organisms, both aerobes and anaerobes, must be identified; the bacteria must be sensitive to the drug or combination of drugs used, and finally the drug must come into direct contact with bacteria in the tissues in an adequate concentration and over a sufficient period of time to allow complete bactericidal or bacteriostatic effect. In acute spreading infections the sulfonamides by mouth or penicillin intravenously or intramuscularly are usually most satisfactory, but in treating a chronic mucous membrane or bone infections much depends on the accessibility of the infecting bacteria to the action of the drug. For example, we have often seen pneumococcal meningitis, secondary to chronic ear or frontal sinus infection, temporarily improve following sulfonamides, or penicillin given intravenously, intramuscularly or intrathecally, only to recur again and again until the primary focus has been sought out and drained surgically.

The antibacterial substance penicillin is produced by only a few of the many hundreds of strains of the mold penicillium notatum. Fleming announced the discovery of penicillin in 1929, but more than a decade elapsed before its chemotherapeutic possibilities were fully realized. Since 1940 many reports on methods of preparation, purification and clinical application have been published in England

and in this country, but with few exceptions these studies have been directed toward the use of penicillin in the treatment of pneumonia, meningitis, septicemia and other systemic infections. It is not yet possible to make penicillin synthetically and the available supply of the sodium or calcium salt of penicillin for intramuscular or intravenous injection is still very limited, but crude or unrefined penicillin can be made in any bacteriologic laboratory and is often very effective for local application.

Otolaryngologists have an exceptional opportunity for developing the local use of antibiotic substances in the treatment of infection, since most of the organisms in ear and upper air passage infections are gram-positive and sensitive to the action of one or more of these drugs.

Acute infections that are not walled off by the natural defenses of the body and those extending to the meninges or blood stream must be treated by saturating the entire body with antibacterial drugs, but the local use of these substances to aid in the cure of sinus, ear and pharynx infections has been ignored, with the exception of sulfonamide powders, which are usually applied as a routine measure at the conclusion of an operation, with no knowledge of the identity of the infecting bacteria or their sensitivity or resistance to this drug.

The discussion in this paper is limited to (1) the local use of a solution of sulfadiazine to inhibit the growth of pyogenic organisms in the nose, nasopharynx and pharynx and thus prevent many of the ear and sinus complications of the common cold; (2) the local use of tyrothricin or crude penicillin in the treatment of acute and chronic infections of the ears and upper air passages. If the conditions of successful chemotherapy mentioned above are fulfilled, it is possible to cure an acute maxillary sinus infection with one irrigation, or to so completely sterilize a wound, that following an extensive surgical operation for acute osteomyelitis of the frontal sinus and skull the incision heals per primam with no redness, swelling, suppuration, or other evidence of wound infection; (3) The local treatment and irradiation of hyperplastic lymphoid tissue in the nasopharynx for the prevention or cure of impaired hearing,

secondary to partial or complete obstruction of the eustachian tubes for recurring colds with ear or sinus complications and for some cases of asthmatic bronchitis, especially in children.

JOHNS HOPKINS HOSPITAL.

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DISCUSSION

DR. THOMAS E. CARMODY: I would like to ask a question. When you spoke of the radium salt you spoke of the time but not of the amount of radium salt used.

I would also say one thing you speak of one irrigation curing maxillary sinus cases. Of course we don't know whether we can call it a cure or not, but we have found that same thing with the salts we have used. However, we have found for years that one irrigation sometimes will clear up a sinus anyway, no matter what you irrigated it with.

DR. HOWARD C. BALLENGER: I would like to get the opinion of Dr. Proctor and his associates on the use of powdered sulfa drugs instead of the solution on the nasal mucosa for acute infections. I have used powdered sulfathiazole in the nose and throat in a fairly large series of cases because it stays in contact with the mucosa a

much longer time than the various solutions. If the patient refrains from blowing the nose the powder can be demonstrated the next day.

I have had very good success with this method of treatment.

Dr. John G. McLaurin, Dallas, Texas (by invitation): One finding that was brought out in the presentation of this paper is most remarkable, if the observation was correct, but I have never seen it occur.

I have reference to the disappearance, under the influence of radon, of the lymphoid tissue from the pharynx to the extent that the pharyngeal mucosa appeared to be as smooth and as void of lymphoid tissue as the mucous membrane of the nasal septum. I have used radon and radium in small capsules with an eye on them, so that the capsules could be threaded into the end of a No. 2 Eustachian cannula. The capsule extended just beyond the end of the cannula, so that when the cannula was placed along the floor of the nose the radium capsule could be placed in the nasopharynx.

After a systematic use of radium thus applied, I have seen a shrinkage or atrophy of the lymphoid tissue in the postnasal space, but I have never seen an effect produced where the appearance of the mucosa approached that of the septal mucosa. The shrinkage of lymphoid tissue around the Eustachian orifices and other chronically infected masses of lymphoid tissue in the nasopharynx would certainly be of great value in many instances, but I feel quite sure that a complete disappearance of such tissue in the nasopharynx, as had been suggested, would be detrimental to the patient. This lymphoid tissue is one of our first lines of defense against infections entering through the upper respiratory tract, and I believe that its complete removal by any means would make a patient less likely to satisfactorily handle such infections.

If the lymphoid tissue is of such large amount that it is obstructive either to the nasopharynx or to the Eustachian orifices, or if chronic infection exists in it that is susceptible to acute exacerbations, then it would certainly be well to get a definite shrinkage of the lymphoid tissue or even remove it, if necessary, to create better ventilation to

the Eustachian tubes and postnasal space and also to prevent recurrences of infection.

I realize that in the oropharynx behind each posterior tonsillar pillar there are lateral rolls of lymphoid tissue, and the upper limits of these rolls frequently extend to the orifices of the Eustachian tubes. These are sometimes called the tubular tonsils and they are frequently the site of recurrent attacks of oropharyngitis and nasopharyngitis, associated with blocking of the Eustachian tubes. Radium exposure will have a satisfactory influence in many instances in controlling this type of disturbance.

I want to reiterate that I have never seen a complete disappearance of this lymphoid tissue by radium exposure, and I seriously doubt that from the standpoint of the patient's health it would be well to have it completely disappear.

Dr. DeForest C. Jarvis: I have been very much interested in the subject of penicillin, for there is a variety of penicillium which grows particularly well on apples and apple juice. It is a very stable variety and keeps very well under ordinary conditions.

From experimental work which I have carried out on cows, calves, bulls, horses and chickens, it seems to be as valuable as *Penicillium Notatum,* about which we are hearing so much at the present time. It is effective when given by mouth, when applied locally, when vaporized and from experimental work which I recently completed on cows and calves it seems to be effective when injected into the muscle. Toxic effects are extremely uncommon.

In furuncle of the auditory canal one application generally seems all that is necessary.

Apple penicillium by mouth and locally is useful in the relief of eczema.

In hay fever when apple penicillium is vaporized it seems to open the nose, stops the watery discharge and sneezing.

In streptococcic sore throat, it seems to render the patient symptom free in twenty-four hours.

The common name for apple penicillium is mother of vinegar.

Dr. Bernard J. McMahon: I want to ask how long do you continue the treatment, and what tendency is there toward recurrence in these infections of the sinuses, ears, and so forth with penicillin?

Dr. Donald F. Proctor (Closing): The applicator being passed around is designed to hold 50 mg. of a radium salt. The radium containing chamber is made of monel metal and is 0.3 mm. in thickness. This permits the passage of more beta rays than the applicator used in the treatment of the nasopharynx with large amounts of radon—500 to 1000 millicuries. The radon applicator is screened with 1 mm. of brass, and the number of minutes it is left in place increases from day to day, because radon loses about 1/6 of its strength every 24 hours. On the other hand the time required for an adequate and safe dosage with the monel metal applicator (6 minutes 40 seconds on each side) is fixed and never varies from year to year. This applicator is practical and safe and with it a patient may be treated on both sides of the nasopharynx in a little less than 14 minutes.

It should be emphasized again that the otolaryngologist, who examines the patient and knows the location and extent of the lymphoid hyperplasia, should carry out the radium treatments. This does not mean that the otolaryngologist is entering a field of therapy in which he has had no training. He treats only one area (the nasopharynx) for only one condition (hyperplastic lymphoid tissue), and at all times uses a fixed dosage which years of experience have shown to be effective and safe. No special training in radiology is necessary in order to place an applicator containing a known amount of radium on the exact spot in the nasopharynx where it is most needed. Lymphoid tissue is so much more sensitive to irradiation than the surrounding tissues that no burns or other complications will ever occur, provided the doctor adheres strictly to the prescribed dosage. The otolaryngologist must never attempt to treat malignant growths in the nasopharynx or elsewhere with this applicator.

Lymphoid tissue in some individuals is much more resistant to irradiation than in others. This is true for both children and adults. The proper treatment in a resistant case is operative removal of adenoids, supplemented with irradiation, rather than to risk a burn by increasing the dosage. Infection in the sinuses or

nasopharynx may so stimulate the growth of lymphoid tissue that the effect of irradiation is counteracted. In such a case operation and local chemotherapy, in addition to irradiation, may be necessary to obtain a satisfactory result.

When the nasopharynx is acutely infected irradiation should be postponed. Even with a subacute infection a full dose of irradiation may cause an acute nasophayngitis, but if the usual dose is divided, half being given at the first treatment and the other half a week later, the reaction is much less or entirely absent. In such cases local chemotherapy is a valuable aid.

Irradiation treatments should be given at intervals of a month. Full dosage treatments at shorter intervals may cause symptoms of nasopharyngitis, and if the periods are lengthened the effect of previous treatment may be partially or totally lost. As a rule three treatments at intervals of a month are necessary to obtain the best result. The change in the appearance and amount of hyperplastic lymphoid tissue comes very gradually and may not be complete for two or three months after the third irradiation treatment. The indication for further irradiation, operation or any change in therapy is based on the nasopharyngoscopic appearance of the nasopharynx.

Tyrothricin comes in alcoholic solution. For use at operation we dilute 1 cc. of the alcoholic solution with 100 cc. of distilled water and apply fully to the wound and surroundings. Any trace of salt or mineral in the water causes the tyrothricin to precipitate into clumps, but when distilled water is used the tyrothricin remains in a fine suspension and thus facilitates an even distribution throughout the wound. It must be remembered that tyrothricin, to be effective, must come into actual contact with bacteria, and that only certain bacteria are sensitive to its bactericidal action.

TRAUMATIC DEFORMITIES OF THE NASAL SEPTUM

SAMUEL SALINGER, M.D.

CHICAGO

For a number of years following the early development of the submucous resection of the septum by Killian, Ballenger, Freer and others, rhinologists generally have felt that the technique was adequate. In recent years, however, unsatisfactory results in certain types of cases have led to at least a partial revision of our ideas on the subject and to a number of modified procedures. The object of these newer procedures is not only to relieve the patient of his nasal obstruction, but at the same time prevent distortion or collapse of the nasal tip and to correct such concomitant deformities as may be in intimate association with the deviation.

A careful analysis of a large series of deviated septums has convinced me that a great many of the cases we see are the result of trauma, recent or ancient (generally the latter). It is therefore, fitting that we consider first the primary care of injuries to the nose before discussing the treatment of such deformities as may result from inadequate primary care.

Trauma to the nose is exceedingly common for obvious reasons, but unfortunately unless it is severe, it frequently is overlooked or neglected. This is particularly true in children who sustain innumerable falls when learning to walk and later at play. Even minor trauma at this stage may have serious consequences because of the immature state of the constituent nasal framework, since ossification is not complete until after puberty. The nasal bridge and septum, being partly cartilaginous, fail to fracture completely. Instead, they may bend or curve slightly as a result of the injury and continue then to develop into a deformity which becomes fully manifest only in later years. A child whose nose has been injured sufficiently to cause an external swelling, bruises or hemorrhage followed by nasal obstruction should be carefully examined under a general anesthetic, if necessary. The septum and turbinates should

be adequately shrunk with adrenalin and cocaine, and examined for the following: subperichondrial hemorrhage, lacerations of the mucosa, suspicious bends or malposition. I do not find the septal cartilage dislocated from the vomeral groove as frequently as mentioned by Metzenbaum, but when it does occur, if seen early, it can be easily corrected. However, if the condition has existed for more than a week I find it very difficult to set the cartilage back in the groove.

Hematomata are quite common and should be thoroughly evacuated. It is true that in many cases infection will not take place and the blood clot will be absorbed. However, it is not feasible that one run the risk, knowing the destructive effects of a septal abscess, particularly in a child. Besides, there is no question in my mind that the organization of the blood clot leads to fibrosis and interferes with the further growth of the cartilage. Therefore, it is advisable to incise the mucous membrane, evacuate the clot and then pack firmly to prevent further bleeding. The treatment, here, parallels that of hematoma of the auricle.

Where the septum is found to be bent or curved it may be associated with a depression of the nasal bridge, the nasal bones being crowded between the ascending processes of the superior maxilla. X-ray examination may be helpful in determining this point, but external palpation is generally sufficient. In such cases it will not be possible to eliminate the septal curve without at the same time elevating the nasal bridge. This is accomplished by intranasal pressure with a heavy dull elevator or an instrument which I designed for this purpose.[1] The septum can then be pushed back into place and retained by means of packing or a wax plug. In some of these cases it is necessary to apply a copper molded splint over the nasal bridge to overcome the spreading tendency and reshape the immature bones.[2]

Considering now the more extensive injuries to the septum resulting from crushing blows, automobile accidents, a fall from a great height and the impact of missiles, one is confronted with a complex situation which must be analyzed step by step. Having in mind the ultimate restoration of normal breathing channels, as well as the complications which may be present therapy is applied only

at such time and to such an extent as will not interfere with the more urgent indications. Since the complicating factors may far outshadow the septal injury in gravity, these must be given first consideration. Shock and hemorrhage must be controlled, but at the same time the nasal passages may be cleared of blood clots by aspiration and packing inserted to prevent further bleeding, and what is more important, prevent the aspiration of blood which in a semi-conscious or unconscious patient may have serious consequences.

After the patient has been placed in the proper surroundings for more detailed examination and treatment, further therapy will depend on the patient's recovery from shock and the elimination of intra-cranial injury as a complicating factor. Treatment of a skull fracture, especially when associated with lacerations of the dura, must take precedence over therapy directed toward the correction of the nasal and facial deformities. These may be deferred as long as a week or ten days, if necessary. Nevertheless, the nasal passages can be kept open during this period by means of aspiration and the use of a sterile vasoconstrictor solution. The only contraindication to intranasal medication would be a demonstrable cerebro-spinal rhinorrhea. The latter, being definite evidence of dural laceration would mean the necessity for early repair at the hands of the neural surgeon.

The principles involved in the care of recent injuries to the septum are first, an accurate appraisal of the extent of the trauma; second, the determination of the effect of the trauma on normal nasal function; and third, a plan for the restoration of normal contour together with normal function. The extent of the trauma may vary from a simple fracture or dislocation of the septum to one in which the septum is lacerated, perforated or comminuted with loss of cartilage, mucous membrane and bone. It is extremely important in all cases not only to replace the broken fragments, using sutures in the cartilaginous portion if necessary, but also to see to it that loss of lining mucosa does not lead to crippling contractures which may permanently block the respiratory passages. I show a case illustrating these points. (Fig. 1) A young woman was seen within an hour of an automobile accident in which the windshield glass

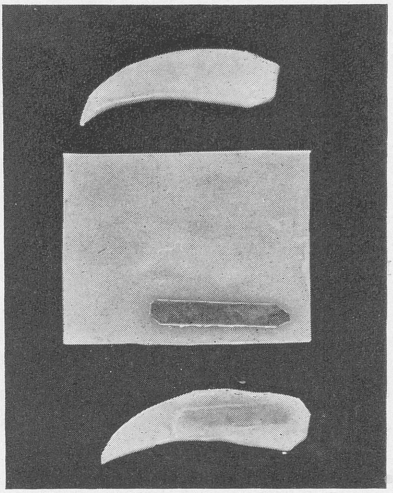

FIG. 1.—Recent laceration cutting across lower half of nose and septum.

FIG. 2.—From above down; wax plug shaped to contour of nasal floor; sheet of dental wax with strip of copper before rolling; wax plug containing copper strip for stiffening.

slashed diagonally across the nose cutting through the left ala, the cartilaginous portion of the septum and the columella, into the right naso-labial fold. When I took these pictures before scrubbing, I failed to notice the depth of the laceration, else my photograph would have shown that the lower half of the nose including a portion of the septum was cut completely across, so that the nose could be lifted as on a hinge disclosing the entire interior. It was quite simple in this case to suture the lacerated septum thus preventing overlapping and subsequent depression of the dorsum.

In another case a man was felled by a heavy box striking him squarely across the nose which was not only crushed, but apparently severed from above downward, remaining attached by the alae. I saw the man for the first time a year or more later, and found that although the nose had been properly sutured in place and had healed (albeit with considerable scarring) he was suffering from an almost

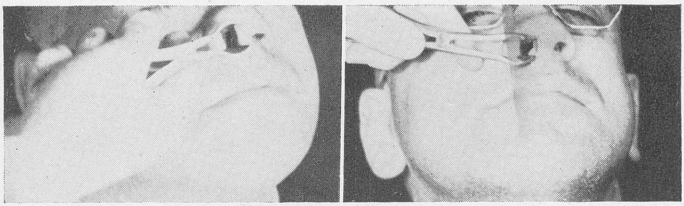

FIG. 3.—Extensive nasal trauma showing partial atresia of vestibule. Enlarged by skin graft.

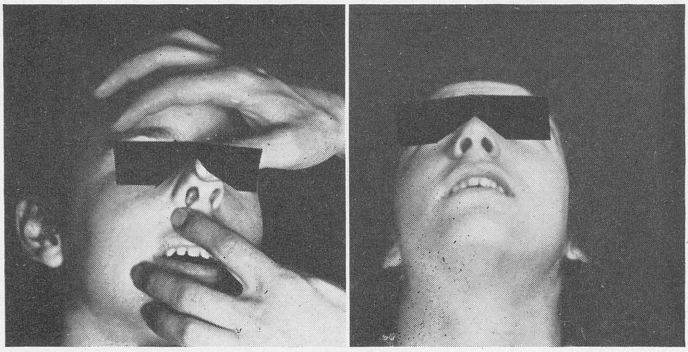

FIG. 4.—Obstruction due to fracture involving the lower end of the septal cartilage. (Patient, aged 13)

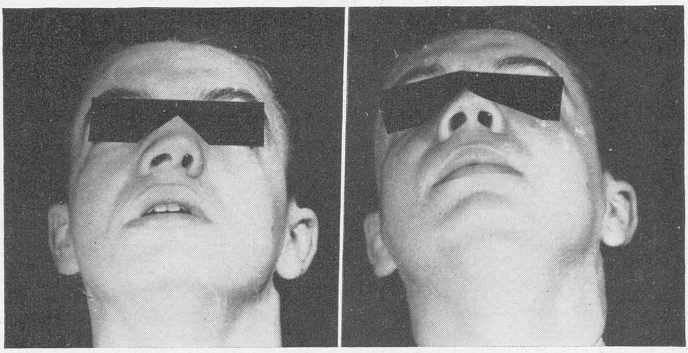

FIG. 5.—Deviation of tip due to septal deformity; dorsum depressed.

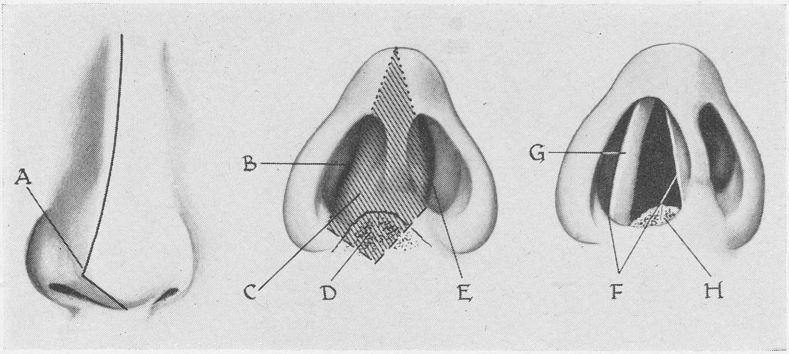

FIG. 6.—Diagram explaining technique in case L.S. (Fig. 4). *A*—angulation lower end of septum. *B*—angle of deviation seen from below; corresponds to *A*. *C*—Lower end of septal cartilage blocking right vestibule. *D*—deviated vomer. *E*—septal border projecting into left vestibule. *G*—angle of deviation exposed after removal of deviated cartilage and mucosa. *F*—right and left mucosa as exposed by speculum; only the left mucosa has been freed; the right mucosa is still adherent. *H*—crest of vomer which is removed to permit repositioning of cartilage.

complete bilateral atresia due to loss of lining mucosa. After opening up the vestibules (Fig. 3) I found the greater part of his septum absent and the space filled with one of his left turbinates, which had to be removed to clear the airway. It was necessary to introduce a skin graft to overcome the atresia which was almost complete on the right side.

In simple fractures of the septum it is usually sufficient to replace the broken fragments where they are retained by means of vaseline gauze packs or wax plugs. It is important when introducing these packs to see that they do not displace torn flaps of mucous membrane, that they are evenly distributed so that pressure will be properly distributed, and that they are left in for a long enough period to guard against recurrence of the deformity. I have kept nasal packs in the nose for as long as a week without deleterious effects, except for the discomfort they caused. Finally, it should be understood that these packs need not necessarily be tight. In most cases the degree of pressure that is required is not as great as is needed,

G.F.TUCKER

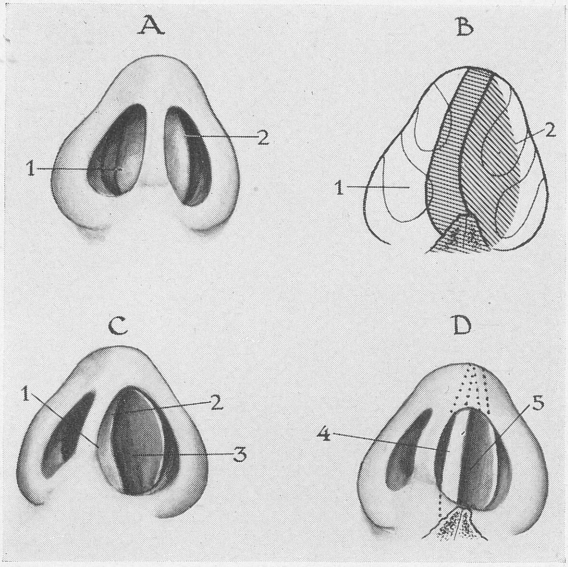

FIG. 7.—Metzenbaum technique. *A*1—angle of deviation; *A*2—exposed free border of septum. *B*—dorsum showing relationship of deviated septum and dislocated lower portion of cartilage to the vomer and the turbinates. *C*—lower portion of septal cartilage exposed as far as angle of deviation by dissection of its mucoperichondrium from the right side only; the mucoperichondrium covering the left side of this portion remains attached. 1—Angle of deviation not quite entirely exposed by dissection of mucosa (2). 3—Deviated cartilage to be preserved. *D*4—shows the cartilage severed at the angle of deviation thus liberating the lower deviated portion which can be displaced to the left by the speculum (not shown). This portion is later replaced in the midline after resection of deviation from 4 backwards and upwards.

for instance, in the control of a severe nose bleed. Also, it should be borne in mind that severe injuries to the septum are usually associated with fractures of the nasal bridge and other facial bones, and that, therefore, the treatment must be coordinated with the other indications.

Where there has been a loss of septal structure it is extremely important to see that the remaining portions of the septum are kept in the midline. Otherwise, adhesions may so distort the passages

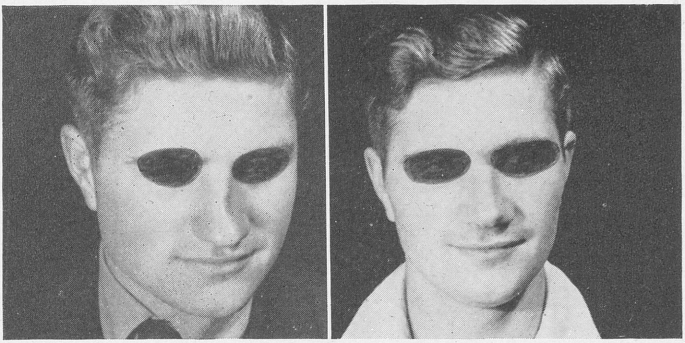

FIG. 8.—Scoliosis involving septum and one nasal bone.

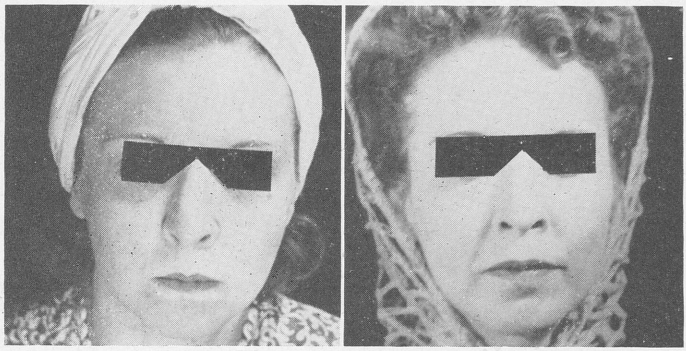

FIG. 9.—Scoliosis involving septum and both nasal bones.

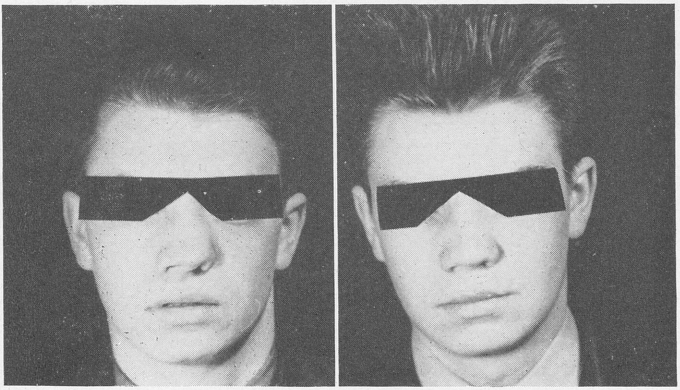

FIG. 10.—Dorsal view of Fig. 5 showing effect of cartilage graft filling dorsal defect.

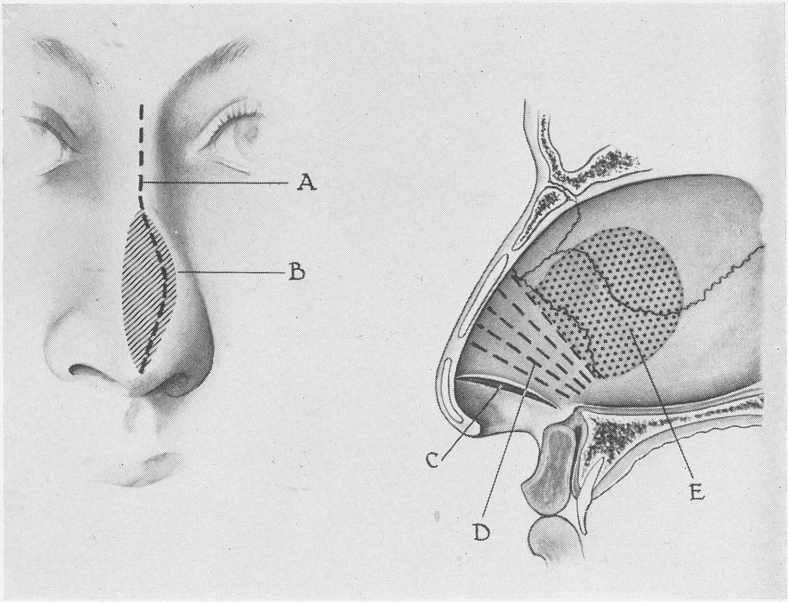

FIG. 11.—Diagram explaining technique in cases shown in Figs. 8 and 9. *A*—dorsal border of septum as visualized and palpated through the skin. *B*—shaded area representing undermining of skin to permit repositioning of cartilage. *C*—incision in septal mucosa. *D*—area of cartilage exposed by dissection of mucoperichondrium showing lines indicating incisions to eliminate the curve . *E*—shaded area representing bone and cartilage removed.

as to subsequently interfere with normal nasal respiration. In such cases I have found that paraffin plugs made out of sheet dental wax and properly shaped are well tolerated within the nose for long periods, are easily removed and replaced whenever indicated (Fig. 2).

Coming now to the septal deformities resulting from inadequate or incomplete primary care, we find that those which give us the most trouble are the ones in which the lower part of the septum has been either dislocated or fractured at an acute angle causing obstruction on both sides (Fig. 4). Also, there are the cases in which

the septum is curved or buckled in the antero-posterior plane giving rise to marked external deformities (Figs. 4 and 5).

In the first instance if the distance from the free septal border to the angle of deviation is short and the nose itself is sufficiently long, it is much simpler to remove all the cartilage and mucosa below the angle of deviation and by removing corresponding wedges from the alar and triangular cartilages achieve a shortening of the nose. Thus, the angle of deviation becomes the free border of the septum and can be maneuvered back to the midline after removing some of the parallel-lying vomer. This is illustrated in Figs. 4 and 6. In this case the associated kyphosis was reduced at the same time.

In most cases, however, it is important that the portion of cartilage lying diagonally across both vestibules be preserved, and here the technique devised by Metzenbaum has its place. Difficulty in replacing this portion of the cartilage is encountered at two points, namely above where the angle of deviation reaches the skin of the nasal dorsum and below where the cartilage overrides the vomer. Since the objective of the procedure is the preservation of that portion of the cartilage lying below the angle of deviation (Fig. 7) it is important to dissect it free only on one side leaving the cartilage attached to the opposite mucosa. When the angle of deviation is reached it must be incised throughout its entire width from dorsal skin down to the nasal floor. This is to facilitate the rotation of the preserved plaque back into the midline. In most cases it also is necessary to free the cartilage from its attachment to the vomer. As a rule the cartilage is found overriding the vomer so that replacement is impossible unless one of two things is done, namely, the overriding portion of the cartilage may be removed or the vomer taken out. In my experience, it has rarely been possible to lift the cartilage back into position on the crest of the vomer. Finally, having freed the cartilage so that it can readily be swung to the midline without tending to spring back to its former position, it is frequently necessary to separate the columellar cartilages from behind, thus forming a groove into which the free border of the cartilage may be fitted. Naturally, that portion of the deviation behind and above the angle of deviation is resected in the usual

manner. The primary incision is closed with one or two silk sutures and the nose packed for 24 to 48 hours.

The second type of deviation which is not amenable to the classic submucous resection is that in which the septal cartilage is buckled or curved away from the midline causing a scoliosis of the nasal bridge. In this type of case we frequently find the entire nasal bridge deviated and it is necessary to refracture the nose bringing the bony arch back to the midline and with it the perpendicular plate of the ethmoid. A submucous resection alone fails to correct either the deformity or the nasal obstruction, because the strip of cartilage which must by this technique be left to support the lower portion of the nose is still curved to one side and holds the mucous membrane like a sheet in its previous position despite removal of considerable underlying cartilage. The procedure which will correct this condition is one in which the cartilage is dissected free on one side only, the dissection being then carried over the dorsal margin directly under the skin which is freed for a distance on both sides forming a pocket into which the cartilage can be pressed after the curve has been eliminated. This is done by making a number of cuts through the width of the cartilage or by removing several narrow strips of cartilage to permit straightening it. Thus, if the nasal bridge is refractured and with it the perpendicular plate brought back to the midline, it will be seen that very little of the septal structure needs to be removed. In most cases the excision takes in the deviated vomer and the sphenoidal process of the quadrangular cartilage which so frequently forms a sharp spur. It is important in these cases to maintain pressure on the previously convex side of the septum for as much as a week or at least until fibrous union has taken place in the hacked cartilage and refractured bones. Figs. 8-11 show the technique described and a typical case.

Occasionally the lower half of the nasal dorsum will be found to be depressed as a result of buckling of the cartilage and no amount of maneuvering can effectively restore the proper height. In these cases it will be necessary at a later date to insert a cartilage graft to fill the defect (Fig. 10).

Another type of deformity is one in which the septum has been dislocated from its dorsal attachment and has carried the tip of the nose with it to one side. As a result the nostril on the deviated side is narrowed and if the cartilage were to be removed in its entirety the tip would sag. In these cases it is necessary to dissect the dorsal skin free from the dorsal margin of the cartilage all the way from the free border back to the point where it begins to deviate. If then, at this point, the cartilage is incised throughout its width it will be possible to reposition it. Here too, it will be helpful to groove the columella from behind to permit receiving the free end of the cartilage.

Occasionally one sees a septum which appears to be collapsed and bulging into both vestibules causing obstruction on both sides. This is usually the result of a previous trauma with abscess formation, absorption of cartilage and replacement with dense fibrous tissue. It is necessary in these cases to dissect out all the fibrous tissue and islands of cartilage, indeed a painstaking and tiresome job which can only be safely accomplished with sharp instruments, good hemostasis and illumination. The liberated mucosa of the two sides is then approximated by a through and through mattress suture, reinforced with packing. Subsequently the dorsum is restored and tip elevated by means of a double cartilage graft.

Summary

1. Trauma to the nose in children should receive careful attention; dislocated septal cartilage replaced, hematomata evacuated and spreading of the nasal bones prevented by proper application of an external molded splint.

2. The primary care of injuries to the septum should include hemostasis, replacement of fragments, careful smoothing out of torn mucosa and proper packing. These must be coordinated with the treatment of the other concomitant injuries.

3. Deformities resulting from inadequate primary care must be corrected with due regard for the necessity of preserving such cartilage as is necessary to the support and contour of the nasal bridge and tip.

25 East Washington St.

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DISCUSSION

DR. THOMAS E. CARMODY: I would like to ask a question. Dr. Salinger, in replacing the cartilage in the Metzenbaum procedure do you sometimes remove part of it in order to get it into the groove?

DR. S. SALINGER (closing): I would like to say that there are other modifications of these procedures some of which I have seen. I know that every individual some time or other in his career has been forced to alter or to improvise a technique according to the indications and I am sure that new techniques will be developed as we go along.

Answering Dr. Carmody: yes, there are times when it is necessary to remove a part of the obstructing cartilage, but the principal objective, naturally, is the preservation of the lower deviated portion which is the chief support of the nasal tip.

TEACHING OTOLARYNGOLOGY IN WARTIME

HARRY P. SCHENCK, CAPTAIN, M.C., U.S.N.R.

OCEANSIDE, CAL.

In any specialty an essential prerequisite for instruction is a sound foundation of general medical training and experience. For this reason, otolaryngologists, as well as medical educators in general, have been keenly interested in the effect upon medical educational standards of war emergency measures primarily concerned with the increased production of doctors. Because wartime modifications in educational methods have influenced the teaching of otolaryngology they deserve careful examination.

The accelerated program for medical education exhibits many revolutionary trends and these are first apparent in premedical training. Here the most important educational experiment consists in the complete elimination of the economic requirement for the study of medicine since those to be educated are no longer dependent upon the ability to pay for their education. Furthermore, the Army and Navy College Training Programs have selected the students on merit alone and regardless of pressure or favoritism. Medical educators are agreed that the present group of premedical students equals or is superior to those selected under the former system.

By subsidizing superior students and those with limited financial resources, by providing undivided time and energy for study, and by selecting students for the study of medicine at an earlier academic period than before the war, medical education may actually be benefited. Some features of acceleration in premedical training, however, have not received general approval. Thus, the elimination of the long summer vacations, increasing the daily and weekly assignments, reduction in elective work and alterations in curriculum continue to be regarded with suspicion.

The Army and Navy are selecting 90 per cent of the medical students. While medical faculties continue to choose the students

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for undergraduate medical instruction, bilateral selection by the school and the student has been eliminated. Upon selection, the student is placed in a pool and neither the school nor the student determine which school shall be attended. After January 1, 1945, the incoming freshman classes will have had only the concentrated military courses.

When, at the onset of hostilities, the medical schools of the nation were confronted by a program entailing a 20 per cent increase in classes, a reduction of the four-year course to three years, and the release of up to 50 per cent of their teachers to the armed forces, it was obvious that a tremendous task lay ahead. In spite of continuous session, modified courses, shortened hospital training and the absence of many former teachers in the services, there has been a determined effort to keep up the quality of the work.

Continuous session has been criticized because it endangered the health of medical students, seriously lowered medical educational standards, prevented orientation of the student, necessitated large classes with minimal personal instruction and fostered an abbreviated curriculum. Whether regimentation will affect the health of the students remains to be determined. The other points of criticism can be profitably examined in the light of the past two years' experience.

In otolaryngology, there has been a definite lowering of educational standards. The deterioration in personal instruction has resulted from depletion of the faculties, however, rather than because of large classes. Curricular changes have, as a rule, not been great, and in some instances the curriculum has actually been augmented. Upon one point there can be no difference of opinion; the accelerated program is definitely increasing the production of doctors.

The clinical faculties in a high percentage of the medical schools of the United States are composed of volunteers. This applies particularly to pedagogy in otolaryngology and other specialties. These faculties were depleted of their younger and most active teachers early in the war. The highly specialized forms of teaching were the ones most affected, and they are continually losing more men formerly regarded as holding key positions. They cannot be replaced.

Moreover, the ranks of the older teachers in our specialty are being constantly thinned by death and infirmity, and at a rate materially increased by the combined burden of an increased teaching schedule and increased private practice.

Clinical instruction in otolaryngology requires teaching material. Serious paucity of such material has resulted from economic improvement in the lower wage groups and ready employment at high wages. The out-patient departments of many of our teaching hospitals are almost deserted. Continuous session, requiring dispensary teaching in the summer months when acute infections of the ear and upper respiratory tract are at a minimum, still further limits clinical instructions.

In spite of these handicaps, undergraduate instruction in otolaryngology compares favorably with that of prewar sessions. Because the object of undergraduate instruction and internship is to equip the individual for the general practice of medicine and not for direct entry to any specialty, the imminence of military service has affected the curriculum very little. If to an already crowded curriculum anything is to be added, certainly nothing in the nature of broad fundamental training should be dropped.

Since younger medical officers have shown deficient training in certain directions the following suggestions are made. There is need for emphasis on hearing tests, malingering tests and the interpretation of audiometer findings. The contributions of aviation medicine to otology are neglected. There is not sufficient attention to the subject of acoustic trauma which, because of the large number of blast injuries, has become of increasing importance. Tropical warfare has demonstrated again that diphtheria is endemic in the tropics and that mycotic and fungoid diseases can reduce the fighting strength of our forces. Instruction in the management of these diseases should be specific and adequate.

The armed forces regard the recent graduate as most desirable, and from this group intern-residents must be chosen. Even in peace time there were more approved internships than eligible candidates. To solve the conflicting interests of military necessity and civilian medical services, two plans were adopted. The 9-9-9 intern-resident

program applies to both militarized and non-militarized personnel in fixing the period of appointments as hospital personnel. It applies to militarized personnel only in the limitation of appointment to one period each as intern, assistant resident and resident. Those not subject to military orders continue to be eligible for prolonged training. Actually, this has afforded two-thirds the former number of interns, one third of whom are deferred to serve as junior residents, and one-half of the assistant residents may be deferred for a third nine-month period as residents. The second plan, the hospital quota program, allots to each hospital a quota made up of both military and non-military house officers.

In each crop of medical students receiving training under this system, several hundred who would normally continue their preparation for careers in medical teaching and research are lost. This is of grave concern, for from this group future leaders in the specialties would normally emerge. There is no provision for the training of a continuous flow of teaching personnel. To minimize a serious permanent loss to medical education, those young men of ability, interested in academic careers, must be returned promptly after the war and every facility provided for their development.

The prospective specialist in otolaryngology should first narrow his interests to his chosen field during service as an assistant resident or resident. It is this phase of instruction which has suffered most.

Regardless of whether the internship is rotating, mixed or straight, the reduction of the internship to nine months lowers educational standards because the total time for patient contacts is reduced. In addition, the depletion of medical and nursing staffs and the dearth of hospital employees make it impossible to impress upon the interns the thoroughness and attention to details essential for the development of sound professional habits. Observations of recent graduates in military service reveals that training in medicine and general surgery has been adequate, but at the expense of instruction in otolaryngology and the other specialties.

The various services have appreciated the need of specialized training for medical officers. This has been provided to some extent

by advanced instruction here and abroad. Such instruction, however, is designed to meet wartime problems and not to make specialists. The larger military hospitals hold regular conferences and staff meetings and service hospitals adjacent to medical schools provide medical officers for special clinical instruction to students. Members of medical faculties are appreciated lecturers before service hospital staffs. None of these represent postgraduate instruction in an acceptable form. Because of the tempo of the war, the rapid expansion of the medical corps, and the availability of reputable and well-trained specialists for supervisory posts, postgraduate training is not of immediate importance to military authorities.

One extensive movement in graduate medical education, Wartime Graduate Medical Meetings, has been in operation for a year and a half. Under the auspices of the American Medical Association, the American College of Surgeons and the American College of Physicians, and with the authorization of the three surgeons general, instruction has been carried out in service hospitals throughout the country. Aided by the deans and faculties of 52 medical schools, facilities have been provided for ward rounds, clinicopathologic conferences, study groups and practical demonstrations. The national faculty of this organization consists largely of the Fellows of the American Otological Society and the American Laryngological Association and other internationally known leaders in this specialty.

Clinical conferences and study groups in the larger medical centers are jointly planned by service hospital authorities, medical school faculties and regional committees of Wartime Graduate Medical Meetings. The ensuing discussions have benefited those in civilian practice and those in active teaching positions even though the primary object has been continuous graduate education to meet the needs of physicians in military service. There has followed a better understanding and appreciation of new clinical problems arising from the war.

Postgraduate education in the pre-war sense is a diminishing entity. Even when such training is obtainable, civilian physicians find it impossible to embark upon a one- to three-year course of

special training. The short brush-up course, while neither producing specialists nor serving as a short cut to specialization, offers the only opportunity to obtain the stimulus and training afforded by contact with medical leaders. Such programs for small groups have been conducted by the Army, Navy, Public Health Service and the American College of Surgeons. It is well within the province of the national otolaryngological societies to develop, operate and direct short courses for small groups. Through their membership all the important clinical facilities in the United States are available. As a temporary measure until the end of the war, this type of instruction will in part compensate for the diminishing availability of organized postgraduate teaching.

In the specialties there is need for thought and planning for the postwar interval. Postwar educational facilities for returning medical officers must be provided if the best interests of the public and the profession are to be served. Those returning to hospital duties will succeed in obtaining adequate supervision and instruction but those, who of necessity, return directly to private practice will need and demand a thorough review of recent developments in otolaryngology or basis instruction in the specialty. The deans and faculties of medical schools should prepare to put into effect immediately upon the cessation of hostilities a plan to provide short courses of two to eight weeks' duration in which concentrated and practical instruction will be provided. The Council on Medical Education and Hospitals of the American Medical Association is cognizant of these problems and is actively engaged in formulating plans which cannot be estimated with reasonable accuracy.

Those engaged in medical pedagogy must formulate positive plans not only for the duration of the war but for the postwar period and its difficult readjustments.

Santa Margarita Ranch.

AEROSINUSITIS—A RESUME

PAUL A. CAMPBELL, LT. COLONEL, M.C.
(by invitation)

RANDOLPH FIELD, TEXAS

As expansion of the activities of man led him to explore the higher reaches of the atmosphere, the depths of the sea, and the earth, the effects of barometric pressure variation upon the closed gas and air containing cavities of the body became more and more evident, and consequently the subject of more and more investigative effort.

