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American Laryngological Association

Fifty-second Volume

TRANSACTIONS

OF THE

FIFTY-SECOND ANNUAL MEETING

OF THE

American
Laryngological Association

HELD AT SWAMPSCOTT, MASS.

MAY 22, 23 AND 24, 1930

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TRANSACTIONS OF THE FIFTY-SECOND ANNUAL CONGRESS
OF THE
AMERICAN LARYNGOLOGICAL ASSOCIATION

HELD AT
SWAMPSCOTT, MASS., MAY 22, 23 AND 24, 1930

DR. LEWIS A. COFFIN, President of the American Laryngological Association, called the Fifty-second Annual Congress to order at the New Ocean House, Swampscott, Mass., at 10 A.M., May 22, 1930.

REGISTRATION

Registration for the entire meeting was as follows:

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ADDRESS OF THE PRESIDENT

LEWIS A. COFFIN, M.D.

WE are assembled in response to the call of our Council for the Fifty-third Annual Meeting of the American Laryngological Association.

I am mindful of the high honor done me by my election to the Presidency of this Association and take this opportunity to thank you, its members, for the vote of regard and confidence which places me in this enviable position.

My first duty, the official announcement of the death of our much-esteemed and beloved President of the last year, Dr. Charles Richardson, fills me with sorrow and sadness. I am sure that each and every member feels that, in the removal of Dr. Richardson from our midst, he has met with a great personal loss. Dr. Delavan, as our historian, will, I am sure, give us a detailed account of Dr. Richardson's life and accomplishments.

Our honored and much-respected Secretary of the Treasury of the United States, at a recent birthday dinner, said: "Age comes on so gradually and unnoticed that one can be unconscious of any material change. But seventy-five years of life leads one into a reflective and contemplative frame of mind and makes one, I think, more appreciative of old associations and friendships, as well as more keenly perceptive of the changes affecting present-day conditions and habits of life."

Try, as I have, to get away from the reminiscent state of mind, it has been impossible of accomplishment, and in that frame of mind I have during the past year had many pleasurable hours: hours spent in the contemplation of the men and their work which has placed our specialty on the high plane which it occupies today; in contemplation of how, at first, apparently independent truths and facts may be woven together to form the great fabric known as acquired knowledge and progress. Tonsils have been the subject of study and discussion as to removal and methods of so doing since X A.D. whereas the much-allied growth, adenoids, was not

discovered and described by Dr. Wilhelm Mayer until A.D. 1868, and then the universal recognition and removal of that growth came about quite slowly, largely on account of the lack of standardization of method of removal. Great impetus was given, in this country at least, by the work of Dr. Hooper, who in 1888 presented a paper showing with what frequency the growths occurred and presenting a forceps which made easy the operation with or without anesthesia.

Strange as it may seem, the man who was one of the first to describe this growth and his method of removal, in New York, was the most vigorous in disagreement with Dr. Hooper as to the frequency of its occurrence, saying that he thought the frequency with which Dr. Hooper had found the condition must be due to some peculiarity of the climate of Boston. It was Dr. Delavan who said at the same meeting that he was surprised that a man of Dr. Beverley Robinson's standing should so express himself in the face of such overwhelming evidence as presented by Dr. Hooper: Dr. Hooper having reported something over 100 cases and putting in evidence nearly as many bottles containing the masses removed. This paper was read in the New York Academy of Medicine. I am sure every laryngologist who heard the paper sent to Codman & Shurtleff, instrument makers of Boston, the following morning for a pair of Hooper's forceps, and the war on adenoids was on and the multiplicity of adenoid forceps and curettes was begun. The combined operation for the removal of adenoids and tonsils was soon in vogue.

I have presented these facts because of the bearing of tonsils and adenoids on other diseases with which we have to deal. During my last years of doing active hospital clinical work the two subjects which were of the greatest interest and concern to me were "sinusitis in children" and "atrophic rhinitis and ozena."

Before discussing the interdependence of these diseases, I wish to call attention to certain historical facts and established truths.

1. As stated above, the combined operation for the removal of tonsils and adenoids was not in vogue until 1889.

2. The first pandemic appearance of la grippe occurred in 1889—occurring in this country in the last quarter of that year and the first quarter of the next. Accompanying and following this dis-

ease there were many cases of sinusitis, which forced attention of laryngologists upon the pneumatic cavities of the head as had never before been known.

3. In a paper by L. W. Dean, on "sinusitis in children," in which he had had the collaboration of pediatricians and pathologists, he has stated that they found 80 per cent of the chronic cases of sinusitis recovered from simply improving nasal drainage by the removal of tonsils and adenoids. Granting this, how much greater a percentage of acute cases would recover from the same treatment, and if prophylactically treated how greater a percentage still would be, so to speak, prophylactically cured!

When one considered that, in 1889, when this infectious grip was raging, comparatively few children had thus been prophylactically protected, one can see what an extensive and fertile field was ready in the noses and sinuses of the children of the land.

Atrophic rhinitis and ozena had been the subject of much speculation for many years, but during the latter part of the nineteenth and the early part of the twentieth century it was so persistent and so disturbing to the afflicted and his family as to be the *bete noir* of the Laryngologist and a nerve-wrecking force to the suffering.

All who have been in our special field of work for over twenty years will recall the many theories advanced as to the cause and cure of this most trying and disgusting disease. Many, and among them some of the best minds and most-authoritative writers, looked upon the condition as an independent disease and offered many pages of literature on the pathology, specific producing organisms, etc., to account for the condition. On the other hand, many of our best thinkers have from early years of the specialty considered it as a complicating sequelum of a diseased sinus or sinuses.

A most convincing paper in support of this contention was presented in 1913 by our fellow-member, Dr. Francis Emerson, before the American Laryngological, Rhinological and Otological Society.

If you have followed me you will no longer wonder that we have had such a harvest of this disease in the period to which attention has been called, and you will as well understand why now, when most children needing it have had tonsils and adenoids removed, atrophic rhinitis and ozena have, practically, been wiped from the map of disease.

For the same reason we see less of chronic sinus disease and therefore less of radical sinus work done. If this be so, we must add a large credit to the tonsil and adenoid operation. Many, both of our own and other specialties, have been shocked at the frequency with which the operation for the removal of tonsils and adenoids has been done and we have heard such critical phrases as the slaughter or massacre of the tonsil. We have all, very probably, shared in this feeling at times. However, I think we must blame, not the thousands of operations, but the disease. We will all hail the day and the man or organization, called for by Dr. Delavan in his paper of last year, that will show us the cause and the method of preventing the condition demanding these operations.

To my mind there is no doubt but that, sometimes, diet or improper feeding of our young will be found to be a contributing factor in the cause of these hypertrophies which become so fertile for infection and so physically detrimental to nasal drainage. I can remember, some twenty years ago, in a discussion of the relationship of gastrointestinal disease to disease of the upper air passage, I said to Dr. Einhorn that if he would show me the throats of his patients I would tell him their diets.

A very interesting and illuminating paper on this subject was read at the Eastern Section of the American Laryngological, Rhinological and Otological Society last winter by Dr. Jarvis of this Association.

I heartily agree with Dr. Delavan that this Association could do no more worthwhile piece of work than to promote a study of these conditions. To this end there should be appointed a promoting or steering committee, consisting of laryngologists, pediatricians, biochemists and pathologists, to direct the study. This much may be said: Our problems multiply; our field widens; the mountains of the unknown invitingly beckon to him who would wrest from them crumbs of knowledge. My belief and abiding faith is that there will ever be men to accept the invitation and thus shall knowledge grow.

Gentlemen, I declare the Fifty-third Meeting of the American Laryngological Association open for such business as may properly come before it.

A NEWER CONCEPT OF HYSTERICAL APHONIA

ROY A. BARLOW, M.D.

THE loss of the voice, the inability to communicate with his fellow-man, is not only of economic importance but also a serious affliction to the individual. The aphonic condition may be the result of a sinister pathological invasion associated with other symptoms, to fill out a clinical picture, and as such may be dealt with according to fairly definite rules and procedure, and is not to be considered in this article other than in a manner of differential diagnosis. This message stresses a condition of aphonia of functional or hysterical origin.

Hysterical aphonia is a more modern phraseology for the older term, "hysterical paralysis of the vocal cords." It is not and never has been a true paralysis of the vocal cords. It is an aphonic manifestation of an hysterical or unstable psychological back-ground.

The anatomy of the larynx and pharynx is familiar to us all. The intrinsic muscles of the larynx supplied by the recurrent laryngeal nerve, the cricothyroid by the superior laryngeal; the auxiliary muscles, such as the stylohyoid, omohyoid, etc., are enervated by the other corresponding cranial nerves. These structures are mentioned for the purpose of reference later.

The individual developing aphonia of a functional type is not psychologically normal. He has been unable to adjust himself to his environment. It may be that he has chosen a career or a business or a profession for which he is congenitally not suited. Frequently the parents have guided his education to fit in with their own desires, rather than the ability of the child. Perhaps circumstances have arisen which necessitate an occupation for which the individual is unfitted or unable to adjust to. It happens frequently that the person has become involved in irregular amorous episodes. Any one or all of these circumstances tend at some time or other to place the patient in a dilemma. The ordinary lay vernacular would say, "he got into a jam." The normal person will manage in some manner to extricate himself or, if

unable to do so, will face the issue and abide by the decision. The psychologically unstable individual will show an entirely different reaction. Mental panic ensues, judgment is warped or lacking entirely, poise is dissipated and the patient seeks escape by any means rather than "stand by the guns."

The result which follows will fall in one of three courses, viz., two permanent and one temporary. The permanent routes of escape will be suicide or insanity. The temporary route would be "nervous breakdown" or some manifestation of an existing complex so presented as to either arouse the sympathy or to render the patient unable to carry on either in whole or in part. It may be a functional paralysis, blindness, deafness, unconsciousness, loss of voice or any one of a number of clinical pictures. This, we must bear in mind, is not a malingerer—it is not the will of the patient—it is a true clinical picture, rendering as much discomfort and anxiety as that due to a real pathological development. It is all a product of the subconscious and beyond the control of the unfortunate victim.

We thus have briefly sketched an hysterical scaffolding for a consideration of the problem in hand, hysterical aphonia. The symptoms and signs are loss of voice. This is not preceded by the usual respiratory infection. It is not associated with fever, pain or the usual manifestations of laryngitis. The patient whispers. He can, however, "clear his throat," and when instructed can phonate during laryngeal examination.

Examination reveals no pathology. There is no paralysis of the cords; there is no ulceration or immobility. The cords move, but there is no tone. Examination of the cords is quite easily made because the pharynx is anesthetic in most hysterical patients. In addition to the pharyngeal anesthesia we frequently find corneal anesthesia, and other signs or stigmata of hysteria.

A careful history will frequently elicit the true crux of the situation. It may be necessary to go back a number of years; no detail is too insignificant.

A careful physical examination should be made, even thoracic roentgen-ray. Too often an aphonic patient is told he has tuberculosis and sent to some distant climate and his economic routine broken up.

I became interested in this group of cases, and together with Dr. Robert West, of the Speech Department of the University of Wisconsin, we carried on a series of studies and observations with the aid of roentgenograms. This work proved very instructive and enlightening. The material though limited in amount has been thoroughly and carefully analyzed and all doubtful findings have been disregarded.

The pictures show quite clearly the soft tissue structure. For the sake of study a number of normals were rayed not only at rest but in phonation. It was noted that for given tones or vibration rates there was always a definite pattern of the larynx and the epipharynx. There was also a definite position or relative position for the hyoid bone as related to the body of the fourth cervical vertebra.

The pattern as outlined by the larynx, base of the tongue and pharynx we have called the "phonic pattern" and will henceforth refer to it as such. This phonic pattern varied in low and high notes. In low notes where the tone is a chest tone the pharynx had a tendency to close, while in high tones the pharynx was opened quite widely. This may be explained on the basis of a secondary resonating column of air.

In the larynx itself the higher the tone the more elevation was noted in the palate and a contraction of the omohyoid muscles.

The analysis of the tone-producing roentgenograms would indicate that in a given person there is a definite position of the larynx and a definite phonic pattern for each tone. The tone depends not only upon the tension of the vocal cords but upon the coördination of the adjoining or auxiliary laryngeal muscles and structures.

The skiagraphs of the hysterical larynx were very interesting. The larynx at rest was not diagnostic. The attempt to phonate, however, resulted in a picture entirely different from any of those studied. The phonic pattern was atypical as compared to those of the normal cases. The dimensions of the pharyngeal spaces were out of proportion to those corresponding in normal cases. There was a general widening of the secondary resonating spaces, which we interpreted as due to a tension of the laryngeal muscles resulting in the victory of the larger, stronger muscles and the opening of the

larynx or in other words a lack of coördination, an unbalanced response.

The study of sound in physics demonstrates that unless the resonating column is in proportion to the nodes and internodes of vibration waves we do not have a full true tone. If the reeds from the small pipe in an organ are placed in vibration in the larger pipes, there is no resultant tone. This then may help explain the lack of tone or loss of voice in these patients.

One patient in particular gave a very instructive demonstration, as she was recovering the phonic pattern improved; but she had a set back due to outside influences and the original hysterical pattern assumed its former outlines. Since that time at various intervals roentgenograms have been taken and the normal pattern has maintained its form.

The consideration of this series of studies brings to light an entirely different concept of "hysterical aphonia." The type of dysfunction is of such a character that no one nerve lesion could be the fault, nor would it be logical to suppose that multiple lesions would cause the same. The same law or tendency reigns here as in other hysterical manifestations, an unreasonable or illogical distribution of disturbed functions.

The desire to speak is present, the stimulus to phonate presents itself by the time it reaches the larynx. There is no destruction of tissue; there is no true paralysis; the lack of phonation is supposedly due to a tension or disproportion of resonating spaces and vibrating medium, or whatever it is, which is essential to the production of the human voice.

The treatment in the past has been directed to the larynx. Steam inhalations and cold and hot compresses have been used. Instillations of menthols, etc., have been advised. Some laryngologists have used faradic electrical current, others have used static spark. There have been cases restored by such procedure.

History gives us case reports where patients have been voiceless for years and, due to some violent, unexpected accident, have spontaneously recovered the voice to full capacity. An auto accident, a fire, an electric storm and recently we have newspaper accounts of aeroplane stunts resulting in voice recovery as well as auditory phenomena, by sudden descent in an unexpected maneuver

in an *aéroplane*. We all have, in our practice, cases of shorter duration, and we have felt instinctively that, having ruled out true pathology, we were dealing with a psychic disturbance.

The true message of this paper and the real object lesson to be derived from this study must be in our sympathetic understanding of these cases. Having made the diagnosis it is utter folly to institute local treatment when the case can be placed in the hands of a competent psychoneurologist. This rule is just as necessary for us to follow as to place a patient whom we have diagnosed as tubercular or syphilitic in the care of a competent physician who looks after those cases.

The treatment then is essentially psychoanalysis. This form of therapy may not necessarily cure the patient; but it is unreasonable to believe that by local measures we can cure a condition which is so deep-seated in its origin: hysterical aphonia is a symptom.

It is even a warning that the patient so afflicted is not a normal person and is beginning to "break" under the stress of struggle for daily bread. It is not enough that we clear up the immediate manifestations, we must see that his environment is adjusted, even, if necessary, a complete change of occupation or surroundings. The patient is unable to adjust to his atmosphere, therefore we must adjust the atmosphere to him.

This aphonia condition may be a warning that subconsciously the patient is using a temporary route of escape from a dilemma, and if we do not take sufficient cognizance of its import he may later follow one of the two permanent routes, insanity or suicide.

This paper is an effort to bring about a clearer understanding of the significance and gravity of the etiological disturbances back of the aphonia; it is a plea to investigate more thoroughly the background of these cases and to see that the patient is placed in the charge of a competent psychoneurologist for the subsequent handling of the problem.

We are going to see more and more clinical pictures such as are herein described; for the business world today is one of the keenest competition and the driving force is tremendous. Greater numbers of individuals will weaken under the strain, and it is our duty to give them every consideration rather than allow them to fall into the hands of some cult or quack form of therapy.

SUMMARY. The roentgen-ray study has given a rather different mental image of the laryngeal changes in hysterical aphonia, primarily dispelling the older term of "hysterical paralysis." It also opens up possibilities of means and aids in the teaching of proper phonation and vocal training. Already it is in use in the instruction of foreign language, and as more data is accumulated we may find this roentgen-ray study itself of value.

Hysterical aphonia should signal to the laryngologist that the patient is failing to adjust to his surroundings, and it should be our duty to see that proper steps are being taken to prevent a recurrence of the disturbance.

DISCUSSION

DR. GEORGE L. RICHARDS, Fall River, Mass.:

I have been very much interested in this question of hysterical aphonia for several years. Dr. Loeb used to say that he cured them by telling them to speak the sweetest name in the English language, "Home, Sweet Home."

Dr. Barlow seems to have gone farther back in the etiology than most of us have done. He speaks about a certain class of cases, the result of the high-pressure way in life in which we now live and are going to live; but what occurs in the case of the younger person who really has not reached any such stage as that?

I have recently had a young French girl who has had a severe case of hysterical aphonia. All the old classical ways of using electricity, "Home Sweet, Home," bad French, trying to grasp her by the thyroid and all the various tricks failed to bring the voice back. Yet after a period of time, all of a sudden, one day she talked again. The voice will remain good a few weeks, perhaps a few months, and then it goes once more. Is it the proper thing for us immediately to send such a case to a psychoanalyst or to have a thorough physical examination or try to find what is the trouble? There may be something in the social status or ups-and-downs of relations with their fellow-girls or with young men or otherwise in the early teens that the average doctor, I for instance, have not gotten hold of. I think this is a very timely thing to discuss.

There is not any pathology as you objectively look at these patients.

I would like to have Dr. Barlow, in the discussion, tell us a little bit more of the way of getting at these patients, or shall we send them all to the present-day psychoanalyst? That is not always possible in the smaller towns.

DR. FRANK ROBERT SPENCER, Boulder, Colo.:

I see a number of girls, eighteen to twenty, in the university who have attacks of hysterical aphonia. As Dr. Barlow has intimated, some mental upset may be responsible for the attack. They often do not require very much study. Those patients who have recurrent acute attacks, however, may have some disturbance of the endocrine system. Many girls eighteen or nineteen, are suffering from hyperthyroidism, either with or without some palpable enlargement of the thyroid gland, and an endocrine study of patients having recurrent attacks offers relief if the proper treatment is given.

Dr. William J. Mayo emphasized an important point in connection with these patients some years ago, and Dr. Barlow has intimated this in his paper. With the increased development of the brain and spinal cord in modern civilized races, with the higher education and strain of civilization, there must be some degeneration of the sympathetic nervous system. Dr. Mayo says this is most frequently observed in the fair sex. He explains that many of the neuroses we encounter in the practice of medicine are due to this. He says none of us live long enough to make any very extended observations along this line, but if we can change their environment for a time and put their minds at ease regarding some worry they have, the hysterical aphonia may disappear in a short time, as you all know.

DR. ERNEST M. SEYDELL, Wichita, Kans.:

Hysterical aphonia is an extremely interesting condition. In the *Denker and Kahler Handbuch for Diseases of the Ear, Nose and Throat* the chapter dealing with this subject is entitled "Psychogenic Disturbances of the Motility of the Laryngeal Muscles." The author, Dr. Amersback, states that the terms psychogenic and hysterical are not identical in meaning.

A psychogenic person may not be hysterical and, more than this, the hysterical reaction is subordinate to the psychogenic. According to this author, any paralytic position of the cords may be simulated by a psychogenic condition. Muck has even reported a case in which there was a unilateral involvement of the larynx and the cord was found fixed in the cadaveric position.

According to Gaupp, we may distinguish two forms of psychogenic disturbances of the larynx: (1) thymogenic or hysterical, which may be caused by severe fright, etc.; (2) idiogenic or psychohypochondriacal, some of which arise through fear and others by hypochondriacal imaginations. I have found that it is much easier to relieve the aphonia in cases which belong in the first group than those which belong in the second. Those belonging in the second group must frequently be referred to a psychiatrist.

My procedure in the treatment of these cases is to make a very careful examination and assure the patient that there is no organic reason for his loss of voice and that I will be able to restore his speech with his coöperation. The treatment is given in a quiet room with no one else present. The patient is first asked to take deep-breathing exercises. Next, firm pressure is made over each side of the larynx and, at the same time, the patient is asked to produce a deep, fluttering tone. This is repeated a number of times and then he is required to hum in a normal, low tone. With this accomplished, it is a simple matter to have the patient articulate—first numbers, then words, and finally speech. I always attempt to overcome the difficulty at the first sitting, as I think this is important.

DR. WILLIAM V. MULLIN, Cleveland, Ohio:

For some time I have been studying the innervation of the larynx, one object being to determine the influence exerted by the sympathetic nervous system on the movements of the vocal cords. We have done considerable work with dogs, but they are unsatisfactory subjects, as it is difficult to judge the effects on a dog's voice. Our conclusion from these experiments so far is that the sympathetic nervous system does not have any direct influence on abduction or adduction. Clinically, however, I feel that it does have some bearing in the type of case discussed by Dr. Barlow.

I have observed one clinical case in a young girl who had a neurocytoma involving the cervical sympathetic ganglion on the right side, with a typical Horner's syndrome. Examination of the vocal cords both before and after operation failed to reveal any disturbance. In Dr. Barlow's cases, aphonia is only one of the symptoms. The patients also are lethargic and much let down, they have cold hands and feet, and are just the opposite of the type described by Dr. Spencer, in which there is hyperthyroidism with hyperfunction of all the senses. It is my belief that hyperthyroidism affects the larynx only as it does all the muscles.

Commenting on Dr. Seydell's discussion, I cannot believe that any form of hysterical aphonia will cause a vocal cord to remain in the midline, as in a true involvement of the recurrent laryngeal nerve. Probably this cord was temporarily held in spasm, and if reexamined later would have been seen to move.

DR. MERVIN C. MYERSON, Brooklyn, N. Y.:

We are all agreed that a successful laryngologist must mix a little psychology with his laryngology. That prompts me to say that we appreciate that hysterical aphonia, when we see it, has a psychological basis. Therefore, I would like to ask Dr. Barlow to explain just what he means by the newer concept of hysterical aphonia.

The matter of a laryngologist bordering on charlatanism when he uses local methods or local applications in the throats of these patients does not quite strike me as a fair statement, because I do believe that we give these patients a little psychological boost when we do something to them that is harmless, of course.

In order to make this study of real value, I should think that we ought to be able to state in some way how many cases of hysterical aphonia have been followed by insanity or by suicide. To my knowledge, I do not know of any, and I have seen some of these cases in the past few years.

We know when an individual is lifting a weight that there is a fixation of the larynx in the open position, that the larynx is fixed in a closed position when the physical effort is very intense. We also know that there is an elevation of the larynx during phonation; we take advantage of this when we examine the larynx. The contraction of the muscles that control the muscular action of the larynx accounts for this. So these x-ray pictures, though very instructive in their way, are rather limited, and I do not think that they have a practical application because they do not tell us anything more than we appreciate in our routine examination of our patients.

I do not mean to be critical.

DR. ROBERT SONNENSCHN, Chicago, Ill.:

There are just two points I would like to mention very briefly. It is needless to tell this Society that, according to the late Sir Felix Semon's law, in a slowly developing paralysis of organic nature it is always the cricoarytenoideus posticus that is first involved. Later, when the nerve is completely out of function, of course, all muscles are involved. In a functional disturbance it is always the abductors,

the most powerful group of muscles in the larynx, that are first involved. It was he and others who called attention to the fact that this hysterical aphonia is a paresis and not a true paralysis, and that it is the adductor group which then is involved.

I mention this because I am very much surprised at the quotation from the *Handbuch* of Muck's case. How that could possibly be a functional disturbance when all the muscles were involved, and one had a picture of complete recurrent paralysis, I do not know. Either it is a very rare exception to Semon's law or else Muck made a mistake.

The second point, as I have seen it, and I am certainly no psychologist, is this: In the treatment of hysteria one must have complete assurance in himself in order to impress the patient with the fact that he will be cured. Therefore, a very careful, accurate diagnosis is necessary, and then you can say to the patient: "You will speak." The simple measure that I have used in the few cases I have seen has been simply to tell the patient to practise breathing at home, and to say: "Tomorrow you will be able to whisper a, e, u." He comes in the next day and I say: "You will be able to speak thus and so," and usually by the third day he is able to yell.

DR. BARLOW (in closing):

Dr. Richards spoke of these girls in their teens. That is just what I mean by the newer concept of hysterical aphonia. Whether the patient is fifty, fifty-five, or whatever the age may be, as a rule, the original disturbance dates back to the teens. It is that complex existing in the subconscious mind, for years held down by the censorship of the conscious mind, which has allowed itself no expression whatever, until finally the constant bombardment of this impetus to get out expresses itself in some visceral disturbance, whether it is a visual disturbance, hysterical deafness, hysterical loss of voice, paralysis of the arm, unconsciousness or what not.

I will briefly give you the history of a girl, aged nineteen years, a teacher in one of the grade schools, who presented herself shortly after the onset of mid-year examinations of her class. As soon as the examinations were over her voice was restored automatically. The following spring at the approach of examination time she again presented herself with a loss of voice, unable to teach her classes. She was a brilliant girl. As Dr. Mullin called to your attention, these individuals usually stand near the head of their class, but they are psychologically unstable and belong in that emotionally illiterate class. That is an expression I am very fond of. Her parents were Swedish and they looked forward to the daughter being a teacher.

To their minds that would bring the greatest credit to their family they could conceive of. The girl was not fitted for a teacher, not because her mental capacity was insufficient, but because she was not the type who could guide the mental activities of other people; nevertheless she was going to be a teacher and she was a teacher, and she graduated from the normal school with high honors. When it came to the practical application of her instruction and teaching her class, she was unable to carry on the work. The only form of escape which she could manifest was the loss of voice, not a malicious thing, though it allowed the temporary escape at examination time when she realized her class was not going to stand up well on the yearly examination in June.

That girl was examined. The history was elicited and upon changing her vocation from that of a teacher to working in an office where she had mostly filing work to do and clerical work in which she did not come in contact with many other individuals, she never had any further manifestations. There were no local measures instituted.

Dr. Spencer spoke of endocrines. We feel that here we have other influences which are brought to bear and they are not true hysteria cases. These cases had been all classified and gone over by the department of psychology. I purposely had that done because I am not a psychologist. So there is no question about the diagnosis of their psychological attitude.

I have never seen the position of recurrent paralysis of the vocal cords as quoted in the *Handbuch*. No doubt these patients are of low-grade sympathetic or, we will say, unbalanced sympathetic system individuals. They are congenitally that way. They are the individuals who cannot stand the normal wear and tear of life, especially as we have it today. The normal individual is the person who can adjust himself to whatever circumstance or condition arises in life and carry on with the material and tools he has at hand. When a person talks about being fitted for one particular phase of work, or qualified to follow one or only one profession, and the minute he gets out of that profession he makes a miserable failure of himself, right there he diagnoses his own case as a psychologically unstable individual, because if he were stable and normal (and there is no criticism meant here), he would adjust himself to his environment, and where we find a man making a failure in life because he is not in the proper groove, then that individual congenitally is not normal to start with. It is no disgrace at all, he cannot help it, but I am simply offering that as a differential of the type of patient.

I did not mean to give the implication that the loss of voice would be followed by suicide. My statement was to the effect that these hysterical manifestations were a method of escape from a dilemma and that sometimes these might go on to the more permanent type of suicide or permanent insanity.

The x-rays, as I said in my summary, were not of startling character and brought out nothing in particular. They were exceedingly interesting to me, and I thought they might be of interest to you.

We may treat our patients locally in the office. We should treat them locally in the office until we have made up our minds exactly what the condition is, and also to gain the confidence of the patient; but when we spray the larynx or do some office manipulation and tell the patient he is cured, we have not cured him because he will present himself with that same condition again if the circumstances arise the second time, as they did with this girl on the second examination period.

We must keep these patients under our observation long enough to gain their confidence, and I might say also that you cannot get anywhere with them until they fall in love with you. That is the one means the psychoanalyst assumes, that the patient must fall in love with the examiner and so get the confidence to unfold the story which dates back perhaps to puberty.

WHAT CLIMATE FOR UPPER RESPIRATORY DISEASES?

JOHN F. BARNHILL, M.D.

"Go to Arizona and you will be well in a week." This statement was made to a patient who had been operated on for a chronic frontal-sinus suppuration, but who continued to suffer severely because of failure of the wound to drain properly or to heal. The patient was in despair and, of course, eager to journey at once to a land so full of promise. The advice was given by a consulting surgeon in a tone of authority that seemed to admit no argument or doubt. The speaker did not state just where the patient should go in Arizona, nor did he give any directions as to her mode of life after arrival. The inference clearly was that merely to live anywhere in that region for a brief period was all that was necessary. The surgeon had never been in Arizona and knew almost nothing of its climate or of living conditions there. Considering the pathology present in the affected sinus, the advice was absurd. The patient needed a secondary operation and not climate.

The above circumstance is narrated chiefly to call attention to a practise, not wholly uncommon, whereby patients are urged to change climate without first having used every means to ascertain the exact extent and nature of the affection from which the patient suffers or to determine definitely what region is most suitable to the disease in question. One need scarcely say that such loosely given, unwarranted advice can lead but to grief for all concerned, to financial hardship in many instances, and, of course, ultimately to loss of confidence in medical leadership. Over-enthusiasm on the part of some physicians in recommending climate as a positive cure-all, no doubt, originated to some extent in regions most reputed to possess the valuable asset. Local pride and commercial interests indigenous to favored climatic areas make difficult the path of one who wishes to arrive at exact, practical facts. Boosters' and optimists' clubs, realtors' boards, chambers of commerce, publications of many kinds, and even railroads, have fathered glowing accounts of their respective resort countries, and these have had much influence in shaping

the views of both physicians and laity concerning the value to health from residence there. Careful observers ever since the time of Hippocrates have believed in the value of climate as a therapeutic measure. It is reasonable, therefore, to assume that a view held for so many centuries is still worthy of sincere consideration. Therefore, the criticism just offered is directed against unjustified enthusiasm and is not intended to discourage the employment of a valuable medical asset.

The belief so tenaciously held by the laity and a large portion of the medical profession that the southwestern and southern portions of our country possess more health-giving, health-restoring virtues than the more rigorous regions to the north, has been controverted lately, but not shaken, by geographers and other scientific workers, including some physicians. The geographers claim to have proven that the climate of the more northerly region is better than that lying to the south, and certain physicians have concluded that in the matter of taking cold, an important item in the inception of many serious diseases, the question of climate is of little or no importance. Since this conclusion is quite revolutionary, it is, perhaps, well to state the bare facts on which it is based. I shall confine these chiefly to the investigation of W. H. Barrows, of Stanford University, whose work and deductions are similar to others. Barrows' researches relate to the question of the relative frequency of colds among students in widely separated universities and under vastly different conditions of climatic environment. At Stanford, where the climate is mild, equable and, indeed, rather ideal, practically the same number of men per 100, among those investigated, suffered from coryza under every circumstance, as to mode of living, as suffered from the same affection among students at Harvard, which is located in a comparatively rigorous climate. Likewise, women students of Stanford and women students of Wellsley—the latter, also, in a cold, comparatively disagreeable climate—living under similar conditions in every particular except climate, also were affected in each institution by practically the same number of infections of the upper air tract. Barrows drew the conclusion from these studies, as did all the others who pursued similar investigations, that climate is not a major factor either in the production or avoidance of colds.

The conclusions of geographers, although conducted along entirely different lines from those of Barrows and others, are equally interesting and one may add, rather surprising. Huntington, a professor at Yale, discusses, among other questions relating to climate and civilization, the important item of the ideal climate, and leads the reader to infer that the most healthful parts of the United States center about New England, the St. Lawrence River country and the region around the Great Lakes. Huntington's conclusions are based not alone upon his own extensive researches, but also upon the researches of many other presumably noted geographers. Among other interesting things concerning the ideal climate, Huntington states that the average annual temperature should be near 64° F.; that neither extreme heat nor extreme cold is ideal, but that regions having a considerable difference between day and night temperatures should be regarded as favorable conditions. He, further, argues that sections having frequent, violent storms are most suitable places of residence as regards health; that an atmosphere well saturated with moisture is better than a dry one, and that but a moderate number of days of sunshine is preferable to a constant or almost constant succession of days having clear skies. It must be evident from a study of the work of this author that he does not regard those regions of the United States which are commonly regarded as having a climate of superior quality, as any better, if as good, as those parts which are known to be cold, damp, changeable, and, altogether, wholly disagreeable during the winter season. In any attempt, therefore, to estimate the value of climate in relation to disease, the physician must form his own estimate as to which of the foregoing opposed opinions is most nearly correct.

This brief paper is an effort to analyze existing differences of opinion, and to arrive, if possible, at some conclusion which may be used as a safe guide in practice. To do this I have consulted, in addition to the research work already quoted, the records of the United States Weather Bureau, reports of insurance companies, a few of which have lately given considerable intelligent effort to ascertain the cause of infections of the upper air tract; otolaryngologists who are known to be competent observers and reliable reporters; and last, I have turned to whatever profit I might,

my own observations, gained by extensive travel and numerous brief periods of residence in favored resort places. I fully realize that my own personal observance covers periods entirely too brief to be of great value except in the matter of estimating the worth of other information.

I regret that some of what I have to say seems personal and dogmatic, but this treatment of the subject appears necessary since our present knowledge is limited and, therefore, all discussion must be more or less doctrinal. I trust such errors as are made will be regarded as errors of observation and will not be looked upon as expressions of bias or prejudice.

Statistical information, as well as my own tabulation of facts, leads to the conclusion that there is no perfect climate anywhere. Many boosters say there is, but even the greatest enthusiasts of the most-favored regions privately admit defects in their environment. These deficiencies vary in different regions: in one, that may be widely heralded as perfect in every respect, it is admitted that there is too much rain or fog, or the difference between the temperature of midday and midnight is too great. In another it is too dry, and there may be too much sunshine. In still another, perfection of climate is marred only by an occasional drop in temperature, of from 15° to 25° F., which is usually accompanied by disagreeable winds and lasts three or four days. Then, too, in some otherwise splendid climatic areas of the desert, occasional dust storms occur which lower the climatic rating.

All the above-enumerated imperfections are but trivialities to those in good health, and may mean little even to those who suffer from changeable temperatures, provided proper clothing, shelter and heat are provided. Mention is made of them for the reason that suffering and ultimate harm is almost certain to come to those who seek such climate in the belief that no bad features are ever met with, and consequently go unprepared to meet them.

The crowded waiting-rooms and busy lives of physicians who practice in health-resort places seem to belie the reputation of such regions as health-giving localities, and to indicate that the climate is neither a preventive of disease nor a cure for any affection. While physicians admit that infections of the upper air tract are frequently met with in their favored climatic environ-

ment, they are quick to explain the circumstance on the ground that such places are filled with visitors from less-favored regions and that these bring with them the foci of infection from which they formerly suffered. They seem certain that the course of such infection is not so severe and not so prolonged as in less-favored climatic regions. All stoutly maintain that complications occur with less frequency and that sequelæ are less common than elsewhere. They are keen to assert that those who take cold readily in the good climate, do so not because of infection from without, but rather from autogenous sources which the patients carried with them from a former environment. These arguments are, I believe, substantially correct, for it is undoubtedly a fact that a large percentage of all disease treated in resort places is imported; and in the case of upper respiratory infections it is a fact, usually demonstrable, that persons who harbor chronic foci of infection often reinfect themselves, producing thereby all the symptoms of a primary acute rhinitis or other inflammatory ailment. It is just this type of patient—the one who carries a chronic focus—who is seldom free from cold in a rigorous climate, that most readily suffers a breakdown even in the most favorable climate on the occasion of sudden temperature changes. This brings back again the question of what constitutes a favorable climate. Clinical experience is not in accord with Huntington that the most favorable climate is one in which *the difference in temperature between day and night is quite marked*. Indeed clinical experience convinces one that the exact opposite is true, and that but slight changes during the twenty-four hours have great advantages in the climatic therapeutics of a large number of infectious conditions of the upper air tract.

A mild, equable climate, *one having no marked or sudden disturbances of temperature*, is best because it is most suitable for living in the open. The benefits to be derived from outdoor life are easily understood if it is recalled that a majority of all diseases of the respiratory apparatus are of bacterial origin, and that such affections are spread from one person to another chiefly in confined, stuffy places. This fact is exemplified during the winter season in every region of the north. Confinement indoors and close contact with infectious individuals is common, with the result that many

suffer more or less constantly from October to May from some form of respiratory affection. Should these same persons change residence to some salubrious region of the south or southwest, where outdoor life is almost constantly invited and practised, most former infections will disappear. I have personally witnessed at one playground in south Florida a striking example of this fact. The place is called the old man's paradise. It would seem well named, for one may see on any day during the winter season thousands of aged men and women at play in the open, all wildly gesticulating, talking loudly, or even quarreling violently, just as children do at sport. All are having a happy time of it and few are ever heard to cough or sneeze.

While witnessing an outdoor exhibition such as this in midwinter in which so large a number of elderly people are joyously participating, the observant physician inevitably recalls his clinical experience with old people in a rigorous climate, and honestly concludes that were these same men and women in their northern home, and practically all would be confined in stuffy, heated rooms, many would be suffering greatly from respiratory affections, and not a few would be seriously or even fatally ill from infection or some sequel of infection.

What I have just described at the old man's paradise I have seen enacted differently under different circumstances and environmental conditions from Los Angeles across the continent to Miami Beach, for the climate of many favored places is such as to encourage life in the open with comfort, and the effect on those who avail themselves of the advantage is much the same everywhere. An enthusiast once stated to me as a fact that when old people live as long as they can in a bad climate, should they then come to his most favored climate, they always live ten years longer. There is, one must admit, some basis of truth in this assertion.

The preceding discussion of this subject has little value unless from it some practical deductions may be drawn. Two questions have chief interest: What class of patients do best in the most-favored climates, and which among such favored regions is best for the individual patient? These questions can be answered only in a very broad and general way. First, as to what type patient? The class of patient most benefited is that characterized by the

person whose resistance is low, who takes cold easily and who suffers more or less constantly from some form of inflammation of the upper air tract from the first frost in autumn to the last frost in spring. This group includes moderately mild cases of paranasal sinus infection, especially of the ethmoids; rhinitis, pharyngitis, bronchitis and asthma due to bronchial inflammation. The type embraces a large percentage of all diseases affecting the upper respiratory tract. Mild though intractable suppurative processes of the middle ear and mastoid process often promptly improve or heal completely in a favorable climatic environment. However, it is neither justifiable nor expedient to send a large percentage of such patients away from home environment. Change of climate is in most instances still a rich man's experiment, and, therefore, to send any one away unless that person has ample resources or is fully assured beforehand of means of comfortable existence in the new environment, is to approach dangerously near to the commission of an inexcusable wrong.

Types which are but little benefited, or are not at all improved, by climate include accessory nasal-sinus suppuration in which the affected sinuses have suffered marked pathologic change either of mucosa or osseous structure; also affections of the middle ear in which areas of necrotic bone or diseased and decaying ossicles are present, and mastoiditis the chief pathology of which is cholesteatoma, sequestra or bony necrosis. No stage of syphilis of the nose, throat or ear is directly benefited, and, of course, affections such as otosclerosis are not improved, except through mental and physical betterment which sometimes are helped as are other bodily ailments. Another class but little if any benefited is that of nasal deformity, either congenital or acquired, but which results in more or less complete obstruction to respiration. It is, of course, irrational to advise patients such as these to change climate in the hope of receiving permanent benefit. Seriously affected sinuses, chronically enlarged and infected adenoids and tonsils, nasal deformities from whatever cause and mastoiditis of a nature above described should be operated on in any climate. After operative measures have been carried out successfully, residence in a favorable climate is undoubtedly beneficial.

Second, how may the physician select the most suitable climate

for the individual patient? This question is difficult to answer satisfactorily for the reason that our most-favored regions have not been well and scientifically mapped and charted from the standpoints of health and sanitation, and consequently much of the knowledge most readily available is little more than private opinion. L. I. Dulin, statistician for the Metropolitan Life Insurance Company, comments, as have others, on our tardiness in capitalizing and utilizing our valuable climatic assets. Dulin says: "The Committee on Public Health Climatology of the American Public Health Association has been trying over the past few years to secure some support for the preparation of an atlas of medical or public-health climatology for the United States similar to Europe. In Switzerland, physicians also have been most active for nearly half a century in collecting physiological data in association with climatic data. We have no effort of that kind under way in the United States at the present time."

Fortunately the wide extent of our southern and southwestern country provides a great variety of choice in the selection of a region having a climate suitable to the comfort of the patient and the needs of almost any disease. Including a broad belt from ocean to ocean, as it does, and crossing mountain ranges, plateaus, deserts and fertile plains, many environments may be found that are cold in elevated places, subtropical near sea level in the extreme south, hot and dry with perpetual sunshine in desert stretches, and cool and pleasant with considerable fog in certain coast districts.

Most patients who suffer from infection of the upper air tract, and from bronchial inflammation do best in a region that is moderately dry, has an abundant but not perpetual sunshine, and one that *is not characterized by marked and sudden temperature changes*. In brief, a region which provides a climate that makes continuous life out of doors possible, comfortable and pleasurable. It should of course, be understood that the most nearly ideal climate is usually valueless until modern sanitation, including provision for pure water, correct housing, proper drainage and the elimination of insect pests, has been added. The most favorable climate of any region must forever be linked with the advances of civilization and science.

There are numerous regions in our southwestern and southern

states which closely approach the conditions of most favorable climate. All are, of course, not equally good in every respect, but many well-known resort places in Arizona, New Mexico and Texas have praiseworthy climate to which properly selected cases may be sent with assurance of benefit. However, sections which most nearly attain the ideal are the coastal regions of southern California and southern Florida. And, assuming that the item much emphasized in this paper is correct, namely, that a close approach to equality of temperature of day and night is best for the type of affections under consideration, then, of these two the southeastern coast of the latter, from Palm Beach to Key West, has some advantage, chiefly because of close proximity of the Gulf Stream. The nearness of the great natural warm-water current assures the neighboring shoreland of a climate almost constantly suitable to outdoor life, and hence is well adapted to the needs of those who suffer from certain well-known types of respiratory disease of the upper air tract.

There is substantial basis for the assertion that in portions of our southernmost states there are climatic conditions that are equal, and in very many instances quite superior to much vaunted resorts in foreign lands. And, while it is true that much of this vast stretch is not developed, and is not at present suited to the reception of ailing visitors, there are numerous regions and many institutions already established that are giving commendable, highly satisfactory service to those more seriously sick. There are, also, climatic environments where, chiefly through living in the open, those less seriously infected may regain health in a wholly pleasant and agreeable way.

Because of the very limited time for presentation of this paper it has been impossible to present the subject with any degree of fulness. On this account I wish now, in closing, to emphasize the following points:

1. There is regrettable laxity on the part of some members of the profession in prescribing climate.
2. There is no perfect climate. Occasionally unpleasant conditions arise, even in the best, which may cause discomfort with back sets, and even danger to life, unless anticipated and sensibly met.

3. The best climate is unfavorable unless a modern sanitary environment has also been established.

4. The most valued features of any climate, in so far as they relate to benefit of respiratory infections, are mildness and *freedom from marked change of temperature from midday to midnight*.

5. Respiratory diseases most benefited are those infections the pathology of which is not greatly advanced and emphasized.

6. That climate has best therapeutic value in the relief of respiratory infections which permits a maximum of pleasurable, comfortable outdoor life.

DISCUSSION

DR. GEORGE L. RICHARDS, Fall River, Mass.:

I think he omitted Cape Cod altogether. If he was never down there, I will take him down and show him a climate more perfect the year round than either California or Florida. We have the requisite amount of moisture and stimulus that you have in southern Florida. I was once in southern California for a while and I nearly froze to death every night and baked every day at noon. We do not do that on Cape Cod.

Seriously, we tried out in New England for some years several tuberculosis sanatoriums. The patients used to be sent to all these other climates. We have found that our tuberculous death-rate has diminished under careful attention to the hygiene of the disease in our state sanatoria right here in the climate of Massachusetts. There is a steadily decreasing death-rate and a steadily decreasing number of tuberculous patients who see us in our office.

I think climate is a question of teaching a patient adaptability.

DR. NORVAL H. PIERCE, Delmar, Calif.:

It is not all climate, either. It is a matter of whether you are going to live in a smoky, noisy locale where the atmosphere is surcharged with the exhalations of innumerable automobiles, or whether you are going to live in a place that is without stress, is peaceful and quiet and where the weather sometimes, and rather frequently, is such that it approaches one's concept of heaven. I think that is more important than whether a certain geographical part of our country has a greater death-rate or a lesser death-rate, whether the temperature registers high at noon and low at night. There is a good deal more than just

temperature, and there is a good deal more, perhaps, than mere sunlight. But this I can say, that if you can live outdoors always, if you have the opportunity of living outdoors in the sunlight, that contributes to health.

There is not the slightest doubt in my mind of that. We have, perhaps, just as much catarrhal inflammation out on the West Coast; we have probably just as much sinus disease. There is no place that is more disagreeable, perhaps, than the centers of our large cities out there. That is true, one after another, of all large cities; but I know from my personal experience that I have arisen from the dead, as it were, and I attribute my ascension largely to the climate of southern California.

DR. GORDON BERRY, Worcester, Mass.:

The "weather" is not only an easy subject to discuss, but he who investigates will find it a fascinating study. My own searchings bring me to the conclusion that a moderate, changeable climate serves well in chronic catarrhal or obstructive forms of deafness. I have tried and found wanting the foggy climate of the seashore, the damp climate of the river basin, the harsh dryness of Colorado. Similarly, one may avoid, if he will, the warm humidity of the South, the extreme cold of the North, the relative lack of sunshine along the Great Lakes.

We have all observed benefits gained by a trip to some other climate. Is this due solely to the rest that is gained and to the change in the mode of living? Usually these cases continue to gain for a year or two. Then, if they remain on in this new environment, they begin to be subject to the prevailing catarrhal troubles of that district. Are they reverting to former unfortunate hygienic or dietetic habits, or have they become susceptible to endemic infections which they can avoid only by moving on again? Any otologist who shares with me the handicap of partial deafness, and who has as yet failed to find the ideal climate, has at least this cheering thought (if ever he reaches this Utopia), his hearing will be better, but he may starve for lack of ear-work to do.

DR. BURT R. SHURLY, Detroit, Mich.:

It seems to me that Dr. Barnhill has brought out one of our most important therapeutic problems. The modern trend of our people is to increase very greatly in knowledge, and our patients now have a very decided way of thinking and they are all susceptible to a very confused amount of advice that is handed to them about climate. If we could standardize our way of thinking and our way of advice,

as Dr. Barnhill has outlined, it would be exceedingly valuable to have a considerable knowledge of climate and of exactly the best thing to do for the individual. It is an individual study.

For many years I have enjoyed the literature of the Climatological Society, which has brought out today the very best literature that we have for the American patient, and if you will glance over that literature you will find that there has always been in the last few years a continuously running argument as to the value of climatic treatment in the various phases of disease, particularly tuberculosis. A number of years ago it was my privilege to be on a ship with 250 young men, absolutely perfect specimens, having just passed the navy examination. There was an acute respiratory disease throughout the entire ship, so that more than 50 per cent of the men were laid low with acute tracheobronchitis and the various complicating conditions.

We sailed out of Newport News for Key West, and in three days that entire sickness was cleared up with a climatic treatment. It made me feel that our advice about the open-window method of treating our cases of acute bronchitis and acute respiratory infection was all wrong, and that the study of the individual demanded that they be given a warmer air and that they do better with warmer air, contrary to the general education of the public which is to keep your windows open at all times for all troubles, and that is generally carried out.

I believe that the same old problem of common sense and the study of the individual, and a very careful knowledge accumulated in regard to our various climates is exceedingly valuable to our specialty.

DR. BARNHILL (in closing):

The real facts concerning the value of climate in the treatment of infections of the upper air tract are easily understood. Such diseases result from the action of pathogenic bacteria on sensitive, susceptible persons. The chief and almost the only cause of such infections is close contact of a well person with an infected individual. Close contact of one with another is necessary and almost wholly unavoidable in cold, changeable climates. In a climate that approaches the ideal there is no such necessity. Everyone lives practically in the open, night and day; hence there is no such thing as crowding, no necessity for close contact, and of course no opportunity for transmitting infection from one to another. It is largely on the basis of outdoor life that I have classified climate as good or bad, for the diseases in question. On this simple plan any part of our vast domain may be analyzed, as to this quality, and just in proportion as to how much of each

day one may spend in the open pleasantly and comfortably, the value of the climate of that place may be reckoned as to its nearness of approach toward the ideal.

Answering Dr. Richard's argument to the effect that regions like Cape Cod have nowhere any superior climate, I would say that this is in a measure true, except for the very class of persons considered in my paper. Those who suffer from frequent infection of the upper air tract do so largely because they are peculiarly susceptible to the influence of cold, and especially to the sudden changes of temperature characteristic of such climates. I am fully aware that an ideal climate may be created indoors almost anywhere by making use of modern methods of heat and ventilation in the home, and that the environment is helpful so long as the person remains indoors to use it. But those not confined to bed will not stay indoors constantly. It is the sudden change from indoor warmth to outdoor cold that seriously affects those whose bodily resistance is so much lowered that they are unable to meet the change.

Those who suffer from tuberculosis usually do well in any climate, provided they are properly housed and fed, and remain in bed where sudden changes may be obviated. It is chiefly those who are ambulatory, who are out and in, who go into crowds and who must withstand the sudden changes incident to life both indoors and outdoors in a rigorous climate, that do best in a climate that permits constant life in the open.

AN ENORMOUS FRONTAL-LOBE ABSCESS FOLLOWING INFECTION OF THE FRONTAL SINUS: OPERATION; RECOVERY

ROSS HALL SKILLERN, M.D.

AND

GEORGE M. COATES, M.D.

It was just thirty years ago that the first report of a recovery of frontal-lobe abscess following operation was published, and it was looked upon then as unique and not apt to occur again in the near future. Since that time and up to the present, we have been able to collect about 177 cases of frontal-lobe abscess, most of which had undergone operation, with a mortality of approximately 75 per cent, and which would place the number of recoveries at about 44.

This high mortality was due to the fact that these abscesses were situated in the silent area of the brain and many remained unrecognized (about 45 per cent) until death occurred and an autopsy was made. As late as 1924, one of us (Skillern) made the statement that he did not recall a case where the operator opened up the frontal sinus and drained a blind abscess in the frontal lobe, that was diagnosed primarily.² This holds good almost up to today, for if we carefully review the literature we will note that almost every case had undergone one or more previous operations for frontal sinusitis when the brain abscess certainly had been present.

In making this statement we refer, of course, to those cases which were due to a previous frontal-sinus infection. Eagleton,³ in 1922, reported 150 cases of frontal-lobe abscess, 64 of which certainly took their origin from the frontal sinus. We have been able to augment these by 27, so that the total number to date counts 92, which is about one-half of the 177 reported. While the mortality following operation is still very high (about 70 per cent), it is steadily becoming lower, due to the improvement in surgical methods made by such men as Elsberg, Kerr and particu-

larly King⁴ even though the latter in our opinion has gone a trifle too far in his radicalistic tendencies. The cause of this high mortality—according to Eagleton⁵ who, in 1925, said it was over 80 per cent—was largely due to imperfect methods of treatment. It is on this very account that we are reporting our last case and discussing in some detail the after-treatments which have proven most efficacious in our hands.

This case is particularly striking from a number of standpoints, the most interesting being her serious condition immediately preceding operation and the almost instant improvement, with uneventful recovery, after the abscess had been located and drained.

Lydia W., aged twenty years, West Chester, Pa. Previous history as to her local disease negative except that at eleven years of age she was kicked in the right frontal region by a horse. There was apparently no concussion and no fracture, but a large skin-flap just above the hairline was raised. This was sutured and healed readily with little swelling. A scar is still evident when the hair is parted.

Present illness began December 8, 1929, with a cold in the head, soon followed by fairly severe right frontal headache. On December 9th, she rode horseback for several hours on a raw, cold day. On her return, headache was worse and remained so. Dr. Klevan, of West Chester, was called in and found her right frontal region tender, the right nasal mucosa swollen and pus in the right nostril. He treated this by ephedrine packs followed by neosilvol.

She was first seen by one of us (Coates) on December 11th in consultation. A roentgen-ray taken at that time showed a somewhat cloudy right frontal sinus. Examination revealed an engorged mucous membrane, right middle turbinate hypertrophied and swollen and crowded between the septum and the lateral nasal wall, much thick mucopus in the nostril and, after cleansing, pus was seen coming from beneath the anterior end of the right middle turbinate. She had a massive edema of the right upper eyelid, which could not be raised. The forehead, also, was edematous above the brow, more marked on the right, but extending across the midline to the left. Tenderness was marked. Shrinking and suction produced a considerable amount of pus, with some relief of the constant headache. Her temperature had been fairly steady between 100° and 102° F., with pulse around 100. She complained a good deal of headache, but had no other symptoms of note.