Until high altitude flight became somewhat common, very few persons had been subjected to great barometric pressure change. The numbers of aviators, deep sea divers, caisson workers, and those in other occupations requiring exposure to pressure changes were never great. Consequently, interest in pressure effects was limited to a few investigators, who for the most part confined themselves to the more spectacular effects—namely, decompression sickness and anoxia.

World War II, however, produced an incentive for mass high altitude flights, as the man "on top" was demonstrated to have great tactical advantage. Furthermore, flight at high altitude was proven to have certain economical implications, for the simple reason that thin air decreases the friction to which an air foil is subjected. Consequently, air speed for any given power output is greater. In fact, at 35,000 feet it is some seventy per cent greater than at ground level.

Over-the-weather flights are also of great advantage. Weather is still the Nemesis of the airman, and he is willing to sacrifice much in the way of comfort to avoid it.

Simulated altitude flight by means of decompression or pressure chambers has been of interest to research groups since the middle

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From the Research Laboratory of the Army Air Forces School of Aviation Medicine, Randolph Field, Texas.

of the nineteenth century. However, mass employment of simulated flight in the indoctrination and classification of flying personnel awaited the Altitude Training Program, one of the outstanding achievements of World War II. This procedure consists of a simulated flight to high altitude for the purpose of acquainting flying personnel with oxygen equipment and indoctrinating them in the use of oxygen at altitude.

The magnitude of the program can be realized when one considers that 275,000 air crew members received this training in 1943.[1] When one adds this number to the large numbers of men participating in aerial flight, one may understand that at this time a considerable number of people are being subjected to extreme barometric pressure change—the environmental factor which in certain instances may lead to the development of aerosinusitis.

Historical: Paul Bert, in his monumental volume, "La Pression Barometrique,"[2] published in 1878, did not allude directly to the effect of barometric pressure variation upon the nasal accessory sinuses. However, he did refer to the experience of M. Cezanne, who reported that during caisson work his employees sometimes experienced an issue of blood from the nose and throat, and that at times headaches persisted for several days. Again, he quoted Babbington and Cuthbert as reporting in 1861 that some of their workers suffered from nosebleed and headaches. The first detailed description of the entity later to be known as aerosinusitis was written by Marchoux and Nepper[3] in 1919, who after experiments in a decompressure chamber stated:

"Our observations lead us to believe that great importance is to be attached to the integrity of the rhinopharyngeal mucosa. Any obstruction which prevents the rapid establishment of pressure equilibrium between the cranial air spaces and the outside atmosphere involves the possibility of a series of misfortunes in which pain is one of the least and syncope one of the most serious. All become more marked as the pilot approaches land and at a time when he needs his faculties for a safe landing. While the atmospheric pressure decreases by 10 cm. of mercury between sea level and an altitude of 1,000 meters, it does not vary more than 4 cm. between

6,000 and 7,000 meters. It is therefore in the lower layers of the atmosphere that the defect is most felt."

Since that statement, several treatises concerning the effects of barotrauma on the sinuses have been published by various investigators, including Behnke and Willmon,[4] Brouwer,[5] Pastore,[6] Von Diringshofen,[7] Hermann,[8-9] Salem,[10] and this observer.[11-12]

Definition: Aerosinusitis[11] is an acute or chronic inflammation of one or more of the nasal accessory sinuses produced by a barometric pressure difference between the air or gas inside the sinus and that of the surrounding atmosphere. It is commonly characterized by congestion and inflammation of the lining structures. Pain over the area of the sinuses is usually present. Mucosal or submucosal hemorrhage may occur. The condition may at times result in temporary or permanent change in the mucous membranes lining the sinuses, depending upon the amount of barotrauma involved.

Incidence: Aerosinusitis, in terms of an entire population, is a very rare condition. In terms of that portion of the population which is being exposed to extremes of barometric pressure change, either in simulated or actual flight or in deep sea diving, it is not so rare. In the Altitude Training Program, depending more or less upon the month of the year, the geographical position of the altitude chamber, and using residual pain over the sinuses as the criterion, the incidence has been reported to vary from practically none to three or four per cent of those making flights above 25,000 feet. In a series of 35,000 simulated flights, again using this extreme criterion, the incidence was one and nine-tenths per cent. In a similar series of 9,000 man flights, only five-one hundredths of one per cent presented sinus symptoms serious enough to require hospitalization. Statistics of actual aerial operations are still meager. From evidence arriving from the field, it would seem that the incidence is only a small fraction of that occurring in the Altitude Training Program. From the standpoint of anatomical incidence, the larger sinuses are more often involved than the smaller ones. Thus, the frontal and maxillary sinuses are the sites of predilection. It has been the experience of this observer that frontal sinuses are involved more often than the maxillary sinuses. However, Wright[13] reports the maxillary sinuses to be the site of greatest incidence in his series.

In terms of seasonal influence, the incidence, as would be expected, more or less parallels that of infectious rhinopharyngitis.

Physical Considerations: The density of the atmosphere which surrounds the earth's surface decreases with altitude. This decrease is such that at about 18,000 feet the atmosphere is only half as dense as at sea level; about one-fourth as dense at 34,000 feet; and about one-eighth as dense at 48,000 feet. As can be seen diagrammatically in Figure 1, the change is not linear per unit of altitude but curvilinar.

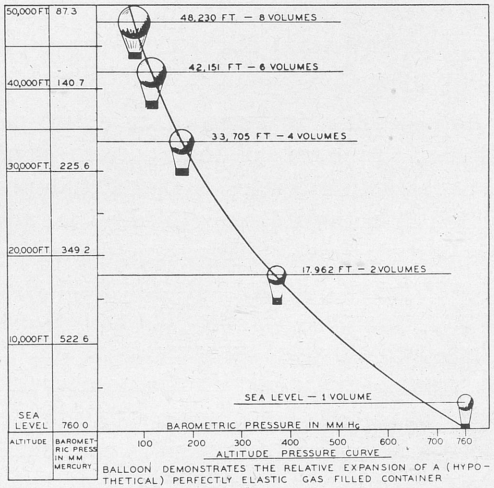


FIG. 1

Any material moving from a region of greater density to a region of lesser density, as in ascent, expands until equilibrium is established between the forces acting inside the material and those outside.[12] In the opposite manner, movement from a region of lesser density to that of greater density produces contraction.

If a closed gas or air containing cavity is moved from a region of greater density to one of lesser density, as in ascent, it will expand. The degree of expansion is limited by the pressure differential between the air or gas inside of the cavity and that outside, and also by the restraint afforded by the elasticity of the material which makes up the structure. On descent, the opposite effect occurs when the forces are applied in an opposite manner.

On the other hand, a cavity with an opening accessible to the outside is affected during ascent or descent, only in that the contents of the cavity flow outward during ascent. On descent, material from the outside is drawn inward until equilibrium is established. (Fig. 2).

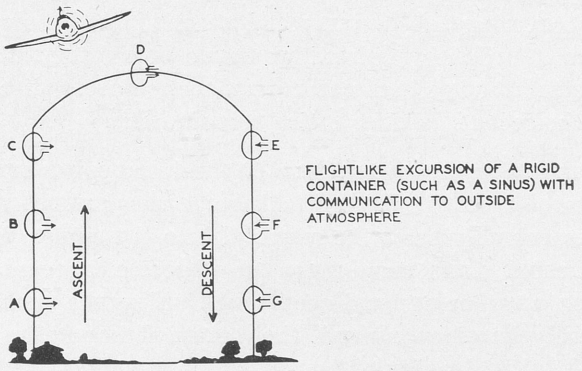


FIG. 2

Categories: As was pointed out, a cavity, such as a normal sinus with an unobstructed ostium, will during ascent allow its gaseous contents to move outward. On descent, the flow of air or gas is in the opposite direction. As this movement is not accompanied by any change in the structure of the sinus, no sensation is felt and no symptoms occur.[11]

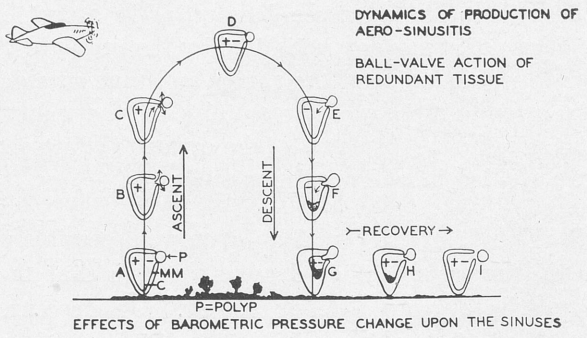


FIG. 3

This free flow of gaseous matter may be altered or prevented by two conditions. First of all, by the presence of pus, fluid, or similar substance covering the ostium; and second, by the obstruction of the ostium afforded by redundant tissue or anatomical deformity. The first situation has been categorized as *non-obstructive aerosinusitis,* and the second as *obstructive aerosinusitis.*

Non-obstructive aerosinusitis: If fluid, mucous, or pus covers the ostium of a sinus, changes in barometric pressure taking place during descent may press the material into the sinus. In this manner, uninfected sinus membranes may become infected. Infection is, however, a rather unusual occurrence, unless the membranes have been severely barotraumatized, their natural immunity lowered, or they are insulted by virulent organisms.

A few such cases have been reported during the recent influenzal outbreak. The usual history has been that of a mild aerosinusitis following a simulated flight with apparent recovery after a period of twenty-four hours or so. However, from three to five days later, in certain instances, a rather serious purulent sinusitis has developed.

Obstructive aerosinusitis: When the ostium of a sinus is blocked by swollen or redundant tissue or an anatomical deformity, a flap valve may be formed during pressure change, and the second category, that of *obstructive aerosinusitis,* results.

The forces' extent can best be understood by referring to a diagram illustrating the cycle of events resulting from an aerial excursion, be it simulated or real.

Let us consider the case of a maxillary sinus with a small polyp or bit of redundant tissue situated in such a manner that it can be pressed against the opening, thus forming a flap valve.

During ascent, the flow of the air or gaseous content is outward. Consequently, the flap is merely pushed aside and the pressure equalized. On descent, however, the situation is quite different, as the heavier outside atmosphere presses the flap against the ostium, or as you may wish to consider it, the lighter inside air sucks the tissue into the ostium. An air-tight seal is thus formed. As descent is continued, a pressure differential develops between the air or gas inside the cavity and that outside. The result is that of a partial vacuum.

Equalization of forces acting and re-acting is a law of nature. The relative negative pressure inside the sinus can be equalized by any space filling phenomena. The most usual space filling phenomena are swelling of the mucous membranes or fluid production, either into the cavity of the sinus or into or under the lining membranes. As soon as sufficient space is filled, the flap valve is released and recovery begins.

Pathological changes inside the sinuses: At least two observers have examined directly the tissue alterations which have taken place following extreme barotrauma in man. Hermann[8-9] has reported mucosal detachment and submucosal hematoma. Wright was forced to perform a Caldwell Luc procedure upon a maxillary sinus which failed to respond to conservative mangement. He reported:[13] "The periosteum of the canine fossa was somewhat bound down by fibrous reaction. The bone was thin and easily removed. On the opening of the sinus a bluish membrane bulged out under pressure. Incision of the membrane revealed a cavity entirely filled by many blood clots enclosed in membranous sacs and not free in the antral cavity. There was no free space in the antrum not filled by these rounded enclosed clots of old blood. There was a dark red color to

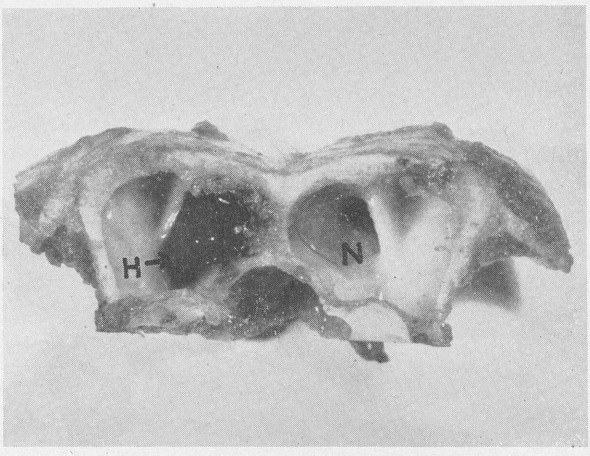


FIG. 4.—Gross appearance of hematoma—*H*, after sinuses were uncapped. *N* is normal control sinus which was not obstructed.

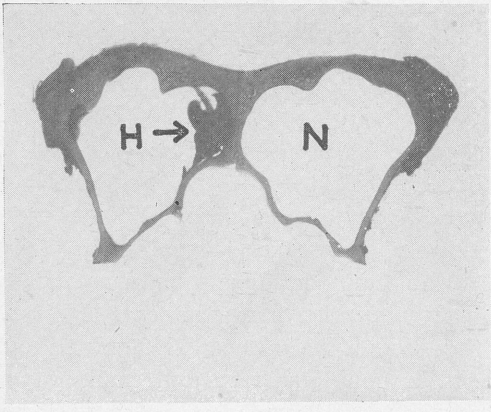


FIG. 5.—Transverse section through sinus showing hematoma—*H*. *N* is normal control sinus.

all the structures. No blood could be evacuated without incising the membrane covering the clot. The entire membrane with the clots was removed and an intranasal opening was made. Microscopic examination showed the bleeding to have taken place in the subepithelial tissues of the mucous membrane."

In our laboratory we have produced aerosinusitis in a dog by the following procedure. The animal was placed under nembutal anesthesia and ascended to 28,000 feet in an altitude chamber. During ascent oxygen was administered as necessary. At 28,000 feet the chamber was leveled off and the area about the exit of the left nasal frontal duct was rather tightly packed with vaseline gauze. The animal was then returned to ground level.

After one hour, examination of the sinus in which the ostium had been obstructed disclosed a large hematoma extending upward from the internal orifice of the nasal frontal duct and occupying the principal part of the medial wall of the septum (Fig. 4).

There were also multiple small hemorrhages in the mucosa. The mucosa of the unobstructed sinus was normal. A transverse section of the sinus is seen in Figure 5.

Microscopic studies showed that the small hemorrhages arose between the epithelium and the periosteum in highly vascularized areas of the mucosa (Figs. 6a, 6b, and 6c).

Presumably the large hematoma had a similar origin with the blood spreading along an indefinite line of cleavage between the dense fibrous tissue of the periosteum and the loose-textured connective tissue of the mucosa. At no point was the epithelial continuity of the mucosa broken. It is of interest that the superficial part of the hematoma was composed primarily of red blood cells while the deeper part consisted principally of coagulated plasma with networks of fibrin enmeshing scattered leukocytes (Fig. 7).

The experimental lesions were similar to those found in the surgically excised mucosa of a maxillary sinus of the patient seen by Wright and Boyd,[13] who kindly loaned me a section of the tissue demonstrated in Figure 8.

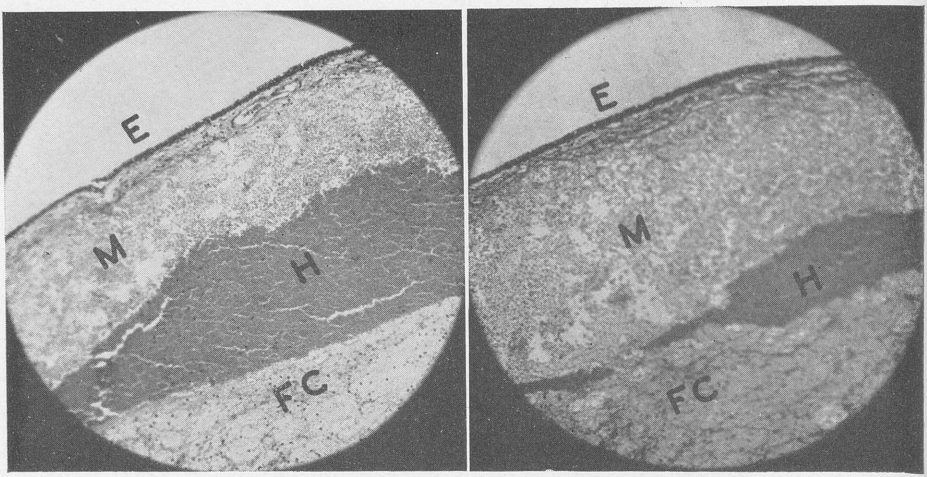


FIG. 6a FIG. 6b

Hemorrhagic areas in the mucosa.

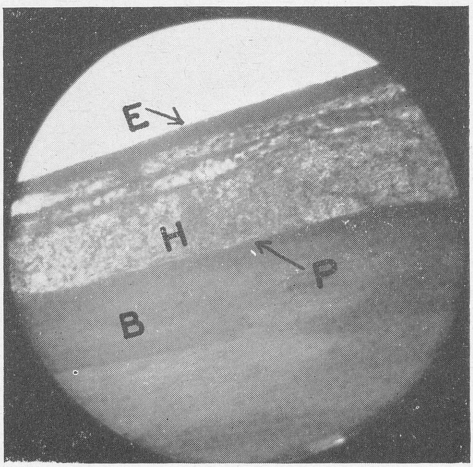


FIG. 6c.—*E*—Epithelium; *M*—Mucosa; *H*—Hemorrhage; *FC*—Fibrin clot; *P*—Periosteum; *B*—Bone.

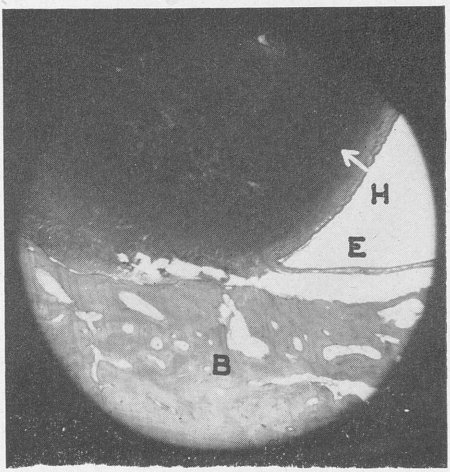


FIG. 7.—*B*—Bone. *E*—Reflected epithelium and mucosa. *H*—Large hematoma.

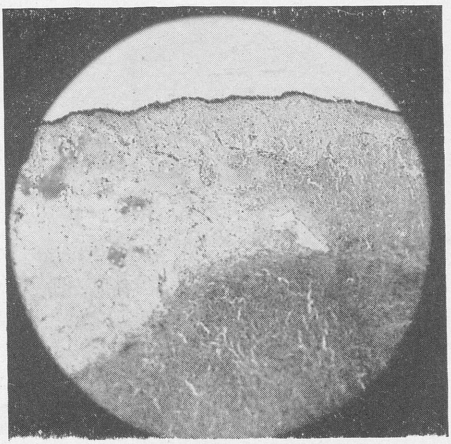
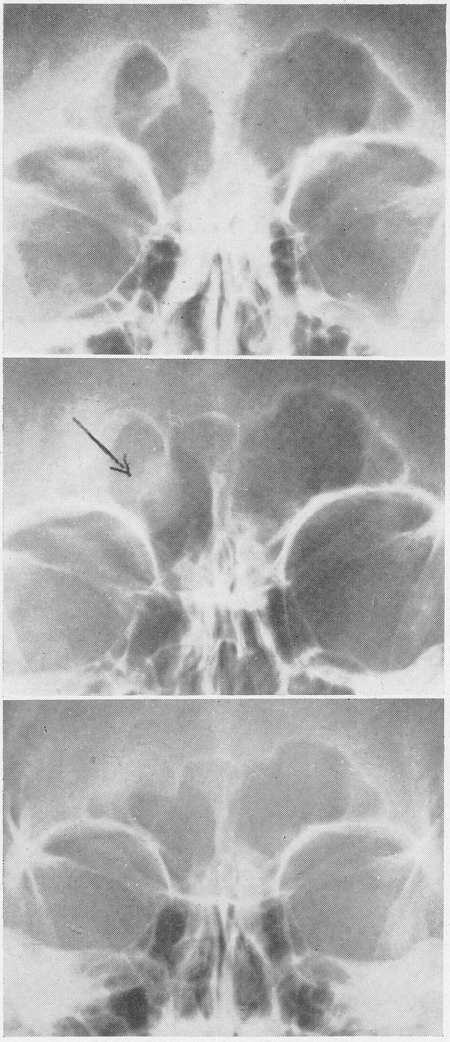


FIG. 8.—Mucosal hematoma from maxillary sinus of case seen by Wright and Boyd.

Roentgenographic Findings: In view of the spectacular pathological changes noted at times, similar unusual roentgenographic findings would be expected. In many instances, especially in *obstructive aerosinusitis*, this is the case. Of most interest is the mucosal hematoma, illustrated in the following series of roentgenographic plates.



Serial x-ray photographs demonstrating mucosal hematoma formation and resolution.

9a—Normal sinus, July 6, 1943.

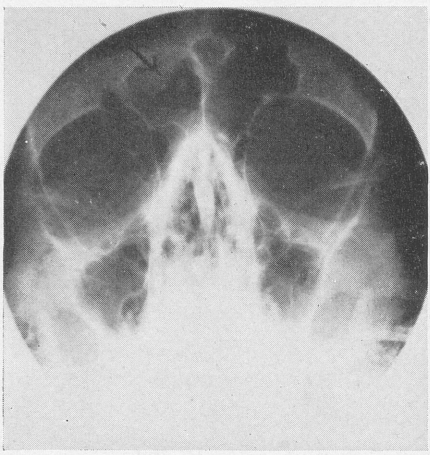
9b—Mucosal hematoma following altitude chamber flight, August 11, 1943.

9c—Resolution, January 25, 1944.

The condition illustrated followed an ascent to 38,000 feet in an altitude chamber, where the subject remained for a short period of time. Descent was then made at a rate of 6,000 feet per minute. When a level of 26,000 feet was reached, he suddenly noticed a sharp pain in his left supra-orbital region. The sensation was described as feeling as though something had burst over and to the nasal side of his eye. The pain was relieved slowly by leveling off and then re-ascending about 1,000 feet. Descent was then begun again. During this descent it was necessary, because of pain, to level off five or six times before reaching ground level. There were no painful after effects. However, an x-ray picture of his sinuses was taken (Fig. 9b) which on comparison with a routine x-ray picture (Fig. 9a) taken a month before demonstrated a large dome-shaped swelling of mucous membrane in the left frontal sinus, which had not been present at the time of the previous x-ray picture. Serial x-ray pictures taken subsequently demonstrated a slow resolution of the process with complete recovery about five months later (Fig. 9c). Roentgenographs (Fig. 10) are random roentgenographic studies illustrating various hematomas which have been seen in our laboratory.

The roentgenographic findings in *non-obstructive aerosinusitis* are in most instances not diagnostic, as the amount of fluid or pus pressed into the sinus is never large.

Diagnosis: Aerosinusitis must be differentiated from purulent and catarrhal sinusitis. The differentiation is usually simplified by the elicitation of a history of pain over one or more of the sinuses during or shortly after exposure to extensive barometric pressure change. In the more severe cases, especially those in which there has been stripping of the mucous membrane or mucosal or submucosal hematoma, the patient may describe the onset of the pain in such terms as "feeling as though a bee had stung him over the eye," as "being struck by a sharp piece of metal," or as "being struck in the head with a club or ball bat." In the less spectacular instances the pain may be described as having slowly developed after return to ground level. Epistaxis occurring during or after exposure to barotrauma is extremely suggestive. Roentgenologic plates disclosing opacity or a thickened lining membrane in the absence of pre-



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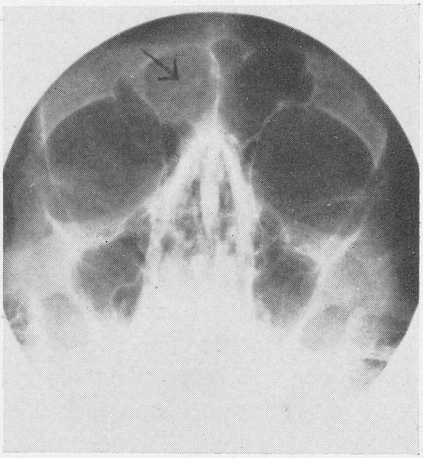

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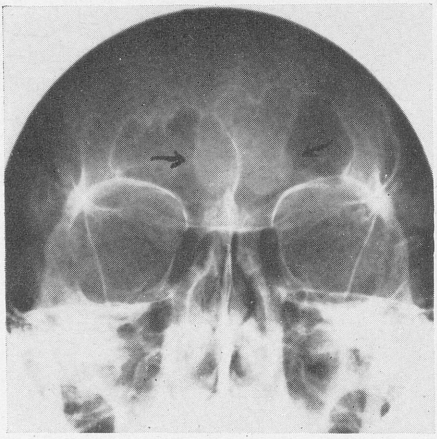

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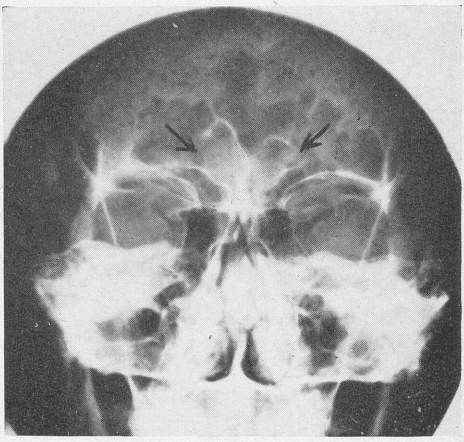

10d

FIG. 10.—Random roentgenographic studies illustrating mucosal hematomas. *a*—demonstrates aerosinusitis with hematoma. *b*—same case, showing half resolution two months later.

vious sinusitis are strongly indicative. This is especially true if mucosal or submucosal hematomas are demonstrated. Leukocytosis and fever in most instances are absent in aerosinusitis. It must be borne in mind that aerosinusitis can occur in a sinus which is already infected, thereby altering the clinical picture of each of the entities.

Course: The milder cases of this manifestation of barotrauma are self-limited, resolution taking place in from a few hours to a few days. The more severe cases may run a course of from a few days to a few weeks. Absorption of mucosal or submucosal hematomas may necessitate a period of from two to six months.

Complications: If the barotraumaized tissues do not become infected, recovery is usually rapid and unspectacular. Should the mucous membranes become infected, as rarely occurs, a severe purulent sinusitis may result. Hemorrhage into the cavity is not rare. As the hemorrhage is space filling, it is a mechanism by which the inside and outside pressures are equalized, thereby releasing the flap valve and usually alleviating the symptoms other than epistaxis. Mucosal and submucosal hemorrhages are absorbed after a time.

Treatment: From the standpoint of prophylaxis, those who have upper respiratory infections, nasal allergy, or nasal obstruction from anatomical deformity or polypi should not undergo great barometric pressure change until it is known that adequate nasal and sinal ventilation exist. This, of course, is difficult to ascertain exactly. However, in most cases freedom from obvious obstruction or pathology can be accepted as presumptive evidence. To err on the safe side, the Altitude Training Program has adopted a test flight to an altitude of 5,000 feet, which serves as an elimination procedure for those with either blocked sinuses or ears.

In developed cases, the otorhinolaryngologist is called upon to use his best judgment for establishing ventilation. Non-obstructive aerosinusitis usually responds to the simplest forms of treatment. Shrinkage of the offending structures will usually allow normal ciliary activity to evacuate a sinus of extraneous materials which may have been pressed into it.

Obstructive aerosinusitis presents a somewhat different problem. Here the primary objective is to equalize the pressures inside and outside the cavity. A return to the altitude at which the block formed, then emptying the nose of secretion, followed by shrinkage of the offending tissues and slow return to earth would seem the ideal procedure. However, before attempting such a measure, one should know of the underlying pathology before subjecting the patient to further barotrauma.

Any procedure which will increase fluid production inside the sinus cavity will aid pressure equalization. Heat of some form thus seems indicated. At times it is necessary to mechanically move a turbinate or a polyp to break up the block. Usually, however, shrinkage by any of the usual vasoconstrictor agents will suffice.

Following recovery from aerosinusitis, the nasal passages should be carefully inspected and any obstruction removed before further exposure to altitudinal change. In a few instances, a submucous resection may be necessary.

In conclusion, it must be pointed out that the pressurization of aircraft should offer the best method for the prevention of any of the body's reactions to altitude or to barometric pressure change. Thus, as is often the case in Aviation Medicine, the ultimate solution to the Flight Surgeon's problem must, to a great degree, be made the responsibility of the aeronautical designer and engineer.

RANDOLPH FIELD, TEXAS.

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DISCUSSION

DR. RALPH A. FENTON: Mr. Chairman and Fellows, you have listened to an exposition by one of Aviation Medicine's most brilliant physicists. This is one of our own group of otologists, a man who came into the Air Service from the Reserve and to whom the Air Service recognizes its great debt. He has benefited all of us, in civilian as well as in military medicine. He has, through experimental facilities which he has himself designed and carried into effect at the Air Medical School in Texas produced authoritative statements upon matters which have puzzled many of us who have had to do with traumatisms of this type in civilians.

Those of us who live in the West have to deal occasionally with matters of this type in persons who "go over the hump," as they say, past the top of the Rockies, and at times, particularly in the winter, are obliged to fly rather high to get over storms. Of course they don't reach the extreme elevations which are simulated in the pressure chamber which is used for training purposes; but such people, particularly those in whom latent sinusitis exists, middle aged business men, people who get on the plane, tired and worn out, go to a great height; formerly (before they began omitting the Salt Lake stop) they would have to climb down to 4,000 feet, say from 16,000 or 17,000, (which is the height reached to avoid a storm in the Rockies); some of those people have had a lot of trouble.

The same thing occurs in traveling down the Pacific Coast from Portland to San Francisco, going over the top of the high moun-

tains at the northern boundary of California, and some individuals become extremely unhappy. Those who have had existing sinusitis may develop serious complications. This has caused considerable concern to the personnel of the air lines and also to their nurses, many of whom have now been withdrawn from active participation in caring for the civilian population, and for whom girls who lack the medical training of nurses are being substituted.

I would ask one question: whether in Colonel Campbell's judgment, the use of oxygen is of any particular value in obviating such disagreeable consequences of altitude flight.

Dr. Robert F. Ridpath: I would like to ask the essayist if he has noted an increase in other symptoms which are associated with sinus involvement such as headaches and pain, during the ascent or the descent in the particular cases he mentions. Are they more severe or less? Also is there vertigo?

Dr. Thomas C. Galloway: Professor Ivy at Northwestern University and Dr. Greenwood have been doing some interesting experiments with a low-pressure chamber in the treatment of respiratory infections. In an "altitude chamber" in some instances a very striking improvement occurs in acute sinusitis.

They are not quite ready to explain exactly how they get this result although probably they get it partly by differences in pressure inside and outside the sinuses, partly by increased blood flow. The return to normal pressure is made more slowly than it would ever be made in an airplane, but the results are reported to be gratifyingly good. With the group around the medical school, who have access to this chamber, there has been a considerable vogue of that treatment. Would Colonel Campbell think that this might be a rather dangerous procedure under some conditions?

Dr. M. Martyn Kafka (by invitation): I have enjoyed listening to Colonel Campbell's talk very much and I feel that his presentation has been excellent.

I have a few comments to make regarding this subject. As a former U. S. Army Flight Surgeon I have had the pleasure of being

associated with army pilots. I know of many of their problems related to aerosinusitis. As a matter of fact, I have treated many such conditions not only as an Army Flight Surgeon but also as an Attending Consultant Flight Surgeon engaged by the Republic Aviation Corporation at Farmingdale, L. I. While at the Republic plant I had the opportunity of watching over the Thunderbolt test pilots who were constantly flying the untried fighter planes to very high altitudes. Some of the test fliers had reached altitudes as high as 40,000 to 42,000 feet. During these high flights some of the test pilots developed aerosinusitis. One aviator had formerly been attached to "The Flying Tigers" stationed in China. One morning he was testing an untried Thunderbolt at 40,000 feet. He suddenly felt an excruciating pain in his left antrum. It was so intense as to necessitate his landing his plane immediately. Upon careful examination I observed a cavity in one of his left upper pre-molar teeth, communicating with the left antrum. It appears that when he was aloft at 40,000 feet the mucous membrane of his nose became markedly edematous. Evidently, air was accumulating in the left antrum through the tooth cavity while the nasal mucous membrane was gradually getting swollen. When the cavity of the premolar became suddenly closed the expansion of the air in the left antrum greatly increased which caused the pain. I treated his congested membranes with an astringent. He immediately felt much relieved and stated he had to continue with his testing. Due to the war effort I permitted him to resume his work. However, I requested that he return for further treatment. I also instructed him to visit a dentist. Later he reported that following several test flights he never experienced this pain again. He continued to use an astringent nasal spray before each test flight.

I feel that the first duty of the Flight Surgeon should be to shrink the nasal mucous membranes prior to each test flight. Furthermore, each test pilot should have a periodical dental examination.

Another pilot had been suffering from bronchial asthma. Nevertheless, because of the urgency of the war effort he was assigned to test planes at an altitude not higher than 15,000 feet. He was instructed to use oxygen at altitudes above 15,000 feet under all circumstances, as there were occasions when he had to fly at altitudes

as high as 20,000 feet. At no time did he develop any asthmatic symptoms.

In addition, there were some individuals in the low pressure chamber in high altitudes ranging from 40,000 to 46,000 feet who developed symptoms of air embolism. One man had severe pain around the cervical vertebrae. Another experienced agonizing pains over his right shoulder joint in altitudes above 35,000 feet. Another had severe frontal headaches at an altitude of 40,000 feet. The cause of his pain was due to congestion of the nasal membrane.

In closing I would like to ask Colonel Campbell if nasal astringents are routinely used in low pressure chamber work in his physiological investigations; also does he believe that pilots should routinely receive nasal shrinkage before beginning their test flights.

Dr. Rosenberg (by invitation): I might mention what has been described as "office" air otitic sinusitis. I think we have all seen the results of too severe suction in treatments of sinusitis.

Hematomas will certainly occur in certain instances.

I wonder whether the sinus showing diffusion might not be allergic. In certain allergic conditions we find that a sinus diffusely opaque to x-ray may in a few minutes be completely clear. Might not that in certain instances be a hematoma?

Dr. S. Salinger: The picture which Colonel Campbell presented is very clear.

Considering the predisposing causes, I wonder whether the size of the cavity bears any relationship to the incidence of these hemorrhages. The illustrations which I have seen have all seemed to be in rather large frontal sinuses.

Lt. Colonel P. A. Campbell (closing): Gentlemen, first of all I would like to have on record that this presentation would have been impossible had it not been for the coöperation which I have had with the various physiological units in the field and of my colleagues who, knowing of my interest in this particular subject, have loaned me case records of x-ray pictures, incidence statistics, etc.

Second I wish to thank Dr. Fenton for the nice compliment. In answer to the questions he asked, in our cases at least the use of oxygen has not proven of much value in the treatment of aerosinusitis. Aerosinusitis seems to be a chance condition. As I told you a little while ago, it is a very rare condition.

In order to produce aerosinusitis the right obstruction has to be in the right place at the right time and when there are three variables the probabilities of all getting together at once are rather low.

In answer to Dr. Ridpath, who asked if chronic sinusitis is aggravated during aerial flight, I believe it is. We have not seen extreme exacerbations in those people but we advise everyone not to fly if they have productive colds. If they have sinusitis they should not fly if they possibly can avoid it. If they must fly, then shrinkage with some of the better vasoconstrictors will in most instances carry them through their journeys in good shape.

Dr. Galloway spoke of therapeutic possibilities. If you remember the diagrams, sufficient ascent could completely empty a sinus. That is, if a person were taken high enough. At 18,000 feet half the sinus could be emptied; at 36,000 feet about three-quarters; and at 48,000 feet seven-eighths of the sinus. Naturally, it is difficult to go that high.

Just how you get those people down again I don't know, and you must get them down. You can't leave them up there. When you come down the forces act in the opposite manner and I suppose if you can keep everything open during the descent that you could arrive on ground level with the sinus remaining empty.

Most people feel better at altitude. For some reason or other in the early stages of anoxia the symptoms are the same as in the early stages of intoxication, that is, the subject feels better.

I wonder if many of the people who were improved by altitude flights merely felt better because their sinuses were empty and they were anoxic.

I do not believe that a septal resection will aid many of these cases. If there is definite impingement of the septum on the tur-

binate, pressing it against the ostium, then a submucous resection is indicated.

Dr. Kafka, I have already answered your question. Many of these instances are chance conditions. We must have the right thing at the right place at the right time. Septal deviation must be in the right place.

Now then concerning whether or not we were dealing with submucosal hematomas. In the first few instances the x-rays we saw were inconclusive. We thought possibly we were dealing with something not a submucosal hematoma, and if Dr. Law will remember, I pestered him with some of my early cases. He seemed to believe that we were right in our diagnosis of submucosal hematoma. Since that time they have been seen in dogs. Now we are pretty certain that we are dealing with a true submucosal hematoma.

In answer to Dr. Salinger concerning the relationship of the size of the sinus and the incidence of aerosinusitis, I am certain there is very definite relationship. The larger sinuses are the ones in which incidence is greatest. They are the only ones in which we see submucosal hematomas. That I believe is due to the relative capacity.

Thank you, gentlemen. It has been an honor to appear before you.

SUMMARY OF SOME KNOWN FACTS CONCERNING THE COMMON COLD

A. C. HILDING, M.D.

DULUTH, MINN.

It seemed to me that it would be of interest and value to summarize all the known facts of the common cold. I scarcely realized how much is actually known. Nor did I realize how difficult it would be to evaluate some of the published items as to their being established facts, or mere probabilities. A better title for this paper would have been, "A Brief Discussion on the Common Cold."

For the purposes of this paper, our meaning of the common cold is that widespread, mild, afebrile, epidemic rhinitis occurring principally during autumn and spring months. It is characterized by copious, thin, mucinous, nasal discharge and a self-limited course of two or three days, which is often followed by or complicated by suppurative infections of the nose, sinuses, ears and lower air passages. There are undoubtedly a number of conditions very similar to the common cold. These may be difficult to differentiate from it at times, but that does not invalidate our conception of the cold as a disease entity with a specific etiology.

It is now well established that the etiological factor is a filterable virus. Kruse[1] determined this in 1914. It was corroborated by Foster[2] in 1916 and again by the masterly work of Dochez and his group[3, 4] in the 1920's. It seems to me that the work of this group is a model piece of careful research and deserves special mention.

They first determined the nature of the normal bacterial flora in the nasopharynx in a group of individuals over a period of months. Then they looked for essential changes in this flora during attacks of colds and found none. They sought for a laboratory animal that was susceptible to colds and found it in the chimpanzee. They studied the symptoms of spontaneous colds in these apes and determined that they were similar to the disease in man. Then, isolating a colony of chimpanzees and exposing them to a patient with a cold,

they proved that these apes were susceptible to human colds. Again quarantining a colony of apes and keeping them under strict surgical asepsis, they produced colds with the filtered nasopharyngeal washings from patients with colds. They ruled out the tiny filter-passing gram-negative organisms, so frequently found, as the cause by demonstrating that colds were produced when these were absent, that filtrates from normal individuals containing these gram-negative filter passing organisms failed to produce colds, and that fresh cultures of this organism failed to produce colds.

They cultured and subcultured the virus and found that it was still active, after the eighty-eighth subculture. They transmitted colds from one ape to another by means of the virus. They then studied the virus itself and learned the following facts.

It grows on chick embryo medium under anaerobic conditions, but not under aerobic. It becomes inactivated at 56° after a short exposure. It retains its activity for two weeks at icebox temperature and for four months if frozen and desiccated. When passed from one individual to another, it retains but does not enhance its virulence. A culture loses its activity rapidly if removed from the incubator and placed on ice. Acacia in the culture prevents this loss. They differentiated it from the virus of herpes by means of intracerebral inoculations in the rabbit.

They further determined[4] that this virus promoted the growth of pathogenic organisms in the throats of patients with colds.

Olitsky and McCartney[5] found that virus actively transmitted colds if removed during the first 20 hours of symptoms. There is some evidence that contagiousness begins even a few hours before the onset of symptoms.[6] The incubation period was found to be between 8 and 48 hours.

Chapman and Hyde[7] discovered antigenically that the cold virus was different from that of influenza. Shibley, et al.[4] demonstrated on their apes that the cold virus does not protect against influenza.

The normal bacterial flora has been studied by many workers in various parts of the world. It is essentially the same everywhere and contains alpha and gamma streptococci, Neisseria catarrhalis,

diphtheroids, staphylococcus albus and aureus, beta hemolytic streptococcus, pneumococcus and Hemophilus influenzae.[8] Burky and Smillie[9] and their co-workers who studied isolated communities found the flora to be essentially the same in Labrador, Alabama, and the Virgin Islands. The flora at Spitzbergen also was found by Paul and Freese[10] to be very similar excepting that pneumococcus and hemolytic streptococcus were rare. At some seasons and during some years, one type of organism will predominate above others. These bacteria play a big role in colds, not as a primary cause, but as secondary invaders. They do not seem to have the power to initiate infection. It is these organisms which produce suppurative rhinitis, sinusitis, otitis media, bronchitis and pneumonia. They may be carried by a large portion of the population in a community with impunity until an epidemic of colds occurs, and then they invade, giving to the epidemic its characteristic complications. For instance, Smillie,[11] in 1940, reported a study from an orphanage. Pneumococcus type 14, was present in all of the children, but all were well. Then an epidemic of colds went through the institution. The numbers of pneumococcus, type 14, increased in the throats of the children. Several developed otitis media and two contracted pneumonia. Pneumococcus, type 14, was recovered from the sputum of these two and from the ear discharges of those having otitis media.

Just what the relation between the cold virus and these pathogenic bacteria may be is not clear. Smillie in the study just quoted, Shibley, Mills and Dochez[4] and others have found that the growth of pathogenic organisms is stimulated or promoted by the virus. Whether this is due to direct stimulation of the bacteria, to opening up of better growth conditions, or to reduction of tissue defense does not seem to have been determined.

It has long been held that exposure to sudden temperature changes, especially if it involves chilling and wetting, causes colds. This is such a universal experience that it cannot be disregarded. There have been several controlled observations which seem to indicate definitely that exposure is at least a factor in causation of colds. Shade[12] reported a four-fold increase in incidence of respiratory infection among 17,000 German troops when the weather became severe. Gahwyler[13] made controlled observations on two different oc-

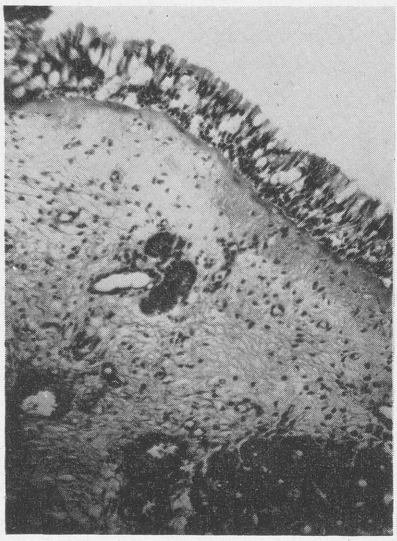

FIG. 1.—Edema of submucosa in early stages of the cold. Specimen removed two hours after onset of symptoms.

FIG. 2.—Necrosis at surface of epithelium on third day.

casions. The first involved 2,700 men sent into the wet trenches compared with 5,300 kept in the barracks and the second 4,500 men kept out in a sharp wind for three days compared with 3,500 kept in their quarters. In both instances the incidence of colds was about four times as great in the exposed group as in the protected one. In both instances, apparently there was chilling from wind or dampness. Subjection to dry subzero weather does not seem to have the effect of promoting colds. In fact, in my home community we are apt to be rather free of colds in subzero weather. This fact has often been noted in connection with arctic explorers and other outdoor people subjected to very cold weather.

Mudd, Grant and Goldman[14] measured temperature changes in the nose and throat following chilling of distant portions of the body. Under such circumstances, the temperature drop of the nasal mucosa was as much as 6° C.

Spiesman[15] reports a similar study on 63 individuals in which he was able to divide his subjects into three groups according to the promptness, character and duration of the temperature change in the nose. The first group was normal and manifested a prompt drop in temperature of the nasal mucosa with a return to normal in about ten minutes. The second group was composed largely of individuals who frequently suffered from colds. In this group, both the temperature drop and the return to normal were delayed. Then there was a third, or hypersensitive, group in which there was a rise instead of a fall.

These studies would seem to supply experimental basis for the relation of colds to chilling.

In summing up the etiological factors it appears to be reasonably certain that the cause of colds is a specific filterable virus, that bacteria are secondary invaders and that chilling is a prominent predisposing factor and acts by reducing the temperature in the nasal mucosa.

The cold is a widespread communicable disease. According to Smiley,[16] the incidence in the U. S. Navy is about 2,500 per thousand per year. He submits evidence[17] that most colds occur in cold susceptible groups. Keefer[6] reports 2-3 colds per year per person among civilians. Greenspan[18] reviewed ten surveys and reported the figures as varying between 277 to almost 4,300 per thousand per year. Coryza is about equally prevalent everywhere. Mild climates seem to have no advantage over the severe ones in this respect.

Reports from institutions, families and isolated communities as well as the experimental studies reported, establish the fact of the contagiousness of colds.[3, 10, 11] Smillie reported outbreaks on St. John's Island, an isolated community in Alabama and in Laborador with a single source of infection in each case. In each he observed and recorded the spread through the community in question.

The spread could be controlled, theoretically by isolation, since the spread is largely by droplet infection. But people will not submit to isolation. For some strange reason, cold sufferers consider themselves as self-sacrificing good sports when they remain at work rather than at home.

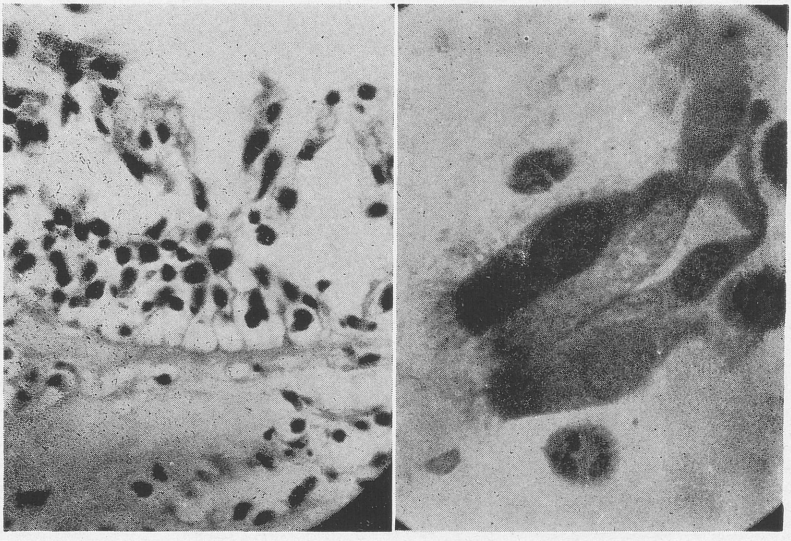

FIG. 3.—Vacuolization of epithelial cells on second day of cold. Some of the loosened cells appear foamy.

FIG. 4.—Group of cells removed from the nasal mucosa by scraping gently on the second day. Two of the cells appear to be secreting mucus. The nucleus of one of these stains poorly as though the cell were necrotic.

There are three peaks in the incidence each year in most communities. They are September-October, January-February, and April-May. It is my impression that the winter peak in our community comes considerably later than January-February. That is the period of our dry, cold weather when we are reasonably free of colds. The incubation period according to Keefer[6] is 24 to 36 hours and he offers evidence that the patient is most infectious during the first day and may be capable of spreading the disease as much as four or five hours before his symptoms begin.

The pathological picture seen in the nose during colds is that of an inflammatory reaction of the mucous membrane. It is swollen, red and covered with profuse discharge. The air passages are frequently occluded by swollen membrane, secretion, or both.

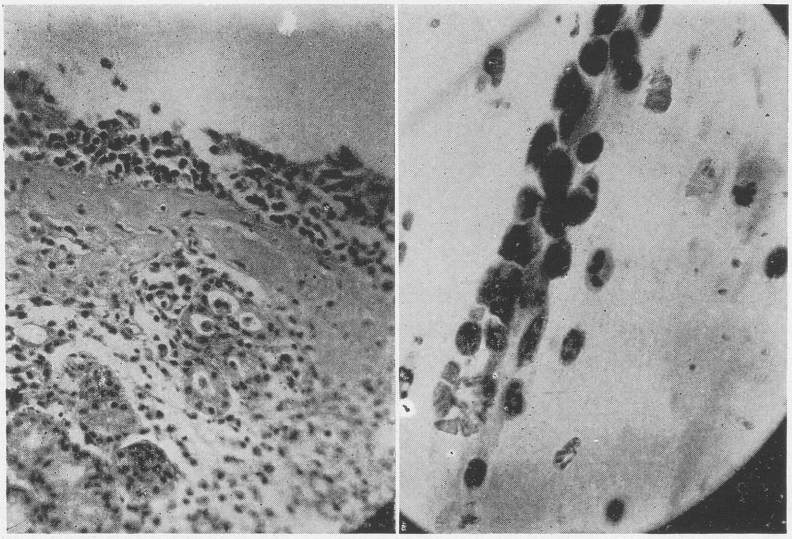

FIG. 5.—Disintegration of the epithelium on the third day. It is possible that this appearance might be artifact.

FIG. 6.—Secretion from the third day of a cold containing many replacement or stellate epithelial cells from deep in the mucosa.

Fifteen years ago, we took 25 specimens for biopsy from the inferior turbinates of cold sufferers during various stages of the disease and about 100 scrapings from the epithelium.[19] From these we attempted to reconstruct the steps of the pathological changes in the mucous membrane. It was not easy to differentiate between changes caused by disease and those caused by crushing action of the instruments which removed the specimens. Judging as carefully as possible, we reported the steps to be about as follows. First, there develops an edema of the submucosa, followed by an infiltration of macrophages, and a few granulocytes. The first change to appear in the epithelium is a separation of the cells as though a fluid formed between them. They loosen and the surface cells begin to slough off. Some become vacuolated or foamy in appearance. A necrotic zone may form next to the surface or single cells or groups of cells may show necrosis. This necrosis may extend deep. Then almost the entire epithelium disintegrates; it looks as though it had been

exploded. Many and sometimes all of the columnar cells are gone by the third day. Some of the stellate cells close to the basement membrane also slough out. During this stage, the nasal secretion may contain many epithelial cells, many of which are surmounted by vigorously active cilia. Sometimes there may be only a single layer of cells left on the basement membrane. The cells that remain proliferate after the most acute stage has passed until they are many layers deep. A syncytium forms at the surface. Finally the surface cells begin to lengthen until they again look like columnar cells. The cellular infiltration of the submucosa is largely composed of variously shaped mononuclear cells with a scattering of polymorphonuclears and some eosinophiles. The edema soon decreases, but the cellular infiltrate increases. By the second or third day, many granulocytes and macrophages are found in the epithelium on their way to the surface. They increase in number as the purulent stage develops.

Repairs seems to take place even while there is still much secretion in the nose. A many layered epithelium full of granulocytes was found on the eighth day, and, on the tenth day, the surface cells showed elongation and on the fourteenth day, definite columnar form.

No bacteria were ever found in the epithelium or submucosa, only in the secretion. Neither were any inclusion bodies ever found.

That is the way it looked 15 years ago. I still believe that this picture is essentially correct. The changes in the submucosa were definite and there was not much chance for error. There is no doubt that the surface cells slough out of the epithelium because large numbers of living ciliated cells are found floating in the secretion and on the second and third days going down as it were with all cilia firing. I have watched ciliated cells under the microscope shake themselves loose from the epithelium especially under stimulation of a slightly alkaline pH and actually have a movie of one such cell. Cells from deep in the epithelium are also found in large numbers in the secretion.

There is still the possibility, however, that the steps which we observed in the biopsy specimens were produced or at least modified

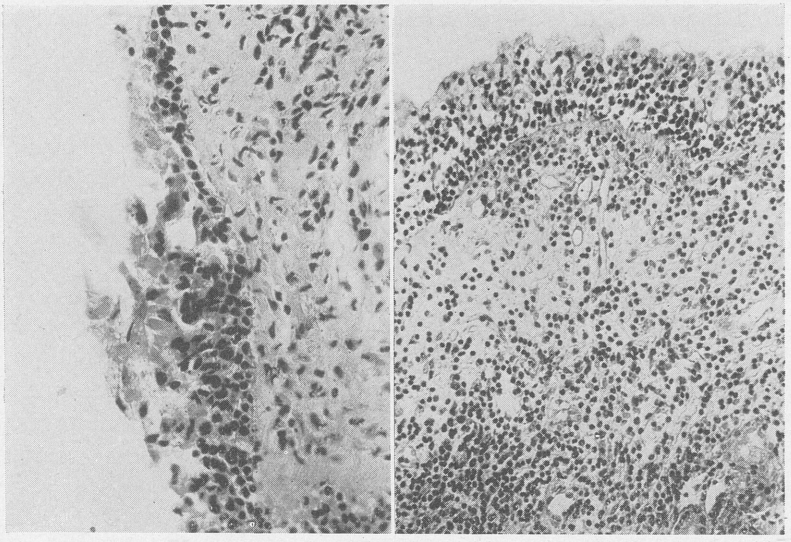

FIG. 7.—Specimen removed on fifth day. In one small spot only one layer of cells remains upon the basement membrane. Here also the question of mechanical damage must be considered.

FIG. 8.—Epithelium on the tenth day of a cold—stage of convalescence. The epithelium appears to be regenerating.

by the crushing action of the instrument used for excision. It is so easy to knock off the cells and so difficult to avoid doing so. We tried to confine our study to areas which had been protected by folds, or which were overlaid by the secretion or necrotic cells that looked as though they were produced where they lay. Mechanical damage was excluded as far as possible.

Those areas which showed newly developing columnar cells are open to doubt. When the surface cells have been lost and the deeper cells grow out, elongate and become ciliated, they must of necessity pass through such a transitional stage. Likewise, however, there is in the normal epithelium a transitional zone between the stratified areas in the fore part of the nasal cavity and the ciliated columnar further back. These zones are inconstant in position. It is possible

that the biopsy of the specimen taken on the fourteenth day of a cold which showed what we took to be newly developing cells, might have been taken from a transitional zone. Perhaps that area had always looked the way we found it to be.

There is doubt also as to what constitutes necrosis from the action of toxins in cells. Cells which have just secreted or are about to secrete mucus look necrotic. Physiologists have told me that it is not known if a secreting epithelial cell survives to secrete again.

Since this study was made 15 years ago, no one seems to have done anything similar to it. It should be repeated by someone else. Shope found very similar changes in the bronchial epithelium of swine afflicted with swine influenza. He described fragmentation of the epithelium and loss of surface cells and published photographs which were strikingly similar to the appearances in these specimens just described.

The outstanding clinical feature of a cold is the profuse, thin secretion. The physical differences between this and the normal secretion are obvious. The chemical differences have not been well worked out.

I made some attempts to study the lysozyme content about ten years ago,[20] and in seven cases studied found that the lytic power of the secretion was reduced at the onset of symptoms (and in one case several hours before) and increased again just before improvement began. It seemed quite definite that this was not a dilution phenomenon because the lytic power was found low before there was much of any secretion and high when improvement had begun even in the presence of copious secretion. This study was simply a preliminary one on a small group. Cahn-Bronner[21] reported similar studies in 1942. He corroborated our findings and went further. He found that the lytic power was not reduced in the copious secretion of hay fever and was therefore convinced that the change in colds is not a dilution phenomenon.

The pH has been tested by a number of observers. Buhrmester[22] found the average pH increased very slightly during colds, that is, the secretion was more alkaline. Fabricant[23] found a very consider-

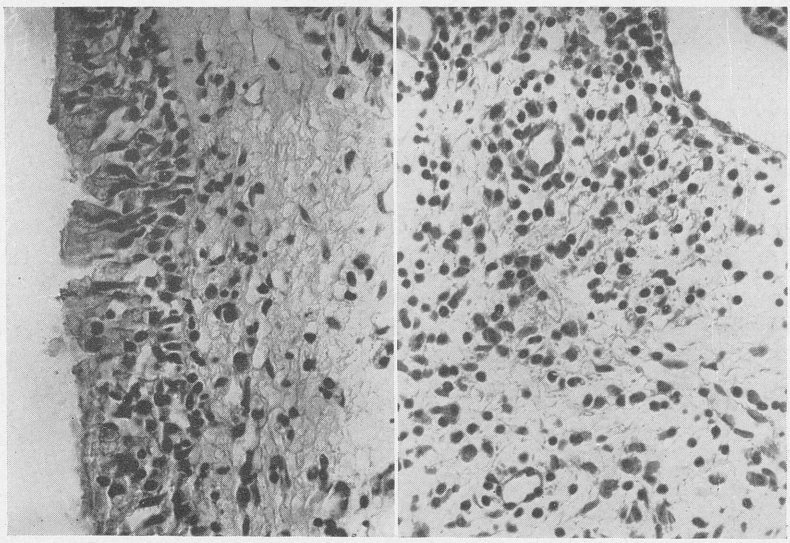

FIG. 9.—Epithelium on the fourteenth day. Imperfect columnar cells are present. They might be in the process of regeneration. On the other hand, such imperfect cells are found in normal epithelium in the zone of transition from squamous to ciliated cells in the fore part of the nose.

FIG. 10.—Cellular infiltration on the third day.

able increase and felt that it was of considerable significance. In my tests,[19] it was increased slightly on an average during the acute stage of watery discharge and slightly reduced during the purulent stage. Mittermaier[24] found it reduced during the purulent stage. The slight rise during the acute stage in our series seemed to be too small to be of much significance. My readings were all higher than those taken in situ, probably because of the escape of CO_2. The normal mucus has a range very close to neutrality—7.0.[25] The low readings found by Fabricant[23] have not been corroborated and were apparently due to a fault in technic as pointed out by Nungester.

I have recently made some tests to determine if an increase in pH (alkalinity) is in itself irritating or destructive in the nose as suggested by Fabricant. The material consisted of a series of bottles

containing physiologic saline each with a different pH. The range used was from 6.8 up to 8.4. This saline was dropped into the noses of a number of volunteers in comparatively large quantities (half a dropper full or more) at frequent intervals. Up to pH 8.4 it was found to be so bland as to be practically without sensation. There was no increase of secretion or any other change noted.

Apparently there have been no studies on the pathologic changes in distant parts of the body. The symptoms would indicate that there must be some. For instance, the delayed reaction of the nasal temperature to chilling of the back which was reported by Spiesman;[15] the extreme sensitiveness to any draft experienced by the patient; the sweating and polyuria, which frequently occur, and the salivation, which sometimes occurs, would seem to indicate some disturbance of the sympathetic-autonomic balance. Spiesman presents some evidence indicating that emotions have a bearing.

There has been a very impressive amount of effort expended upon preventing colds. The results have been disappointing.

Vaccination by bacterial vaccines has been given very thorough trial by the subcutaneous and oral routes and even locally in the mucous membrane. Reports have been both favorable,[26-29] and unfavorable.[30-34] One author who presents strikingly good results from subcutaneous vaccination writes that, "No control series was used, as the primary purpose of the inoculations was to do something for the students and not to test the vaccine. It did not seem fair to exclude students wishing vaccination simply to provide a control group for statistics." Another author, using the same method, reduced the incidence of colds in a group of students equally successfully—in fact by 55 per cent.[31] However, he vaccinated a control group with normal saline and obtained even more favorable results in this control group, namely 61 per cent reduction.

Vaccination by bacterial vaccines both by mouth and subcutaneously appear at this time to be valueless for reducing either the incidence or severity of colds.

Vaccination by nasal spray[29] has a scientific basis in the fact that immunity may be local. Walsh has demonstrated that the antibody

titer of the mucosa can be raised as compared with that of the blood and other tissues by means of local application of bacterial vaccines. His results look promising as far as complicating bacteria are concerned, but are not aimed at the virus itself. This work should be pursued further and corroborated by others.

Unfortunately, both the cold virus and the group of pathogenic organisms usually found in the nasopharyngeal flora are very weak producers of antigens. One would scarcely expect any notable results from vaccination. The immunity following an attack of coryza lasts only three or four weeks. Likewise, infections by staphylococci and streptococci in general produce no lasting immunity in patients who recover.

These studies in large groups of students or industrial workers are severely handicapped by the human element and must be controlled with exceedingly great care. This fact is demonstrated by the control groups which were so strikingly benefited by injections of saline. Students who come requesting vaccination, even before the study is begun, are already a selected group in whom the results are probably heavily weighed by the will to believe.

Dochez[3] tried vaccination with heat killed virus by hypodermic injection. The results seemed promising in apes but were negative in man. He suggests that the reason might lie in the supposition that immunity is not general, but may be local in the nose. This virus vaccine should be tried in a nasal spray. Unlike the cold virus, the influenza virus does produce an antigen and reports on results from vaccination appear to be favorable.[35]

Vitamins have been tried in large groups of patients and have been found to be without value in preventing colds.[36] Crampton[37] reports favorably upon the use of vitamins but offers no control series.

The sulphonamides have been tried and most reports indicate negative results as far as the cold itself is concerned. On the other hand, they have very definite value in the prevention[8, 38] and treatment of complications. That does not mean that they should be used routinely.

McCord[39] and McConnell[40] both made studies of the relation of air conditioning (as it exists now in office buildings) and the incidence of coryza. Each recorded the number of colds among employees working in an air-conditioned building as compared with a similar group working in an old building in which there was no air conditioning. In both studies, the incidence was the same in both groups. Both of these studies are open to question, because the employees remained under the study conditions only eight hours in each twenty-four.

There is another type of air conditioning which is aimed at destruction of bacteria and viruses floating in the atmosphere which seems to be very effective in stopping the spread of infection. The purification can be accomplished either by aerosols,[41] antiseptic chemicals, or by ultra-violet light.[42] The latter seems to be very promising. It can be used either by subjecting the whole room to ultra-violet rays, or just the source of air, or it can be used in the form of barriers of light. Wells states that the foundations for efficiently purifying air and checking its purity are as well established as the control of the purity of the supply of drinking water. He believes that such control and purification of air supply is necessary to free us from air-borne infection. He likens the contamination of air supply by the nasopharynx to the contamination of water supply by the excreta from the intestinal tract. Perhaps in this we have an effective method of preventing colds in schools and factories.

The hardening process has long been advocated as a cold preventative; that is, cold baths, outdoor exercise and calisthenics. It may have value, but has not been subjected to enough well controlled studies to have established a value.[18] Outdoor individuals do have fewer colds, but they are also subjected to fewer contacts with infection.

In general, treatment has been as disappointing as prevention and it is just as difficult to determine the value of the suggested remedies. Colds are self-limited unless complicated. Some are abortive and are over in a few hours. Some last only 24 to 48 hours. And others, when complicated, run a course covering several weeks. When symptoms disappear after the exhibition of some remedy, it is di fi-

cult to know if the remedy exerted some beneficial influence, or if the attack was about to subside anyway.

Codeine and papaverine[43] seem to have some definite value in shortening the course. Under some circumstances this remedy should doubtless be used.

Steaming often gives symptomatic relief.

If serious complications threaten, then the sulfonamides are indicated.

Isolation should be practiced whenever feasible. It not only prevents spread of the cold virus, but protects the sufferer from bacterial infection of his damaged epithelium by droplets from other individuals. It seems to me that this is very important. If bacterial infection can be prevented, the attack will soon be over because the virus stage is so short.

Of all of the many nasal drops which have been advocated, simple ephedrine in saline solution seems to give about as much relief from nasal obstruction as anything else. This is of course purely symptomatic treatment, and I fear is not resting upon a firm scientific basis. Why do we want to open the nasal passages when nature has closed them? Perhaps the closure is purposeful. In my practice, I have done the opposite. As soon as there is any sign or symptom of an approaching cold, I have instructed my patients to block the nostril (or nostrils) with clean cotton, tightly enough to reduce materially the inflow of air. The cotton must be changed frequently. It seems to me that the irritation is greatly reduced by this simple measure and often the swelling does not become great enough to obstruct the air passage.

Much treatment has been directed toward the cilia, generally with a view toward stimulation. But do we know if the cilia need stimulation during a cold? Perhaps they are working at capacity as it is. In some cases, they are largely destroyed. Under such circumstances stimulation would seem to be irrational.

If stimulation is desired, it can be had by means of a mildly alkaline solution (8.0—8.5) which contains magnesium and calcium salts.[44] At pH 8.5 in Tyrode's solution, the cilia will continue active

for 24 hours. Acids, on the other hand, paralyze ciliary action. It ceases entirely if the pH drops to 6.4 or less. If magnesium or calcium ions are omitted, the tissue breaks up and action ceases. The sodium ion according to Negus is the strongest inhibitor of ciliary action.

Conclusions

These facts about the cold seem to be fairly well established. It is a widespread, contagious epidemic disease which occurs in all climates, especially in the autumn and spring months. It is spread by droplet infection and can be controlled by isolation like other communicable diseases. It is caused by a specific virus and is frequently followed immediately by complications due to bacterial infection. These are purulent rhinitis, otitis media, laryngitis, bronchitis. Chilling and exposure are definite etiologic factors probably because of prolonged reduced temperature in the nasal mucosa. Immunity following attacks is very brief both as far as the virus and the complicating bacteria are concerned. Vaccination is unsuccessful, apparently because both the virus and the bacteria have such poor antigenic properties.

Prevention by the giving of vitamins, or by so-called "hardening processes" are also largely unsuccessful.

The local pathology centers about injury to the epithelium, presumably by direct action of the toxins on the epithelial cells, and consists in destruction of the superficial cells. There is a marked submucosal cellular reaction. The secretion increases tremendously in volume and contains a lower percentage of mucin. There is at least some indication that the lysozyme content decreases even before the volume increases. The change in pH is small, if any, and tends toward the alkaline side.

Systemic pathological changes are largely unknown.

Treatment is not very successful and consists principally in symptomatic measures and attempts to prevent complications.

324 West Superior Street.

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DISCUSSION

Dr. George B. Wood: I want to congratulate the Association for this very excellent paper presented by Dr. Hilding.

There should be no more doubt in our minds after listening to this presentation that the initial etiologic factor in the production of colds is a virus infection. However, we must recognize that the virus infection itself is usually of minor importance. However, it produces the necessary insult to the mucosa that permits the invasion of the pyogenic organisms and it is this secondary infection that leads to serious complications. If the ordinary vaccines, autogenous or stock, have any effect it can only be in relation to the secondary pyogenic infection. Personally, I have little faith in either the therapeutic or prophylactic use of vaccines as far as colds are concerned.

The insult to the mucosa necessary for the successful inoculation of pyogenic organisms can be brought about not only by virus infections but also by mechanical and traumatic means. This has been amply demonstrated by animal experimentation. Years ago I found that it was almost impossible to infect the frontal sinus of dogs by simple inoculation with pyrogenic organisms. If, however, a drop of croton oil was put in the sinus 24 hours before inoculation, infection invariably followed. There has been other experimental work along the same lines so that I feel that the pyogenic element of the upper respiratory infection is not due so much to a synergetic

relation between the virus and the bacteria as it is to the reduction of the local vital resistance of the tissue by the virus.

I want to say a word concerning the technique of making cultures from the nasal fossae. In 1909 Cobb read a paper before this Association in which he showed that if the technique of taking cultures avoided contamination from the anterior inch and a half of the nasal fossa, the majority of cultures thus made from the normal individual were sterile. He used an inverted rubber sleeve to convey his applicator into the nasal fossa thus avoiding nostril contamination. The same thing can be more simply and readily accomplished by the use of a small glass tube, the ends of which have been drawn out to not quite capillary size. After sterilization, with suction apparatus attached to one end, the other end can be passed by direct vision to the region of the sinus ostium or other area in the nasal fossa from which it is desired to make the culture. After the secretion has been sucked into the tube the suction apparatus is removed and the ends of the glass tube sealed with a Bunsen burner and the tube sent to the laboratory for investigation. By this method I have been surprised at the number of sterile cultures that I have gotten from nasal fossae.

Dr. H. Marshall Taylor: Doctor Hilding has referred to the experimental work done by Mudd, Grant and Goldman, who reported that chilling of the body surfaces causes a fall in the temperature of the nasal and pharyngeal mucous membrane, and that chilling of the nasal mucous membrane and change in vasomotor tone may so disturb the equilibrium between the host and the bacteria of the nose as to favor infection.

This experimentation was carried on when the patient was at rest. Could Doctor Hilding tell us were the individual taking exercise, with increased respiration, would this lowering of the temperature of the nasal mucous membrane occur? The individual does not lose body temperature in a cold medium when taking exercise rapidly as he does when his body is at rest.

Dr. S. Salinger: There are two items which I think probably will appear in the paper when it is published and should be mentioned at this time. One is the effect of drying of the mucous mem-

brane, on the ciliary activity, as a predisposing factor to colds, a fact which Dr. Proetz has written about some years ago. The other is the native constitution of the individual. Whether he is of the sympathetic or parasympathetic type has a bearing on his vasomotor reactions to changes in temperature and consequently on his susceptibility to infection.

These are two factors which in my experience have much to do with the incidence of respiratory infections and I wonder if Dr. Hilding has anything to say in this connection.

Dr. Arthur W. Proetz: I should like to ask Dr. Hilding if he knows whether the cold-susceptible fraction of the subjects, that is, those susceptible to chilling are by any chance in the hypothyroid group.

Dr. A. C. Hilding (closing): Dr. Wood's remarks concerning the virus, recall one of its important characteristics which was brought out by Paul and Frees in their study at Spitzbergen. The virus is comparatively short lived in a given host. It must be transferred from host to host rather frequently, or it will die out in a community. The population at Spitzbergen is rather small and is isolated from the rest of the world for seven months of the year. When the first boat arrives in the spring, an epidemic begins and within a month ninety per cent of the people have had colds. Another epidemic begins late in the summer and then gradually dies away in the autumn after the shipping season has closed. There are practically no colds in the community all winter.

These workers interpreted these facts about as follows: The virus is short lived and the immunity of short duration. No one is immune when the first boat arrives. Those who are first attacked recover and have time to lose their immunity before the shipping season is over. Some of these get a second attack. However, when the last boat sails away in the fall, practically everyone has had a cold so recently that everyone is immune. Therefore the virus dies out and there are no colds through the winter.

In a community such as New York, however, there are enough susceptible people at all times to harbor and transfer the virus.

I do not know about the relation of chilling and exercise. It is conceivable that a cold-susceptible individual might prevent the lowering of the intranasal temperature by exercise.

Drying is undoubtedly a predisposing factor if it is sufficient to dry the film of secretion. Under such circumstances, ciliary action ceases and you have conditions favoring infection.

It would be highly desirable if someone would make a study on the sympathetic-parasympathetic balance during colds. There seems to be very little known about this subject.

I cannot answer Dr. Proetz's question concerning hypothyroidism in cold susceptible groups. There was no reference to this condition in any of the articles reviewed.

Thank you again so much.

STEREOSCOPIC VERSUS PLAIN FILMS IN ACCESSORY SINUS EXAMINATIONS

FREDERICK M. LAW, M.D.

NEW YORK, N. Y.

Roentgenography of the nasal accessory sinuses is the most difficult of the many branches, and requires the most painstaking care in the technique. A poorly made set of films is worse than useless. There must be a sufficient number of films made in various angles, and it is astonishing how many times I have films referred for interpretation and find there are only two films. Such a set is of no value whatever.

There are three methods for this examination: (1) plain or flat films; (2) stereoscopic films; (3) sectional or planigraphic films.

All roentgenologists can make plain or stereoscopic films but only a few have access to the equipment needed for planigraphic films. The most generally satisfactory examination is with stereoscopic films. Very few rhinolaryngologists have their own stereoscope, but all hospitals and roentgenologists have the instrument. In case none is convenient, it is possible to so train the eyes that stereoscopic vision can be obtained without the use of any extra equipment. It is very simple and, once it is understood, requires little practice to obtain a good third dimensional impression.

Of course, there are certain individuals who cannot relax and fuse the two images, even with the best of mechanical apparatus. In this case he still has the conventional plain film to look at, but the stereoscopic pair is available, if necessary.

The planigraph is a wonderful achievement and in a few cases is valuable in sinus work, but it requires special training in the technique and the interpretation. It is indispensable in the examination of the larynx in the posterior anterior position as it does remove the image of the spine and shows the extent of involvement of the soft tissues of the larynx.

Why are not more stereoscopic examinations made? There are several reasons: the additional expense to the roentgenologist; his inability to collect a larger fee to compensate; the lack of training of the technician; the roentgenologist's inability to fuse the images and appreciate the added value of the third dimension on a film.

When you examine a disarticulated skull you see depth, so why not see depth in the x-ray film? It is a great help and once you realize the added information you will insist upon stereoscopic films for all your cases. It is easier to visualize pathology when the film depicts the condition as it would appear if you were looking into the sinus rather than at it.

When will stereoscopic films give added information?

1. In all cases of fracture of the skull.

2. In all cases of fracture of the nasal bones. They enable one to see which side is fractured and how much displacement exists.

3. In cases in which the film taken in the Waters position shows an apparent tumor in the antrum. A stereoscopic film may show this tumor to be due to the image of the cheek or upper lip superimposed on the antrum.

4. In cases in which the film taken in the Waters position is negative, while the film taken in the 23° angle position is positive. A stereoscopic film may show this opacity to be due to an increased density of the floor of the posterior cranial fossa, which will be placed posterior to the antrum.

5. In cases in which it is difficult to determine whether the right or left ethmoid shows a change in the appearance of the cell structure as viewed on the lateral film.

6. In cases in which it is difficult to determine on which side a large agger cell exists.

7. In all cases of enlargement of the sella turcica.

8. In all cases of bony involvement of the skull.

9. In cases in which there is a suspicion of absence of a frontal sinus or a four plus involvement of a frontal as indicated by a faint

circular line of density where the superior border of the frontal should be. A stereoscopic film may show this line to be part of the occipital suture superimposed on the frontal region, and not the superior border of the frontal, thus proving the absence of a frontal sinus.

10. In cases in which the lateral film shows a suspicion of a partition dividing the antrum into two sections. A stereoscopic film will prove this apparent partition to be the posterior border of the malar bone.

11. In all cases for examination of the petrous pyramid.

12. In all cases for examination for salivary calculus.

13. In cases in which an apparent soft tissue tumor is shown on lateral films of the pharynx. A stereoscopic film may show this to be caused by the lobe of the ear and to be entirely outside the skull, not in the pharynx.

There is one precaution to be observed when examining each one of a stereoscopic pair of posterior anterior views as separate plain films. The necessary shift in the position of the tube may cause one antrum to be more opaque than the other. This opacity may be manifest in both views but on opposite sides. The stereoscope will fuse them both and give you a true image. If you do not use a stereoscope, then consider the condition of both antra to be as registered by the lesser opacity. The increased density is caused by the angulation of the tube and shifts as the tube is modified to one side or the other. Of course, if one or both antra are badly involved, this precaution does not follow.

14. In cases of malignancy involving the antra. The stereoscopic film enables one to determine more accurately the degree of bony involvement.

From the above, it is evident that stereoscopic views are not absolutely essential in all cases but the roentgenologist does not know the clinical evidence and it is embarrasing to have to request a second examination to clear up some disputed point.

Many of you send your patient to the roentgenologist with a request for a sinus examination without giving any information about

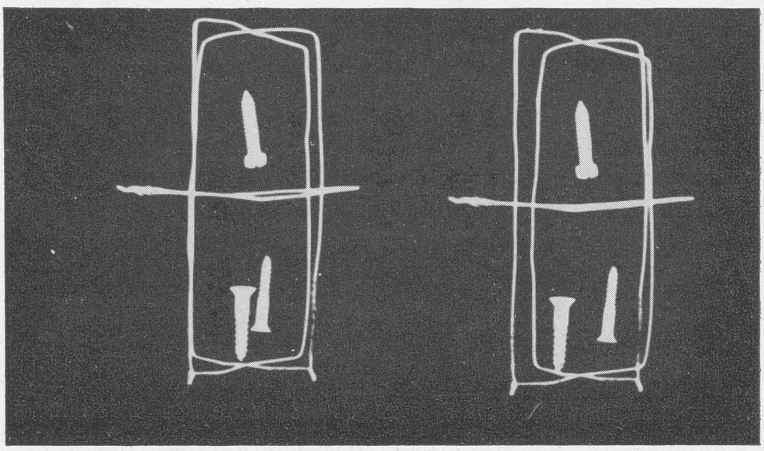

FIG. 1.

the case. Some patients object to questioning by the roentgenologist and thus he may be handicapped by lack of information. A brief note, or phone call, would greatly facilitate the technique and aid in the interpretation.

Perhaps you are saying I am bigoted and have a special desire for stereoscopic films but I find them of great assistance, and am sure you would gain more information if you could succeed in the technique of obtaining stereoscopic vision without the use of expensive and bulky apparatus.

I have given you the reasons for wishing stereoscopic films; now I will try to demonstrate to you the method of obtaining third dimensional impression. Fig. 1 shows a pair of stereoscopic films of a card board box, bound with wire, and suspended within are three screws at different levels. The idea is for you to converge your eyes so that there will be crossed vision. You should see the left hand image with the right eye and vice versa. If you were viewing two films in the illuminating box, one alongside the other, you should step back three or four feet and follow my directions.

Place the open hands against the side of the orbits, folding the thumb in. Keep the hands straight, like blinders on a horse. Close the left eye and swing the right hand over like a gate, so that the image of the right hand film is obscured. Keep the hands there. Open the left eye and close the right eye. Swing the left hand over until the image of the left hand film is obscured. Keep both hands steady. Now open both eyes. If you see two images try to focus on one or the other, try to pull them together. You should see everything in the third dimension, the wires forming a square and the screws suspended with the square. If unsuccessful, start all over again. You must relax and converge.

After you have mastered the method you can dispense with your hands and converge the eyes at will and see stereoscopially. Thus you could have a pair of stereoscopic films in the operating room and simply look up at them and see the part you are operating upon with the same sensation of depth as the actual structure on the table.

The best films to practice on are chest films and those of an empty skull. Many of you may never have seen a stereoscopic image and not understand what it means. If you have available an old-fashioned parlor hand stereoscope and some pictures, you will understand what that impression of a third dimension really means.

140 East 54th Street.

VENTRICULAR LARYNGOCELE

H. MARSHALL TAYLOR, M.D.

JACKSONVILLE, FLA.

It was in 300 A. D. that Galen[1] first made mention of the laryngeal ventricle. Recognition of this recess was credited to him by Morgagni,[2] who gave an incomplete description of it in 1741 and for whom it is named. Mackenzie[3] advocated calling it the ventricle of Galen. Eighty-eight years after Morgagni's account was published, Larrey, the French army surgeon, first mentioned the air sac which at times develops from the saccus of this ventricle. The term laryngocele, now commonly applied to this anomaly, was introduced in 1867 by Virchow,[4] who described the sac in these words:

"I have found a dilatation of the ventricle of Morgagni, up to the present unknown as it seems, which one may call 'ventricular laryngocele.' One sees, in these cases, small elongated sacs with thin walls, coming off from the upper part of the ventricle by a somewhat narrow orifice, and sometimes to the upper border of the thyroid cartilage, and even to the hyoid bone, where they terminate by an end in the form of a club; most frequently they are found only on one side; once, however, I have met them on both sides. Their walls are smooth, their interior is lined with ciliated epithelium, and they are filled with air."

Confusion in nomenclature makes questionable the number of cases of laryngocele reported up to the present time, but at best it is small. Hipple,[5] in 1910, reviewed the world literature on the subject of pathologic distension of the appendix of the ventricle into an air-containing cyst and found only 20 cases reported. In but 12 of these cases was the condition obeserved in life. Three years later, Hansberg[6] stated that 24 cases had been published up to that time. In 1921, Iglauer[7] reported a case. Moore,[8] whose comprehensive article was published in 1922, has been misquoted in regard to collecting 85 cases of laryngocele in the literature. The confusion arose because his article was on the complete subject of "The So-Called Prolapse of the Laryngeal Ventricle and Eversion of the Sacculus" and

was not confined to a discussion of laryngoceles. He merely cited 10 cases as examples of ventricular laryngocele as described by Virchow.[4]

In the recent American literature, Jackson and Jackson[9] in their textbook published in 1937 mentioned two cases. Other authors reporting cases are Freedman,[10] two cases and Simpson,[11] one case in 1938; Lindsay,[12] one case in 1940; Allman and Cordray,[13] two cases in 1942; Lothrop,[14] one case in 1943; and Schall,[15] one case in 1944, In Schall's case the laryngocele was associated with carcinoma of the larynx; the author believed it to be the first case of the kind report ed in the literature. It is of interest that during the 66 years of the existence of the American Laryngological Association there appears but one report of a case of laryngocele in the Transactions of that senior laryngological society; in 1914, Shambaugh[16] presented a case in a paper entitled "Laryngocele Ventricularis."

The proper clinical conception of laryngocele in man is dependent in large measure upon adequate understanding of the evolution and physiologic aspects of the ventricle of Morgagni. This portion of the larynx is a horizontal space, elliptical in shape. It is situated between the true and the false vocal cords. Anteriorly from the roof there arises a vertical pouch known anatomically as the sacculus. Moore,[8] citing Rudinger,[17] stated that the ventricles are relatively much larger in the male and added that occasionally they are greatly overdeveloped.

There is a close resemblance between the larynx of mammals and that of reptiles and birds. One striking difference, however, is that the ventricle is observed only in mammalian life. The human larynx was not originally designed to serve as an organ of speech, but instead was primarily intended as a valve for guarding the pulmonary system at its entrance by exercising the primitive function of keeping everything out of the lungs but air. Negus[18] concluded that air sacs are not developed for purposes of voice and noted that many animals without them are as noisy or more so than those with them. Among the examples he cited are the roar of the lion, which has no air sacs, and the seldom heard voice of the Anubis baboon with its enormous air cavities; also, the howler monkey with thin

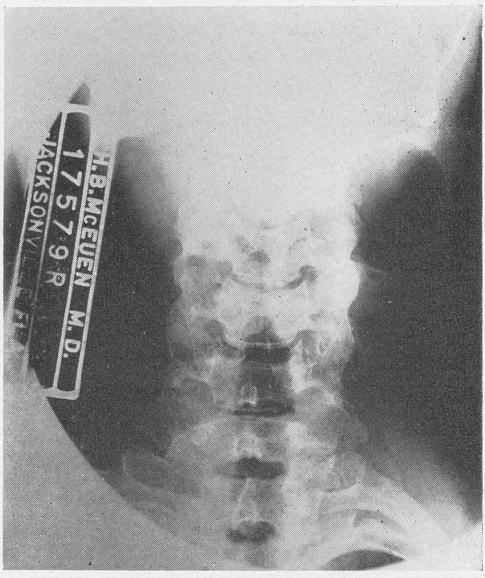

FIG. 1.—Anterior posterior view of air sac.

osseous air sacs is especially noisy, and the lemur without these sacs is likwise extremely noisy.

As viewed by this author, the structure of the larynx in the different mammals differs in accordance with the relative quantity of oxygen needed and of carbon dioxide given off by the lungs. This requirement is influenced by the mode of life. In anthropoid apes, for example, temporary cessation of respiration is involved in the fixation of the bony structure of the chest required to give the pectoral muscles full play as the forelimbs grasp, hug, strike and climb. Compensation for the interruption of the vital function of normal respiration was attained by the evolution of air sacs arising from the ventricle of the larynx. Filled with oxygen, and during expiration with carbon dioxide, they serve as mixing chambers and are used with the least amount of exertion by the lungs.[12]

It would appear that the sacculus, which is a constant finding in man, is a degenerated relic of air sacs leading out of the ventricle,

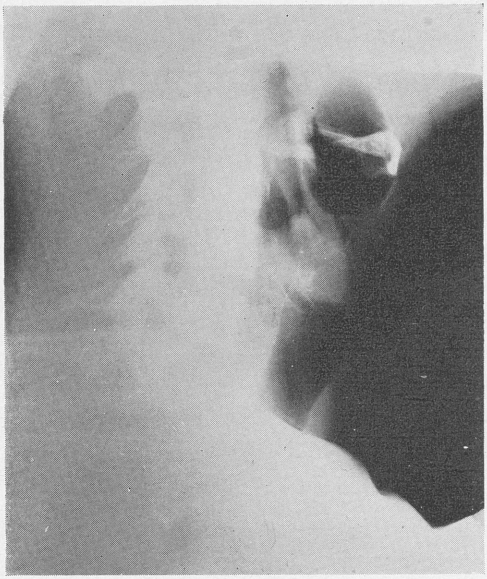

FIG. 2.—Lateral view of air sac.

or perhaps their incomplete precursors, with a function directed to rebreathing air.[12,-18] It contains lymphoid tissue and mucous glands, which without doubt help to supply the continuous flow of mucus required to keep the borders of the vocal cords lubricated during phonation. This recess varies in size from a simple depression near the anterior end of the ventricle to a narrow prolongation extending up to the level of the thyroid cartilage and, rarely, even higher. Comparison of the human sacculus with the excessively large air sac of the chimpanzee or the orangutan, as Negus[18] noted, clearly indicates evolutionary developmental changes degenerative in character. It is from this appendix that the ventricular laryngocele develops.

Lindsay[12] described three types of laryngocele. The internal laryngocele, commonly observed, is a cystic dilatation in which the hernial protrusion of the mucous membrane lies within the larynx. The superior external laryngocele, occurring less frequently, appears as a swelling in the neck after the sac has perforated and projects as a hernia through the thyrohyoid membrane. Also, the internal and

external laryngocele may occur in combination, joined by a small isthmus. In this mixed type the extralaryngeal portion is frequently large and has usually protruded through the thyrohyoid membrane.

A preexisting pathologic condition is not necessary to the development of a laryngocele for the condition is observed in the normal larynx. Etiologic factors mentioned in the literature include coughing, whooping cough, singing, horn blowing, glass blowing, weight lifting, vomiting, straining at stool and childbirth. The known occurrence of a congenitally long appendix of the ventricle may also be of etiologic significance.[12] In all cases there is present an abnormally increased intraglottic pressure, to which the ventricles and, in turn, their appendices are exposed. Too, it is probable that tumors, syphilis, tuberculosis and other diseases affecting the larynx may so weaken the walls of this organ that a laryngocele may arise, especially in the presence of other etiologic factors.

If the mucosal hernia is small, there are no symptoms. The size of the air sac may, however, increase to the extent that it causes embarrassed respiration and even dyspnea, and it may also affect the normal fuction of the vocal mechanism. The internal laryngocele, which sometimes extends to the aryepiglottic fold or even to the base of the tongue, dilates on forced expiration and, if large, may produce some obstruction accompanied by hoarseness.