She was seen again on the 15th, at which time slight improvement was shown, the edema being less and the pain considerably improved. The bacteriological examination revealed a pure culture of *Staphylococcus albus hemolyticus*. Dr. Klevan had given her one dose of eolan and prepared a vaccine from the staphylococcus organism.

On December 18th, the condition being in no wise improved, the anterior end of the middle turbinate was removed under local anesthesia. As there was distinct evidence of pus under the upper eyelid below the middle of the brow, this was incised and a small amount of thick white pus evacuated at a depth of about $\frac{1}{2}$ inch, the organism being again the *Staphylococcus hemolyticus*. No fistula into the frontal sinus was discovered and no necrotic bone felt with a probe. Dr. Klevan reported that following these procedures, during the course of the next three weeks, the nose drained freely for a while, later ceasing. The opening through the upper eyelid drained for three days and then closed. The edema of the upper eyelid disappeared almost entirely, as also the edema over the forehead. The headaches, however, continued and there was some photophobia. The temperature was normal after about December 25th. However, she did not do well, gradually losing strength and complaining of constant and increasing frontal headache, which required morphine for its control.

On January 8th, her condition became much worse; she was markedly weaker and had a subnormal temperature, with a pulse at times below 50. On January 10th, conditions became so alarming that she was brought in an ambulance from West Chester to The Graduate Hospital in Philadelphia, a distance of thirty miles. She had to be assisted downstairs into the ambulance and is said to have fainted on the way down. One-half hour after admission she became semicomatose, being aroused with the greatest of difficulty and only after hesitation answering questions, *yes* or *no*. In another half hour she developed a complete hemiplegia on the left side, with some weakness of the left facial muscles.

Previous History. Has had measles, pertussis, mild influenza in the spring of 1929. Patient was not a strong child, but after her thirteenth birthday she put on weight and seemed healthy in every way until 1926, when she had amenorrhea for ten months. Tonsils and adenoids were removed in 1920.

Physical Examination on admission. A well-developed and a fairly well nourished, pale, white female, aged about twenty years, who was first seen lying on her right side in a stuporous condition with eyes closed and breathing about fifteen times per minute. The head was

drawn back and the muscles and neck stiffened. No thyroid enlargement and no cervical lymphadenopathy. Right pupil was dilated, with the right eye directed down. Left pupil contracted and in a central position. No exophthalmus. Extraocular movement could not be tested because of patient's lethargy. The ears were negative. The nose, throat and mouth normal. Lung expansion free and equal, but deep and slow. Tactile fremitus normal, percussion note normal and breath sounds normal. The apex beat was in the fifth interspace, midclavicular line. No enlargement, murmur or displacement. Rate about 68. Abdomen negative, no muscular twitching or convulsions. Patient was stuporous and unable to coöperate at all. Did not respond to any stimuli. Knee-jerks were hypertonic, the Achilles reflexes present. There was bilateral ankle clonus, but the Babinski was absent on both sides, as were also the confirmatory tests. The reflexes of the upper extremities were present on both sides and there was no spasticity.

On admission, temperature, 98.7° F.; pulse, 68; respiration, 20. One hour after admission the patient was seen by Dr. T. H. Weisenberg, who made the following notes: "When I first saw the patient, she responded to questions, giving the right answers, with the exception that she thought I was Dr. Coates. She was able to open her eyes and at that time her pupils were small but equally dilated. She was able to look up, down, but not to right or left. It was impossible for me to determine whether this was due to paralysis or her mental state, more probably the latter. There was some weakness in the lower left face. Her history obtained from the relatives showed that she had recently complained of headache, which was progressively worse, and nausea and vomiting for the past two days. She has also been drowsy and somewhat lethargic, but there were no convulsive phenomena or ocular symptoms with the exception of some photophobia. On returning to the room after one-half hour, she was found much more stupid, the head drawn backward and to the right: could no longer answer questions; the left pupil was rigid and small, the right pupil dilated to its maximum and not responding to light. During the dictation of these notes the right eye deviated downward, with total lack of response to any type of stimulation. When first examined, power in right hand was normal and left hand diminished. At the second examination, the left foot was turned outward, the left hand in decerebrate position. Right knee-jerk was increased; there was an abortive clonus; plantar irritation gave no response. Left knee-jerk was more prompt than the right and clonus more persistent. Abdominal reflexes absent both sides. The evidence points to right frontal-lobe abscess, with

even now a basal meningitic involvement. Fifteen minutes later there was present a bilateral Babinski. She used right arm freely, but not the left. The right pupil had come down to same size as the left and the deviation of the right eye downward had disappeared. Throughout the examination she seemed to have a protrusion of the right eye."

She was then seen by Dr. Edmund M. Spaeth, who gave the following eye report: O. D., widely dilated and fixed; O. S., pinpoint and fixed. A short time before the pupil O. D. had been pinpoint, as was now that of the left (after about ten-minute interval). Slight exophthalmos, O. D., with widening of palpebral fissure. The eye was held closed. The right eye is in hypoduction. Fundus, O. D., disk swollen and pale; margin blurred and edematous. The elevation of this nerve head is at least 2 diopters. There is no hemorrhage in the retina. O. S., nerve head seen also swollen and edematous, with blurred margins. The elevation in this eye is from 1 to 1.2 diopters. In both eyes the veins of the retina are engorged and tortuous. There is no hemorrhage on the left.

The patient was taken immediately to the operating room and the following operation performed:

Operation January 10th. The usual Killian incision, but slightly beneath the eyebrow. After the periosteum was retracted, a small hole was made into the sinus, which was apparently dry. The anterior wall of the sinus was now removed and the interior appeared to have regenerated as far as inflammatory symptoms were concerned except at the extreme upper apex. At this point, where the anterior and posterior sinus walls came together, a drop of pus was present. On probing it was found that the diploë appeared softened and somewhat purulent. A vertical incision was now made up the midforehead to gain necessary room, and after turning back the flap the external table over this area, although apparently healthy, was gradually removed in an upward direction until this osteomyelitic process of the medullary substance was completely uncovered and normal bone was encountered on all sides.

The internal table above and the posterior sinus wall below were sponged and carefully inspected in order to discover a possible focus of penetrating infection, but no discoloration or softening could be detected. Despite this negative finding, the extreme gravity of the patient's condition demanded instant action, so the internal table was opened above the upper limits of the sinus cavity and almost immediately a few drops of heavy white pus followed the introduction

of a probe between the dura and internal table. It was decided to lay bare a considerable portion of the frontal lobe, so the bone above the sinus cavity, as well as the posterior sinus wall, was resected. The dura above began to bulge and did not pulsate, while that portion directly behind the sinus cavity showed no bulging and pulsated faintly.

After a quick consultation, a scalpel was introduced into the bulging portion through the dura, directly backward, to the depth of $\frac{1}{2}$, $\frac{3}{4}$ and finally 1 inch, which was followed only by a gush of dark blood. As the patient was now moribund and further operative interference could do no harm and might possibly be of benefit, the scalpel was pushed further inward and backward, when suddenly a gush of pus under great pressure welled out of the wound alongside of the scalpel blade, dividing into two streams as it flowed down over the patient's face and hair. The scalpel was quickly removed and a pair of forceps inserted, opened and removed, thus opening widely the deep wound. Another gush of pus followed in even greater volume than the first.

With a long, thin bistoury, a portion of the brain with the dura, about the size of a small thimble, was resected so as to give free access to the abscess. The abscess cavity was now packed with gauze, following which more purulent material was evacuated. The bleeding had now become so intense from the brain and meninges and from the abscess cavity that, if allowed to continue, it would soon have exsanguinated the patient. As all of the purulent secretion appeared to have been evacuated, the cavity was snugly packed with seamed iodoform gauze, 1 inch wide, over 10 feet being required to fill the cavity and sinus. The wound was, of course, left open and the patient returned to her room in a precarious condition. After about seventy minutes consciousness seemed to be trying to return, which surely was a most welcome manifestation, as she had been brought to the operating room to all intents and purposes moribund. In the course of a few hours complete consciousness returned, but she was still a mighty sick girl. She was put under morphia and atrophin and provision was made for blood transfusion at the earliest possible moment.

Postoperative Treatment and Course. After operation the patient, in the course of an hour, began to respond to suggestions. Both pupils were equal and about the same plane, but the eye signs quite variable during the next few hours. Blood-pressure immediately after operation was 110/80, but two hours later was 90/40. A Type 4 donor was located, cross-agglutinated, and at 9.25 p.m. the patient was given 500 cc. of whole blood by the citrate method as well as 200 cc. of

normal saline. The pulse remained of good quality and rate. At this time the patient was seen to move both arms and legs and, when asked to do so, put both arms under the bedclothing. Before the transfusion the temperature was 102° F. rectal, pulse 90, respiration 20. An hour later it was the same, except the pulse had come down to 80. Patient was unable to answer questions. General blood examination immediately postoperative: erythrocytes, 3,850,000; leukocytes, 17,450; hemoglobin, 68 per cent; small lymphocytes, 7; polymorphonuclears, 93. The urine showed a faint trace of albumin, but was otherwise negative. Culture from the brain abscess eventually showed a *Staphylococcus albus*.

Laboratory Report from Dr. Eugene A. Case. "Examination of tissue removed at operation: The brain tissue shows a perivascular cellular infiltration of lymphocytes, plasma cells and mononuclears. The pia arachnoid is also infiltrated with similar cells. The sections do not show the lining of the abscess cavity."

Patient had a good night following operation and the next day, temperature, pulse and respiration remained about the same, with recession of the temperature to 99.4° F. at its lowest point. The pulse was full and regular and of good quality. Blood-pressure, 98/70. External dressings were changed and the patient's mental condition seemed clearer. She was given an enema and fluid by rectum, and morphine sulphate for restlessness. Dr. Weisenberg reported patient much better, clearer mentally but still somewhat stupid. The left hemiplegia was about the same as previously, although the weakness of the face was slightly more marked. No further evidence of progressive meningitis. Under dilated pupils, Dr. Spaeth reported the eye condition unchanged. Blood examination was about the same as previously, except that the leukocytes had gone up to 24,550. The highest elevation of temperature for the twenty-four hours was 101° F.

On January 12th, her general condition was about the same, sleeping most of the time. White blood count had fallen to 17,740; blood-pressure, 110/70. Temperature ranged from 99° to 102° F., and pulse from 108 to 120. Patient's neck was still stiff; there was no nausea.

Fundus examination by Dr. Spaeth showed the papilledema unchanged, but a definite decrease in the tortuosity and the engorgement of the retinal vessels. Patient conversed rationally with members of her family and did not complain of any discomfort or headache.

On January 13th the temperature and pulse range were about the same as the previous day. There was some restlessness; taking fluids by mouth very well. The outer dressing was removed; 18 inches of

iodoform gauze were removed from the wound. The edge of the wound was clean; no evidence of herniation. Dr. Weisenberg reported patient better mentally and physically from the neurological viewpoint. Weakness of the left side of the face was less. The Babinski was absent. Dr. Spaeth reported a definite bilateral paralysis of the external recti, slightly more marked on the left. Pupil reaction normal, but the left slightly smaller than the right. Papilledema about the same. Leukocytes, 14,350.

January 14th. Patient slightly irrational at times. Leukocytes 12,700. The wound was dressed and 18 inches of gauze removed, making a total of 36 inches. Neurological signs improved. There was some mental upset in that the patient did not recognize people by sight, calling them by wrong names. Eye condition unchanged.

January 15th. Temperature range between 100° and 101.4° F.; pulse, 92 to 110; respiration, 20. Patient resting well; general condition very good; neck still stiff; no headache. Thirty inches of gauze were removed from the wound, making a total of 66 inches. Patient still dull at times. This day all reflexes were absent with the exception of the Achilles, which could only be obtained slightly. No further signs of meningitic involvement. At this time a very fine area of hemorrhagic extravasation was noted in O. S. between superior temporal artery and vein just as they enter the nerve head. Leukocytes, 17,550.

From now on the patient showed definite improvement day by day, the temperature and pulse gradually coming down and the stiff neck clearing up. Her mental condition shortly became entirely clear, but sometimes the knee-jerks were absent. The gauze packing of the wound was removed a little at a time daily. On the 17th, the hemorrhage of the left eye had entirely disappeared and there was definite improvement in both eyes. The hemiplegia had also practically disappeared.

On the 18th, 18 inches of gauze packing was removed from the abscess cavity itself, the previous removals having been only from the frontal sinus. By the 20th, 8 feet 2 inches of packing had been removed from the sinus and abscess cavity. Dr. Spaeth reported diplopia field unremoved, diplopia constant in all fields, due to a convergent squint, paralysis of the external rectus more extensive on the left than on the right. Papilledema was beginning to recede in both eyes. Leukocyte count still showed an increase up to 16,000.

On the 22d, twelve days after operation, all of the packing had been removed from the abscess cavity, which was cleansed and immediately repacked daily. Her general condition was splendid; bright

and cheerful; no headache or complaints; noticeable improvement in eye condition. Within ten days, although there was still some diplopia present, both eyes could be rotated externally to a degree almost normal. The abscess cavity continued to drain, but in the course of two or three weeks the frontal-sinus cavity became largely obliterated by granulations; stiffness of the neck had entirely disappeared and on the 27th, pulsations in the frontal lobe of the brain were noticed.

On the 31st, papilledema had been reduced to 1 diopter in each eye. Leukocytes were 14,000 and temperature not over 99.8° F., with pulse 100 to 106.

A small sequestrum was removed from the wound on February 6th, at which time the patient was bothered by an erosion of an unerupted right third lower molar tooth, which caused some pain and temperature disturbance for a day or so, but which quickly subsided under treatment.

By February 10th, reflexes in the legs were practically normal, though slightly stronger on the right side. General condition very much improved. Ocular movements still slightly restricted in upper deviation of the left eye. Pupils equal and reacted promptly and freely to light. In both eyes there was still some slight blurring present in the upper pole; somewhat less in the left.

After February 14th, leukocyte counts were discontinued and the temperature remained practically normal. At this time the cavity of the abscess was estimated to be from $1\frac{1}{2}$ to 2 inches deep, packing being removed and replaced with fresh gauze daily.

After February 19th, the abscess was gently syringed daily with bichloride of mercury 1 to 5000, strengthened in the course of two weeks to 1 to 3000. Constant improvement was noticed, with the external wound healing nicely, with, of course, some deformity, and the eyes in all respects constantly improving. Drainage was less marked after the irrigations were started, but the daily packing of the abscess cavity with $\frac{1}{2}$ -inch iodoform gauze was continued.

She was kept in bed until March 10th, after which time she gradually spent more and more time out of bed and was permitted to leave the hospital for short walks and drives after March 25th.

By April 7th, the eyes were practically normal and the depth of the cavity in the brain reduced to about $\frac{1}{2}$ inch. Granulations at the lower part of the wound over the supraorbital ridge continued to discharge a small amount of pus and bare bone was felt with the probe. The remainder of the sinus cavity had by now been obliterated by firm, organized granulations.

On April 10th, the remaining $\frac{1}{2}$ inch of packing was forced out of the abscess cavity, which promptly closed up. It was reopened with the probe, but as no fluid was found it was allowed to close permanently.

April 11th. Two small sequestra were removed from under the granulations and the sinus in the brain was observed to be definitely healed.

April 18th. Two more very small sequestra were removed under a small area of granulation tissue down near the supraorbital ridge, about $\frac{3}{4}$ inch below the site of the abscess opening.

April 19th. Discharged from hospital. (This could have been done long before, but she resided at some distance from the city, which would have made it very inconvenient to come in daily for dressing.)

April 26th, 1930. Wound entirely healed.

The following cases represent those which have been reported as recovering following operation:

Male, aged twenty years. (HERZFELD: *Berlin. klin. Wchnschr.*, November 25, 1901, S. 1180.) History of frontal sinusitis following cold; after six weeks became decidedly worse. Some temperature; pulse about 50; pain in head growing steadier in intensity; beginning stiffness of neck.

Operation. Left frontal sinus opened; mucosa greatly swollen and discolored and bulges upward through the wound, which, on being opened, contained only a small amount of fetid pus with granulations. Posterior sinus wall found carious, so removed, and at lower part extremely fetid pus flowed out from between the dura and bone, the source of which it was not possible to determine. The underlying dura was discolored and failed to pulsate, and, on being incised, gave forth a considerable quantity of very fetid pus. On retracting the dura a fistula extending into the brain was discovered. This fistula was dilated with the forceps, after which it was possible to put one's finger into an abscess hole up to the second phalanx both laterally and superiorly. (Pus showed pneumococcus.)

The cavity was packed loosely with iodoform gauze and dressed externally with the same. The operation was immediately successful. Headache relieved entirely. Temperature became normal, but pulse remained low. Profuse purulent secretion for six days, after which time it diminished and healing quickly followed, so that the external skin wound at the end of five weeks became fully closed. One now

could note only a slight pulsation, which became less and less until it has practically disappeared. Patient entirely well, with all faculties preserved. (No details concerning the dressings are given. A remarkable fact was that the eye-grounds at no time showed any changes.)

Male. (RAWLING: *Trans. Med. Soc. of London*, 1907, xxx, 375.) Frontal headache, with edema of right upper eyelid.

May, 1905. *Operation*: Roof of orbit exposed.

June. Complete coma and paralysis left side of body and face. Second operation: Skull opened and frontal lobe exposed; abscess being found and evacuated.

July 15th. Discharged cured.

1906. (Early). Another abscess in same situation; evacuated.

1906. October 13th: Headache severe; drowsy and irritable; bulging over site of operation.

Operation, but no abscess found; marked hernia of brain, which was shaved away.

October 24th. Abscess again sought and found. Abscess walls removed *en masse*; recovery. Frontal sinus operated upon later. (*Staphylococcus pyogenes aureus*.)

(While it is stated that "Another abscess was found," one cannot help thinking it was a refilling of the same abscess, caused by too early discontinuance of drainage.)

Male, aged thirty-one years. (CARGILL, TURNER and THOMSON: *Proc. Royal Soc. of Med.* (Laryngol. Section), 1907-1908, p. 125.) Left orbital cellulitis. Incised and pus evacuated. Two weeks later, great depression, slow cerebration, irritable, temperature and pulse rate subnormal; optic neuritis marked; left frontal sinus discharging pus and bone at bottom clearly carious.

Operation. Frontal sinus opened; posterior wall softened and easily broken down, exposing cerebral surface covered with dirty, gray-looking granulations. No dura could be found. The brain pulsated, with no bulging. Exploratory needle brought pus, 3 to 4 ounces being evacuated. (*Staphylococcus aureus*.)

The after-treatment was not described. Patient is now quite well.

Male, aged twelve and a half years. (DONALIES: *Archiv. f. Ohrenh.*, 1908, lxxv, 199.) Brought into the office a very sick boy. Pain in head; swollen nose and pus; some fever; pulse, 98, and answered

questions slowly but correctly. There was a fluctuating swelling in midforehead. History of traumatism four weeks previous.

Diagnosis. Acute purulent bilateral frontal sinusitis; subperiosteal abscess; probable intracranial complication.

Operation. Abscess opened by vertical incision and both frontal sinuses opened. No improvement in general condition.

October 15th. Patient's head stiff and drawn backward; pulse, slow; both frontals discharging profusely.

October 15th. More apathetic, sleepy; condition worse.

October 21st. Spasms of left side of body and face.

Operation. Posterior wall of right frontal sinus removed. Dura somewhat hyperemic, but otherwise apparently normal. Dura incised and frontal lobe punctured; no pus found. The left frontal was now attacked, and on opening the posterior wall a few drops of thick pus were seen on the dura. The dura itself was discolored, but no break was found in continuity. It was now incised through the discolored portion and, at a depth of 2 mm., an abscess was found about $1\frac{1}{2}$ cm. in diameter. Immediate improvement followed the operation; temperature lower and pulse higher. Uneventful recovery. Wound closed November 8th.

(No mention is made of manner or method of postoperative treatment. The curious part of this case was the spasms on the diseased side.)

Male, aged twenty-eight years. (WIENER: *New York Med. Record*, October 22, 1910, p. 715). Four years ago external operation for frontal sinusitis, which was followed by a secondary operation one year later. Since then recurrent attacks of excruciating headache radiating from right frontal sinus. Fistulous opening at inner third of brow discharging pus. Patient of late forgetful and irritable. Marked tenderness over right frontal sinus. Brain abscess suspected and operation advised but refused. Two weeks later became semi-stuporous, passing from one convulsion into another; these being brought about by the collateral edema.

Operation. Right frontal sinus exposed, and found to have been completely eradicated. On posterior wall a small black spot was noted, which admitted a probe to the depth of $3\frac{1}{2}$ inches. One-half dram of pus followed its removal. Incision enlarged and scalp elevated and area laid bare about size of one's hand. A spot in the dura about the size of a twenty-five cent piece was found discolored, with

granulations. A crucial incision was made in the dura into the abscess cavity. Pus permitted to flow out and cavity sponged until clean, which appeared to be about the size of a small child's fist, containing approximately 4 ounces of pus. Cavity wiped with 90 per cent alcohol and packed (not too firmly) with $\frac{1}{2}$ -inch selvaged iodoform gauze.

On the following day the patient felt perfectly comfortable, and outside dressings changed. On the third day internal packing removed, with little secretion in cavity. The cavity was permitted to fill up with granulations and was completely closed in seven weeks. Patient has all faculties and for the past year has had no recurrence of symptoms. He is actively engaged at his former occupation as a painter and appears to be perfectly well.

(This case appears to be the first one operated upon for brain abscess with recovery that had not undergone previous operations for other conditions. All of these cases with fistula formation seem to respond particularly well to surgical interference.)

Male, aged nineteen years. (RISCHE: *Ztschr. f. Ohrenh.*, 1911, lxii, 231.) One month ago swelling noted over left eye, which quickly spread to upper lid. Incised; abscess evacuated. Next day pain worse, with vomiting. Temperature, 39° C.

Operation. Fistula into frontal sinus; anterior wall softened; granulations in frontal sinus; perforation in cerebral wall. Posterior wall resected. Dura covered with granulations and did not pulsate. Needle-puncture into the frontal lobe brought pus at depth of 2 cm. A small knife was now introduced 2 cm. and about $1\frac{1}{2}$ to 2 tablespoonsful of pus evacuated. Rubber drainage-tube in abscess cavity and iodoform gauze external in sinus.

September 8th (next day). Temperature fell to 37.6° C.

September 9th. Dressing soaked with pus. Changed, also new drain and tampon; dressing changed daily.

September 17th. Rubber drainage tube discarded and strip of gauze substituted.

September 19th. Vomited during night and complained of headache and looked pale. Pulse, 58 and small. The dressings removed and opening in the dura dilated, whereupon several cubic centimeters of pus gushed out. A new rubber tube was again inserted. Unfavorable symptoms at once disappeared.

September 27th. Rubber drain again discarded.

October 10th. Both brain and dura entirely healed. External wound healed.

October 31st. Patient discharged.

(This case shows the danger of removing the means of drainage too soon.)

Male, aged thirty years. (BERENS: *Laryngoscope*, 1916, p. 1083.) History of an old pansinusitis, with external radical operation (January 1913). Three years later, frontal headache with large swelling of cicatrix in right frontal region.

Operation. January 5, 1918. Old frontal wound reopened with pus under slight pressure. Sequestrum on posterior wall, which, on removal, yielded about 4 drams of pus (extradural). Granulations were present which were hiding a perforation through which a probe was passed in an upward and inward direction for a distance of $2\frac{1}{8}$ inches. This opening was gently enlarged with forceps and a horse-hair inserted into the stem of the abscess. (Does not state if more pus was evacuated.) Wet gauze dressing. Free drainage followed. More strands of horsehair were added daily for a week until the fistula had become as large as a leadpencil. A soft rubber drainage-tube was then substituted and the discharge became so profuse that for two weeks it was necessary to change the dressings every six hours. Discharge practically ceased at end of five weeks, but wound not allowed to heal until April 18th.

General Symptoms After Operations. Much frontal headache; restless and irritable; pupils contracted. Temperature, 100° to 101° F. (Culture: *Streptococcus hemolyticus*, pure.)

Female, married. (LYNCH: *Trans. Am. Laryngol. Assn.*, 1917, p. 144.) More than fifteen years suffered with headaches. Right maxillary sinus irrigated for a year with only varying relief. Middle turbinate removed; frontal opened intranasally; no relief. Leukocytes, 11,000. One year later (1913), external frontal; no improvement. In 1916, posterior ethmoid and sphenoid full of pus; treatments relieved headaches, but old symptoms soon returned. Posterior ethmoid cells and sphenoid opened, exposing cavernous sinus and dura; progressed nicely for five days, then symptoms of meningitis, which, however, after three days subsided. Four weeks later, symptoms reappeared, with 17,000 leukocytes. Mental dulness almost to coma; roused only with greatest difficulty.

Operation. Old frontal wound opened, which contained about 15 drops of milky pus; posterior sinus wall removed. Meninges appeared normal. Dura split; needle introduced into frontal lobe and, at depth of 1 inch, 3 drams of yellow-green pus withdrawn. After preparation of opening for a wire-gauze drain, the needle was reintroduced and 4 more drams of pus withdrawn. The brain was searched for other abscess cavity, but no evidence could be found. Finally, a small rubber-tissue drain was introduced into the brain to the depth from which the pus had been aspirated. Patient became conscious after recovery from anesthetic, and in six hours began to talk. Daily dressings revealed no pus, and on sixth day drain removed and brain explored again for pus, but without result. Wound kept open for two weeks, then allowed to heal. Recovery rapid; headaches gone; mentality clear and bright; leukocytes down to 5800. (*Staphylococcus aureus* in pure culture.)

(This case, on account of its chronicity, undoubtedly had a marked abscess wall which collapsed when the purulent secretion was withdrawn. It would seem that while it may not always be necessary to lengthily drain these cases to obtain a cure, nevertheless the danger of secondary abscess formation is so great that one takes a chance in not so doing. To be on the safe side, it is much better to allow the cavity to slowly fill up with granulation tissue even though the duration of the hospitalization is considerably lengthened.)

Male, aged twenty-two years. (LEEGAARD: *Ann. Otol., Rhinol. and Laryngol.*, 1919, p. 108.) Sinus discharge for several months. Became worse after exposure from sleeping in a tent that was damp. Trouble centered around left eye. Radical operation on left frontal sinus. Discharged from hospital after six weeks, apparently well. Two weeks afterward was again admitted to hospital, suffering great pain in left sinus, coupled with dizziness. Old wound opened; no pus in sinus; posterior wall removed; dura bulging. Needle puncture brought thick pus, at depth of $1\frac{1}{2}$ cm., 35 cc. being evacuated slowly and carefully. (Culture showed diplococci.) Split drainage tube inserted 1 cm. Next day tube lay outside of dural opening. Strip of gauze inserted to keep opening patulous. Dressings changed daily, opening in dura separated each time with forceps.

This dural opening closed one month after operation. Exposed

portion of dura began to bulge and at the end of six weeks reached size of walnut. Gradually subsided and disappeared in about two months. Slight increases in evening temperature continued for three months, then ceased. Patient's general condition excellent; operation scar firm; slight diplopia, which cleared up in about nine weeks.

(After the first day, gauze packing was apparently used and drainage tube discarded. Hernia disappeared without special treatment.)

Male, aged fifteen years. (KERR: *Archiv. of Surgery*, 1923, vii, 297.) Two years before had a submucous resection, which was followed by cerebrospinal rhinorrhea. Two months ago, rhinorrhea suddenly ceased and pneumococcus meningitis developed. Recovery followed spinal punctures.

After a period of time (not stated), headache with vomiting appeared with choked disk. Leukocytosis, 16,000. Neurologic examination negative. Symptoms pointed to infection in right frontal-sinus region and diagnosis of frontal-lobe abscess was made.

Operation. Craniotomy was performed and several ounces of greenish pus (pneumococcus, Type IV) were evacuated from an abscess about 3 cm. deep in right frontal lobe. Drainage was maintained for six months, followed by complete cure. (No details were given concerning technique of operation or after treatment.)

October 27th. Male, aged twenty-three years. (SWINDELLS and RANKIN: *British Med. Jour.*, 1924, ii, 622.) Edema of left upper and lower eyelids; tenderness over left maxilla; temperature, 103 °F.; pulse, 50.

November 7th. Headache increased.

November 9th. *Operation.* Left antrum drained through canine fossa; left frontal sinus opened, which contained glairy, inspissated pus. Patient greatly improved.

November 19th. Headache again severe. Patient became violent and abusive toward the nurses. Mind clouded; condition weak.

November 27th. Handgrips weak; right weaker; semicomatose; pulse, 48.

November 28th. *Third Operation.* Skull trephined above and to left of frontal sinus. Dura bulged into opening with no pulsation; marked granulating area. Dura incised and pus found at depth of $\frac{3}{4}$ inch (micrococci and staphylococci), 5 ounces collected. A gauze

drain, $2\frac{1}{2}$ inches. Next day patient entirely changed; mind clear and calm. Twelve days after, two epileptiform seizures and other similar attacks.

January 6th. All packing removed.

January 20th. Wound allowed to close.

February 6th. Discharged cured.

(Five ounces of pus is perhaps the largest frontal-lobe abscess reported.)

Female, aged ten years. (WISHART: *Jour. Laryngol.*, 1924, vol. 39 p. 638.) Operated upon for chronic frontal sinusitis. Abscess found while probing through defect in posterior wall. One-half ounce of pus evacuated. (*Staphylococcus albus*.) Drained with rubber tube. Uninterrupted recovery.

Male, aged thirty-nine years. (JESSEMAN: *Boston Med. and Surg. Jour.*, 1925, xcii, 739.) Right frontal sinusitis.

Operation. February 23d. Intranasal, anterior end middle turbinate and anterior ethmoid cells opened.

February 25th. Convulsion which lasted ten minutes. Semi-conscious. Diagnosis, brain abscess.

Operation. February 25th. Frontal sinus opened and filled with thick, dark-colored pus. Entire membrane pyogenic. Entire front wall removed. No defect found in posterior wall. Large opening made into this wall. Dura darkish and appeared under tension, without pulsating. Incision through dura brought no pus, but at depth of $\frac{1}{4}$ inch an abscess was opened which contained a large amount of dark-colored pus. Two rubber tubes used for drainage. Wound allowed to remain open.

Following days, pain improved, drowsiness less marked and mind clear. Profuse discharge. Temperature, 101° F. by axilla. Rubber drains not changed for one week, then only one returned.

March 20th. Drains omitted.

April 15th. Plastic closure of wound, with healing by first intention.

April 23d. Dismissed from hospital.

(It is most unusual to find an abscess at such a superficial depth, the drainage difficulties in such a case would be markedly lessened.)

Male, aged fourteen years. (ARNOLDSON and BOSTROM: *Acta oto-laryngologica*, 1925, viii, 346.) May 15th, admitted to hospital for acute frontal sinusitis.

Operation, May 24th. Intranasal. Removal of middle turbinate and passage into frontal sinus enlarged. No pus.

May 29th. Frontal sinus opened externally; contained pus and granulation tissue. Posterior wall removed; dura discolored and tense. Puncture brought fetid pus (sterile). Opening in dura enlarged and an abscess uncovered the size of an egg. Drain introduced. Tampon of iodoform gauze. Cure. Discharged June 22d.

(Authors do not state manner of dressings or postoperative progress of patient. It will be noted that two weeks in the hospital elapsed before the presence of the brain abscess was recognized and operation performed.)

CASE I (Boss: *Ztschr. f. Laryngol. u. Rhinol.*, 1926, xiii, 128.)

(Tendency to secondary abscess formation is well shown in Boss's Case I, which required five incisions.)

Original operation (puncture of frontal lobe to depth of 3 cm.), November 4th. The second followed in two and a half months, January 19th. The third in a little over three months, April 22d. The fourth in nine days, May 8th. The fifth about six months, November 5th. Patient died in two days. Autopsy showed another abscess in frontal lobe which had not been opened.

(To our minds, all this was due to the fact that drainage had not been kept up, and after each operation the patient was permitted to leave the hospital too soon. His second case went on to a more favorable conclusion.)

CASE II. Male, five days after an automobile accident complained of pain in right frontal sinus with edema of right eyelid.

August 14th. Local treatment for acute frontal sinusitis.

August 18th. Headache worse. Middle turbinate removed. Improvement in headache and edema.

August 28th. Symptoms return, even more marked.

August 30th. Killian radical on frontal sinus. Sinus filled with gray-green pus.

September 4th. Condition much better—wound closed.

September 6th. Headache reappeared and temperature 38.5° C.

September 11th. Wound reopened; considerable pus appeared at inner angle; headache becoming unbearable; temperature, 38.8° C. (During following nine days patient's condition gradually worse.)

September 20th. Continued vomiting; headache and beginning somnolence. Neurological examination, beginning right facial paralysis.

Operation. Removal of apparently intact posterior sinus wall; dura showed bluish-red discoloration about the size of a quarter; pulsation present; puncture, 1 cm. deep, brought 1 ounce gray-green pus.

September 24th. Dilatation of passage to abscess cavity; tamponade.

September 25th. Dressing changed daily; irrigation with Rivanol 1 to 5000; uninterrupted healing.

(This is a typical case of frontal sinus infection, with frontal-lobe abscess formation, where the latter remained unrecognized until it began to affect certain vital centers. Continued headache after a radical operation, other things being eliminated, is pathognomonic for meningeal involvement; the longer it continues without stiffness of the neck, the more indicative of some form of brain abscess.)

Female, aged twenty-eight years. (CALLFAS: *Trans. Am. Laryngol., Rhinol. and Otol. Soc.*, 1926, p. 436, Case II.) History of cold after swimming. Much pain and swelling in and around left orbit. Patient semiconscious.

Operation, August 8th. Incision below supraorbital arch, and rubber drainage tube inserted. Next day both ethmoids exenterated, followed by some improvement. Blood positive for staphylococcus. Next three months variable, with abscesses in breast and thigh.

November 24th. Patient much worse. Gave very slow response to questions. Neurological findings suggest a lesion in left hemisphere.

Operation, November 25th. Left frontal sinus opened; posterior wall badly diseased; needle puncture brought thick yellow pus at depth of 3 cm. (*Staphylococcus aureus*.) Next day patient showed improvement, which continued until she left the hospital.

(Reporter does not mention kind of dressings or treatment except intravenous injections of 1 per cent solution of mercurochrome, which seemed to do more harm than good. Perforated rubber tubing may have been used for drainage. Callfas uses a rubber tube, splitting the protruding end into four segments, which are folded back and stitched to skin; the tube is gradually shortened as

indicated. This case is typical of the manner in which some of these frontal-lobe abscesses gradually progress.)

Male, aged twenty-two years. Syphilitic. (MATHIEU and PERON: *Paris médical*, October 2, 1926, p. 270.)

Operation. Cure.

Male, aged twenty-eight years. (PORTMAN: *Bull. et mém. Soc. méd. et chir. de Bordeaux*, 1926, p. 365.)

Operation. Cure.

(WINTER and KALT: *Rev. d'oto-neuro-ocul.*, 1926, p. 702.)

Operation. Cure.

Female, aged twenty-three years. (ALVIN: *Lyon médical*, 1927, cxl, 127.) During a voyage to South Africa contracted a heavy cold. Disembarked at Tunis and was operated upon for a collection of pus above left eye. Arrived at Marseilles April 19th. Shortly afterward vomiting set in and loss of memory. Pulse below 60. Reflexes diminished.

Operation, May 1st. Frontal sinus opened. Two fistulas found on floor, one surrounded by granulations, resection of the inferior wall of the sinus, with drainage into the nose. (Author gives no further data regarding operation or dressing.) One hour after operation pulse returned to 80. Lumbar puncture showed no sign of meningitis. At the end of thirty-six hours, pulse fell again to 60 and the torpor, which had diminished, reappeared. Temperature, 38° C.

Second Operation, May 9th. Resecting of part of the anterior sinus wall a fistula was found at the top of the internal wall which had been overlooked at the first intervention. Resection of the internal wall showed meninges and dura apparently normal. (No further operating was done.) During the afternoon the patient's condition became much worse. Pulse below 55, with incontinence; semicomatose.

Third Operation, May 10th. No anesthesia required. Dura incised; frontal lobe punctured, using a large trocar, with immediate evacuation of an abscess containing about a large glassful of serous pus (*streptococcus*). Immediately after the operation she seemed to return to life. While she had not spoken for twenty-four hours, she responded to questions and her physical state seemed completely changed. Pulse revived to 80. (No description of technique or

drainage is given.) During the following days the suppuration was profuse. Four days after operation erysipelas appeared on face. Antistreptococcic serum administered. The temperature fell rapidly and at the same time the suppuration diminished.

May 31st. Drain removed.

June 26th. Central hernia the size of the palm of one's hand.

October 30th. Discharged.

(It is unfortunate that the author does not go into details regarding the technique of finishing the operation, as well as the dressings and subsequent treatment. Such information is of inestimable value to those having similar cases under their care.)

Male, aged nineteen years. (MACKENZIE BROWN: *Ann. Otol., Rhinol. and Laryngol.*, 1927, p. 710.) After swimming, developed pain and tenderness over left frontal sinus. Temperature, 103° F. Four days later, swelling appeared. Opened. (Streptococci.) Well for four days. Severe headache, ptosis of left eyelid. Patient vomited several times during the day. Became unconscious.

Sinus opened through old incision. Posterior wall removed, followed by about 1 dram of pus (extradural). Dura thickened and covered with granulations; no perforation found. Drain placed in sinus and wound closed. Improvement followed, but on the sixth day after the operation he complained of headache and soon became unconscious. Old wound reopened; dura opened and $\frac{1}{2}$ ounce of pus was found $\frac{1}{2}$ inch from the surface (streptococci). Drain inserted into abscess cavity (kind not stated). Consciousness returned next day and no headache. Complete recovery followed. (Author gives no detail regarding dressings nor length of stay in hospital.)

(IMPERATORI: *Trans. Am. Laryngol. Assn.*, 1927, p. 34.) Reported 5 cases of frontal lobe abscess following frontal sinusitis. All of these underwent operation with 2 complete cures.

CASE I. Male, aged twenty-seven years. Headache in frontal region for seven weeks. Swelling over left frontal incised; considerable pus evacuated. Headache persisted.

Radical Operation. Inner table of both frontal sinuses necrosed with epidural abscess. Sinus found, which led into right frontal lobe. Abscess evacuated containing about 1 ounce of pus. No culture reported. Drain placed in frontal lobe and wound left wide open. Headache disappeared within a week. (The writer does not go into detail regarding the postoperative treatment.)

CASE II. Female, aged fourteen years. Headache for three weeks with increasing severity and periods of violent delirium. Temperature, 103° to 105° F. Fluctuating mass over frontal region.

Radical Operation. Both frontals contained pus; inner wall apparently not involved; no improvement. Four days later wound again opened. Portion of posterior wall removed; dura congested, no pulsation. Search into left frontal lobe proved negative. Small epidural abscess above frontal sinus on right which covered a sinus leading into the frontal lobe. Evacuation of 2 ounces pus. No cultures reported. Pulsation immediately returned. (No detail of postoperative dressings. Patient left hospital in ten days. Author states he is still living.)

January 25th, 1927. Male, aged twenty-one years. (SANDES and HALLORAN: *Med. Jour. of Australia*, August 27, 1927, p. 298.) Headache and vomiting for three months following a head cold; cerebation slow; double papilledema.

January 26th, 1927. Patient became comatose; pulse, 130.

Operation. Trephine in right frontal bone midway between eyebrow and hair of scalp. Exploratory needle brought pus. Needle track dilated and glove-drain inserted. Eight ounces of pus were slowly evacuated. (*Staphylococcus aureus*.)

February 1st. Clear mentally and condition greatly improved. Much fluid was escaping and portion of abscess wall extruding.

March 30th. Small piece of granulating cerebral tissue removed from center of wound, revealing a cavity, the probe sinking in 2 inches, with drain inserted.

April 21st. Patient discharged, with no evidence of hernia and healed scar. He was practically blind in right eye from secondary optic atrophy.

(The enormous amount of pus evacuated makes this case noteworthy. We have been unable to find another case of frontal-lobe abscess where more than 5 ounces were found, and this amount in only one instance.)

Male, aged ten years. (MOTLEY: *Southern Med. and Surg. Jour.*, 1927, p. 243.) Acute exacerbation of chronic purulent left pansinusitis, with orbital cellulitis.

Operation, September 9th. Radical frontal, ethmoid and sphenoid (external) on right side; dura not exposed.

September 30th. Discharged; condition good.

October 18th. Slight swelling and purulent discharge through scar; roentgen-ray showed area of osteomyelitis.

Second Operation. Sequestrum about 2 cm. in diameter removed, which involved only outer table.

October 30th. Nausea and vomiting. Disks show $2\frac{1}{2}$ diopters choking.

Third Operation. Dura exposed 2 by $1\frac{1}{2}$ inches and painted with iodine. After waiting forty-eight hours for walling off, needle introduced and pus found at depth of 1 cm. Crucial incision through dura and Mosher drain placed inside of abscess; 1 ounce thick creamy pus evacuated. (Staphylococcus.) Iodoform-gauze packing. Dressings changed twice daily. Prompt relief to both nausea and vomiting. Wire basket removed from abscess two weeks after operation.

December 15th. Fourteen weeks later. Patient up and about. Small brain hernia, 3 cm. in diameter, which receded rapidly after spinal punctures and magnesium sulphate dehydration. Discharged. Remained well for past six months, when last seen.

CASE I. Male, aged forty years. (SPROWL: *Ann. Otol. Rhinol. and Laryngol.*, 1928, p. 922.) Four months previously had developed a head cold with pain and tenderness over right eye, which has persisted. Two months later vomiting began. The headaches increased in severity, with impairment of vision and vertigo. Frontal sinus tender; thick, greenish, purulent discharge from right nose; fundus revealed a bilateral papilledema. Temperature, 101.4° F.; pulse, 56; white cell count, 16,000.

Operation, April 8th. Frontal sinus full of thick greenish pus under pressure. A large portion of the posterior wall was necrotic and lying free in the sinus. Through this perforation, which was about the size of a dime, the dura was seen covered with perforations which surrounded a fistula from which pus was exuding. The fistula was enlarged and 3 ounces of pus evacuated from the frontal lobe. (Streptococcus hemolyticus.) The needle entered to a depth of 3 inches without reaching bottom. A rubber tube was introduced for drainage and sutured to the skin flap; iodoform packing around the tube. Wound dressed daily. Convalescence uneventful. The tube was gradually shortened and entirely removed on May 26th (forty-eight days). Wound entirely healed on June 4th. Patient last seen over two years later, with no recurrence of symptoms.

CASE II. Male, aged fourteen years. On August 8th admitted to hospital with marked swelling and tenderness of right upper eyelid

and frontal sinus; edema of bulbar conjunctiva; pupils equal; fundi negative. Temperature, 104.2° F.; pulse, 124; leukocytes, 18,700.

Operation, August 10th. Brow incision, with evacuation of large orbital abscess. Cigarette drain and wound left open. Patient improved daily and, although still a slight discharge from wound, was discharged on September 3d.

On October 13th wound still discharging. Skiagraph shows unusual shadow over right orbit, presumably due to bone destruction.

October 20th. Headache again appeared; tenderness over frontal bone. Temperature, 98° F.; pulse, 84. Neurologic examination negative. Right disk, beginning optic neuritis. Leukocytes, 16,400.

Operation, October 21st. In outer angle of frontal sinus both plates necrotic. Small perforation in posterior plate, with granulations on underlying dura. Osteomyelitis in outer plate of sinus, extending above. On enlarging perforation the dura bulged upward into the wound. Incision $\frac{1}{2}$ inch long in dura. Three puncture wounds in different directions were made into the frontal lobe, but no pus appeared. The dural incision was packed with iodoform gauze and the external wound left open; dressings changed daily being soiled with cerebrospinal fluid. On third day a small hernia appeared through opening in dura, which increased in size. During the next fourteen days patient gradually became worse, and on the fifteenth severe headache and nausea appeared and the papilledema increased to $1\frac{1}{2}$ mm. Babinski positive.

Operation, November 6th. Old incision enlarged and considerable softened bone removed. Dural wound enlarged and a brain searcher introduced just above the hernia and at the depth of 1 inch an abscess found. The cavity was unroofed, as described by King, and $\frac{1}{2}$ ounce of pus evacuated. The wound was dressed daily by covering the hernia with rubber tissue coated with vaselin and gauze over this. (No mention is made of the abscess cavity itself.) On the third day the hernia became quite large, but slowly subsided after spinal punctures.

January 5th. The wound was entirely healed and the patient left the hospital. He remained free from symptoms until January 25th, when he returned complaining of severe pain around the eyes and appeared ill. The wound was bulging and the brain seemed tense. Leukocytes, 20,400. Kernig positive. The following day was distinctly worse.

January 27th. Old incision again opened. Brain searcher directed backward. Located a large superficial abscess containing about $1\frac{1}{2}$

ounces of thick green pus (pneumococcus.) This abscess was also unroofed and the brain permitted to herniate.

Convalescence was prolonged but uneventful. The large hernia was gradually reduced with spinal taps.

March 19th. Wound completely healed and patient discharged from hospital one week later.

(This case is particularly illuminating, for it shows in the first place the possibility of overlooking a frontal-lobe abscess; second, the tendency to the formation of secondary abscess even under skilful management, and third, the gradual disappearance of the hernia following spinal tapping. No less remarkable is the resistance shown by the younger patient after the purulent collection has been thoroughly evacuated.)

Male, aged six years. (LEARMONTH: *Surg. Clinics of North America*, August, 1929, p. 979.) Acute right frontal sinusitis, with fistula over right supraorbital area.

Operation, October 17th, 1927. Wound broke down and drained until January. Examination of nose and sinuses negative; roentgen-rays negative.

Diagnosis. Osteomyelitis of low virulence.

Treatment. Ultra-violet ray. Sinus healed after one year (February, 1928).

July. Swelling reappeared over frontal sinus, with headaches. Incision healed and relieved all symptoms.

September 24th. Sudden attacks of headache with projectile vomiting. Headache only relieved by lumbar puncture. Examination showed choked disks, 3 to 4 diopters. Vision greatly impaired, 6/30. Leukocytes, 16,300.

Diagnosis. Abscess of frontal lobe.

Operation, October 29th. Both frontal sinuses chronically inflamed; posterior bony wall appeared perfectly normal. This wall removed; dura apparently healthy. A blunt cannula was passed into the frontal lobe and encountered resistance at depth of 4.5 cm. A needle was introduced, and 15 cc. thick, grayish pus withdrawn by a syringe (*Staphylococcus*). A tract was then made into the abscess and two drainage tubes placed in position and sutured. Iodoform gauze. Patient not much improved.

Operation, November 8th. Passed needles in different directions, and on the lateral aspect of the frontal lobe encountered a second

abscess, from which 20 cc. pus was evacuated. Opening made into this abscess through the temple. The wall between the two abscesses was broken down and a rubber tube passed from the frontal opening to the temporal opening.

Postoperative course, uneventful.

November 17th. Patient left hospital.

December 18th. Tube removed and wounds kept open with gauze soaked in 1 to 1000 acriflavine.

January 4th. Patient dismissed, entirely cured.

(This case shows the importance of searching for other collections of pus when the symptoms do not show improvement. Many fatal cases reported, in which this was not done, give a similar history to this one before the secondary operation.)

Male, aged twenty-years. (MAYER: *Wiener Med. Wchnschr.*, 1929, lxxix, 253.) Operation; recovery.

Male, aged nineteen years. (J. E. J. KING: Personal communication, 1930.) Six weeks ago had a severe head cold, followed by left frontal sinusitis, which was operated upon. Since has had severe headaches. Vomits several times daily, which is projectile and not preceded by nausea. No fever; pulse, 58. Vision good, although fundus shows slight choking of disk, with punctate hemorrhages. Appears to be mentally sluggish.

Diagnosis. Brain abscess, right frontal lobe.

Operation, November 9th. Trephine opening over right frontal lobe. By means of an exploratory puncture with a brain cannula into the right frontal lobe the typical resistance of a brain abscess was encountered at a depth of 3 cm. After the abscess had been located, a circular opening in the bone, about $1\frac{1}{2}$ inches in diameter, was made directly over the extreme end of the frontal lobe and the dura incised in a stellate fashion. The frontal lobe was then incised, and after removing a portion of the extreme tip, a good exposure of the yet unopened abscess cavity was obtained. Before opening the abscess, small iodoform gauze strips were introduced beneath the dura to prevent spreading of infection to meninges. An incision was now made into the abscess cavity and thick, yellow, odorless pus removed by sponging and washing-out with Dakin's solution. A section of the abscess wall which had a firm capsule was excised, but not as much as was desired, in order to allow complete herniation. Gauze dressing

wet with Dakin's solution and rubber dam over abscess. Hourly instillation of Dakin's solution.

Patient's condition showed immediate improvement. Pulse up to 90-96.

November 11th. First dressing. Operative area clean. Dressing removed except around site of defect and hernia. Patient alert; talks coherently; no paralysis. Temperature, 99.6° F.; pulse, 78.

November 12th. Second dressing. Hernia projects 1 inch above skull. Dakinization every hour continued. Patient much improved. Soft diet.

November 14th. All original gauze removed and partially replaced. Dakinization.

November 22d. Abscess cavity completely obliterated.

January 12th. Hernia found completely collapsed.

February 2d. Sinus closed; patient discharged, completely cured.

(The author goes into minute detail regarding the daily appearance of the wound, as well as the dressings and the exact medication. Particular attention is paid to the hernia and the manner and method of treating same.)

In reviewing the treatment of brain abscess, at least as far as the frontal lobe is concerned, it seems to us that great stress is laid upon certain procedures which appear to be quite unnecessary, while other measures of the utmost importance are only given passing mention or for the most part entirely ignored. It is our purpose to present our experiences concerning the former, and to enter into some detail regarding the latter, to the extent that their importance would seem to indicate.

In order to cure a brain abscess, we must first recognize its presence. Eagleton stated, in 1922, that at least 45 per cent were diagnosed only on the autopsy table. Since that time some improvement has occurred, but it is only within the past two years that expert neurological examinations have played a great rôle in arriving at definite preoperative knowledge, so that one of the prime requisites for an ultimate cure lies in the early recognition of the pathological condition. After the diagnosis of a frontal-lobe abscess is definitely established, what procedure shall be adopted? If we study in detail those cases reported in which a

fistula has formed, we are at once impressed by the fact that most of these recovered.

Now in considering the operative interference, why not attempt to emulate Nature as much as possible by making a fistula that is of sufficient size for adequate drainage, and by packing to keep it patulous until the abscess cavity is closed by the slowly forming granulation tissue? This, briefly, is the method followed in our case just reported. This will necessitate approaching the meninges through the diseased frontal sinus; in other words, through a septic area. We have found that a clean field is apparently, in such cases, unnecessary; therefore, packing off the intradural space around the operative field is probably a waste of time. It was previously thought that the high mortality was due to exposing the meninges to purulent secretion, with resulting meningitis. This has not been proven to be the fact, for if the patient survives the operation, the cause of death is usually a secondary abscess, at least as far as the frontal lobe is concerned. Suppose the approach has been made through the frontal sinus, the posterior wall of which has been removed and the dura exposed: what is the next step? To find, of course, the abscess. We believe the frontal lobe should be penetrated with a bistoury until the purulent secretion is located. Penetration can be made to a depth of $2\frac{1}{2}$ cm. without danger to the lateral ventricles, and if the knife blade is not higher than $1\frac{1}{2}$ cm. from the floor the penetration can go as far as 4 to 5 cm. When pus appears, a grooved director may be introduced, then a hole made directly into the abscess cavity the size of a leadpencil, by cutting away that much dura and brain tissue. This opening should be large enough for adequate drainage, but too small for brain herniation, which is one of the banes of post-operative treatment. The abscess cavity can now be completely evacuated by sponging with gauze strips, although severe hemorrhage will probably be encountered, which however, is controlled by the next step. The cavity is washed out with 1 to 3000 bichloride solution, then packed snugly, but not too tightly, with 1-inch seamed idoform gauze. The main purpose of this is to not only control the hemorrhage, but to prevent collapse and adhesion of the abscess cavity walls which would cause the retention of pus

in the recesses and ultimately lead to the formation of a secondary abscess. There is, of course, the question as between the use of gauze packing and rubber drainage-tubes, but we believe if a straight drainage tract into the abscess cavity is procured that the gauze will suffice, as we can then control the caliber of the tract by the pressure of the gauze.

The first intracranial dressing should be left ten days or two weeks, and the gauze then removed piecemeal, but the outside layers of gauze can be changed once or even twice daily, depending upon the secretion, which usually is very profuse. This, to our mind, is the crux of the entire situation—that is, the drainage—which should be ample, continuous and particularly lengthy, for most of the failures have resulted from too early discontinuation of this essential part of the treatment.

The iodoform gauze is slowly removed on succeeding days, taking only that which has become loose and does not adhere to the brain substance. As the tract becomes shallowed the iodoform gauze is entirely removed at each dressing and replaced with fresh. In the meantime the patient should be confined to bed for at least five to six weeks. Finally the tract will close, and then will come the plastic work of reconstruction.

CONCLUSIONS. 1. All cases of frontal sinusitis about to undergo an external operation, that show any symptom pointing to the possibility of intracranial involvement (unusual headache, fundus changes and, particularly, some disturbances in the reflexes), should have a thorough neurological examination.

2. The approach to the frontal-lobe abscess should be through and above the diseased frontal sinus.

3. The abscess, when found, should have straight drainage about the size of a leadpencil. This will not only suffice, but prevent a hernia forming. (If no capsule has formed, on account of being in the acute stage, a larger opening may be necessary.)

4. The drainage should be ample, continuous and lengthy until granulations fill the cavity.

It is our belief that close application to these principles will greatly reduce the mortality of frontal-lobe abscess, at least in those following infection of the frontal sinus.

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3. Eagleton: Brain Abscess, Macmillan, 1922, p. 251.
4. King: Treatment of Brain Abscess, *Surg., Gynec. and Obst.*, November, 1924, p. 554.
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DISCUSSION

DR. CORNELIUS G. COAKLEY, New York City:

Mr. President, I think the readers of the paper ought to be congratulated upon the apparently wonderful results they got in this case. I say "apparently," because the experience that many of us have had with very large abscesses is that they will apparently get well and a few months later develop abscesses from just what the Doctor has tried and we hope did succeed in doing, namely, to have that abscess thoroughly obliterated. I should be glad to get the report of this case in one or two years from now. I hope that he succeeds. It has been a wonderful result so far as it has gone.

DR. HENRY B. ORTON, Newark, N. J.:

I want to congratulate the authors on their wonderful result. There is one point—that the patient was kicked in the head. In Dr. Eagleton's service in the city hospital we have felt that all cases of fracture of the frontal sinus that has involved both plates will eventually come to either a meningitis or a frontal lobe abscess if not operated on.

DR. JOHN E. MACKENTY, New York City:

I have nothing to say except to repeat what Dr. Coakley has said, that one never knows how long it will take to cure a frontal-lobe abscess. I had one patient that lived six months following the evacuation of a large frontal-lobe abscess, only to die at the end from the same disease.

I think the authors are to be congratulated. I think their method of treating the condition is excellent.

DR. SKILLERN (in closing):

That which Dr. Coakley and Dr. Mackenty said is just exactly what I wanted to bring out. Both of these gentlemen have had experience along this line. Precisely what I was afraid of in this case, is that we were going to have a secondary abscess. I quite agree with them that she is not out of the woods yet, but our idea in treating it the way we did was to try to very gradually close it up. You see it was three months that we kept her in the hospital. In that way we hoped that we might overcome the possibility of a secondary abscess. I think nearly all of these cases that die are from secondary abscess and not encephalitis, as a great many believe.

I have also in this article looked over the different cases from 1900 to date of those that have recovered and have jotted down the synopsis of all those cases and after each one made some criticism regarding what that particular case brought up. I hope it is going to be of benefit to any of use who are unfortunate enough to have another one of these cases for then we can note how the different men handle these cases and perhaps strike the very one that we happen to have at that time.

AN UNEXTRACTED FOREIGN BODY IN THE ESOPHAGUS PENETRATING THE THORACIC AORTA OF A TEN-MONTHS' INFANT

HENRY L. SWAIN, M.D.

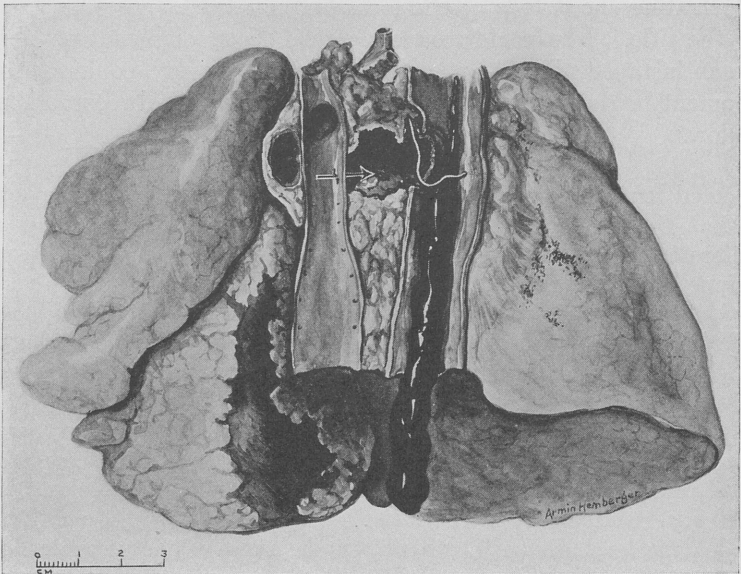
ALLOW me to present most briefly the history of and the pictures illustrating the details of the local findings in the case described in the title. I have always wondered if there might not possibly be an instance where "to remove or not to remove" could for the moment be debated. The history from the hospital records is as follows:

Aged ten months. Entered January 23, 1930. Died January 24, 1930. Onset of the present illness began rather suddenly ten days before admission with vomiting and a slight fever daily. Intestinal gripe being prevalent, that was the diagnosis.

About 3 A.M. of January 23, patient vomited about 50 cc. of blood and became very pale. The competent physician who was in charge advised hospital examination. Entered early forenoon. Temperature 104° F. Child well developed and overnourished. Sits up in bed off and on. Heart, lungs and abdomen were normal. At noon, temperature was 98.6° F. The stools contained a large amount of comparatively fresh blood. 150 cc. of citrated whole blood transfusion. At 6.00 P.M. roentgen-ray of chest disclosed a foreign body, 3½ cm. long, about the level of the fourth rib, to the right of the sternum. It appeared like a bent, irregularly shaped, fine hairpin. About 9.00 P.M. 30 cc. of fresh blood was vomited, $\frac{1}{60}$ grain of morphine was given for restlessness. Normal saline 450 cc. hypodermoclysis. (At this time I was consulted over the phone.)

At 11.45 P.M., an esophagoscopy was done by Dr. Charles Flynn. The lumen was almost full of bloodclots at the level of the fourth rib. Only a small portion of the anterior esophageal wall was visible, as blood oozed steadily after the clots were removed. Adrenalin on sponges did not check the oozing. No portion of foreign body was visible. In his opinion and that of the roentgen-ray and other consultants the foreign body may have escaped into the mediastinum. No bleeding from the bronchi. No foreign body was seen.

January 24, at 10.00 A.M. the child suddenly became very pale, pulseless and respirations ceased. A partial postmortem was granted. There was a moderate inflammation of the mediastinum, posterior to the esophagus at the level of fourth rib. When the esophagus was opened an inflammatory area about 3 cm. in diameter bulged into the lumen. A nickle-plated fine wire was lying horizontally in the lumen, one end was through the middle of the bulge into thoracic aorta, and the other end had penetrated the esophageal wall. The stomach and intestines were well filled with freshly clotted blood.



I present the roentgen-ray picture, a slide of the colored drawing now being passed around and a piece of wire showing the various bends, no two of which were in the same plane. This is as nearly as I could make it to be like the specimen, which for many reasons is allowed to remain intact.

It would seem quite evident that the wire had first been in the aorta itself, had worked loose or back toward the esophagus, and as the wire was both in and out of the opening a splendid opportunity was afforded for the formation of a false aneurysm. The

distention of this practically pulled the wire out of the aorta and bulged into the lumen of the esophagus. After a time this aneurysm began to leak into the esophagus and then the first marked hemorrhage.

To have been guided by the roentgen-ray alone, one could hardly have been given the correct picture. Even the genius of a Jackson or a Mosher would have been taxed to remove so crooked a wire.

As a consultant I advised, as the history relates, to wait for a couple of hours until Dr. Flynn, who is attending in bronchoscopy, could get to the hospital and look things over. For reasons plainly evident in the picture he was unable to locate the wire at all—too much blood, both clotted and free, to have even the merest glance into the esophagus. Then the displacement of the esophagus to the left, by what was in the living child a much bigger false aneurysm, made details still more difficult of orientation. If one had happened to have worked under fluoroscope, one would certainly have plunged into the thin-coated aneurysm and death on the table would have resulted, as eventually transpired in bed.

It seems to me that the result was inevitable, unless some chance had directed the attention of the parents to the possibility of a foreign body much earlier than was finally done by the profuse hemorrhage.

DISCUSSION

DR. HENRY B. ORTON, Newark, N. J.:

I would like to ask whether we should esophagoscope in the face of mediastinitis, or whether we should keep our hands off until such time as Nature has tried to wall off and then go into the chest? I am reporting 6 cases at the Bronchoscopic Society, and only 1 is alive at the present time, and that one we kept our hands off.

DYSPHAGIA OF SEPTIC ORIGIN RESEMBLING FOREIGN BODY IN THE ESOPHAGUS

WILLIAM B. CHAMBERLIN, M.D.

POSSIBLY the title of the following report is incorrect, or at least misleading. I have called it a dysphagia of toxic origin because there was present a localized or focal infection and no other cause could be found. Whether the designation as idiopathic or even hysterical dysphagia would have been more illuminating, I leave to my auditors to decide.

Bobbie H., aged five and a half years, was referred to me on September 18, 1929, by Dr. McKenzie of Warren, Ohio, with the following history. On awakening four days previously he manifested some hoarseness and complained of inability to swallow. There was a croupy cough and he complained that he was choking. Following this there was some daily fever, with a maximum of 102° F., until twenty-four hours previous, when the temperature had suddenly risen to 106° F., following a peritoneal infusion. Although there was no history of a foreign body having been ingested, a roentgen-ray was taken which was entirely negative. A bismuth meal was somewhat unsatisfactory and apparently progressed as far as the sixth cervical vertebra and was then promptly regurgitated. A stomach tube passed without obstruction through the esophagus. Throughout his life Bobbie had had a capricious appetite and was given to "tantrums" when his wishes were opposed. Such was his history on admission to Lakeside Hospital.

Physical examination showed some redness of the right tympanic membrane, while Dr. Blankenhorn, who was called in consultation, was able to diagnose a definite pneumonia at the hilus of the right lung. The child was most coöperative and made apparently every effort to swallow. On fluoroscopic examination the bismuth meal would be churned about a few minutes in the mouth and then spat out. It was noticed, however, that a thin stream of bismuth passed downward into the stomach. The tube encountered no resistance during gavage.

At the suggestion of Dr. Blankenhorn a laryngoscopy and esophagoscopy were performed the day following his admission. The former showed a normal larynx, while the esophagoscope passed freely into

the stomach, disclosing no abnormalities *en route*. Four days later the patient was seen in consultation by Dr. H. J. Gerstenberger. The condition was unchanged except that the right tympanic membrane showed more congestion. Two days later a paracentesis was performed in my absence by my assistant, Dr. I. F. Weidlein. A serous discharge followed, which soon became purulent. Ten days later the right mastoid was exenterated. There had been pain, fever and tenderness, together with a prolapse of the canal wall. The mastoid process was large and pneumatic, containing pus and granulation tissue. The sinus and dura were not exposed. Meanwhile the lung had entirely cleared, but the inability to swallow had continued as on admission.

At first Bobbie had made strenuous resistance to the introduction of the tube, but this soon ceased and he would introduce the tube himself through the right nostril. Ten days after the mastoid operation he was discharged from the hospital. The ear was dry, but there was still some discharge from the mastoid wound. Food and water were still taken by tube only, which Bobbie still introduced himself. Meanwhile he was left alone in a room surrounded with tempting foods of various kinds to see if he would attempt to swallow, but no such attempts were made, although the child was quite alone and did not know that he was being observed.

Six weeks after the onset of the dysphagia the mother noticed that he was apparently swallowing saliva and possibly some fluids. At this time another fluoroscopic examination was made. According to the report of Dr. Blankenhorn "he was at first complacent about being examined and hopped onto the table, but soon began to cry and begged to be taken off, saying that he could not swallow. When given a mouthful of moderately thick barium this was seen to pass through the esophagus and down into the stomach in a normal way. He insisted, however, that he could not swallow. At the end of the examination he was quite cheerful and volunteered the information that he did not swallow anything. It was apparent, however, that he swallowed quite naturally and then spat up afterward." Ten days later he said that he swallowed everything, although his mother had been forced to rely on partial feedings by tube following the fluoroscopic examination in order to keep him properly nourished. The mastoid wound had been healed for some time.

The foregoing case presents some unusual features in diagnosis. A careful search of the literature has failed to discover a similar

history. The cases of so-called hysterical dysphagia reported by Plummer, Vinson and others were all in adults and, while there was an inability to swallow solid foods, soft foods and liquids were still swallowed freely. The dysphagia in these reported cases always followed choking attacks and difficulty in swallowing solid foods, usually meat. In addition it was associated with enlargement of the spleen and a secondary anemia, probably due, as Vinson says, to the quality of the foods taken. There was often associated an hypothyroidism, attributed by Plummer to a disuse atrophy. The difficulty was attributed to a spasm of the pharyngo-esophagus muscle and was always cured by the passage of a sound, no matter what size; in other words, by suggestion.

Inasmuch as a sore throat had been complained of, the possibility of a diphtheritic paralysis was considered. The throat on admission showed no signs of disease and smears were negative, as was also the Schick test. Another possibility could also have been an anterior poliomyelitis, although there were no other signs and the spinal puncture was negative.

The remaining possibilities were a spasm of the esophagus or a paralysis. Against the former is the fact that both the tube and the esophagoscope passed freely into the stomach, no resistance being encountered. A paralysis is accordingly the only apparent diagnosis remaining. That this was functional is evidenced by the history of the case. What was the predisposing cause of this functional disorder it is difficult to say. The only apparent cause was the infection in the middle ear and mastoid process. Still these, as well as the pulmonary involvement, might have been secondary to the dysphagia and the forced feeding. So, as stated at the beginning of my paper, whether we shall call this a case of dysphagia of septic origin or a case of hysterical dysphagia I leave to my auditors to determine.

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DISCUSSION

DR. BURT R. SHURLY, Detroit, Mich.:

This is an exceedingly interesting and unusual condition in children and yet it is one that often brings with it a great number of puzzling features. Some years ago I had a very similar case in a young adult who for weeks had been unable to swallow except under very unusual and queer conditions, and then only one or two special varieties of food. Any other variety she was absolutely unable to swallow. Complete examination led us to believe that there could be nothing but functional disturbance. This woman was unable to eat at the table or in the presence of anyone at the table, but she could stand at the sideboard and take one or two varieties of food. She was able to eat some peanuts. Of course one would believe that there must be with this a mental aberration or some approaching insanity perhaps, but the case was dealt with by having the patient come to the office with a bottle of milk and a sandwich. She had been absolutely unable to drink milk or any fluid substance for weeks. We got out the galvanic current with the laryngeal electrode and passed it down the esophagus and by the proper mental suggestions, such as we would use with hysterical aphonia, we were gradually able to accomplish the deglutition of food in the office, so for some days she brought her lunch with her every day. We succeeded in finally getting the milk and the sandwich down, together with additional ingredients each day.

The story reads like a fairy story. She went home and was later married. I have seen her with two perfectly normal children and absolutely no return of this functional dysphagia. In other words, it simulated hysterical aphonia just as much as anything could. The relief was absolute by these methods of electrical stimulation and suggestion, and she was able to eat at the table. It was an exceedingly interesting case and required no psychoanalysis or any other peculiar specialist, but just the ordinary old throat doctor.

Could I add one other case along this same line? Early one morning I was called to see a patient who had swallowed his false teeth. He was some nine miles away. He was the chauffeur of a family. I told them to bring him down to the hospital. I got in the machine with him and went to the hospital. This man was absolutely unable to swallow. For four hours he had tried to get a glass of water down and had absolutely lost the power of deglutition. We took him to the hospital and x-rayed the esophagus. We laid him down on the x-ray

table and when the x-ray plate came back, there was no foreign body to be seen. He had slept in a flannel undershirt. When we got him up, out of the folds of his undershirt dropped his false teeth. The man was soon able to swallow perfectly again.

I simply bring that out to show the most marvelous mental relationship that there is over the laryngeal muscles and the muscles of deglutition and one of the things that we should not forget is that wonderful and marvelous psychic relationship which we see constantly with all patients of this type.

I have seen many young children of this age exhibiting all the various hysterical manifestations. I think it is increasing. It is stated now that one out of every two beds in the hospitals of the country has a mental or nervous case, that there is an increase from 56 per hundred thousand to 554 per hundred thousand in the number of mental cases in the last few years hospitalized in our country. I believe that relationship is a growing unrest of the nervous system and that it is one that becomes more prominent, one that should be more constantly recognized by those of us who work in this field.

DR. CHAMBERLIN: Did she give a history of choking on solid food before it began?

DR. SHURLY: She choked on every food.

DR. CHAMBERLIN: These cases of Plummer's gave a definite history of choking on a piece of meat at the table and forever after they could not swallow.

DR. SHURLY: She would spit out everything. There was absolute relief by the methods I gave her.

DR. MERVIN C. MYERSON, Brooklyn, N. Y.:

May I ask Dr. Chamberlin whether the barium-meal film showed a picture that resembled that of spasm of the esophagus?

DR. CHAMBERLIN: Very much, yes.

DR. MYERSON: Then I would offer the suggestion that perhaps because this child had a pneumonia that was localized to the hilum area that the child very likely had a mediastinum pleurisy which may have been responsible for a periesophagitis, which caused a spasm of the esophagus. Then the psychic element later entered into the situation after the pneumonia was healed and the child being fairly bright and smart and having a fuss made over him continued his difficulty in swallowing for the effect it had on his family and those about him. I think that that might be an explanation worth considering.

DR. LEWIS A. COFFIN, New York City:

I have been much interested in Dr. Chamberlin's paper and case. I have not had children to deal with with this trouble but have had several adults, one of them being myself.

My first case was a woman about thirty years of age, who came to see me when I was working at the Manhattan Eye, Ear and Throat Hospital. I had been highly recommended to her. She was tall, frail and neurotic. She declared that she could not swallow solid food and that liquids were swallowed with difficulty.

Esophageal bougies entered the stomach easily without obstruction. I made up my mind the condition was due to hysteria. I told the lady that I was sure she would not obey orders and that, therefore, I must refuse to treat her. She begged, and after some apparent hesitation I agreed to see her the following morning at my office. When she appeared the next morning, I had in the drawer of my desk a hard-boiled egg and some Graham crackers. I began the visit by telling her that I was sorry I had made the appointment as I was certain she would not follow my advice.

She protested that she would and asked why I did not try her, when I opened the drawer, took out the egg and said "Eat this." She said "Oh, Doctor you know I cannot do that." After a little sparring of the same kind, she said "I'll try," and I am sure she was much astonished when the egg had disappeared. She ate one or two of the crackers. I praised and encouraged her efforts and instructed the woman accompanying her that I wished to see her on the following morning, but told her she must not bring her down unless she had eaten such and such a breakfast. They came down. The woman was discharged. Some years later, at some function in New York, a large lady approached me and said: "Dr. Coffin, you do not know me. I came to see you from Hudson because I could not swallow." She said she weighed 220 pounds.

Now as to myself. I had this feeling here about the midsternum region that I could not swallow a thing. The feeling was accompanied with considerable pain. I went over to see Dr. Chappell, one of our old members. He could see no reason for my symptoms and advised the expectant plan of treatment. I had, I believe, three different attacks, and made up my mind that it was all due to stomach indigestion.

My son is a doctor in general practice. One day about a year ago, as I was leaving the office, he said to me: "I wish you would look at this man. He says he is unable to swallow. He says it pains him

so at the midsternum region." I said to him: "I think it is gastro-intestinal," and my explanation was this. On account of stomach indigestion the esophagus contains a volume of gas, the bolus of food in the act of swallowing meets this volume of gas and gives rise to the sensation of obstruction and, of course, pain. Attention to the stomach digestion relieved the young man entirely.

I can easily conceive that a child would, under these conditions, refrain from swallowing to the extent of a seeming inability. The child reported by Dr. Chamberlin was young, he had a diseased mastoid and probably had a considerable gastrointestinal disorder corresponding with the findings of Marriot in children suffering from mastoid.

Whereas there may be an hysterical element in these cases, my belief is that it has its foundation in reality.

DR. LOUIS H. CLERF, Philadelphia:

It is a question whether we should accept dysphagia of toxic origin as a clinical entity. There are cases of dysphagia, however, which are extremely toxic and one can find no cause for the difficulty in swallowing. A man who recently came under my observation at the Jefferson Hospital had been ill for about ten days. He appeared very toxic. Dysphagia began four days before and was progressive. Malignancy was suspected and a barium study of the esophagus indicated a block of the lumen at the cricopharyngeal level. Indirect and direct examination of the larynx and pharynx and esophagoscopic study of the esophagus revealed nothing abnormal. A neurologist who examined the patient found no evidence of organic disease. A feeding tube was inserted and left *in situ*. Swallowing improved, but never returned to normal. A recent report from the patient's physician was that the patient succumbed to the infection. We were unable to determine its character.

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

To me the tremendously interesting feature of the report is the age of the patient. I had never known of anybody as young as that having either spasm or difficulty of that type in swallowing except the two types that the Doctor spoke of, diphtheritic paralysis or the oncoming of anterior poliomyelitis. I have had 3 of these last myself and made the diagnosis over the telephone in 1 case from a description of the symptoms. Sad to say, it proved to be true, and the patient died.

The youngest person out of the child age that I ever had with that

sort of a spasm, outside of the familiar ones we see in the female so often, was a man of the tramp species. He was in his twenties and unable to swallow at all. He was dehydrated and in a very sad state. It was in the days long ago. He could get whisky in those days for small sums. Simple cocainization of the entrance to the esophagus allowed him to swallow water with ease and he promptly went out and got drunk. He did not believe it was right to drink pure water, and so he adulterated his water. From then on he had no trouble at all. The spasm, I think, was produced by a wound or other sensitive spot at the entrance of the esophagus.

This subject has always greatly interested me because the professor with whom I worked forty-five years ago, as a student in Germany, was about the first man who ever had, to my knowledge, any physical explanation for the globus hystericus, and it was he who called attention to the fact that it might be the lingual tonsil when enlarged. But what was more to the point, that the tremendous big plexus of bloodvessels upon which the lingual tonsil lies is capable of considerable engorgement, and during that period of engorgement the patient would have an added desire to swallow, would be conscious of the feeling of something, the "globus," which he would try and remove by swallowing. This exaggerates into spasm in one of unstable nervous system. So through all these years these cases have always attracted my attention, and I surely have missed it if any came in that were as young as yours, Dr. Chamberlin.

DR. CHAMBERLIN (in closing):

I just wondered if the first case that Dr. Shurly spoke of, this young woman, might not be typical of those cases which Plummer first reported, and afterward Vincent, which is called (especially by the English laryngologists at the present time) Plummer's disease.

In regard to Dr. Myerson's question, of course, spasm followed in that case. The pediatricist and the internist who were called in thought that our case was one of spasm, but I cannot imagine a case of spasm where a tube could be passed so readily and easily. The child would take the tube and pass it down himself. Manifestly he was not going to pass that tube if it caused him any pain.

I suppose that Dr. Coffin's case should properly have been referred to the psychoanalyst, unless Dr. Coffin still retained those powers which Dr. Barlow mentioned yesterday.

I agree with Dr. Clerf that we should hesitate in ascribing a toxic origin to these cases, and as I said both at the beginning and the end

of my paper, I do not know that I am right in ascribing the origin in this case to a localized toxicity, and yet I do not know of any other cause.

Dr. Swain made reference to the age of the patient. The age made it an extremely interesting case for us. I wish to assure you that we have searched the literature fully and have been utterly unable to find any reference in the literature to such a condition occurring in a patient of this age. If any of you should come upon anything in the literature which might have a bearing I should be greatly obliged if you would let me know about it.

LATERAL TRANSTHYROID PHARYNGOTOMY

TROTTER'S OPERATION FOR MALIGNANT CONDITIONS OF THE LARYNGOPHARYNX*

HENRY BOYLAN ORTON, M.D.

DRAWBACKS to operations on the laryngopharynx may be classified, according to Trotter,¹ into three groups: (1) The uncertainty of cure, which is the most important; (2) the danger of the operation and the distresses of convalescence, and (3) the mutilation and disability that may be left by an operation otherwise perfectly successful. This still holds true.

Intrinsic cancer is being well attacked by both laryngofissure and laryngectomy by Fellows of the American Laryngological Association. But I think that something can be done for patients who are suffering from so-called extrinsic cancer of the larynx. Better terms are epilaryngeal cancer, a growth originating on the epiglottis, aryepiglottic folds, pyriform sinus or lateral wall of the pharynx, and hypopharyngeal cancer, occurring in the lower portion of the pharynx.

In this presentation I claim nothing original, for I quote freely from Mr. Trotter's articles and personal communications, but by plaster casts and photographs I shall attempt to show a procedure by which to attack malignant conditions of the pharynx: lateral transthyroid pharyngotomy. I shall limit myself to that portion known as the laryngopharynx.

A few years ago, while visiting in England, I saw Mr. Trotter with comparative ease remove by this route a postericoid carcinoma, an operation in which heretofore the larynx as well has been sacrificed. I became enthusiastic over it, and since that time I have had occasion to perform this operation on 9 patients. If my results are not good, it is due to the operator and not to the operation. In these 9 cases I had an operative mortality of two,

* The dissections and casts were made with the assistance of Dr. H. P. Mosher in his laboratory at the Harvard University Medical School in February, 1930.

or 22.3 per cent, both of the patients dying within thirty-six hours of septic pneumonia. Three patients had a recurrence in the glands in six months, and 1 patient in nine months, following operation; 1 lived for one and a half years, and 2 are living at this writing, 1 patient now for over one year and the other for eight months.

Microscopically I am concerned only with the squamous-cell epithelioma. Mr. Trotter's classification shows (Fig. 1A) the hypopharyngeal group which involves the cricoid region and the posterior pharyngeal wall, and which is practically limited to women. Three of my cases belonged to this group.

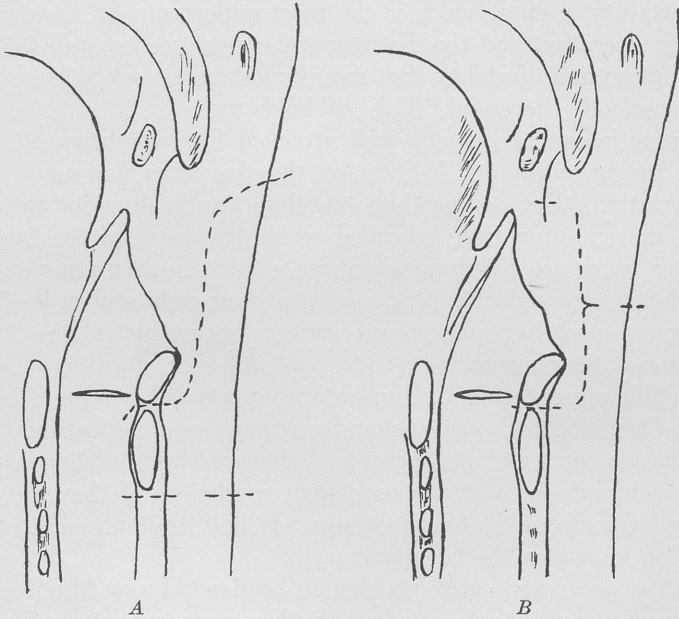


FIG. 1.—A, the hypopharyngeal group, practically limited to women (Wilfred Trotter); B, the epilaryngeal group, the type ordinarily occurring in men (Wilfred Trotter).

Fig. 1B shows the group known as the epilaryngeal, which involves the arytenoids, aryepiglottic folds, epiglottis, pyriform sinus and possibly the base of the tongue. This group is almost entirely limited to men. Six of my cases belonged to this group.

In the order named, a growth involving the epiglottis, aryepiglottic folds, lateral wall of the pharynx, postericoid and pyriform

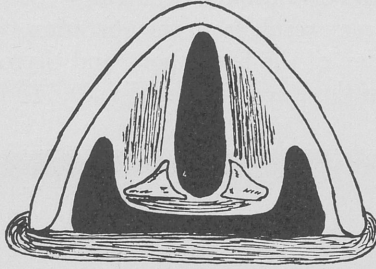


FIG. 2.—Diagram of section through the pyriform sinus of the pharynx to show its relation externally to the thyroid cartilage and internally to the lateral wall of the larynx (Wilfred Trotter).

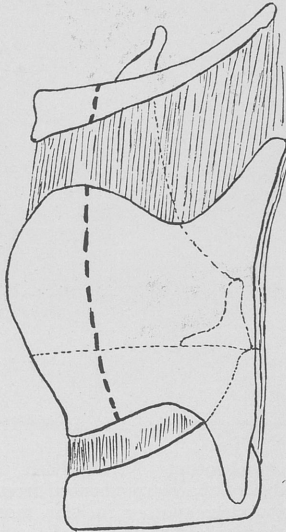


FIG. 3.—The finely dotted lines indicate the relative positions of the aryepiglottic fold, the arytenoid cartilage and the vocal cord. The thickly dotted lines show where the great cornu and the thyroid ala are divided in the operation. When these parts have been removed, the laryngopharynx is accessible (Wilfred Trotter).

sinus gives the best prognosis. If the growth has had its origin from the lateral wall of the larynx, it soon involves the pyriform sinus and thyroid cartilage, and nothing short of complete laryn-

gectomy, with a partial or entire cervical esophagus, will be of any use. This group is the most discouraging one for any operative procedure, yet if the condition is seen early enough, a few additional years of life may result from the operation. By the removal of the shaded portion of the hyoid bone and thyroid cartilage one has an approach to these conditions (Fig. 3).

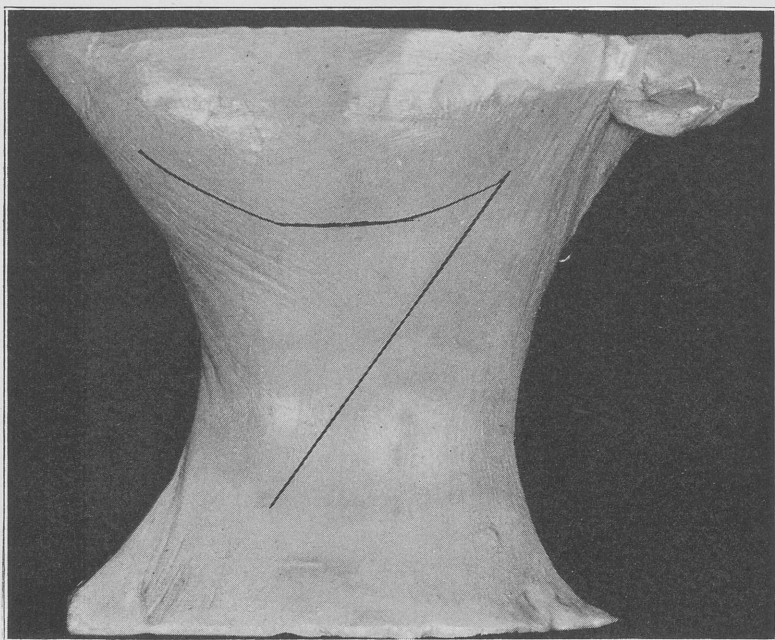


FIG. 4.—A preliminary tracheotomy having been performed, an incision is made along the anterior edge of the sternomastoid from the angle of the jaw to the suprasternal notch; from this, another incision more or less at right angles extending along the lower border of the jaw to the symphysis. These incisions vary, depending on the type of operation and the amount of tissue to be removed.

After a preliminary tracheotomy, the incision for ordinary trans-thyroid pharyngotomy would be along the anterior border of the sternomastoid muscle, reflecting anteriorly and posteriorly the muscles of the larynx. But as most of these patients, by the time they are examined, have enlarged glands of the neck, from either infection or lymphatic extension, it becomes necessary to perform

a resection, and the former incision is inadequate. We then make an additional incision more or less at right angles to the original one, running from the angle of the lower jaw downward, forward and upward to the symphysis (Fig. 4).

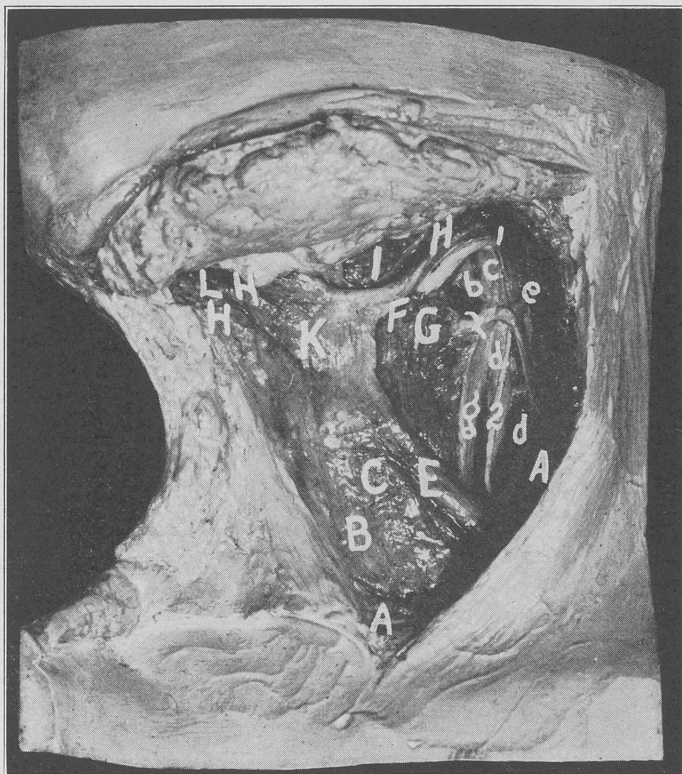


FIG. 5.—This shows the skin and platysma retracted. The cervical fascia, superficial lymphatic glands and submaxillary gland are removed. The anterior edge of the sternomastoid has been retracted outward to expose the structures lying beneath it. The carotid sheath is opened, showing the vessels. The muscles shown are: A, the sternomastoid; B, the sternohyoid; C, the sternothyroid; D, the thyroid; E, the omohyoid; F, the stylohyoid (cut); G, the hyoglossus; H, the digastric; I, the submaxillary fossa; K, the suprahyoid aponeurosis; L, the mylohyoid, and Z, the pomum adami. The vessels are: a, the common carotid; b, the external carotid; c, the internal carotid; d, the internal jugular vein; e, the anterior division of the temporomaxillary vein joining the facial; f, the facial, and g, the carotid sheath. The nerves are: (1) the hypoglossal, and (2) the descendens noni.

These flaps and the platysma are turned back; the superficial fascia, the submaxillary gland and the deep fascia are likewise removed upward and backward as far as the spinal accessory nerve and posterior triangle. This step exposes the deep vessels of the neck and the muscles surrounding the larynx (Fig. 5).

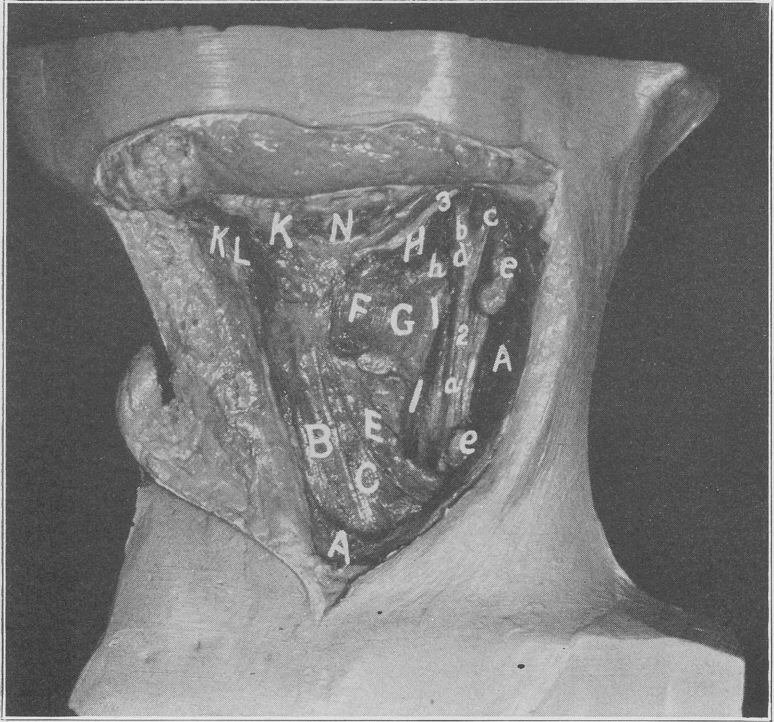


FIG. 6.—This shows the deep cervical fascia removed and the internal jugular vein ligated and resected. All veins and arteries coming off anteriorly have been ligated and cut. The pneumogastric nerve is seen. The thyrohyoid muscle is cut, exposing the thyrohyoid membrane. The inferior constrictor has been separated from the thyroid cartilage. The hyoid bone is also exposed. The muscles shown are: A, the sternomastoid; B, the sternohyoid; C, the sternothyroid; E, the omohyoid; F, the thyrohyoid (cut); G, the thyrohyoid membrane; H, the hyoglossus; I, the inferior constrictor; K, the digastric; L, the mylohyoid; M, the suprahyoid aponeurosis and N, the submaxillary fossa. The vessels are: a, the common carotid; b, the external carotid; c, the internal carotid; d, the superior laryngeal; e, the internal jugular ligated and resected; f, the thyroid cartilage-cornua, and h, the hyoid bone. The nerves are: (1) the pneumogastric; (2) the descendens noni, and (3) the hypoglossal.

All the vessels that come off anteriorly from the great vessels are ligated and cut. The internal jugular is ligated and resected. The external carotid may or may not be ligated. The sterno-

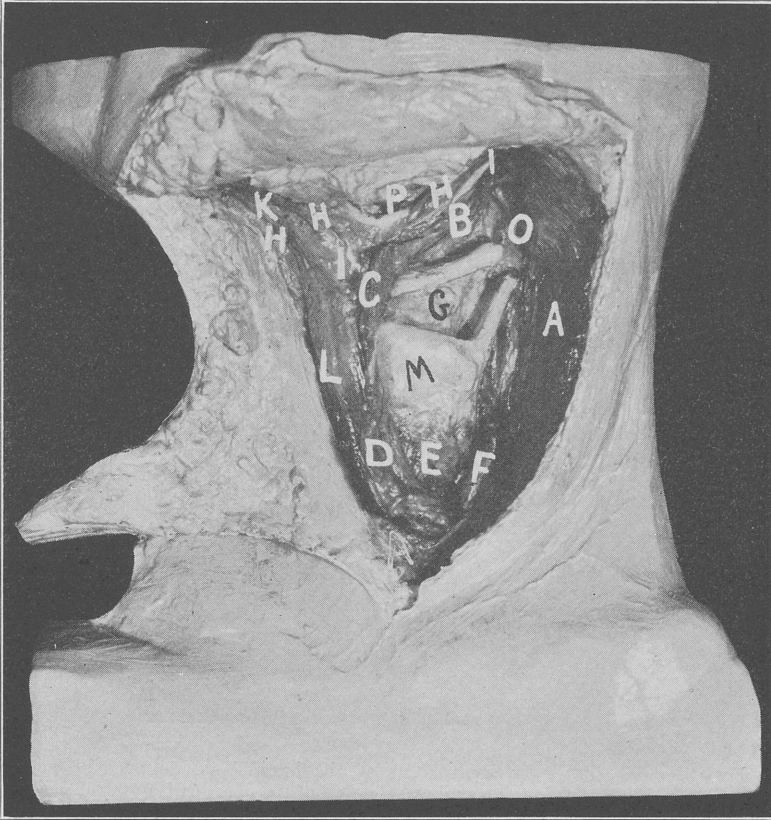


FIG. 7.—This shows the anterior edge of the sternomastoid sutured to the prevertebral fascia covering and protecting the great vessels. The area is drained by a counteropening posteriorly. The muscles have been reflected from the hyoid bone and the thyroid cartilage. The constrictors of the pharynx are reflected, and the great cornu of the hyoid bone and thyroid cartilage exposed for removal. The muscles are: A, the sternomastoid sutured to the prevertebral fascia; B, the hyoglossus; C, the thyrohyoid; D, the sternothyroid; E, the cricothyroid; F, the inferior constrictor; G, the thyrohyoid membrane; H, the digastric; I, the suprahyoid aponeurosis; K, the mylohyoid; L, the sternohyoid; M, the thyroid cartilage; N, the great cornu of the hyoid; O, the middle constrictor, and P, the submaxillary fossa. The nerve shown (1) is the hypoglossal.

mastoid may or may not be removed, depending on the glandular involvement (Fig. 6).

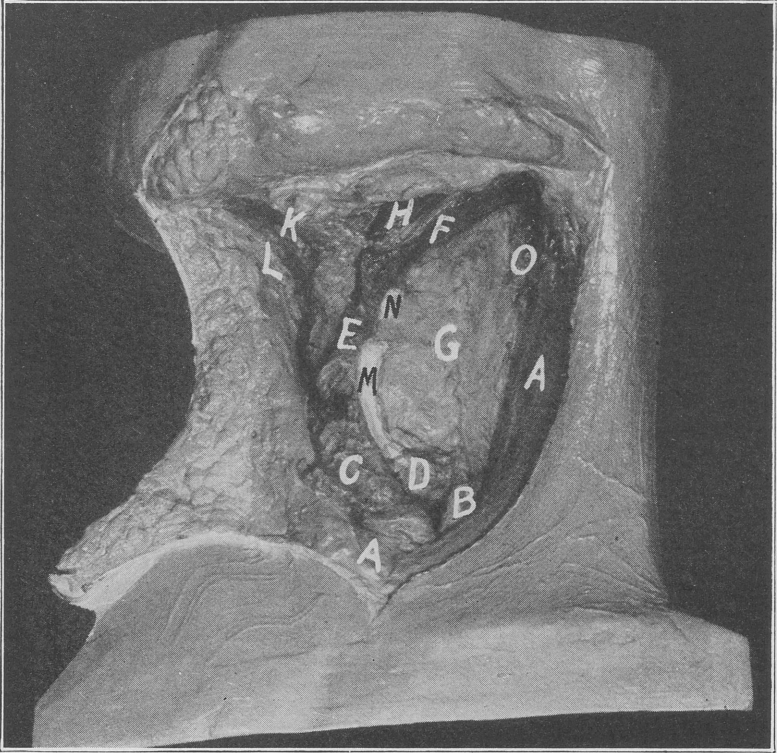


FIG. 8.—This shows the great cornu of the hyoid bone removed as well as the greater half of the thyroid cartilage; the wall of the pharynx is now well exposed and ready for incision. The muscles and structures shown are: A, the sternomastoid; B, the inferior constrictor; C, the sternothyroid; D, the cricothyroid; E, the thyrohyoid; F, the hyoglossus; G, the pharynx ready to be opened; H, the submaxillary fossa; K, the digastric; L, the mylohyoid; M, the thyroid cartilage removed; N, the great cornu of the hyoid removed, and O, the middle constrictor.

The next step is to suture the sternomastoid to the prevertebral fascia; this covers the great vessels and thus protects them from infection when the pharynx is opened. The area is drained by a counter-opening posteriorly (Fig. 7).

The following muscles are then reflected from the hyoid bone and thyroid cartilage: the hypoglossal, thyrohyoid, sternohyoid, sternothyroid, stylopharyngeus, and the inferior and middle

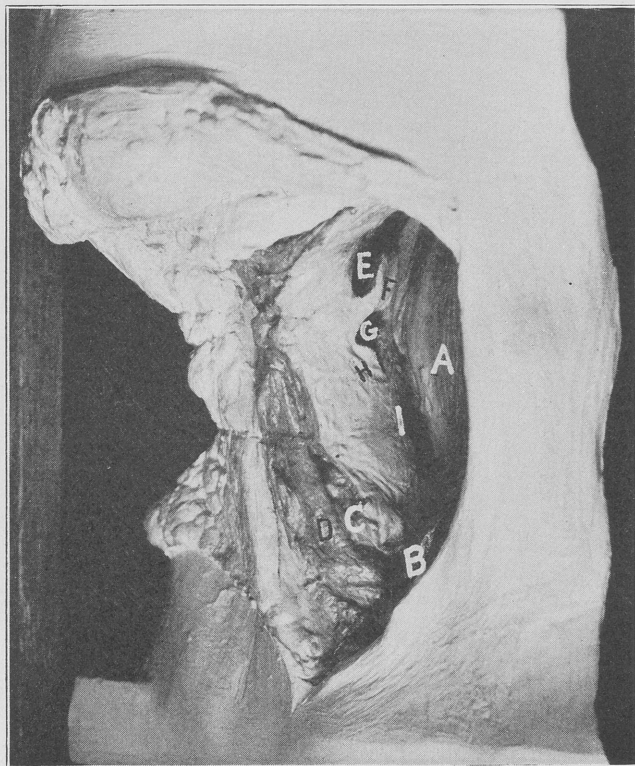


FIG. 9.—This shows the pharynx opened, exposing the base of the tongue, epiglottis, arytenoids and postcricoid regions. This gives an excellent approach to a growth in either the epilarynx or the hypopharynx. The muscles and structures shown are: A, the posterior wall of the pharynx; B, the sternomastoid; C, the cricothyroid; D, the sternothyroid; E, the base of the tongue; F, the epiglottis; G, opening into the larynx; H, the arytenoids, and I, the cricoid.

constrictors of the pharynx. One is then ready to remove, by means of heavy scissors, the exposed portions of the hyoid bone and the greater portion of the thyroid cartilage (Fig. 8).

This permits one to palpate the growth through the pharyngeal

aponeurosis and mucous membrane, and to decide where to make the opening into the pharynx so as to be well around the malignant area; or if the growth is too large to remove, one may back out, so to speak, and not proceed any further (Fig. 9).

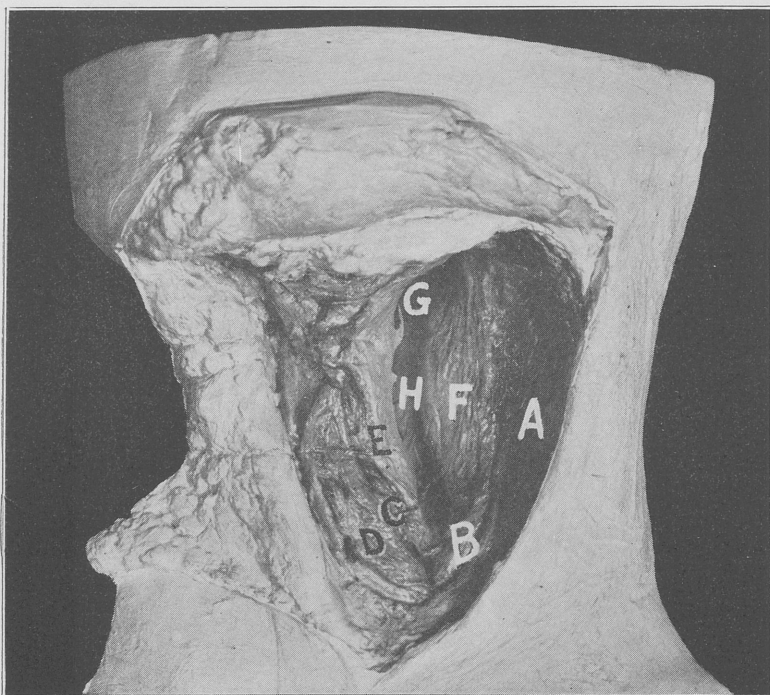


FIG. 10.—The muscles and structures shown are: A, the sternomastoid; B, the inferior constrictor; C, the cricothyroid; D, the sternothyroid; E, the cricoid; F, the postpharyngeal wall; G, the epiglottis, and H, the arytenoids.

The next step is to open the pharynx, an incision which gives an approach to either a hypopharyngeal or an epilaryngeal growth, and which may be continued upward by going through the mandible, as in one of my cases, to remove a growth higher up (Fig. 10).

Before the operation, one has a fair idea of how much tissue it will be necessary to remove, and the flap is made sufficiently large to fill up the defect. The picture shows a flap of skin turned in to

make up a defect in a postericoid carcinoma. A feeding tube is inserted into the esophagus. Later, a plastic operation is performed to close the neck (Fig. 11).

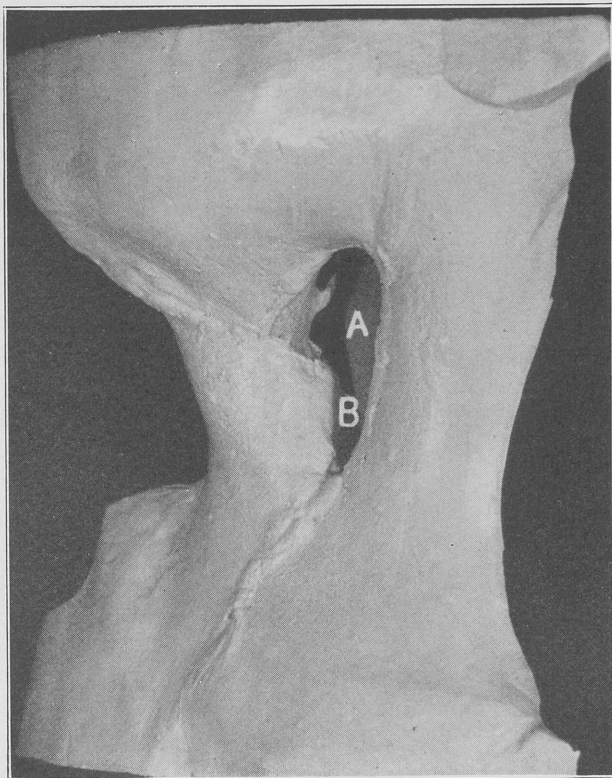


FIG. 11.—This shows the closure for postericoid growth, when it has become necessary to turn in a flap of skin from the neck to make good the defect in the pharynx. This gutter is closed later by a plastic flap. *A*, the posterior pharyngeal wall, and *B*, the skin flap turned in.

As illustrated in Fig. 12*A*, if the growth is on the anterior wall of the esophagus, the flap will be made from *A* to *B*. The black portion is removed and then the flap is turned in from *B*. If the growth occurs posteriorly, the flap is turned in from *B* over the muscles and vessels, as shown in Fig. 12*B*.

REPORT OF CASES. CASE I.—M. T., a white woman, aged sixty years, married, was referred to me on March 30, 1928, for difficulty in swallowing. Nothing in the family history was of any importance. Her general health had been good until September, 1927, when she had trouble with swallowing, without pain. She had a slightly enlarged thyroid gland for which she had been treated at one of the local hospitals; at another hospital she had had her tonsils removed. The only other complaint that she had besides slight trouble in swallowing was the expectoration of considerable phlegm which accumulated in her mouth. The Wassermann test, urinalysis, *x-ray* examination of the chest and blood chemistry all gave negative results.

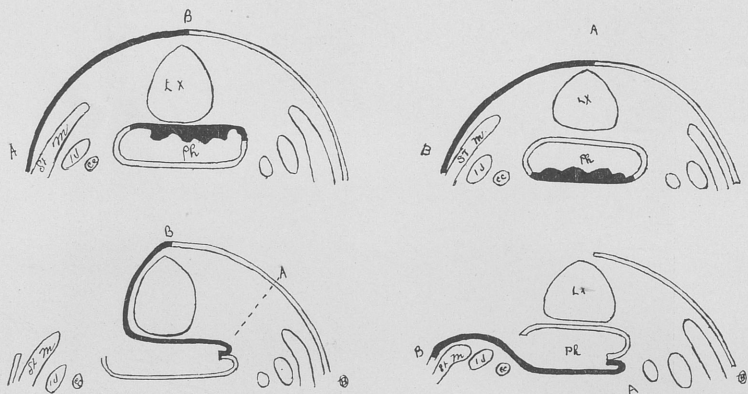


FIG. 12.—A, disposition of skin flap in operation for removal of a hypopharyngeal growth-tumor on the anterior wall (Wilfred Trotter); B, disposition of skin flap in operation for removal of a hypopharyngeal growth-tumor on the posterior wall (Wilfred Trotter).

Laryngeal examination showed the left cord and arytenoid fixed and a mass in the postericoid region, involving the entire hypopharynx. The clinical diagnosis was carcinoma, and an operation was suggested.

I did not see this patient again until November, 1928, ten months later, when she was admitted to the City Hospital. There had been a gradual increase of difficulty in swallowing but no pain; the patient stated that she had had a number of *x-ray* treatments. She had had no difficulty in breathing. She had lost about 50 pounds (22.7 kg.) in the previous four months. Laryngeal examination on November 1 showed an extensive growth, which seemed to spring from the posterior wall of the esophagus and which extended well into the pyriform

sinus; both arytenoids were edematous. This growth apparently had progressed considerably in ten months. The patient and her family wanted something done, although I could promise them no hope.

On November 2 I performed a transthyroid pharyngotomy. A tracheotomy was performed and a No. 4 tracheotomy tube was inserted. An incision about 3 inches (7.6 cm.) long was made across the median line of the neck; then an incision was made from the upper portion to the angle of the jaw and a lower incision to the clavicle. The skin was reflected backward, the fascia to the posterior border of the sternomastoid was removed along with the resection of the internal jugular; the external carotid was ligated, and the submaxillary gland on the left side was removed. The left side of the thyroid cartilage was removed. The sternomastoid was sutured to the prevertebral fascia. The inferior constrictor was cut through, exposing the lateral wall of the pharynx; this was opened and the growth inspected. The skin was reflected into the pharynx; the upper part of the flap was sewed to the pharyngeal mucous membrane. The same procedure was followed below. The feeding tube was passed into the esophagus and anchored at the opening of the neck. A dressing was applied. The patient died on the fourth day following operation. The pathologic report showed carcinoma.