When the sac has perforated through the thyrohyoid membrane and appears as a swelling in the neck, coughing, crying, physical exertion with the upper extremities, or straining at stool may cause the superior external laryngocele to become enlarged. It can be emptied by pressure, and the sound of escaping air, or air and fluid if infection is present, may be heard. Local discomfort and headache may accompany the distension if there is pressure on the large veins of the neck. When a laryngocele becomes infected and a pyocele develops, the patient on coughing may expectorate pus.

An external swelling of the neck with convexity of the ventricular band, some decrease of this mass on pressure accompanied by a hissing sound, and the presence of a tympanitic note on percussion should establish a clinical diagnosis, which may readily be confirmed by roentgenograms showing a sac filled with air. In the earlier

cases, when a laryngocele was suspected the introduction of an aspirating needle to determine the contents of the tumor-like swelling was a diagnostic measure. This procedure which might introduce infection, became unnecessary with the advent of the roentgenogram. Roentgen studies now make easier the diagnosis of a laryngocele and offer but another evidence of the fact that no laryngeal examination is complete without a roentgen study.

Report of a Case

J. C. F., a white man aged 35, was referred to me with a tentative diagnosis of carcinoma of the larynx. He could not recall when he had not had a recurring hoarseness and a swelling on the left side of the neck. He related that since childhood he had been fond of singing and possessed a voice of considerable volume. Oftentimes, on singing, he had noticed that the swelling would increase in size; also, the forced expiration associated with muscular exertion, such as the lifting of a weight and particularly straining at stool, would cause the mass to become larger. During the two years immediately preceding the examination, he had noticed enlargement of the swelling, increased hoarseness and occasionally embarrassment on forced inspiration. At times, on pressure over the mass he had observed that quick reduction in its size was accompanied by a squeaking or hissing sound.

Local examination disclosed on the left side of the neck the presence of a tumor-like swelling about the size of a tangerine, which extended above the thyroid cartilage and increased in size on coughing. The patient voluntarily percussed this external mass, which gave forth a tympanitic sound. Indirect laryngoscopy showed a swelling involving the left ventricular band, which at its greatest convexity projected beyond the median line of the glottis occluding entirely the view of the left vocal cord. A roentgen study established the diagnosis of ventricular laryngocele.

On July 10, 1942, with the cooperation of Dr. Kenneth Morris, the tumor was successfully removed without rupture. An incision was made on the left side of the neck at the level of the thyroid cartilage, extending from the middle of the sternomastoid muscle forward to about an inch from the midline. When the platysma

muscle was cut and the superficial fascia opened, a thin-walled sac protruded between the sternohyoid muscle medially and the sternomastoid muscle posteriorly. It was in rather intimate contact with the submaxillary and lingual vessels above and the carotid vessels laterally. The sac was dissected free down to the pharynx where the neck could be seen coming from the posterior lateral aspect of the pharynx just above the thyroid cartilage. The neck of the sac was doubly ligated with crochet cotton and cut between the ligatures. The sac was removed intact. The stump was enfolded with several cotton sutures and muscle tissue, and fascia was used to cover the stump. A small rubber dam drain was inserted to the deepest portion of the wound, and a small amount of sulfanilamide was sprinkled around the stump of the sac. The wound was closed around the dam with interrupted cotton sutures.

The pathologic report, made by Dr. L. Y. Dyrenforth, follows:

"*Gross*: This mass is about 5 cm. in diameter and is open at one side by a small neck of wrinkled and edematous tissue, giving it somewhat the appearance of the closed part of a toy balloon. The wall is uniformly about 2 mm. in thickness, but at the opening the tissues are edematous and consequently thicker than this. The interior is fairly smooth, but near the opening there is distinct rugation, which becomes more pronounced as the aperture is approached. The interior surface is translucent gray, with a distinctly mucosal aspect. The external surface is slightly ragged. A quantity of limpid serous fluid is present in the unopened specimen, which does not completely submerge in a beaker of ordinary 10 per cent formalin.

"*Microscopic*: The wall of this sac is characterized by a uniform consistent inflammatory reaction. This reaction manifests itself by clumps of round cells and other mononuclears of inflammatory origin. These occur mostly in multiple foci and are sprinkled through the adventitia. There are considerable numbers of well developed lymphoid follicles also in the loose tissue beneath the mucous membrane. These contain well developed follicle formations and germinal centers. Capillary blood vessels are abundant. There are definite areas of granulation tissue near the surface. The mucous mem-

brane is characterized by a high columnar epithelium suggestive of ciliated variety. There is an abundant inflammatory reaction beneath this. The deeper layers of the stroma contain muscle fibers and loose fatty tissue.

"*Diagnosis*: Laryngeal cyst showing inflammatory changes in the wall."

The patient had an uneventful recovery. The drain was removed on the third day, and the sutures were removed on the seventh day. Healing was completed at the end of two weeks. The wound was examined again after three months had elapsed; it was well healed with no evidence of the laryngocele. It was noted at this time that the patient's voice which had previously been hoarse, was now practically normal in tone. Approximately two years later, on May 22, 1944, the patient was again examined. There was no evidence of recurrence of the cyst. Both the larynx and the voice had returned to normal.

Summary

A survey of the subject of laryngocele in man including its phylogenetic aspects is presented.

One case is reported, in which the air sac was successfully removed intact. After approximately two years there was no evidence of recurrence.

111 West Adams Street.

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DISCUSSION

DR. CHEVALIER L. JACKSON: I have listened to Dr. Taylor's presentation with particular interest, because though I have seen several cases of laryngocele, I had seen none that needed external operation until very recently.

This particular patient is a man who is on service with the Red Cross. He has been prevented from going overseas because of his having a large laryngocele, visible on the outside and behaving similar to Dr. Taylor's case in that slight pressure will make the sac empty itself into the interior of the larynx. He has been given an

appointment to come back very shortly for operation, so I will follow Dr. Taylor's example and I hope I can tell you next year about a similarly successful result. It might be worth mentioning that we obtained a beautiful demonstration of this sac by planigraphy. I think planigraphy does afford an excellent means of visualizing and graphically recording the form and relations of a laryngocele.

DR. LEROY A. SCHALL: Mr. President, in the past few years I have seen four cases of laryngocele at the Massachusetts Eye and Ear Infirmary. Two of these cases were reported by Drs. Allman and Cordray in The Annals of September 1942.

One bilateral case which I watched develop was a case where there was a persistent hoarseness and after repeated biopsies, looking for cancer, the correct diagnosis was made.

I reported a case, at the Eastern Section of the Triological Society, of a laryngocele associated with cancer of the larynx. This was a patient on whom I did a total laryngectomy.

Although I realized that it is dangerous to claim priority, I said I thought perhaps that this was the first case of laryngocele associated with cancer of the larynx in the literature.

I wish to acknowledge the fact that Dr. Gordon B. New in an article on cysts of the larynx had reported a case similiar to mine.

I appreciate Dr. Taylor's article very much. This is an interesting subject and I know I have been particularly fortunate in having seen four cases.

DR. SAMUEL IGLAUER: Mr. Chairman and Gentlemen, Dr. Taylor was kind enough to refer to a case which I reported in 1921.

When I first saw this child he was about eight years old and nothing was done. He did not require an operation apparently and went along till I saw him about twenty years later and he still had his laryngocele without any particular trouble.

I think later he was in the Army and he died from disease or injury. In other words, unless the laryngocele produces symptoms there is no particular occasion to operate on it.

The term "laryngocele" is perhaps unfortunate. I would not dare to question Vergo. After all it is an aerocele rather than a laryngocele.

Dr. Taylor suggests that these sacs are a sort of accessory lung and when the ape closes his glottis he has a sac on which he can draw for oxygen. I don't believe there would be much absorption of oxygen from a sac of that size.

Since Dr. Taylor is so interested in swimming I would suggest that maybe this sac is a life preserver which the monkey wears inside his neck.

I think I also read somewhere that the animal blows up this sac to scare his enemy, giving him a frightful appearance.

It was reported by several authors that there were 85 cases of this kind in Moore's paper. However, he was dealing with various lesions of the larynx and had only about ten or twelve cases of true laryngocele.

The diagnosis, can of course be made with the x-ray as was done in my patient some twenty years ago. If you have the patient perform the Valsalva Act and blow out his sac it will show up much better in the x-ray.

A child may be given a whistle to blow to distend the sac.

There is no objection that I can see to putting in a needle. I don't believe it would carry infection in. Of course, if the sac becomes infected then you have a deep abscess in the neck and it is much more serious.

Dr. W. Likely Simpson: I just want to bring out one or two points that my case, kindly mentioned by Dr. Taylor, presented. This was an external laryngocele or aerocele of traumatic origin. This man had had a slight encounter with one of his friends and the gentleman had placed his heel upon the region of the larynx and traumatized his neck about a year before this. When I opened him in the midline there was an opening through the left thyroid cartilage about a quarter of an inch in diameter, and through this opening air escaped into the cavity about the size of a small egg. This

aerocele could be expressed by external pressure or could be inflated at will.

After the usual examination, filling the sac with lipiodol for x-ray studies is helpful.

Dr. George B. Wood: Although I have not had much clinical experienced with this rather rare condition I understand that we can have two distinct types of laryngocele. One is the so-called idiopathic or developmental type and the other is of traumatic or pathologic origin. They can be readily differentiated by histologic examination of the sac. In the idiopathic type the sac is lined with epithelium from the laryngeal mucosa, while in the traumatic type the air is directly in the connective tissue spaces and surrounded by inflammatory capsule, without a lining epithelium.

President Imperatori: Thirty years ago I saw a patient, on whom Dr. Harmon Smith had made a diagnosis of what he termed pneumatocele; the diagnosis being arrived at by palpation and auscultation.

Our x-ray apparatus was not functioning at that time in this particular hospital. I don't think we had an x-ray at that time. I recall that this was the first time that I had been instructed in palpation of the larynx; that is, moving the larynx back and forth in the tissues of the neck and crepitating the cartilages on the spinal column. Palpation of the larynx should be practiced more frequently—for obvious reasons.

I had the opportunity of seeing the x-rays of this patient that Dr. Taylor has shown and certainly I congratulate him on his very fine surgical procedure and the results obtained.

Dr. F. M. Law: The x-ray examination in this particular type of case is given by means of planigraph or tomograph, whichever you choose, and plain lateral films of the neck.

A tumor ordinarily will not show in the neck in the lateral film, because the density of the tumor mass is the same as the density of the neck but in this type of case that area is filled with air and will show because the density is less than the surrounding tissue.

A diagnosis is possible with the stereoscopic lateral film, but more possible with the modern planigraph or sectional examination. That is a very simple procedure, but one precaution to the technician who makes the film. A lateral film of the neck will show a suspicion of aerocele and then the tomograph or planigraph is made, but in that particular type of case it must be made during inspiration and expiration. In inspiration it may not fill with air and may not be visible, but with expiration it does show.

DR. H. M. TAYLOR (closing): I wish to thank the members of the Association for the discussion of my paper.

I believe Dr. Iglauer could read with interest and profit "The Mechanism of the Larynx," by Negus.

The laryngeal air sacs have only two functions. One: Filled with oxygen, and during expiration with carbon dioxide, they serve as mixing chambers and are used with the least amount of exertion by the lungs. Two: They house mucous glands whose function it is to keep the neighboring parts moist.

PATHOLOGY OF NASAL MUCOUS MEMBRANE AND SUGGESTIONS AS TO TREATMENT

LE ROY A. SCHALL, M. D.

BOSTON, MASS.

From the title, this paper falls naturally into sections; the first on the tissue changes of nasal mucosa is somewhat scientific; the second, dealing with treatment, is, I hope, practical.

Respiratory mucous membrane consists of epithelium, basement membrane, and a tunica propria or stroma.

The epithelium is of a pseudocolumnar ciliated variety, and in the embryo and infant is uniform throughout the respiratory portion of the nasal mucosa. In the adult, from repeated nasal infections, from traumatization of respiratory blasts of air or from trauma of faulty nasal hygiene, nature may try to protect traumatized areas by a metaplasia from columnar epithelium to stratified squamous epithelium. Because of the absence of cilia, these are the so-called silent areas of the nose and are found over the anterior tip of the middle turbinate, the anterior tip of the inferior turbinate, and the inferior portion of the septum. The natural motion of the cilia is toward the ostia, and in the normal nose the surface epithelium is covered by a thin sheet of mucus.

The surface cells rest on the basement membrane. This thin band of undifferentiated tissue limits the epithelial cells from the underlying tissue. Its exact origin is doubtful but it is thought to be derived from connective tissue. It is canaliculated, and through the canaliculi, wandering cells may escape to the surface.

The tunica propria consists of a fibro-elastic connective tissue network—superficial, loosely interwoven, deep, more compact and condensed. Where the mucous membrane overlies cartilage, the condensed deep layer of the stroma forms the so-called perichondrium

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From the Department of Otolaryngology, Massachusetts Eye and Ear Infirmary, Boston, Mass.

and, where it overlies bone, the periosteum. The tunica propria is the supporting structure. It gives support to the glands, vessels, nerves and lymphatics. Within the stroma are various tissue cells—polymorphonuclear leucocytes, histiocytes, endothelial cells, fibroblasts, connective tissue cells, and the small round lymphoid cells. The specialized venous cavernous spaces are particularly numerous in the inferior turbinate, to a lesser degree in the middle turbinate, and may also be found at the tubercle of the septum.

In acute rhinitis the normal histology is disturbed. The temporary vasoconstriction of the vessels is followed by a vasodilation. The polymorphonuclear leucocytes at first lag along the vessel wall, then cling to the wall and many by diapedesis pass through the wall and appear in the tissue spaces. The defense mechanism of the tissue becomes active, the wandering tissue cells migrate to the source of irritation, so that there is an increase of the wandering cells in the mucosa. With the slowing of the blood stream the blood plasma by transudation escapes into the tissue spaces. The cavernous spaces are dilated and temporarily lose their power to contract. With an excessive blood supply to the glands there is an excessive stimulation of the glands with a consequent excessive amount of secretion.

No two rhinologists agree on the treatment of an acute rhinitis Although they all agree that the ideal treatment is bed rest at the first sign of a cold, the local treatment varies from those who perhaps overtreat the local condition to those who condemn all local medication. I am opposed to permitting or encouraging a patient to make a trip to the doctor's office on a cold wintry day to receive a treatment for a head cold. In my opinion, the first principle of treatment is nasal ventilation.

The late Dr. D. Campbell Smyth taught that vasoconstrictors should be used often enough to open the nose and then used often enough to keep it open. This is, I believe, good therapeutics. The vasoconstrictors should be isotonic and of a pH compatible with that of the nasal secretion. They should not irritate. The radio announcement that the stinging of the nose drops means they are penetrating the tissues is a gross misstatement of fact. Nasal vasoconstrictors should not sting or irritate. Test your medication on your-

self and find out how often you could not or would not take your own medicine.

Vasoconstrictors contract the dilated vessels and cavernous spaces. The blood supply is diminished and shrinkage of the swollen mucosa permits nasal ventilation. With the diminution of the blood supply there is less stimulation of the glands and therefore less secretion. Man, like the horse that can't breathe through its mouth, is more comfortable if he can breathe through his nose. George Catlin, the artist, after his life with the Indians, wrote in 1861 a small book entitled "Shut Your Mouth." You can only shut your mouth when you can breathe through the nose, and medication that permits nasal breathing is, in my opinion, medication based on nasal pathology.

I have not been impressed with the efficiency of bacteriostatic or bacteriocidal drugs used in conjunction or in combination with vasoconstrictors. Limited bacteriological investigations of their efficiency in a selected group of patients has failed to show any demonstrable diminution of bacteria by their use. I am not sure about the efficiency of the use of finely divided sulfonamides insufflated over the nasal mucosa after careful shrinkage. Some patients appear to be benefited by their use.

When the nasal secretion is so thick and sticky that the patient cannot properly clear his nose, I am not opposed to a gentle irrigation with Ringer's solution, but I am opposed to the use of nasal irrigations by the patient. I believe their use starts a vicious cycle and that they do more harm than good.

Spot suction is of great value in freeing the nasal cavity of excessive secretion. Massive suction is dangerous, as it may cause further trauma to a diseased membrane.

The correction of a coexisting constipation and the use of a "Dover's Powder" are two old remedies that have proven their worth.

Chronic Rhinitis—In chronic turgescent rhinitis the pathology of the nasal mucosa is characterized by dilated cavernous sinuses that have lost their power to contract. Associated with it there is an increase of the fibroconnective tissue of the stroma. The polymorphonuclear leucocyte has disappeared from the tissue spaces and

there is an increase of the small lymphocytes, particularly around the glands and the ducts of the glands.

The increased vascularity of the mucosa means increased activity of the glands. The secretion is tenacious and sticky and with a blocked nose there is postnasal dropping and increased postnasal secretion.

The treatment depends upon the etiology, and the etiological factors are many. Jarvis' classification of causes is practical and should be utilized. A nasal airway unilaterally obstructed by deviations and spurs of the nasal septum will in time by overwork of the open side cause a compensatory hypertrophy of the mucosa of that side. It is the blocking of the former good side of the nose that brings the patient to the physician.- Climatic changes in temperature and in humidity; congestion of other body organs—such as the congestion of the stomach mucosa from overeating or overdrinking; congestion of the uterine mucosa from menstruation or from pregnancy; congestion due to constipation or congestion due to a tired out nasal mucosa from trying to fulfill its physiological function, not only all day but all night long—from the mistaken idea that to be rugged and robust you should let the wintry blasts blow over you —all these effect the nasal mucosa. We too often forget that of all the animals man is the only one that does not protect his nose while he sleeps.

The treatment of chronic turgescence of the nasal mucosa should be based on the correction of the etiological factor or factors producing the congestion. Normal nasal functioning should be restored by equalizing nasal ventilation by the correction of septal deviations and spurs, by correcting dietetic errors and by eliminating constipation. We should emphasize the relation of skin temperature to nasal congestion although we will not succeed in persuading anyone to dress properly. Bare legs, bare heads and bare chests are in fashion, even if the nose runs and the secretion trickles down the throat.

As many of my patients have been relieved by the simple expedient of partly closing the windows and sleeping in a room that is not under 50° F., as by any other means.

When anatomic, physiologic and dietetic errors are corrected, improvement offtimes is hastened by destruction of the dilated cavernous spaces. This may be accomplished by subepithelial electrocoagulation or by the careful injection of sclerosing fluids into the stroma.

Vasomotor Rhinitis—The pathologic changes produced by vasomotor rhinitis are expressed by a swelling of the superficial epithelial cells, at times by a desquamation of the cells, by an alteration of the basement membrane, and the condition is characterized by dilated tissue spaces caused by transudation of serum into the stroma. There is an infiltration of cells, not only eosinophiles but also small round lymphocytes, histiocytes and plasma cells, an infiltration most marked just beneath the basement membrane. The cavernous spaces as well as the capillaries are dilated.

To enter into a discussion of the treatment of vasomotor rhinitis is beyond the scope of this paper. Suffice it to say that after a careful search for a specific allergen has failed, or when after an exhaustive effort to produce immunity by adequate treatment of suspected allergens, then certain local measures are of value. Vasoconstrictors give but temporary relief. Realizing that the basic pathology is one of edema with dilated cavernous spaces, subepithelial electrocoagulation or destruction of the dilated spaces and cavernous sinuses by the careful injection of sclerosing fluid seems logical. Small reactionary polypi may likewise be destroyed by the careful use of the electrocoagulating needle.

Atrophic Rhinitis—The etiological factors of atrophic rhinitis will not be discussed. The pathology is that of a metaplasia of the pseudostratified columnar ciliated epithelium to that of stratified squamous epithelium. The basement membrane is thickened. The stroma shows an increased fibrosis. The fibroconnective tissue becomes increased, the vessels are thickened and the glands, due to both the decreased blood supply and the fibrosis, undergo atrophy. Due to the absence of cilia the secretion stagnates, secondary infection occurs, and scabs and crusts form. With the complete atrophy of the glands and when there is no longer any glandular

secretion, the scabs, crust and odor disappear, so that atrophic rhinitis is said to be a self-limited disease.

Many therapeutic agents have been tried in this disease—at one time glucose applied locally on strips of gauze, at another the local use of estrogenic substances. At the present time, the local use of prostigmin is being used. Glucose and estrogen appeared to give some relief while the patient was under active treatment, but the scabs and odor re-appeared as soon as the frequent cleansing of the nasal cavities ceased. At the Infirmary we are attempting to duplicate the results of Zoskin and Bernheimer[1] and substantiated by Henner and Busby[2] by the use of prostigmin methylsulfate.

I am of the opinion, however, that careful cleansing of the nose is the best therapeutic measure we have, although I admit it is treating the effect and not the cause of the disease.

Operative procedures to narrow the wide nares have in my hands failed.

SUMMARY

The histology of respiratory nasal mucosa, the pathological changes seen in acute rhinitis, chronic turgescent rhinitis, vasomotor rhinitis and atrophic rhinitis, together with the highly controversial subject as to their treatment, is herewith presented.

243 CHARLES STREET.

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DISCUSSION

DR. ARTHUR W. PROETZ: Mr. Chairman and Gentlemen, having instigated this paper, I listened rather carefully for something in the treatment phase with which I might find fault and so justify the opening statement of the essayist. I must confess that I agree with him practically throughout.

It seems to me that this paper is a very sound one. Listening every so often to such a paper will serve to keep our feet on the ground and to let us know that people who think are still resorting to simple logical remedies and not to the fancy things that we hear about on the air, as Dr. Schall says.

There are two very small practical matters I would like to discuss. I don't quite agree with Dr. Schall regarding irrigation. I have been off and on irrigation in acute conditions for a long time and finally abandoned it because I believe it spreads the infection to some extent.

But more important is the fact that one can remove the secretions better by capillary suction than by irrigation. Irrigation simply washes the surface. If one uses a thin suction tube to take hold of the secretion before any medication is used, at almost any point he can draw the whole mass of mucopus away just as you tear a table cloth off the table and in that way attain what irrigations would not reach, because the latter dilute and destroy the continuity and tenacity of the secretion.

The other thing is the treatment of the dry nose. A few years ago I began to experiment with solutions for that purpose.

This simple solution, which proved surprisingly effective is five per cent glycerine, five per cent ethyl alcohol in physiological sodium chloride solution. Some scent needs to be put into it because for some reason the mixture has a very stagnant odor. I use a few drops of spirits eau de cologne and stain it yellow with sunset yellow, (2 drops per ounce of a 1% solution).

It should be made up carefully. If the odor is not corrected people won't use it. These drops have to be administered in large quantities, a half dropperful in each nostril three, four or five times a day. In cases of atrophic rhinitis and of senile rhinitis sicca it is very useful.

I use it also in the bronchi of children with bronchitis and with acute tracheo-bronchitis to good effect.

PRESIDENT IMPERATORI: I would like to state what my experience has been in a family of four young girls from seven years up to fourteen, and a mother and father, all with atrophic rhinitis.

Under treatment for the past year with the estrogenic substance the children, and particularly the younger child, have apparently been cured. The fourteen year old girl is partially cured and the mother and father remain practically the same, that is, estimating a cure on the amount of crusts, size of the turbinates, etc.

With the estrogenic substance applied to the adult the odor has disappeared entirely.

I offer that simply from my own clinical experience.

DR. T. E. CARMODY: There are several things which Dr. Schall mentioned which seemed to me quite important. One is that he cannot control his own family any more than can any of the rest of us. However, I think that the possibility of some of these people going with fewer clothes is better than we formerly thought.

I remember a statement of an Indian in Montreal when someone said to him, "Will you not be cold with nothing around you?"

He turned to the speaker and said, "Is your face cold?" The speaker said, "No," to which the Indian replied, "Then me all face."

So I think if we go with fewer clothes, even these girls going with socks or without socks, maybe will get along all right.

Years ago in treating tuberculosis we used to have the windows wide open. If the doctor came in and found the window only half way open he thought the patient would not get well.

I had a patient about this time who asked me whether she should sleep on the porch or stay inside. I said to sleep on the porch. I continued to treat her, as we did in those days in the office and her larynx got very much better and about three months later I said, "Aren't you glad that you slept outside?" She said, "I didn't."

Another thing which I know we use a great deal are the silver salts and I am glad that Dr. Schall limits those. However, in some cases I believe that they are good, but I think that neosilvol has done a great deal of harm because every mother uses it because it does not stain and I have seen quite a large number of cases of argyria following its use. There have been a number of cases re-

ported. I have one patient who used it for sixteen years and he is just about as black as you can have them.

The ciliary action is of course interfered with by almost any drug put in the nose, as has been proven by Proetz. I have always been against irrigation but I think the suction with a small tube, as Dr. Proetz suggests, is excellent and I don't believe that we should use suction with a great deal of force because we probably will rupture some of the vessels in the mucous membrane.

Dr. Thomas C. Galloway: We agree too easily perhaps with Dr. Schall because his logic is sound, but it seems to me that there are certain fundamental conceptions in the philosophy of therapeusis that have to be considered. At one time doctors talked of breaking the fever in infection. Now the conception is that fever may be a conservative process.

The normal physiology of the nose has been very beautifully worked out by Proetz, Hilding and others, and yet there has been remarkably little work done on the process of inflammation which leads to healing.

We assume that certain pictures are physiological and we strive to reproduce those. We used to say that excessive secretion was abnormal, so we gave atropine. I think none of us now believe that atropine is anything but pernicious in the treatment of acute cold. May it not even be possible that if we produce the processes of inflammation that we may be promoting healing?

An analogy is given by the use of x-ray in the treatment of acute infections. When one treats acute cervical adenitis with a proper dose of x-ray one heightens the intensity and hurries the tempo of inflammation so that a process which might take from two to six weeks to resolve may under the influence of x-ray in twenty-four to seventy-two hours go through the normal stages of inflammation, whether the end result be regression, or localization and suppuration.

Likewise I still believe that under certain conditions if one sees an upper respiratory inflammation very early, if it is not a generalized and widespread infection that is already beyond the use of any

local applications, one may by the judicious use of packs of hypertonic magnesium sulfate, or of mild silver salts, especially the partially ionized mild silver protein, get real benefit.

Dr. Fred Z. Havens: Referring to the matter of the fresh-air fiend who may do himself more harm than good, you might be interested to know that there is a scientific basis for an argument against the necessity of sleeping outdoors or with open windows.

A few years ago a friend of mine, who is a heating and ventilating engineer, gave me one of his textbooks. In browsing through it I learned that there is at the University of Illinois a research institute devoted to the study of heating and ventilating problems. They have tested various types of construction with reference to the amount of air which will pass through the walls. They refer to this as infiltration and they have found in their researches that with the best possible modern construction there is sufficient infiltration interchange between the inside and outside air to provide a complete change seven times an hour.

Dr. L. A. Schall (closing): Frankly, I am disappointed that you agreed as much as you do and that there was not more disagreement and more discussion. I thought this paper would stir up a lot of discussion.

I agree with Dr. Proetz that localized spot suction is of great value.

In my paper I said I am not opposed to careful irrigation when necessary. I also have used packs. One such treatment yesterday gave me great relief. I am not opposed to this type of treatment, if done carefully and gently.

Thank you very much for listening to me.

INTER-AMERICAN RELATIONS IN OTOLARYNGOLOGY

A Report

CHEVALIER L. JACKSON, M.D.

PHILADELPHIA, PA.

During the past few years, many of our medical societies have been starting to direct attention to the promotion of better relations and understanding between physicians and surgeons of this country and those of the other American republics. This has been in part due to the Good Neighbor Policy of our government, expressed through both the State Department and the Office of the Coordinator of Inter-American Affairs. Even without these influences, however, such a movement is a natural consequence of the disruption of the previous relations of all the Americas with Europe.

I shall take the liberty, in this paper, of quoting and paraphrasing passages from a report prepared for the American Academy of Ophthalmology and Otolaryngology.[1] In this report I sketched briefly the history of international relations in otolaryngology as shown by the succession of international congresses that have been held since the First International Otological Congress, which took place in the United States, under the presidency of Dr. John Roosa about 70 years ago. The next one was held in Milan, and subsequent congresses met at Bâle, Brussels, Florence, London, Bordeaux and Budapest; the Ninth, the last before the outbreak of World War I, was held in Boston in 1912, with Clarence Blake as president.

The war caused a hiatus of ten years, but the Tenth International Congress of Otology was held in Paris in 1922 under the presidency of Professor Sébileau. This congress was not truly "international", because representatives of Germany and Austria were not invited. Six years later a Congress was held in Copenhagen under the presidency of Professor Schmiegelow a Corresponding Fellow of this Association, which it was decided to call the First International Congress of Oto-Rhino-Laryngology, instead of the Eleventh International Otological Congress. This Congress was a successor to

the ten Otological Congresses, and three "Laryngo-Rhinological" Congresses. It seemed as if the succession of International Congresses was to be permanently resumed. A fine Congress of Oto-Rhino-Laryngology (the Second) was held in Madrid in 1932, with Professor Tapia as president, and the Third International Congress of Oto-Rhino-Laryngology took place in Berlin in 1936 under the presidency of Professor Carl Von Eicken. This was a great success, and a well balanced representation of otorhinolaryngologists from many nations of the world participated. Arthur Proetz, Horace Newhart, and I were among those that attended from the United States. The British, French and Belgian delegations were especially numerous, which seemed a sign pointing toward the development of real friendship and collaboration. In Berlin it was voted that the next Congress of Oto-Rhino-Laryngology (the Fourth) should take place in Amsterdam in 1940, under the presidency of Professor Burger. Plans for this congress were being energetically prosecuted until the spread of World War II made it obvious that cancellation would be necessary.

Let us outline briefly some aspects of the history of otolaryngology on the South American Continent. According to Dr. Aristides Moll, Secretary of the Pan-American Sanitary Bureau, otolaryngology was "among the first of the specialties to forge ahead, usually tagging along close behind her elder sister, ophthalmology"! As early as 1868 a Mexican, Iglesias, published a Handbook (in French). Dr. Carlos Desverine of Havana was one of the first Latin Americans to devote himself to the specialty (1887), and Dr. Conde Flores of Venezuela was another pioneer (1895). Institutions for the deaf were established in Rio in 1856, Buenos Aires in 1857, Mexico in 1869 and Havana in 1925.

In 1936 a historic meeting was held in São Paulo, Brazil, under the presidency of Dr. Mario Ottoni Rezende, which was called the Paulista Otorhinolaryngological Week. At this meeting it was decided to hold a First Brazilian Congress of Oto-Rhino-Laryngology, after an interval of two years. This First Brazilian Congress took place in 1938, in Rio de Janeiro, with Professor Marinho as president, and at its conclusion a decision was reached to join with colleagues of the other nations of South America in a First South

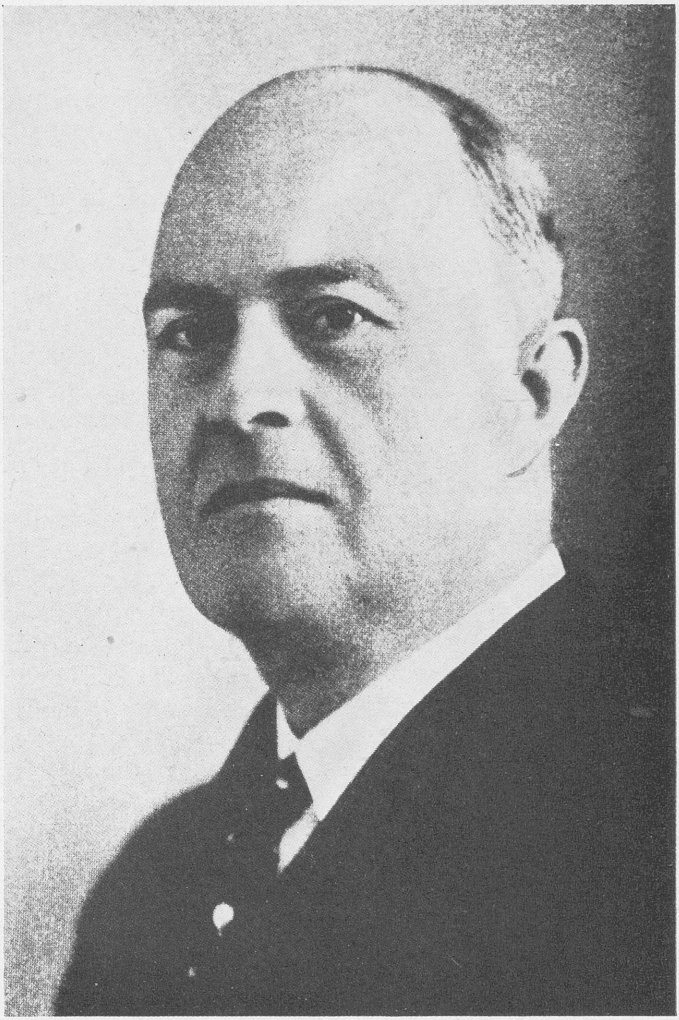

Fig. 1.—Dr. Eliseo V. Segura, Ex-Professor of Otorhinolaryngology of the University of Buenos Aires (1919-1942); President of the National Academy of Medicine of Argentina. Dr. Segura is the acknowledged dean of Latin American Otolaryngologists, and was President of the First South American Congress of Otorhinolaryngology held in Buenos Aires in 1940.

American Congress of Otorhinolaryngology. This First South American Congress of Otorhinolaryngology was held in Buenos Aires in 1940, under the presidency of the Dean of Latin American Otolaryngologists, Prof. Eliseo Segura (Fig. 1) and at its conclusion a First Latin American Congress was planned, to be held in São Paulo, Brazil, in 1943, under the presidency of Professor Paula Santos. However, since it was deemed impracticable to hold a Congress at this time in São Paulo, it was decided to have, instead, a Second South American Congress in Montevideo, with Professor Justo Alonso (Fig. 2) as president. This Congress is to take place in November of this year (1944). In the light of this historical background, there can scarcely be any doubt of the advisability of taking steps toward the organization of a Pan American Congress of Otolaryngology.

Thus far, the only occasions on which the otolaryngologists of the United States have met in scientific session with those of the other American Republics have been during the Congresses of the Pan American Medical Association. At the Fourth Congress of this Association held in Dallas, Texas, in 1933, Dr. Chevalier Jackson Sr. was president of the Section on Otolaryngology; he was again president of the section the following year for the Fifth Congress, which was a "floating" Congress stopping at Havana, Panama, Caracas and San Juan. The Sixth Congress ws held in Rio in 1935 and the Section was presided over by Dr. Lee Hurd. Finally, the Seventh Congress of the Pan American Medical Association took place in Havana in 1938, and Dr. William E. Sauer presided. The speaker had the honor of being a sort of "perennial" secretary of the Section in all of these congresses.

On January 19, 1942, Dr. Wherry wrote me that the Program Committee of the Academy had appointed me as chairman of a committee to develop plans for a Pan American Congress of Otolaryngology. Because of the increasing difficulties in communications and transportation resulting from our progressively more complete involvement in the war effort, nothing was done during the ensuing months except to write informally to some of our Latin American correspondents concerning the project of a Pan American Congress. Throughout the spring months of 1943, Dr. Babbitt, then president

Fig. 2.—Dr. Justo M. Alonso, for 25 years Professor of Otorhinolaryngology at the University of Montevideo. Professor Alonso will be President of the Second South American Congress of Otorhinolaryngology, to take place in Montevideo, October 11th to 15th, 1944.

of the Academy, sent letters to a number of representative otolaryngologists in the various other American republics, telling them about the meeting of the Academy in Chicago in October 1943 and inviting them to attend. Despite the great difficulties in communication and transportation due to chaotic international conditions imposed by war, a very gratifying response rewarded our efforts. A number of distinguished Latin American otolaryngologists made the trip to Chicago, and a number of others accepted the invitation to attend, but were prevented from reaching Chicago by circumstances beyond their control. The great majority of those who did not accept wrote us expressing their appreciation of the invitation and their warm sympathy with the idea of establishing some sort of Pan American organization in otolaryngology.

The entire meeting of the Academy was pervaded with the spirit of inter-American friendship and cooperation. It was very appropriately keynoted by the magnificent address of the Guest of Honor, Dr. Ralph Fenton, on "The Unity of Medical Science in the Americas" on Sunday afternoon. At the Inter-American Dinner on Wednesday night, President Babbitt welcomed the Latin American guests in Spanish, and they were then introduced individually. Addresses of the evening were given by Mr. William L. Schurz of the Division of Cultural Relations of the State Department, and Colonel Albert Dreisbach, of the Office of the Coordinator of Inter-American Affairs.

Our Committee on Inter-American Relations met for the first time at breakfast on Sunday morning, October 10th. At this meeting Dr. Gradle was asked to speak with regard to the project of a Pan American Congress of Otolaryngology, from his experience in connection with the establishment of the Pan American Congress of Ophthalmology. He outlined briefly the story of the ophthalmological organization and made numerous suggestions regarding the proposed otolaryngological one. Various members of the Committee, and the Latin American colleagues present, discussed the project of a Pan American Congress of Otolaryngology. Several of the members of our Committee expressed the opinion that there would be numerous advantages in holding the first Pan American Congress in conjunction with, or immediately following an annual

meeting of the American Academy, as the First Pan American Congress of Ophthalmology had been held. Subsequent congresses would be held in the other American republics, perhaps in conjunction with the meeting of another of the Latin American societies.

At the conclusion of the meeting it was moved by Dr. Fenton that "the academy be requested to sponsor the establishment of a Pan American Congress of Otolaryngology and to finance its initial meeting, this initial meeting to be held in conjunction with a meeting of the academy." This motion was seconded by Dr. Theobald, and passed. Two additional meetings of the Committee were held before the close of the Academy sessions, and the Committee was informed that the Council of the Academy had agreed to sponsor a Pan American Congress of Otolaryngology to take place in connection with the annual meeting of the Academy in 1945.

The Academy Committee is continuing as both a Committee on Inter-American Relations in Otolaryngology and a Committee on Pan American Congress of Otolaryngology. The following is a complete list of the members of the Committee as it now stands: *Executive Committee*: Chevalier L. Jackson, Chairman, James A. Babbitt, Ralph Fenton, Juan Font, Arthur Proetz, Frank Spencer, Gordon B. New. *Members*: Victor Alfaro, Howard C. Ballenger, Wesley Bowers, Mackenzie Brown, Thomas Carmody, George M. Coates, William Gill, Perry Goldsmith, Paul Holinger, Charles J. Imperatori, Francis Lejeune, James M. Robb, William E. Sauer, LeRoy Schall, Marshall Taylor, Walter Theobald. *Consultants*: Harris P. Mosher, Moacyr Alvaro, Conrad Berens, Harry Gradle, Forrest J. Pinkerton. *Ex-Officio*: Lawrence T. Post, President, William L. Benedict, Executive Secretary. The majority of these men are members of the American Laryngological Association.

During the current year this committee is keeping in touch with all of the Latin American colleagues with whom contact has been established, and establishing contact with additional ones. Letters are being written to the various societies of otolaryngology in the other American republics, acquainting them with the intention of the American Academy to sponsor a Pan American Congress in 1945, and extending to them an invitation to join with the Academy in

organizing it. The Mexican Society of Ophthalmology and Otolaryngology already assures us of its fullest cooperation. At this meeting of the American Laryngological Association, the Mexican Society is officially represented by its Vice President, Dr. Pedro Berruecos, and its Secretary, Dr. Manuel Lopez y Lopez. The Cuban Society has pledged its support through its President, Dr. Pedro Hernandez Gonzalo, who attended the 1943 meeting of the Academy and the meetings of our Inter-American Committee. The support of the Colombian Society of Ophthalmology and Otorhinolaryngology is pledged by its President, Dr. Jorge Suarez-Hoyos in a letter just received.

Professor Justo Alonso, President of the Second South American Congress, has been informed of the decision of the American Academy to sponsor the establishment of a Pan American Congress of Otolaryngology and finance its initial meeting, to be held in conjunction with the Annual Meeting of the Academy, in 1945. Professor Alonso has already indicated his approval of the idea of organizing a Pan American Congress, and he has expressed his willingness to receive as delegates to the Second South American Congress this November members of the American Academy whose names we send him. He has already been sent the names of the members of our committee, and we should be glad to send on the names of any other otolaryngologists who are interested.

Preliminary steps are being taken to carry out the suggestion of Dr. Paul Holinger, that we endeavor to "promote the exchange of post-graduate students and residents in otolaryngology between the United States and the other American Republics." A form letter is being prepared, to be sent to all of the institutions offering approved residencies in otolaryngology, asking them what opportunities they have to offer for Latin American postgraduate students and residents. One man, has already been given a residency at the Manhattan Eye and Ear Hospital through the efforts of our Committee Dr. Victorino Marquez of Venezuela.

In conclusion, I may say that in organizing the Pan American Congress for 1945 the Academy Committee will look for moral support and wise counsel to the American Laryngological Association.

Needless to say, the Association will at all times be kept officially informed regarding plans for the Pan American Congress, since its secretary, and its present president, are members of the Academy Committee. I hope that our new president will consent to become a member of this committee also. Finally, I take the liberty of suggesting that some representative Latin American otolaryngologists be made Honorary or Corresponding Fellows of this Association. We have, in the past, had two distinguished Cuban otolaryngologists as Corresponding Fellows, Dr. Carlos María Desverine y Galdos, and Dr. Enrique Fernandez Soto, but at the present time we have no Latin American members.

An appendix has been prepared for submission to the Council of the Association, in which are given: 1) a list of Latin American Otolaryngological Societies, and 2) a list of Latin American otolaryngological journals, with all of which I believe our journals should establish exchanges, and to which I believe we should encourage our libraries to subscribe.

255 South 17th St.

Reference

1. Report of Committee on Inter-American Relations in Otolaryngology, Trans. Amer. Acad. Ophth. & Otolaryng. Jan.-Feb., 1944.

LIST OF LATIN AMERICAN OTO-RHINO-LARYNGOLOGICAL SOCIETIES

Argentina:

Sociedad de Otorrinolaringología de Buenos Aires.
Sociedad Rioplatense de Otorrinolaringología, Buenos Aires.
Sociedad Argentina de Otorrinolaringología de la Associación Médica Argentina.
Sociedad Argentina de Bronco-esofagología.
Sociedad de Otorrinolaringología y Broncoesofagoscopía de Cordoba.

Brazil:

Sociedade Oto-Rino-Laringologia de Rio de Janeiro.

Sociedade de Oftalmologia e Oto-Rino-Laringologia de Rio Grande do Sul, (Porto Alegre).
Sociedade de Oftalmologia e Oto-Rino-Laringologia da Baía.
Sección de Otorrinolaringologia de la Associacão Paulista de Medicina.

Chile:
Sociedad Chilena de Otorrinolaringología (Santiago).

Colombia:
Sociedad Colombiana Oftalmológica y Otorrinolaringológica (Bogotá).

Cuba:
Sociedad Cubana de Oto-Laringología.

Mexico:
Sociedad Mexicana de Oftalmología y Otorrinolaringología.

Paraguay:
Sociedad de Oftalmología y Otorrinolaringología del Paraguay (Asunción).

Uruguay:
Sociedad de Oto-Rino-Laringología del Uruguay.

Venezuela:
Sociedad de Otorrinolaringología, Oftalmología y Neurología (Caracas).