CASE II.—S. K., a white woman, aged fifty-seven years, married, consulted me on January 17, 1929, complaining of inability to swallow. Her father died of cancer, and her husband died of some internal growth (?). The personal history was negative. The present illness began in August, 1928, with difficulty in swallowing. She noticed some enlarged glands on the side of the neck which at times were painful, the pain radiating to the right ear. Laryngeal examination showed the left cord abducting and adducting normally, with a mass in the postericoid region. The right cord was interfered with somewhat in these movements by a mass in the right aryepiglottic fold and posterior lateral wall of the pharynx. Urinalysis, the Wassermann test, blood chemistry and *x*-ray examination of the chest gave negative results.

An operation was performed on January 28, 1929. A tracheotomy was performed under procaine hydrochloride anesthesia, followed by intratracheal anesthesia. An incision was made so as to cover in the defect caused by the removal of a growth from the posterior wall of the larynx. The fascia and glands along the deep vessel were removed. The internal jugular was ligated and resected. The sub-

maxillary gland was removed. The muscles of the larynx and the hyoid bone were freed; the hyoid bone and a portion of the thyroid cartilage were removed, exposing the wall of the pharynx. The growth was then palpated and found to extend from above the larynx to below the third ring of the trachea. Because of this extension, it was decided not to enter the pharynx. On February 4, gastrostomy was performed. The patient refused further operation, and left the hospital in February. She died on June 9. The pathologic report on a specimen showed it to be undifferentiated carcinoma of the esophagus.

CASE III.—H. B., a white man, aged fifty-six years, married, was referred to me in June, 1929. The personal history was negative. The patient was well until three months before examination, when he noticed a small ulceration on the buccal surface of the right cheek. This area became more and more eroded, until it reached the size of a quarter. It then involved not only the buccal mucous membrane, but also the jaw and the side of the mouth. This became so painful that the patient had to seek medical aid. There were some enlarged cervical glands on the right side. Urinalysis, the Wassermann test, blood chemistry and *x*-ray examination of the chest gave negative results.

On June 10, under rectal anesthesia, an incision was made in the middle of the chin downward and outward to the middle of the clavicle on the right side. Flaps were turned backward and forward. The glands of the neck, the deep superficial fascia and the submaxillary gland were removed. The external carotid artery was ligated. An incision was then carried upward through the lip, and the entire right side of the face was turned back. The jaw was incised at the ramus, and 3 inches of the right side of the jaw were excised. Then by means of diathermy the right half of the tongue, the floor of the mouth and the side of the pharynx were removed. The patient was discharged from the hospital three weeks later. I did not see him again until October 3, when a slight recurrence on the inner surface of the ramus of the right jaw was observed. He was admitted to the hospital on October 7 for further operation by means of surgical diathermy. While rectal anesthesia was being administered, he suddenly ceased to breathe. The pathologic report showed squamous-cell carcinoma.

CASE IV.—J. E., a white man, aged forty years, married, was admitted to the hospital on December 16, 1929, with a complaint of difficulty in swallowing. His mother had died of cancer at the age

of fifty-eight. Nothing in the antecedent personal history was of interest. The patient had had trouble with his throat for a year, and had gradually encountered difficulty in swallowing. In attempting to swallow food he readily gagged, and he felt that there was an obstruction in his throat. The condition became progressively worse. He complained of slight hoarseness. Laryngeal examination showed a large growth springing from the right side of the pyriform sinus, which caused a sluggish movement of the right arytenoid. Urinalysis, blood chemistry, the Wassermann test and x-ray examination of the chest gave negative results.

An operation was performed on January 6, 1930. A transverse tracheotomy was performed, and then a laryngoscopy for direct inspection. I was under the impression that the growth sprang more from the posterior wall, as a probe would not pass around it. An incision was made from the angle of the jaw downward and forward beyond the median line, then downward over the left side of the thyroid cartilage to just above the tracheotomy opening and then backward beyond the border of the sternomastoid muscle. This flap was dissected backward; the glands of the neck were dissected, and several glands were found along the carotid sheath. The internal jugular vein was ligated and resected. The submaxillary gland was removed along with the deep fascia. The sternomastoid muscle was sutured to the prevertebral fascia protecting the great vessels. The muscles were freed from the hyoid bone and thyroid cartilage, exposing the lateral wall of the pharynx, which was opened; on inspection, the growth proved to be so extensive that it involved the entire pyriform sinus. It was then decided to remove the entire growth and the larynx from above downward, with about 2 inches (5 cm.) of the cervical esophagus. All bleeding was controlled. A skin flap was turned in; the upper part of the esophagus was sutured to the skin and the lower part to the lower portion of the skin flap, thereby forming the posterior wall of a new esophagus. A feeding tube was inserted through the nostril, and the trachea was sutured to the skin. An extensive operative procedure was thus employed. The man died on January 9, three days after operation. The pathologic report showed the specimen removed to be a squamous-cell carcinoma, Type IV.

CASE V.—J. D., a white man, aged fifty-four years, married, was referred to me on October 12, 1929, complaining of pain on the left side of his tongue.

Laryngeal examination showed a postcricoid carcinoma on the right side. Urinalysis, the Wassermann test, blood chemistry and roentgen examination of the chest yielded negative results.

An operation was performed on November 4. After preliminary tracheotomy an incision was made so that a flap could be turned in to form the anterior wall of the esophagus to replace the amount of tissue that was to be removed. The fascia and the glands along the vessels were removed. The jugular vein was ligated and resected. The external carotid was ligated. The submaxillary gland was removed. The sternomastoid was sutured to the prevertebral fascia. The pharynx was exposed but was not opened. The dissection of the gland took a long time because *x*-ray and radium treatment had fused the various planes, making dissection difficult. The skin was closed with the intention of completing the operation later. The patient did not return for observation as soon as I expected; when seen, he stated that he did not wish to have anything further done. The growth was in the postcricoid region, involving the pyriform sinus. The patient was discharged from the hospital on November 16, but continued to have *x*-ray and radium treatment. He became worse, and died on March 5, 1930.

A section was made for pathologic examination. The gland was found to be entirely replaced by invading groups of carcinomatous cells and fibrous productive tissue. The carcinomatous cells appeared moderately active, with larger cells and mitosis present, but there was also considerable evidence of differentiation in the formation of epithelial pearls. There was considerable protective fibrosis. The diagnosis was grade 3 carcinoma in the lymph gland.

CASE VI.—E. W., a white woman, aged fifty-five years, married, was referred to me on October 29, 1927, with a complaint of difficulty in swallowing.

Laryngeal examination showed both arytenoids to be slightly edematous. Both cords abducted and adducted normally. On phonation there was seen in the postcricoid region of the esophagus the upper surface of an irregular mass with ulceration in the center; some cervical glands were palpable on the left side. Urinalysis, the Wassermann test, blood chemistry and *x*-ray examination of the chest yielded negative results.

On November 19, the entire mass, including the anterior wall of the esophagus and larynx, was removed with about 2 inches of the posterior wall. The growth was a squamous-cell carcinoma. Fig. 13 shows the extent of the growth.

On February 14, 1928, a gastrostomy was performed to enable the patient to be fed while we were reconstructing the anterior wall of the esophagus. Fig. 14*A* shows the upper opening and lower opening of the esophagus with the trough forming its posterior wall. After the gastrostomy was performed, the nasal feeding tube was removed.

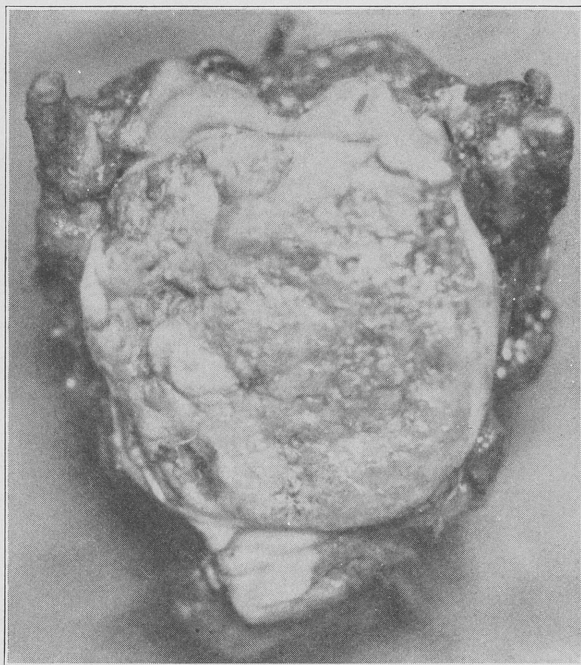


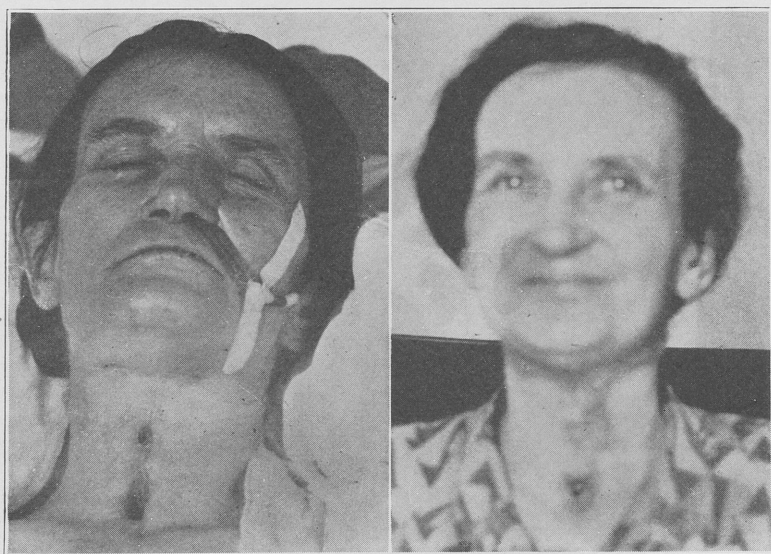
FIG. 13.—Case VI. Squamous-cell carcinoma.

On March 1, 1928, the edges of this trough were undermined. A flap was turned down from the chin to form the anterior wall. The parts undermined were brought together in the median line; a double row of sutures was made to prevent leakage. Fig. 14*B*, shows the patient after operation.

On pathologic examination, no evidence of papillary ingrowth into the tumor area was found. The diagnosis was squamous-cell carcinoma.

CASE VII.—P. D., a white man, aged sixty-three years, married, was referred to me on March 16, 1929, for a swelling of the neck of three weeks' duration. He had been troubled with occasional hoarseness.

Laryngeal examination showed the right arytenoid and cord moving freely on abduction and adduction; above the left arytenoid there was an irregular mass. Fig. 15 shows the extent of the swelling before operation. Urinalysis, the Wassermann test, blood chemistry and x-ray examination of the chest yielded negative results.



A

B

FIG. 14.—Case VI. A, appearance of patient after gastrostomy; B, appearance of patient after operation.

An operation was performed on April 8. Under rectal anesthesia a low transverse tracheotomy was performed. An incision was made along the anterior border of the sternomastoid muscle from the angle of the jaw downward to the level of the hyoid bone, going beyond the median line of the neck, then downward to within 1 cm. of the tracheotomy wound and then backward to the anterior border of the sternomastoid. The skin flaps turned back exposed the muscles of the entire left side of the neck to the angle of the jaw. The deep fascia of the

neck and the internal jugular vein were removed along with the submaxillary gland; this exposed a mass of glands (Fig. 16 *A*) below the

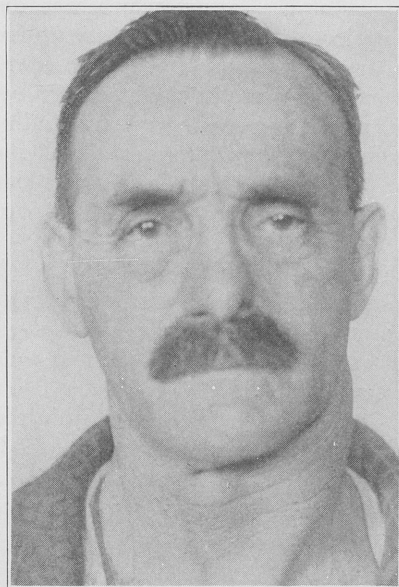
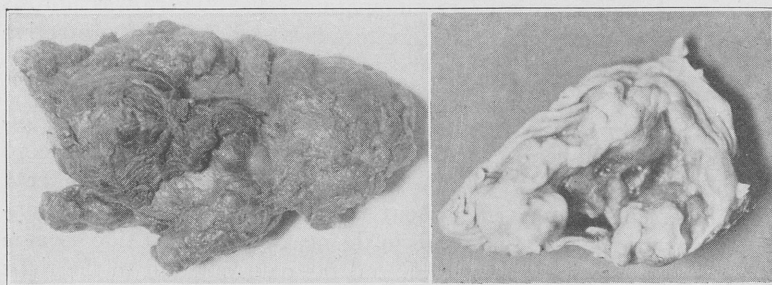


FIG. 15.—Case VII. Swelling of the neck before operation.



A

B

FIG. 16.—Case VII. *A*, mass of glands removed; *B*, postericoid growth.

sternomastoid muscle. This mass involved the deep vessels of the neck. The carotid artery was dissected free from it. The vagus nerve

was freed from this mass, and the upper part of the jugular vein was ligated and cut at the angle of the jaw; the muscle, vein and mass of glands were removed. The pharynx was opened, exposing a carcinoma of the posterior part of the larynx which was dissected free and removed. The anterior esophageal wall was sutured to the mucous membrane of the larynx; the pharynx was closed with three drains. The edges of the incision were brought together by means of clips. The dissection of glands was extensive and adherent to various structures. Exposing the pharynx, a very small postericoid growth was removed (Fig. 16*B*). There was not much loss of tissue in the esophagus; it was not necessary, therefore, to turn in the flap to make up a defect.

The patient recovered from his operation, got along without his tracheotomy tube and had a good voice. On August 21 he had a recurrence of the growth in the posterior triangle. Deep *x*-ray treatment and radium treatment was given without much benefit. The patient died on December 19.

There had been no recurrence of growth within the pharynx. It is quite noticeable that in these cases the recurrences have been in the glands and not at the point of original growth.

Pathologic examination of the tissue on April 17 revealed an invasive growth composed of groups and strands of carcinoma cells. The growth was very cellular, with only slight fibrous production in the stroma. The cells were undifferentiated and showed numerous mitotic forms. The diagnosis was carcinoma. On April 27, examination of a gland in the neck revealed extensive invasion and replacement by active and undifferentiated carcinomatous cells. There was fibrosis and an acute inflammatory reaction which was of doubtful protective value. The diagnosis was secondary carcinoma of the lymph nodes.

CASE VIII.—C. F., a white man, aged sixty years, married, consulted me on October 15, 1929, for sore throat and swollen glands of the neck. He had always enjoyed good health. On September 1 he had consulted a physician about soreness in the throat. The condition grew worse, and the glands in the neck on the right side became enlarged; swallowing was painful, and the pain radiated to the right ear and to the right side of head. At times he expectorated some blood.

Laryngeal examination revealed an ulceration on base of the tongue, involving the epiglottis and aryepiglottic fold, and overgrowth of tissue in the glosso-epiglottic fold, extending upward to the palatoglossal muscle. Abduction and adduction were normal. Urinalysis,

the Wassermann test, blood chemistry and *x*-ray examination of the chest yielded negative results.

The patient entered the hospital on October 23, and on October 28 a transthyroid pharyngotomy was performed. Under rectal anesthesia I made a block dissection of the neck, removing the deep cervical fascia and exposing deep structures of the neck. The submaxillary gland was removed; the muscles were separated from the hyoid bone on the right side and the right alæ of the thyroid cartilage

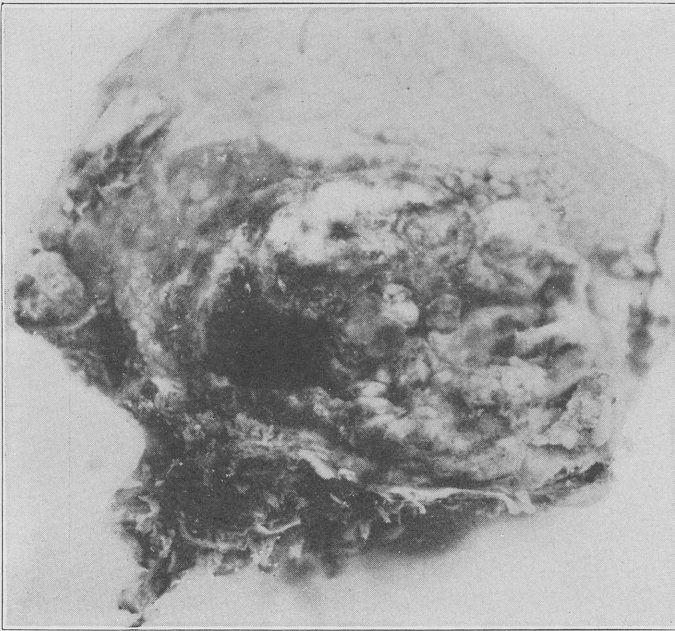


FIG. 17.—Case VIII. Growth removed from the posterior border of the tongue.

on the same side; part of the hyoid bone and thyroid cartilage were then removed. The anterior border of the sternomastoid was sutured to the prevertebral fascia. The lateral wall of the pharynx (Fig. 17) was opened and the growth on the posterior border of the tongue inspected. Then the epiglottis, right aryepiglottic fold and posterior third of the tongue as well as part of the palatoglossal muscle were removed by diathermy. The pharynx was closed with two layers of sutures; three drains in the neck and skin were brought together

with skin clips. The patient was discharged from the hospital on November 27.

On pathologic examination the tongue was seen to be ulcerated and had a growth about 2 cm. in diameter at the base. There was an extensive carcinomatous growth of the tongue in which all layers of

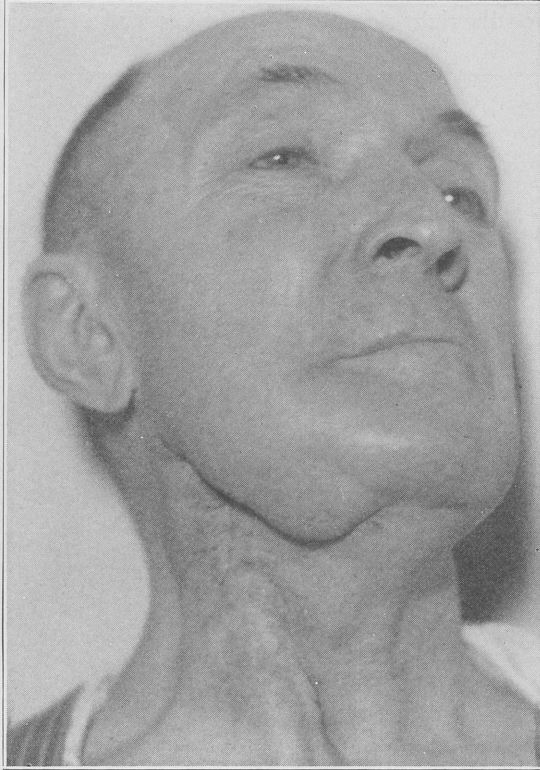
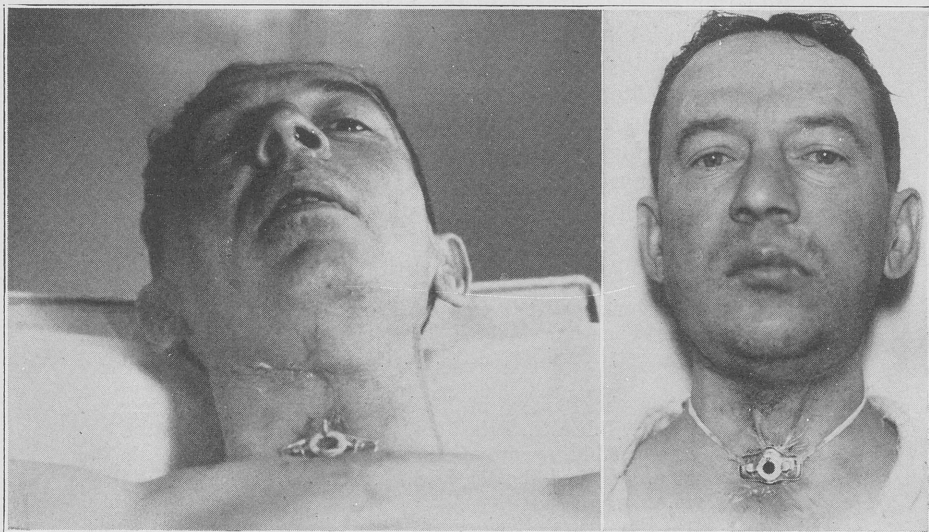


FIG. 18.—Case VIII. Appearance of patient after discharge from the hospital.

the surface epithelium were involved. There were numerous epithelial pearls derived from the squamous superficial layer. The growth was extensive and of a high grade of malignancy. Sections from two lymph nodes showed carcinomatous involvement of the same type as the primary growth.

CASE IX.—J.K., a white man, aged forty-three years, married, was referred to me on July 3, 1929. The patient had enjoyed good health until nine months (October, 1928) before, when he began to complain of soreness on the right side of the throat and pain radiating to the right ear. Laryngeal examination showed a large ulceration in the right pyriform sinus involving the lateral wall of the larynx. Urinalysis, the Wassermann test, blood chemistry and x-ray examination of the chest yielded negative results.



A

B

FIG. 19.—Case IX. A, appearance of patient following operation; B, appearance of patient on discharge from the hospital.

Under rectal anesthesia, on July 8, an incision was made on the right side of the neck to form a flap which would cover the defect on the posterior wall of the esophagus, for I intended to remove not only the larynx, but the anterior and posterior walls of the esophagus. The deep fascia and the submaxillary gland were removed. The internal jugular vein was resected on that side. The larynx was skeletonized and the sternomastoid muscle on the right side was sutured to the prevertebral fascia. The trachea was amputated at the second tracheal ring. Then with diathermy the entire mass, including the

anterior and posterior wall of the esophagus, was removed. All bleeding was controlled. The upper flap of skin was sutured to the mucous membrane of the anterior portion of the hypopharynx. The triangular flap of skin was then turned in and sutured to the posterior surface of the hypopharynx and to the skin on the opposite side. The lower portion of this flap was sutured to the posterior wall of the amputated esophagus just above the tracheal opening. This made three sides of a new esophagus. In September, a plastic operation was performed to form the anterior wall. The sides of the trough were undermined and sutured in the median line. This left a raw surface to fill in. A swinging flap was made from the left side of the neck with the pedicle uppermost. The sides were sutured to the undermined edges along the trough. Fig. 19 *B* shows the patient at the time of his discharge from the hospital, on January 19, 1930.

Pathologic examination revealed hypertrophy of the epithelium in which the cells showed malignant changes; this was invading the stroma in small groups. The diagnosis was epithelioma.

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DISCUSSION

DR. JOHN F. BARNHILL, Indianapolis, Ind.:

Dr. Orton is to be congratulated for taking up a question in surgery which has been pretty well discarded, at least on this side of the ocean. It has been presumed that the question was settled in its disfavor. This new technic, or comparatively new plan of operation, which the Doctor follows, must have merit, since otherwise he would not be able to report such extraordinary results.

The operation, as I see it, goes a bit further than the block dissection which we had formerly done in these cases. The procedure is difficult

for the reason shown in the pictures: There are no normal landmarks to follow. When landmarks are present, when fascia, muscles and other structures are not matted together by infiltration, it is perfectly easy, after making the preliminary incision, to do a great deal of the dissection with one's finger; but in instances such as Dr. Orton has shown no such technic is possible and block dissection is necessary.

Some years ago I did a number of these operations, hoping that much could be done by following the procedure. In some of these patients almost all structures on the affected side of the neck were infiltrated, including the carotid sheath and its contents. In several I even went so far as to take away the carotid sheath, including, of course, the pneumogastric nerve. In such instances I feared at first that this would have serious effect on the heart and respiration, but this was not the case. These patients recovered rapidly, even after as complete dissection as Dr. Orton has shown; but, unfortunately, the recovery was not permanent.

The question in cancer always is, even though not so extensive as shown by Dr. Orton, has metastasis beyond the possible reach of surgery already taken place? In some of my cases, almost before the wound had thoroughly healed, evidences of metastasis were found in other parts of the body, and since none of the patients lived beyond a year and a half, we have lately hesitated to do this operation. The technic provides a beautiful piece of surgical neck artistry, and Dr. Orton has presented it in such a way as to again entice me into trying the plan he has advocated and so well illustrated, although I still believe the odds are very much against success. The lymphatic supply of this region is denser than that of almost any other part of the body. It is very difficult, therefore, to limit the early spread of the disease. However, with the newer diathermic instruments which the doctor mentioned using, one wonders if it is not possible to seal off the lymph vessels better than was formerly done, and thus give notable advantages over former methods.

A large amount of tissue always must be removed, and in some a fistula follows, through which food and liquids leak. This misfortune is, of course, annoying to the patient and troublesome to the physician. So, altogether, my own view is that not too much hope should be entertained concerning lasting benefits or certain comforts to the patient from the operation described.

DR. WILLIAM V. MULLIN, Cleveland, Ohio:

I should like to ask Dr. Orton if he ever considers radiation in any form immediately before or after operation. I presume, also, that he

makes every effort to ascertain whether metastasis has taken place. We find it very difficult, for example, to determine the presence of a metastatic growth in the chest from an x-ray taken at an early stage. In one of our cases of laryngeal cancer it was impossible to decide, although there appeared to be some indefinite markings in the roentgenogram of the chest. Laryngectomy was performed, but the patient died, and postmortem examination revealed a definite metastasis in the lung.

A later case of extensive malignancy of the larynx might have offered a satisfactory result from laryngectomy in the absence of metastasis. Comparison of this patient's roentgenograms with those taken in the earlier case, however, revealed similar markings in the chest, and we, therefore, advised against operation.

Doubtless this is an individual problem, since some patients are willing to grasp at anything that will offer a respite even if only for a year or two. The question for the physician to decide is whether or not he is justified in subjecting them to such a procedure.

DR. HARRIS P. MOSHER, Boston, Mass.:

This is a very splendidly prepared and presented paper. I have been one of the doubters about the operability of cases of this type and, to be perfectly frank, I have sidestepped them. This operation to me is a very welcome one indeed. It completes the systematic surgery of the side of the neck. Up to now, in my mind, all I have had has been the approach after a block dissection, getting in and getting out as best I could, but here comes along an operation which is in step, systematic, and gives a very splendid exposure. From the anatomical and surgical standpoint it completes, in my mind, the surgery of the side of the neck.

I first heard of Mr. Trotter's work through Dr. Beck, and I got here ahead of Dr. Beck. Dr. Beck, I imagine, is coming next. Then comes along Dr. Orton, and we see the procedure on the cadaver and we talk over his results. So if the case is operable, and if there are no suggestions of metastasis, it seems to me that here you have a perfectly definite, beautiful anatomical procedure to carry out.

To speak on one or two more or less detached points: I am very much in favor of a two-stage operation. There were two deaths promptly following operation in this series, of septic pneumonia. I will frankly say that a two-stage operation, in my opinion, might and probably would have avoided those two deaths from septic pneumonia.

About diathermy: At the Eye and Ear Infirmary we have had

1 or 2 cases. Those were done by the two-stage method, and in connection with diathermy. I have very great hopes for diathermy with these cases.

Now the skin-flap: the skin-flaps are very pretty in diagrams, but they are not always as easy to carry out. In one of the cases at the Eye and Ear Infirmary, not as extensive as these, a procedure was used which I think will help out and will avoid the necessity of a skin-flap in many cases. That is to use a paraffin plug in the esophagus, with a feeding tube carried through that and not attempt to turn in a skin-flap, letting the epidermization come in from the neck. I feel strongly that that will be a help in these cases which are not of the most extreme type. The case that I have in mind healed very beautifully with the paraffin plug in with the feeding tube through it, put in right after the operation.

Now about the casts: I have lugged around casts all my life. I have had the usual disappointment that about one man out of ten would look at them. I had the very pleasant experience in London, however, of having just the reverse. I carried some casts there, and I never had as many men look casts over and ask about them as I had there, and I hope that with these casts here on the table, at the end of the discussion you will go up and look at them. I have some pride in those because I had a little bit to do with them. Dr. Orton came to town a week before my postgraduate course and stayed there for part of the course. We got him started on the casts and then he finished them up during the first week of the course. We had a very nice time together. He proved a very good workman.

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

As Dr. Barnhill said, I too, have had a fling with the block dissection in these extensive malignant diseases.

A number of years ago I had the temerity to present before one of the largest societies the work that I had done in the Cook County Hospital for a number of years with this type of dissection, and at times I was taken to task for doing such a horrible thing. The answer, and consensus of opinion, was to let people die in peace, because most of the cases that I performed block dissection on either died from mediastinitis, embolic processes in the lung or recurrences. Consequently, I stopped doing this extensive work until it was my privilege and pleasure to visit in London three years ago, when I saw the work of Mr. Trotter, who showed me every point of the work. I had the good fortune of being there at the same time as Dr. Gordon New, of

Rochester, Minnesota, and, therefore, we had the opportunity to discuss the subject thoroughly.

Having seen this work first hand, I wish to say that it is not the fault of the operator if the results are not good, but it is the fault of the occasional operator in this work. If any of you have the opportunity of doing as many cases as Mr. Trotter, you will perfect that technic and the results will be very much better. We, in this country, could not, even at the largest institutions, have the opportunities this gentleman has in coöperation. There is only one other place where I have seen such splendid coöperation in cancer work and that was in Stockholm with Dr. Gunnar Holmgren and his associates, who get all the cases of carcinoma of the upper jaw sent to them from Norway, Sweden and the upper parts of Europe, and, therefore, can develop the technic to the perfection that they do. It is due to the large material that these men have that they can develop the technic, and we, who have the occasional opportunity, must not be disappointed when we have these losses or less brilliant results.

I am tremendously pleased that Dr. Orton brought this matter to our attention. When I first returned from Europe I received a consultant appointment on the staff of the Veterans' Bureau Hospital, the Hines Hospital, where there are all types of these cancer cases and thus had the pleasure to try out this extensive operation. I say pleasure, because the people are most grateful, especially if you do not hurt them, to have a chance for their life, for they know that without it they must die, and they do have a great deal of pain, etc., before death. The results that I have thus far obtained have been good. I am not citing any statistics, we certainly must not do that, as it is too early, but the following points stand out in my mind:

I have been able to operate most of the cases either by rectal or spinal anesthesia. I do not mean making the injection at the upper part of the spine, that is dangerous, but injecting low, say at the dorsal region and waiting three-quarters of an hour before you begin to operate on the neck. That is one thing that has been very gratifying in these cases, namely, they may be operated under either of these two methods of anesthesia and great danger is avoided from pneumonic complications.

The second point is that I have been removing the carotid arteries and the jugular veins in most of the cases because they were usually involved, and when they were not I was afraid to leave them for fear of causing embolic processes. In one case, where I left the common carotid artery completely dissected, I had an arteritis develop under

my own eye; the artery swelled out and produced an aneurysm, necessitating ligation.

In closing, I would like Dr. Orton to speak of the ligations of the common carotid, as to the number of cases of hemiplegia that followed from the sudden ligation. I now compress the artery and do not ligate it at once—a gradual compression by means of a Neff clamp. It takes forty-eight hours before the ligation of the artery is completed.

Another thing that Dr. Orton touched upon is the use of radiation. I think there are a good many cases of recurrences from covering up these large surgical defects where the cancer was removed too early. I am sure the cases I saw of Mr. Trotter, as well as Mr. Negus, are covered with skin much later. These large dissections are left open for observation for recurrence and radiation, both x-ray and radium, and subsequently covering these parts by large pediculated plastic flaps, such as taken from the back of the neck and bringing them forward.

One of the cases that Dr. Orton reported, of the resection of the lower jaw, is considered as big work. I have had one case where I had to resect one-half of the lower jaw in completing the operation and the end-result was very satisfactory, especially from the physiological point of view. However, it is work for the younger men. It tires one out to do an operation like that and I will be very glad to see the younger men take it up. It is a life-saving proposition. It certainly was worth while to come here and hear that paper.

DR. MERVIN C. MYERSON, Brooklyn, N. Y.:

I cannot speak from experience. I just did a case two weeks ago and the patient died thirty-six hours later, no doubt of septic pneumonia; but I can say from that little experience, that one must not only be fearless and have a good surgical and anatomical knowledge but have a strong back, as Dr. Beck intimated.

I did not intend to rise to speak, but Dr. Beck's mention of spinal anesthesia for work in the upper parts of the body tempted me to rise and give this information, which, perhaps, he is not acquainted with: This type of spinal anesthesia originated in Brooklyn, and those who are going to follow this type of spinal anesthesia should know that at the King's County Hospital the medical examiner had 2 cases to examine postmortem where some of the enterprising gentlemen who undertook this type of spinal anesthesia had used spinal anesthesia for tonsillectomy, so that this type of spinal anesthesia is not without its danger.

I know that most of the surgical men around Greater New York are very cautious about undertaking this form of spinal anesthesia.

DR. ORTON (in closing):

Dr. Barnhill asked about diathermy and the fistula following. I think in 4 of these cases we used diathermy. We had a fistula following in the man where we resected the posterior part of his tongue and the lateral wall of his pharynx, and this fistula remained in there for three weeks, but it gradually closed in. We did not have to do any further plastic.

This procedure gives an exposure, and you must have the exposure before you attempt to remove a mass, know just how far you can get around it, and then when you visualized this mass, go completely around it. We take out everything we have to.

Dr. Mullin asked about radiation. I did not cite all my cases. They are in here and will be published.

The preoperative preparation as to metastasis: All these cases have been examined as to Wassermanns, sputums, blood chemistry, x-rays, and every complete medical examination beforehand.

I quite agree with Dr. Mosher and his two-stage. These operations have been extremely interesting to me for the simple reason that in some of them we get a metastasis on the opposite side of the neck. Instead of doing a block dissection on one side, I know in the future we will do a block dissection on both sides. To do that it would make the operative procedure a little bit too long for any one man to stand. I will unquestionably do it in two stages.

I am very fond of diathermy and will be very glad to try the paraffin plug the first opportunity I have.

I like rectal anesthesia, although it has its danger. One case I cite here, in which we did a transmandibular pharyngotomy, had a recurrence on the jaw about four or five months later, just a small thing, and we were going to give him another rectal anesthesia for his diathermy, but during the anesthesia he suddenly stopped breathing before anything was done at all.

DR. SHURLY: How about radiation after operation?

DR. ORTON: That has been done.

DR. BARNHILL: You said you had no hemiplegia. How many ligations did you make?

DR. ORTON: The external was tied off in three, I think.

THE DISPLACEMENT METHOD IN SINUS DIAGNOSIS AND TREATMENT: ITS ADVANTAGES AND LIMITATIONS*

ARTHUR W. PROETZ, M.D.

FOUR years ago I presented to this Association the preliminary report of a method of sinus diagnosis and treatment to which the name "displacement" was given. It is essentially a means of introducing fluids into sinuses without the trauma of instrumentation. The satisfactory results which have attended its use prompt this complete presentation of its advantages and its limitations as observed by numerous clinicians. It has been the source of much information regarding the physiology of sinuses and has uncovered some significant phases in their pathology.

This paper must confine itself largely to the statement of conclusions, both for economy of time and because accounts of the experiments and observations upon which these conclusions are based have been recorded in various journals. The method consists in the introduction of fluids into the sinuses either for treatment or for diagnosis and has not been changed since the first presentation. The use of radiopaque oils has been supplemented by a special and very definite technique of radiography and interpretation which is presented in detail.

THE DISPLACEMENT METHOD.¹ "1. The patient is placed in the supine position, with his head projecting beyond the top of the treatment chair or table. The occipito-atloid joint is extended until the tip of the chin and the external auditory meatuses are in the same vertical plane. This renders the sphenoid sinus, with its ostium upturned, the most dependent structure in the nasal chamber. A V-shaped pocket thus occurs at the junction of the face of the sphenoid with the cribriform plate of the ethmoid.

"2. The fluid to be introduced is now allowed to flow into the

* From the Department of Otolaryngology, Washington University School of Medicine, St. Louis.

¹ Quoted from Proetz, A. W.: Displacement Irrigation of Nasal Sinuses, Tr. Am. Laryngol. Assn., 1926, p. 252.

nostrils from a syringe and comes to rest in this pocket, submerging the ostia of the sinuses. It does not enter the sinuses, as their ostia are of insufficient size to permit the escape of the contained air.

"3. Gentle suction (not more than 180 mm.) is now applied intermittently to one nostril, the other being closed, the palate and tongue being held in the "K" position to seal the pharynx. Any of the ordinary vacuum appliances will suit, provided that the degree of tension can be controlled. When the suction is first applied, a bubble of air escapes from the sinus through its upturned ostium. On releasing the suction this air is replaced by a drop of the overlying fluid. The process is repeated until the sinus is full; about a dozen alternations usually suffice to accomplish this.

"4. The patient is returned to the erect position, and the fluid remains *in situ* for an indefinite period (eight hours to several days)."

The type of syringes and suction apparatus used is of no consequence provided that a maximum of 180 mm. vacuum can be controlled. For convenience two types of special syringes have been designed.

TREATMENT. Any fluid commonly employed for treatment of the nasal chambers may be introduced into the sinuses but by far the best results have been obtained with the use of a simple, mild, stable astringent, namely 0.5 per cent ephedrine sulphate in normal saline solution.

One avoids the displacement method in early acute nasal infections, partly because of the possibility of spreading infection, although no instances of this accident have been reported, but chiefly because suction increases the hyperemia, adds to the patient's discomfort and further impedes drainage. As soon as the free draining, purulent stage is reached, the process may be materially shortened by irrigating every second day.

The method finds its chief function in those cases which have passed the acute stage and persist through a long and stubborn subacute one. This is especially true of pansinusitis which resists treatment chiefly because of the inaccessibility of the trouble. The annoying drainage of chronic sinusitis often yields to it in a remarkably short time (five to ten irrigations).

Eye disorders including retrobulbar neuritis, retinitis and uveitis arising from sinus infections have yielded to this treatment, which has been resorted to only after sinus drainage has been demonstrated by radiopaque injection to be good. I am aware that cases of this nature may show a pronounced tendency to recover spontaneously but the irrigation has unquestionably played an important part in the cases observed.

It is effective in the copiously draining sinuses of children as young as two years of age. The method of introduction in children does not vary from the routine employed in adults except that it is well to gag the mouth open to prevent the child's blowing the liquid from his nose before the vacuum can be applied to pass it into the sinuses.

The salient points in the rationale of this treatment are: (1) That the solution rests in the sinuses for a prolonged period where it can cope effectively with the retained exudates which it dilutes and (2) that by gradual drainage, the astringents employed keep the ostia free over a prolonged period of time. In diseased sinuses which are badly drained and in which the noxious infective material acts without intermission for days and weeks, the actual time during which treatment solutions can remain in contact with the membrane must be of paramount importance in the success of any treatment.

DIAGNOSIS. For roentgen-ray diagnosis, opaque oils are introduced. My own experience is limited to lipiodol, brominol, campiodol, lipo-iodine and iodipin. At present my choice lies between lipiodol diluted 50 per cent with some inert oil and brominol (light), undiluted, with a preference for the latter owing to its low natural viscosity (without heating) and to its pleasant ethereal odor which renders it unobjectionable to patients during the seventy-two-hour period of observation.

Certain fundamentals must be considered before any roentgen-ray diagnosis with radiopaques is attempted. Obvious as it may seem, this statement is prompted by the frequency with which false deductions appear in the current literature of the subject—deductions based upon radiographs accompanying them which have been made without regard to the simplest laws of physics.

To begin with, no sinus can be filled by any method beyond its

normal ostium. This being the case, the only sinus completely filled is one whose ostium is at the exact top at the moment of filling. This occurrence must be exceedingly rare; and it follows therefore, that for diagnostic purposes all sinuses should be regarded as less than completely filled.

Air bubbles in the oil occupying the central portion of the top of the cavity, if roentgen-rayed vertically from above will cast an irregular shadow on the film, which is more than likely to be misinterpreted. For this reason, all fluid radiopaques should be rayed with the beam horizontal and the patient vertical. In this position gravity keeps the oil in contact with the membrane and discrepancies in contour cannot be artifacts. This position has the added advantage of sharply indicating the oil level in each cell. In the case of overlapping cells, hence overlapping shadows, these oil levels distinguish individual cells in the group. This becomes especially helpful in the case of a posterior ethmoid cell overlapping a sphenoid in which distinction anatomically is often very difficult, but very important.

By the displacement method the most satisfactory filling occurs in the sphenoids and the ethmoids. The antra usually admit enough oil for the purpose of diagnosis. It is not recommended for frontal-sinus investigations as the oil admitted is rarely sufficient for diagnostic purposes. The degree of filling in the antrum depends upon the distance between the ostium and the top of the cell, since in the inverted position this is the portion capable of filling.¹

The simple outlining of sinuses by means of radiopaques which in most clinics is regarded as their sole purpose is in our experience the very least of their functions. Fluid radiopaques having been introduced into the sinuses by displacement, since there is no irritation involved in the introduction, can be observed over a period of time and regarded as a fairly accurate indication of physiological drainage.

Four factors are necessary for successful filling: (1) The proper position of the head; (2) fluid covering the ostium; (3) intermittent

¹ The excursion of fluid in and out of the antral ostium during displacement is often sufficient to dislodge an entire antrum full of mucopus into the nose and in not a few cases may be employed instead of the usual irrigation for this purpose.

vacuum; and (4) a patent ostium. The first three are readily controlled. If then a certain cell fails to fill, one may assume that

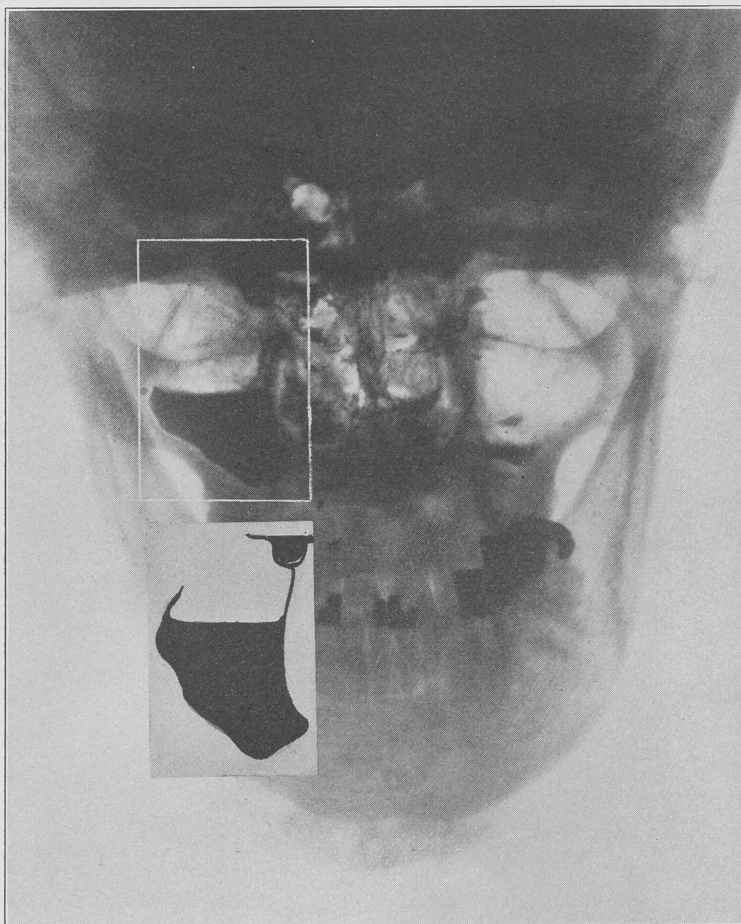


FIG. 1.—Case I. General radiopaque filling in an allergic individual, in an interval between asthmatic attacks.

the ostium obstructs the entrance of the fluid. Such failure to fill at all indicates some gross obstruction incapable of being overcome by the 180 mm. of vacuum.

Having once been filled, the emptying time is a much more delicate indication of physiological drainage. The normal emptying time for all sinuses is ninety-six hours but as only a trace remains after seventy-two hours, this period suffices for routine

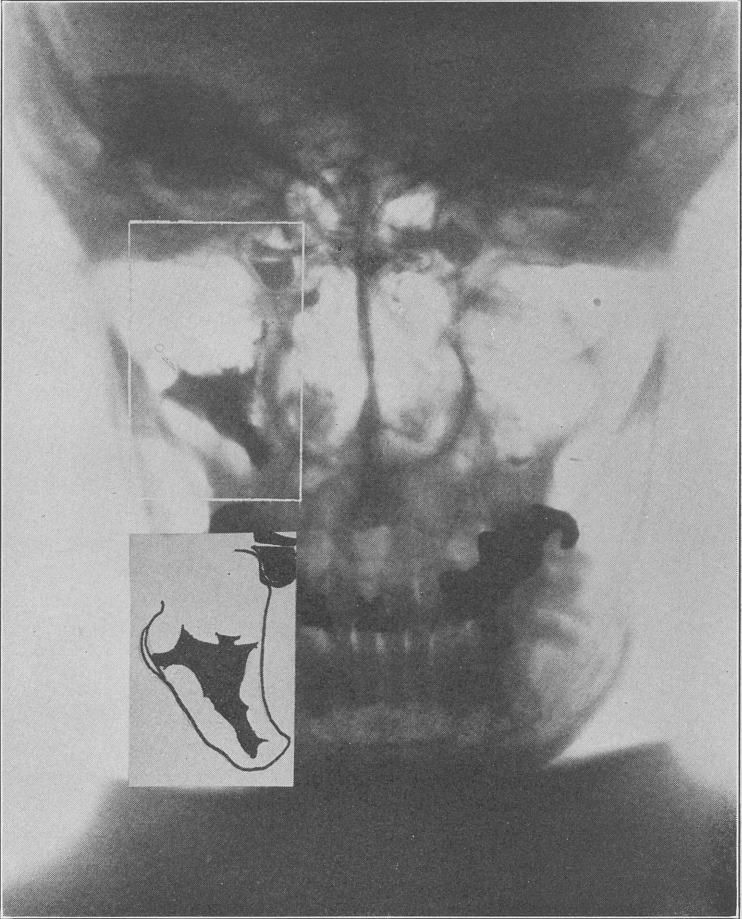


FIG. 2.—Case I. The same patient as in Fig. 1. Film exposed seventy-two hours later, immediately following a severe asthmatic attack precipitated by contact with goose feathers. Note the extreme swelling of the antral mucosa while that of the sphenoid shown above remains normal.

observations. The retention is especially significant if it occurs in a single cell, while other cells empty at the normal rate.

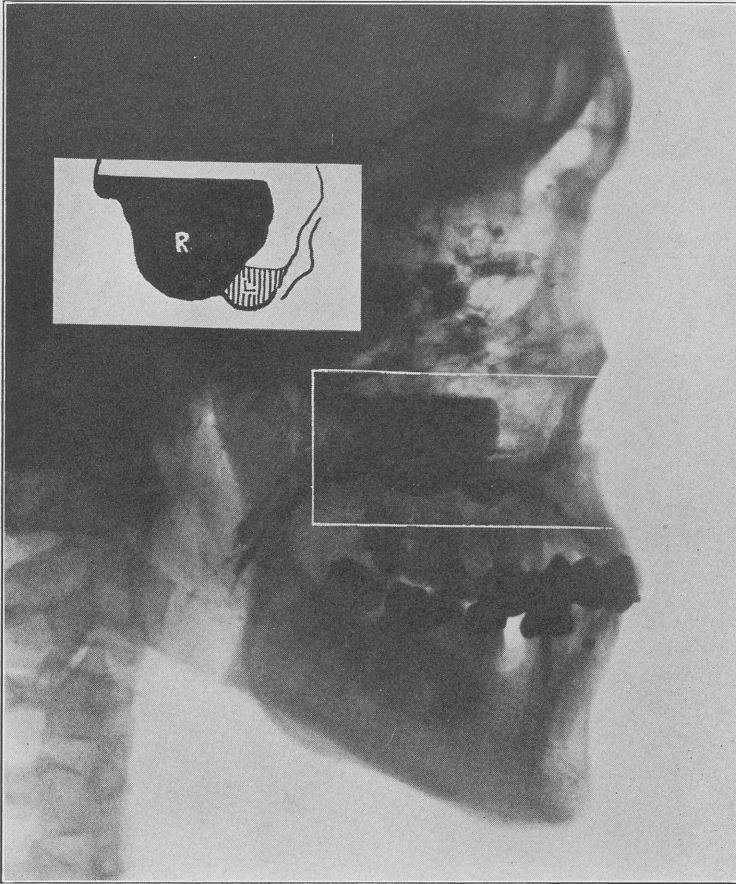


FIG. 3.—Case I. Lateral view of Fig. 1. Note normal contact lines in both maxillary sinuses.

RADIOGRAPHY. The technique of radiography with radiopaques is extremely simple but all important. Beside the partial filling and the horizontal beam already referred to, accurate angulation is highly desirable. By this means, seventy-two-hour films can

be accurately compared to initial films. Furthermore, when angulation is always the same, the observer readily accustoms

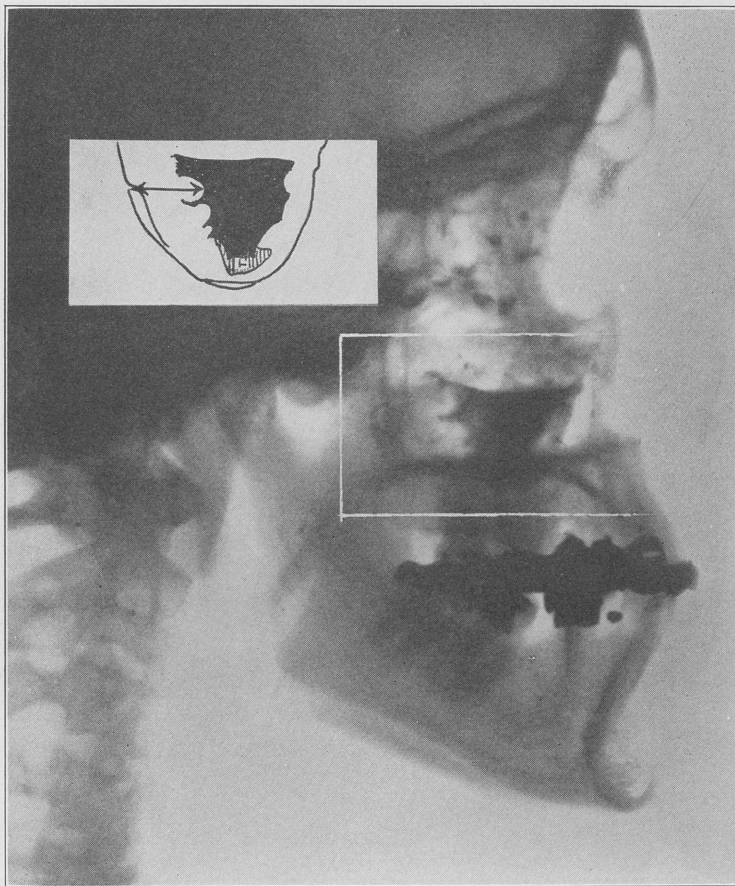


FIG. 4.—Case I. Lateral view of Fig. 2. Note thickened membrane on the right. The left is practically unchanged.

himself to the normal picture and quickly detects small deviations from it. The shadows of radiopaques are so sharply defined that overlapping of bony parts becomes less important and the complicated angulations resorted to, to prevent them, are unnecessary,

although they may be retained with the exception that the film becomes vertical and the beam horizontal. Film-head-tube relations should then remain the same. Our present practice is to make: (1) A simple postero-anterior view, the horizontal beam passing through the so-called "base line" (external auditory canal to nasofrontal suture), the film before the face being perpendicular



FIG. 5.—Case II. Both maxillaries containing radiopaques before allergic attack.

to this line; (2) a lateral view made by retaining the base line horizontal and revolving the head in a vertical axis through 90 degrees; and (3) a "ground plan" made by tilting back the head until the vertex rests against the vertical film and the base line itself is vertical. The horizontal beam is now projected from beneath the chin so that the teeth overlap the frontal sinuses and

all other sinuses are clearly shown in the "ground plan." In other words, our representations correspond to an architect's working drawing, front elevation, side elevation, ground plan, each at 90 degrees from the other. If the proper head positions are maintained in making these films, they can actually be laid upon a view-box in such a way that accurate projection lines can be carried from one to the other.¹

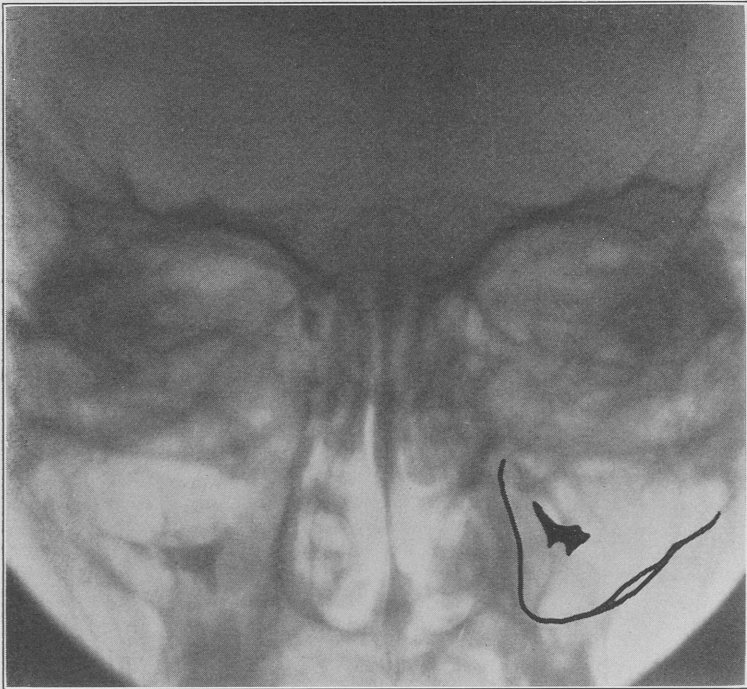


FIG. 6.—Case II. The same patient three days later, following an acute allergic attack.

Dr. Edwin C. Ernst, through whose painstaking coöperation the radiography of displacement has been developed, supplies the following data:

¹ Allen, Kenneth D. A.: Sinus Radiography for the Proetz Method of Sinus Study, *Ann. Otol., Rhinol. and Laryngol.*, December, 1928, pp. 1212-1222.

Tube distance, 31 inches.

Current, 20 milliampères.

Potential, 68 to 78 kilovolts for postero-anterior positions; 58 kilovolts for lateral positions.

Exposure, seven to eight seconds for postero-anterior positions; four seconds for lateral positions.

Immobilization of the head is imperative.

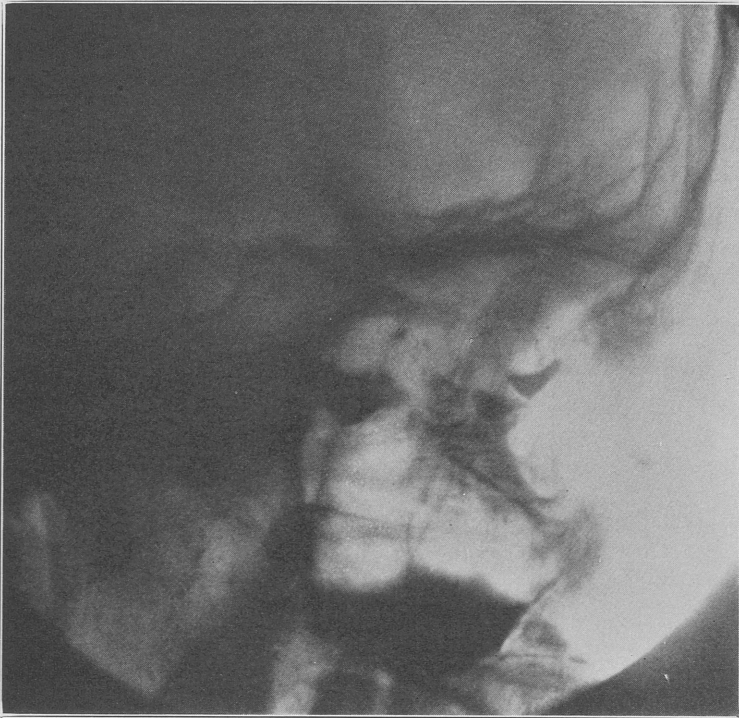


FIG. 7.—Case II. Lateral view of Fig. 5.

ALLERGY. As a general rule, active allergy in the nose completely prevents filling by displacement. Apparently the flap-valve action of the edematous tissues effectively obstructs the interchange of air and fluid. This obstruction is so pronounced

and so constant that one has come to regard complete failure to fill as a strong indication of allergy.

Our routine serial radiograms have recently led to two findings which we regard as significant. The first is that, under the influence of a sudden allergic attack, the sinus membrane may swell from

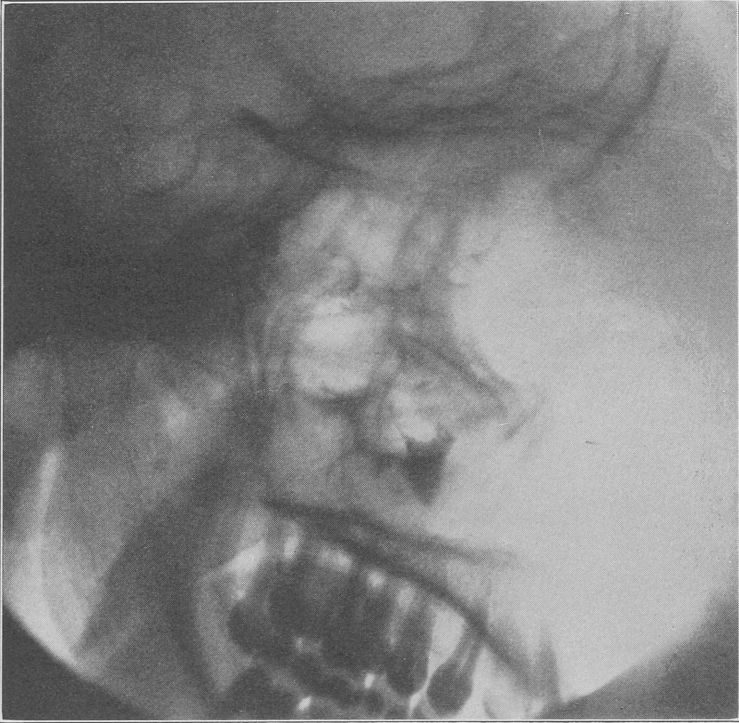


FIG. 8.—Case II. Lateral view of Fig 6, showing the typical rounded contours of the acute edema of allergy.

its normal thickness to practical obliteration of the sinus cavity within a few hours; and that it may subside from this state to its normal thickness in the same short time. This reaction may be confined to a single cell while the others remain unaffected. This was demonstrated in several patients exposed to their allergens between the first and the second observation.

The second is that such labile membranes can and should be distinguished from true hyperplastic membranes in allergic individuals when any surgical intervention is contemplated. Shadows cast by the two types of membrane are not likely to be confused. The transitory allergic swelling is very pronounced and is characterized by the convex outlines commonly associated with polyps. It varies with the allergic state of the patient. The true hyperplasias are likely to be moderate; the outlines are smooth and follow the contours of the sinus. They are more dense than the first type and are not affected by asthmatic or other allergic crises. These impressions have been confirmed histologically by Hansel.

Individuals have been encountered who are sensitive to olive oil and its derivatives, oil of sesame and poppy seed oil which enter into the composition of some of the radiopaques. The reactions have ordinarily been confined to the sinus membrane but in the case of the poppy-seed-allergic individual severe urticaria of hands and feet occurred.

* * * * *

The following articles contain the clinical and experimental data upon which this report is based:

1. Proetz, A. W.: Displacement Irrigation, a New Method of Diagnosis and Treatment of Sinuses, Tr. Washington University Med. Soc., December 14, 1925. *Journal Missouri State Med. Soc.*, February, 1926, p. 77.
2. ——— Displacement of Nasal Sinuses, *Arch. Otolaryngol.*, 1926, iv, 1.
3. ——— A New Procedure in Sinus Diagnosis, Tr. Am. Laryngol. Assn., June, 1926, pp. 252-257.
4. ——— A Special Syringe for Displacement Irrigation of the Nasal Sinuses, *Arch. Otolaryngol.*, 1926, iv, 424.
5. ——— Physics of Sinus Drainage, *Ann. Otol., Rhinol. and Laryngol.*, 1927, xxxvi, 23.
6. ——— Iodized Oil in Sinus Diagnosis, *Missouri State Med. Jour.*, 1927, xxiv, 229.
7. ——— Further Data on the Displacement Method in Sinuses, *Ann. Otol., Rhinol. and Laryngol.*, 1927, xxxvi, 297.
8. ——— Visualization of the Sphenoid with Studies of the Exposed Pterygoid Canal, Tr. Washington University Med. Soc., October 10, 1927.
9. ——— Visualization of Sinus Drainage, *Ann. Otol., Rhinol. and Laryngol.*, 1927, xxxvi, 978.
10. ——— Sources of Error in Sinus Radiography with Fluid Contrast Media, *Ann. Otol., Rhinol. and Laryngol.*, 1928, xxxvii, 806.
11. ——— Physics and Physiology of Sinuses, *Ann. Otol., Rhinol. and Laryngol.*, 1929, xxxviii, 964.
12. ——— Sudden Allergic Reactions Localized in the Antrum, *Ann. Otol., Rhinol. and Laryngol.*, 1930, xxxix, 87.

13. Proetz, A. W.: Allergic Reaction in the Maxillary Sinus, *Jour. Allergy*, May, 1930, vol. i.

14. Proetz, A. W. and Ernst, Edwin C.: Sinus Mapping by the Displacement Method, *Radiology*, June, 1927, p. 502.

15. Ernst, Edwin C. and Proetz, A. W.: Contrast Media in Sinus Radiography; Precision Methods and Apparatus, Tr. Second Internat. Radiologic Congress, Stockholm, 1928.

DISCUSSION

DR. FRANK R. SPENCER, Boulder, Colo.:

Dr. Seydell has just asked me to mention a patient of one of the men in Denver, Dr. C. Howard Darrow, who is on the faculty at the University of Colorado.

He had a man under his care who had been kicked severely in the forehead. He developed a badly infected frontal sinus and subsequently a frontal lobe abscess. During the course of treatment Dr. Darrow used lipiodol for injection into the frontal sinus. It went back into the frontal lobe abscess, into the ventricles and the spinal canal. Shortly after this he developed meningitis and it was so diagnosed by one of our best neurologists, Dr. C. S. Bluemel. The roentgenograms showed the lipiodol in the ventricles and in the spinal canal. It is very interesting to see how far the lipiodol travels and how treacherous a thing it is to inject lipiodol under pressure unless you know where it is going. The most interesting feature of this case is that the patient made a complete recovery, or apparently a complete recovery, after rather a stormy attack of meningitis. He was presented by Dr. Darrow before the Mid-western Section Meeting of the Triological Society in Denver last January. He is not entirely well, however, because he is still having some discharge from the frontal sinus. He may have further trouble with the frontal lobe abscess, although apparently this has healed.

DR. HENRY M. GOODYEAR, Cincinnati, Ohio:

I think Dr. Proetz always demonstrates the method and the use of opaque oils very clearly with his slides and diagrams.

Late in 1925 I started using 40 per cent lipiodol in making the diagnosis of sinus conditions, especially of the antra, and the method that was used is still followed. The antrum is filled when possible through the normal opening with a double-curved cannula. While it is being filled, the number of cubic centimeters that go in from the syringe are

measured and counted so that you can often tell something about the sinus by the number of cubic centimeters that enter the antrum. That is, it may hold only 2 or 3 cc., so that you are pretty sure that there is something in the antrum that is taking the space that would be occupied by the oil. If the sinus holds from 15, even up to 30 cc., as occurred in one of my recent cases, then you are pretty sure that the antrum must be a fairly normal one to hold that much oil.

Dr. Proetz said that you can fill a sinus only as high as its opening, that you may have a projecting partition from the upper wall forming a pocket which will not fill in the top of the antrum. This is true, but it is very rare that you have this condition present to begin with, and if it does not fill by the direct method then of course by the insufflation or displacement method, with a small amount of fluid on the floor, the chances are you not going to cast any opaque shadow in this particular recess. But if you fill completely and immediately pack the middle meatus with cotton, then very often the oily film extends above the ostium because you get your cotton in before the viscous oil runs out, especially if the opening is small and you are using lipiodol in oil.

If you take the patient in the sitting posture, there may be a space up high that is not filled, but if you take that same patient in the recumbent position, usually you get an outline of the whole upper area of the antrum.

As far as the dilution is concerned, Dr. Proetz said he was using 50 per cent solution of lipiodol in oil. As far as my observation is concerned as to dilution, I find that if one can dilute lipiodol two to one with ordinary liquid albolene, it gives a better shadow than full strength brominol.

Another thing about using brominol is that it costs \$1.50 for 25 cc. and you use it full strength. Lipiodol costs \$1.75 for 20 cc. and you dilute it two-thirds with oil, and of course it cuts down the expense of the injection. Some sinuses will hold more than 20 cc., and at that rate you would be using \$1.50 worth of oil on each side.

As to the filling of the frontal sinus, Dr. Proetz said very little. It is rather difficult to fill, but by placing the head in the knee-chest position and dropping the oil in the nose followed by suction to produce a vacuum, you can get brominol to run into the frontal sinus.

As far as using the displacement method in the sphenoid is concerned, all of our cases have been done by directly filling the sphenoid sinus through a cannula. Usually you can get into the normal opening, and if you can not, it is not usually difficult to puncture the anterior wall.

As far as the ethmoids are concerned, I think it requires a lot of work, and, of course, very careful work with exact angulation to get very much out of displacement diagnosis in ethmoid conditions. In the first place your patient has to come back in twenty-four hours, or seventy-two hours for more plates, which sometimes makes it rather expensive. If the ethmoid area is suspected, it is often about as easy to cocaine and press the middle turbinate over and examine directly.

Another point that I have noticed is that lipiodol will often stay in the antrum for even a couple of weeks. I would like to ask Dr. Proetz how long after injection he has observed oil in the sinuses?

I recall one patient who had an abscess following the extraction of a tooth. The abscess extended up along the cheek and into the temporal region. In order to try to find the fistula, I put some lipiodol in the opening up in the temporal region. Now, a year and a half after that oil was put in, I can still see a small tumor mass in the cheek, from which I was able to obtain oil many months after its injection.

On the strength of this I thought I would try it for some depressions that needed filling instead of using bone, to see what would happen. I thought it might encourage fibrous tissue and help to fill the depression. In one case, a postoperative frontal sinus that had quite a deformity and a wide thin scar closely adherent to the bone, I injected oil under the skin. After making several injections the scar tissue was loosened considerably from its bound-down position, with apparently some deposit of new fibrous tissue under the skin which decreased the deformity considerably.

DR. RALPH A. FENTON, Portland, Oregon:

I would like to ask Dr. Proetz if he has had, in addition to these allergic phenomena from various oils which I think some of us have noticed in using this very excellent method, an exacerbation of thyroid symptoms in susceptible people. We have happened by chance to see two persons in whom thyroid symptoms seemed to be speeded up by the iodine-containing oils. I think it is also worthy of note that Dr. Proetz is contributing distinctly to our literature by giving us definite data for our *x*-ray men and our *x*-ray technicians, which we have hitherto lacked.

DR. WILLIAM B. CHAMBERLIN, Cleveland, Ohio:

I would like to ask Dr. Proetz two questions. In the first place, he spoke of not using any astringent in the nose, such as ephedrine or

adrenalin, in order to secure a better dilatation of the opening. I would like to ask what the very definite objection to that is?

Second, he spoke of treatment of accessory sinus disease by this method, and in the treatment the only drug he mentioned was ephedrine. If such is the case, why should one resort to this displacement method? Why could not he accomplish quite as much by simply packing the nose with adrenalin, following it with ephedrine alone in the first place? Evidently the ephedrine has no other efficacy than in promoting drainage.

DR. FREDERICK M. LAW, New York City:

We have used lipiodol at the Manhattan with a great deal of success. Personally we find the injection method more satisfactory. I feel that the best contribution Dr. Proetz has made is in the allergic type of case. I think that is very interesting. The demonstration of a clear area remaining after the swelling of the mucosa is a distinct contribution.

For outlining the sinus, irrespective of which one, if you gentlemen will spend the time with your *x*-ray men and learn to read plates or films stereoscopically, spending the time that he spends, you will learn to see these shadows almost as clearly as though you had lipiodol in the sinus. Understand, I say stereoscopically. Unfortunately everyone can not see stereoscopically, but those men at the Manhattan who have used the stereoscope (and Dr. Faulkner will bear me out in this) can appreciate the distinct shadow that a markedly thickened membrane will make. This is particularly the case in the ethmoids.

Dr. Eggston's contribution today describing the types of change occurring in the periosteum in the bone is distinctly and clearly shown in every perfectly made roentgenogram of the sinus. If you remember, two years ago Dr. Fowler made a contribution describing the changes occurring in progressive deafness. At that time we showed the *x*-rays which demonstrated the pathology brought out today by Dr. Eggston.

If you will go over your films stereoscopically, picking first a normal case to learn the type and appearance of the ethmoidal structure, then take your pathological cases, you will discover marked variation in the appearance of the ethmoid cells. First you will see the fuzz and semi-transparency of a thickened bone; the other type is the thin, fine, clean cut, markedly condensed type. There may be complete absorption of all or some of the ethmoidal septa. These are the appearances that are obscured by your lipiodol injection. This change occurs in the walls

of all the sinuses, and can be made out if you properly, carefully study your *x-ray*. Do not put them up in the box and stand off five feet and say, "Oh, there is some opacity in that sinus." That is not what the *x-ray* is for. Learn to study your films, learn the technic of the *x-ray* man, and together you will make a diagnosis in the majority of cases. Study your plates carefully.

DR. SWAIN: If we could all have Dr. Law's films to study it would be very easy.

CHAIRMAN CHAMBERLIN: I would like to emphasize a point which Dr. Law made there in regard to the value of stereoscopic plates. I think this was emphasized to me during the past winter by some very beautiful plates which Dr. Lee Hurd showed, wherein one could see very distinctly the outline in the antra of the thickened mucosa.

DR. LAW: I want to say this, that it is necessary to have perfect technic, not technic that will produce a pretty picture. Unfortunately, the roentgenographers have no standard when it comes to sinus work. A patient will bring a film to you made in Timbuctoo. You bring it to the *x-ray* man or perhaps you look at it yourself for an interpretation. You are accustomed to reading a certain type of plate. The chances are you will not be able to read that plate. The fact that the same technic used in Timbuctoo does not correspond to what you are accustomed to may cause you to make an error in your interpretation. It is quite possible to take a sinus case showing a pathology in an antrum, which is the easiest thing to show, and with the same angle but with a difference in voltage or exposure, to make an entirely different looking antrum.

So if you yourselves talk to the *x-ray* men and get them to produce a film which shows contrast, not that dense dead black and a clean-cut white, but one in which the shadow of a normal antrum is just off the gray (if you get too much contrast you burn out the soft tissue of the mucous membrane), you will see that mucous membrane and you can pick out each individual ethmoid cell on either side. If one of them shows a thickened membrane and the others are clear, you can detect it. Insist upon a perfect technic: a film which shows detail, shows distinct, clearcut outlines of not too great a contrast. That is the secret of a good film.

CHAIRMAN CHAMBERLIN: I might add in corroboration of what Dr. Law has said that *x-ray* men are rather hard to talk to. Most of them occupy a very elevated plane as far as the clinician is concerned. These plates, Dr. Law, of which you speak, not only are

brought to you but are brought to us from some dentist or some occasional x-ray man in a country town and they refuse to have plates made again.

If there is no further discussion, Dr. Proetz will be asked to close.

DR. PROETZ (in closing):

Whenever I open a discussion on displacement, it sooner or later deteriorates into a general discussion of lipiodol, and they are two different things; so I am going to confine my remarks as much as possible to those things that do apply to displacement.

Dr. Law will be as surprised as anybody to hear that I do think that one can tell as much about a sinus without lipiodol in it as with lipiodol, if one studies them carefully, that is, as far as the topography is concerned.

Naturally we presuppose a good picture and we presuppose a picture made by a man with whom we are familiar and whose shadows we are able to interpret, but when I talk about lipiodol in connection with displacement, I have reference particularly to drainage time, and one can learn much about the physiology or pathology of the sinuses of an individual case from the drainage time that the mere topographical information derived from lipiodol does not reveal.

Everybody works along some line or other in trying to make his diagnosis and finally he develops a technic in which he is interested and which he develops to a much greater extent than anybody else. We probably get more out of displacement where I am because we have developed it farther. Whatever method *you* use, you get more out of than I do because I am not familiar with it, and I think no doubt this accounts for the preferences and dislikes for one method or another.

Dr. Chamberlin asked about the use of astringents. In treatment, I do use astringents because I feel that any method to which we can resort to get the fluid in is legitimate. When we are trying to make an observation of emptying time, however, if we pack that nose with adrenalin, we get an abnormal filling; that individual pathological cell, which otherwise under ordinary circumstances does not fill, will fill under astringents and the diagnostic point is lost. We do not want that. We want that cell to be just as it is, so that it won't fill and we can spot it.

Then again the reaction from the adrenalin will bring about more or less hyperemia, which will in turn interfere with the emptying-time of that cell, so we want to leave the nose in as nearly its average con-

dition as we can for diagnosis. For treatment, on the other hand, we certainly want to use every means at our command to open the sinus.

As to the help that we get from displacement over ordinary treatment of the nose, by displacement we get treatment solutions inside the sinus where the disease is. If the wind carries a lot of dust down the hall and fills up a lot of rooms with dust, we do not want to sweep the hall in order to clean the rooms. We have got to get inside the room, which we ordinarily do not do with sinus treatment. So when we put our solution in, and it stays there for twenty-four hours, we certainly get a more logical type of treatment from our solution than we do if we simply put it into the nose.

In answer to Dr. Fenton's question about the effect of lipiodol on the thyroid, I cannot tell him very much about that because shortly after we began this work we abandoned lipiodol and used brominol. I have never seen any thyroid effect in any case under my observation or of the people with whom I have been in contact.

As to these cases in which lipiodol was found in the cranial cavity and in the spinal canal, I must emphasize the fact that this was invariably an accident of injection and not of displacement. I do not see how it would be possible to introduce oil into the cranial cavity by displacement, as it contains no air to displace.

I hardly know what to say to Dr. Goodyear. I rather felt after he got through talking that I had better abandon displacement entirely and say no more about it; but I will go into details with him personally and not occupy too much of your time.

NONSUPPURATIVE ETHMOIDITIS

EDWIN MCGINNIS, A.B., M.D.

INFECTION of the nose and accessory sinuses has for many years been of the most intense interest to me. Perhaps my own personal need for help in that region of my anatomy was the first determining factor of my interest in that field of our specialty. In my routine examination of nurses, medical students, college students and the men coming up before the draft boards for the Army at the time of the World War, I made the observation that about 65 per cent of the individuals had normal noses. In a rhinological clinic or private practice about 10 per cent of the noses coming in for examination are normal. I also felt a curiosity to discover whether here, in the nose, might not be the cause of that prevalent illness that attacks the human; the common cold, which is spread and aggravated by modern crowded conditions at home and at work, and especially, perhaps, in such crowds as those of the movie theater, where, in the fall, spring and winter, there are always some coughers and sneezers.

Now the theory of focal infection as causation of general disease is a rather well-established fact. The good end-results of a complete tonsillectomy and adenectomy, as against a partial removal of these structures is also acknowledged. Apical tooth infection can be demonstrated by the roentgen-ray, and the presence or absence of pathogenic organisms demonstrated by bacterial examination. But my routine examinations proved the amazing fact that even though every vestige of tonsil and all the teeth had been removed, some patients still suffered the ill effects of infection. Dr. John F. Barnhill has very aptly given one possible explanation, "The general infection can act as an infecting agent, even though the primary focus has been eliminated."

Reasoning, however, from the nonnormal noses, and the persistence of infective trouble after the removal of the other possible foci of infection, it seemed logical to conclude that the general infection was not started by a tonsillitis, nor by a tooth, but the the nose or the sinuses were the seat of the original infection.

There is agreement among us as to the evidence of sinus infection in individuals when there is present in the nares a considerable amount of purulent discharge. There is also agreement when, on intranasal inspection, a stream of pus is exuding from the sinus openings. Roentgen-ray and transillumination are usually confirmatory. An interesting observation is that the patient's protective mechanism is very active when fighting an acute suppurative sinus infection. The patient's resistance, antibodies and lymphatic glands rise to the emergency, though, of course, in a percentage of cases, there is a blood-stream invasion so that general infection results. In the diagnosis of *these* cases we are in agreement, though we may disagree as to their treatment.

The type of case that has awakened my great interest is the type usually referred by a medical man for a general checkup. These patients have often made "the rounds." They are usually people with some gastric upset; joint, or headache cases; some of which have been diagnosed as sciatic or migraine cases, as having various eye lesions, etc. The history often shows these patients to have had sinus examinations and treatments. Roentgen-ray has been an unsuccessful aid to diagnosis. The most outstanding fact in their history is that they have frequent colds of unusually long duration. These patients also admit a postnasal discharge. Some of them have a deep-seated eyeache, a frontal headache, or occasionally, occipital headache. Most of them do not have to blow the nose much for cleansing purposes. Palpation of the cervical glands at the angle of the jaw discovers a palpable, tender gland.

Stereoscopic roentgen-ray of the sinuses, if done by Dr. Fred Law, would probably show thickening of the lining membranes of the cells. But the ordinary "garden variety" of roentgen-rays do not usually show much deviation from normal.

Intranasal examination, carefully done, will bring out some of the points emphasized by Skillern, in the symposium at the meeting of the American Academy last fall at Atlantic City. I always do a careful shrinking of the nasal mucosa, watching for any change in the color, for congestion of the surface of the mucosa of the middle turbinal, and I make a careful investigation of the middle meatus whenever possible. Very often it is possible to wipe out a thin greenish discharge from the middle meatus. This discharge

makes one suspicious of infection of the lining mucosa of the ethmoid cells by bacteria which are not pyogenic bacteria. Evidence of the correctness of this suspicion I have repeatedly obtained by laboratory investigation of cultures. These cultures prove the presence of hemolytic *Staphylococcus albus*, *aureus*, and the *Streptococcus viridans*. We occasionally find the hemolytic streptococcus in cells which show no pus.

Not long ago I was asked to examine a young woman, as part of a general checkup, and my only discovery that helped toward diagnosis was the history of a greenish postnasal discharge. She had lost weight, and a tentative diagnosis of exophthalmic goiter had been made. Examination revealed an almost normal nose. At the request of the attending man, Dr. Paul Rhoads, an associate of Dr. George Dick, I did an intranasal plastic operation opening the ethmoidal cells. Dr. Rhoads recovered the *Streptococcus viridans* from bits of the lining membrane of the anterior ethmoid cells. These were pathogenic to experimental animals, and the autopsy of one animal revealed an acute congestion of the suprarenals and a swollen hemorrhagic area in the duodenal mucosa. Small doses, injected over a period of time, would probably have produced a duodenal ulcer. The patient made an uneventful recovery from the operation, and has since gained in weight and strength.

If these nasal cases are recognized, then naturally their treatment becomes subject for discussion. If one goes over the literature, one finds there are various ideas as to the best treatment. Twenty years ago a long ocean voyage was the happy means of cure advised. Nowadays some of these patients seek the southwest for the sun's ultra-violet rays; some at home use various lamps. Drs. Dean and Daniels would have plenty of vitamin A in the diet. Dr. Stucky uses the aid of a breakfast food. Some treat these cases with intranasal packs.

I have deemed surgical aid the most effective. The thought came to me that it would be wise to make abnormal noses, where symptoms such as I have described above are present, as nearly normal as possible. This can be done by intranasal plastic surgery. The gratifying thing about it is that it works, and that the patients with the reconstructed internal noses live afterward very

much as do the people with the normal noses. That is, they have fewer intranasal upsets; fewer colds; infections clear up faster with less general upset; they gain in weight, and lose their general infective symptoms. Of course the question arises, "Why pick on the ethmoid?" The majority of the cases in my series had clear antrums, frontals and sphenoids, but if one or more of the paranasal cells are involved these are also operated upon.

DISCUSSION

DR. FRANK B. KISTNER, Portland, Oregon:

I have been interested for a long time in nonsuppurative sinusitis, particularly in its relation to systemic disease. As a consequence, I have had considerable curiosity as to the kinds of organisms to be found in these sinuses and where they are harbored in the tissues.

We have cultured washings from those sinuses that were accessible, as the sphenoid and the antrum; have made swab cultures from the various sinuses, ethmoid and sphenoid and antrum, at the time of operation and have ground and cultured the tissues removed at operation. Cultures of washings from sinuses gave a surprising number of perfectly sterile cultures—swab cultures gave a larger number of positive cultures, usually with the streptococcus predominating. Cultures of the tissues gave almost uniformly a growth of streptococci. I think possibly these two slides may tell us why we get such a variable result from washings and surface cultures and uniformly over 90 per cent positive cultures from the tissues.

We have stained for bacteria in the tissues of quite a number of cases of latent sinusitis. The bacteria were only rarely found on the surface; occasionally found in the loose subepithelial layer and as seen in these slides they are most numerous in the deeper layers near or on the periosteum.

DR. RALPH A. FENTON, Portland, Oregon:

I regret my unfamiliarity with the details of Dr. McGinnis' conservative operation, which should have been placed in a recent publication regarding these intranasal operations, and I can see much to recommend it; but I am wondering if such a conservative operation may not occasionally lead to the reclosure of these ethmoidal cells. He speaks of a gutter drain being cut down through them. I am wondering if,

in certain cases, where one or two cells are alone infected, as Skillern and Mosher have both pointed out, is frequently the case, whether other cells not previously involved might become infected by the lengthwise gutter drain of this type. The same thing obtains, of course, in Pratt's operation, where the work is carried out under the turbinate without very much exposure of the deeper field. At least that is the criticism which has been made of both operations.

DR. E. ROSS FAULKNER, New York City:

There is just one part of Dr. McGinnis' demonstration on which I want to speak, that is the partial ethmoidectomy. Perhaps I have had a more unfortunate experience than most of the men from the fact that probably 50 per cent of the ethmoid operations that I do are secondaries, and it is a very difficult operation to do after somebody has done a partial ethmoidectomy.

Years ago I used to attempt to do partial ethmoidectomies, but I do not believe I have done one for a good many years, and I do not ever expect to try to do another. If the ethmoid is sufficiently diseased to demand an operation, I think they had better all be cleaned out. I am inclined to think that a partial ethmoidectomy is almost in the same category as a partial mastoidectomy. I have done a great many secondaries in these cases that had had a partial ethmoidectomy. They have been done all over the country. They come back with eye symptoms or some obscure lesion, due to a focal infection that the partial ethmoidectomy has been more or less responsible for. When you open ethmoid cells, you leave some ragged edges there. I do not see how epithelium of any kind is going to grow over those ragged edges and make a closed wound afterward. I do not see how the cells that are not open proximal to the ones you have opened would not get enough traumatism from an operation like that to block them entirely. From my experience that is just about what has happened.

DR. JOHN F. BARNHILL, Indianapolis, Ind.:

I rise more to ask a question than otherwise. Everywhere one hears discussions on partial ethmoidectomy and complete ethmoidectomy. When complete ethmoidectomy is referred to, usually there seems to be gross, though, perhaps, unintentional exaggeration. Dr. Faulkner says he does a complete ethmoidectomy. One may rightly wish to know exactly what he means by that. Does he mean that he cleans out absolutely every cell and leaves the ethmoidal box a free and open space?

DR. FAULKNER: Yes; clean out the whole business.

DR. BARNHILL: I have no intention or wish to cast any reflection on Dr. Faulkner's skill and ability to do this operation as he has stated. I only wish to call attention to the loose way in which the term is often used. I have several times tried to do a complete ethmoidectomy on the cadaver and have found it difficult to accomplish. I have, also, tested postgraduate students who have declared that they, as a rule, do a complete ethmoidectomy on the living, and have had them do one on the cadaver, after which a complete dissection of that head was made and it was found that but partial exenteration had been done.

CHAIRMAN CHAMBERLIN: I think if one wants to acquire deep humility, providing he has not it already, he should do just what Dr. Barnhill has said; he should do a complete ethmoidectomy on the cadaver and then dissection of the head, and see how many cells he has left. It is very illuminating.

DR. MCGINNIS (in closing):

I am sorry there was not more discussion. I am indebted to Dr. Shea for having Dr. Kistner show his slides, and they illustrate what I have suspected for a long time. I have tried to get slides of the stained mucosa done at our hospital, but I am told that it is quite a delicate operation; however, Dr. Kistner has done this for us. We have done the next best thing, that is, we recovered the organisms from the crushed tissue.

As to the operation: This is not intended to be a partial ethmoidectomy, but is a drainage operation on the ethmoid labyrinths, and probably should be called an ethmoidotomy. In this operation we do not intend to remove the cell mucosa. In my student days a boil or carbuncle was treated by a wide-open crucial incision, but nowadays the surgeon is more conservative.

Dr. Fenton brought out the point about infecting cells that are not already infected. With this procedure you can stop when you come to normal cells. This answers Dr. Faulkner's objection also. We do not aim to have any ragged edges or surfaces left after the surgery is done. I do not like to see noses that have had the middle turbinal, the middle turbinal plate and the ethmoid cells eradicated. I do not think that is the aim of true surgery.

I am very glad that Dr. Barnhill brought out the fact that it is pretty hard to remove all the ethmoid cells, and this was illustrated in my experience by a case that had a large posterior ethmoid cell which came down behind the antrum and a molar root had penetrated into

it. It would have been a physical impossibility to have destroyed this cell.

The point of this paper is that there are hundreds of individuals who are suffering from a focal infection located in the nasal cells, especially the ethmoid cells, and there is very little local evidence of such infection. I have been very much gratified by the number of satisfied patients, and even cures of general diseases after the elimination, by drainage, of the intranasal cell infection.

SPOROTHRIX INFECTION INVOLVING THE BONES OF THE FACE

FREDERICK T. HILL, M.D.

SPOROTRICHOSIS is a rather uncommon disease, caused by the invasion of tissue by the *Sporotrichum*, one of the Fungi imperfecti. The disease is usually localized in the skin and subcutaneous tissues, although the mucous membranes, musculatures and bones may occasionally be involved. Sporotrichosis of the bones, according to Altschul,¹ "belongs to the rarest of diseases." Beurmann and Gougerot² state that the tibia is the bone most frequently involved. Although quite a few cases of sporothrix infection of the eye and adnexa have been reported, only 14 references to involvement of the bones of the face were found in the literature. Because of the difficulty of pathological diagnosis it may be that many cases have been overlooked. The histological changes are not very characteristic and the demonstration of the organism in the tissues is extremely difficult. In the case I shall report the first pathological diagnosis was "giant-cell sarcoma." Sporothrix grows well on glucose agar, however, if sufficient time is given, so this may be the diagnostic method of choice. Two different strains are mentioned in the literature: *Sporothrix schenkii* and *Sporothrix beurmanni*. There is some doubt as to whether this distinction is justifiable, however, as there are good grounds for considering them as identical.

Fage,³ in 1908, reported a case of sporotrichosis with multiple gummatous lesions on various parts of the body, one involving the bony wall of the orbit. In 1909, Bonnet⁴ reported a case with multiple lesions, one involving the frontal bone. The same year, Beurmann and Gougerot⁵ reported a case in a man, aged sixty-five years, with multiple subcutaneous gummata and abscesses. The nasal bone was involved. This case resulted fatally. In Velter's⁶ case (1910), the infection seemed to originate in the wall of the orbit and later extended in the temporomalar region. Morax's⁷ case (1910), showed involvement of the inner wall of the orbit.

Pautrier and Richou⁸ (1911), reported a case of sporotrichosis of the nose in which the lesion invaded the vomer, causing a perforation of the septum. This subsided under treatment with potassium iodide, together with local applications of iodine. In one of the cases reported by Lapersonne,⁹ in 1912, there was considerable destruction of the external table of the frontal bone.

D'Agata,¹⁰ in 1916, reported a case in which there were two bone lesions, one involving the tibia, the other on the upper jaw. Both lesions were of the nature of an osteoperiostitis. *Sporotrichum beurmanni* was cultured from necrotic material removed from the jaw lesion.

Aloin and Vallin,¹¹ in 1920, reported a case in which the first lesion noted was in the right submaxillary region. This broke down, with the formation of a fistula. Later there was a fluctuant area of swelling over the frontal region. At operation it was found that the frontal bone was necrosed and the dura covered with fungoid growths. Under treatment with potassium iodide the lesions healed. In 1921, Schloffer¹² reported a case in which there were swellings on the jaw and other bones of the skull. *Sporotrichum beurmanni* was isolated from the pus from these areas and also obtained from the blood. In 1922, Warfield¹³ reported a case with multiple gummata due to sporothrix infection. One lesion had eroded the frontal bone.

In Gifford's¹⁴ case, reported in 1922, in which the infection originated in the lacrimal sac, there was extension into the ethmoidal cells. After intranasal exenteration, followed by treatment with potassium iodide and local applications of tincture of iodine, the lesion slowly healed. *Sporotrichum schenckii* was cultured from this case.

In 1923, Mason and Frost¹⁵ reported a case of sporotrichosis in a woman, aged fifty-three years, a native of California. The primary lesion was on the thigh. Eleven months later, a firm, painless nodule developed on the forehead, apparently arising from the periosteum of the frontal bone. *Sporothrix* was cultured from this lesion. In spite of treatment the case resulted fatally.

In 1926, Altschul¹ reported a case in a woman, aged sixty-three years, in which the first symptom noted was pain in the lower jaw. A hard, immovable tumor slowly developed on the left mandible.

The roentgenogram showed several sharply outlined areas of rarefaction, resembling a multilocular bone-cyst in the jaw. A portion of the tumor was removed and showed a fibrous osteomyelitis. Sporothrix was cultured from this tissue. Subsequently similar tumors developed on the forehead and various parts of the cranium. A roentgenogram of the skull showed it to be "studded with large and small areas of rarefaction." Potassium iodide and roentgen-ray therapy were given without effect. Death was due to pneumonia. Necropsy showed defects in the frontal and parietal

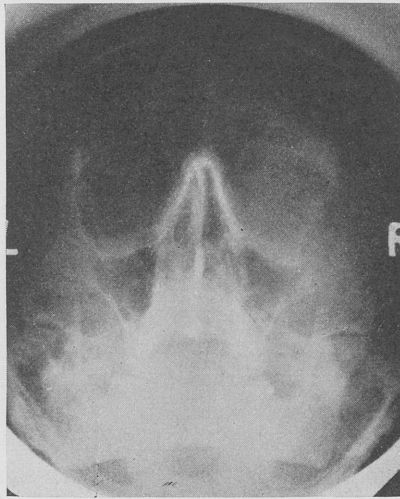


FIG. 1.—X-ray before operation showing destruction of malar and outer angle of orbit.

bones, through which extruded grayish-red material. There was a swelling in the left mandible, with a small, oozing fistula. There were large plaques of tumorlike growths on the dura mater which had partially eroded the bone of the skullcap. There were, also, bone lesions in other parts of the body. The author noted the similarity of the roentgen findings in this case to those of multiple myeloma.

G. G., aged thirty-two years, of French-Canadian parentage and by occupation a telephone linesman, had been under the care of Dr.

H. F. Hill for the past three years, consulting him first for a small growth on his right upper eyelid. This had been noted twelve years before, at which time it was about the size and shape of the head of a match. This had slowly increased in size until it was about 6 mm. in diameter. There were no other symptoms. One year previously this had been opened and curetted by his family physician. Wassermann test was negative. Removal of the growth had been attempted by Dr. H. F. Hill. The first pathological report obtained was "giant-cell sarcoma." Another pathologist gave the report of "giant-cell inflammatory tissue from a fungous growth." This was confirmed by Dr.

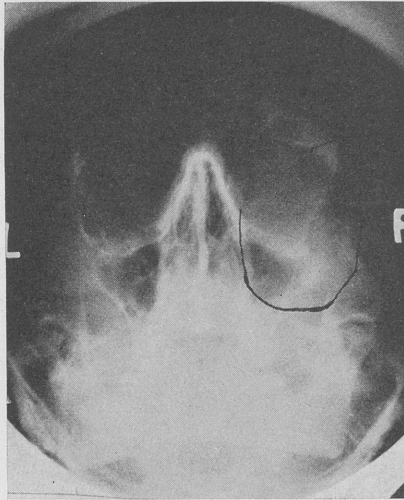


FIG. 2.—Showing bone removed at operation.

F. A. Verhoeff as sporothrix. Subsequently the growth had extended laterally beneath the skin, infiltrating the outer canthus, with the formation of a fistula. Large doses of potassium iodide and roentgen-ray treatments had been given without effect. Salvarsan had been used intravenously. Locally iodine and gentian violet had been employed. From time to time the larger lesions had been removed. At the last removal, six months before, the periosteum at the outer edge of the orbit had been found involved. During the previous year and a half, enlarged lymph nodes had been removed from both inguinal regions and the right side of his neck by his family physician.

Some of this material had been examined but no organisms were found.

When the patient was referred to me there was marked chemosis and exophthalmos, with an indurated nodular swelling extending downward from the outer edge of the orbit and involving the malar bone. He had been suffering constant, intense pain for the previous three weeks, the first discomfort he had had. Roentgen examination showed partial destruction of the right malar bone and of the external angular process of the frontal bone, with areas of rarefaction; antrum cloudy. Radical surgery seemed to offer the only hope of relief.

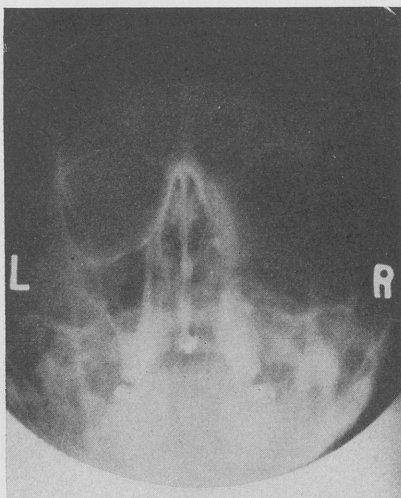


FIG. 3.—X-ray after operation.

Under rectal anesthesia, after removal of the eye by Dr. H. F. Hill, an attempt was made to exenterate the entire mass. This was done by a combination of rongeur forceps on the bone and surgical diathermy on the soft parts, going well around the involved areas. The lower eyelid and the major portion of the upper were sacrificed. As the growth had apparently burrowed beneath the skin and did not involve it lower down, a flap with its base on the nose was thrown back and the underlying structures removed. The orbit was completely exenterated, its outer wall removed well out to the zygomatic arch and the floor taken away, exposing the interior of the antrum and removing its lateral wall. The bone had the peculiar appearance

and feel of "rotten ice." Where the growth had invaded the soft parts it had a dense, fibrous appearance. The membrane lining of the antrum was apparently not involved, nor had the growth extended into the ethmoid region. After the removal was completed the skin flap was sutured across the lower portion of the opening and it was lightly packed with iodoform gauze.

He reacted well from the operation and experienced almost immediate relief from the previously constant pain. Strange to say, he had little difficulty in chewing, such as one might expect from the removal of the anterior anchorage of his zygoma. After the packing was removed, daily exposure to the Kromayer lamp was given. Under this treatment, together with the applications of gentian violet,

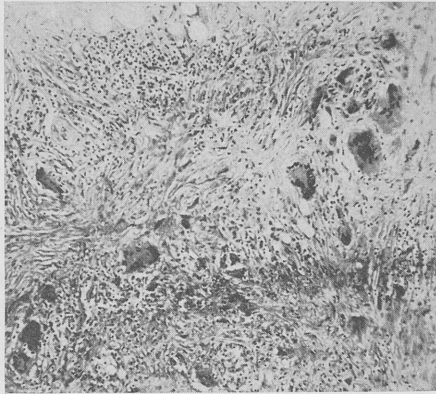


FIG. 4.—Section stained with hematoxylin and eosin showing giant-cells and fibrosis.

healing slowly took place. The orbit gradually filled in and became epidermatized. Cultures taken at various times were negative for sporothrix. The outlook appeared quite optimistic until four months afterward, when a nodular swelling was noted in the canine fossa, extending backward. At this time he had a return of the pain in this region.

Under local anesthesia, an incision was made in the canine fossa exposing a dense, white, fibrous mass, similar to that removed at the previous operation. This was found to be much larger than expected and to extend backward almost to the ramus of the mandible. In order to dissect this free it was necessary to reopen the old incision in the cheek, fully exposing the site of the previous operation. Because

of this we were able to check up on the condition of the old operative field. In the upper portion, where the floor of the orbit had been removed, there was no sign of recurrence. There was still no evidence of involvement of the antral lining membrane. Infected bone was found at the outer inferior angle of the antrum, in what remained of the lateral wall. As a result of the previous operation the quadratus labii superiores, canines and zygomaticus muscles had been practically obliterated. At this time the process had involved the superficial portion of the masseter. The growth exhibited a peculiar fibrous characteristic where it had invaded muscle, showing distinct lines of cleavage, something like vegetable root-fibers. There was almost no bleeding. Cutting into the growth was almost like cutting

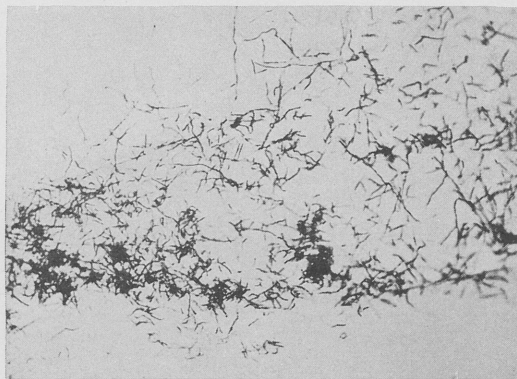


FIG. 5.—Section of culture media showing sporothrix growing on glucose agar, stained with Verhoeff's modified Gram stain.

a raw potato. Removal was very difficult and resulted in tearing into Steno's duct, so that there was some free flow of parotid secretion into the wound subsequently. The canine fossa incision was closed, but the upper one kept open for better observation of the field. This was lightly packed with iodoform gauze, which was removed after forty-eight hours. Exposure to the Kromayer lamp was resumed and has been continued since at intervals of about three days.

Since this operation there has been freedom from the pain. He is feeling well and is out and doing about as he likes. Recent roentgenograms of his skull showed no further bone involvement. We were especially on the lookout for areas of rarefaction in the skullcap, as noted by Altschul. The possibilities of metastatic involvement of

the lungs and—bearing in mind the enlargement of the inguinal nodes—of invasion of the pelvis, warrant careful consideration. Roentgen examination of these areas have so far been perfectly negative. One cannot help but be very pessimistic regarding the prognosis. The continued slow progression of the process, despite therapy and surgery, makes one dubious of the possibility of cure. Had radical surgery been applied earlier, when the lesion was apparently regional and circumscribed, there would have been better chance of complete eradication.

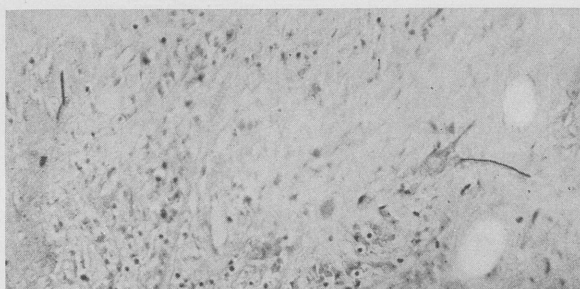


FIG. 6.—Section stained with Verhoeff's modified Gram stain showing sporothrix in tissue.

It has been puzzling to account for the etiology of this infection. According to Gifford,¹⁶ sporotrichosis most commonly involves the hand, following some superficial wound or abrasion. This patient at no time has had any lesion on his hands, or elsewhere superficially on his body, except the eyelid. In inquiring into his occupation it seems that his work as a linesman was on cross-country telephone lines. During his noonday lunch-hour he was in the habit of taking naps, lying face downward on the ground. It might be possible that he got his primary infection on his eyelid in this manner. However, this disease is extremely rare in New England, although not so uncommon in the Mississippi basin, and this man has always lived in Maine.

PATHOLOGY. The pathological work on this case was done by Dr. F. A. Verhoeff, whose isolation of the *Sporotrichum* earlier in the course of the disease had established the diagnosis. His report is as follows:

All the tissues submitted for examination, including the bone tissue, except the lymph gland, show the same inflammatory process. The normal tissue has been pervaded with granulation tissue which has gone on to the formation of dense, fibrous tissue. The tissue contains large numbers of foreign-body giant-cells, but no tubercles resembling tuberculosis or syphilis. Some of the tissue shows considerable infiltration with lymphocytes; some, only moderate infiltration with these cells. By ordinary methods of staining no organisms can be found in the tissues, but Verhoeff's modified Gram stain shows filamentous organisms scattered through the tissues, some of them of great length. Some of them are surrounded by giant-cells; others are not. The lymph gland shows the lymph sinuses distended with epithelioid cells. No organisms can be found in the lymph gland.

Smears from the pus from the lesion in the lids show sporothrix filaments in considerable numbers. A culture taken on glucose-agar showed the organism in four days. Transplants from the original growth on glucose-agar and Sabouraud's media failed to grow. Pieces of the original culture media were fixed in formalin, sectioned and stained in Verhoeff's modified Gram stain. These show the organisms growing down into the medium in characteristic fashion.

Potassium iodide has been considered as almost a specific in these cases. Gifford¹⁶ says that in large doses it nearly always brings about a prompt recovery. However, in this case prolonged treatment with potassium iodide, to the point of toleration, for four years, has been useless. Several of the cases with bone involvement, reported in the literature, also have shown no improvement and have resulted fatally. It would seem probable that any effect that potassium iodide has in these cases is produced by breaking down the fibrosis, as in tertiary lues, and thus allowing the natural defensive powers of the body, or such other therapeutic agents as may be employed, to act upon the organism, rather than by any specific reaction for the *Sporotrichum* inherent in the drug itself.

SUMMARY. A case of sporotrichosis is reported, of twelve years' duration, beginning with a small, circumscribed lesion on the eyelid. This was apparently activated by curettage into a slow but persistent progression, finally involving the bones of the face. Iodine, salvarsan and roentgen-ray therapy have been of no avail. There seems to have been a predilection toward, first,

bone, and later, muscular involvement; with little tendency to invasion of the skin or mucous membranes. Pain was a late symptom and apparently due to pressure. Radical surgery, at the time instituted, has only been palliative, at least from present indications.

CONCLUSIONS. From the meager material available it is useless to attempt to draw conclusions. However the following points seem to present themselves for consideration:

1. Sporotrichosis of the bones is extremely rare.
2. Its diagnosis is difficult and may be overlooked or confused with neoplastic disease. Cultural diagnosis is easier.
3. Its prognosis is dubious. Potassium iodide therapy is not always successful.
4. Incomplete surgery is inadvisable. If surgery is undertaken, it should be radical and done when the lesion is circumscribed, if possible.

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DISCUSSION

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

What form of iodine was used in cases of actinomycosis? We have found Lugol's solution works best, and I do not know whether it might have been used in this case.

DR. RALPH A. FENTON, Portland, Oregon:

I would like to ask Dr. Hill, if there is no further discussion, a question as to whether other cases reported in the literature had been handled by surgical diathermy?

DR. HILL (in closing):

In answer to Dr. Carmody's question I will say that in the previous four years before I saw the case, I cannot take any credit for the treatment. That was carried on by my brother, who is an eye man. I think he tried out every form of iodine. I know Lugol's solution was used. Tincture of iodine was used locally and injected into the tissues, and tremendous doses of potassium iodid were used, to the extent that he had drug rash and gastrointestinal upsets.

As far as I can find, Dr. Fenton, in the cases reported in the literature there was no evidence of diathermy having been used before.

A STUDY OF THE BLOOD IN THE VARIOUS ANGINAS

JOHN J. SHEA, M.A., M.D.

HEMATOLOGY is undergoing radical changes. The relationship of the anginas, and the hematopoietic system is uncertain, for in one instance an infection of the throat may be the initial lesion, and in another the terminal manifestation of a hematopoietic disease. The medical student of ten years ago, was taught a theoretical classification of the diseases of the blood, which was not practical when applied in the routine of medicine. Today, we seek a better knowledge of the possibilities of the hematopoietic response to infection, and this knowledge can best be obtained through a closer study of the blood findings in the anginas.

Turck,¹ in 1907, described a hematopoietic disease, which has become known as agranulocytic angina. Schults,² in 1922, again drew attention to the disease, and since then many cases have been reported in the literature. I wish to report 1 true case, and 2 cases, 1 from neoparsphenamine and 1 of an unknown origin, whose blood pictures were those of an agranulocytic angina.

The older physiologist considered the blood as a special form of connective tissue having a fluid matrix. The modern conception is, that the blood is a mixed secretion, whose formed elements are the result of parent cells living outside the blood stream. These hematopoietic tissues differ in the embryo, and the adult. The fetal blood is formed as a result of the activities of the blood islets in the yolk sac, the primitive liver and the reticulo-endothelial system. The capillaries of the formative bone-marrow, and the lymphoid follicles of the spleen, and other lymphoid structures next add to this natal blood. The process changes gradually, until in adult life there exist groups of parent cells, outside the blood stream, with four major hemic units in the blood. From the parent myeloblasts come the granular cells, which are the polymorphonuclear leukocytes, and the monocytes; from the lymphoblasts are derived the lymphocytes; and from the normoblasts, the red blood cells.