LATIN AMERICAN OTOLARYNGOLOGICAL JOURNALS

Argentina:
Revista Argentina de Oto-Rino-Laringología
Constitución 2948, Buenos Aires.
Revista de la Sociedad Argentina de Otorrinolaringología
Santa Fé 1171, Buenos Aires.
Revista de Oto-Rino-Laringología y de Bronco-Esofagología
Calle Colón 627, Cordoba.

Brazil:
Anais de Oto-Rinó-Laringologia,
Rua Imperatriz 218, Recife.

Arquivos de Clínica Oftalmológicae Oto-Rino-Laringológica,
Galeria Chaves, 1 andar, Porto Alegre.

Revista Brasileira de Oto-Rino-Laringologia
Rua S. Bento 181, São Paulo.

Chile:

Revista de Otorrinolaringología
Dr. Arturo Tello, Rosas 910, 2° piso, Santiago.

Cuba:

Revista Cubana de Oto-Neuro-Oftalmiatría
Aptdo. 970, Habana.

Mexico:

Anales de la Sociedad Mexicana de Oftalmología y Oto-Rino-Laringología Escuela de Medicina, esq. Brasil y Venezuela, Mexico, D. F.

Paraguay:

Anales de Oftalmología y Oto-Rino-Laringología del Paraguay
Calle Gnel. Martinez esq. Convención, Asunción.

Uruguay:

Anales de Oto-Rino-Laringología del Uruguay
18 de Julio 1270, Montevideo.

Venezuela:

Archivos Venezolanos de la Sociedad de Otorrinolaringología-Oftalmología-Neurología
Aptdo. 1428, Caracas.

DISCUSSION

Dr. Ralph A. Fenton: The industry and care with which Dr. Jackson as chairman of this committee has approached this task is comparable to that of Dr. Gradle's organization of the ophthalmological side of this matter. It is high time that we, along with other professions and groups in the United States, should take cognizance of the brilliant training and of the splendid institutions which exist for the benefit of our colleagues in Latin America.

Such was the text of what I had to say at Chicago: that I felt that we here in North America were somewhat ignorant of the splendid traditions of medical science in Latin America and that we should place all of our facilities at the command of our colleagues and should work with them toward better health for the citizens of their countries, as well as for our own.

PRESIDENT IMPERATORI: I am very much in favor of the continuance of these committees and the furtherance of the fine work that has been started by the Academy, and particularly the work of Dr. Jackson.

The only way we can know the South Americans is to talk with them and have them know what we think about certain things and to know what they think about us. In the postwar years it will be more or less necessary to understand these people better.

I feel very strongly about this, having lived in Colombia long enough to understand to a degree the temperament of our neighbors to the south.

There is no profession which can enter a country more easily than the medical profession and we should be the spearhead to establish these good relations.

Dr. Jackson, I want to thank you for your fine report and I hope that your enthusiasm will continue.

SOME PROBLEMS IN THE SURGICAL TREATMENT OF BILATERAL ABDUCTOR PARALYSIS OF THE LARYNX

JOSEPH D. KELLY, M.D.

NEW YORK, N. Y.

The object of this paper is to acquaint those laryngologists who have not had experience in the surgical treatment of bilateral abductor paralysis of the larynx with some of the problems I have met in the handling of these cases by arytenoidectomy. If the case is one where the patient has not had a tracheotomy, it is very likely that the operator will feel that a straight line incision, from the hyoid bone to the sternum, will be the incision of choice because it is natural to believe that you will be able to get a complete exposure of the trachea and the thyroid cartilage and thus obviate the necessity for a horizontal incision in addition to the tracheotomy incision, which so far has been a part of an arytenoidectomy.

If you have a case in which one thyroid operation has been performed by an excellent operator, where the pretracheal muscles have not been severed and where there was primary healing without distortion and the formation of scar tissue, it is probable that you will have no trouble in exposing the muscles covering the thyroid cartilage to your satisfaction. However, you must now completely expose the right wing of the thyroid cartilage and reflect upward the thyrohyoid muscle to expose that part of the thyroid cartilage through which you are to make the window for the extraction of the arytenoid. This is sometimes most difficult to accomplish under the best conditions without using a horizontal incision. When using the straight midline incision you must exert an unusual amount of traction upon the pretracheal tissue to expose the thyroid cartilage properly or you must displace the larynx in such a way that it will be a long and difficult task to locate and remove the arytenoid cartilage. I would not advise the midline incision for the beginner.

If, however, a patient comes to you for relief of his paralysis and he has had two or three thyroid operations together with x-ray treatment, the probability is that there is nothing left of the pretracheal muscles but a mass of scar tissue which is adherent to all the surrounding structures, and it may be barely possible to find a line of cleavage along which you can cut, even when using the horizontal incision. It would, therefore, be impossible to separate and retract this tissue if you were using a midline incision. I feel, therefore, that the easiest and proper incision for the expeditious performance of the arytenoid operation is the horizontal incision.

In short, fat necks with a small thyroid cartilage and trachea, you may find some difficulty occasionally in locating the lower border of the thyroid cartilage. It was a surprise to me when I first had the experience of spending some time dissecting the cricoid cartilage free of scar tissue, thinking it was the thyroid cartilage. This disorientation was brought about by the fact that I was operating upon a short, thick neck, full of scar tissue, where only with the most careful and tedious dissection was I able to expose the upper part of the trachea. This mistake on my part was due to the fact that I had made my incision at the lower border of the cricoid cartilage instead of the lower border of the thyroid cartilage. When I have a difficult case now, I make the horizontal incision longer and dissect the skin and subcutaneous tissues well upward and push my index finger over the trachea and up over the thyroid cartilage until I orient myself by feeling the thyroid notch. This accident may not happen to you, but it may shorten the time of the operation if you will use the patience necessary to insure the proper position for the horizontal incision.

I might further add that the majority of the 34 patients which I personally have operated upon have had two or more thyroid operations and necks filled with scar tissue, so that most careful dissection was necessary in some of them in order to avoid the vessels within the carotid sheath. In some of these cases the scar tissue will form about the carotid sheath and pull it from under the protecting margin of the sternocleidomastoid muscle. When this happens, care must be exercised not to cut into, or to put the point of a sharp dissecting scissors through, the jugular vein. Sometimes the

thyroid cartilage itself will be so covered with scar and muscle tissue that it is difficult to expose satisfactorily the area in which you want to make your window. The posterior border of the thyroid cartilage and the attachment of the inferior constrictor muscle may be so thickened and fixed in scar tissue that a satisfactory digital examination for the location of the arytenoid cartilage cannot be made. If circumstances and the case warrant it, a submucous dissection of this area may be performed. So far I have tried this in only one case.

Occasionally the arytenoid cartilage will be very elusive, especially in young people. The tissues are soft and pliable; the arytenoid cartilage is freely movable and may be placed rather deep in a broad larynx. Under such circumstances the position of the arytenoid cartilage and the facet of the cricoid cartilage may be readily located by placing the index finger of the left hand behind the posterior border of the thyroid cartilage and feeling the upper border of the cricoid cartilage through the fibers of the inferior constrictor muscle. If the point of the dissecting scissors is then pushed through the fibers of the arytenoid muscles which are exposed after making the window, you will be able to locate the position of the arytenoid cartilage and feel it move between the point of the scissors and the tip of your finger. By this means it is easier to find the location of the crico-arytenoid articulation and sever the capsule. In a like manner, the fibers of the arytenoid muscles may be separated and cut to expose the outer surface of the arytenoid cartilage. From there on it is a matter of patience and judgment until the arytenoid cartilage is delivered to your satisfaction.

In making the window through the thyroid cartilage, care should be exercised not to make the window too large. In our earlier cases we were content in placing the suture fixing the cord away from the middle line through the external perichondrium of the thyroid cartilage, but now we are fixing this suture by putting it through the cartilage and the perichondrium at the upper part of the anterior border of the window. If the window is too large, the suture may be placed too far forward on the cord to give a satisfactory voice.

The problem which made us change to putting the fixing suture through the thyroid cartilage was the severe paroxysms of coughing which sometimes follow the introduction of a tracheotomy tube into the larynx of a patient who has never before worn one. Occasionally it is very difficult to control this coughing and I believe that in one of our recent cases this coughing caused the breaking away of the suture which had been placed through the external perichondrium alone. Because of this coughing complication, we have recently had to remove the tracheotomy tube in one case on the second day, and in another on the third day. I know that I have made the statement that I believed that some of our good results were brought about by keeping the tracheotomy tube in place for as long as four, five or six weeks, but in nervous, excitable patients we must choose between what we would like to do and what is the best thing to do for the individual when an emergency arises. Both of these patients had their tracheotomy tubes corked two hours before the tube was removed. This was done to make sure that there would be no immediate dyspnea. We find that the corking of the tube alone sometimes relieves the coughing.

This complication of postoperative coughing brings up the problem of the best intralaryngeal area in which to place the fixation suture. If this suture is not put securely into the fibers of the thyroarytenoideus muscle or into the fibrous tissues of the cord itself, it may give way at this point just as well as from the external perichondrium. If the fixing suture is placed too far posteriorly into the muscle fibers of the crico-arytenoideus, it will not be very secure and it will be difficult to get the proper separation of the cords without too much distortion of the tissues in the posterior part of the larynx. In the presence of severe coughing a suture in this area may pull through these muscle fibers. Also, because of its proximity to the inferior constrictor muscle, severe gagging and vomiting may affect its position. It has been our practice to use the laryngoscope before determining the position in which to place the suture. The anesthetist or the assistant uses the scope and the operator with a small, fine hook pulls the cord from the median line of the larynx at several points. The location giving the desired airway with the least amount of separation of the cords anteriorly is

the point of choice. This is usually just anterior to the location of the vocal process of the arytenoid cartilage. It might be of interest to note here, that one of the laryngologists watching this operation suggested that we use more than one suture in fixing the cord. I think this is a good suggestion and we have followed it in one case. However, we must be careful not to separate the cords too much if we are anxious to retain a good speaking voice.

After the cord has been fixed, the closure of the laryngeal mucous membrane is the next step if there have been any lacerations of this tissue. The lacerations should be sutured with a fine eye needle, using triple O catgut, and the fibers of the arytenoid muscles should also be pulled together. After suturing the thyrohyoid muscle as nearly as possible to its original attachment to the thyroid cartilage, the wound is ready to close. We still use a drain which is removed anywhere from the second to the fifth day.

The next group of surgical problems to be considered are those which we have encountered in attempting to correct our failures by reoperating upon the same side. I have tried this in three cases and failed. The first patient I operated upon the second time on the same side was one I first operated on in November of 1941 and re-operated on in February 1942. At this time I went through the old incision, uncovered the window previously made in the thyroid cartilage, and attempted to put a suture into the location of the cord and further displace it from the median line of the larynx. This was a failure so far as the patient was concerned. However, I felt that I had accomplished something at the time. This patient later developed a granuloma of the larynx which was removed by our bronchoscopic department. I made no further efforts in secondary operations until after I had had a successful result in a case of laryngeal stenosis.[1]

This was the case of a young man 23 years of age who had a laryngeal obstruction with fixation of both arytenoid cartilages. He had been in the hospital some 76 times in the course of three years. He had considerable hypertrophy of the mucous membranes of his larynx and a thickening of his thyroid and arytenoid cartilages. It was thought that removal of one or both of his arytenoids might increase his airway. He was prepared for an arytenoidectomy, and

at the time of operation it was found that he had an old perichondritis causing obstruction. One arytenoid cartilage was removed but on laryngoscopic examination the amount of space gained was negligible. I then decided to remove all the cartilage from one side of his larynx and sew the internal perichondrium and the thyrohyoid muscle to the median surface of the pretracheal muscles, hoping that the formation of scar tissue and the fixation of this side of the larynx to the under surface of the muscles mentioned would widen the larynx laterally. The result was most encouraging. Two months following this operation he had sufficient airway to allow us to close a tracheal fistula which had existed since he was two years old—the result of necrosis of the tracheal rings following a diphtheria infection complicated by a tracheotomy. He was able to get a gainful occupation for the first time in his life.

With this experience to justify my again attempting to correct failures by operating on the same side, I re-operated upon two of the early failures within a short time—one a nurse from the South and the other a housewife from upper New York State. In the case of the nurse, I removed the right wing of the thyroid cartilage and carried out the procedure of suturing the internal perichondrium of the larynx to the pretracheal tissues, as I had done in the young man. In the housewife, in addition to removing the right wing of the thyroid cartilage, I did a thyrotomy and performed an intratracheal removal of the arytenoid cartilage on the opposite side. Removal of the arytenoid cartilage at this time was done because on laryngoscopic examination at the time of operation, we were unable to widen the larynx by pulling on the sutures put through the internal perichondrium. I thought that the intratracheal removal of the opposite arytenoid cartilage would give me more airway.

The results in these two cases were disappointing. I have not heard from the nurse since three weeks after her operation, which was last summer, and I take it for granted that her result was not satisfactory. However, the lady from upper New York State has been a most grateful and obliging patient, and in her case there was a perceptible narrowing of the larynx after this operation and the action of the inferior constrictor and cricopharyngeus muscles can be seen on indirect examination through the right side of her

larynx. The last time I saw her there was a distinct movement of the right side of the larynx each time she gagged or strained when I was examining her. The arytenoid prominence on the right side, which previous to the second operation remained firm in its position, now dropped forward toward the interior of the larynx. While both of these cases were between 50 and 65 years of age, which I believe is the unfavorable period for consistent good results, I am nevertheless convinced that we must conserve the integrity of the thyroid cartilage, as far as possible, for the success of any operation to relieve bilateral abductor paralysis of the larynx.

The one point that I did not take into consideration when drawing an analogy between the success that I had with the young man with laryngeal obstruction and the possibility of getting results on these two patients by following the same technique, was that in the young man I was dealing with an obstruction and not with a paralysis of the muscles of the larynx. Within the last three weeks I have done a secondary operation on a young lady who had two previous operations for the relief of her laryngeal paralysis at another institution. The first operation was done about three years ago by the King technique, and the second operation was done about six or eight months later, using the technique of the arytenoidectomy. She came to our clinic about a year ago and we told her that we thought she had sufficient airway to carry on without her tracheotomy tube, but she was reluctant to do this because of the distress that she experienced at night. She was becoming very nervous and felt that she could not hold a position. We finally persuaded her to let us remove the tracheotomy tube without closing the tracheotomy opening and encouraged her to seek employment, which she did. However, after following our advice for some months, she came to the hospital and said she was ready to have her third operation if I thought it would give her more airway. We did the usual arytenoidectomy on her left side. On exposure of the arytenoid cartilage it was noted that the cartilage was very large and after it was delivered, the left cord fell away from the median line, giving the patient sufficient airway without placing a fixation suture. However, in view of the fact that this patient had been operated upon on two previous occasions for relief of her paralysis, I was hesi-

tant about not putting in a fixation suture although it gave us more separation of the cords than we deemed necessary. There is no question of the success of this last operation from the standpoint of an airway, and I am hoping she will have a useful voice.

In closing I think there are two more problems which are worth mentioning at this time. One is the question of primary healing. I noticed that in two of our elderly patients the wounds did not heal readily. In one the wound broke down and several bits of cartilage were extruded. The wound healed with an ugly scar although the result was very satisfactory. In this particular case we were longer than usual in doing this operation and several times I could feel the heat of the two lights we were using through my rubber gloves but thought nothing about it. However, when in a second case of an elderly person who should have made an uneventful recovery there was trouble with healing, I began to realize that the heat from the lights we were using was drying out and devitalizing the tissues in these old people and preventing primary repair. I have now changed our procedure and we use one light until the thyroid cartilage is exposed and from there on the operation proceeds with the reflected light of a head mirror. We have had no breaking down of wounds in elderly people since making this change.

The other problem to be considered is that in doing this operation under local anesthesia and injecting the intratracheal tissues through the window made in the thyroid cartilage, care must be taken not to inject too much solution. If the tissues are flooded, it is more difficult to locate and extract the arytenoid cartilage and, in separating the cords, you may have too much separation when the fluid has been absorbed and the tissues return to normal.

These are some of the problems we have met in the surgical treatment of bilateral abductor paralysis of the larynx. If the experiences outlined here prevent the laryngologists who may be enthusiastic enough to undertake this work to avoid some of the embarrassment and pitfalls we have experienced, I will feel that our efforts have not been in vain.

140 East 54th Street.

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DISCUSSION

DR. MCCALL (by invitation): In any of the suggested procedures for the relief of this condition the most important point in the operation is the identification and fixation of the arytenoid cartilage. The relationship between the arytenoid and the thyroid ala varies greatly in different larynges. It is not an easy matter to determine just where to make the window in the ala.

To overcome this difficulty after we have exposed the thyroid ala we introduce the laryngoscope. The interarytenoideus is severed. The arytenoid is mobilized by disarticulation from the cricoid cartilage.

The thyroid ala is exposed. After that is done an anterior commissuroscope is introduced in the larynx. The end of the scope fixes the arytenoid cartilage and it is pressed laterally against the thyroid ala. After the room has been darkened and by transillumination it is a relatively easy matter to determine just where the window should be made.

The window is then made; and the laryngoscope is introduced in the larynx. The inter-arytenoideus is severed. The arytenoid is mobilized by disarticulating it from the cricoid and a full curved needle with full suture is introduced in the arytenoid cartilage.

The suture is then fed through the window in the ala and traction delivers the arytenoid cartilage.

After the suture is fed through the window which you have made, traction delivers the arytenoid through the window. It is then fixed to the perichondrium of the thyroid. We do not believe it is necessary to do an arytenoidectomy.

Of course, Dr. Kelly has had a lot more experience and a great many more cases than we have had.

DR. MERVIN C. MYERSON: There are many pitfalls in this work, but after one does a few operations, he comes to a decision as to what

his procedure should be. Even after a decision is made, one keeps trying to improve.

Two months ago, while working in the morgue, I decided there was no reason why one could not plicate the cord in a new position with a specially curved needle through a laryngo-fissure approach.

Dr. Imperatori suggested such a procedure to me a year ago. A suture is passed through the tissues including the thyroid cartilage and plicates the vocal cord in an outward position without arytenoid removal. This was done in two cases. In the first case unfortunately we operated on a patient who had had previous surgery. He developed a perichondritis with some necrosis of the cartilage. The second appears to have been successful.

As you all know, the thyroid cartilage of the female is much more vulnerable than that of the male; because of its thinness and its vulnerability, the incidence of infection of the cartilage is much greater.

The second case yielded a perfect result. If we can use a laryngo-fissure exposure and place a suture through the posterior end of the vocal cord, tying the suture in the subcutaneous tissues outside of the thyroid cartilage, I believe we are simplifying this apparently complicated procedure.

Dr. Thomas C. Galloway: Mr. Chairman, why should we remove the arytenoid in this operation?

One advantage of leaving it is that the procedure is then much more easily done. There is much less danger of penetrating the larynx and getting contamination with pharyngeal secretions, there is much less trauma to the cord structures, and there is left a guy-line including the arytenoid and the vocal process, through which one can insert a fixation suture and deliver the arytenoid. There is room for it in the window and it helps occupy that dead space.

As Seed pointed out, not only should the articulation be well dissected and the arytenoid mobilized but the suture should be placed anterior to the center of rotation of the arytenoid.

The direct laryngoscope I think also is an important aid. If one makes a small window in the thyroid cartilage with a curet, he

then can place a sharp hook quite accurately through the muscular processes of the arytenoid through the external window by watching perorally the movement of the arytenoid.

Dr. Francis L. Weille: I believe it was last year that I had the privilege of following one of these cases which had been done for us in Boston by Dr. Kelly.

The thing which puzzled me was why, afterward, with the excellent space he obtained in this particular case the patient did not aspirate foreign bodies or fluid. I would like, in view of Negus's idea of the physiology of the larynx, to ask Dr. Kelly if he would tell me why these patients do not do this.

I tried the patient with water the day after operation and he could swallow without any trouble at all and without coughing.

Dr. J. D. Kelly (closing): Answering Drs. McCall, Myerson and Galloway I would say that I have no argument with the man who gets results by another method and have said so in my last two papers on this subject.

Dr. Weille I can answer by saying that I discovered that the operation did not interfere with deglutition by giving my second patient a drink of water before he left the operating table. The operation was done under local anesthesia.

EXTRA-LARYNGEAL SURGICAL APPROACH FOR ARYTENOIDECTOMY. BILATERAL ABDUCTOR PARALYSIS OF THE LARYNX

HENRY BOYLAN ORTON, M.D.

NEWARK, NEW JERSEY

Laryngologists have long recognized that surgery of the neck, especially thyroidectomies, is one of the most frequent causes of bilateral abductor paralysis of the larynx. Other conditions may also cause abductor paralysis all of which are well known to the members of this association and time need not be taken here to enumerate them.

Surgeons and laryngologists for years have been devising methods whereby these patients could have relief from their condition. Today, some form of arytenoidectomy, with fixation of cord laterally, seems to be the operative procedure. I wish to place before this association, an extralaryngeal approach to the arytenoid.

Knowledge of the part in question is necessary to allow the surgeon access with the least disturbance, and to enable him to carry out his task with as little effect on function as may be.

Trotter,[1] after more than 20 years of experience, established the operation of lateral pharyngotomy by the transthyroid route, as a method of general usefulness in surgery of the pharynx and laryngopharynx. Unfortunately, this method, or slight modifications thereof, is not used as frequently as it should be; for instance, removing the lateral half of the hyoid bone enables one to have perfect access in removing a neurofibroma of the pharynx without contamination of the mucous membrane of the pharynx. Then by removal of the cornua of the hyoid bone and the lateral thyroid cartilage gives access to the laryngopharynx. And now by removing the posterior half of the thyroid ala gives adequate access to the arytenoid without entering the pharynx or larynx. The first step in the actual attack on the arytenoid itself must be proper exposure. Application of this procedure is now described.

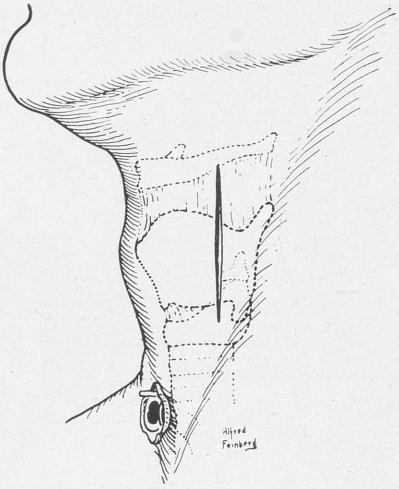


FIG. 1.—Shows the longitudinal incision over the lateral half of the thyroid ala.

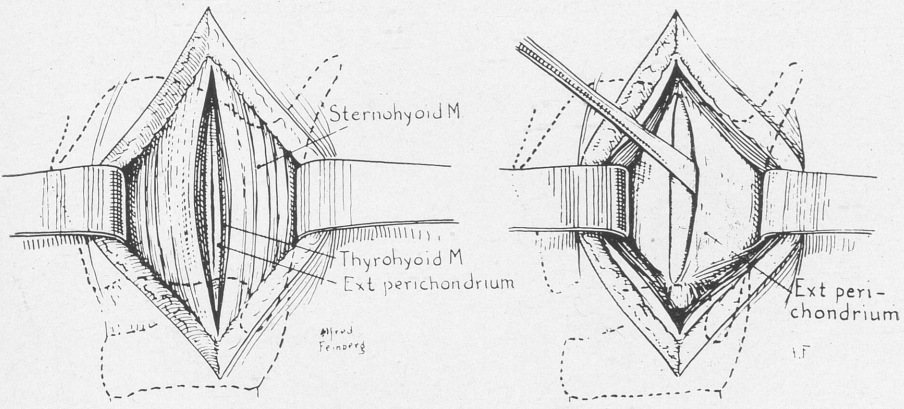
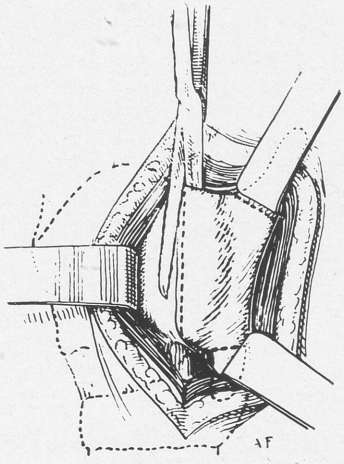


FIG. 2—Splitting of the sternohyoid and thyrohyoid muscles and incision of the external perichondrium in line of incision.

FIG. 3.—Elevation of the external perichondrium from the posterior half of the thyroid ala. This separation is carried around the under surface of same.



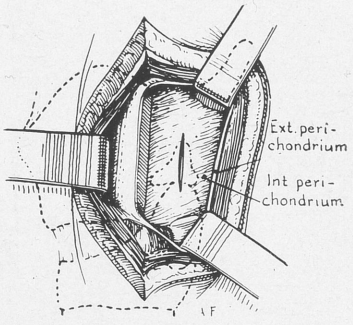
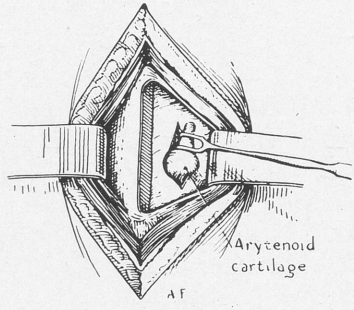


FIG. 4.—Shows the perichondrium entirely separated and the posterior portion of the thyroid ala being removed by heavy shears.

FIG. 5.—Shows the internal perichondrium and the pharyngeal aponeurosis, and by palpation the arytenoid can be definitely identified. It also shows the incision over the arytenoid through its muscular attachments.



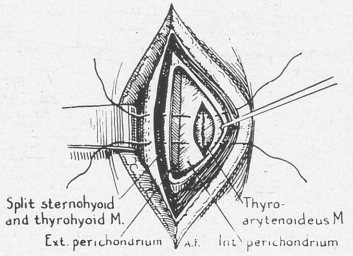


FIG. 6.—Shows the arytenoid cartilage being removed from its muscular attachments.

FIG. 7.—Shows the insertion of sutures, which has the tendency of pulling outward that side of the larynx.

Lateral Transthyroid Approach

Position of Patient: The position is the same as for a complete block dissection of the neck. The chin is rotated laterally and a small sand bag or pillow is placed under the shoulder of the side to be operated on; this position gives a flat surface to that side of the neck.

Anesthesia: May be either avertin, ether, or local depending on the operator.

Incision: After proper preparation of the surgical field, a longitudinal incision is made over the lateral half of the thyroid ala, on the side to be operated on extending downward from a point midway between the hyoid bone and thyroid cartilage to the level of the cricoid cartilage. The incision is through the skin and the platysma. The sternohyoid, thyro-hyoid and sterno thyroid muscles are split in the line of incision. The omohyoid muscle is lateral to the incision. With retraction laterally and mesially of the muscles, the perichondrium is incised in line of incision, the perichondrium with attachments of the inferior constrictor muscle is elevated from the posterior half of the thyroid cartilage. This separation is carried around under the surface of the thyroid cartilage. With complete separation of the thyroid cartilage from the external and internal perichondrium, the posterior lateral half of the thyroid ala is then removed by means of heavy scissors.

The removal of this portion of thyroid cartilage exposes the laryngopharyngeal aponeurosis and then by palpation at the lower angle of the wound the arytenoid is located. Incision is then made through the internal perichondrium on to the arytenoid cartilage, and by sharp dissection the entire arytenoid cartilage is freed from its muscular attachments and removed.

Two or three fine chromic catgut sutures are placed through the thyroarytenoideus muscle to the external perichondrium and the anterior split sterno-thyroid and thyrohyoid muscles. A small cigarette drain is inserted, fascia closed by two or three interrupted sutures and skin closed.

This cigarette drain is removed in three or four days. No feeding tube is used.

SUMMARY: The advantages of the procedure herein described are:

1. The lateral transthyroid approach gives access to a wider field of operation.

2. The longitudinal incision and splitting the sternohyoid and thyrohyoid instead of the transverse incision.

3. Direct identification of the arytenoid cartilage by palpation beneath the laryngopharyngeal aponeurosis.

4. Arytenoid is under complete vision during the entire arytenoidectomy.

5. A direct extra laryngeal surgical attack to the side of the larynx.

The procedure has proven very satisfactory and from letters I have received from other men who have followed this procedure their results have been very gratifying; their comments being the ease with which the operation could be accomplished.

I feel quite certain that the approach is sound and time alone will tell if arytenoidectomy is the solution to the problem of Bilateral Abductor Paralysis.

224 DELAVAN AVENUE.

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DISCUSSION

DR. J. D. KELLY: I have nothing but the highest praise for anything that Dr. Orton undertakes and if he can get results with the Trotter incision I think he should use it, but until I can see some of

these cases and compare them with the results which I have gotten by the use of the horizontal incision and the window made in the thyroid cartilage, I will not use the Trotter incision for the reasons outlined in my papers on this subject.

Dr. Lawrence R. Boies: I hestitate to report a single case but I feel I owe it to Dr. Orton because it was his technique that I used.

Three months ago I operated on a thirty-seven year old housewife. She was hospitalized for seven days. Her tracheotomy tube was removed at the end of one month. I saw her last week. She has not quite as good a voice as she had before operation. She has an excellent airway and is very happy over the result.

I was impressed with the relative simplicity with which I could expose and remove the arytenoid cartilage by this approach.

Dr. Mervin C. Myerson: I should like to amplify my previous discussion.

We all know that it is not so much the location of the whole arytenoid cartilage which controls the position of the vocal cord as it is the vocal process of the arytenoid. If, through a laryngo-fissure incision, the vocal process of the arytenoid cartilage is severed and then a suture is placed through the posterior portion of the cord, whether it includes the vocal process or not does not matter, and that structure is pulled outward by a suture which fixes it on the other side of the thyroid cartilage subcutaneously. I think we are accomplishing our objective.

I might add that a fish-hook type of needle is used.

Dr. H. B. Orton (closing): It is perfectly true that one may not have as good a voice after operations on the larynx, as previously. If you do a laryngo-fissure you do not obtain the same type of voice after the removal of the carcinoma as before.

The larynx gives the tone to the voice. The words are formed by the lips and the tongue.

Most of these patients are perfectly happy to have an additional airway and to be able to dispense with the tracheotomy tube.

Scar formation has a tendency to contract outward as long as we have not injured the internal mucous membrane of the larynx.

It seems to me there is a greater tendency for contraction outward than if there were not too much of the thyroid cartilage to interfere. This also applies to Dr. Myerson's remark.

Dr. Kelly and I are both trying to keep away from contamination by pharyngeal secretions. I am trying to do that by the extralaryngeal operation.

I feel quite certain the approach is sound and time alone will tell if arytenoidectomy is the solution to the problem of bilateral abductor paralysis.

CANCER OF THE LARYNX
REPORT OF UNUSUAL CASE

LOUIS H. CLERF, M.D.

PHILADELPHIA, PA.

The expectancy of life of a patient with untreated carcinoma of the larynx is dependent upon a number of factors. For that reason it is hazardous to express an opinion regarding the probable length of life. Few untreated patients survive for more than two or three years after a diagnosis has been established.

The following is of interest because of the original diagnosis and therapeutic recommendations, the subsequent course and duration of symptoms and the final outcome. The following history was secured from the patient's son, a physician.

Male, aged 77 years, consulted me at the Jefferson Hospital during October 1938. There was a history of hoarseness of over 11 years' duration with dyspnea of about one year's duration. During early 1927 the patient developed hoarseness and was referred to the late Dr. J. E. Mackenty by his physician during the latter part of October of the same year. Microscopic study of tissue removed from a tumor of a vocal cord was diagnosed as "an invasive form of epithelial new growth." Dr. Mackenty recommended laryngectomy but the patient refused this and returned home. He continued with his activities and was in charge of a general store for approximately ten years when he began to have dyspnea and was no longer able to carry on his work. Recently the dyspnea became marked and he finally decided to have some plan of surgical treatment carried out.

On examination the patient's general condition seemed excellent for one of 77 years. The outstanding physical findings were limited to the larynx. There was an extensive ulcerated lesion which involved the entire left vocal cord, extended subglottically for a considerable distance and invaded the anterior extremity of the op-

From the Department of Laryngology and Broncho-Esophagology, Jefferson Hospital, Philadelphia, Pa.

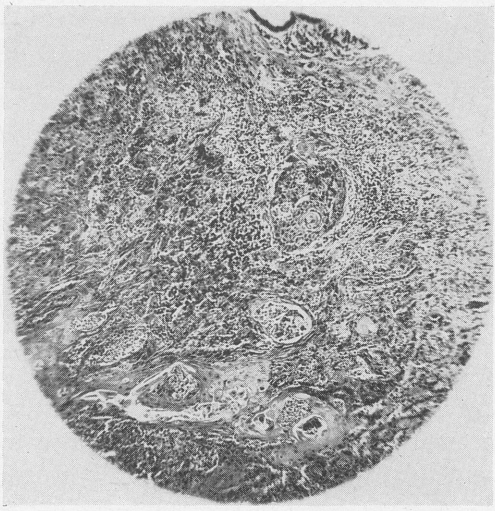


FIG. 1.—Photomicrograph (X83) from biopsy specimen exhibited a bit of mucosa and solid clumps of epithelial cells which are infiltrating. The epithelium is fairly well differentiated and there is formation of keratin (Dr. B. L. Crawford).

posite vocal cord. The left side of the larynx was fixed. The right vocal cord and subglottic tissues appeared edematous but there was no impairment of motility of that side. There was no demonstrable evidence of metatasis. Roentgenological examination of the chest revealed no evidence of pulmonary metastasis and systemic studies all were essentially negative.

A biopsy was done and additional tissue was removed to improve the laryngeal airway as there was marked stenosis. The histologic diagnosis by Dr. Baxter L. Crawford was papillary squamous-cell carcinoma, intermediate grade of malignancy (Fig. 1).

The question of therapy was discussed with the patient and his son. In view of the need of tracheotomy for dyspnea and the absence of any demonstrable metastasis to regional lymph nodes it was decided to do a laryngectomy. Because of subglottic involvement

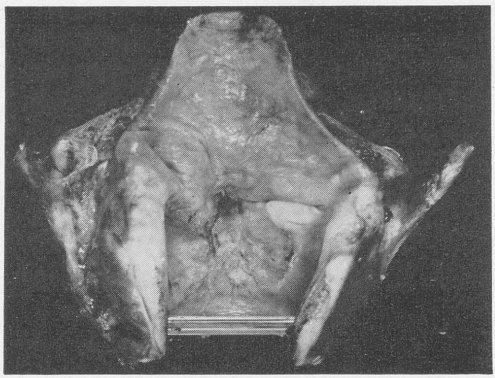


FIG. 2.—Specimen of larynx divided in midline posteriorly exhibited extensive involvement of the entire left vocal cord extending subglottically to the lower border of the cricoid cartilage, also involving the ventricle and anterior extremity of the left ventricular band and crossing the midline anteriorly involving the anterior third of the right vocal cord and corresponding subglottic tissues.

it was deemed advisable to remove the first and second tracheal rings; in addition, a large segment of hyoid bone was removed.

The larynx was divided in the midline posteriorly following removal and the interior inspected. The growth appeared as an extensive ulcerating papillary lesion. It involved the entire left vocal cord extending into the ventricle and involving the anterior extremity of the left ventricular band. It also extended across the midline involving the anterior third of the right vocal cord. There was marked subglottic extension below both cords, particularly anteriorly and on the left side where it extended to the lower border of the cricoid cartilage (Fig. 2).

The patient made a satisfactory postoperative recovery and returned home 24 days following operation. He has continued in excellent condition and there has been no evidence of recurrence now over 5½ years following operation.

Comment

The long interval from the time of the occurrence of symptoms and the original diagnostic studies in 1927 when laryngectomy was recommended and the time that it actually was performed, the comparative freedom from serious symptoms for a period of 10 years in the absence of any therapy with the later development of progressive dyspnea and increasing hoarseness, and the fact that laryngectomy still could be successfully performed 11 years after the original diagnosis was made immediately raise the question of diagnosis.

Should isolated cases of carcinoma which exhibit unusual behaviour be regarded with suspicion and should the pathological material be reexamined in the light of these changes? When the diagnosis of carcinoma was made in 1938 a communication was sent to the pathologist who made the original diagnosis in 1927 and the histologic material was reviewed. There apparently was no reason for changing the diagnosis and this was considered an unusual case. There is no question that the larynx exhibited an extensive cancer when removed in 1938. A more recent review of the original biopsy material by another pathologist suggested that the condition might be considered as a case of Bowen's disease.

Bowen's disease is regarded by many pathologists as a highly specialized form of superficial squamous-cell epithelioma with lateral intra-epithelial spread. It more often is observed in the skin than in the mucous membranes. Its transition into frank cancer more often occurs in mucosal than in cutaneous cases. Brighton and Altmann's case is the first reported instance of a laryngeal localization.

1530 Locust Street.

Reference

1. Brighton, G. R., and Altmann, F.: Bowen's Disease and Superficial Epithelioma of Upper Respiratory and Alimentary Tracts, Arch. Otolaryng. 36:354, 1942.

FIBRO-SARCOMA OF LARYNX

JOHN H. FOSTER, M.D.

HOUSTON, TEXAS

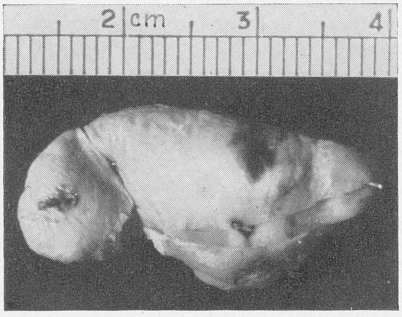
CASE 1.—H. W. A., age 31, was referred to me December 9, 1943 by Dr. F. H. Newton, of Dallas, Texas on account of a laryngeal tumor with considerable dyspnea. The patient, who had been discharged from the Army on November 23, 1943, was the son of a physician in Dallas and had taken one year in medicine before enlisting. The following is the history of his case as written by himself:

"The first sign of this tumor occurred on the morning of July 3, 1943 at Camp Gruber, Okla. After breakfast I coughed a medium hard cough and out flew something and landed on the floor. Upon examination it proved to be a piece of tissue. Hemorrhage followed immediately and lasted about 30 minutes. We were all ready to leave for the desert near Yuma, Arizona and left about an hour later. The only symptom prior to coughing this up was a slight rale on inhaling. The piece of tumor was about the size of an English pea.

"I felt all right and the doctor said it was a granulation. I had had a severe case of bronchitis at Camp Robinson, Arkansas, about 3 months before and he said it might be the healing of that. About the 12th of July I coughed up a piece again upon arising and it was slightly larger than the first. Again bleeding stopped in about 30 minutes and again the doctor said it was a granulation.

"On the 27th of July I coughed up my third piece and it was the one I turned over to Dr. Foster last week. I coughed this one up voluntarily. I couldn't breathe when I lay down and this had gotten worse for the 3 nights previous to the 27th. I sat up and made myself cough and after a few minutes out came the tissue and bleeding lasted again about 30 minutes.

"By this time the doctors realized it was not a granulation. My outfit happened to be the 123rd Station Hospital. I was put in the



Photograph of tumor coughed up.

hospital that night. Capt. Lanschner, the head of E.E.N.T. for the 123rd, examined me by indirect laryngoscopy the next day and also had me x-rayed. He said he could find nothing by either of these and sent me to Torney General Hospital at Palm Springs, California.

"Here I was x-rayed again and the site of the tumor found. On August 10th Capt. Moffett did a laryngoscopy and removed a piece of tissue for examination. He also removed what he said was a papilloma from my right vocal cord. On August 23rd he did another and removed the balance of the tumor. On September 3rd he did another but removed no tissue saying it was looking fine. I was sent home for a 30 day furlough and returned on October 7th. While I was gone a piece of the tissue was sent to the Army Museum in Washington, D. C.

"Capt. Moffett told me upon my return the results of their findings and that he had orders from the Surgeon General to remove the base entirely — endoscopically if possible but to do a laryngofissure if necessary. On October 11th he did a laryngoscopy and removed a small piece of tissue for examination. The next day he was transferred. Before leaving he told me he did not remove the base but believed it entirely possible to remove it endoscopically.

"Here Major Witter the Chief of E.E.N.T. took over. He did not examine me until about 3 weeks later. All during the time from

July 27th until last week my breathing was free and easy and I gained about 15 lbs. in weight. About November 1st the Major examined me by indirect laryngoscopy and said there was nothing there and that I would have to be watched for awhile. The next week they decided to discharge me but changed their minds and the next week examined me again by indirect method and said I was to return to duty.

"They sent in my charts for return to duty but the Chief of Surgical Service bounced them right back and ordered a laryngoscopy. He recommended discharge if my throat was all right. They did a laryngoscopy on November 13th and said there was nothing but a red spot at the site of the tumor. I was discharged on November 23rd. I felt fine and my breathing was normal. I had begun to cough up some dried greenish scabs which they said was probably dried postnasal drip.

"I caught a cold on the train coming home and it settled in my larynx. I thought nothing much of it until I had a slight hemorrhage about two weeks ago. I went to Dallas to see my father who is a doctor and Dr. F. H. Newton a E.E.N.T. specialist there. He recommended Dr. Foster and when I returned home from Dallas my breathing became more difficult so I reported to him right away which was last Thursday."

Examination by indirect laryngoscopy revealed a subglottic tumor that obstructed the air-way to such an extent that there was considerable dyspnea. It seemed to be attached anteriorly. Examination otherwise negative. An x-ray examination was made with the following report:

"There is a tumor measuring 2 cm. in diameter arising from broad base on anterior portion of trachea just distal to larynx with elevation of 1.5 cm. extending into lumen of trachea. The tumor mass is sharply circumscribed, having a somewhat irregular summit, containing no calcifications. The lumen of trachea is considerably narrowed.

"The lungs show normal aeration, free from infiltration or consolidation. The heart and mediastinal structures are within normal limits, bony thorax and pleural cavities negative.

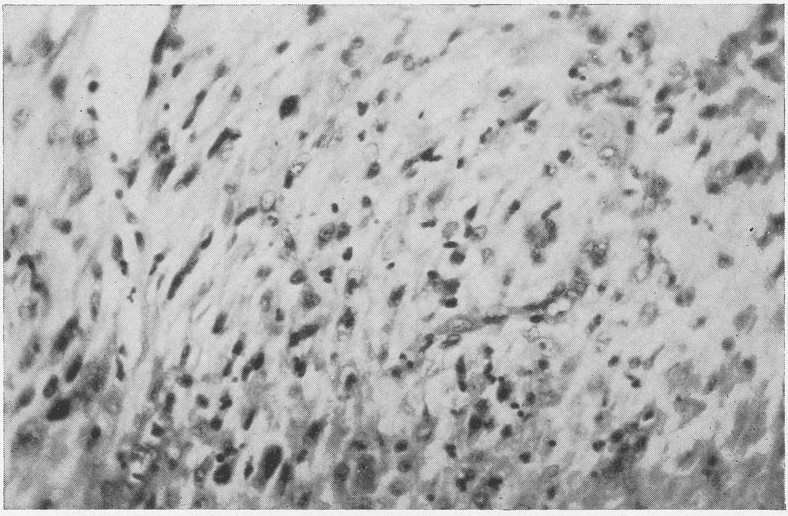


Fig. 2—Photomicrograph, Fibrosarcoma of the Larynx—High Power Section.

X-ray diagnosis: Tumor of upper trachea. Chest negative for pulmonary or skeletal metastasis or other pathology."

Patient had with him a tumor mass that he said had been coughed up on July 27, 1943. It was a smooth oval tumor about the size and shape of a large almond and showed a small area where it had formerly been attached. This was submitted to Dr. Violet Keiller who pronounced it a fibro-sarcoma.

On account of the dyspnea which had come on so rapidly immediate operation was advised, but this was delayed for two days in order for his father to come to Houston.

Operation: On December 11, 1943, under local anesthesia, the tumor was exposed by direct laryngoscopy and found to be attached to the anterior wall in the region of the cricoid isthmus. The greater part was removed with a snare and the base smoothed with punch forceps. The attachment was anterior and to the right and seemed

to include the cricoid isthmus and the tracheal wall for a short distance. There was comparatively little bleeding.

Patient suffered very little reaction and relief of dyspnea was complete. Three days later the application of a radium pack to the neck over the site of the tumor was begun. 50 mg. of radium were applied for 14 hours on three successive nights. My reason for the use of the radium was that although I had never before seen a fibrosarcoma in this region I had seen three in other locations that recurred promptly after thorough removal, but which remained well when removal was followed by the radium pack. One of these grew from the periosteum of the orbit and two from the periosteum of the bones of the face.

Healing in this case was uneventful except that the site of attachment showed granulations for some time. When healing was complete there remained a small smooth, slight elevation which is still present. He has reported regularly for observation and there has been no sign of recurrence.

The following are the pathological reports from: 1. Laboratory of Torney General Hospital; 2. Col. J. E. Ash, American Registry of Pathology, Army Medical Museum; 3. Dr. Violet Keiller, Houston.

1. *Gross Description*: The specimen consists of a piece of tissue measuring 2 x 1 x 0.7 cms. Part of its surface appears to be covered by mucosa which is smooth. The consistency is moderately firm. On cross section, it is composed of greyish-white tissue which is homogeneous in structure.

Microscopic Description: The sections show the mass to be composed of interwoven strands of spindle cells and fibers which are densely packed. The fibers are not well defined, they appear rather coarse and sometimes show suggestions of longitudinal striations. Often the fibers seem to form a synctium. The ends of the fibers are not clearly visualized but they seem to be pointed. The nuclei are large, oval in shape, and vesicular in type. The nucleoli are plainly visible and are frequently eosinophilic. The nuclei vary in size, shape and staining qualities but mitotic figures are rarely encountered. Amitotic division is often suggested and quite a few composite giant

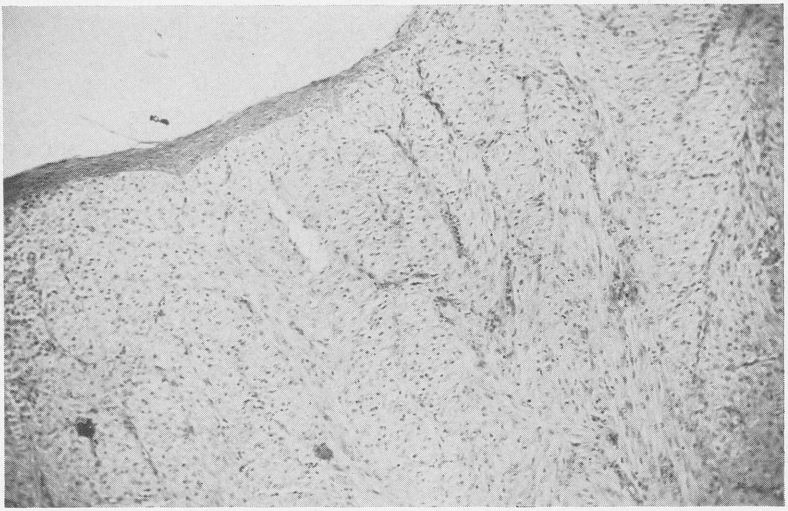


Fig. 3.—Photomicrograph, Fibrosarcoma of the Larynx—Low Power Section.

nuclei are seen. The vessels are moderately numerous and are of the capillary type. There is no necrosis. A few groups of lymphocytes are scattered about and polymorphonuclear leukocytes are not uncommon. The surface is covered with non-keratinizing stratified squamous epithelium which is stretched and thinned out; it appears intact except for a few places where it is replaced by fibrino-purulent exudate and granulation tissue.

P.T.A. Stain: adds nothing of note. No cross striations of the fibers are seen.

Note: The absence of clinical symptoms in presence of a mass of this size in the larynx seems very remarkable. Histologically the picture is that of a tumor of connective tissue origin which is quite cellular and actively growing. There is lack of invasiveness—as seen by the intact surface epithelium—but the irregularity of the nuclei, the mitotic figures and suggestions of amiototic division warrant close observation of the patient regarding recurrence of this growth.

Diagnosis: Cellular fibroma of larynx. Fibrosarcoma?

The Ward Officer requests that an opinion be given concerning the proper treatment for this condition.

2. In the case of H. M. A., Pvt., your S-622, A. M. M. Accession 97690, this is histologically a fibrosarcoma but it has been our experience that these tumors are not so prone to metastasize. Complete surgical eradication is the only recommendation that we can make as regards treatment.

3. *Gross Description*: The material consists of two specimens: the first of these is a tumor mass, brought by the patient, December 8, 1943, with the history that it had been coughed up in July, 1943. The second specimen is tumor tissue removed from the subglottic region by Dr. Foster, December 11, 1943.

The first specimen has been preserved in formalin and is in perfect condition. The tumor is a long ovoid, 2.3 cm. in length, with a curious knob turned up at one end, 1.5 cm. in height; this seems to have been the pedicle of attachment of the tumor. The surface is smooth, and the interior firm, rather soft, homogeneous, white from long fixation. Immediate section showed it to be a fibroblastic sarcoma.

The second specimen is in several pieces, the largest of them 1 cm. in diameter. The entire mass is estimated as 2 cm. by 1.5 cm. by 1 cm. or slightly larger. The tissue is greyish pink, homogeneous, soft but not friable; the appearance of the fresh cut section suggests sarcoma.

Microscopic Description: Section through the length of the first specimen, excluding the "pedicle", shows the tumor to be covered by thin stratified squamous epithelium, reduced by pressure in some areas to two or three layers of cells, showing no proliferative changes, and with clearly defined base. There is no ulceration of the surface, nor is there any inflammatory reaction in the depths of the tumor. This is composed of interlacing bundles of active young fibroblasts, in the form of long spindles, with long oval nuclei, of about the same size. Small vessels with thin but adequate walls are present in moderate number.

In the second specimen the relation of tumor to surface epithelium is similar to that described in the first specimen. Again there is no ulceration of the surface, and while in this case there is slight inflammatory infiltration throughout the entire mass, there is no tendency for the infiltrating cells to concentrate near the tumor surface.

The pattern of the tumor tissue has changed but little since the formation of the first tumor; the most noticeable alteration is in the blood supply. Many more small blood vessels are present and some of these are channels with imperfect walls. No alteration is observed in the rate of growth of the essential tumor cells, nor in the anaplastic character of these, although some increase in the degree of malignancy would have been expected as this is a recurrence, following operative removal after the spontaneous extrusion of the first tumor.

Diagnosis: Fibroblastic sarcoma of larynx, grade 2.

Comment: This case is reported for two reasons:

1. A fibro-sarcoma in this region is sufficiently rare to warrant reporting.

2. The use of radium following removal of fibro-sarcomata has been so satisfactory that I feel that it, too, should be recorded.

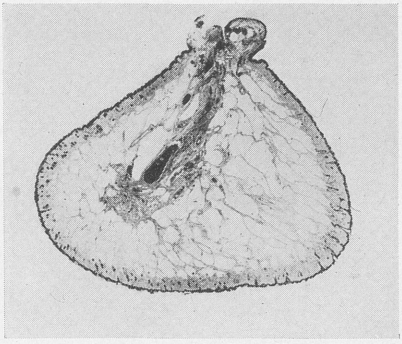


FIG. 1.—Section of Cyst (x3).

CONGENITAL DERMOID OF NASO-PHARYNX

JOHN H. FOSTER, M.D.

HOUSTON, TEXAS

CASE 2.—J. E. H., age 19 days was referred to me February 25, 1944 by D. A. J. Richardson of Jasper, Texas. The mother said that there had been some obstruction in breathing since birth and that in a short time she noticed that the infant got something in its mouth and suckled on it. The baby was taken to Dr. Richardson who discovered some sort of tumor in its throat and referred them to me.

Examination showed a well-nourished baby apparently well. On depressing the tongue a rounded tumor was visible attached to the posterior pharyngeal wall. It was freely movable on a small pedicle and at times disappeared into the nasopharynx; at others came into the mouth.

On account of the pedicle being so slender it was deemed safe to remove the tumor. It was grasped with forceps and removed with a snare. There was no bleeding of any consequence.

The tumor was sent to Dr. Violet Keiller who reported as follows:

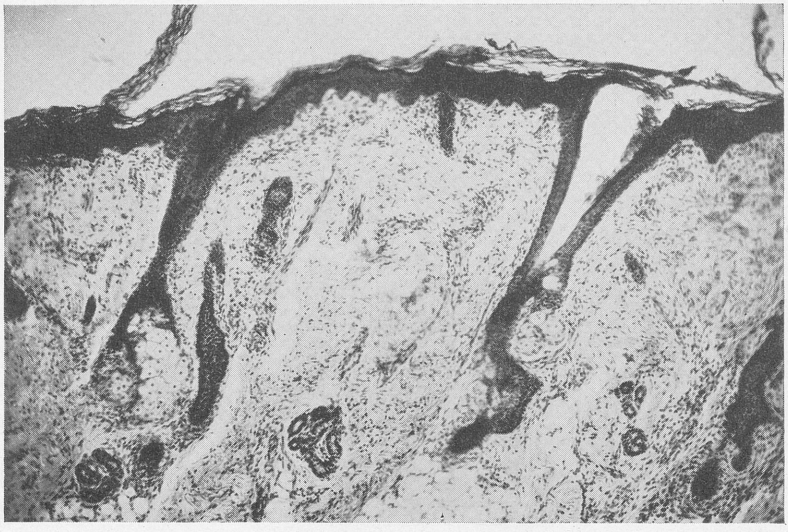


FIG. 2.—High Power Section.

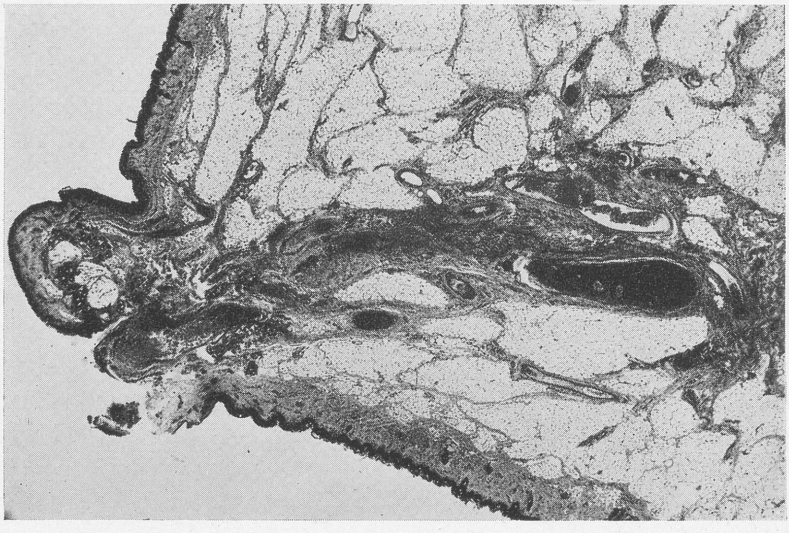


FIG. 3.—Section Through the Pedicle.

Gross: Tumor removed from posterior pharyngeal wall, about the level of the hard palate. It was connected with pharyngeal wall by a narrow pedicle, of rather soft tissue, 4 mm. in diameter, and 2.2 mm. long (as removed.) The tumor is bean-shaped- a rather fat bean, 1.5 cm. long, by 1.2 by 1 cm., and the pedicle is placed like the stalk of a bean but closer to one end than to the other. The pedicle seems to have a small hollow in its center. The surface of the tumor (but not the stalk) is delicate integument, with tiny pits visible with a hand lens, and minute hairs just perceptible to the naked eye. The specimen floats in water, and that and its texture suggest that most or perhaps all of the interior, is composed of fat tissue.

Gross: Tumor of naso-pharynx—reported in part Feb. 27; microscopic report delayed for celloidin sections through entire mass and pedicle.

Microscopic: Epidermis with a thick keratin layer, partly macerated, covers the tumor, except that at a small area near the pedicle there is, on one side of this, a covering of columnar "nasal" epithelium. Here there are a few mucous glands. In the tumor proper, sebaceous and sweat glands are present in orderly arrangement, and minute hair follicles are seen. Fat forms the greater part of the interior of the mass, and in the very center there is a slender bar of cartilage, pointing to the base of the stalk but not entering this. The stalk consists of fibrous tissue, with one or two bundles of nerve fibers—no ganglion cells—and with large, thin walled vessels. There is no evidence of active growth.

Diagnosis: Congenital naso-pharyngeal dermoid.

Comment: This case is reported simply as a rather anomaly found in so young an infant.

NEUROFIBROMA OF THE PHARYNX WITH PARALYSIS OF THE LARYNX FOLLOWING OPERATION

FREDERICK T. HILL, M.D.

WATERVILLE, MAINE

Neurofibroma of the pharynx must be considered as occurring infrequently as the literature contains few reports of such cases. Figi,[1] in his report in 1933, quoted Penfield as suggesting the term, "perineural fibroblastoma" for tumors variously classified as solitary neurofibroma, neurinoma, fibroma of nerves, gliofibrosarcoma, peripheral glioma, schwannoma and acoustic neuroma. Cases have been reported by Motley,[2] and by Hall and Owens,[3] both in 1941, and by Foster[4] in the Transactions of the American Laryngological Association in 1942.

This case is of interest because of the occurrence of a post-operative laryngeal paralysis.

Case Report.—A. P., a young woman of 20, was seen because of obstruction and feeling of fullness in the left side of the throat. She had had a tonsillectomy 8 years before. Just recently she had been admitted to another hospital with a diagnosis of peritonsillar abscess.

Examination disclosed a marked bulging of the left lateral wall of the pharynx extending from the level of the base of the tongue, upward behind the soft palate, almost to the toris tubae. This was rather firm to palpation. Laboratory studies and x-rays of the neck and nasopharynx were negative. The preoperative diagnosis was fibroma or mixed tumor.

Under ether anesthesia the mass was exposed by a longitudinal incision and a large encapsulated tumor, extending nearly to the base of the skull, removed by blunt dissection. This was about the size and shape of a hen's egg, measuring 6 cm. by 4 cm., and was of

FIG. 1.—Photomicrograph of section of tumor; x70. The cells are arranged in regimental formation or palisades, typical of the cells of the neurilemma or Schwann sheaths of the fibers.

yellowish-white color. The incision was closed with silk, and healed promptly.

Histopathological examination (Dr. J. Gottlieb), showed "Neurofibroma or neurinoma, resembling an acoustic nerve tumor. Can be considered as a pure Schwann cell tumor."

Drooping of the left eyelid and a contracted pupil, indicating involvement of the cervical sympathetics, were noted on the second post-operative day. These subsided in two days.

Of more significance was the finding of a left abductor laryngeal paralysis. The patient complained of a weak voice and examination of the previously normal larynx, now disclosed the vocal cord motionless in the mid-line position. I felt that this might be a temporary condition, possibly due to pressure or hemorrhage, but the condition has persisted now for a period of 8 months, the only

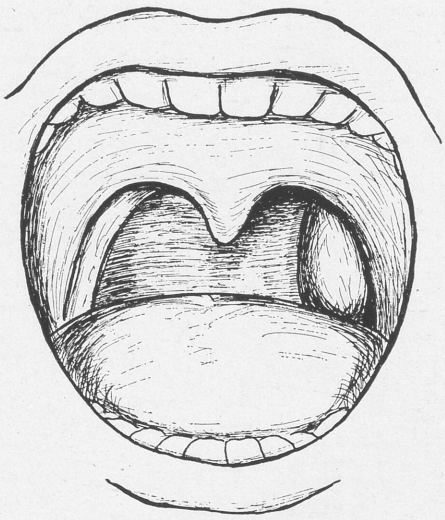


FIG. 2.—Drawing of Tumor of Pharynx.

change being a shift of the cord to the paramedian, or cadaveric position. There is no anesthesia of larynx. She is otherwise perfectly well and has no complaints except for a rather weak voice.

Comment: The findings suggestive of Horner's syndrome, similar to that noted in Foster's case, were due probably to the post-operative reaction, as they promptly subsided. The post-operative development of laryngeal paralysis suggests the vagus nerve origin of this tumor, probably adjacent to the jugular foramen, as palpation of the post-operative cavity revealed it to extend nearly to the base of the skull. Only a portion of the efferent fibers were involved as there was no paralysis of the soft palate or pharynx, nor anesthesia of the larynx. The first finding of the vocal cord in adduction, with a later change to the cadaveric position, suggests progressive involvement, first of the more susceptible abductor fibers, followed later by the adductors.

Possibly operation by means of an external approach would have resulted in less trauma to the vagus nerve. While the intra-oral

procedure was not especially difficult, it was necessary to exert some traction by applying a tenaculum to the tumor. However, the neurological origin of the tumor was not established until after operation, and with the thought that it was a mixed tumor or some simple fibroma, there was a natural inclination to avoid an external scar.

The present laryngeal picture is typical of the usual recurrent laryngeal nerve paralysis, with the vocal cord in the para-median position and the arytenoid slumped forward. It suggests the advisability of keeping in mind that such findings may, at times, be due to pathological conditions at the base of the skull, as well as in the neck or mediastinum.

PROFESSIONAL BUILDING.

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1. FIGI, F. H.: Solitary Neurofibroma of the Pharynx, Arch. Otolaryn. 17:386, March, 1933.

2. MOTLEY, F. E.: Positive Malignancy, Arch. Otolaryn., 34:771, Oct., 1941.

3. HALL, C., and OWENS, H.: Solitary Neurofibroma of the Pharynx, Arch. Otolaryn. 34:1163, Dec., 1941.

4. FOSTER, J. H.: Ganglion Cell Neuroma of the Pharynx, Trans. Amer. Laryn. Assn., 1942.

DISCUSSION

DR. J. H. FOSTER: The appearance of this case is very similar to one that I operated on.

In my case the tumor shelled out perfectly easily. I did not even have to catch up a bleeding spot. There seemed very little trauma. However, my patient had Horner's syndrome for a few days afterward, but no laryngeal trouble.

DR. ANDREW A. EGGSTON: This most interesting case of Dr. Hill's is an exact duplicate of one in the service of Dr. Nigro. This case was in a woman of 30 years. There was an olive-sized mass in the left wall of the pharynx behind the posterior pillar. This patient had paralysis of the left vocal cord and Horner's syndrome following the removal of the mass.

Fibro-neuromas have recently been restudied by Masson and Stout and others by means of tissue culture and special stains. Formerly they were considered more of a fibrous connective tissue but now have been more intimately related to the cells of the neurilemma or sheaths of Schwann of the nerve fibers themselves. Therefore, they are now spoken of as schwannomas or neurolemmomas. They are related to the more fibrous types as seen in Von Recklinhausen's disease. Histologically in the cellular types the cells are arranged in regimental formation or pallisades. They are usually spindle shaped with long fibrillae which run in a parallel way as contrasted with the fibrillar interlacing or reticular formation of connective tissue.

There is a second type however where the reticular net work predominates and there is quite an edema of the structures. This type is illustrated by a tumor which was sublingual. There the reticular bulging or swelling of the nerves can be seen on the side or circumscribing the remaining normal axis cylinders.

These tumors rarely recur or metastasize. However, their position upon some nerves may result in some functional abnormalities after removal. In the case of the pharyngeal growth the laryngeal paralysis has now persisted over a year. I am sure more of the tumors will be recorded as their true nature is recognized and reported, as Dr. Hill has so nicely done in this case.

Dr. R. S. Moore, Syracuse, N. Y. (by invitation): I had one of these tumors of the larynx about fifteen years ago and my purpose is to find out if any of the other men have had any of these tumors inter-laryngeally. The diagnosis was made by Dr. Weiskotten, our pathologist, and Dean of the Medical School.

Because of the rarity of neurofibroma in the larynx he sent slides to Dr. Mallory in Boston and his diagnosis was the same. The tumor was removed by laryngofissure.

I have seen the patient through the last fifteen years and there never has been any recurrence.

ATRESIA OF THE PHARYNX

Operated Upon by the Mackenty Method

CHARLES J. IMPERATORI, M.D.

NEW YORK

J. A., male 3½ years of age, was first seen in October, 1943.

Chief symptoms were some slight difficulty in swallowing, noisy breathing, inability to breathe through his nose and a muffled voice. All these symptoms followed within a few days an operation intended to remove this child's tonsils and adenoids. Since last June, he has neither gained in weight nor stature.

Physical Examination

Weight: 30 lbs.—13.5 Kg. Height: 40 in.—1.016 meters.

Nose: On inspiration alae dilate, while on expiration there is no expiratory blast. Very small amount of mucoid discharge in the inferior meatus, both sides.

Mouth: Teeth and gums normal. Tongue normal. Normal amount of saliva in the mouth.

Oropharynx: Not visible, for there is a web of scar tissue that stretches from the hard palate, including the soft palate and uvula, in a downward direction to the posterior wall of the lower part of the oropharynx, approximately at the level of the dorsum of the tongue, when depressed. No landmarks could be identified in this web of tissue. At the lower part near the junction with the pharyngeal wall, there was a small fistula into which a fine probe could be inserted upwards for approximately 1 cm. When the sutures were in place and traction made, the outline of the uvula could be seen.

Ears: Drums were considerably thickened.

Examination by direct laryngoscopy showed that the epiglottis and laryngeal structures were normal. Wassermann negative.

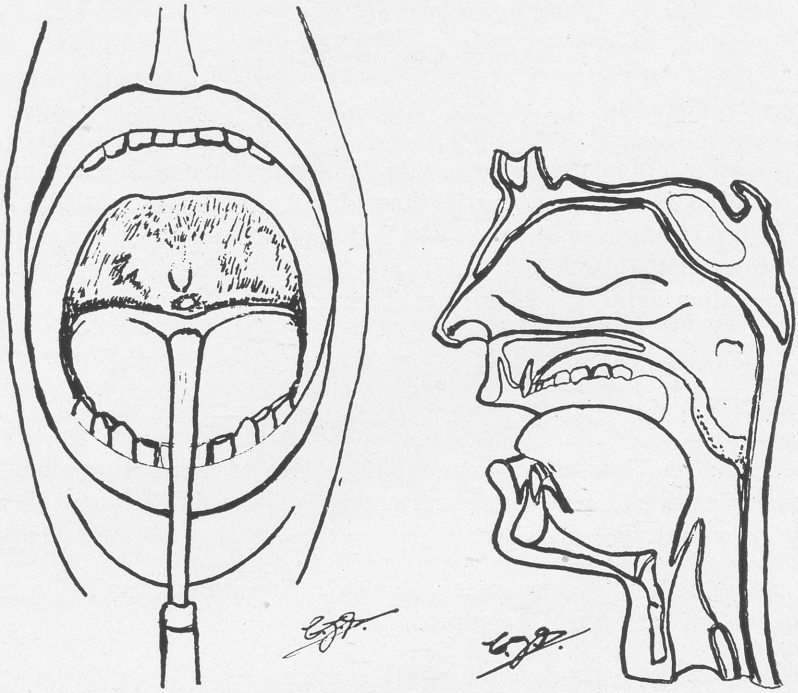


FIG. 1.—This illustration shows the scar tissue stretched across the oropharynx. The uvula can be just outlined imbedded in it and just below it is a small fistula, that, on probing, leads in an upward direction. Because of the irregular course, it did not permit of admittance of the probe into the nasopharynx. However, occasionally, an air bubble could be seen in the opening of the fistula.

FIG. 2.—Lateral view (diagrammatic) of the atresia in relation to the other structures.

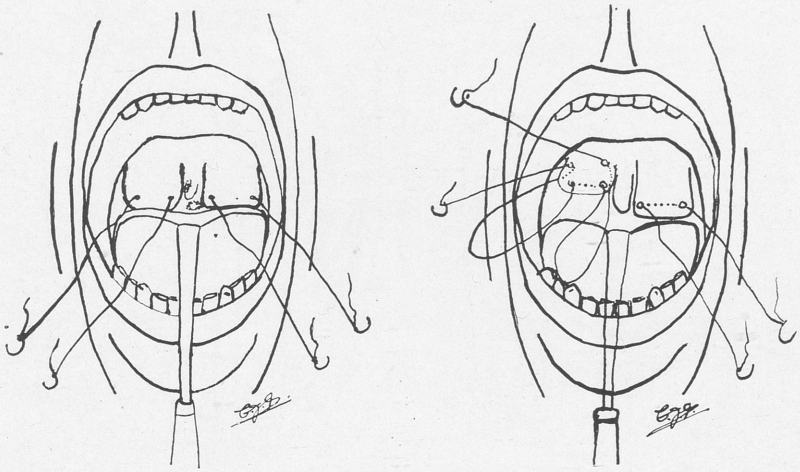


FIG. 3.—Suture retractors are placed in the lower part of the atresia just above its attachment to the lower pharyngeal wall. The suture material should be of a material similar to silkworm gut. In placing them, pass the needle, preferably a No. 2 curved, in a needle holder that will hold the needle at an angle of 45 degrees. Pass the needle through the atresia from within outward and into the depth of the atresia sufficiently that the posterior surface is included. The sutures should emerge near the posterior pillar or what was that anatomical entity.

The suture is then armed with a needle of the same size just used in its proximal end.

This procedure is repeated on the left side and the "U" shaped flaps cut, as illustrated.

The sutures are used as retractors and the adhesions are separated, preferably by sharp dissection.

FIG. 4.—The sutures are now introduced from behind the soft palate and the flaps drawn into the nasopharynx and anchored to the nasopharyngeal surface of the soft palate.

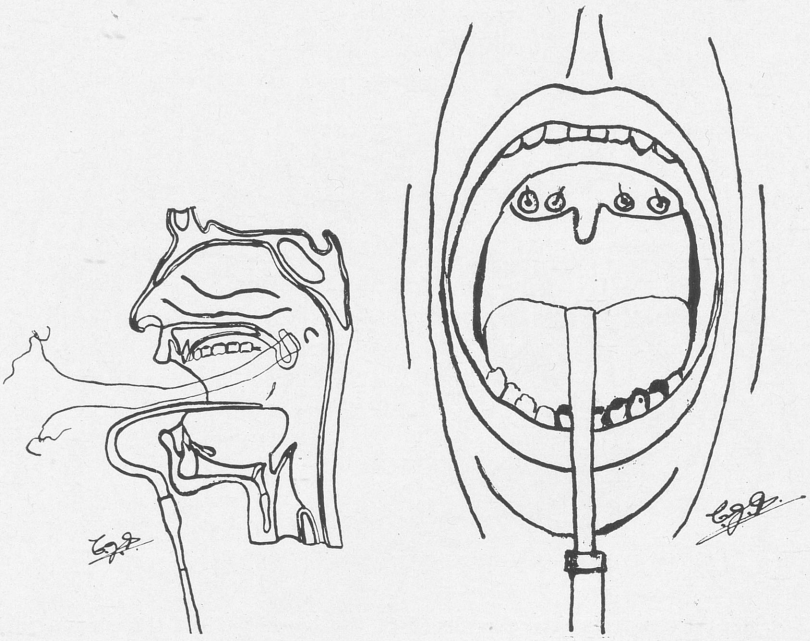


FIG. 5.—This will illustrate a lateral view of a flap drawn up.

FIG. 6.—Completed operation with the sutures anchored with split shot. (It is important not to draw the sutures too tightly or the shot will produce a pressure necrosis.)

Diagnosis: Pharyngeal obstruction produced by a web of scar tissue closing off the oro and nasopharynx.

Operation: November 16th, 1943, followed the procedure as described by Dr. John E. Mackenty in the Transactions of the American Laryngological Association, Vol. 1926, pg. 131. There is a very good description of this operation in the book entitled, "Diseases of the Nose and Throat by Wright and Smith. Lea and Febriger, Phil. 1914, pg. 651."

In dissecting the flaps, as described by Mackenty, the uvula and most of both tonsils were found imbedded in the scar tissue. There was a very prominent projection from the body of the second cervical vertebra and to which most of the scar was attached posteriorly.

The present condition (May 4th, 1944) of this child is very satisfactory. Weight: 35½ lbs.—16.10 Kg. Height: 41.5 in.—1.054 meters.

In order to keep the opening from inverting, particularly on one side, it was necessary at the weekly and later monthly return examinations, to dilate the opening. This was done with a Kelly clamp,—the ends of which were covered with rubber tubing.

The nasopharyngeal opening is approximately two-thirds that at the time of the completed operation. Voice is good, breathing appears to be normal and the expiratory blast from the nostrils appears to be normal, so that there is no difficulty in blowing his nose.

The type of operation that was done by the physician, was not known but it is assumed that the injury to the tissues, most likely, was produced by the use of a curette. Formerly, these injuries were common. Undoubtedly, those individuals, having a prominent tubercle on the atlas or a very prominent body of the axis or more specifically the edge of the lower surface of the odontoid process, were more likely to have this sequel and particularly so when the curette, such as the Gottstein or its modification, the Beckman, was used. Since the introduction of the LaForce adenotome, these sequelae are infrequent.

In examining the reports of the Manhattan Eye, Ear & Throat Hospital, where approximately 100,000 adenoid and tonsil operations have been done in the past ten years, there are but three cases recorded of atresia of the pharynx. Two of these patients were adults and one a young child,—two followed an adenoid and tonsil operation and the other followed a severe throat infection. No details as to the type of tonsil operation are given and no note as to Wassermann reaction, is recorded. In two patients, the Seton type of operation was done, and on the other no details of the operative procedure, were given. No record of final results.

One would conclude that this sequel following an adenoid and tonsil operation is comparatively rare, when the accepted technique is practiced.

108 E. 38TH ST.

DISCUSSION

DR. THOMAS E. CARMODY: There are two points which seem to me to be quite important.

Dr. Mackenty's operation was quite ingenious and I remember when he read the paper. While I have not used that exact method, I have used plastic procedures in these cases and I have seen quite a number of them.

As Dr. Imperatori says, the Gottstein curette of course took off some of the vertebral body. However, it is not the odontoid process because the odontoid process of the axis or second vertebra goes up through the atlas, so probably you could not take out the odontoid process.

I saw one of these cases in the office only a few days before I left home. A plastic operation can be done by taking a flap from the cheek or a flap from the oral side of the palate and turning it under so as to get the raw surface covered. However, that does not cover the raw surface on the posterior wall of the pharynx which must be covered by a split skin flap.

I also remember a case which one of the doctors said was a very peculiar adenoid, because it had bone in it.

PRESIDENT IMPERATORI: With the permission of the Association, I would like to clarify the statement that Dr. Carmody made.

The odontoid process does extend upward. It is really the body of the second cervical there being no body of the first. It means tooth or long tooth, and the lower and anterior part of the odontoid process in a great many cases projects into the pharynx.

St. Clair Thompson speaks about the first cervical. I doubt that very much. It is either the second or the third cervical which is very prominent, and there may be an enlargement of the second cervical

tubercle, or of the third, and the result is the projection anteriorly. When a curette is used with the head extended the result may be to include bone and with the additional injury to the soft parts produce the scar resulting in the atresia.

DR. CARMODY: That is not the odontoid process.

PRESIDENT IMPERATORI: I believe that it is the lower anterior part of the odontoid process, or the tubercle on it.

DR. GORDON BERRY: I am quite sure that I read a reprint by Dr. Rowe of Rochester giving that plastic procedure. I think it may go further back than that but it antedates Dr. Mackenty.

CONGENITAL LYMPHANGIOMATOUS MACROGLOSSIA WITH CYSTIC HYGROMA OF THE NECK

D. M. LIERLE, M.D.

IOWA CITY, IOWA

This presentation consists of a brief discussion of congenital lymphangiomatous macroglossia, with cystic hygroma of the neck. It includes a report of one case which has been treated, and two more which are receiving treatment at the present time.

Cystic hygroma may be congenital or acquired. Its origin frequently is in the submaxillary region, and it may involve the face, the floor of the mouth, and the tongue. The essential pathology consists of a dilatation of the lymphatic spaces. This dilatation is due to obstruction caused by the absence of efferent lymph vessels, or by lymphatics which do not have patent channels. Sich believes that the origin of lymphangiomas is in an isolated vessel or a connective tissue primitive rest which perforates into new lymph vessels. The hygroma usually consists of multilocular cysts lined with endothelium, with fibromuscular walls. These cysts are usually translucent and contain clear lymph fluid, but they also may have milky or chocolate-colored fluid with cholesterin.

Sections of the tongue in lymphangiomatous macroglossia reveal excess fibrous tissue, lymph vessels surrounded by enlarged vessels containing coagulated blood and lymphadenomatous new growth. Fibrous tissue, according to Buttin,[1] may cause muscle fibers to degenerate by pushing them aside and exerting pressure upon them. Consequently only fibrous tissue and large lymph spaces containing blood and lymph may be seen.

The cases in this series were three girls, eleven months, two and three years old, respectively, when admitted to the hospital. All had varying degrees of macroglossia at birth. Two had associated cystic hygroma of the neck at birth. According to the history of the third,

involvement of the neck did not occur until several months after birth. Just previous to hospitalization, the macroglossia and the cysts of the neck began to increase rapidly in size in each case.

Case 1 was first admitted to the hospital at the age of 11 months. The tongue was so large that it could not be retained in the mouth. The obstruction was so great that there was marked difficulty in eating and breathing. The mouth was constantly open, and there was dribbling of saliva. The lower jaw was deformed and the lower lip slightly hypertrophied. The tongue, exposed to the air, had become hard and fissured, and there was hypertrophy of the epithelium. Several areas of bluish discoloration could be seen and there was superficial ulceration of the mucous membrane, causing considerable discomfort.

The treatment consisted of surgical removal of the tumor of the neck. This appeared to arise from the left submaxillary region. It involved the floor of the mouth and extended into the base of the tongue. Since x-ray treatment in angiomata causes swelling of the endothelial cells and a degeneration of the elastic fibers and muscle with a replacement by fibrous tissue, the tongue was treated with divided doses of x-ray. A total of about 3000 roentgens, divided into doses of 400 to 500 roentgens, was used over a period of about four months. For only a short time, there was a definite decrease in the size of the tongue. Later it increased somewhat in size.

Later, a plastic operation was done on the tongue. The anterior portion was amputated and a large wedge-shaped section was removed, so that the tongue was practically normal in size. Microscopic sections revealed the absence of lymph spaces, but there was degeneration of muscle with replacement by a large amount of fibrous tissue. There has been no recurrence in a period of four years.

Cases 2 and 3 are in the hospital at the present time receiving similar treatment.

University Hospital.

References

1. Buttin, H. T., and Spencer, W. J.: Diseases of the Tongue, Cassell & Company, Ltd., 1900.

DISCUSSION

PRESIDENT IMPERATORI: I have never seen a case of hygroma cysticum colli complicated by macroglossia. There is a case which I reported, and apparently cured, in the 1940 Transactions of this Society. In that particular instance the child was suffering so much from dyspnea that the only way it could breathe was with its head in extreme extention, because of the compression of the mass of lymphoid tissue in the neck.

The treatment was to aspirate the various masses of lymphoid enlargement and inject a sclerosing fluid. The same kind of fluid that is used in the treatment of hydrocele was used. The child recovered and a year later was growing normally.

A CASE OF MALIGNANT RECURRING NASAL HEMORRHAGE OF UNDETERMINED ETIOLOGY

SAMUEL SALINGER, M.D.

CHICAGO

I am reporting a case of daily recurring epistaxis in a woman of twenty-three, which has been under my observation for the past three years. Despite every possible pathologic and hematologic test to which I and my colleagues have subjected her, we have been unable to arrive at a satisfactory diagnosis.

I first saw the young lady in July, 1941, when she gave a history of nosebleeds alternating between the two sides over a period of years, increasing in frequency until they were a daily occurrence. Examination at that time revealed a hyperemia of both Kisselbach areas. Despite repeated cauterizations, the bleeding continued. Within a month or two an ulcer developed on the right side of the septum which bled repeatedly despite frequent applications of the cautery and soon eroded through the septum. The nasal chambers were otherwise normal as was the blood examination. In December, 1941, a submucous resection was performed, which was attended by average bleeding. Since the hemorrhages continued, she was referred to an internist who reported only a moderate degree of secondary anemia. X-ray examination failed to reveal any abnormality. Radon paste was packed into the nose several times without result. It was noted at this time that the septum on the right side posterior to the perforation was spongy and swollen. Biopsy from this region was reported as "fibrin with many polymorphonuclear leucocytes; no cells seen." She was given large doses of cevitamic acid and vitamin K without result. Since the hemorrhages persisted, it was decided to try radium. She received a number of radium treatments within the nose totalling 600 mg. hrs. X-rays and planigraphs taken in April, 1942, disclosed an abnormality in the right maxillary sinus. A Caldwell-Luc was performed revealing a very small antrum confined mostly to the nasal side and

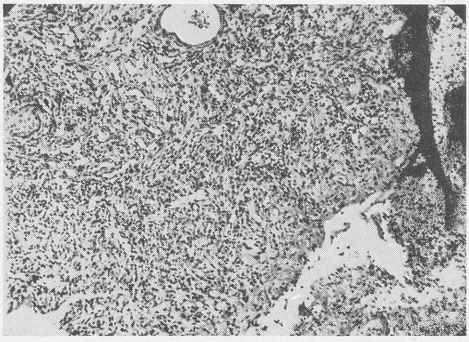


FIG. 1.—Section from septal ulceration. Granulation tissue with foci of necrosis; no evidence of malignancy.

containing a mass of spongy tissue which was thoroughly cleaned out. Histologic examination disclosed "chronic inflammation with granulation tissue." The sinus healed promptly, but the hemorrhages persisted and the granulomatous area in the right side of the septum was still present. Biopsy was reported as "granulation tissue with foci of necrosis." She was given an additional 300 mg. hrs. of radium intranasally. The bacteriologic reports were negative for non-aerobic bacteria. Guinea pig inoculation for Tb. was also negative. We assumed then that the hemorrhage was due to self trauma, and referred her to a neuro-psychiatrist who was unable to confirm our suspicion.

It is interesting to note that despite repeated examinations we were never able to pick out a definite bleeding point, nor was it possible to induce hemorrhage except by direct trauma with a metal instrument.

Another interesting point is the fact that the patient frequently complained of a throbbing sensation in the bridge of the nose, and at times exhibited a cyanotic swollen area which was tender to the touch and which at times would disappear completely. Radium was again applied, and an atrophy of all the nasal structures was beginning to be manifest.

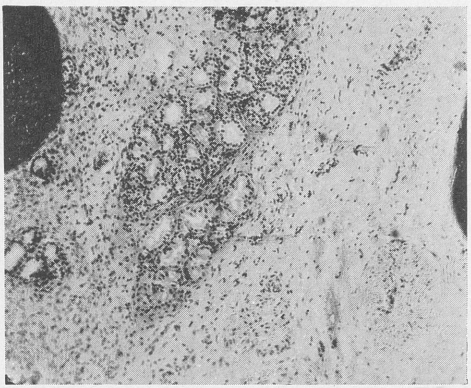


FIG. 2.—Tissue from maxillary sinus. Chronic inflammation with granulation tissue.

The patient was hospitalized several times in 1942 and 1943, and was examined and treated by our hematologist, Dr. Raphael Isaacs, who reported nothing abnormal in the blood except for a marked secondary anemia.

On March 6, 1943, we removed a wedge of skin, bone and mucous membrane constituting the swollen nasal prominence, and submitted the tissue for histologic and bacteriologic study. The report was "organization tissue with periosteal and endosteal fibrosis and new formation of bone." It was negative for tuberculosis, sarcoid, carcinoma, etc. The cultures were reported as staphylococcus, hemolytic streptococcus and anhemolytic streptococcus and staphylococcus.

While in the hospital a search was made among her effects, and on two occasions a safety pin was found which on being tested with benzedrine gave a positive reaction for blood. The patient denied self trauma, and repeated examinations by our neuro-psychiatrist, Dr. Gitelson, failed to elicit the information we desired. It was noted now that the evidences of trauma were repeatedly seen on the left inferior turbinate and the posterior margin of the large septal perforation, the only two areas readily accessible to a right-handed

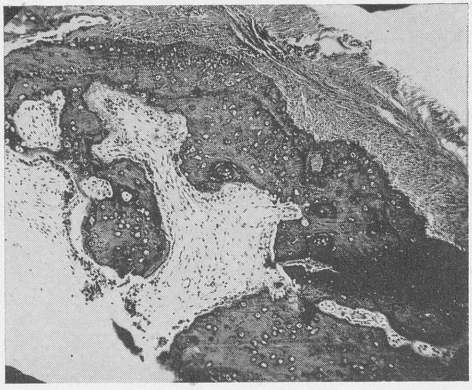


FIG. 3.—Tissue from nasal dorsum. Organization tissue with periosteal and endosteal fibrosis and new formation of bone.

person. The wound over the nasal bridge healed and reopened frequently. When sealed with a metal cap it would heal completely, but would reopen when exposed again. Examination of this lesion by our dermatologist was reported as simple ulcer of probable traumatic origin.

At times the nose was packed with gauze saturated in zinc peroxide and the entire nose covered with a metal cap for two to three days at a time. During such periods there was no bleeding.

Despite a number of transfusions the blood count has remained low (Hg. around 50% and R. B. C. 3,000,000). The patient has developed an allergy to blood and now has to depend on hematinics and liver.

We are convinced that the bleeding is the result of self trauma, but are unable to obtain a confession from the patient. She is definitely not a schizophrenic and thus far can be classed merely as a hysteric with strong fixations. We feel certain that we have eliminated tuberculosis, lues, malignant granuloma, sarcoid, Osler's disease, carcinoma, sarcoma, anaerobic infection, lupus, mycotic infection and all blood dyscrasias, and can only conclude that the case

is one of self-inflicted trauma based on as yet undisclosed psychogenic impulses.

Report of Psychiatrist

"This patient has been studied for nearly two years to begin with by ordinary interview techniques. Blood pressure readings were taken during a number of the interviews and revealed an average increase in the systolic pressure from 120 to 140 at certain points in the interviews. But no bleeding occurred at these points. There were two interviews conducted with the patient attached to a Keeler polygraph. Significant increases in the pulse pressures were noted during these interviews at pertinent points in the interrogation. But no bleeding occurred at these times. A sodium amytal interview was finally conducted and at this time it was made clearly evident that the patient bore great hostility to her father who was a drunkard and whose practice it had been in her childhood to tweak her nose during moments of erotized friendliness towards her. She recalled great fear of her father when during his periods of intoxication he would pursue her and her mother with a knife.

"Because of this evidence of specifically connected hostility and anxiety and because of the failure to elicit this hostility in face to face interviews I have been attempting to carry on a psychotherapy of the patient under hypnosis since January 18, 1944. This was undertaken in the hope that she might be better able to ventilate her feelings with the critical functions of the ego suspended. She has now been hypotized over a period of six weeks.