The bone-marrow houses an emergency supply of polymorphonuclear leukocytes, and can deliver them into the circulation when needed. If the surplus is consumed and the newly-formed ones are not sufficient for the demand, then immature forms will be found in the circulation.

The most common causes for the presence in the blood of immature cells are:

- (a) Hemorrhage.
- (b) Diseases which destroy blood cells.
- (c) Various substances which act as stimulants to the hematopoietic system.
- (d) Hyperplasia of the hematopoietic tissues.

The infections of the throat are characterized by the influence they exert upon the hematopoietic system, and belong to the third class. On the other hand there are primary blood diseases which produce lesions in the mouth, and it is difficult to separate the border-line cases.

The laryngologist has an opportunity of studying the relationship of the anginas and the hematopoietic response in the following diseases:

- (a) Agranulocytic angina.
- (b) Acute infectious mononucleosis.
- (c) Vincent's angina with a lymphoid leukocytosis.
- (d) Acute lymph adenosis.

The anginas are capable of producing four types of changes in the blood:

- (a) Polymorphonuclear leukocytosis as seen in follicular tonsillitis and the septic throats.
- (b) Mononuclear leukocytosis as occurs in infectious mononucleosis.
- (c) Leukopenia with a decrease of granulocytes—agranulocytic angina.
- (d) Leukocytosis with a relative hyogranulocytosis, as seen in the patients suffering from a generalized Vincent's infection.

AGRANULOCYTIC ANGINA. Today we are seeing more of these agranulocytic anginas either because the disease is more frequent, or because the laryngologist is more alert. There are two varieties: one which ends fatally, and is most frequently seen in middle-aged

women, and another, which is only a complex. The etiology of the diseases is not definitely known. Various observers have isolated the following from lesions of the throat in these cases:

- (a) *Bacillus pyocyaneus*.
- (b) Streptococci—hemolytic; streptococci—nonhemolytic.
- (c) *Staphylococcus*.
- (d) Vincent's bacteria.

The toxin which causes the malady is capable of paralyzing the hematopoietic system, especially the bone-marrow, where the myeloblasts are producing granular units. The action of the toxin is the same as that of massive doses of roentgen-rays on the long bones. The initial sore throat is the usual portal of entrance of the infection, but later other mucous membranes, as in the intestines and vagina, present lesions. The throat picture depends on the amount and the location of the lymphoid tissue that it contains, and if the patient has retained her tonsils they will be found to be covered with an exudate. Thereafter the progress of the disease is of a general infection which ends fatally. The true case I am reporting, was clinically diphtheria.

Strangely, the glandular system does not present any uniform enlargements as only the glands immediately adjacent to a lesion enlarge. The logical treatment of these cases is to supply the deficiency, which is blood, and to stimulate the hematopoietic tissues to renewed activity.

Lymphocytes are relatively increased though absolutely decreased. The red blood cells, platelets, and hemoglobin remain within normal limits until the terminal stage, when they take on the picture of a secondary anemia. The bleeding- and coagulation-time remain normal, and the blood of these patients type, and match readily when seeking a donor for transfusion. The blood cultures reported have not been consistent as to any organism.

CASE I.—Mrs. M., aged fifty-one years, white, housewife, was seen in consultation with Dr. Henry G. Rudner at the Baptist Hospital May 6, 1929.³

Chief Complaints. Weakness, sore throat and fever.

Family History. One sister died with a blood dyscrasia (myelogenous leukemia). One uncle died of carcinoma of the throat, and one of her grandmothers died of a malignancy of the eye.

Past History. She had frequent attacks of tonsillitis during childhood, the right tonsil was tonsillotomized at the age of twenty-one years, with good after-results. During the past summer she had "malaria" but did not take quinine. Eight months previous to admission she had neuritis and pyorrhea and her remaining teeth were extracted.

Present Illness. Three weeks previous to admission she had fever with no localizing symptoms. Quinine was administered, but discontinued because of an urticaria. One week later she developed a severe sore throat, and the family doctor gave diphtheria antitoxin, as both tonsils were covered with a pseudomembrane. For one week her throat was sore, and painful, and the fever rose each evening as high as 104° F. A few days previous to admission she had diarrhea, and pain in her knees.

General Examination. General appearance of an acutely sick woman with an icteroid tinge.

Face. Herpes on nose and lips.

Mouth. No teeth present, and the alveolar processes were normal. The tongue was heavily coated with a white fur. The pharynx was infected. There was a necrotic ulcer on the anterior pillar of the left tonsil, and the posterior pillar of the same tonsil was displaced by a swelling from behind which was probably a gland. The tonsils were clean, but had a cadaveric appearance.

Neck. The anterior glands were palpable and tender.

Chest. Numerous bronchial râles. The heart was normal.

Abdomen. Generalized tenderness, but the liver and spleen were not palpable.

Genitalia. Showed a marked irritation with an erosion on the left vulva.

Extremities. Knees were tender but not enlarged, with normal mobility.

Rectum. A small erosion present.

Blood. Red blood count 3,240,000; white blood count 1200; polymorphonuclears 10 per cent; large lymphocytes 10 per cent and small lymphocytes 80 per cent.

Urine. Negative.

Smears from the throat lesion were rich with the bacteria of Vincent. A culture made from the same region was negative for diphtheria bacilli.

Progress. The mouth lesions became more lymphocytic in character, and similar lesions appeared in the rectum. A severe diarrhea

ensued from similar intestinal lesions and the patient gradually grew weaker and died.

The treatment consisted of three whole blood transfusions averaging 200 cc. Deep roentgen-ray therapy was applied to her tonsils, glands

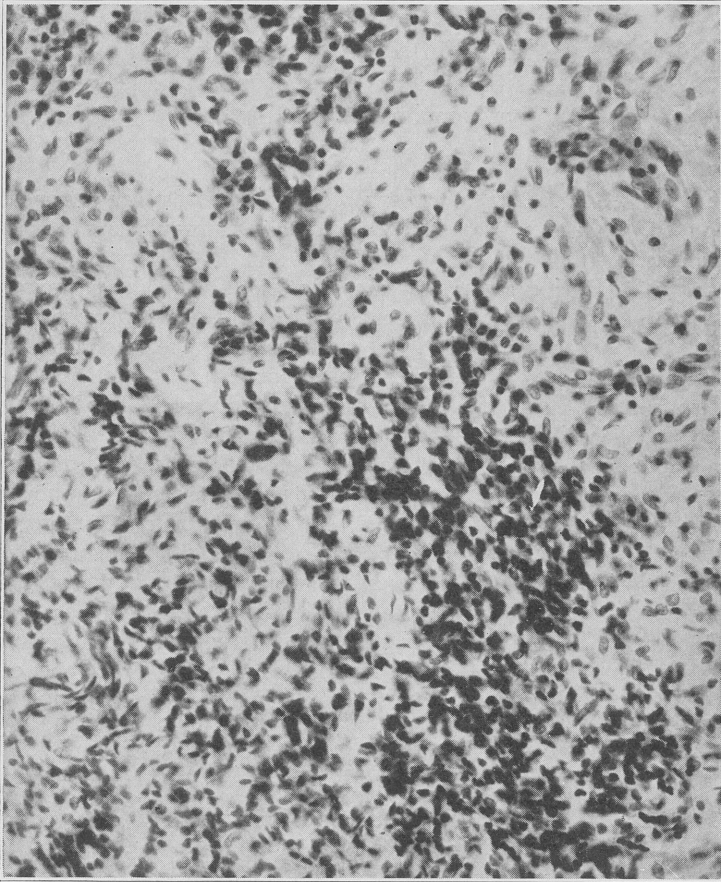


FIG. 1.—Case I. Photomicrograph of tonsils showing an atrophy of the lymphoid elements.

of the neck, pelvic girdle, and the long bones. The blood count was done daily, and descended as low as 500 leukocytes with an average of 10 per cent of the granular cells.

The autopsy revealed that the bone-marrow was aplastic and consisted mostly of fat with a few small areas of marrow cells, consisting chiefly of lymphocytes and other mononuclear cells. The tonsils showed microscopically an atrophy of the lymphoid structure. (Fig. 1.)

A CASE OF ARSENICAL POISONING WITH AN AGRANULOCYTIC BLOOD PICTURE. The intravenous administration of arsenical compounds may at times depress the hematopoietic system and present a symptom-complex resembling acute agranulocytic angina.

CASE II.—Mrs. D. P., aged twenty-six years, was seen in consultation with an acute throat, fever and a rash previously diagnosed "measles."

History. During a physical examination her blood was found to react 4 + to the Wassermann test, and arrangements were made for a course of neoarsphenamine. Three days after the second dose she developed a fever of 104° F. with a macular rash.

Upon admission to the hospital three days later she still had a slight rash, and her blood count was reported as follows: red blood cells, 3,300,000; hemoglobin, 60 per cent; white blood cells, 4400; polymorphonuclears, 2.5 per cent; eosinophiles, 2 per cent; small lymphocytes, 81.5 per cent; large mononuclears, 14 per cent. Her treatment consisted of one blood transfusion of 300 cc. of whole blood, intravenous administration of 7½ grains of sodium thiosulphate every twelve hours for three days, and a supportive diet rich in vitamins.

A chart of her blood shows the gradual increase of the granular elements, and the blood count on discharge was white blood cells, 9100; polymorphonuclears, 74 per cent; small lymphocytes, 25 per cent; large mononuclears, 1 per cent. The course of the blood is shown in Chart I.

The temperature curve was as seen in Chart II.

A CASE OF AGRANULOCYTIC ANGINA COMPLEX OF UNKNOWN CAUSE.

CASE III.—W. S. M., aged sixty years, was admitted to the hospital convalescing from pneumonia. He had a bilateral acute maxillary sinusitis which improved under simple irrigation, and was ready for discharge when he developed an acute angina with a rising

March, 1930,

	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.	22.	23.
Hemoglobin	60%								4,900,000				
Erythrocytes per cubic millimeter	3,300,000	4,100,000		4,100,000					5100	6500	7250	7500	9100
Leukocytes per cubic millimeter	4900	4100		4650		4550	5000	5200					
Polymorphonuclear:													
Neutrophiles	2.5%	2%	4%	8%	13%	22%	36%	37%	49%	52%	60%	62%	74%
Eosinophiles	2%	1%											
Basophiles								1%					
Small lymphocytes	81.5%	56%	64%	68%	61%	62%	64%	58%	42%	42%	36%	32%	25%
Large lymphocytes		3%	2%	1%	1%								
Large mononuclears		36%	30%	23%	24%	15%	10%	5%	9%	6%	4%	6%	1%
Transitionals	14%	2%			1%								

11a

	March, 1930,										April, 1930,		
	22.	23.	24.	25.		27.	28.	29.	30.	31.	1.	2.	
Hemoglobin	80%	Transfusion	85%	
Erythrocytes per cubic millimeter	4,000,000	3,700,000		4,600,000	4,500,000	4,500,000
Leukocytes per cubic millimeter	1800	2150	1550	1550		3950	4000	4300	6300	6900	11,100	10,500	
Polymorphonuclear:													
Neutrophiles	1%	3%	2%	3%		28%	41%	48%	58%	58%	72%	73%	
Eosinophiles													
Basophiles													
Small lymphocytes	97%	96%	90%	82%		60%	52%	40%	33%	40%	24%	20%	
Large lymphocytes	8%		3%	2%	5%	3%	
Large mononuclears	2%	1%	4.5%	5%		8%	5%	7%	9%	2%	4%	7%	
Lymphoblasts	3.5%	2%		1%							

Form 52 8-29 6m

BAPTIST MEMORIAL HOSPITAL MEMPHIS, TENN.

Case No. _____

GRAPHIC CHART

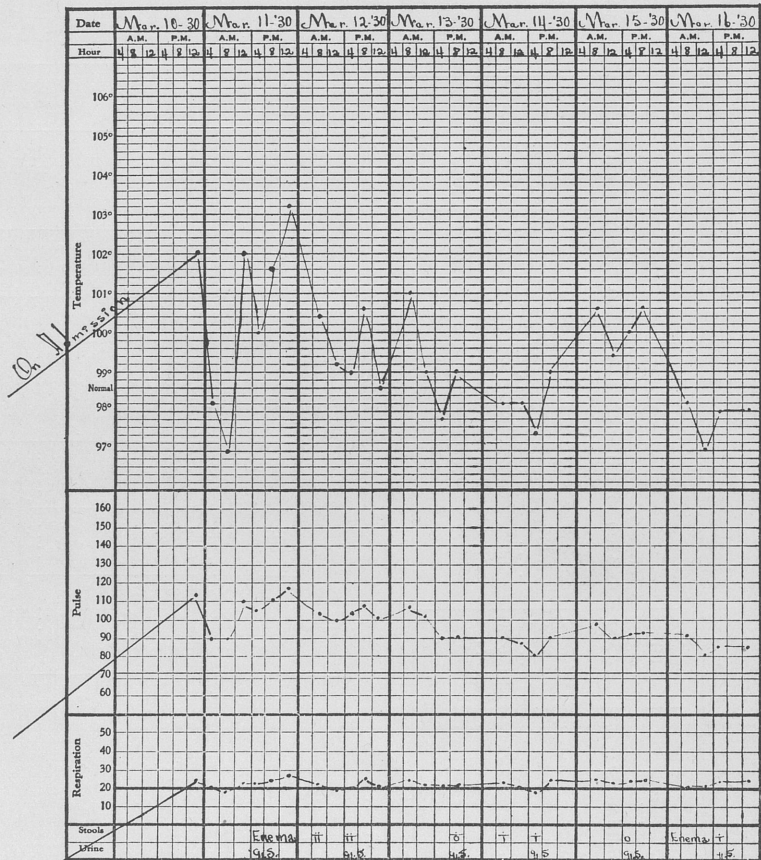
Name Miss Patterson Address 1431 Carr Service of Dr. Rudner and Shea


CHART II.—Case II. Temperature curve.

Form 82 8-29 5m

BAPTIST MEMORIAL HOSPITAL

MEMPHIS, TENN.

Case No. _____

GRAPHIC CHART

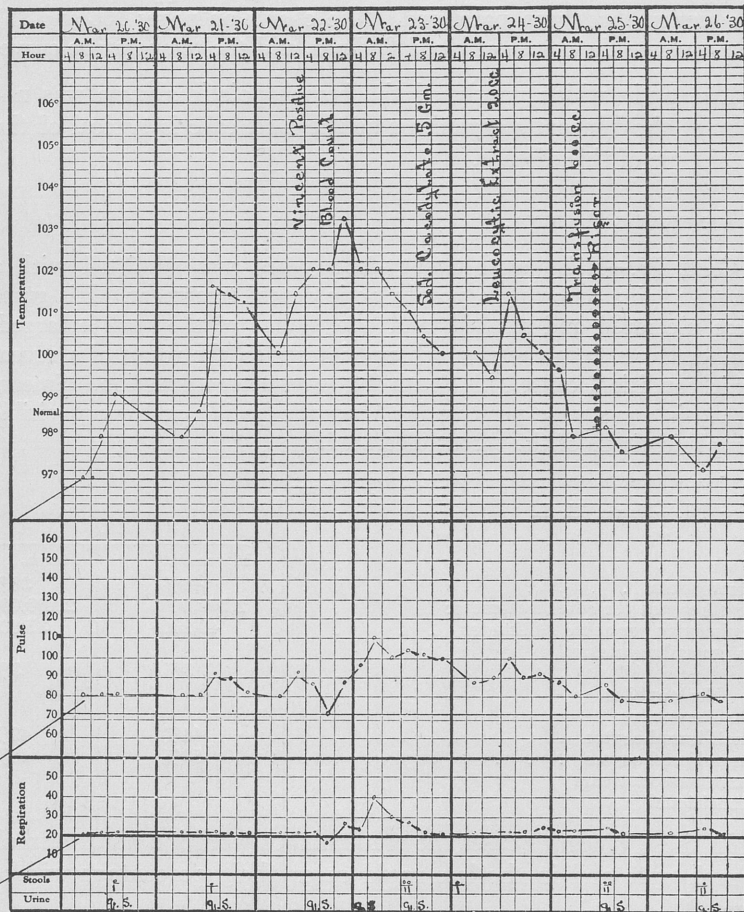
Name Mr. Martin Address 428 Union Service of Dr. Shea

CHART IV.—Case III. Temperature curve during the acute angina.

temperature. A small ulcer was visible on the left tonsil, from which many fusiform bacilli, and spirochetes of Vincent were obtained. The blood picture was a revelation. White blood cells, 1800; polymorphonuclears, 0.1 per cent; small lymphocytes, 97 per cent; large mononuclears, 2 per cent. The granular elements were reduced to 3 per cent, showing the hematopoietic tissue, whose function is their production, had been paralyzed. The charts on pages 166 and 167 demonstrate the course of blood findings, and the temperature curve.

The toxin which paralyzed the hematopoietic system was not that of the Vincent's organisms as the next case is typical of the fatal type of this disease.

VINCENT'S ANGINA.

CASE IV.—E. L., aged twenty-four years, was admitted with a severe angina following a prophylactic dental treatment. His gums were ulcerated and swollen, and the glands of the neck enlarged. Hemorrhages from the stomach and intestines, the result of a severe toxic condition, caused his death five days after admission. Smears from his gums were positive for Vincent's bacilli, and every visible lymphoid tissue of the throat ulcerated.

CHART V.—BLOOD

Case IV.—Name, E. L.

	1923,	
	March, 11.	March, 14
Hemoglobin	46%	38%
Erythrocytes per cubic millimeter	2,700,000	2,100,000
Leukocytes per cubic millimeter	40,200	37,800
Polymorphonuclears:		
Neutrophiles	3%	3%
Eosinophiles		
Basophiles		
Small lymphocytes	44%	49%
Large lymphocytes	53%	48%
Large mononuclears		
Transitional		

In these Vincent's cases the predominating cell is a mature small lymphocyte and in the less rapid cases their percentage will be over 90.

INFECTIOUS MONONUCLEOSIS. The monocytes are normally on the increase during the second stage of leukocytic response to infection, and in chronic malaria the percentage of monocytes is from 6 to 10.

Pfeiffer in 1889 described an acute febrile disease characterized by an angina with enlarged glands, and having a blood picture in which the monocytes and lymphoid leukocytes were increased. Many names have been assigned this condition:

- (a) Glandular fever.
- (b) Infectious mononucleosis.
- (c) Acute benign lymphoblastosis.
- (d) Acute lymphadenosis.

The disease is the result of some toxin stimulating the hemato-poietic system, and the resulting new cell produced being a mononuclear leukocyte.

There are three possibilities:

- (a) Lymphocytes.
- (b) Monocytes.
- (c) Myelocytes.

The third is a possibility, but I have never seen an acute angina with an increase of the myelocytes, but the other two types are more common than is appreciated.

(a) *Lymphocytic Leukosis Following an Angina.* The spreading of Vincent's disease throughout the United States by the return of the soldiers from France, has increased the frequency of this leukosis. Vincent's disease is usually a local pathological entity, but occasionally it may excite a systemic response, and if this response is hemic, there will result a lymphocytic leukosis. The number of leukocytes may reach 30,000 to 40,000, and the percentage of lymphocytes over 90. These patients usually die unless carefully treated. These cases differ from the acute lymph adenosis of which the following is an example:

ACUTE LYMPHADENOSIS.

Mrs. T. S. W., aged twenty-three years, formerly our operating-room nurse, was admitted to the hospital with an acute glandular enlargement, and a blood count of white blood cells, 399,000; small lymphocytes, 16 per cent; large lymphocytes, 6 per cent; polymorphonuclears, 78 per cent. One month previously she had undergone a minor gynecological operation, at which time she was thoroughly examined and found to be in average health. Her progress was rapid, and she died one month after admission.

	1928,			1929,
	Dec. 22.	Dec. 26.	Dec. 30.	Jan. 14.
Red blood cells		1,600,000
Hemoglobin		25%
White blood cells	399,000	202,000	352,000	205,000
Small lymphocytes	16%	42%	50%	88%
Large lymphocytes	6%	29%	30%	5%
Polymorphonuclears	78%	27%	15%	4%
Lymphoblasts	2%	5%	3%

Are these small cells stem cells, or mature small lymphocytes? In this case the granular cells responded in the beginning, and the first differential of 78 per cent is in marked contrast with the 3 per cent of the last count. A thorough study of this young lady was nonproductive of any knowledge of the etiology of her disease. The mouth was carefully watched for Vincent's infection but remained clean though very painful during the last week of her life. The terminal mouth and throat lesions were due to a hyperplasia of the lymphoid tissue in the mucous membrane. The tongue became dry, and coated with old blood, and she complained of the tongue burning similar to that suffered by the pernicious anemia patient. The glands of the neck remained the same size and consistency throughout the course of the disease.

(b) *Mononucleosis*. A febrile condition starting with an angina, and having the usual headaches and malaise, but whose blood study shows a relative and absolute increase of monocytes is the true "infectious mononucleosis." It occurs most frequently in the young adult, and females are more susceptible than males. The etiology is nonspecific and the course is benign. The oxydase stain will differentiate the monocytes.

The glands of the neck are enlarged, but not strikingly so, and the spleen palpable. The first study of the blood with Wright's stain will merely show a relative and absolute increase of the mononuclear leukocytes, but the oxydase process will bring out the granules in some of the cells, thereby separating the lymphocytes and the large mononuclears. The symptoms subside gradually, but the blood picture remains, "mononuclear" for months. The throat lesions in these cases vary from a slight ulceration to extensive inflammations, and the bacterial study of direct smears and cultures gives no help. The presence of Vincent's organism

in the direct smear is common if there is an anaërobic recess, as under a membrane covering a slough. The total white blood cell count does not increase to an alarming degree and remains under 20,000.

Last year I saw in consultation a young married woman with a persistent angina whose blood picture was: red blood cells, 3,800,000; hemoglobin, 75 per cent; white blood cells, 12,100; polymorphonuclears, 32.5 per cent; polymorphonuclear eosinophiles, 2 per cent; small lymphocytes, 58 per cent; large mononuclears, 7.5 per cent; and six months later was: red blood cells, 4,200,000; hemoglobin, 75 per cent; white blood cells, 5800; polymorphonuclears, 50 per cent; polymorphonuclear eosinophiles, 1 per cent; small lymphocytes, 45 per cent; large mononuclears, 4 per cent.

When she was first seen a smear from her tonsil lesion was rich in the organisms of Vincent, and we feared she was to be one of the lymphoid leukocytosis cases, but a study of her blood with an oxydase stain showed a 7.5 per cent of the large mononuclear cells, and we then knew she was of the benign class, "acute infectious mononucleosis."

CONCLUSIONS. 1. The toxins of the various anginas may stimulate or paralyze the hematopoietic system.

2. A closer study should be done of the blood of these angina patients.

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DISCUSSION

DR. RALPH A. FENTON, Portland, Oregon:

We are very much indebted to Dr. Shea for classifying in this careful manner the reaction of the blood to these various difficult anginal cases, and, in passing, for his having brought out the importance of

transfusion as a means of treatment. I think our literature has hitherto been lacking in an effective study of this type; and, while there is a good deal to be found in the literature of the blood, I think comparatively little of that has been made available to us. Too frequently, when we call upon the internist or the hematologist for assistance, he gives us a string of figures and very little in the way of clinical help. The reticuloendothelial system (improperly called endothelial, perhaps) and its relation to the defense reaction of the organism has received comparatively little study by otolaryngologists. Only recently in Europe, and to even a less extent in this country, has any mention been made of the defense reaction arising from the essential elements of the mesenchyme, the circulating tissue which is the blood, and the mesenchymal cells which furnish these defensive histocytes, which are such a valuable sign of recovery in the anginas. These things have not been incorporated in our literature. Only lately papers appeared at the Collegium, Dr. Beck tells me, in London last summer, and a study by Terracol before the Latin Otolaryngological Society only last fall in Madrid. I think these are the only papers which have thus far appeared upon this topic, and they were not in relation to throat complications.

With this start, and with the stimulus which Dr. Shea's important contribution has given us, I feel that many of us will be guided and helped in our treatment. I would like to ask Dr. Shea whether transfusion, was not, from a clinical standpoint in his later cases, the most valuable single weapon he had? In our few cases of extremely severe Vincent's, we have been very greatly helped by repeated transfusions, just as in severe cases of blood-stream infection from the ear.

DR. WILLIAM V. MULLIN, Cleveland, Ohio:

I desire to report a case of an unusual blood dyscrasia.

A woman, aged twenty-eight years, was admitted to the hospital with the diagnosis of acute mastoiditis. A month previous she had not been feeling well, was very nervous and could not sleep. General physical examination and a complete blood examination at that time were entirely negative. She was given a prescription for elixir of luminal. After she had taken about an ounce of this a severe rash appeared. Her physician made a diagnosis of scarlet fever, and the patient was sent to the City Hospital. There it was decided that she did not have scarlet fever, despite desquamation of the skin on the palms of her hands and soles of her feet. She returned home under the care of her physician, and soon after developed pain in her right ear, with an exacerbation of the rash.

When I saw her the exfoliative dermatitis involved practically every part of her body, and a few vesicles along the back were discharging some pus. There was one under her arm and one in the right external auditory canal which produced severe external otitis, accounting for the mastoid pain. There were also a few vesicles along the hard palate. These were negative for Vincent's organism. Cultures from the skin vesicles showed staphylococci. Her temperature was 104° F. The blood picture was as follows: red blood cells, 3,000,000; white blood cells, 3000; hemoglobin, 70 per cent; no polymorphonuclear leukocytes, and 60 per cent small lymphocytes.

In twenty-four hours the red blood cells had dropped to 2,000,000, the white blood cells to 1000, the hemoglobin to 40 per cent, and there were still no polymorphonuclears. Three blood transfusions brought a satisfactory response, although during her three weeks in the hospital she desquamated over and over again, losing every hair on her body. She had some abdominal distress and occasional diarrhea. Because of the desquamation it was difficult to make protein skin tests, but so far as we could determine, her reactions were always negative. Although several times we thought she would not recover, she was finally discharged with a normal blood picture.

About six weeks after her discharge from the hospital, she returned to the Clinic complaining of several attacks of asthma. She was given potassium iodid, and immediately a rash appeared.

This case is of interest because, apparently, only a few doses of luminal caused a breaking down of the bone-marrow to such an extent that it almost terminated fatally.

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

Dr. Pierce advised me to report this case, which I have not seen and only heard of indirectly, but it is so striking as far as angina is concerned, and so fits in with this paper, that I promise to give the details to Dr. Shea as soon as I get them. A young attorney, just graduated, whom I knew personally, I saw on the previous Sunday when he was perfectly well, and on Thursday he was buried. He had died from what Dr. Dick, the expert on scarlet fever, called an unerupted scarlet fever. There was absolutely no chance at all, so far as the blood condition was concerned, for anything to be done for him. The physician was called in on Monday morning and found, as he called it, a fiery red sore throat, a dry tongue and dry skin, with a very high temperature, and the boy was terribly sick. A blood count was made and it was found that the leukocytes were practically absent.

Just two counts were made during this time. The boy was treated in his home. It may be a type of angina with practically no leukocytes. He had transfusions and everything possible was done, but he died.

DR. MERVIN C. MYERSON, Brooklyn, N. Y.:

Dr. Shea deserves a great deal from this society for bringing this subject to the attention of the laryngological profession. This is a very intriguing and very fascinating subject for one who gets to see a good many of these cases. I have been privileged to have the good fortune to see quite a large series of these cases in conjunction with a fine hematologist in New York City, Dr. Nathan Rosenthal.

We all know that any severe toxemia, be it caused by uremia or a leukemia or chemical poisoning from arsenic or mercury, can cause peripheral necrosis, and it is this picture of local reaction and necrosis which leads us to these blood studies in these cases.

There are cases, however, of severe toxemia with blood pictures such as Dr. Shea has presented upon the screen which, peculiarly enough, do not show an anginal picture.

We have had 2 such cases on the medical services at the Mount Sinai Hospital where the patients had rectal and intestinal lesions at autopsy, but no throat lesions.

Then there is another very interesting phase of the subject of angina, and that is that Dr. Rosenthal, in taking an incidental blood count as a preliminary to an appendectomy, and he has done this in two or more patients, has found a rather low polymorphonuclear count, and he has studied one case for over three years and he has found that this patient over a period of time, with blood counts taken every few weeks, let us say, has a variation in the percentage of granulocytes, that he may run down as low as 20 or 12 and up as high as 60, which makes him feel that perhaps these patients are carrying some low-grade toxin which does not assert itself and which when it does assert itself gives us the picture of what we are inclined to call agranulocytic angina.

We see severe streptococcic throats, such as Dr. Beck has spoken of, and in those cases we get superficial ulceration which later becomes deep because of a capillary thrombosis due to the streptococci locally.

Those cases may come in with a very low poly count and very low leukocyte count and if the patients are going on to a fatal termination, the leukocyte count will drop.

Another observation that we have made is the fact that we have

taken blood cultures frequently and systematically in these cases and we found that where we get a streptococcic infection of the blood, which, by the way, we consider secondary to the angina or to the systemic disease, the prognosis is invariably bad. Where, however, we get a negative blood culture, we have a right to expect that the patient has a chance for recovery. In the last series Dr. Rosenthal spoke of 22 cases: he had 6 recoveries.

DR. ERNEST M. SEYDELL, Wichita, Kans.:

I wish to take issue with Dr. Shea in reference to his statement that all cases of true agranulocytic angina are associated with icterus. I recently treated a fatal case in which the white cell count was reduced to 800 and in which there were no granular leukocytes. This woman was never icteric.

Freedman reported 60 cases of agranulocytic angina. Four of these were men. In this report he states that the absence of icterus does not exclude a diagnosis of agranulocytic angina. In Freedman's 60 cases 26 had a bacteriemia and no specific organism was found. An interesting point about these cases which was not brought out in Dr. Shea's paper is the fact that these patients may have several attacks of angina. These may be mild, at first, but the patient may die later from a severe form of the disease.

In one of my cases the patient had three distinct attacks of angina. Each attack was accompanied with considerable prostration and a reduction of the white cell count. The last attack was fatal.

We are also accustomed to think that agranulocytic angina occurs only in adult women. A review of the literature discloses the fact that men are not immune to this disease. Two cases have been reported in children. The first case was in a three-year-old child. The white count dropped to 180 and the child died after an illness of three weeks. The second case was probably only an agranulocytic symptom-complex following furunculosis. This patient recovered.

DR. FREDERICK T. HILL, Waterville, Me.:

I think Dr. Seydell has brought out the question I wanted to ask Dr. Shea. The only 2 cases that I have seen were both in young children about six or seven years of age; both were fatal.

Another point about the treatment of Vincent's angina. Bearing in mind always the grave though perhaps unusual possibility of blood response to Vincent's angina, I have felt inclined this past year to treat the early cases of Vincent's angina by destroying the lesion with

diathermy rather than using any form of medication. I would like to get Dr. Shea's reaction to that. It has been fairly satisfactory to the patient.

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

Dr. Shea said that in the true agranulocytic anginas the patients were middle-aged. I have seen 2 cases in the last couple of years in young women, aged twenty-one and twenty-four years, and 1 in a child aged seven years, I think. I just wondered where his information came from.

DR. WILLIAM B. CHAMBERLIN, Cleveland, Ohio:

In this connection I would like to mention a child of one of the assistants in the clinic, a child aged ten years, who died of agranulocytic angina.

DR. SHEA (in closing):

In answer to Dr. Carmody's question as to the average age of the agranulocytic angina patients, we found that it was 32 years for females, and 37 for males in 43 reported cases in the current literature.

The reticuloendothelial system, referred to by Dr. Fenton, will be the subject of more study in the near future.

Our best treatment has been to supply the deficiency in these cases, and the deficiency is blood, and then to correct what is wrong, and that is to overcome the toxin's influence on the hematopoietic system. When the patient is infected with the toxin, he should respond normally, and when he responds normally he gets well. When the response is too great, or the toxin paralyzes the hematopoietic system, atypical blood pictures are found. If not handled carefully and with judgment, these cases are lost. Some of them are hopeless, such as Dr. Beck's case, of which I will be very glad for further information.

As to Dr. Seydell's question, Schultz, in his original article, gave icterus as one of the diagnostic symptoms, and as the disease is so often called Schultz' disease, I incorporated it in my paper. Our case had a typical jaundice.

PSAMMOMA (BRAIN-SAND TUMOR) REMOVED FROM THE ETHMOID AND SPHENOID AREA*

HENRY M. GOODYEAR, M.D.

PSAMMOMA, or "brain-sand tumors," are of sufficient rarity to justify their being placed on record. Their recognition and removal from the ethmoid area may be accomplished with considerable difficulty and hazard.

In 1867, Virchow coined the term "psammoma" (sand tumor, peculiar to the meninges of the brain and spinal cord). Piersol said they were practically normal in the adult pineal gland in the form of so-called "brain sand." They are found particularly, however, in connection with chronic inflammation and tumor formation, sometimes sarcomatous or endotheliomatous in character.

Karsner, in his *Human Pathology*, describes a psammoma as a tumor made up of many small psammatous bodies of concentrically laminated calcareous deposit, usually microscopic in size, but sometimes attaining a diameter of 2 to 3 mm.

The case which I am about to describe had a psammoma which apparently originated in the region of the cribriform plate and extended up into the left frontal region of the cerebrum and down into the nose.

When the patient was admitted upon the ear, nose and throat service at the Cincinnati General Hospital, he was complaining of severe headache and rapid loss of vision. These symptoms were of only ten days' duration. The vision was 20/30 and the field contracted.

Eight months previous to this time the patient had been on the general surgical service complaining of severe left frontal headache and gradual loss of vision for one month.

At this time Dr. George Heuer removed a tumor from the left frontal area of the cerebrum. As the tumor had extended through

* From the Ear, Nose and Throat Service of the Cincinnati General Hospital.

the dura in the region of the cribriform, a leak of spinal fluid was almost inevitable after operation. This was avoided, however, by carefully suturing a piece of fascia lata in the dura opening where it lived and no evidence of cerebrospinal rhinorrhea ever occurred.

The outer shell of the tumor was bony hard, but the inner portion could be easily carved. It was considered to be epitheliomatous in character. The vision returned eight hours after operation and there was no particular discomfort or disturbance of vision up to his present illness.

Intranasal examination revealed a hard tumor mass, apparently growing out from the septum and completely filling both sides of the nose down to a level of the inferior turbinates.

Roentgenograms showed a dense clouding of the ethmoid and sphenoid areas on both sides and a deep clouding of the left antrum.

Taking advantage of the absence of the left eye, which had been removed sixteen years before, following a traumatic injury, a wide opening was made into the nose by a Killian incision and the removal of a large portion of the frontal process of the maxilla.

Through this opening a bony tumor, which could be easily carved, was pared away. The mass extended so far laterally that the ethmoid labyrinth with the lamina papyracea was removed on both sides, as well as what remained of the cribriform plate. There was no escape of spinal fluid. The sphenoid cavities were large and completely filled with the bony growth, without any definite line of demarcation. The medial wall of the left antrum had disappeared and the sinus cavity was found to be filled by an extension of the tumor mass. The greater part of the nasal septum was removed.

Iodoform packing was used to control rather severe bleeding in the sphenoid area. The vision rapidly returned within twenty-four hours and the headaches have not recurred, nor has there been any evidence of return of the tumor tissue within the nose one year after operation.

A psammoma probably always originates from the dura, and when it extends through this structure and through the cribriform plate it is evident it would be most difficult to remove without a resulting dural fistula and loss of spinal fluid.

The preceding intracranial operation in this case, and fortunate closure of the dura by an insertion of fascia lata, made this nasal operation possible.

A psammoma in the ethmoid area might be most commonly confused with an osteoma, since osteomata seem to have some pre-

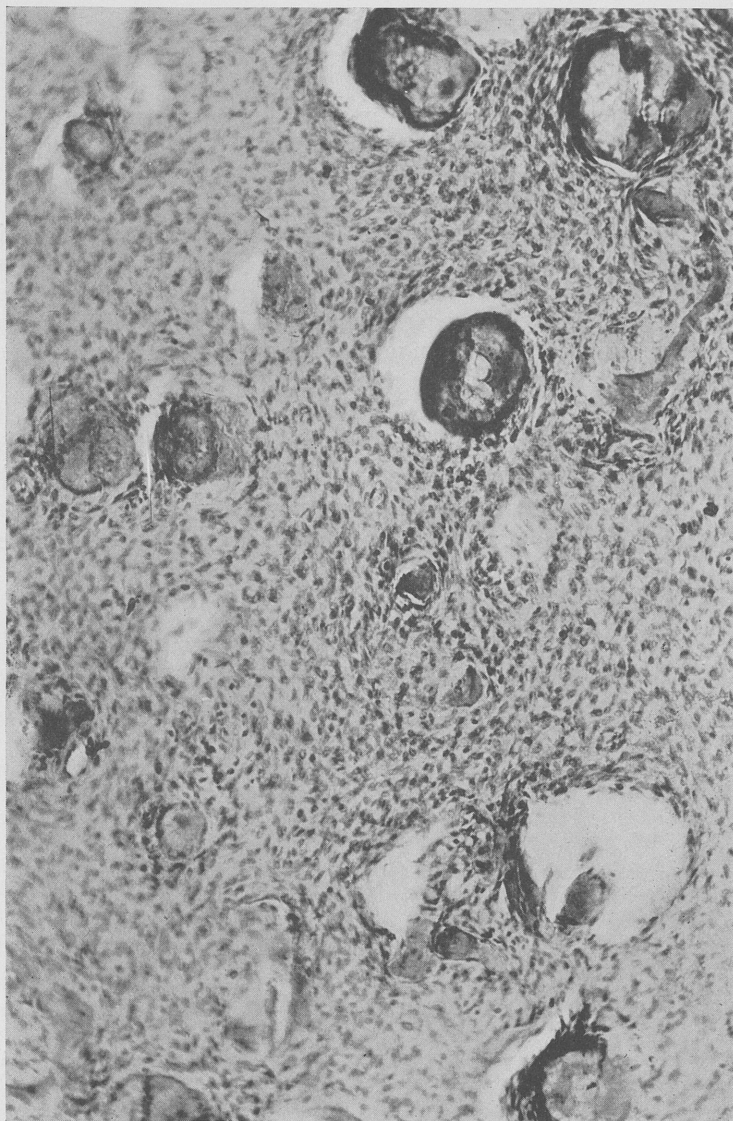


FIG. 1.—Microscopic section (low power) from tumor removed as recorded herewith showing the numerous psammomatous bodies of concentrically laminated calcareous deposit.

dilection for this region and often extend into the ethmoid area, the orbit and the frontal sinus. I reported one such case in 1924.¹

Eight weeks ago I removed a tumor, suspected to be a psammoma, from the ethmoid region in a young woman who was complaining of severe headaches and disturbance of vision.

The tumor apparently grew from the upper portion of the septum and extended through the ethmoid region and into the right orbit. By a Killian incision, the mass was completely removed, with a wide exposure of dura. Above the bony tumor mass was a large collection of pus and the floor and posterior wall of the right frontal sinus had been completely absorbed. On microscopic section the growth proved to be an osteoma rather than a psammoma.

Several years ago I operated an endothelioma in this area. Through an external opening the dura was elevated and the anterior two-thirds of the cribriform plate removed. There was no escape of spinal fluid and the softness of the tumor distinguished it grossly from a psammoma or osteoma.¹

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DISCUSSION

DR. SAMUEL IGLAUER, Cincinnati, Ohio:

I was fortunate enough to have seen this case with Dr. Goodyear at the hospital, and when I saw the x-ray pictures, considering the fact that the surgical service had operated on this man and that Dr. Heuer's first name is George, I said, "Let George do it," because it appeared to me impossible to get that tumor out without a fatal result. However, Dr. Goodyear undertook the operation, and I think he is to be congratulated on the result.

If you recall the picture, you will remember that that tumor involved the entire nasal septum and extended up into the frontal lobe, and it seemed impossible to get the mass out without tearing the dura all to pieces and setting up a meningitis.

One remarkable feature of the case was the rapid return of vision after the tumor had been removed, showing that with pressure on the optic nerve and loss of vision, by removal of the pressure the vision may immediately improve.

DR. WILLIAM V. MULLIN, Cleveland, Ohio:

As I have never seen a psammoma, I shall report another unusual case of a tumor found in the nose.

A woman, aged fifty-four years, gave a history of nasal obstruction five years before she first consulted me. At that time she had had what she thought were polyps removed from both nostrils, but she said that the operator also inserted his finger up behind her soft palate and removed something in that way. The patient was relieved for a year after this operation, and then the nasal obstruction returned. She consulted another physician, who treated the tumor with radium on four different occasions, without affording relief.

When I saw her she was very ill, with a sixth-nerve paralysis on the left side, pronounced deafness of the conduction type, and marked proptosis of both eyes, with complete loss of vision in the left eye and

almost total loss in the right eye. The oculist reported that the venous vessels of the eye showed marked obstruction, and suggested that there might be pressure on the cavernous sinus. A tumor mass filled both nostrils and the nasopharynx, and could be seen hanging below the soft palate. It had eroded the floor of the nose and caused a bulge in the roof of the mouth. Roentgenologic study of the sinuses showed that the entire sphenoid area and the sella turcica were destroyed. The pathological diagnosis of a biopsy specimen was chordoma.

Chordoma is a tumor which arises from the cellular remains of the notochord, and is, therefore, of epithelial origin. There are only about 80 cases reported in the literature, and these are described in two locations, the spheno-occipital region and the sacrococcygeal area. The average age at which these tumors develop is from thirty-five to forty years, those of the spheno-occipital group developing about ten years later than those of the sacrococcygeal group. A positive diagnosis can be made only by the use of the microscope. They always recur when removed, are slowly malignant and occasionally metastasize.

DR. RALPH A. FENTON, Portland, Oregon:

May I ask Dr. Goodyear if he and Dr. Heuer at any time considered the use of radium in this type of case, or if he found any mention of the use of radium implants, radium-containing needles or anything of that sort? On severe neoplastic growths which are by other means inoperable, those things have occasionally been used, and just in passing I would mention one case in which we gave rather massive irradiation to a sphenoid and ethmoid tumor which had somewhat similar eye symptoms (it was not this type of tumor at all, it was not osteomatous in any sense), with almost complete separation of the ethmoid capsule and opening of the sphenoid. Fortunately, he recovered without any surgery at all; we were afraid to go in because of the danger of fatal hemorrhage from the cavernous sinus.

CHAIRMAN CHAMBERLIN: Was that a fibroma?

DR. FENTON: No, it was a carcinoma, one of the slow-growing type.

DR. CHAMBERLIN:

In connection with Dr. Fenton's discussion, I might mention the use of radium in nasopharyngeal fibromata. I have seen 4 of these cases which I reported at one of the previous meetings of the American Laryngological, Rhinological and Otological Society. Strangely enough,

I have not seen any since, except in consultation. The case of Dr. Pitkins, where he used emanation in the case of nasopharyngeal fibroma, apparently was progressing favorably when he suddenly developed meningitis and died. So I mention that as one of the dangers from the use of radium in these inoperable tumors.

DR. GOODYEAR (in closing):

I would like to thank Dr. Iglauder for his discussion. It was due to his kindness and generosity that I had the opportunity to work on this patient, while on Dr. Iglauder's service.

Dr. Mullin mentioned the chordoma. I know little or nothing about them, but I am delighted to have this unusual tumor mentioned in connection with tumors of this area.

In looking up the literature, Dr. Fenton, I went pretty carefully into psammomas, but I do not recall finding anything about psammomas being treated by radium. I do not believe there was any report of such treatment. Of course, the tumor is pretty hard, and it is a question whether radium would act on a tumor as hard as a psammoma. This tumor mass was so hard it was a good deal like carving cartilage.

Your case of carcinoma surely represented a beautiful result of the use of radium.

The epithelioma that I removed from the ethmoid region and mentioned here as one operated in this area, did not have radium applied. I was very much interested in this particular case, as the tumor showed some swelling along the inner canthus and also a tumor mass in the ethmoid area. We took some sections and they were reported non-malignant, so I attempted to remove it. I found that I could expose the floor of the brain and the dura and elevate practically all the way back to the sphenoid. The cribriform plate was removed on one side, and yet the patient had no reaction afterward. In fact, when I went to the hospital the next day (the man was pretty close to seventy years of age), his bed was empty and I found him in another room, sitting beside the window. His temperature was normal, and he went along uneventfully until he died later of some metastasis.

I also appreciate Dr. Chamberlin's report in regard to fibromas.

PATHOLOGY OF ACCESSORY SINUS DISEASES

ANDREW A. EGGSTON, M.D.

THE processes involved in the production of pathological changes in the mucous membrane and bony tissues of the nose and communicating sinuses will always be a fruitful field for investigation. The pathological anatomy resulting has been variously described and has received a varied clinical terminology. In most cases of chronic sinusitis the clinical manifestation expresses altered physiology, which is the sequence of microscopically deranged histology, the exact nature of which has not been clearly defined. A careful study of surgical material from both acute and chronic sinusitis has been made for a number of years and certain changes have been so universally observed that some definite explanation of the pathology involved has been formulated. This presentation, while of a more pathological nature, will be of basic and fundamental value if these observations prove correct, and will clarify the processes involved in the production of sinus disease resulting ultimately in the clinical application of this knowledge to the treatment of these troublesome conditions.

Tissues of the maxillary, frontal, ethmoid and sphenoidal sinuses involved in inflammatory processes have been examined. The pathological changes observed were strikingly similar in character. The changes of paramount value in classification, have been of a vascular nature.

The associated pathological changes of the contiguous soft and bony tissues are secondary to the vascular changes. The tissues of the sinuses are supplied with blood by arteries and drained by veins and lymphatics, the same as other structures of the body. However, the tissues in question have a circulatory system unique in structure and physiology. The blood supply is particularly excessive and peculiarly sensitive to extrinsic and intrinsic changes. The proper circulation of fluids through these tissues varies momentarily and a suitable adjustment to altered conditions is of prime importance. Therefore, any pathology of the vascular structures

should be carefully noted. The lesions observed in the tissues from the sinuses have been so universal that an attempt to explain the changes in the adjacent structures have been made with the vascular system as a basis.

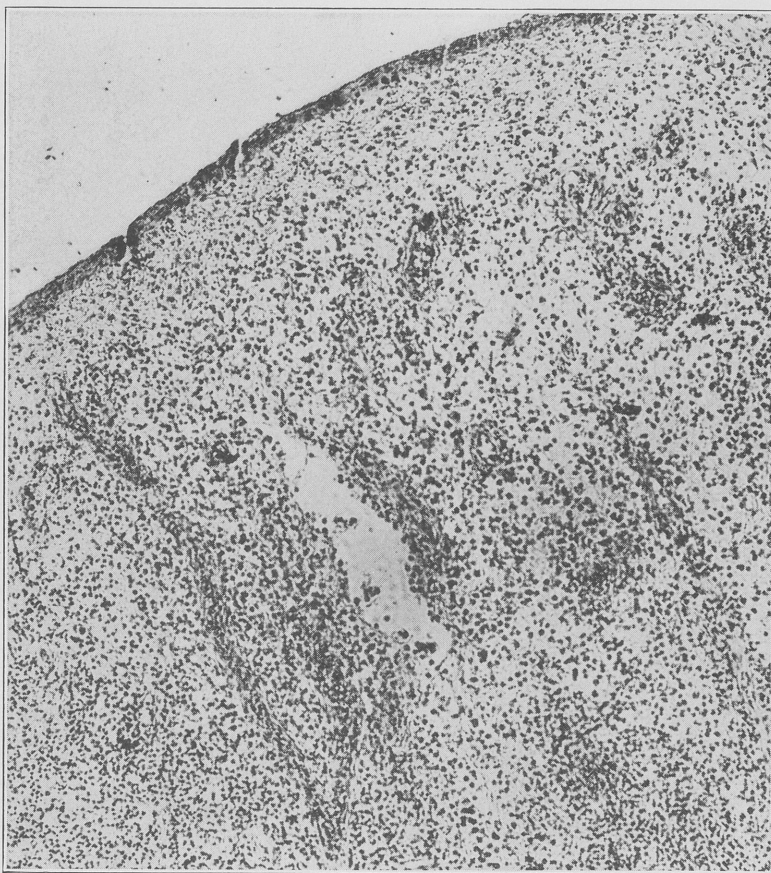


FIG. 1.—Section of polypoid tissue showing thin mucous membrane, edema and diffuse inflammatory reactions of tunica propria. The perivascular concentration of cells is well illustrated both in cross and longitudinal sections of the bloodvessels.

The type of pathology observed permits of a classification which is practical as a working basis, but for exact presentation must

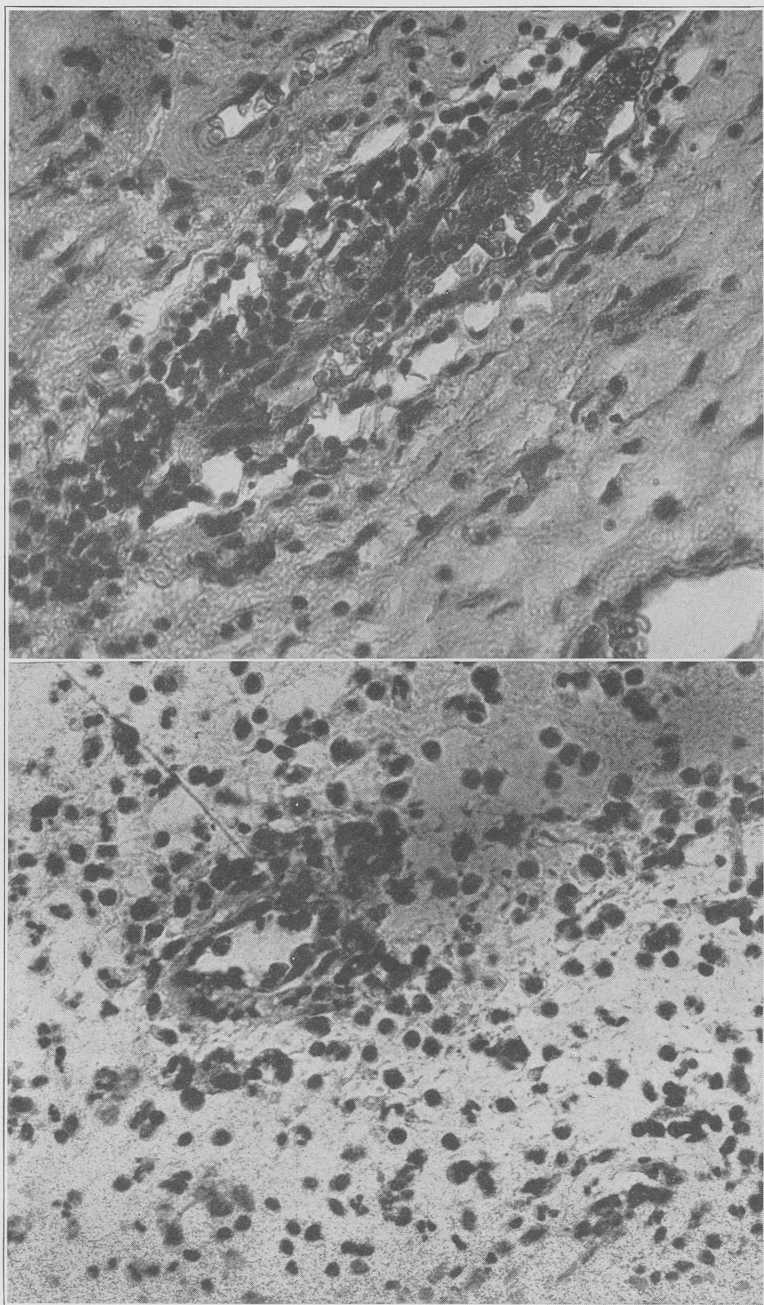


FIG. 2.—A larger magnification of the cross-section of a blood-vessel of a polyp showing perivascular infiltration by mononuclear and polynuclear cells.

FIG. 3.—Longitudinal section of a vein in a polypoid sinusitis showing perivascular infiltration with monocytes. The edema of the stroma is also shown.

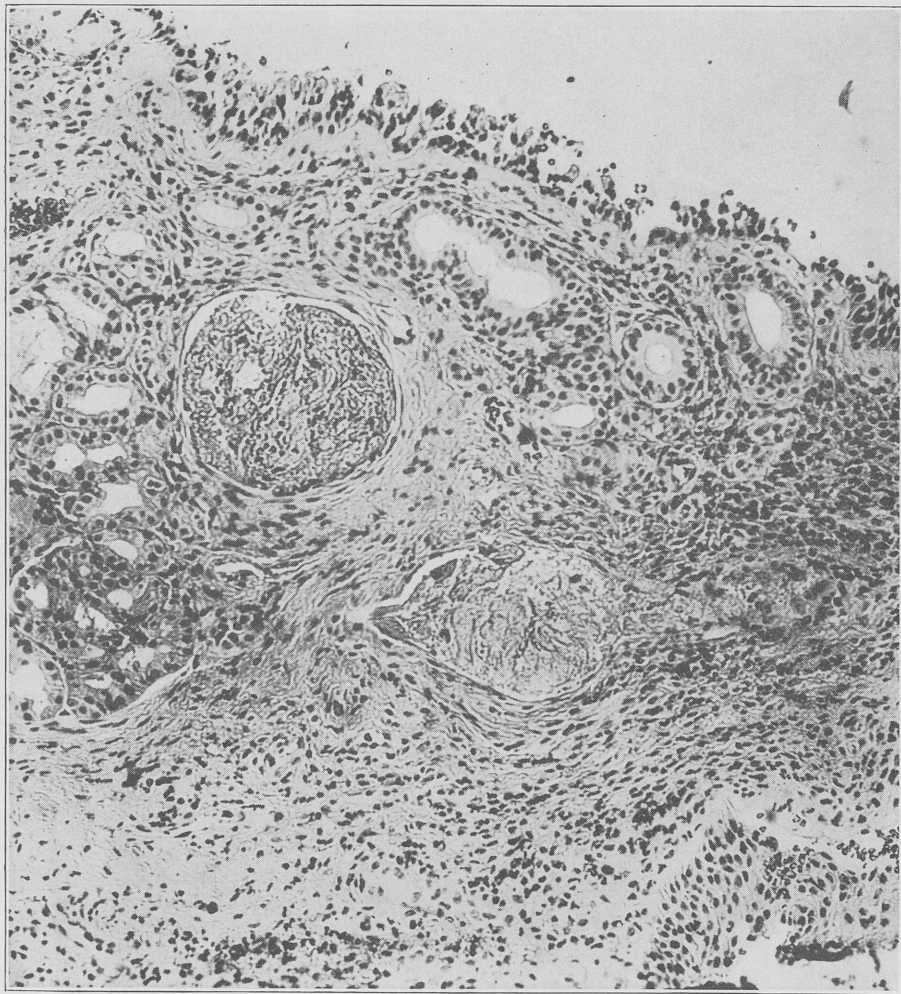


FIG. 4.—Section of antrum mucosa showing epithelial desquamation with some metaplasia in the right lower region. Also early stages of thrombosis are shown in the two bloodvessels. The upper one, cut in cross-section, is more advanced and shows beginning organization. There is perivascular inflammatory reaction which is also evident in the stroma generally.

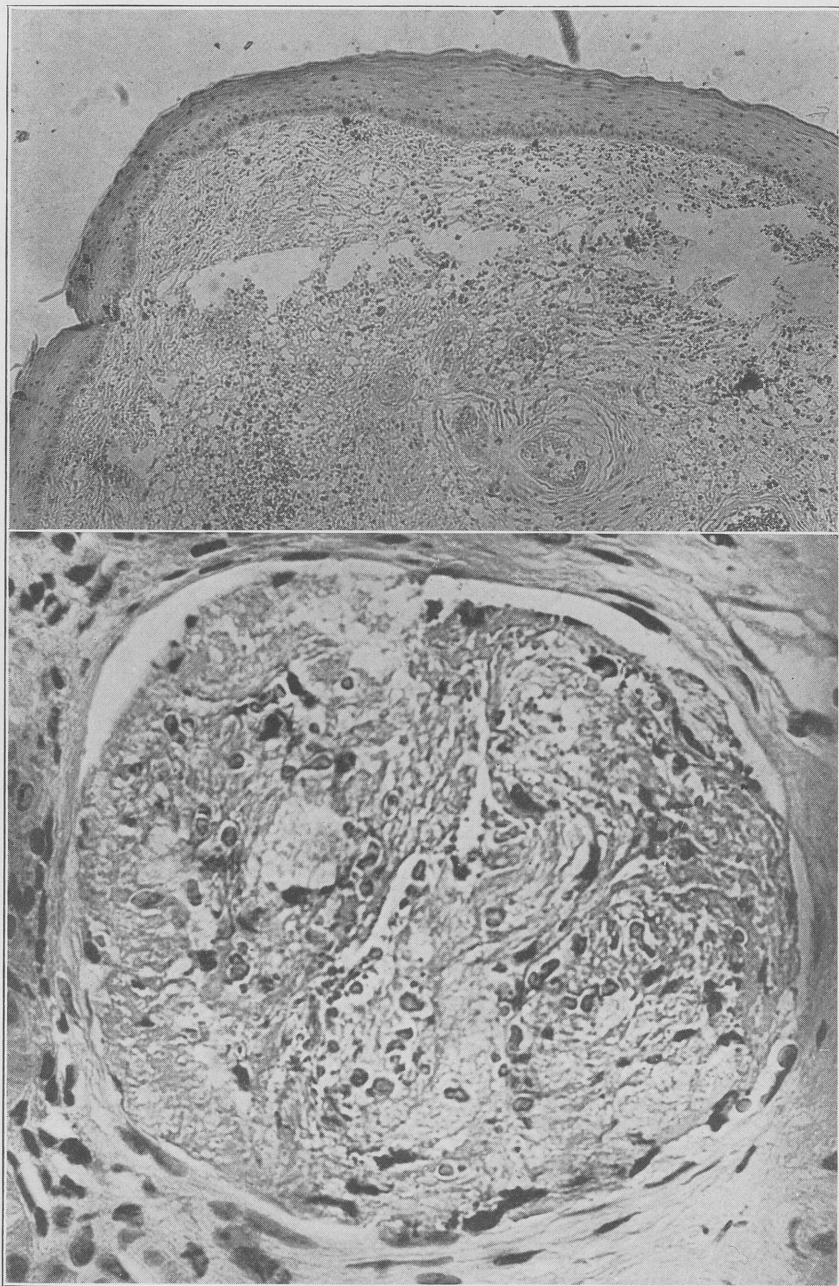


FIG. 6.—A section of polyp from the vocal cord which shows several chronic varicosities with thrombi, a finding so characteristic of polypi of the larynx and other regions of the body and easily demonstrated in hemorrhoids. The surface mucosa in this region shows beginning thinning due to edema and pressure in the submucous tissues.

FIG. 5.—Higher magnification of thrombosis shown in Fig. 4, showing invasion of the clot and fibrin by fibroblasts in the early stages of organization.

necessarily present the subject in a more abstract manner than is possible in the clinical classification of patients. However, most cases will fall rather loosely into two distinct groups and a simultaneous association of these permit of a combined type.

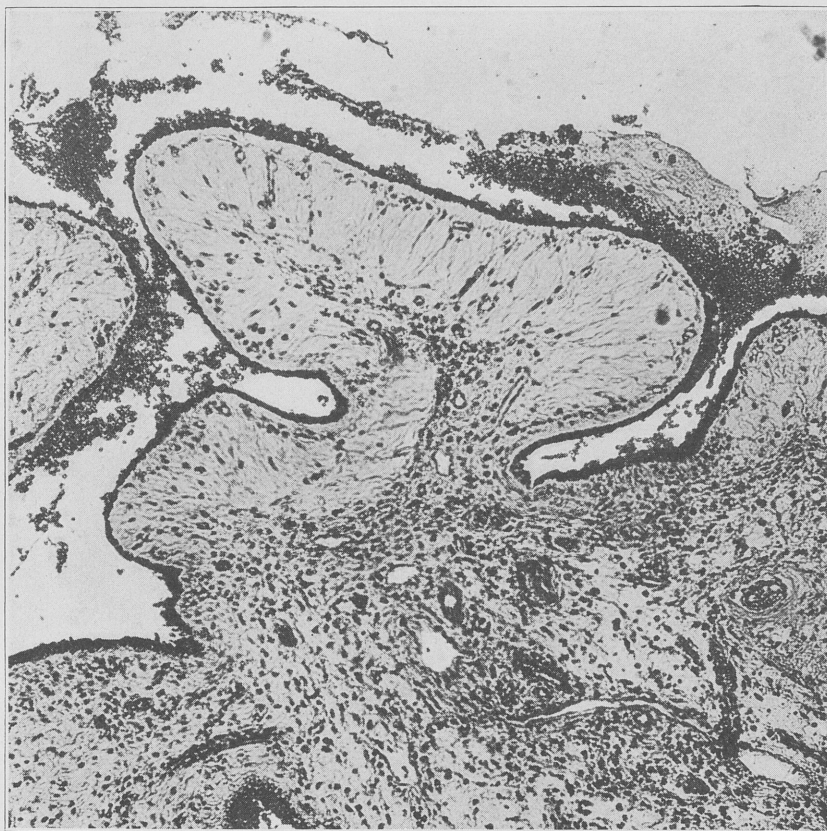


FIG. 7.—Polypoid hyperplastic ethmoidal sinusitis showing small or young polypi. The mucosa is atrophic and metaplastic. Some blood and lymph vessels are dilated. Some are thrombotic and show marked perivascular cellular infiltration.

The pathological histology of acute sinusitis reveals practically the same vascular and cellular reaction of the soft tissue. Briefly, these changes are, dilated and congested bloodvessels with cellular

stasis, migration of leukocytes into the tissue, the appearance of lymphocytes, plasma cells and other cellular forms in the walls of



FIG. 8.—Early polypoid ethmoid sinusitis with metaplastic mucosa and some of the mucosa showing active secretion, revealed by the many goblet cells present. There is edema of the stroma with dilated vessels. Several veins are sectioned longitudinally in the tunica propria showing marked phlebitis.

the vessels and the immediate perivascular tissue, associated with diffusion of serum into the adjacent structures. These reactions are primarily in the walls of the bloodvessels and perivascular

spaces and secondarily in the soft tissues. The acute inflammatory changes disappear as the irritation subsides, but it is a pathological axiom that every inflammatory process, however small, leaves a

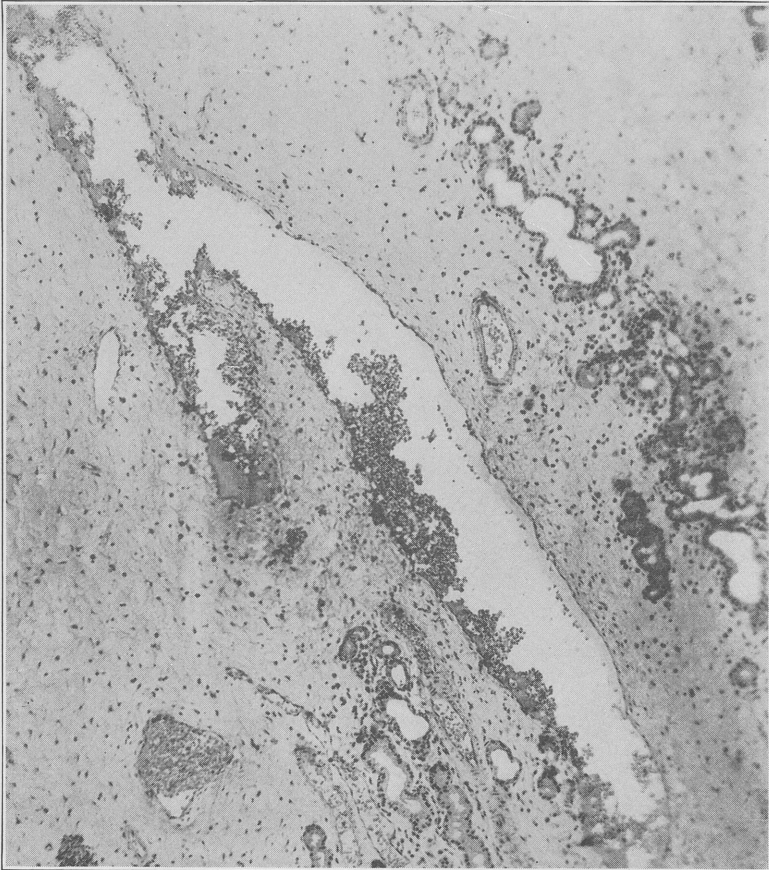


FIG. 9.—Shows a polypoid mucous membrane with dilated veins and lymphatics and some edema and lymphocytic infiltration. Some of the glands are atrophic and cystic.

disturbed tissue relationship, usually in the form of an increased fibrous tissue content. It is also known that the fibrosis leaves the tissue more sensitive to subsequent attacks. Repeated infec-



FIG. 10.—Markedly cystic mucous glands frequently found in polypoid tissue. The glands are filled with secretion which is pent up due to obstruction of the outlet channels.

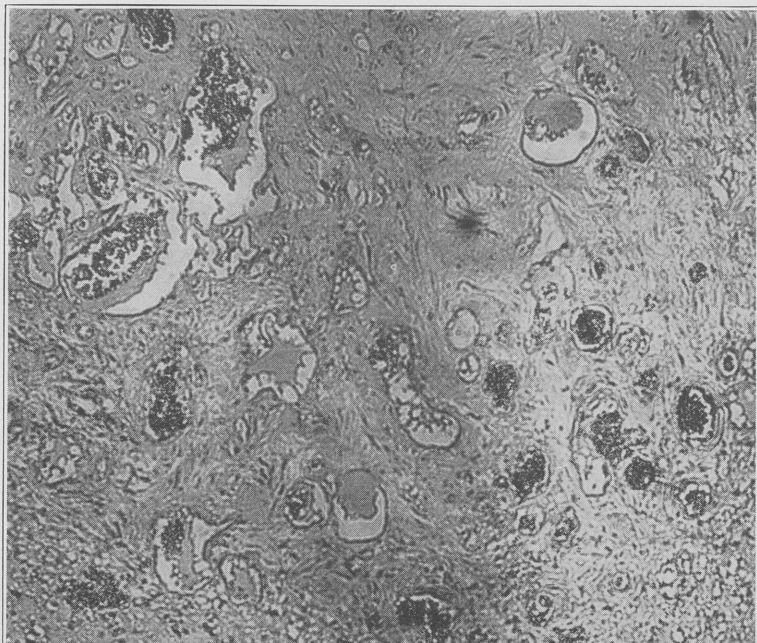


FIG. 11.—Dilated veins and lymphatics with blood stasis in a nasal polyp. The dilated channels are probably proximal to the obstructed zones of the vessels.

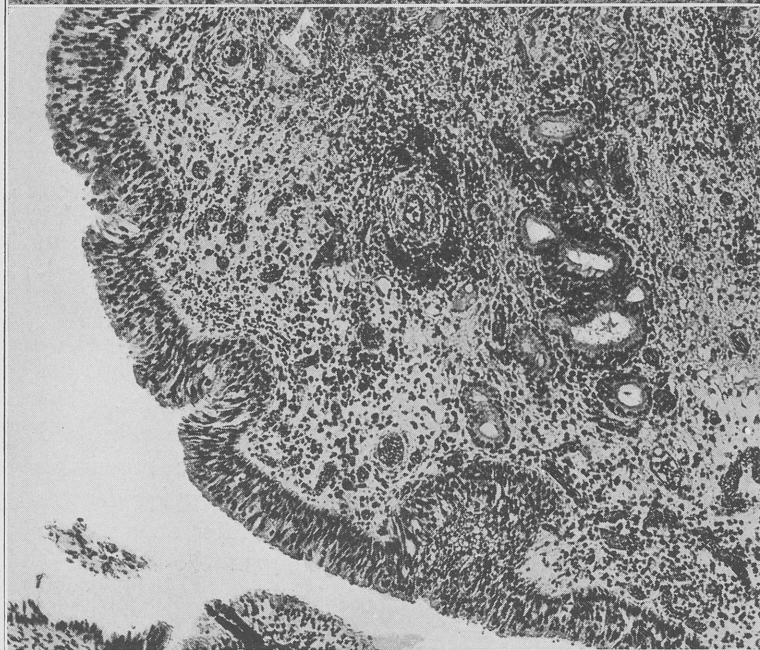


FIG. 12.—Mucosa of nasal sinus shows thickened epithelium with diffuse mononuclear reaction of the tunica propria. The marked perivascular mantelling with mononuclear cells is strikingly illustrated.

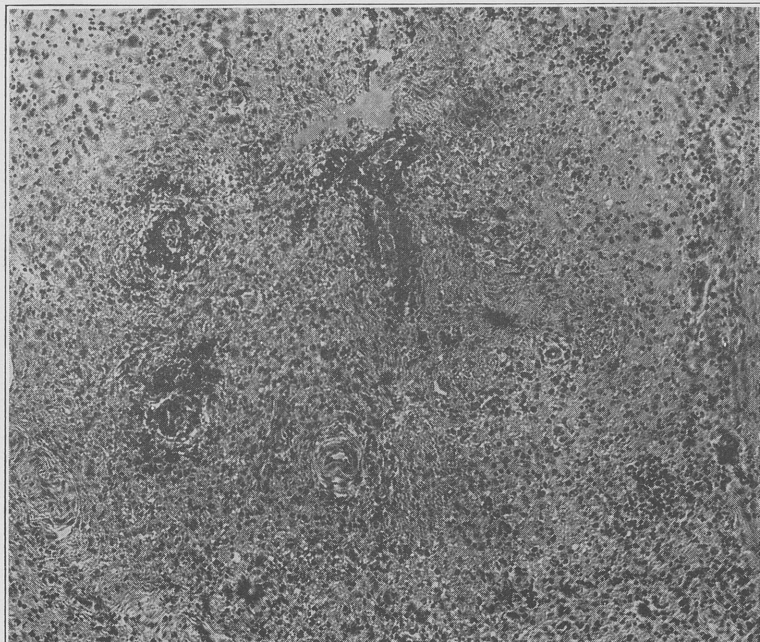


FIG. 13.—Another case of chronic fibrotic sinusitis showing periarthritis with chronic fibrosis of some of the vessels.

tions and inflammation ultimately produce fibrous changes of great pathological importance. If the chief change in acute inflammation is vascular, it logically follows that the greatest chronic changes are also of a vascular nature. The distribution of the changes may be arterial, venous or lymphatic. The type of chronic pathological changes found in the soft and bony tissues depends upon which of the vascular channels are involved.

The acute processes are not easily demonstrated, because they rarely demand surgical treatment and therefore a scarcity of material exists for study. However, enough of the changes have come under our observation to demonstrate the pathological processes responsible for the gross changes found in the terminal stages. These terminal stages are the types with which this presentation is concerned:

1. Hypertrophic or polypoid, characterized by thickened and edematous changes in the mucous membrane and periosteum, usually associated with polypoid masses of the soft tissue and rarefaction and osteoporosis of the bone.

2. Atrophic, fibrotic, sclerotic or arteriosclerotic, characterized by an increase in the fibrous connective tissue in the stroma, thinning of mucous membrane, metaplasia of surface epithelium, associated with thickening of the periosteum and a condensing osteitis.

3. The third type results from a combination of the hypertrophic and fibrotic changes in the same patient, or even in the same sinus cavity. This type is characterized by rugæ and sulci, with the production of a papillated type of membrane.

Hypertrophic sinusitis is the most common variety and is characterized by increase in the volume of the mucous membrane. In the earlier stages there is an increase in the thickness of the epithelial layers with some infiltration by leukocytes, chiefly lymphocytes. The stroma shows edema with marked infiltration of mononuclear wandering cells, consisting chiefly of lymphocytes and plasma cells, and in many instances, eosinophiles. The cellular reaction is most marked in the walls of the veins and lymph channels and the immediately adjacent stroma, presenting a definite picture of phlebitis, lymphangitis, with varying degrees of periphlebitis and perilymphangitis. In the later stages there is marked edema of the stroma with filling of the interstitial

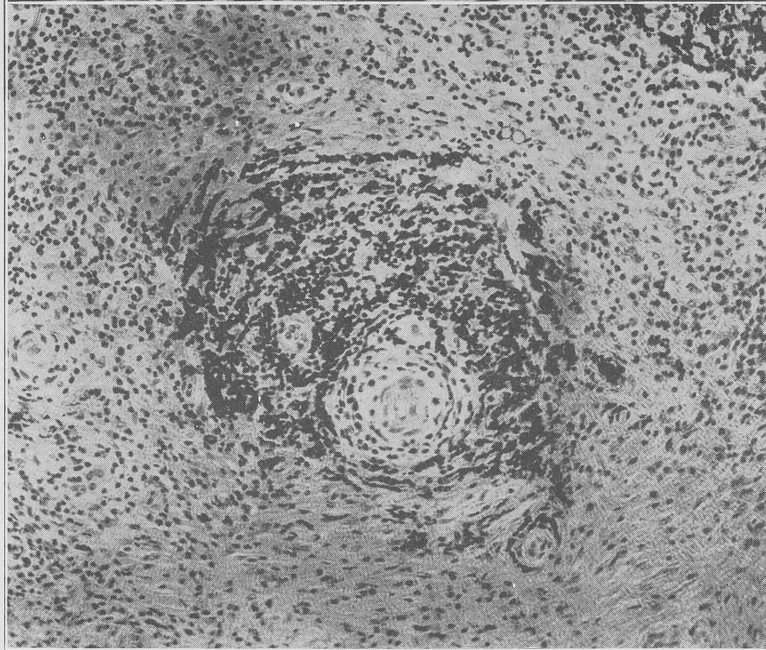


FIG. 14.—Higher magnification of one of the vessels shown in Fig. 13. The perivascular inflammatory infiltration of lymphocytes is characteristic.

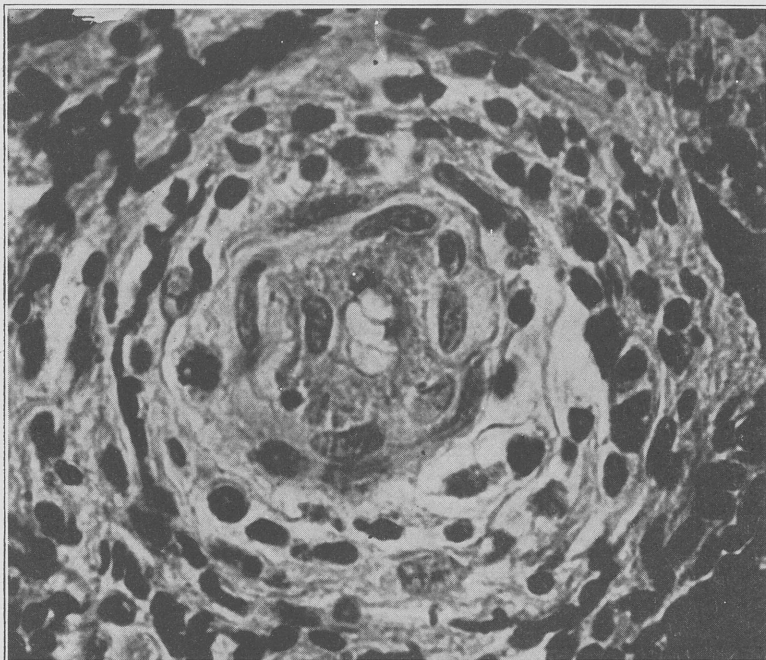


FIG. 15.—A very high magnification of cross section of a small arteriole showing in addition to the perivascular mantle of monocytes a marked proliferation of the endothelial cells of the intima of the vessel.

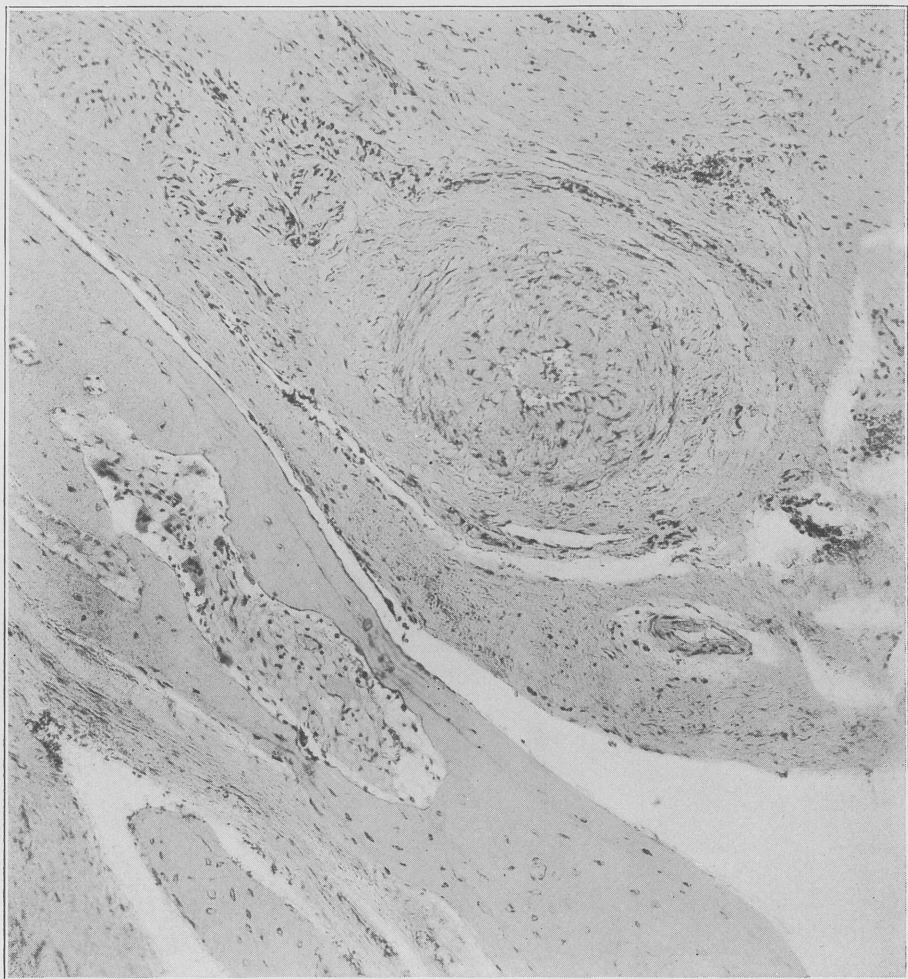


FIG. 16.—Shows two arterioles with marked fibrosis and thickened vessel walls. The larger shows also a marked endarteritis with fracture of the elastic lamina and fibrous connective tissue replacement. Areas of inflammation are noticeable in the stroma adjacent to the vessels. There are many osteoclastic giant cells in the lake of the trabecula of bone near the left of the picture.

spaces with fluid and separation of the fibrous tissue fibrillæ. There is an increase in the quantity of fibrous tissue with a decrease in the amount of elastic tissue in the mucous membrane. This turgescence of tissue continues with finally a production of polypoid masses. The surface epithelium becomes thin due to the pressure. The mucous glands show cystic degeneration due to obstruction of the excretory ducts from the pressure of the stroma. The vascular changes consist of inflammatory infiltration of the veins and lymphatics followed by an increase in the fibrous tissue which replaces the smooth muscle and elastic tissue, consequently reducing the resiliency and contractility of these structures. These changes produce partial or complete obstruction of the venous and lymphatic channels, resulting in the interference of the drainage of the areas supplied. This increased stagnation of the fluids results in further extravasation into the fibrous tissue spaces increasing the tendency to polypoid hypertrophy. In some instances actual thrombi can be demonstrated in the veins producing complete obstruction. As the result of localized thrombi there will result dilatation of the vessels proximal to the obstruction, which makes more difficult the true interpretation of the processes involved. The bloodvessels in nearly all instances show varying stages of inflammatory process, representing acute, sub-acute and chronic changes. This manner of producing polypoid changes in the nose is similar to the processes involved in the production of polyps in the larynx, cervix, intestinal tract and rectum. Hemorrhoids are polypoid masses containing thrombotic and dilated veins with edema. As a result of venous and lymphatic obstruction, there will be changes in the soft tissues and involvement of the periosteum and bone. The periosteum becomes broad and edematous and its fibers are widely separated by fluid and, ultimately, the bony structures will show signs of absorption and osteoporosis. The bone may become necrotic and sequestered, and, in some instances, septa or plates will disappear; in other places bony cysts, spurs and deviations may result. The normal growth of bone is due to the activity of osteoblasts which have the faculty of absorbing and depositing bony salts from the circulating fluids. If the circulation is interfered with bony absorption may replace formation. Giant cells, so-called osteoclasts, osteoblasts or wandering cells appear in great numbers.

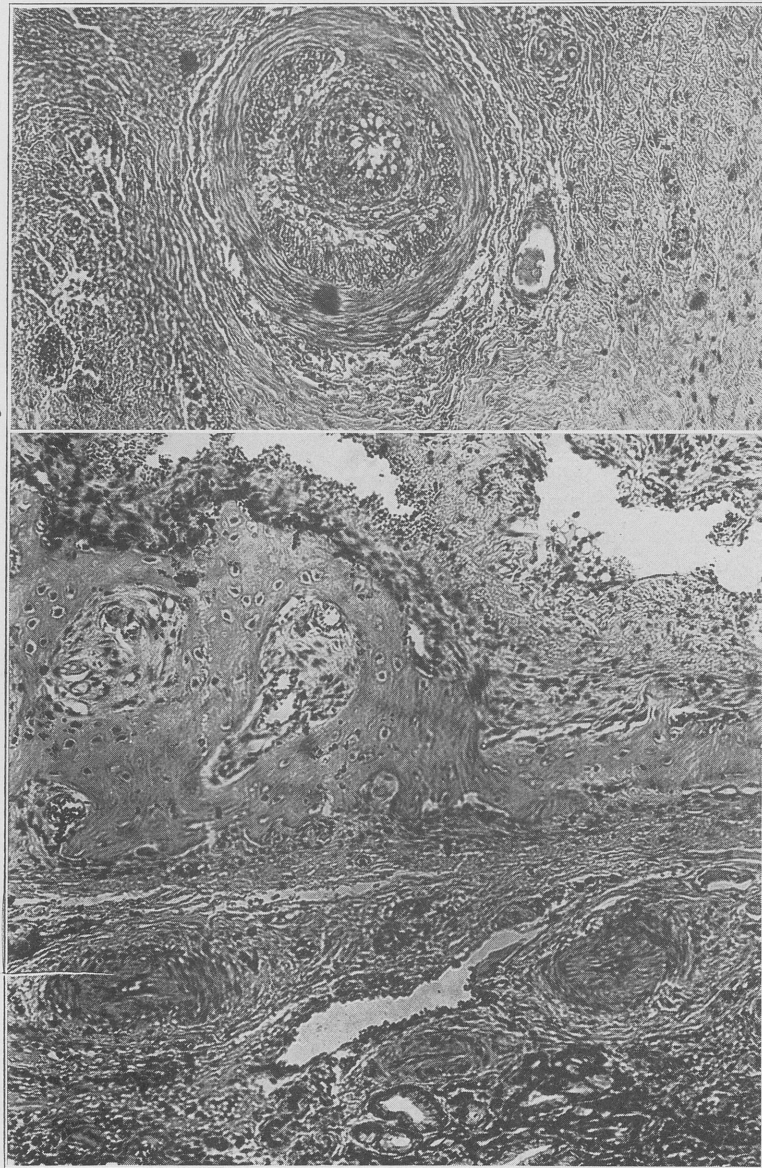


FIG. 17.—Marked arterial fibrosis with obliteration of the canals. Marked periostitis with evidence of osteogenesis and thickening of the bony structures. The periosteum shows monocyte reaction.

FIG. 18.—Higher magnification of an arteriole in chronic fibrous sinusitis showing complete disarrangement of muscular coats and of the elastic lamina due to the marked increase in fibrous tissue.

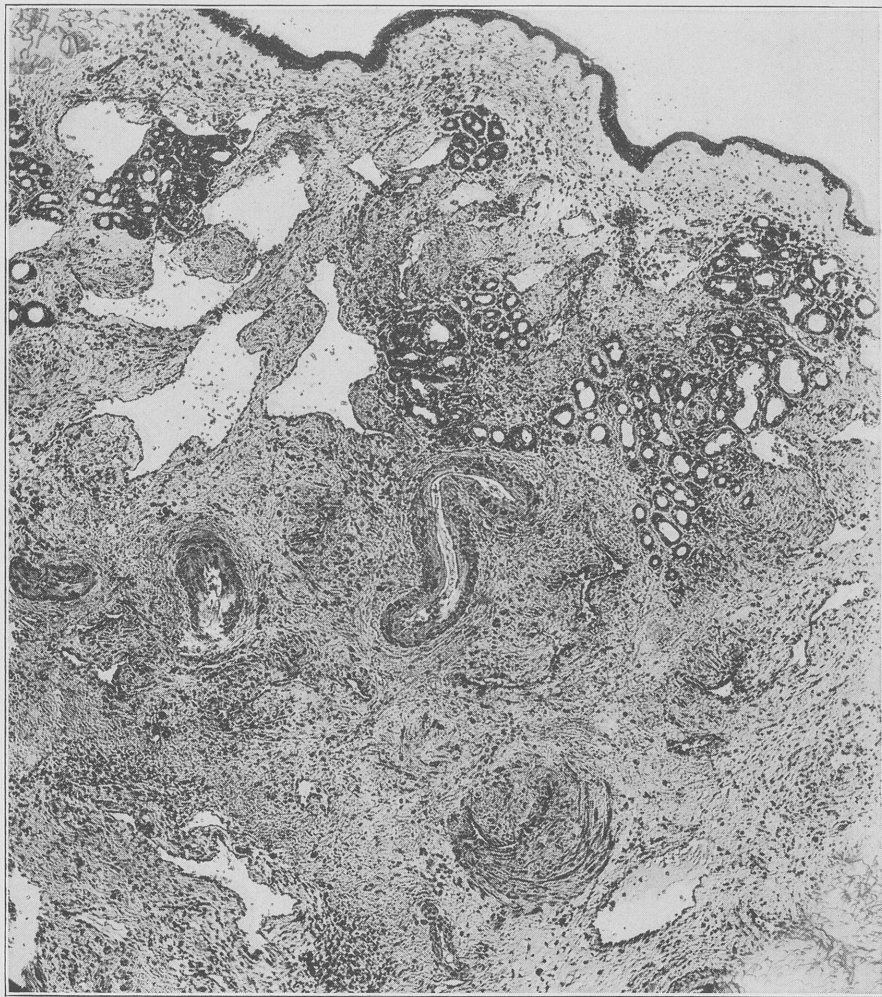


FIG. 19.—An advanced stage of chronic fibrotic sinusitis characterized by metaplasia of the surface epithelium into squamous instead of columnar cells. The tunica propria shows diffuse fibrosis. The arterioles are sclerotic and obliterated. The lymph and venous channels are dilated. The secretory glands are atrophic.

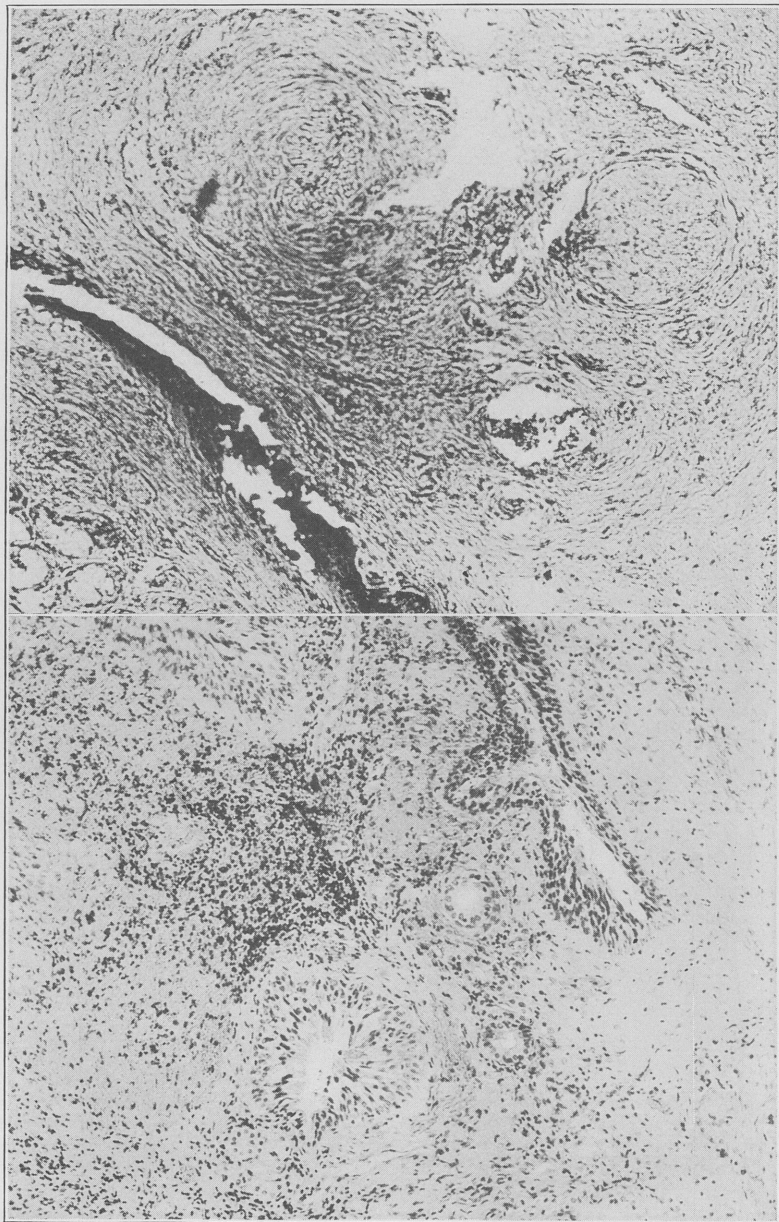


FIG. 20.—A higher magnification showing an early stage in the metaplasia of the surface epithelium.

FIG. 21.—An advanced and sclerotic stage of arteriole fibrosis with destructive changes in the bony trabecula.

The atrophic or fibrous type of sinusitis presents a picture which is an exact counterpart of the hypertrophic type. A decrease in the volume of tissue is characteristic, however there may not be

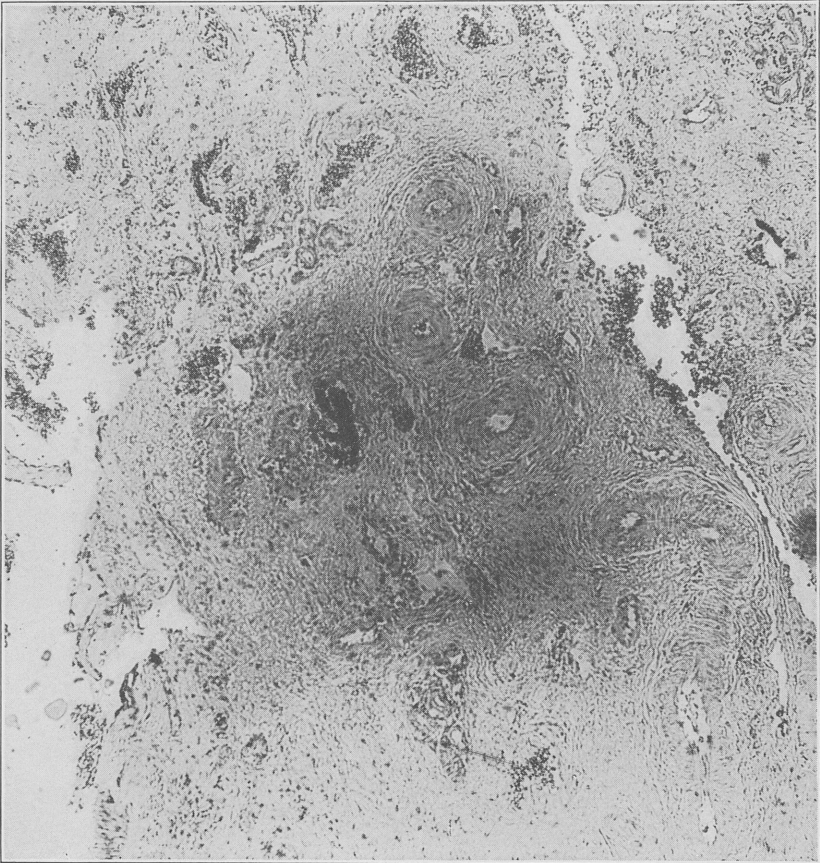


FIG. 22.—Tissues of chronic sinusitis showing an involvement of all the blood-vessels, the veins being affected as well as the arteries. The tunica propria shows diffuse fibrosis.

a gross reduction in the thickness of the membrane, depending upon the volume of scar tissue present. In the earlier stages there is very little atrophy, but in the later stages marked reduction of the soft tissue and bony structures occurs.

The fibrotic type in its most advanced stage simulates the changes found in ozena, with a loss of secretion, crust formation and putrid odor, but there are many cases that never attain this

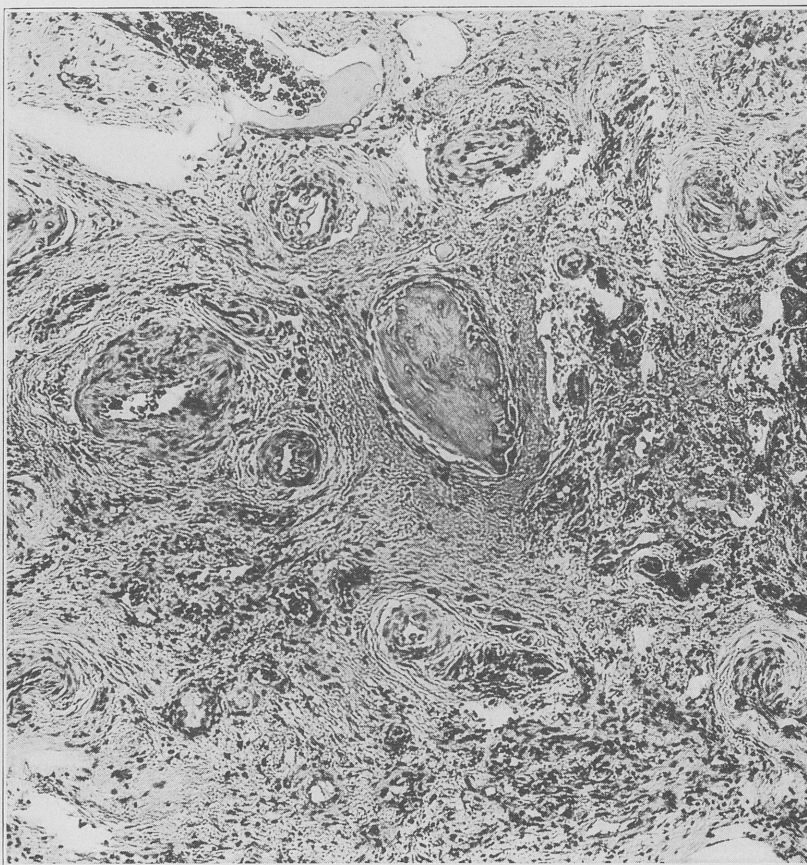


FIG. 23.—Another case showing fibrous involvement of all the blood channels. There is a diffuse fibrosis and periostitis present.

stage of severity. Undoubtedly, the processes involved are of a similar nature. In the milder cases of fibrosis there is no necrosis of tissue and consequently no actual crust formation, but putrefactive odor is usually absent in the early stages. Microscopically,

there is considerable metaplasia of the surface epithelium with desquamation. In some areas the epithelium of the mucous membrane may be thickened, forming papillæ, while in other localities it may be thin and becomes of a squamous nature. There is usually a marked condensation of the submucous stroma characterized by dense bands of fibrous tissue. In the early stages this is fibroblastic, but later shows marked hyaline degeneration. This is particularly noticeable in the submucous membrane of the surface epithelium and around the glands. The secretory glands are usually small, atrophic and inactive. If the afferent ducts are obstructed they become cystic. Cellular reaction of the stroma usually are lymphocytes, plasma cells and other wandering monocytes. There is, in general, a greater simplicity of the variety of the cells than is found in the hypertrophic type. The most striking changes occur in and around the afferent bloodvessels in contrast to the efferent or drainage vessels in the polypoid type. The vessels involved in fibrotic sinusitis are the arteries and arterioles which supply nourishment and not the veins and lymphatics which drain the areas as in the case of the hypertrophic type. Usually endarteritis, arteritis or periarteritis can be demonstrated. In the earlier stage there is considerable cellular reaction around the vessels and an increase in the fibrous tissue. In the later stages the vessel walls are distinctly thickened. The lumen of the vessels is decreased and in some instances arteriosclerotic changes with complete occlusion of the vessels occur. There is degeneration of the elastic fibers and smooth muscles in the walls of the vessels which interferes with the resiliency and elasticity of the structures. The veins and lymphatics show decreased fluid content and in some instances are collapsed. There is irregularity of the bone, due to perichondric and periosteal inflammation, resulting in alternating areas of cartilage and bone formation interspersed by areas of absorption and, in the more acute processes, necrosis of the bone. If the arteriole vascular involvement is of a relatively subacute nature, so characteristic of syphilis, there will be marked necrosis of the soft tissue and bone. In atrophic rhinitis the process is of a more chronic nature and necrosis occurs only in the advanced stages.



FIG. 24.—Bony changes which illustrate a condensing osteitis with thickening of the trabeculae, a process which may occur simultaneously with osteoporosis in the same patient. The osteoblasts are shown around the margin of the trabeculae of bone.

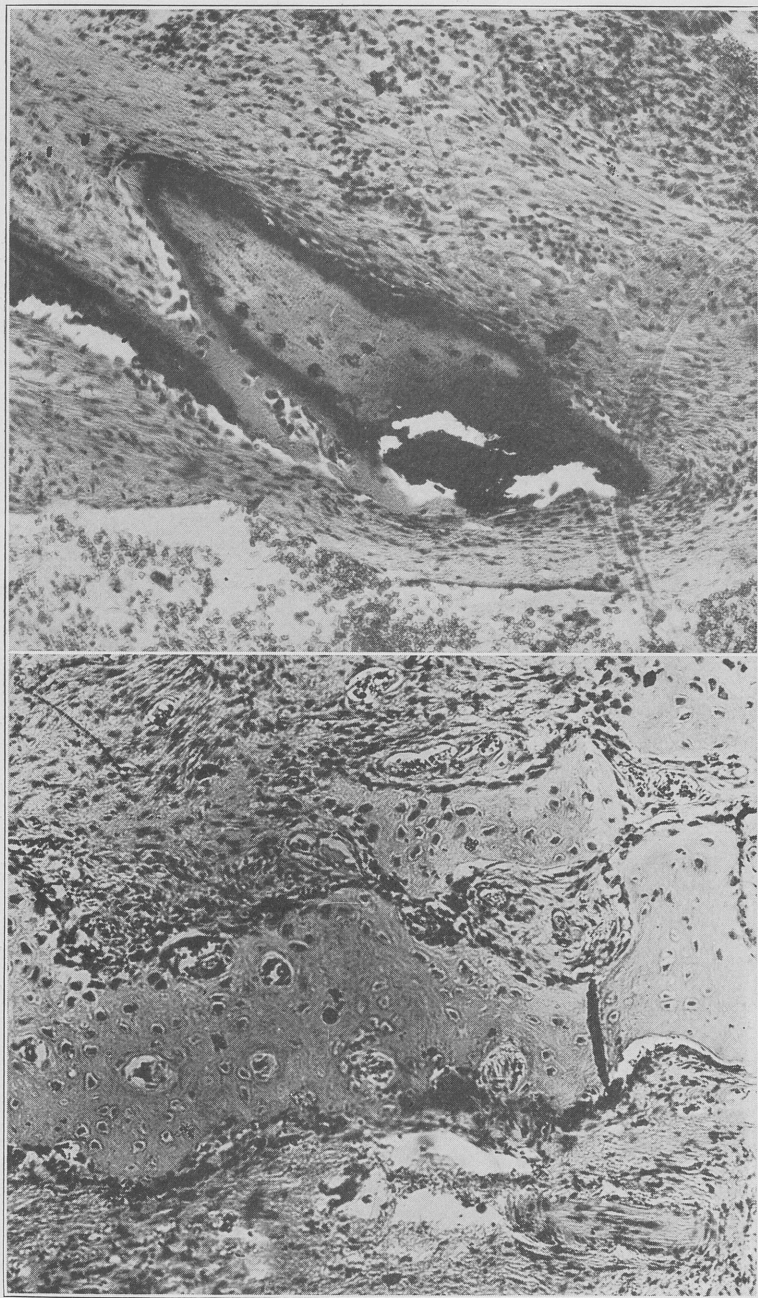


FIG. 26.—Bony trabeculae showing precipitation of the calcium salts and loss of histological bone.

FIG. 25.—Illustration of the periostitis and thickening of the bone trabeculae. A process usually associated with a chronic fibrotic sinusitis.

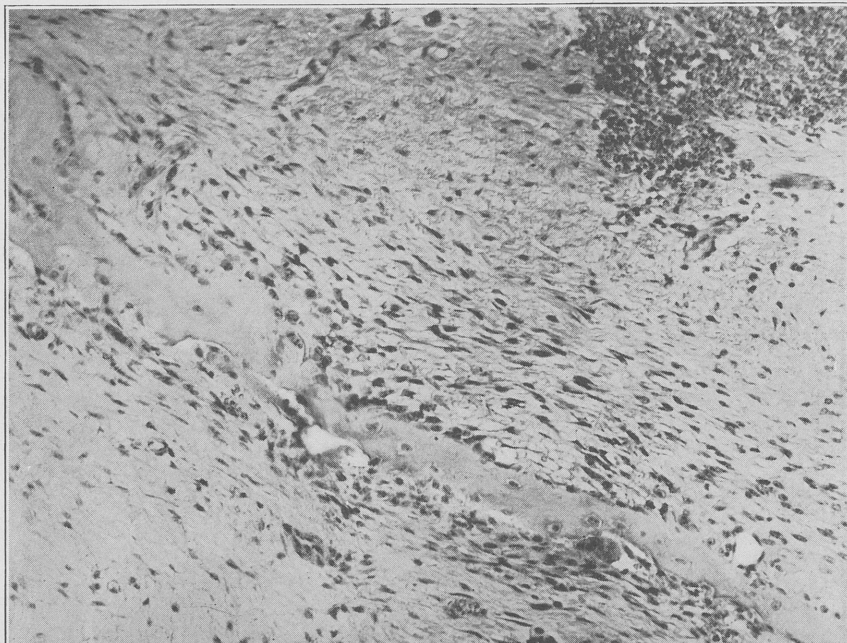


FIG. 28.—A spicule of bone showing osteoclasts on the left side and osteoblasts on the right side, probably illustrating both

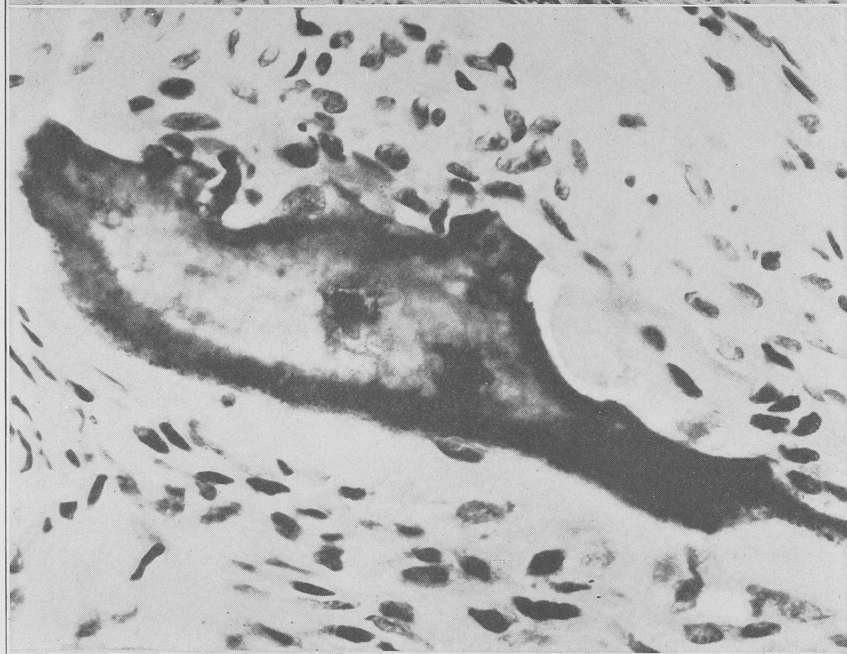


FIG. 27.—Trabecula of bone showing osteoblastic absorption near the lower part of the picture. The osteoclastic giant cell

The third, or combined types of chronic sinusitis, consists of an involvement of both the afferent and efferent vascular channels.

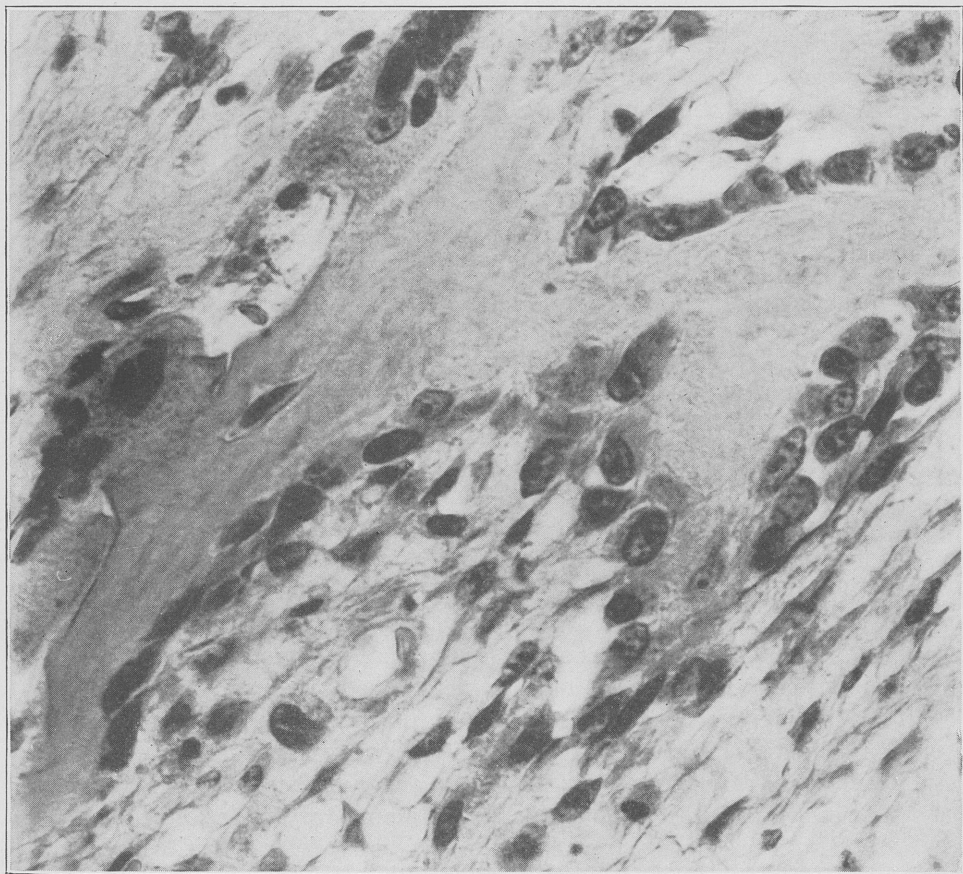


FIG. 29.—A higher magnification illustrating an osteoclast of the left side of the bone. One cell is seen near the upper margin of the bone and the other near the left hand margin of the picture. The peninsula of bone extending to the right illustrates osteogenic cells.