"As is obvious from the clinical facts, there has been no effect of this treatment on the patient's major complaint. Interesting discoveries have been made, for example, that the patient adheres as rigidly to her defensive symptoms under hypnosis as do patients normally without benefit of hypnosis. This is contrary to the general impression that hypnosis is supposed to reduce the resistance of the individual. Certainly, this girl is the most resistive individual I have ever encountered despite the fact that she goes into a profound trance, the depth of which is verified by the fact of complete post-hypnotic amnesia.

"The work has been largely directed towards finding out what the directions and intensities of her hostilities are which would make it necessary for her to turn so much self-destructiveness on herself. It seems that there is a great deal of envy of her father, whose drunkenness and bad behavior, failure to support the family, were too easily forgiven by the mother. Nevertheless, despite all these actual reasons in the life situation, the patient's rigid moral and ethical attitudes will not permit her to allow any experience or recognition in herself, of the fact of resentment, even less giving vent to it in any degree. However, this is the direction in which treatment to this point has gone.

"I am entertaining on illusions as to the prognosis psychiatrically in this case which remains extremely guarded, especially in view of the fact that a diagnosis of the presenting symptoms as such has not been made, and in view of the fact that the mechanism of the induction of bleeding on the basis of psychic tensions is in no way clear."—Maxwell Gitelson, M.D.

25 East Washington Street.

FIBROMA OF THE ETHMOID AND FRONTAL REGION

W. LIKELY SIMPSON, M.D.

MEMPHIS, TENN.

Tumors in the region of the ethmoid and frontal sinuses are sufficiently common to be well known by all who do sinus surgery. However, it is believed that this case is worthy of being reported in detail, because of the tremendous size of the tumor, the technical difficulties attending its removal, including the problem of the management of exposed brain with or without dural covering, the surgical principle of complete exenteration of infected sinal mucosa that is emphasized, and the studies made during the postoperative period of the function of deglutition and phonation. By reference to the latter is meant motion pictures that were made of superior and posterior aspects of the uvula, pillars, and pharyngeal musculature during the various stages of swallowing and of phonation in cooperation with the Department of Anatomy of the University of Tennessee Medical School.

Report of a Case

C. B., a colored male 63 years of age, was admitted to the John Gaston Hospital on January 17, 1943, with a history of having a growth in the upper part of his nose for five or more years. During the last two or three days there had been severe aching and pain in the left frontal region, with increased lacrimation in the left eye. Headaches had been so severe that he had to stop his work. He had not been able to breathe through the left nostril for a long time. The patient stated that the left eye had been gradually pushed to the side. He had intermittent epistaxis during he last few months. He claimed to have had a similar tumor removed from his nose in 1933. However, reference to his previous record indicated only that nasal polyps were removed in 1933. He had no other opera-

From the Department of Otolaryngology, University of Tennessee School of Medicine.

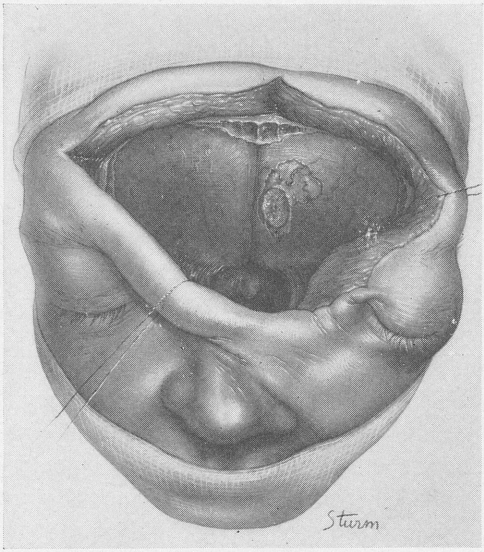


FIG. 1.—Drawing of post-operative field as viewed from in front.

tions nor any serious illness except the trouble with his nose. His family history was essentially negative for malignancy, tuberculosis, diabetes, and insanity.

Physicial Examination. The temperature was 99° F.; pulse, 100; respiration, 20; and blood pressure, 120/70. The patient was a well-developed, rather poorly nourished elderly colored male who was not acutely ill but whose face was greatly deformed by the presence of a bulging mass situated between the eyes, extending upwards on to the forehead. The left eye was situated laterally and downwards from its normal position. The mass was firm, immobile, not tender, not nodular, and not attached to the underlying skin but fixed to the deep structures. The left eye was congested and hyperemic. He was able to count fingers with the left eye but its axis was markedly divergent from that of the right eye.

The bridge of the nose was widened and prominent and the nasal bones could be palpated. Anterior rhinoscopy revealed a mass that

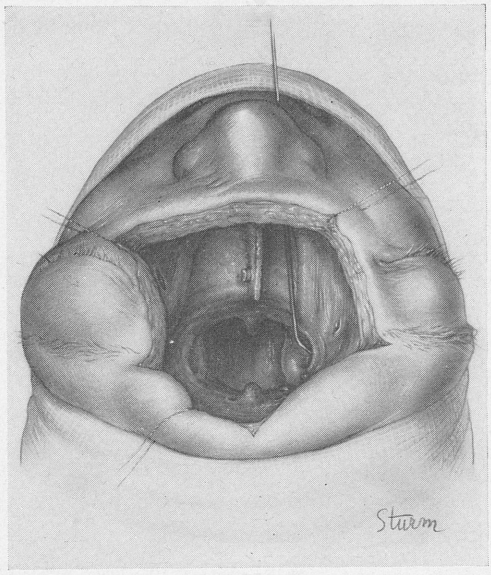


FIG. 2.—Drawing of post-operative field as viewed from head of table.

completely occluded the left side of the nose. The septum was deviated to the right, producing a partial obstruction of the right side. The mass seen in the left fossa was firm, red, smooth, slightly tender and slightly movable with an applicator.

The mouth and throat presented nothing of interest other than dental caries and poor oral hygiene. The ears were clinically negative with normal hearing bilaterally.

The lung fields were resonant and vascular. The heart was enlarged downward into the region of the P.M.I. in the sixth and seventh interspaces to the left of the midclavicular line. However, the heart sounds were clear and regular and there were no murmurs present.

The remainder of the physical examination was within normal limits.

The admission blood picture showed a mild secondary anemia. The urinalysis was negative. The Kahn test was negative.

X-ray films made the day after admission disclosed a destructive process involving the maxilla, malar and frontal bones on the left, as well as the ethmoids and the anterior part of the sphenoids.

On January 19, 1943, the mass in the left fossa of the nose was biopsied and the pathological report was probable fibrosarcoma with low-grade malignancy. Due to the magnitude of the tumor, it was not considered to be operable.

However, at this time the patient developed fever and pain, with increase in the size of the bulging mass. This became fluctuant very shortly and believing that an abscess was present an incision was made and drainage was carried out. The incision through the skin and subcutaneous tissues was made over the most prominent part of the fluctuation, situated midway between the center of the nose and the inner canthus of the left eye, and a large amount of thick yellow pus was evacuated. After the pus was evacuated, the anterior surface of the tumor was observed to be smooth, glistening, apparently encapsulated, and we considered at this time the possibility of enucleation of the mass.

The patient's general condition improved following drainage of the abscess but culture of the pus was positive for staphylococcus aureus and small gram-negative bacilli, morphologically Koch-Weeks bacilli.

On January 30, 1943, the tumor mass was removed in toto. It was attached to the dura by a pedicle from its posterior superior aspect, in the region of the posterior ethmoids. The dura was removed with the tumor over an area of about two inches in diameter overlying the left frontal lobe. There was a true retrobulbar abscess which was evacuated and drained. All of the sinuses on both sides of the nose were in a suppurative condition. Sulfanilamide was dusted into the wound after hemostasis was secured and the cavity was packed with iodoform gauze.

Pathological Report. Grossly the tumor was yellowish-white in color with grooves and ridges on its surface, corresponding to the

sulci and gyri on the surface of the brain with which it had been in contact. It was well encapsulated and attached by a pedicle, apparently to the dura. On the cut surface it had a yellowish-white, whorled appearance almost identical with that of a uterine fibroid. The cut surface tended to bulge forward. Numerous small calcified areas were visible on this surface. The consistency was very tough.

Microscopic section showed numerous wide, swirling bundles of fibrous connective tissue. Isolated areas were more cellular, however, and in these were spindle-shaped cells. These more cellular areas were scattered at random throughout the section and had little regularity. Some of the sections were covered by a pseudostratified columnar ciliated epithelium while others had a simple mesothelial surface.

The postoperative course was uneventful, except that the area of the exposed brain where the dura had been removed gradually bulged forward and downward into the cavity. Within a short time healthy granulations covered all the raw areas. The cavity reduced itself to approximately half its size, and the contents of the left orbit gradually swung back towards the midline. Vision of the left eye gradually improved until he could see almost as well with the left eye as with the right.

Considerable pus exuded from the remnants of the frontal sinuses, the sphenoid and maxillary sinuses. On March 30, 1943, the remnants of both frontal sinuses and the left antrum were exenterated.

On May 1, 1943, the sphenoidal sinuses were exenterated. During the course of this procedure the internal carotid artery on the left was injured. Profuse hemorrhage resulted, but this was quickly and easily controlled by means of an iodoform pack introduced into the sphenoid sinuses and filling the cavity. On May 6, the packing was removed and hemorrhage again resulted so it was repacked.

On May 9, 1943, hemorrhage resulted spontaneously while the patient was straining at stool. He was immediately taken to the operating room, the sphenoid repacked, and the left internal carotid artery ligated just above the bifurcation of the common carotid.

The postoperative course has been uneventful since that time, and there has been no further hemorrhage. The packing in the sphenoid was left in situ for three weeks; the loose packing of the large cavity formerly occupied by the tumor was changed at frequent intervals. No bleeding from the sphenoid resulted when the packing was loosened and gradually removed three weeks after ligation of the left carotid artery (approximately six weeks after the original injury to the artery).

899 Madison Avenue.

CASE REPORT OF A CAROTID BODY TUMOR

PIERRE VIOLE, M.D.

LOS ANGELES, CALIF.

Experience with this tumor has been chiefly confined to the past fifty years, during which time a total of only some 250 cases have been reported. These were largely individual cases; hence the experience of any one operator has been limited, and literature pertaining to it is scant.

Marchand in 1891 appears to have been the first to fully describe this neoplasm, after excision of a carotid body tumor by Riegner in 1880. Through the succeeding decade a scattered few cases made their way into medical literature, and in 1903 Scudder[1] reported a case diagnosed as "perithelioma", a tumor of nine years' standing, which was successfully removed. This was the first case of surgery of a carotid body tumor authentically reported in this country.

Relatively little was known of the function of this gland until about 1930; it had not been considered essential to well-being or even to life itself. In more recent years it has been demonstrated that the respiration and circulation are influenced by the carotid body, due to chemoreceptors present in the gland which react to arterial blood changes. Schmidt and Comroe[2] state more conclusively that the role played by carotid bodies, is a significant one in the control of respiration, and that changes in chemical composition of the blood affect the respiration through both the carotid body and the respiratory center itself. Twombly, Simon and Steinberger,[3] in an article on this subject published in 1943, state that experiments show the carotid body to be more sensitive than the respiratory center to the respiratory poisons, such as nicotine, cyanide and lobeline and to anoxia, whereas the center itself is more sensitive to changes in carbon dioxide content and pH of the blood.

From the Department of Otolaryngology of the University of Southern California School of Medicine.

As is true of other tumors, their cause is not known. It is thought there may be a hereditary factor in the development of this growth, since a number of familial cases have been reported. Both sexes seem to be equally susceptible, both white and negroid races are involved, the age of patients varying from early childhood to senility.

These tumors develop slowly, usually over a period of years. Often the only symptom noticed by the patient is an external swelling in the area of the gland itself, which may appear to remain almost static over a prolonged time, until a sudden acceleration may suggest medical investigation. The growth is usually round or oval, smooth, and rather elastic though compressible. It is movable from side to side but not up and down. The tumor may not be apparent at first glance, but with the head extended, close inspection under a good illuminator may reveal a swelling behind and below the angle of the jaw. Eventually pressure due to the growth may produce some hoarseness, cough, possible nausea, vomiting, or tinnitus, and occasionally local pain is experienced. However, these patients are usually symptom-free for many years before such developments call the tumor to their serious attention.

This neoplasm is predominantly unilateral, though a few cases have been reported where the patient had a growth on both sides of the neck. These may occur simultaneously, or one may develop after removal of the other, sometimes a number of years subsequent to the original operation. In the discussion of this subject by Gratiot[4] he cites a case reported by Lund in which the right gland was removed 29 years following excision of the left. He adds that 15 to 20 per cent of carotid body tumors become locally malignant after remaining benign for years, with only rare metastases in the regional lymph nodes, distant metastases being unconfirmed.

Without the somewhat precarious procedure of aspiration biopsy, pre-operative carotid body tumor is difficult to diagnose. It is often logically confused with other cervical conditions, such as inflammatory lymph nodes, tubercular nodes, malignant lymphomas, branchial-cleft cysts, aberrant thyroid tissue, lipoma and sarcoma. Gratiot differentiates it from such other growths by the fact that it is the only "single, solid, slow-growing tumor, intimately attached to

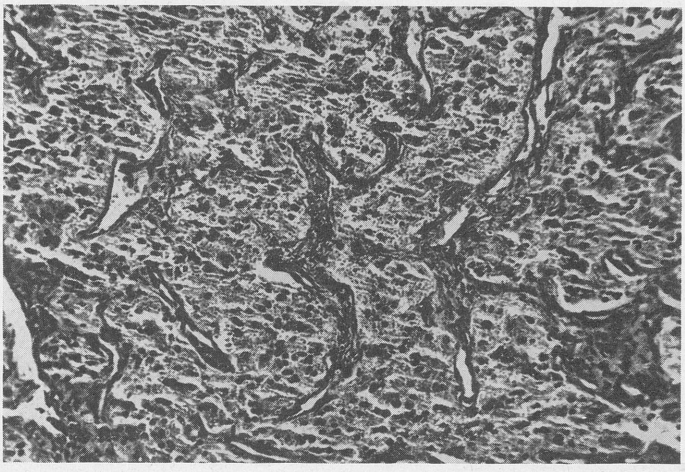


FIG. 1.—Photomicrograph of carotid body tumor revealing characteristic paraganglioma formation.

the carotid bifurcation," and concludes that exploration may be the only recourse left to the operator to establish an accurate diagnosis. Laboratory findings offer little conclusive evidence, as blood, urine and x-ray examinations may all be negative in the presence of the tumor. Surgical procedure is the universally accepted treatment. In the article above referred to by Twombly et al., they state that perhaps the most important point in diagnosis is to consider any slow-growing cervical mass as a possible carotid body tumor.

Ewing[5] considers this neoplasm in a class by itself histologically, describing the structure as complex and in general that of an alveolar perithelioma, but varying in many details. He states a skillful enucleation is usually successful in preventing recurrence, but cites an estimated 35 per cent mortality from the operation.

J. C., a Japanese male, 59 years of age, entered Los Angeles General Hospital September 3, 1942, complaining of a swelling in the right side of his neck of three months' duration. Apparently he had been in good health until the onset of this condition, when he became conscious of difficulty in swallowing and diminished appetite.

Just prior to his appearance at the hospital he had been seen in New Mexico, at which time a biopsy was made and a tentative diagnosis of carcinoma was recorded.

The past history was noncontributory.

Physical examination revealed a Japanese adult in no apparent distress, the only significant findings being as follows:

A 4 x 6 cm. mass projected into the naso- and oro-pharynx from the right lateral pharyngeal wall. The mucosa over the mass appeared normal. The mass occupied about one-third of the oro- and naso-pharynx, and seemed soft but nonfluctuant on palpation.

In the neck at the upper portion of the anterior border of the right sternocleidomastoid muscle a firm mass was palpable, which moved with swallowing.

No other glands were palpable. The urine and the blood were negative.

Three biopsies were attempted at the hospital. We were unable to corroborate the biopsy previously made and were not able to make any diagnosis.

The results of the biopsies were:

Sept. 17, 1942—Miscellaneous pharyngeal mucous membrane, glands and muscle. No pathological diagnosis.

Sept. 26, 1942—Biopsy attempted by dissection of neck. No tissue obtained for section.

Oct. 5, 1942—Pharynx section showed only connective tissue, many blood vessels, and some adipose tissue. Tumor could not be diagnosed.

On October 14, 1942, the patient was taken to the operating room for excision of the pharyngeal tumor. The operative procedure consisted of ligation of the right common carotid to control hemorrhage, and as the tumor presented itself primarily in the pharynx, it was decided to enucleate the mass through the oral approach. The mass was semi-soft, moderately fluctuant, and movable from side to side. The enucleation was accompanied with considerable bleeding.

The patient was returned to bed in a state of shock and, in spite of all supportive measures attempted, expired within 24 hours following surgery.

The specimen consisted of an irregularly oval portion of tissue 4 x 3.5 x 1.8 cm. The tissue was yellow-brown in color and presented numerous fibrous bands on all aspects. On section a mottled yellow-brown appearance was observed, suggesting a rather fatty component, although its firm consistency argued against this possibility.

Microscopic examination revealed a tumor having the typical structure of a "paraganglioma" of the carotid body. There was no evidence of malignancy. A very thorough autopsy was performed. No metastatic lesions were encountered which could be related to the tumor in question.

Summary

This case of a carotid body tumor, also diagnosed as "paraganglioma of the carotid body", is presented primarily because of the relatively infrequent occurrence of this type of tumor, the problem of differential diagnosis which it presents, and the seriousness of operative procedure.

1930 Wilshire Boulevard.

References

1. Scudder, C. L.: Tumor of the Intercarotid Body, Am. J. M. Sc. 126:384, 1903.

2. Schmidt, C. F., and Comroe, J. H., Jr.: Functions of Carotid and Aortic Bodies, Physiol. Rev. 20:115, 1940.

3. Twombly, G. H., Simon, L. G., and Steinberger, L.: Tumor of Carotid Gland, Conn. S. Med. J. 7:172, 1943.

4. Gratiot, J. H.: Carotid Body Tumors, Surg. Gynec. & Obst. 77:177, 1943.

5. Ewing, James: Neoplastic Diseases, 4th Ed., Philadelphia, W. B. Saunders Co., 1940, p. 384.

DISCUSSION

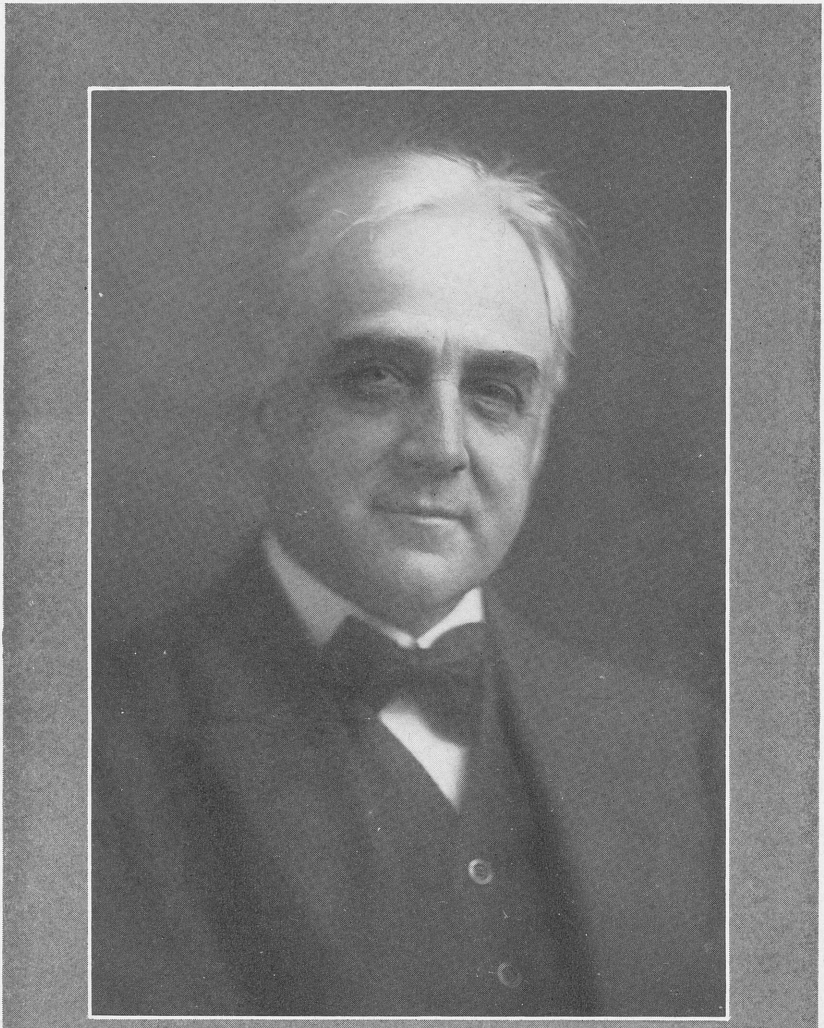
Walter B. Hoover, M.D., Boston, Mass. (by invitation): This is really an unusual tumor. Within the past seven months it has been my privilege to see two patients with tumor of the carotid body. One

was a man of 25 years. An operation had been attempted elsewhere from outside and inside the throat, but without success. An accurate diagnosis had not been made. I thought removal might be attempted from within the pharynx. An intratracheal tube was inserted and we were ready to operate from the outside as well as from the pharynx. The tumor was attached to the base of the skull and was very difficult to separate from its attachment, but this was finally accomplished after about two and a half hours of careful dissection. Bleeding was troublesome because there were a number of large vessels running along the tumor, which seemed at first to be not only a carotid body tumor but also a hemangioma. The tumor was pressed down into the neck and the pharyngeal incision was closed. The tumor was then separated or dissected from the area of the bifurcation of the carotid by an external approach.

The second case was that of a woman of 55 years in whom a large tumor presented in the pharynx up behind the palate, pushing the palate forward and extending to the base of the skull, and also presenting below the angle of the jaw in the neck. We prepared the patient by compressing the common carotid artery ten days before operation so that it could be tied off if necessary. Having had previous experience, we approached this tumor from the outside. It was not necessary to tie off the carotid artery, and the tumor was successfully removed.

In each case we performed a tracheotomy, inserted a feeding tube, and left a pack in place in the space from which the tumor was removed. A transfusion was given each patient. The pack was removed from the neck seven days after operation.

Memorials



Thomas Hubbard

THOMAS HUBBARD

1859-1943

Dr. Thomas Hubbard, Active Fellow 1895, Emeritus Fellow 1927, died in Toledo, Ohio, where he had practiced Otolaryngology for many years, on the 5th of July, 1943. He was well known to the older fellows of our Association who admired him for his many fine, manly qualities as well as for his excellent scientific work, but since his retirement nearly twenty years ago, he is unknown to many of our younger members.

Dr. Hubbard was born July 22, 1859 of old New England stock, his forebears having settled in Middletown, Connecticut, about 1650. His father, Amos Fisk Hubbard, a banker, son of Colonel Wm. Hubbard, came to Ohio in 1834 and settled in Ashtabula, where his uncle, Dr. John C. Hubbard, practiced medicine for nearly fifty years. His early school years were spent in Ashtabula and his college training was obtained at the University of Michigan, where he chiefly studied chemistry and attended medical lectures from 1880 to 1882 (Ph.C.). He then matriculated in the School of Medicine of the University of Pennsylvania, graduating in 1885. Two years later, he began the study of laryngology (and otology) in Vienna with Schrötter, Politzer and others, and returned to Europe in 1908 to study bronchoscopy with Killian at Freiburg, Germany. Always especially interested in diseases of the larynx, he took up the then new science of bronchoscopy with enthusiasm and many of his later papers were concerned with this technique of which he had made himself a master. He had early made a specialty of the treatment of the laryngeal complications of diphtheria and this led naturally to surgery for the relief of dyspnea and for foreign bodies in the throat, air passages and esophagus. In 1913, the title of his Presidential address was "The Air One Breathes—A Study of Humidity and Dust Contents" and in regard to this particular interest of his, part of a letter written in 1939 follows: "That work was really begun in 1884 when I gave a report on the carbonic acid gas present in the respired air of the lecture ampitheatre at Old Penn in collaboration with

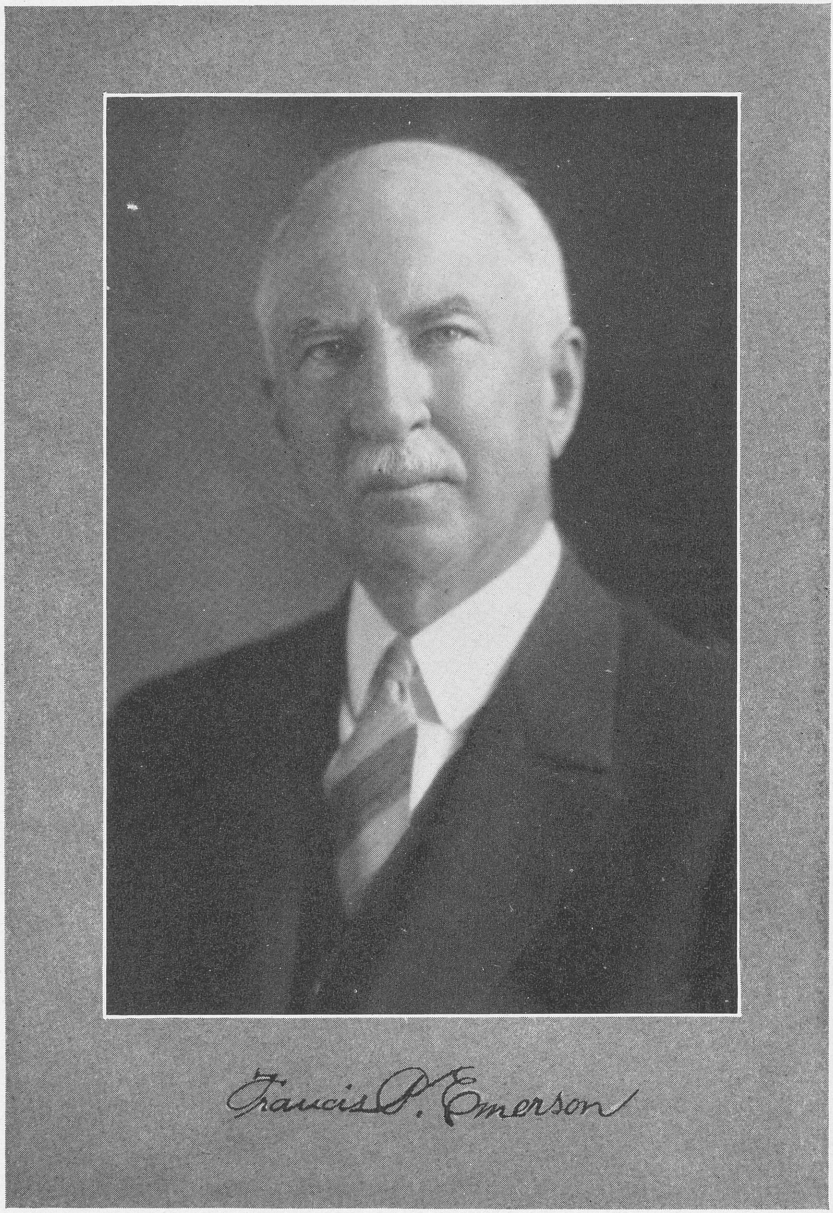
John Marshall, assistant to the Professor of Chemistry (Dr. Marshall succeeded his father-in-law, Prof. Wormley as Prof. of Chemistry and also was Dean of the School of Medicine). This was followed by a paper entitled "Hot-Air Comfort" in 1925. Both of these papers introduced the term "conditioning the air", one twenty years and the other ten years in advance of the period when it became a popular craze. I studied Ventilation when in Vienna, at the Congress of Hygiene and Demography in 1887-8. All that is my background of training in the art of Ventilation. Applied Hygiene has attraction for me."

Dr. Hubbard was President of the Association in 1913, having been elected an active fellow in 1895. He practiced medicine in Toledo, Ohio, until incapacitated by a hemiplegia, but returned to Ashtabula after his illness. A beautifully wrought weather-vane, mounted on a thirty-foot steel shaft, was presented by him to the Ashtabula Public Library in memory of his grandparents and this weather-vane was fashioned by his own hands after his illness. In an endeavor to re-educate his muscles, he took up wood carving and made a very notable success of it, as anyone who has seen samples of his exquisite handiwork, will testify. His particular design was a folding carved book-rest made from a single piece of wood and it was known as the "Dr. Hubbard book-rest". He also became an excellent typist and even learned again to write in longhand.

From 1893 to 1896, Dr. Hubbard was secretary of the Ohio State Medical Society and was President of the Toledo Academy of Medicine in 1906. He was also a Fellow of the American Otological Society, the American Laryngological, Rhinological and Otological Society, the American Bronchoscopic Society, the Northwest Ohio Medical Society, of which he was President, and many others. He was laryngologist to the St. Vincent's and Flower Hospitals, and bronchoscopist to St. Vincent's, Flower, Toledo and Robinwood Hospitals.

Dr. Hubbard was married in 1889 to Charissa Graves and had two sons, Holland A. and Pomeroy Graves Hubbard. He was a member of Trinity Episcopal Church and in politics was a Jeffersonian Democrat.

A regular attendant at the meetings of our Association and the author of six papers presented at our Annual Congresses, he was a kind friend, a skillful scientist and a highly esteemed Fellow, whose chief medical interest lay in diseases of the laryngo-tracheo-bronchial tree and in hygiene and air-conditioning, a term that he was one of the first to use.



Francis P. Emerson

FRANCIS PATTEN EMERSON

1862-1944

Dr. Francis P. Emerson was born at Candia, N. H. in 1862, the son of Moses F. Emerson and Abbie Patten, both of Candia. He died at Summit Road, Franklin, Mass. on January 19, 1944 in the home of his daughter with whom he had lived since his retirement over ten years ago.

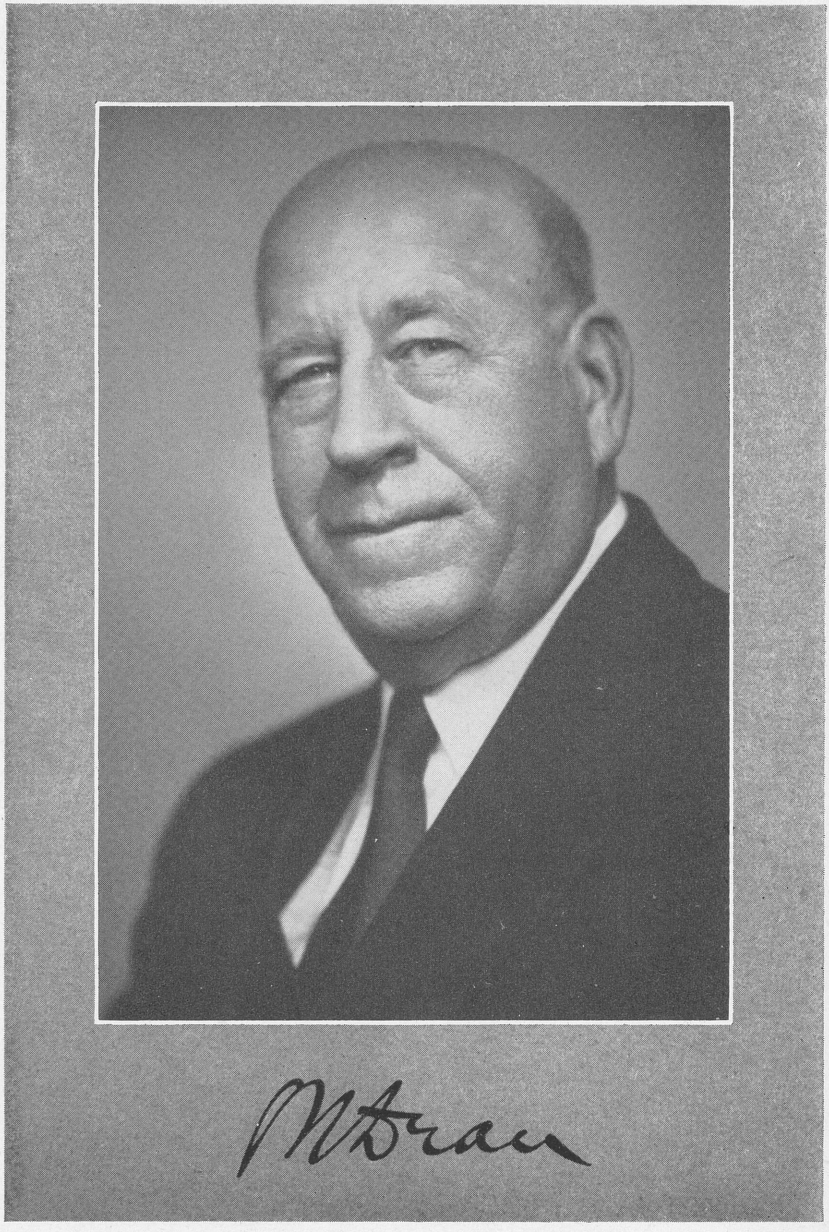
His preliminary education was obtained in Pembroke Academy in Candia and he received his medical degree from the College of Physicians and Surgeons (now Columbia) in New York City in 1886 and it was there, after his graduation, that he began the study of laryngology under the tutelage of Drs. Powers and Newcombe. On going to Massachusetts, he became connected with the Boston Dispensary and with the Massachusetts Eye and Ear Infirmary and he served on the staff of the latter institution as aural surgeon, as well as to the Massachusetts General Hospital, until his retirement. He held the rank of Instructor in Otology in the Harvard Medical School, was a staff member of the Brooks Hospital of Brookline, Mass., of the Massachusetts Women's Hospital and Consulting Aural Surgeon to the Adams Nervine Hospital at Jamaica Plains.

During World War 1, Dr. Emerson, with the rank of Major, M.C., served as Chief of the Division of Head Surgery in the Base Hospital and Chief of the Emergency Hospital for Influenza patients at Camp Lee, Virginia in 1918-1919. He had been married in 1886, the year of his graduation to Miss Rena Colby and had two children, Guy C. Emerson and Mrs. Donald B. Chapman. His chief diversions were travel and golf and he belonged to the following clubs and societies: Washington A. F. and A. M., Boston Chamber of Commerce, Military Order of the World War, Harvard Club of Boston, Weston Golf Club and Franklin Country Club.

Dr. Emerson's major medical interest was Otology and the majority of his twenty-old medical papers dealt with this subject, although he presented two papers before the Association during his

comparatively short active fellowship, to which he was elected in 1921, becoming a Corresponding Fellow in 1930. He was Second Vice-President in 1928, was President of the New England Laryngological and Otological Society in 1928-29, and Chairman of the Section on Otology, Rhinology and Laryngology of the American Medical Association in 1916-17. He was also a member of the American Association for the Advancement of Science.

Failing eyesight had compelled Dr. Emerson's retirement from practice and active hospital work and for the last years of his life he had been practically blind and much broken in health.



LEE WALLACE DEAN, M.D.

1873-1944

Lee Wallace Dean, Sr., late Professor of Otolaryngology in Washington University School of Medicine, St. Louis, and President of the American Laryngological Association in 1924, died February 9, 1944.

He was born in Muscatine, Iowa, March 28, 1873, and received his early education in that state. Subsequently he attended the State University where the degree of Bachelor of Science was conferred upon him in 1894. Two years later he received the degrees of Master of Science and Doctor of Medicine.

After a year of graduate work in Vienna, Dr. Dean returned to Iowa and three years later was made Head of the Department and Professor of Otolaryngology and Oral Surgery. In 1912 he became Dean of the College of Medicine, and held both of these positions until 1927 when he came to Washington University as Head of the Department and Professor of Otolaryngology in the School of Medicine, and Laryngologist to the hospitals associated with this institution. Following his retirement in 1940 he was made Emeritus Professor.

During the First World War he served as Commanding Officer of General Hospital No. 54 with the rank of Lieutenant Colonel.

Dr. Dean was a Fellow and at one time President of each of the other national special societies: of the American Otological Society in 1932; of the American Laryngological, Rhinological and Otological Society in 1937; of the American Academy of Ophthalmology and Otolaryngology in 1937. He was a member also of the Association of American Peroral Endoscopists, the American Broncho-Esophagological Association, and La Société de Laringologie des Hôpitaux de Paris. From 1937 until his death he was Co-Editor of the Annals of Otology, Rhinology and Laryngology. At the time of his death he was still an active member of the American Board of Otolaryngology.

The honor which he most prized was the DeRoaldes Medal which this Association awarded him in 1937.

Dr. Dean will be remembered chiefly as an educator, for his every thought revolved about the development of young men in the specialty and the improvement of standards in the teaching of otolaryngology. His absorbing interest and the subject of most of his contributions to the literature dealt with the infection of sinuses in infants and young children. In his later years, the various phases of allergy occupied his attention and he inspired many original investigations on allergy of the nose and ears among his colleagues in the Department at Washington University.

Even after his removal to St. Louis he remained the proud owner of a number of farms in Iowa which he visited regularly.

He and Mrs. Dean were ardent hunters and fishermen. In season they roamed the Dakotas, or the shores of Florida, together. These expeditions were invariably followed by a series of sumptuous dinners in honor of their many friends.

Mrs. Dean, the former Ella Bailey, was in ill health even before the death of her husband and survived him less than a year. Their son, Lee Wallace Dean, Jr., resides with his family in St. Louis and is a member of the staff of the Department of Otolaryngology in Washington University.

A. W. P.

Executive Sessions

1944

FIRST EXECUTIVE SESSION

JUNE 7, 1944

The First Executive Session of the Sixty-sixth Annual Meeting was called to order at the Hotel Waldorf-Astoria, New York, N. Y. by President Charles J. Imperatori immediately following the close of the First Scientific Session on Wednesday, June 7, 1944.

On recommendation of the Council, the following were elected:

To Emeritus Fellowship:
DR. RALPH BUTLER

To Active Fellowship:
DR. HUGH G. BEATTY
DR. LAWRENCE R. BOIES
DR. FRED Z. HAVENS
DR. GORDON D. HOOPLE
DR. MARVIN F. JONES

There were no recommendations to Honorary or Corresponding Fellowship.

Dr. Thomas E. Carmody and Dr. C. Stewart Nash were appointed to serve as an Auditing Committee.

The Association elected the following to serve as a Nominating Committee: Dr. Harris P. Mosher, Dr. Ralph A. Fenton, Dr. Thomas E. Carmody, Dr. George M. Coates, Dr. William E. Grove.

The newly elected Fellows were inducted into the Association and, there being no further business, the session adjourned.

ANNUAL BUSINESS MEETING

JUNE 8, 1944

The Annual Business Meeting of the Association was called to order at 12 noon on Thursday, June 8, 1944 by President Charles J. Imperatori.

REPORT OF THE SECRETARY

At the Annual Meeting of the Council, held in New York City January 9, 1943, it was voted "in view of the heavy demands upon the transportation facilities of the country in pursuit of the war, as well as upon the time and energies of the Fellows" to omit the Sixty-fifth Annual Meeting which should have been held in the Spring of 1943.

After providing for a mail ballot to replace the election customarily held at the Annual Meeting, the Council recommended:

For Active Fellowship:

DR. ANDERSON C. HILDING, 324 West Superior St., Duluth, Minn.
DR. O. E. VANALYEA, 135 S. LaSalle St., Chicago, Ill.

For Corresponding Fellowship:

DR. ANDREW A. EGGSTON, 635 Park Ave., New York, N. Y.

For Emeritus Fellowship:

DR. HARRINGTON B. GRAHAM
DR. SAMUEL MCCULLAGH

For Honorary Fellowship:

DR. ROBERT LEVY
DR. FIELDING O. LEWIS
DR. NORVAL H. PIERCE

The mail ballot was favorable in each case.

The resignation of Dr. Walter Stevenson was accepted with regret.

The Newcomb Award for 1943 was presented to Dr. George M. Coates.

At its Second Meeting, held in New York on June 6, 1943, the Council strongly expressed itself in favor of holding the Sixty-sixth Annual Congress should conditions render this at all feasible.

The report of the DeRoaldes Award Committee was received, naming Dr. Ralph A. Fenton for that honor. A certificate was given to Dr. Fenton and the awarding of the medal was postponed until this could be struck and properly presented.

At the recommendation of the Secretary, the Council directed that a volume of the Transactions be published in 1943 despite the omission of the Annual Congress and that in lieu of the usual scientific papers an Index be prepared listing all papers read before this Association since its inception, and their authors.

This volume appeared early in 1944 and the Secretary is pleased to report that the demand for it from non-members has been gratifying, as will be shown in the Treasurer's Report.

The Annual Meeting of the Council was held in New York City on January 10, 1944. It was decided at this meeting to hold the Annual Congress at the Hotel Waldorf-Astoria, New York City on June 7 and 8, 1944.

It was decided, as an emergency measure, to limit the Sessions to two days, and the Secretary was instructed to bring before the Membership an amendment to the By-laws making the change permanent. The proposed change is in response to a general sentiment among the Fellows of the four national Societies in favor of a plan which will permit all of the meetings to be held within the space of one week.

Dr. Samuel Salinger was appointed Abstract Editor and Dr. Harold I. Lillie was elected Delegate to the Board of Governors of the American College of Surgeons.

Since June 1943 the Association has lost through death Dr. Thomas Hubbard, Toledo, Ohio, Emeritus Fellow, on July 5, 1943;

Dr. Francis P. Emerson, Franklin, Mass., on January 19, 1944; and Dr. Lee Wallace Dean, Sr., St. Louis, Mo., on February 9, 1944.

The Membership on May 10, 1944 was:

Honorary Fellows	5
Corresponding Fellows	17
Emeritus Fellows	21
Active Fellows	95
Total	138

Respectfully submitted,

ARTHUR W. PROETZ,
Secretary.

The report was accepted and filed.

June 7, 1944

REPORT OF THE AUDITING COMMITTEE

Gentlemen:

Your Auditing Committee makes the following report:

This is to certify that we have examined the books of the Treasurer of the American Laryngological Association for the fiscal year 1943-1944 and find the same correct. These books have already been audited by the Treasurer of Colby College, Waterville, Maine, and found to be in order and according to sound accounting procedure.

C. STEWART NASH

THOMAS E. CARMODY

The report was accepted and filed.

The Treasurer then read his report.

Report of the Treasurer

Balance Sheet May 15, 1944

ASSETS

Cash in bank		$ 2,457.44	
Cash due from Trust Funds		419.98	$ 2,877.42
Trust Fund Investments:	Book Value		
Bonds:			
$3,000 U. S. Treasury 3 1/8 1946-49	$3307.20		
*1,000 U. S. Treasury 2 3/4 1948-51	1101.20		
1,000 U. S. Treasury 2 3/4 1958-63	1101.20		
3,000 U. S. Defense Bonds 2½ Series G-1953	3000.00		
1,000 U. S. Defense Bonds 2½ Series G-1954	1000.00		
3,000 U. S. Treasury 2½ 1964-69	3000.00		
1,000 U. S. Treasury 2¼ 1956-59	1003.50		
		13,513.10	
Savings Deposits			
Waterville Savings Bank	2542.18		
Less-Due General Cash (over-invested)	419.98		
	2122.20	2,122.20	15,635.30
Total Assets			$18,512.72

LIABILITIES AND CAPITAL

Trust Funds:			
Casselberry Fund	5000.00		
Johnston Fund	4731.23		
DeRoaldes Fund	500.00		
Dr. James Edward Newcomb Prize Fund	500.00		
Dr. James Edward Newcomb Trust Fund	2000.00		
Memorial Fund	1979.19	14,710.42	
Unused Income:			
Casselberry Fund	119.55		
Johnston Fund	116.60		

DeRoaldes Fund	214.48		
Dr. James Edward Newcomb Prize Fund	59.52		
Dr. James Edward Newcomb Trust Fund	252.22		
Memorial Fund	159.51	924.88	$15,635.30
General Capital and Surplus			2,877.42
Total Liabilities and Capital			$18,512.72

*Recent financial reports have included in security holdings
$2,000 U. S. Treasury 2 3/4 1948-51 @ $2202.40
These reports should have read
$1,000 U. S. Treasury 2 3/4 1948-51 @ 1101.20
1,000 U. S. Treasury 2 3/4 1958-63 @ 1101.20

STATEMENT OF INCOME AND EXPENSE

FOR THE PERIOD MAY 14, 1943 TO MAY 15, 1944

Income:		
Dues	$ 2,200.00	
Sale of Transactions	835.00	
Income from Trust Fund	364.37	$ 3,399.37
Expenditures:		
Honorariums—Officers (Allowance for clerical hire)	400.00	
Council Meeting	435.81	
Cost of Transactions	1,093.15	
Postage	75.91	
Stationery and Printing	206.06	
Miscellaneous Expense	7.16	
Treasurer's Bond	37.50	2,255.59
Excess of Income over Expenditures		1,143.78

RECONCILIATION OF GENERAL CAPITAL AND SURPLUS

General Capital and Surplus—May 14, 1943		$ 2,098.01
Excess of Income over Expenditure	$1,143.78	
Income Prorated to Unused Income of Trust Funds	364.37	779.41
General Capital and Surplus—May 15, 1944		2,877.42

Note: Securities Purchased	Cost
$3,000 U. S. Treasury 2½ 1964-69	$ 3,000.00
1,000 U. S. Treasury 2¼ 1056-59	1,003.50

Securities Sold	Book Value	Received
$5,250 Walnut St. Corporation Income Bonds 1950 c stock	$ 1,120.00	$ 2,622.21
3,000 Wm. K. Brandt 3-1942	420.00	449.58
In accordance with the vote of the Council the profit on the securities sold was used as follows—		
Johnston Fund	$1248.77	
Casselberry Fund	283.02	

DISPOSITION OF TRUST FUND INCOME

Total Income on Trust Funds		$ 364.37
Prorated as follows:		
Casselberry Fund	$ 123.88	
Johnston Fund	116.60	
DeRoaldes Fund	12.39	
Dr. James E. Newcomb Prize Fund	12.39	
Dr. James E. Newcomb Trust Fund	49.92	
Memorial Fund	49.19	364.37

STATEMENT OF INCOME (TRUST FUND)

	Unused Income 5/14/43	Income	Trans. to Principal of Fund	Expended	Unused Income 5/15/44
Casselberry Fund	$172.27	$123.88	$172.27	$ 4.33	$119.35
Johnston Fund	127.97	116.60	127.97	.....	116.60
DeRoaldes Fund	202.09	12.39	.....	.....	214.48
Dr. James E. Newcomb Prize Fund	47.13	12.39	.....	.....	59.52
Dr. James E. Newcomb Trust Fund	205.30	49.92	.....	.....	255.22
Memorial Fund	110.32	49.19	.....	.....	159.51
	$865.08	$364.37	$300.24	$ 4.33	$924.88

While the distribution of gain from the sale of securities of a pooled Endowment Fund to the Principal of a specific fund violates the principle of such a fund—this has seemed advisable in order to bring funds to their original amount. In view of the fact that all of the trust funds of the Association are now at their original amount with the exception of the Johnston Fund it is recommended that that fund now be fully restored by transferring income prorated to this fund to the principal, $116.60, and by transferring $152.17 from General Capital and Surplus. It is further recommended that in the future, 7½% of the income from trust funds, be set aside as a Reserve for Losses in Investment.

Respectfully submitted,

FREDERICK T. HILL,

Treasurer.

May 26, 1944

This is to certify that I have examined the books of the American Laryngological Association for the fiscal year 1943-44. I have verified all cash balances. The financial reports presented correctly reflect the financial condition and operations of the Association, and the accounts are kept in accordance with sound accounting procedure.

A. G. EUSTIS,

Treasurer, Colby College, Waterville, Maine.

The report was accepted and filed.

REPORT OF THE NOMINATING COMMITTEE

The Nominating Committee is pleased to recommend the following Fellows to serve as a Board of Directors of the Association for the year 1944-45:

DR. HAROLD I. LILLIE, Rochester, Minn.
DR. JOHN J. SHEA, Memphis, Tenn.
DR. THOMAS C. GALLOWAY, Evanston, Ill.
DR. ARTHUR W. PROETZ, St. Louis, Mo.
DR. FREDERICK T. HILL, Waterville, Maine
DR. LOUIS H. CLERF, Philadelphia, Pa.
DR. JAMES A. BABBITT, Philadelphia, Pa.
DR. GORDON BERRY, Worcester, Mass.
DR. THOMAS E. CARMODY, Denver, Colo.
DR. CHARLES J. IMPERATORI, New York, N. Y.

(Signed)

HARRIS P. MOSHER,

Chairman.

The report was accepted and filed.

Report of the Candidates' Nominating Committee

The Candidates' Nominating Committee consists of the following: Drs. Fletcher B. Woodward, John Mackenzie Brown, Thomas C. Galloway, Arthur W. Proetz and George M. Coates, Chairman.

The work of this Committee is entirely informal. Its purpose is to, from time to time, conduct a survey of the otolaryngologists of the country, particularly the younger men who are coming into prominence through their writings, teaching appointments and attendance at medical meetings. The members of the Committee submit from time to time suggestions as to possible candidates for Fellowship and these suggestions are presented to the Council for its information.

The Committee furthermore charges itself with the duty of seeing that promising young men are encouraged to attend the meetings of our Society where they may become acquainted with our Fellows. During the past years, many of those suggested by the Committee have become Fellows but the Committee itself does not as a Committee file nomination blanks. Individually, the members of the Committee may encourage the nomination of those they think worthy by other Fellows of the Association. Nearly all of the Fellows elected during the last few years have been acted upon in this way.

It does not seem proper to give the list of those under consideration at an open meeting of the Association. Therefore, no list is appended. It may be said, however, that there are some fifteen or twenty names under consideration at the present time.

Respectfully submitted for the Committee,

George M. Coates,

Chairman.

The report was accepted and filed.

Report of the American Board of Otolaryngology

The American Board of Otolaryngology has continued for the past year its routine of two examinations. For the one to be held this June about a hundred candidates are expected from the present registration. At the examination last fall some sixty candidates appeared.

The time required before a candidate can appear before the Board has been shortened slightly on account of the wartime shortening of House Officer services.

Respectfully submitted,

Harris P. Mosher

The report was accepted and filed.

Report of the Biographer and Historian

The Historian, Dr. George M. Coates, reported the deaths of Dr. Thomas Hubbard, Dr. Francis P. Emerson and Dr. L. W. Dean, Sr.

The report which consisted of the memorials to these Fellows, which appear in the current volume of the Transactions was accepted and filed.

By unanimous vote of the Fellows present, Article 4, Paragraph 4 of the Constitution was amended to read as follows:

> "Each Fellow shall be expected to comment confidentially to the Secretary upon the proposed candidates and these comments shall be in the hands of the Secretary before the fifteenth of December next following."

By unanimous vote of the Fellows present, without discussion, Article 3, Section 1, Paragraph 4 of the By-laws was amended to read:

"Second Day, General Business Meeting at which only Fellows of the Association shall be present. This shall include Reports of the Secretary, the Treasurer and all Committees, and new and unfinished business. The remainder of the second day shall be devoted to the presentation of scientific papers." And paragraph 5, dealing with the third day, was deleted.

The Newcomb Award for 1944 was presented to Dr. Charles J. Imperatori.

Report of the Memorial Fund Committee

The Committee reports a curtailment of activities because of the war. During the past three years Drs. Berry, Taylor, Shurly, and Proetz have successively retired from the Committee and Drs. Babbitt, Macpherson, Grove and Berry have been appointed by the Council, each for a term of five years. Present members have been continued in office an extra year because of the 1943 off-year.

The Committee's primary function is to administer wisely the income from the Fund, in the furtherance of laryngological research. We are persuaded that no body can better assure the wise use of such funds for such research down through succeeding years than the American Laryngological Association through its carefully chosen committee. But first we need working capital. The assessment idea promised an income of about one thousand dollars a year; but the uncertainty of war urged its temporary abandonment.

There are two other sources for the fulfilling of this so worthwhile effort. One is to place a clause in our wills so that unforseen contingencies cannot prevent our intent to pass on to succeeding generations the torch that was so carefully kept aflame for us. The other is for those who are enjoying a better than normal income just now (and there are some) to informally pledge and give a selected amount each year until a desired total has been attained. In these

days such tax-deductible gifts are a tempting and easy way to help any project in which we take particular interest and pride. The Committee would greatly appreciate an opportunity to discuss one of these two possibilities with any Fellow of the Association.

Respectfully submitted,

Dr. Gordon Berry, *Chairman*

The report was accepted and filed.

The Chairman of the Memorial Fund Committee announced the recent gift of $5,000 to the Association from Mrs. Lee Wallace Dean, Sr., in memory of Dr. Dean. By general acclamation the Secretary was instructed to write to Mrs. Dean expressing the grateful appreciation of her gift.

Report of the Committee on the Rehabilitation of Deafened Soldiers

This Committee has corresponded with the Surgeons General of the Army and the Navy and with the Director of the Veterans' Administration, offering the services of the Association in any capacity that might be helpful to the war effort, especially with regard to the rehabilitation program.

At first it was planned that all discharged casualties be taken care of by the Veterans' Administration. More recently the Army has established three centers over the country for the rehabilitation of deafened soldiers. These are staffed by competent specialists, have evolved an elaborate plan of treatment, and are well equipped. Some of the members of the staffs have asked advice from various members of your Committee.

Of late the Navy has established a rehabilitation center at the U. S. Naval Hospital in Philadelphia and is arranging for another on the Pacific Coast. When these two arms of the military service have completed these rehabilitation measures, the men are either

returned to duty or discharged into civilian life to be picked by the Veterans' Administration. It is anticipated by these services that the civilian otologist and civilian rehabilitation agencies will then come into play.

The Academy of Ophthalmology and Otolaryngology, through its Committee on Hearing Conservation is fostering a nation-wide program of rehabilitation for civilians and discharged military casualties. This Conservation Committee also concerns itself with speech rehabilitation. It would welcome official representation from the American Laryngological Association.

It is recommended that this reporting committee be discharged and that two Fellows be appointed by the President as our delegates to the Academy Committee.

Respectfully submitted,

HARRIS P. MOSHER,

Chairman.

The report was accepted and filed.

There being no further business, the meeting adjourned at 1 P. M.

FINAL EXECUTIVE SESSION

JUNE 8, 1944

The Final Executive Session was called to order by President Imperatori on Thursday afternoon, June 8, 1944.

A motion was passed to accept the Report of the Nominating Committee, and to direct the Secretary to cast a ballot for the incoming Board of Directors. The Meeting recessed briefly while the new Board of Directors retired and elected the following officers for the ensuing year:

HAROLD I. LILLIE, M.D., *President*
JOHN J. SHEA, M.D., *First Vice-President*
THOMAS C. GALLOWAY, M.D., *Second Vice-President*
ARTHUR W. PROETZ, M.D., *Secretary*
FREDERICK T. HILL, M.D., *Treasurer*
LOUIS H. CLERF, M.D., *Librarian and Historian*
JAMES A. BABBITT, M.D., *First Councillor*
GORDON BERRY, M.D., *Second Councillor*
THOMAS E. CARMODY, M.D., *Third Councillor*
CHARLES J. IMPERATORI, M.D., *Fourth Councillor*

The President-Elect was conducted to the rostrum and addressed the Association briefly.

There being no further business, the Meeting adjourned *sine die*.

List of Fellows

1944

FELLOWS

IN THE ARMED FORCES OF THE UNITED STATES

Rea E. Ashley

Edwin N. Broyles

Edward H. Campbell

Norton Canfield

Gordon D. Hoople

William W. Morrison

Lyman G. Richards

Harry P. Schenck

LIST OF FELLOWS

Date Indicates Year Admitted to Active Fellowship.

Attendance at meetings is shown by the figures following each name. Asterisk (*) indicates year paper was read.

Active Fellows

1942. Ashley, Rea E., 384 Post Street, San Francisco, California.

1923. Babbitt, James A., 1912 Spruce Street, Philadelphia, 3, Pennsylvania, 1923, '24*, '25, '26, '27, '28, '29*, '30, '31, '32, '33*, '34, '35, '36, '37, '38, '39, '40*, '41, '42, '44.

1936. Ballenger, Howard C., 723 Elm Street, Winnetka, Illinois, 1936, '37, '38*, '40, '41, '42.

1944. Beatty, Hugh G., 150 East Broad Street, Columbus, Ohio, 1944.

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, '39, '40, '41*, '42, '44.

1938. Blassingame, Charles Decatur, 414 P. and S. Building, Memphis, Tennessee, 1938, '39, '41, '42, '44.

1944. Boies, Lawrence R., 1631 Medical Arts Building, Minneapolis, Minnesota, 1944.

1941. Bowers Wesley C., 17 East 61st Street, New York City, 1941, '42, '44.

1932. Brown, John Mackenzie, 1136 W. 6th Street, Los Angeles, 14, California, 1935*, '37, '40*, '41, '42, '44.

1940. Broyles, Edwin N., 1100 North Charles Street, Baltimore, Maryland, 1940, '41*, '42*.

1913. Butler, Ralph, 1930 Chestnut Street, Philadelphia, Pennsylvania, 1920*, '21, '23, '24, '27, '29, '31, '32, '37, '38, '41, '42.

1930. Campbell, Edward Hastings, 2035 Delancey Street, Philadelphia, Pennsylvania, 1930, '31*, '32*, '33, '35, '38, '39*, '40, '41.

1942. Canfield, Norton, 789 Howard Avenue, New Haven, Connecticut, 1942.

1923. Carmody, Thomas E., 806 Metropolitan Building, Denver, Colorado, 1924, '26*, '27, '28, '30, '32*, '34, '35, '37*, '38, '39*, '40, '41, '42*, '44.

1925. Clerf, Louis H., 1530 Locust Street, Philadelphia, Pennsylvania, 1925, '27*, '28, '29*, '30*, '32, '33, '34, '35, '36*, '37, '38, '39, '40, '41*, '42*, '44*.

1917. Coates, George Morrison, 1721 Pine Street, Philadelphia, 3, Pennsylvania, 1920, '21, '22, '23, '24, '25, '26, '27, '28, '29, '30, '31, '32, '33, '34, '35, '36, '37*, '38, '39, '40, '41, '42, '44.

1939. Cody, Claude Carr, 1306 Walker Avenue, Houston, Texas, 1939, '41, '42 '44.

1937. Crowe, Samuel James, 4332 North Charles Street, Baltimore, 18, Maryland, 1937, '38*, '40, '42, ('44*).

1941. Cunning, Daniel S., 121 East 60th Street, New York City, 1941, '42, '44.

1941. Davis, Warren B., 135 South Eighteenth Street, Philadelphia, Pennsylvania, 1941, '42*.

1941. Dixon, Fred W., 1027 Rose Building, Cleveland, Ohio, 1941, '42*, '44.

1923. Fenton, Ralph Albert, 806 Medical Arts Building, Tenth and Taylor Streets, Portland, Oregon, 1923, '25*, '26, '28, '30, '32, '34, '35, '37*, '39, '40*, '42, '44.

1930. Figi, Frederick A., Mayo Clinic, Rochester, Minnesota, 1930, '32*, '34, '35, '38*, '42*.

1933. Foster, John H., 1304 Walker Avenue, Houston, Texas, 1934, '35, '37, '38, '39*, '40, 42, '44*.

1940. Fuchs, Valentine H., 1112 American Bank Building, New Orleans, Louisiana, 1940.

1932. Furstenberg, Albert Carl, 201 South Main Street, Ann Arbor, Michigan, 1933*, '36*, '38*.

1936. Galloway, Thomas Cyrus, 635 Church Street, Evanston, Illinois, 1936, '37, '38*, '39, '40, '41, '42*.

1940. Gatewood, E. Trible, Professional Building, Richmond, Virginia, 1940, '41, '42*, '44.

1940. Gill, William D., 705 East Houston Street, San Antonio, Texas, 1940, '41*.

1928. Gittins, Thomas Roy, 408 Davidson Building, Sioux City, Iowa, 1928, '31, '32*, '34, '36, '39*, '40, '42*.

1940. Goodale, Robert L., 330 Dartmouth Street, Boston, Massachusetts, 1940, '41, '42*, '44*.

1928. Goodyear, Henry M., 556 Doctors' Building, Cincinnati, Ohio, 1928, '29, '30*, '31, '32, '33, '34*, '35, '36, '37, '38, '39, '40, '42, '44.

1934. Grove, William E., 324 East Wisconsin Avenue, Milwaukee, 2, Wisconsin, 1934, '35, '36*, '37, '38, '39, '40, '41, '42*, '44.

1940. Hansel, French K., 634 North Grand Avenue, St. Louis, Missouri, 1940, '42.

1933. Harkness, Gordon Follette, 2410 River Drive, Davenport, Iowa, 1934, '35, '36*, '40, '41, '44.

1944. Havens, Fred Z., 102 Second Avenue S-W., Rochester, Minnesota, 1944.

1942. Heatly, Clyde A., 11 North Goodman Street, Rochester, N. Y., 1942.

1943. Hilding, Anderson C., 324 West Superior Street, Duluth, Minnesota, 1944*.

1928. Hill, Frederick Thayer, Professional Building, Waterville, Maine, 1928, '29*, '30*, '31, '32, '33, '34, '35, '36, '37*, '38*, '39, '40, '41*, '42, '44*.

1944. Hoople, Gordon D., Medical Arts Building, Syracuse, New York.

1938. Hourn, George E., University Club Building, St. Louis, Missouri, 1938, '40, '44.

1930. Houser, Karl Musser, 2035 Delancey Street, Philadelphia, Pennsylvania, 1930, '32*, '33, '34*, '37, '38, '39, '40, '41, '44.

1939. Hunt, Westley Marshall, 907 Fifth Avenue, New York City, 1939, '40*, '41, '42, '44.

1925. Iglauer, Samuel, 707 Race Street, Cincinnati, Ohio, 1926, '27, '30, '32, '33*, '35*, '36, '38, '39*, '41, '42*, '44.

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, '39*, '40*, '41*, '42*, '44*.

1938. Jackson, Chevalier Lawrence, 255 South Seventeenth Street, Philadelphia, Pennsylvania, 1938, '39, '40, '41*, '42, '44*.

1928. Jarvis, DeForest Clinton, Quarry Bank Building, Barre, Vermont, 1928, '30, '32*, '33, '34*, '35, '37*, '38, '39, '40, '41*, '44.

1939. Johnston, William Howard, 1515 State Street, Santa Barbara, California, 1939, '40, '41, '42, '44.

1944. Jones, Marvin F., 121 East 60th Street, New York City, 1944.

1942. Kelly, Joseph D., 140 East Fifty-fourth Street, New York City, 1942, '44*.

1921. Kernan, John D., Jr., 103 East Seventy-eighth Street, New York City, 1921*, '22, '25, '26, '27, '29, '31, '34*, '35*, '36, '37*, '39, '40*, '42*.

1942. King, Edward D., 1002 Carew Tower, Cincinnati, 2, Ohio, 1942.

1929. Kistner, Frank B., 600 Mayer Building, Portland, Oregon, 1930, 35, '37, '40.

1936. LeJeune, Francis Ernest, 632 Maison Blanche, New Orleans, Louisiana, 1937.

1933. Lierle, Dean M., University Hospital, Iowa City, Iowa, 1933, '34, '35*, '37, '38, '39, '40*, '42*, '44*.

1922. Lillie, Harold I., Mayo Clinic, Rochester, Minnesota, 1923*, '24*, '25, '26*, '28, '31*, '33, '36*, '37, '38, '40, '44.

1935. Looper, Edward A., 104 West Madison Avenue, Baltimore, Maryland, 1935, '36, '37, '38*, '39, '41, '42, '44.

1927. Lukens, Robert M., 1923 Spruce Street, Philadelphia, Pennsylvania, 1927, '28, '31*, '32, '35, '36, '38, '39, '40*, '42.

1939. Lupton, Irving Martin, 806 Medical Arts Building, Portland, Oregon.

1929. Lyman, Harry Webster, 906 Carleton Building, St. Louis, Missouri, 1929, '31, '34, '35*, '36, '38, '39, '42, '44.

1928. Macpherson, Duncan, 920 Fifth Avenue, New York City, 1928, '29, '30, '31, '33, '34*, '39, '40, '41, '44.

1941. Martin, Robert C., 384 Post Street, San Francisco, California, '41, '44.

1939. McCaskey, Carl Heber, 608 Guaranty Building, Indianapolis, Indiana, 1939, '40, '41, '42*, '44.

1933. McMahon, Bernard J., Missouri Theatre Bldg., St. Louis, Missouri, 1934*, '35, '36, '38*, '41, '44.

1931. McNally, William James, 1509 Sherbrooke Street West, Montreal, Canada, 1931, '33, '34*, '39, '44.

1940. Morrison, William W. (Commander) M.C. U.S.N.R. Navy Dispensary, Navy Department, Washington, D. C., 1940, '41*, '44.

1904. Mosher, Harris P., 127 Front Street, Marblehead, Massachusetts, 1920, '21*, '22, '23*, '24, '25, '26, '27, '28*, '29, '30, '31*, '32*, '33, '34, '36, '37, '38, '39, '40, '41, '42, '44.

1939. Myers, John Llewellyn, 1115 Grand Avenue, Kansas City, Missouri, 1939, '40*, '44.

1927. Myerson, Mervin C., 136 East Sixty-fourth Street, New York City, 1927, '28*, '29, '30, '31*, '32*, '33*, '34*, '35, '36, '37, 38, '39*, '40, '41*, '42, '44.

1937. Nash, Carlton Stewart, Medical Arts Building, Rochester, New York, 1937, '38*, '40, '42, '44.

1922. New, Gordon B., Mayo Clinic, Rochester, Minnesota, 1922, '24, '25*, '26*, '28*, '31*, '33*, '35*, '37*, '39*, '41*.

1923. Newhart, Horace, 527 Medical Arts Building, Minneapolis, Minnesota, 1923, '25*, '26, '28, '30, '33, '34, '35, '36, '38, '39, '40, '41, '42.

1927. Orton, Henry Boylan, 224 Delavan Avenue, Newark, New Jersey, 1927, '28, '29, '30*, '31, '32, '33*, '35, '36, '37, '38*, '40, '41, '42, '44*.

1927. Porter, Charles Terrell, 520 Commonwealth Avenue, Boston, Massachusetts, 1927, '28, '30, '31*, '32, '35, '37, '38, '39*, '40, '41, '42, '44.

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*, '39*, '40, '41, '42*, '44.

1934. Richards, Lyman Gilder, 319 Longwood Avenue, Boston, Massachusetts, 1934, '35, '36*, '37, '38, '39.

1930. Ridpath, Robert F., 1737 Chestnut Street, Philadelphia, Pennsylvania, 1930, '31, '33, '37, '38*, '40, '41*, '44.

1938. Salinger, Samuel, 25 E. Washington Street, Chicago, Illinois, 1938, '39, '40*, '42, '44*.

1921. Sauer, William E., 3720 Washington Boulevard, St. Louis, Missouri, 1921, '23, '25, '28, '31, '32, '35, '37, '39*, '41, '44.

1932. Schall, Leroy Allen, 403 Commonwealth Avenue, Boston, Massachusetts, 1934*, '35, '38, '40*, '41, '42, '44*.

1934. Schenck, Harry P., 1912 Spruce Street, Philadelphia, Pennsylvania, 1934, '35, '36*, '37, '38, '39, '40, '41*, ('44*).

1930. Seydell, Ernest M., First National Bank Building, Wichita, Kansas, 1930, '31*, '33*, '34, '35, '37*, '38, '39, '40*, '41, '42.

1928. Shea, John Joseph, 1018 Madison Avenue, Memphis, Tennessee, 1928, '29, '30*, '31, '32*, '33, '34, '35, '36, '37, '38, '40, '41*, '42.

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, '39, '40, '41, '42, '44.

1939. Simpson, W. Likely, 512 Physicians' and Surgeons' Building, Memphis, Tennessee, 1939, '41, '44*.

1923. Spencer, Frank Robert, Physicians' Building, Boulder, Colorado, 1923, '24*, '26, '27*, '28, '30, '32*, '34, '35, '37, '38, '40*, '41, '42.

1924. Taylor, Hermon Marshall, 111 West Adams Street, Jacksonville, Florida, 1924, '25*, '26, '27, '28, '31, '34, '35*, '36, '37, '38, '39*, '40, '41, '42, '44*.

1937. Tobey, George L., Jr., 403 Commonwealth Avenue, Boston, Massachusetts, 1937, '38, '39.

1927. Tobey, Harold G., 403 Commonwealth Avenue, Boston, Massachusetts, 1927, '29*, '30*, '31, '32, '35, '37, '39, '41.

1925. Tucker, Gabriel, 250 South Eighteenth Street, Philadelphia, Pennsylvania, 1925, '26*, '27*, '29, '30, '31, '32, '33, '34, '35*, '36, '37, '38, '39, '40*, '41*, '42.

1943. Van Alyea, Oliver E., 135 South La Salle Street, Chicago, Illinois, 1944.

1941. Viole, Pierre P., 1930 Wilshire Boulevard, Los Angeles, California, 1944*.

1933. Wanamaker, Allison T., 817 Summit Avenue, Seattle, Washington, 1933, '37, '41.

1940. Weille, Francis L., 395 Commonwealth Avenue, Boston, Massachusetts, 1940, '41, '42, '44.

1922. White, Francis W., 471 Park Avenue, New York City, 1922, '23, '25, '26, '27, '28, '29, '31, '32, '37, '39, '40, '41, '42, '44.

1939. Williams, Horace James, 5908 Greene Street, Germantown, Philadelphia, 1939, '40, '41, '42.

1942. Wishart, D. E. Staunton, 170 St. George Street, Toronto, Canada, 1942, 44.

1905. Wood, George B., Suite 504, Physicians' Building, Twentieth and Chestnut Sreets, Philadelphia, Pennsylvania, 1921, '22*, '23*, '25*, '28, '29, '31*, '32, '34, '37*, '38, '39, '40, '41, '42, '44.

1940. Wood, V. Visscher, 3720 Washington Boulevard, St. Louis, Missouri, 1940, '41, '42*.

1935. Woodward, Fletcher D., 104 East Market Street, Charlottesville, Virginia, 1935, '36*, '37, '38, '39*, '40, '42, '44.

Honorary Fellows

1939. (1907) Jackson, Chevalier, 255 South Seventeenth Street, Philadelphia, Pennsylvania, 1939*, '40*.

1943. (1914) Levy, Robert, Metropolitan Building, Denver, Colorado.

1943. (1918) Lewis, Fielding O., Providence Road, Media, Pennsylvania.

1937. Nager, F. B., Freiestrasse 30, Zurich, Switzerland.

1943. (1906) Pierce, Norval H., Del Mar, San Diego County, California.

Corresponding Fellows

1942. Batson, Oscar V., 133 South Thirty-sixth Street, Philadelphia, 4, Pennsylvania, 1942*.

1938. Blair, Vilray P., 508 North Grand Avenue, St. Louis Missouri.

1940. Colledge, Lionel, 2 Upper Wimpole Street, London, W. 1, England.

1943. Eggston, Andrew O., 653 Park Avenue, New York City, 1944.

1901. Grant, Sir James Dundas, K. B. E., Flat 4, Sheffield Terrace, London, W. 8, England.

1931. Lasagna, Francesco, Royal University of Parma, Parma, Italy.

1926. Law, Frederick M., 140 East Fifty-fourth Street, New York City, 1934, '35, '36, '37, '38, '39*, '40, '41, '42, '44*.

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, Surrey, England.

1930. Negus, Victor E., 149 Harley Street, London, W I, England, 1938*.

1941. Patterson, Norman, Nobs Creek, Ashbridge Park, Berkhamsted, Herts, England.

1937. Portmann, Georges, 25 bis Cours de Verdun, Bordeaux, France.

1924. Schaeffer, J. Parsons, Jefferson Medical College, Philadelphia, Pennsylvania, 1934.

1896. Schmiegelow, Ernst, 18 Norregade, Copenhagen, Denmark.

1936. Vialle, Jacques, 27 Rue de France, Nice (Alpes Maritimes), France.

Emeritus Fellows

1941. (1923) Barlow, Roy A., Winchester, Virginia.

1937. (1915) Barnes, Harry Aldrich, Pembroke, Massachusetts.

1941. (1922) Goldsmith, Perry G., 513 Medical Arts Building, Toronto, Canada.

1936. (1898) Goodale, Joseph L., Argilla Road, Ipswich, Massachusetts.

1943. (1932) Graham, Harrington B., 516 Sutter Street, San Francisco, California.

1940. (1917) Greene, Joseph B., 331 Haywood Building, Asheville, North Carolina.

1933. (1905) Halsted, Thomas H., 475 Fifth Avenue, New York City, 17.

1927. (1896) Hardie, T. Melville, 30 N. Michigan Boulevard, Chicago, Illinois.

1940. (1915) Hastings, Hill, 306 Medical Office Building, 1136 West Sixth Street, Los Angeles, California.

1918. (1888) HINKEL, F. WHITEHILL, 28 Norwood Avenue, Buffalo, New York.

1942. (1919) HURD, LEE MAIDMENT, Box F., Rowayton, Connecticut.

1933. (1904) INGERSOLL, JOHN M., 1700 S. Bayshore Lane, Miami, Florida.

1938. (1918) KENYON, ELMER L., 185 North Wabash Avenue, Chicago, Illinois.

1939. (1923) LARGE, SECORD HERBERT, 170 Eddy Road, Cleveland, Ohio.

1943. (1921) MCCULLAGH, SAMUEL, 116 E. 63 St., New York, 21, N. Y.

1941 (1903) PACKARD, FRANCIS R., 302 South Nineteenth Street, Philadelphia, Pennsylvania.

1942. (1931) POTTS, JOHN B., 1620 Medical Arts Building, Omaha, Nebraska.

1940. (1923) SEWALL, EDWARD C., 430 El Escarpado, Stanford University, California.

1933. (1907) SHAMBAUGH, GEORGE E., 122 South Michigan Avenue, Chicago, Illinois.

1941. (1892) WATSON, ARTHUR W., 2013 Locust Street, Philadelphia, Pennsylvania.

1939. (1908) WILSON, J. GORDON, Old Bennington, Vermont.

BENEFACTORS

Dr. Arthur W. deRoaldes

Dr. William E. Casselberry

Dr. Samuel Johnston

Dr. James E. Newcomb

Dr. Cornelius G. Coakley

Obverse of the deRoaldes medal

RECIPIENTS OF THE DEROALDES AWARD

1928. Dr. Chevalier Jackson.

1931. Dr. D. Bryson Delavan.

1934. Dr. Harris P. Mosher.

1937. Dr. Lee Wallace Dean.

1943. Dr. Ralph A. Fenton.

RECIPIENTS OF THE CASSELBERRY AWARD

1923. Dr. George Fetterolf and Dr. Herbert Fox.

1928. Dr. Ralph A. Fenton and Dr. O. Larsell.

1929. Dr. Richard A. Kern and Dr. Harry P. Schenck.

1929. Dr. Edward H. Campbell.

1931. Dr. Arthur W. Proetz.

1934. Dr. Anderson C. Hilding.

1936. Dr. Francis E. LeJeune: Dr. Joel J. Pressman.

1939. Dr. H. Marshall Taylor: Dr. Brien T. King.

1940. Dr. French K. Hansel.

1941. Dr. Noah D. Fabricant.

RECIPIENTS OF THE NEWCOMB AWARD

1941. Dr. Burt R. Shurly.

1942. Dr. Francis R. Packard.

1943. Dr. George M. Coates.

1944. Dr. Charles J. Imperatori

PAST OFFICERS

Presidents

(Year of Election)

1879. Louis Elsberg
1880. J. Solis Cohen
1881. F. I. Knight
1882. G. M. Lefferts
1883. F. H. Bosworth
1884. E. L. Shurly
1885. Harrison Allen
1886. E. Fletcher Ingals
1887. R. P. Lincoln
1888. E. C. Morgan
1889. J. N. Mackenzie
1890. W. C. Glasgow
1891. S. W. Langmaid
1892. M. J. Asch
1893. D. Bryson Delavan
1894. J. O. Roe
1895. W. H. Daly
1896. C. H. Knight
1897. T. R. French
1898. W. E. Casselberry
1899. Samuel Johnston
1900. H. L. Swain
1901. J. W. Farlow
1902. J. H. Bryan
1903. J. H. Hartman
1904. C. C. Rice
1905. J. W. Gleitsmann
1906. A. W. deRoaldes
1907. H. S. Birkett
1908. A. Coolidge, Jr.
1909. J. E. Logan
1910. D. Braden Kyle
1911. James E. Newcomb
1912. George A. Leland
1913. Thomas Hubbard
1914. Alexander W. MacCoy
1915. G. Hudson Makuen
1916. Joseph L. Goodale
1917. Thomas H. Halsted
1918. Cornelius G. Coakley
1919. Norval H. Pierce
1920. Harris P. Mosher
1921. Harmon Smith
1922. Emil Mayer
1923. J. Payson Clark
1924. Lee Wallace Dean
1925. Greenfield Sluder
1926. Chevalier Jackson
1927. D. Bryson Delavan
1928. Charles W. Richardson
1929. Lewis A. Coffin
1930. Francis R. Packard
1931. George E. Shambaugh
1932. George Fetterolf
1933. George M. Coates
1934. Dunbar Roy
1935. Burt R. Shurly
1936. William B. Chamberlin
1937. John F. Barnhill
1938. George B. Wood
1939. James A. Babbitt
1940. Gordon Berry
1941. Thomas E. Carmody
1942-3. Charles J. Imperatori

Vice-Presidents

1879.	F. H. Davis	
1880.	W. C. Glasgow	J. O. Roe
1881.	E. L. Shurly	W. Porter
1882.	C. Seiler	E. F. Ingals
1883.	S. W. Langmaid	S. Johnston
1884.	J. H. Hartman	W. H. Daly
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. F. Shambaugh
1914.	Clement F. Theisen	Lewis A. Coffin
1915.	J. Gordon Wilson	Christian R. Holmes

1916.	Thomas H. Halsted	Greenfield Sluder
1917.	John Edwin Rhodes	D. Crosby Greene
1918.	G. E. Shambaugh	John R. Winslow
1919.	Francis R. Packard	Harmon Smith
1920.	Harmon Smith	W. B. Chamberlin
1921.	Dunbar Roy	Robert C. Lynch
1922.	George Fetterolf	Lorenzo B. Lockard
1923.	Hubert Arrowsmith	Joseph B. Greene
1924.	Ross H. Skillern	Gordon Berry
1925.	John E. Mackenty	Robert Levy
1926.	Lewis A. Coffin	William V. Mullin
1927.	Charles W. Richardson	Hill Hastings
1928.	Robert Clyde Lynch	Francis P. Emerson
1929.	William B. Chamberlin	Ralph Albert Fenton
1930.	Harris P. Mosher	James A. Babbitt
1931.	Joseph B. Greene	E. Ross Faulkner
1932.	Gordon Berry	Frank 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
1938.	Ralph A. Fenton	Frederick T. Hill
1939.	John H. Foster	Thomas R. Gittins
1940.	Charles T. Porter	Gordon F. Harkness
1941.	Arthur W. Proetz	Henry B. Orton
1942-3.	Harold I. Lillie	Dean M. Lierle

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	1933.	William V. Mullin
1918.	D. Bryson Delavan	1935.	James A. Babbitt
1919.	J. M. Ingersoll	1939.	Charles J. Imperatori
1920.	George M. Coates	1942.	Arthur W. Proetz

Treasurers

1912. J. Payson Clark
1923. George Fetterolf
1932. William V. Mullin
1933. James A. Babbitt
1935. Charles J. Imperatori
1939. Frederick T. Hill

Librarians

1879. F. H. Bosworth
1883. T. R. French
1903. J. H. Bryan
1930. John F. Barnhill
1934. Burt R. Shurly
1935. George M. Coates

Librarian and Historian

1936. George M. Coates

PAST MEMBERS

Dates indicate original election to the Association

**Deceased*

Honorary Fellows

*1908. Barnhill, John F., Miami Beach, Florida.
*1893. Birkett, Herbert S., Montreal, Canada.
*1878. Bosworth, Francke H., New York City.
*1878. Cohen, J. Solis-, Philadelphia, Pa.
*1918. Dean, Lee Wallace, St. Louis, Mo.
*1881. Delavan, D. Bryson, New York City.
*1893. deRoaldes, Arthur W., New Orleans, La.
*1890. De La Sota Y Lastra, Ramon, Seville, Spain.
*1879. French, Thomas R., Brooklyn, N. Y.
*1880. Garcia, Senor Manuel, London, England.
*1903. Harris, Thos. J., New York City.
*1878. Johnston, Samuel, Baltimore, Md.
*1878. Lefferts, George Morewood, Katonah, N. Y.
*1883. Mackenzie, John N., Baltimore, Md.
*1881. Mackenzie, Sir Morell, London, England.
*1910. Massei, Ferdinand, Naples, Italy.
*1910. Moure, J. J. E., Bordeaux, France.
*1918. Oliver, H. K., Boston, Mass.
*1909. Semon, Sir Felix, Great Missenden, England.
*1889. Swain, Henry L., New Haven, Conn.
*1914. Thomson, Sir St. Clair, London, Eng.
*1903. Tilley, Herbert, London, England.
*1914. Wagner, Clinton, New York City.
*1890. Wright, Jonathan, Pleasantville, N. Y.

Corresponding Fellows

*1892. Browne, Lennox, London, England.
*1901. Collier, Mayo, Kearsney Abbey, Kent, England.
*1893. Desvernine, Carlos M., Havana, Cuba.
*1930. Emerson, Francis P., Franklin, Mass.
*1936. Fraser, John S., Edinburgh, Scotland.

*1887. Gouguenheim, A., Paris, France.
*1894. Holden, Edgar, Newark, N. J.
*1919. Kelly, Adam Brown, Helensburgh, Scotland.
*1881. Labus, Carlo, Milan, Italy.
*1902. Lermoyez, Marcel, Paris, France.
*1897. Luc, H., Paris, France.
*1894. MacIntyre, John, Glasgow, Scotland.
*1920. McKenzie, Dan, London, England.
*1880. Meyer, Wilhelm, Copenhagen, Denmark.
*1896. Mygind, Holger, Copenhagen, Denmark.
*1919. Paterson, Donald Rose, Cardiff, Wales.
*1919. Rogers, John, Jr., New York City.
*1894. Sajous, C. E. DeM., Philadelphia, Pa.
*1940. Soto, E. Fernandez, Havana, Cuba.
*1881. Thornton, Pugin, London, England.
*1913. Turner, A. Logan, Edinburgh, Scotland.
*1880. Whistler, W. McNeil, London, England.
*1901. Wingrave, Wyatt, Lyme Regis, Dorset, England.
*1894. Wolfenden, R. Norris, Kent, England.

Emeritus Fellows

*1928. Beck, Joseph C., Chicago, Illinois.
*1901. Brown, J. Price, Toronto, Canada.
*1891. Bryan, Joseph H., Washington, D. C.
*1897. Clark, J. Payson, Boston, Mass.
*1899. Cobb, Frederick C., Bradenton, Florida.
*1905. Coffin, Lewis A., New York City.
*1893. Coolidge, Algernon, Boston, Mass.
*1913. Dabney, Virginius, Washington, D. C.
*1882. De Blois, Thomas Amory, Boston, Mass.
*1892. Farlow, John W., Boston, Mass.
*1922. Forbes, Henry H., New York City.
*1905. Freer, Otto T., Chicago, Ill.
*1880. Gleitsmann, Joseph W., New York City.
*1906. Greene, D. Crosby, Jr., Boston, Mass.
*1895. Hopkins, Frederick E., Springfield, Mass.
*1927. Hubbard, Thomas, Toledo, Ohio.
*1885. Knight, Charles H., New York City.
*1894. Leland, George A., Boston, Mass.

*1943. Lincoln, William R., Cleveland, Ohio.
*1886. MacCoy, Alexander W., Philadelphia, Pa.
*1896. Mayer, Emil, New York City.
*1923. McKinney, Richmond, Memphis, Tenn.
*1901. Myles, Robert C., New York City.
*1878. Porter, William, Ocean Springs, Miss.
*1903. Renner, W. Scott, Buffalo, N. Y.
*1897. Rhodes, John Edwin, Chicago, Ill.
*1884. Rice, Clarence C., New York City.
*1905. Richards, George L., South Yarmouth, Mass.
*1878. Robinson, Beverly, New York City.
*1908. Smith, Harmon, New York City.
*1892. 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.
*1906. Ballenger, William L., Chicago, Ill.
*1880. Bean, C. E., St. Paul, Minn.
*1904. Berens, T. Passmore, New York City.
*1924. Bigelow, Nolton, Providence, R. I.
*1893. Bliss, Arthur Ames, Philadelphia, Pa.
*1895. Boylan, J. E., Cincinnati, Ohio.
*1892. Brown, Moreau R., Chicago, Ill.
*1933. Buckley, Robert E., New York City.
*1915. Canfield, R. Bishop, Ann Arbor, Mich.
*1934. Carmack, John Walter, Indianapolis, Ind.
*1889. Casselberry, William E., Chicago, Ill.
*1883. Chamberlain, C. W., Hartford, Conn.
*1917. Chamberlin, William B., Cleveland, Ohio.
*1896. Chappell, W. F., New York City.
*1882. Chapman, S. Hartwell, New Haven, Conn.
*1902. Coakley, Cornelius G., New York City.
*1913. Coffin, Rockwell C., Boston, Mass.
1918. Cox, 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.
*1935. Eves, Curtis C., Philadelphia, Pa.
*1919. Faulkner, E. Ross, New York City.
*1914. Fetterolf, George, Philadelphia, Pa.
*1901. Freeman, Walter J., Philadelphia, Pa.
*1917. Friedberg, Stanton A., Chicago, Ill.
*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.
*1913. Goldstein, Max A., St. Louis, Mo.
*1905. Grayson, Charles P., Philadelphia, Pa.
*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. Shurly, E. L., Detroit, Mich.
*1892. Simpson, William Kelly, New York City.
*1919. Skillern, Ross H., Philadelphia, Pa.
*1909. Sluder, Greenfield, St. Louis, Mo.
*1879. Smith, Andrew H., Geneva, N. Y.
*1932. Smyth, Duncan Campbell, Boston, Mass.
*1928. Sonnenschein, Robert, Chicago, Ill.
1924. Stein, Otto J., Chicago, Ill.
*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.
*1938. Vail, Harris H., Cincinnati, Ohio.
*1936. Voislawsky, Antonie P., New York City.
1896. Ward, Marshall R., Pittsburgh, Pa.
*1879. Ward, Whitfield, New York City.
*1886. Westbrook, Benjamin F., Brooklyn, N. Y.
*1924. Wherry, William P., Omaha, Neb.
*1924. White, Leon E., Boston, Mass.
*1922. Wishart, David J. G., Toronto, Ontario.
*1896. Woolen, Green V., Indianapolis, Ind.

G.F.TUCKER