Arterioles, as well as the venules and lymphatics, are involved in adjacent areas of the mucous membrane. The surface of the membrane reveals a rather nodular or trabeculated appearance

produced by alternate areas of edema and hypertrophy, interspersed by fibrotic or atrophic zones. As the fibrous tissue contracts the hypertrophic nodules become more apparent, forming rugæ and sulci, resulting in a papillated topography. In the maxillary sinus it is not uncommon to find rather large areas of hypertrophy and polyps, while other areas show atrophy. This variety of pathology is particularly difficult to classify because the tissue removed may be from a hypertrophic or atrophic area, and unless the source of tissue is carefully correlated with the pathology the processes involved may be erroneously interpreted. The bony changes in this type are of an irregular nature. In some places there is osteoporosis and softening with absorption, while in others, there is otitis and exostoses. In many instances, in spite of the areas of atrophy, there is a general redundancy of fibrous tissue and therefore the whole membrane appears thickened. The microscopic character depends upon the combined involvement of the arteries, veins and lymphatics. There will be present arteritis, phlebitis and lymphangitis, with perivascular involvement of the adjacent structures and the secondary tissue changes characteristic of both atrophic and hypertrophic nature.

The vascular basis of the morbid changes in sinusitis receives therapeutic support in the employment by clinicians of drugs which directly affect the vascular system. It is also true that no tissue in the body is more prone to vascular changes from extrinsic and intrinsic causes than the mucous membrane in the nasal cavities.

The physiological duties which the mucous membrane of the nose subserves has no analogy in any other structure of the body. This is particularly true in relation to the quantity of blood which passes through this region daily, with momentary variation in the quantity as the caliber of the bloodvessels changes with every variation in the temperature, humidity or other atmospheric stimuli. Therefore, the slightest tissue alteration that influences the resiliency and contractility of the vessels will produce altered function.

The understanding of the pathological histology of chronic sinusitis must be based upon the correlated studies of the clinical studies of the clinician, roentgenologist and pathologist. A thorough correlation of the findings is of most importance in under-

standing of the processes involved. The anatomy and histology of the nose is so highly complicated and there is so much mutilation of the tissue with a loss of relationship when tissues are surgically removed that it is very difficult to visualize the sequence of events in the formation of the morbid anatomy. It is therefore of great importance that the exact source of the tissue be known if an accurate pathological interpretation is possible.

These observations are presented in a spirit of investigation and are certainly not meant to be the final word in the explanation of so complicated a condition as chronic sinusitis. Thorough investigation convinces me that the study here presented constitutes a new angle of approach and I trust will serve a useful purpose in the treatment of these cases.

Acknowledgement and thanks are hereby extended to Dr. Lewis A. Coffin for his stimulating interest and helpful suggestions in these investigations.

DISCUSSION

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

I think this presentation will be of greatest interest to Dr. Law and to myself, because, since the American Board has been functioning for the examination of candidates in the department of pathology, it has been a struggle with the candidate coming in for an examination when he had not had any instruction along this line.

There has not been a great deal of work done along the line of histological pathology in nose and throat conditions, and the presentation made here today, I think, will stimulate many men to do this type of work.

The presentation of histological specimens on black and white as presented here is of value, to be sure, but no comparison to when we have the tinted specimens. They are of much more value.

The interesting point that the Doctor continually stressed is the pathology (I am talking now from a clinical and therapeutic standpoint), the vascular changes, the degenerative changes that are secondary to the vascular changes, and he said that in syphilis this is so common. That is, of course, the characteristic thing, endovascular and perivascular changes in lues, and from the specimens that were shown one would infer that there was a great deal of that type of

disease, if it is to be inferred that endovascular and perivascular tissue is present.

I would like to know if these specimens are the routine specimens from postoperative work. Are they from the antrum or the ethmoid? Perhaps I should say the spicules from the ethmoid region.

Also, the question of his strictness about getting these specimens from the clinics, and the length of time before he got them in his hands for preparation, because you must consider a good many changes in specimens from manipulations as well as from atmospheric changes, and dry specimens lying around the operating room a day or two are often sent to the laboratory.

As I say, there is much to be discussed about the details of this paper. We are not here for that purpose, but surely the minute pathology is important in a discussion of the subject.

DR. RALPH A. FENTON, Portland, Oregon:

May I ask the essayist if in his judgment the occurrence of a perivascular infiltration may not often be an indication of a defense reaction rather than of a process already degenerative? It would seem to us, in relatively acute cases, that perivascular infiltration is an evidence of defense rather than of a change which will result in fibrosis or in permanent change of the membrane; it is a step toward repair. Of course it was difficult in the rapid sequence of the slides to be sure which process was being referred to, and I would like to have that clarified in the final statement.

DR. FRANK R. SPENCER, Boulder, Colo.:

I would like to ask the essayist if he will tell us whether some of these changes account for the fact that a small percentage of sinus cases in the hands of all operators have to be reoperated, and if it is the bone changes which are responsible for continuation of the sinus disease?

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

I understood that you said the osteoblasts, when they cannot get their proper supply of calcium from the blood, owing to the existence of inflammation, take the calcium from the bone and, therefore, osteoblasts become osteoclasts in a certain negative phase. Do the osteoclasts ever get to be active and keep on doing it even if one gets more calcium in his blood? Do they become permanently active absorbents of bone?

DR. FRANK B. KISTNER, Portland, Oregon:

I understood Dr. Beck to say that he considered the perivascular infiltration suspicious of lues. In our own investigation we have frequently suspected that it was luetic, but we have many, many cases in which by no diagnostic means could we pin lues on the individual clinically. I think the perivascular fibrosis is quite characteristic of a long-standing infection whether it be a purulent sinusitis or a non-purulent sinusitis.

DR. E. ROSS FAULKNER, New York City:

I want to commend Dr. Eggston for his very careful piece of work. All these things add a little to our knowledge. Even when they do not add any new knowledge they corroborate old findings and make us a little more certain of what is going on. It is very interesting that the microscopic findings in these things correspond very accurately to your gross findings.

After all, inflammatory processes everywhere are a vascular phenomenon and the end-results in the course of an inflammatory process vary a great deal, according to the tissue involved. While there is no exact analogy of paranasal sinuses to other parts of the body, the inflammatory process really carries on in about the same way as it does elsewhere. Thus you get a fibrosis or a caries or necrosis from inflammatory processes in the sinuses, and you get also your polypoid formation or thickening of the tissue the same as you do in other parts of the body. Some of these changes correspond to the type of infection. I believe the correlation between the bacteriology and the gross and microscopic findings from inflammatory processes has never been thoroughly worked out in the nose.

You consider the general specific qualities of infections as we know them in all parts of the body. The syphilitic infection is characterized by inflammatory changes leading to sclerosis and fibrosis wherever it occurs. The tuberculous process is characterized by peculiar softening, a specific process which breaks down into a cold abscess. The pus organisms, depending a little bit upon which tissue is involved, produce various changes such as necrosis, caries and sclerosis: If it is in a dense bony tissue or in a dense fibrous tissue, and you get your sequestrum or carbuncle on cancellous bone, it may produce necrosis or caries or sclerosis depending upon the severity of the inflammation. There is no question about it that various infections in the nose have very different characteristics not only in their acute phenomena but in their end-results. There are some acute infections in the nose that produce a very strange pathology, which may never be recognized. The

general practitioner believes that any acute process in the nose is accompanied by a very acute coryza. That is not always the case. One of the strange pathological changes that occur in the nose of a very virulent streptococcic infection is often a dry nose with practically no coryza at all. There is no secretion until perhaps at the end of four or five days or a week. With that phenomenon, the earliest manifestation you may see is a hemorrhagic condition. I remember seeing 3 cases in one epidemic in which the only manifestation of acute process of the nose was quite a severe epistaxis. These things might be studied further, and the various pathologic changes, both acute and chronic process, corresponding to the bacteriological findings, and their correlation would be worth the object of someone's careful attention.

DR. EGGSTON (in closing):

I wish to thank the Association for the opportunity of presenting this dry, yet fundamental paper. It is usually difficult to arouse very much interest in pathology among clinicians who have not had the time or opportunity to continue studies in this direction. However, once interested in it, I do not think there is any subject which is so stimulating and fascinating as histological pathology.

Most of the tissues upon which the observations presented are based, were taken from private cases that have been thoroughly examined in a general manner and upon whom Wassermann tests and other blood examinations had been made. The tissues were all fixed immediately after removal. In the one case of distinct arteritis, with perivascular mononuclear cells typical of syphilis, referred to by Dr. Beck, the Wassermann and clinical signs were negative. There are nonspecific inflammatory reactions in the nasal mucosa that simulate syphilis, and the fact that syphilis, a vascular disease, will produce destruction of bone and soft tissues in the nose is an argument in favor of the fact that, in the basic vascular changes; particularly in the arteritis types, there exists an analogy of the processes.

The explanation of the mechanism of the production of pathological lesions is one that requires a great deal of visualization and application of the knowledge of physiology and pathological processes. Sometimes the best one can do is to arrive at conclusions that will act as a working basis even though later they may prove somewhat inaccurate.

I thought that sufficient evidence of perivascular changes was found in these sections to furnish a basis from which further work could

proceed in order to prove or disprove the theory that the vascular changes are the important ones.

I concur with Dr. Beck that too little work and study have been devoted to the histological changes in the diseases of the accessory sinuses. Of course, it is very difficult to obtain material and it is also very difficult to draw accurate conclusions from material not properly correlated with the *x*-ray and clinical observations. I hope this paper will stimulate further investigation.

Perivascular reactions in acute inflammation are undoubtedly defense reactions. In the chronic processes there is a continuation of perivascular irritation by toxins, which accounts for the perivascular infiltration resulting in continued injury with fibrosis as any inflammatory process leaves permanent damage to the tissues.

The chronic process ultimately results in gross and permanent changes which will essentially affect the contractility and resiliency of the vessels, altering the function of the delicate mucous membrane of the nose and accessory sinuses.

The vascular changes that we have noted here are not unlike arteriosclerotic changes which occur in the kidneys in so-called arteriosclerotic nephritis and to a certain extent in the capillaries, the basis of a glomerular nephritis. Vascular changes in the kidneys form a basis of classification of nephritis.

An ostitis or a periostitis undoubtedly add to the likelihood of a recurrence of symptoms of acute sinusitis, as any infection may become latent in infected bone.

There is a great deal of controversy about the respective functions of osteoblasts and osteoclasts. Histologists know they have something to do with the production and absorption of bone. Some say they are entirely different cells; some say they are identical, but assume different function, as the chemical changes of the occasion demands.

THE GENERAL PRINCIPLES OF ALLERGY

JOHN A. KOLMER, M.D., D.S.C., LL.D.

PHILADELPHIA

EACH year the list of substances harmless for the majority of human beings but responsible for the production of unpleasant symptoms and lesions in others is constantly increasing so that cases of hypersensitiveness are now known to appear in every speciality in medicine and are of growing clinical interest and importance. And this is especially true since there is also an increasing appreciation of the fact that certain symptoms hitherto variously explained may be indeed due to this unsuspected state in some individuals.

TERMINOLOGY. Unfortunately a rather large list of names has been given to this condition which may prove confusing to many practitioners. Probably the best known is that of "anaphylaxis," coined by Richet, to mean without protection or the opposite to prophylaxis. From this standpoint it is a good name for those individuals who acquire hypersensitiveness instead of a resistance or immunity to serum or other substances.

But the term "allergy" coined by von Pirquet would appear to be a better one since it means altered reactivity of cells and tissues which appears to be the fundamental mechanism. This is usually in the form of increased or exaggerated activity so that certain cells or tissues react excessively with the production of lesions and symptoms to various substances harmless for the majority of persons and it also includes the opposite state of reduced reactivity in which, by various therapeutic measures, the exaggerated susceptibility and reactivity of the tissues may be reduced.

Additional names are those of hypersensitiveness, idiosyncrasy, intolerance, hyperergy and atopy, the last being coined by Coca and Cooke to mean "strange disease," for designating those states particularly influenced by heredity like hay fever and asthma. But it would appear that the term "allergy" meets the essential

requirements in the present state of our knowledge and is worthy of general adoption although some of the others may be employed for refinements in terminology, my own custom being to use allergy as the general term, anaphylaxis as a sub-term when the exciting agent is known to be a protein substance, while hypersensitiveness is used when its chemistry is unknown or believed to be of a non-protein nature as in allergy to metallic compounds like mercury and arsenic.

Furthermore and unfortunately, a rather large and confusing list of names has been used for naming the exciting agents of allergy like anaphylactogens, allergens, sensitinogens, sensibiligen, atopens, etc. It will be noticed that all end with "en" as in immunology a word with this ending means the antigen or producing agent. But if the word allergy is used it is proper to speak of the exciting agent as allergen, if we wish to be consistent in terminology.

As will be discussed shortly in more detail it is now known that an antibody is involved in most allergic phenomena or reactions and this too has come in for a wide variety of names like anaphylactin, allergin, sensitizin and reagin, the last mentioned being coined by Coca and Cooke for designating the antibody involved in hay fever and asthma. Here it will be noticed that the ending "in" is used in conformity with nomenclature in immunology in which a word with this ending means antibody.

When the allergic antibody or allergin is in or upon cells the latter are said to be "sensitized" and this constitutes the fundamental state as it accounts for the altered (usually exaggerated) reactivity of the cells.

THEORIES OF THE MECHANISM OF ALLERGY. When the exciting agent or allergen comes in contact with the antibody or allergin in these sensitized cells a colloidal shock reaction is believed to occur resulting in the production of lesions and symptoms. This is the so-called "cellular theory" of allergy and so many facts have been accumulated in support of it as the underlying mechanism that it has been quite widely accepted.

The antibody or allergin however, may also occur in the body fluids or humors and some students of the subject are of the opinion that when the exciting agent or allergen meets with it in the blood that a reaction occurs between the two with the production of

poison usually called "anaphylatoxin" and held responsible for the lesions and symptoms. This is the fundamental conception of several theories usually listed under the general designation of the "humoral theory" of allergy but at present the cellular theory has been more widely adopted.

Indeed it would appear that the presence of the allergic antibody in the blood may prevent allergic serum reactions on the assumption that it prevents the access of the exciting agent or allergen to sensitized cells and thereby prevents instead of produces an allergic or shock reaction. This is now called "antianaphylaxis by antibody production" but should not be confused with "desensitization" since the latter term means the removal of antibody from sensitized cells and constitutes the basis of the present-day method of treating hay fever, asthma and other allergic states by the prolonged administration of an extract of the exciting agent or allergen on the assumption that minor reactions are produced in the sensitized cells between allergen and allergin which gradually exhausts the latter and thereby renders them less susceptible to the former.

On the other hand, the presence of antibody in the blood may not entirely prevent allergic reactions by preventing access of allergen to sensitized cells. The sera of allergic individuals have been injected into the skins of normal individuals and the infiltrated skins have thereby acquired a temporary hypersensitiveness to the exciting agents and show the presence of free antibody in the injected sera as shown by Prausnitz and Küstner and yet the same antibody in the blood of the allergic individuals does not protect them against reactions. There is probably an important quantitative factor not understood at present.

If these simple propositions are brought into relation to hay fever for example, it means that the pollens are the exciting agents or allergens; that while all persons come in contact with them only some produce an antibody; that this antibody sensitizes the body cells and when the individual thereafter inspires pollen into the respiratory tract or has it fall into his eyes, a reaction takes place between the pollen and the antibody in the cells which expresses itself clinically by hyperemia, cellular and serous exudation and the symptoms of hay fever.

The one and essential difference between the normal and allergic individual therefore is that the latter produces antibody to pollens, dandruffs, foods, etc., while the former does not or at least not to a detectable degree. Furthermore that allergic lesions and symptoms do not occur unless antibody is produced and if the cellular theory is correct, not until sufficient antibody has sensitized enough cells to result in allergic reactions of sufficient degree to produce lesions and symptoms. In other words some kinds of antibody, like the antitoxins, afford protection against disease, but in allergy the very opposite occurs in that an antibody is responsible for the disease resulting in the rules of immunity "standing on their heads" as Besredka is stated to have remarked. Indeed here is a disease that does not appear until antibody is produced or until enough of it has united with (sensitized) certain of the body cells.

Why some persons (roughly 10 per cent) have this unfortunate capacity for producing allergic antibody to foreign serum, pollens, dandruffs, orris root, glue, silk, cotton, foods, drugs, ivy and a host of substances is not known and constitutes the greatest riddle of all.

NATURAL AND ACQUIRED ALLERGY. It is doubtful if any child is actually born with an allergic state but it may be manifest quite early during childhood and especially allergy to horse and other foreign sera. Most of the sudden and always tragic allergic deaths have occurred in children receiving their first dose of diphtheria or tetanus antitoxins for prophylactic purposes. How or why they developed such exquisite allergic sensitiveness is unknown. The majority had never received a previous injection of serum and all such constitute true examples of "natural" allergy. In some instances one or both of the parents are known to have had a similar allergy to serum or such allergic diseases as hay fever or asthma, suggesting that while the child may not be born allergic yet that it had inherited something either in an increased capacity for making allergic antibody or tissues unusually susceptible to sensitization and the effects of allergic shocks.

Fortunately such pure examples of natural allergy are comparatively rare although there is also an apparent inheritable factor in probably the majority of those who have hay fever and asthma.

No one appears to be born with either disease but either may become manifest during childhood. The victims of hay fever and asthma and for that matter all other clinical types of allergy appear to have either inherited a greatly increased capacity for producing allergic antibody or are peculiarly subject to the effects of allergic reactions. No one can escape contact with pollens, dandruffs, foods and many other allergens but fortunately the majority of us either do not produce antibody to them or if antibody is produced it does not sensitize enough of our cells to make us conscious of the lesions and symptoms of allergic shocks; or, at least, it does not sensitize cells of such physiological importance to produce symptoms of allergy. For example, an individual may show a positive skin reaction to milk or wheat without eczema, urticaria, gastrointestinal or other demonstrable clinical evidences of allergy to these substances. In other words the skin is allergic but it may be that the gastrointestinal mucosa has escaped sensitization which accounts for the absence of allergic symptoms.

In general terms Coca, Doerr and other students of allergy believe that this important inheritable factor is an increased capacity for making allergic antibody in which the mechanism is unknown. I have long been of the opinion that it may be the inherited constitutional state of an unstable or excitable vasomotor system. The involuntary muscle of the body would appear to be especially involved in allergic sensitization and the lesions of acute allergic shock, as in hay fever and asthma, would appear to be vasodilation which I believe may be due to paralysis of the muscle of bloodvessels and resulting in acute congestion with serous and cellular exudation and for which adrenalin constitutes a physiological antidote because of its vasoconstricting action. In other words that more human beings *are* allergic to one or more substances than hitherto surmised but that those who have inherited this constitutional state are more apt to show the lesions and symptoms of allergic reactions when allergen meets with allergin in cells so situated that the resulting shock is capable of producing lesions and symptoms.

Furthermore, I believe that an unstable vasomotor system is basically responsible for dermographism and those cases of so-called "physical allergy" (Duke) due to heat and cold.

Inheritance therefore, appears to play an important rôle in many cases of allergy and especially in hay fever and asthma. But it is apparently absent in some cases in so far as one may judge by family histories and it is certain that some persons may acquire sensitiveness to horse and other foreign sera as well as to certain drugs like arsphenamine and its congeners without it. Furthermore inheritance does not appear to play any essential rôle in allergies to tuberculin, luetin and other organisms which are acquired as the result of chronic infection. But the inheritable factor undoubtedly intensifies either the degree of allergic sensitization or the severity of allergic reactions; for example, acquired allergy to horse serum following the injection of an antitoxin or other foreign serum is rarely as dangerous as the natural type in which there has been no previous sensitizing injection of serum.

KINDS AND CHEMISTRY OF ALLERGENS. One of the perplexing and confusing phases of allergy is the ever-increasing list of substances known to excite allergic reactions. Every year sees the list enlarged so that the diagnosis and treatment of allergic diseases is fast and properly becoming a speciality in medicine.

Theoretically at least every pollen may be an allergen although, practically, only those that are wind-borne possess much importance. But so many pollens have been identified with hay fever that this is indeed a perennial disease in the United States. And because of the large variety of pollens known to cause hay fever the term itself is quite obsolete as hay fever is only that form of allergy due to the pollens of the grasses and such weeds as may occur in hay whereas in truth the pollens of many trees, flowers and weeds may produce the disease. Likewise the older term of "rose-cold" is obsolete as the pollen of the rose is but an exceptional offender. A much better name is "pollen disease" or pollenosis.

But hay fever is simple as compared to allergic asthma. Here the list of allergens not only embraces the pollens and foods but the effluvia, hairs and feathers of all of the lower animals as well as silk, cotton, glue, house dust, orris root and a multitude of other substances. The list is already a large one but the end is not yet even in sight so that the detection of allergy by skin tests may require testing with such a large number of different allergens that it has become a speciality in medicine as previously mentioned.

Whether or not bacteria and their products may produce sensitization and allergic reactions is still in doubt; personally I believe that they may and that some cases of asthma may be due to allergy to streptococci, staphylococci and other bacteria growing in the upper and lower respiratory tract including the tonsils, abscessed teeth and nasal accessory sinuses. Furthermore I believe that tuberculin hypersensitiveness is an allergic phenomenon.

The proteins of allergens are believed to be the chief sensitizing substances from the chemical standpoint. No one as yet has demonstrated allergy to protein-free lipoids. But it may be that allergy may occur to certain carbohydrates although it would appear at present that these substances may be carbohydrate-protein moieties rather than pure carbohydrates.

As the matter stands at present it would appear that any protein agent in which the protein has not been hydrolyzed as far as the amino-acids may be, theoretically at least, an allergen. Just how drugs produce allergy is not known and especially in the case of such metallic compounds as mercury and arsenic. Wolff-Eisner has suggested that drugs may combine with and sufficiently alter the chemistry of body proteins to produce a foreign protein as the allergen but this has not yet been proven; neither has it been definitely disproven.

DIAGNOSIS OF ALLERGY. A thorough and painstaking history is always of great aid and importance in the diagnosis of allergy.

Not infrequently the patient knows or strongly suspects the substance or substances responsible for the attacks.

Thorough diagnosis, that is to say, the detection of all or at least the majority of exciting agents is most essential in treatment. Hence there is usually no short-cut road to diagnosis unless it be in the case of weed hay fever which, around Philadelphia, develops about August 15 and is chiefly due to the ragweeds.

Skin tests have proven of great aid. In the case of pollens and such allergies as those of the dandruffs, hairs and feathers of the lower animals, orris root, glue, silk, etc., I believe that the cutaneous or scratch tests are sufficiently sensitive; furthermore, they are safer and more specific than the intracutaneous tests and permit the conduct of a larger number at one time. But the latter are more sensitive and especially required in tests employing the

food and bacterial allergens. In every community there should be at least one specialist in this line of work fully equipped with a large set of allergens for cutaneous and intracutaneous tests and possessing sufficient experience for conducting and interpreting the results.

In suspected allergy to horse or other alien serum it is always advisable to first use the scratch test; or a drop of 1 to 10 dilution may be placed in an eye. This is especially true in natural allergy to serum where the degree of sensitiveness may be so extreme as to make the intracutaneous injection of 0.1 cc. of a 1 to 10 or even a 1 to 100 dilution very dangerous. Individuals may react to an allergen of horse hair or dandruff and not to serum but I believe that the administration of serum to such is always dangerous and to be undertaken only with very special precautions.

Cases of allergic asthma, vasomotor rhinitis, eczema and recurrent urticaria especially require thorough testing; hay fever is relatively simple in comparison.

Skin tests depend upon the principle that in allergy the skin shares in the sensitization. This however, appears to be subject to error in both a positive and negative way. Apparently the skin may react to substances which do not appear to have any possible relationship to the production of symptoms. Occasionally skin reactions occur to substances which do not produce symptoms when inhaled or swallowed by the patient. In other words it would appear that the skin may be sensitive when the mucosa of the respiratory or gastrointestinal tracts is not.

The reverse is sometimes apparent in that allergens inhaled or swallowed produce symptoms while skin tests with them yield negative reactions. The detection of antibody in the serum by the passive transfer method of Prausnitz and Küstner is occasionally helpful.

Skin tests alone therefore are not as yet the royal road to diagnosis. Much is yet to be learned about the whole subject. The history and actual experience of the patient with different allergens are still of great importance so that the exact diagnosis of allergy requires a great deal of experience, skill, detective ability of the highest order, time and infinite patience.

PROPHYLAXIS AND TREATMENT OF ALLERGY. In my opinion it is well for persons with a family history of hay fever, asthma or some other allergy to avoid all possible sensitization by serum. At present I do not immunize such children against diphtheria with toxin-antitoxin but employ diphtheria toxoid.

I also think it advisable for such persons to exercise particular care against acquiring focal infections or if such are found, to have them drained or removed if at all possible in order to reduce the chances of acquiring sensitization to bacteria, which I believe is the cause of some cases of allergic asthma.

If serum is to be given to an individual known or suspected to be allergic to it, I believe that it is advisable to first give a subcutaneous injection of adrenalin and atropin followed by the injection of very small amounts of serum at a time by one of the several well known methods of so-called serum desensitization.

In my opinion horse serum should never be given to an individual with a *natural* allergy to the horse and especially if positive skin reactions to serum are elicited. Furthermore, horse serum, like diphtheria and tetanus antitoxins, should never be given to a stranger, that is, to one with whom the physician is totally unacquainted, until a few questions are asked about symptoms referable to allergy and especially asthma.

The fact that a person gives a history of having had a previous injection of horse serum without a reaction is the best possible guarantee that he or she does not possess the dangerous natural allergy. Acquired sensitization is rarely as dangerous. Indeed I personally never hesitate giving such an individual a subsequent dose of serum by subcutaneous, intramuscular or subthecal injection although an immediate or delayed reaction may occur and especially that type known as "serum sickness." But I am much more careful if serum is to be given intravenously because here the stage is set for a severe reaction if the patient is sensitized, since the allergin is brought so suddenly in contact with sensitized cells. It is always advisable to have in readiness a syringe carrying 1 cc. of 1 to 1000 adrenalin for intramuscular injection if an acute shock reaction occurs.

The first and most important principle in the treatment of allergy is to avoid all possible contact with the allergen or allergens. Exact

diagnosis or the detection of the exciting agents is, therefore, very essential. The physician must give the patient all possible aid. For example he must teach him or her that a food must be avoided not only as such, but as it enters into the cooking of other foods. This renders allergy to basic foods like milk and wheat very difficult to handle. As far as possible the mystery in the minds of patients should be explained away; for example, how they may react to hog hair even though there are no hogs in the vicinity if hog hair happens to be in their mattress.

Substances borne in the air are especially difficult or impossible to avoid. Air filters are yielding encouraging results. A sea voyage usually affords immediate relief; a change of residence to a locality free of the exciting agent may be helpful.

Otherwise desensitization is the only treatment to be tried but may not be required to all substances giving positive skin reactions.

For example, insect-borne pollens and those that are very heavy may be disregarded. Those like ragweed and grass which are light and borne for long distances in the air are especially important. The allergist therefore, should possess a good working knowledge of botany and the physician should be acquainted with the fauna of his locality. As a general rule the commercial laboratories preparing extracts for skin testing and treatment are in position to furnish reliable information. The specialist in allergy should be prepared to advise the physician in these particulars and outline the therapeutic program.

According to present conceptions desensitization depends upon the principle of desensitizing cells, that is, upon producing slight but frequently repeated allergic reactions by administering an extract of the allergen and thereby exhausting or removing the antibody from the cells.

But there is an increasing realization of the fact that this is more difficult in practice than "on paper." In hay fever I give at least 18 injections at intervals of three to seven days (depending upon reactions) and beginning early enough to finish the series before the season opens. It is necessary to begin with a high dilution of pollen extract; usually 0.1 cc. of a 1 to 10,000 solution is well borne followed by progressive doses until the course finishes with the injection of 0.5 cc. of 1 to 100 with no severe reaction.

The same principle is used in the treatment of asthma except that more caution is required with the first doses.

I have given up trying to desensitize persons with a *natural* allergy to horse serum; I doubt if it can be carried to the point of rendering an injection of serum entirely safe.

I have also given up the hope of desensitizing individuals with exquisite allergies to foods by feeding the allergens in capsules. Part way desensitization can be secured but not to the point (in my experience) where the food can be eaten freely and without unpleasant and even dangerous reactions.

Unfortunately skin tests are not reliable indices of desensitization; that is to say, the reactions may be positive when the patient is desensitized to a satisfactory degree.

In hay fever I believe that at least three preseasonal courses should be given in succession even though marked or even complete relief from symptoms during the season follows the first or second.

In the treatment of hay fever, asthma and vasomotor rhinitis I believe that a very careful search for chronic foci of infection should always be part of the program and especially in asthma. Surgical drainage or removal of foci with the administration of autogenous vaccines and especially those prepared of the unheated but sterile toxins of the organisms is always part of my therapeutic program.

Hay fever, asthma and other allergies are still indeed and in fact "strange diseases" concerning which much has been learned and for the victims of which much may be done; desensitization is always worth while under proper conditions if there is at the outset a thorough understanding on the part of both physician and patient that there is as yet no short or "royal road" to recovery as there is much, very much, yet to be learned on etiology and treatment.

THE PATHOLOGY OF ALLERGIC TISSUE AS SEEN IN THE NOSE AND ACCESSORY SINUSES

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STUDIES of pathologic changes in the tissues of the nose and accessory sinuses in asthma and in vasomotor rhinitis have been few. Outstanding ones have been reported by Finck¹ and Hansel.² An investigation has recently been carried out under the auspices of the Harvard Medical School at the Anaphylaxis Clinic of the Massachusetts General Hospital and at the Mosher Laboratory of the Massachusetts Eye and Ear Infirmary. It has had as one of its purposes the study of pathologic tissues removed from the nose and accessory sinuses of patients having asthma or vasomotor rhinitis or both.

The material which has been studied has been taken from 26 allergic patients. Eighteen of the cases had asthma; of these 15 had primary and 3 secondary sinus operations. By "secondary operation" is meant a radical operation upon a sinus which has previously been operated upon radically. The remaining 8 patients had vasomotor rhinitis; 3 of the 8 had asthma also. In every case a radical sinus operation of greater or less extent was done. From the antrum, ethmoid, sphenoid, and from various locations in the nose, a total of 160 specimens of tissue were taken for examination. For instance, in every radical antrum operation the antral mucous membrane in the region of the canine fossa and in the region of the osteum was examined as well as the main mass of antral lining. In such a case the nasal mucous membrane of the inferior meatus was always examined also.

GROSS PATHOLOGY. The sinus membrane was found to be moderately to greatly thickened, the thickness varying from 3 to 15 mm. It was often polypoid. Polyps would occasionally fill the antrum. In 3 instances the antral lining was found to harbor multiple pea-sized pus pockets. These pockets were apparently buried in the tissue itself and not draining; on opening them the

pus which they contained appeared to be under pressure. Three large antral cysts were found, 2 in vasomotor sinuses, 1 in an asthmatic sinus. In the secondary operative cases the sinus lining was found thickened with some areas polypoid and other areas fibrotic. In some sinuses there was no secretion; in others secretion was present but varied in type. The most characteristic was thick, very tenacious mucus.

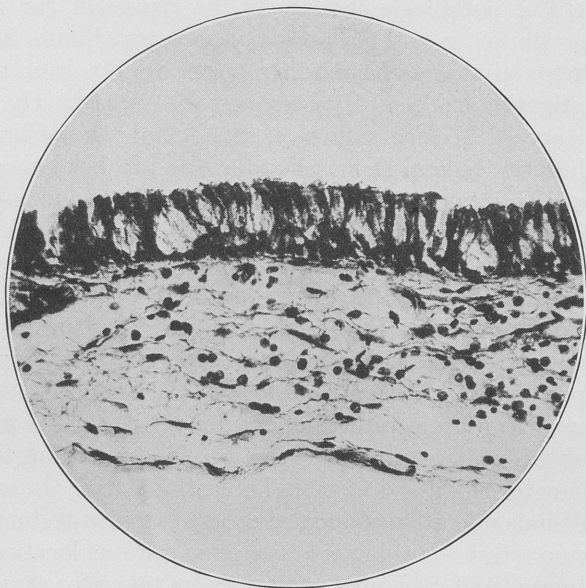


FIG. 1.—Photomicrograph of normal maxillary sinus mucous membrane. $\times 222$

MICROSCOPY. Microscopic examination of the antral secretion is of interest. Smear examination occasionally revealed large numbers of eosinophiles but more frequently the differential count was not found to be remarkable. Only 4 antra were found to be sterile; 3 of these contained secretion and 1 a cyst. A variety of organisms was found in the remaining antral cultures, the streptococcus predominating.

The microscopic examination of the 160 tissue specimens embraced over a thousand observations. In each specimen an obser-

vation was made concerning each tissue component as well as concerning miscellaneous points of interest.

Epithelium. Pseudo-stratified ciliated columnar epithelium was the usual finding. At times, however, it showed metaplasia to columnar, to stratified squamous and to cuboidal epithelium. All gradations between these various extremes were seen.

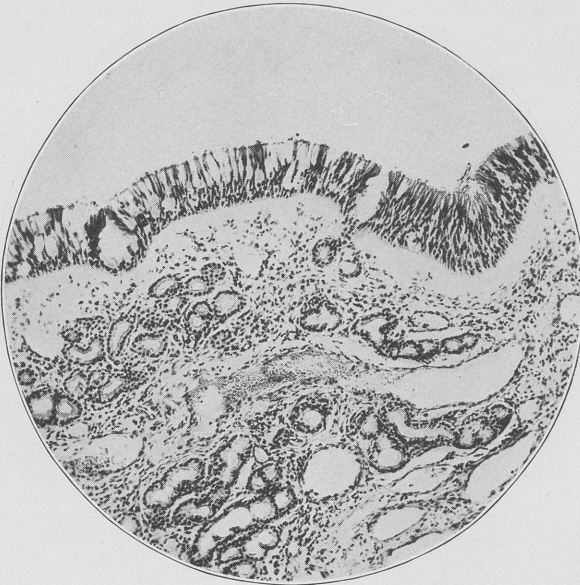


FIG. 2—Photomicrograph of left middle turbinate mucous membrane taken from a patient having asthma. $\times 75$. The epithelium is typical. Note the marked increased thickness of the basement membrane.

An almost total absence of epithelium was sometimes found and may be ascribed in many cases to methods of sponging at operation. One deduces that thousands of cells are destroyed every time a sponge is forcibly passed over the epithelial surface.

At times the epithelium was partly desquamated in process of being sectioned and stained for tissue examination. Many specimens have shown an epithelium containing numerous cells with foamy, pale-staining cytoplasm. One would suppose that these are merely goblet cells secreting mucus and many of them undoubt-

edly are such cells. An important problem is to determine whether they may at times be ordinary ciliated columnar mucous membrane cells which have taken over a new function in that they are attempting to secrete mucus. This problem can be solved only by having intricately prepared serial sections made and specially stained from extremely freshly removed tissue.³

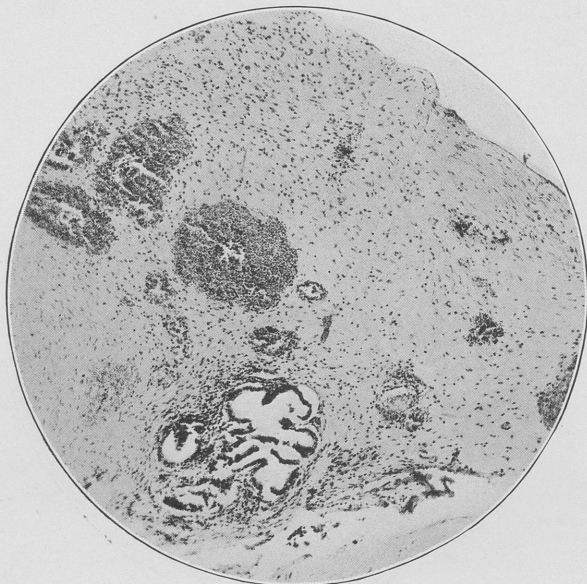


FIG. 3.—Photomicrograph of maxillary sinus mucous membrane removed from a patient having asthma. $\times 60$. Note the entire absence of epithelium. There had been no previous sinus operation in this case.

Basement Membrane. The basement membrane was universally thickened in the nasal preparations, at times extremely so. The sinus sections showed less thickening though it was still marked and in many cases extremely marked. A basement membrane may vary in thickness in the same section very appreciably. Assuming the normal thickness of the basement membrane to be a slender but definitely defined line visible with the ordinary low-power objective, a marked increase in thickness would be ten or more times the normal. The usual normal histological structures

in the basement membrane were seen. In addition it was frequently found to be vacuolated and frequently sent finger-like processes into the tunica propria. Very frequently it lost its elastic fibrillated appearance and became hyaline.

The most interesting changes seen in a basement membrane were those following repeated cauterization with strong silver nitrate solution over a period of months. In one case the membrane was found to become very thin in many places, though areas were

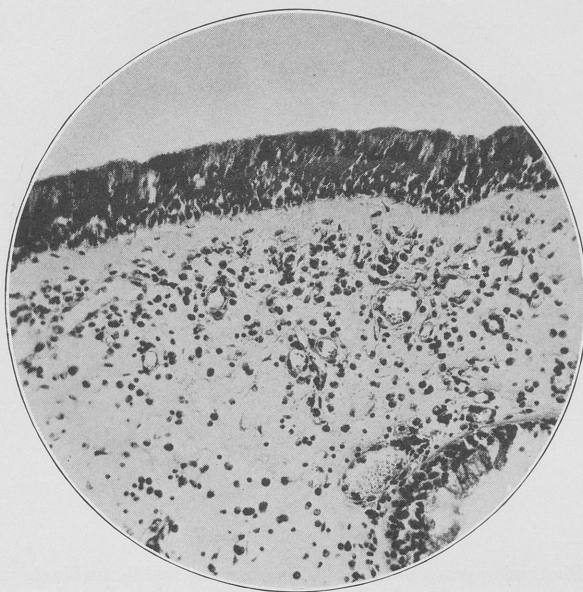


FIG. 4.—Photomicrograph of antral lining removed from an asthmatic patient. $\times 135$. Note the marked thickening of the basement membrane.

left where it was still markedly thickened. The clinical improvement in this case may possibly be ascribed to the increased filtrability of the basement membrane, allowing intercellular tissue fluids to escape more easily into the nose, thus diminishing the chronic edematous state of the tissue.

Tunica Propria. The tunica propria was found to vary widely in the tissues examined. Ordinarily it consisted of moderately loose connective tissue becoming denser toward bone and looser

toward basement membrane. There were all variations between extremely loose edematous and very dense, almost tendinous, connective tissue. Various gradations might be seen in the same antrum by cutting sections from different portions of the sinus lining. There was a tendency toward increased density of the connective tissue around the bloodvessels. This tendency was less in regard to gland structures. Cauterization of nasal mucous

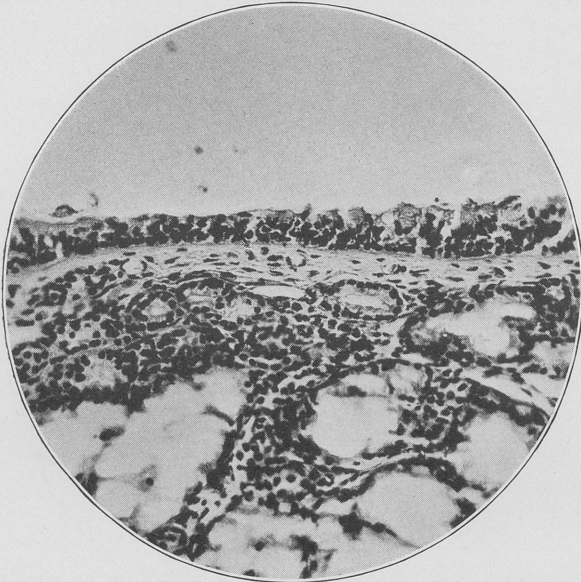


FIG. 5.—Photomicrograph of the anterior tip of the middle turbinate taken from a patient having vasomotor rhinitis. $\times 196$. This tissue had been cauterized with 50 per cent silver nitrate repeatedly during a period of months. It shows marked thinning of the basement membrane.

membrane with 50 per cent silver nitrate at intervals over a long period of time in the case previously mentioned produced superficial sclerosis only, without evidence of deep seated changes in the mucous membrane.

Edema. Of a total of 160 specimens, 54 were found to show edema microscopically.

In the asthmatics about 30 per cent of the nasal specimens and about 40 per cent of the sinus specimens showed edema.

In the patients upon whom secondary operations were performed, only about 20 per cent showed edema.

In the vasomotor rhinitis cases, about one-third showed edema both in the nasal and sinus specimens.

Fibrosis. Of the total of 160 specimens, 63 were found to show either marked fibrosis of the tunica propria or spotty areas of fibrosis.



FIG. 6.—The specimen is the same as in Fig. 5. Here, however, the basement membrane is very thick.

Of the asthmatic patients, one-third showed fibrosis in both the nasal specimens and the sinus specimens.

In the patients upon whom secondary operations were done, a little more than one-half of the specimens both from the nose and from sinuses showed fibrosis.

In the vasomotor rhinitis cases, about one-third showed fibrosis in both the nose and sinuses.

Vessels. Mucous membrane vascularity in the nose and accessory sinuses is variable. Only infrequently does one find a scanty

supply in the tissue sections examined. Usually the notation is either "moderately vascular" or "richly vascular." It is very common to find large vessels lying deeply in the tunica propria, smaller vessels ascending more or less perpendicularly toward the surface and spreading out in a network just under and parallel to the basement membrane.

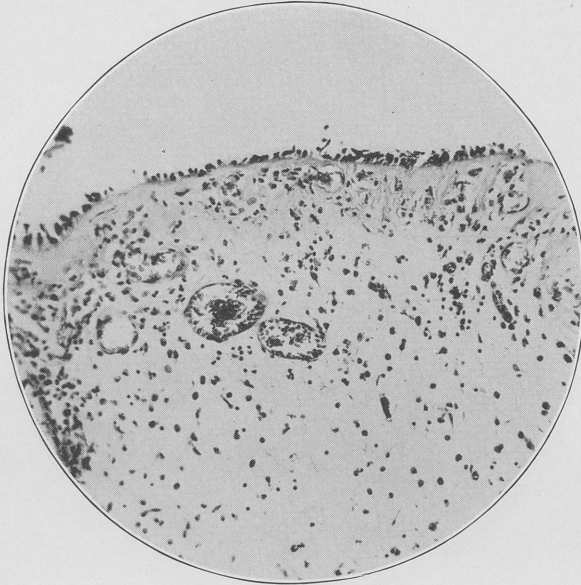


FIG. 7.—Photomicrograph of maxillary sinus mucous membrane removed from a patient having asthma. $\times 131$. There is marked edema of the tunica propria.

Unusual sections are of considerable interest. Three antral lining sections were found to be so richly vascular as to simulate an inferior turbinate.

The antral lining is ordinarily rich in small vessels in vasomotor rhinitis, and the nasal mucous membrane is highly vascular. In the asthmatic, the nasal mucous membrane is equally richly vascular but the sinus lining is more variable. It may be rather poorly supplied with vessels though the contrary is usually true. The 3 antral linings mentioned above, simulating cavernous nasal tissue, were in asthmatic patients. In only 1 case was the region

of the osteum found to have a scanty blood supply and in only 3 was it moderate. In the other cases it was either rich or extremely rich. Occasionally a vessel may be seen containing several polymorphonuclear leukocytes in a small cross-section, without any such cells in the tunica propria.

No smooth muscle hypertrophy of vessel walls was observed.

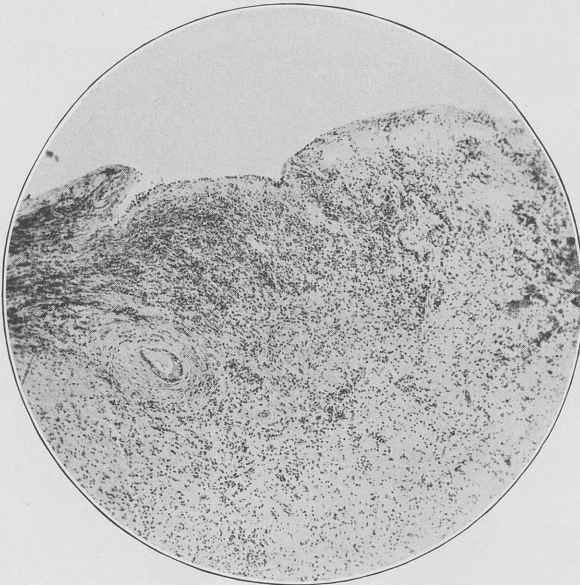


FIG. 8.—Photomicrograph of maxillary sinus mucous membrane removed at a secondary operation upon a patient having asthma. $\times 40$. The tunica propria shows marked fibrosis.

Glands. Normally, the glands of the nasal and accessory sinus mucous membrane are said to be tubo-alveolar, containing in the acini both serous and mucous cells. In the present study the ordinary tissue section, whether from nasal or sinus mucous membrane, showed (1) pale-staining, apparently actively secreting mucous gland acini; (2) trapezoid-shaped cuboidal epithelial cells lining acini which were frequently dilated; such cells were dark-staining and finely granular; the dilation was often very marked; (3) mixtures of (1) and (2) in varying proportions in the same

acinus. The ducts of such glands were identified by their lining of ciliated columnar epithelium. Such ducts were rather scarce. Frequently the same type of cell was seen in tubules opening on the surface as was seen in the acinus itself.

An interesting point about these glands was that the mucous glands, instead of taking the usual pale stain with hematoxylin and eosin, showed fibrillary cytoplasmic changes and took a faintly

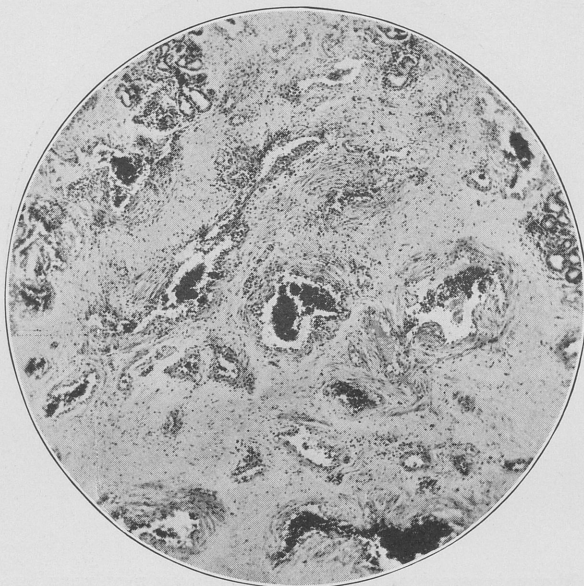


FIG. 9.—Photomicrograph of inferior turbinate mucous membrane removed from an asthmatic patient. $\times 40$. Three *antral* linings of the series studied showed vascularity approaching that seen above.

pinkish tinge near the basement membrane. This was suggestive of these glands being originally more serous than mucous with metaplasia of the serous gland cells to mucous cells. An important factor in determining the type of nasal secretion in allergic patients is the predominance of one type of gland cell over the other.

Of considerable interest was the finding of pinkish-staining secretion in some of the dilated serous glands and ducts. This was indicative of obstruction, the result being dilation of the gland

lumen and of the ducts, even going on to the ordinary retention cyst.

Infiltrating Cells. In the nasal tissues from asthmatics, plasma cells and lymphocytes were found to be about twice as common as eosinophiles and eosinophilic plasma cells. The lymphocyte was by far the commonest cell observed. An almost entire absence of infiltrating cells was seen in 2 specimens, and in 2 other specimens lymphoid nodules were observed. Polymorphonuclear leukocytes were conspicuous by their absence.

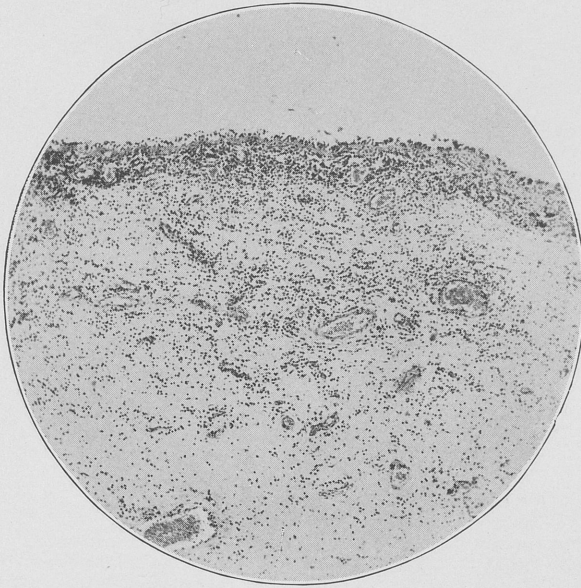


FIG. 10.—Photomicrograph of maxillary sinus mucous membrane removed from an asthmatic patient. $\times 50$. There is a rich supply of small bloodvessels.

In asthmatic sinus tissues the ratio of plasma cells and lymphocytes to eosinophiles and eosinophilic plasma cells was about 4 to 3. Eosinophilic plasma cells were one-half as common as eosinophiles. Lymphoid nodules were observed in 10 specimens. Polymorphonuclear leukocytes were absent in the asthmatic sinus tissues.

In nasal sinus specimens removed from asthmatic patients at

secondary operations eosinophiles and eosinophilic plasma cells occurred about twice as often as lymphocytes and plasma cells. Polymorphonuclear leukocytes were not observed in such tissues.

In vasomotor rhinitis nasal tissue specimens, there was a very slight predominance of eosinophiles and eosinophilic plasma cells over lymphocytes and plasma cells. Only one lymphoid nodule was observed. No polymorphonuclear leukocytes were seen in any of the nasal specimens. Four specimens showed a practically entire absence of infiltrating cells.

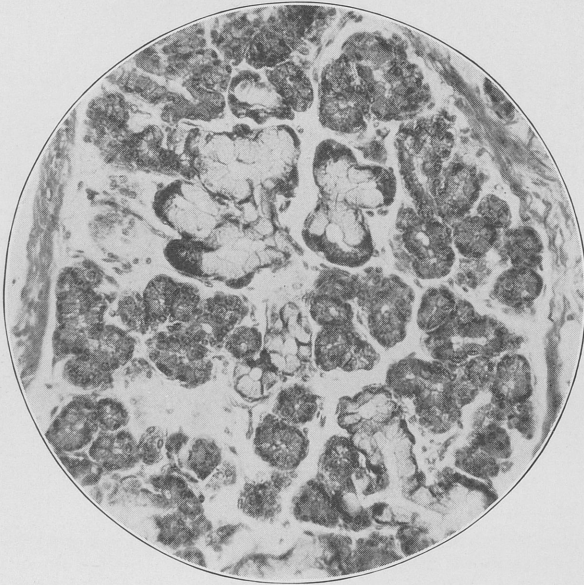


FIG. 11.—Photomicrograph of glands seen in a section of ox epiglottis. $\times 95$. It contrasts the pale-staining mucous glands with the dark-staining serous glands.

In the vasomotor rhinitis sinus mucous membrane, the eosinophile and eosinophilic plasma cells predominated over lymphocytes and ordinary plasma cells by a ratio of about 3 to 2. Lymphoid nodules were observed in 3 instances. Polymorphonuclear leukocytes were seen in only 3 of the sinus tissue specimens.

Regeneration. It is of interest to note that tissue element regeneration was present in each of the secondary operative cases.

TYPES OF VASOMOTOR NASAL MUCOUS MEMBRANE. For years Tobey⁴ has distinguished in vasomotor rhinitis patients two types of nasal mucous membrane—the red and the pale. What are the histological changes occurring in these tissues? Both have pseudo-stratified ciliated columnar epithelium, but that of the pale membrane appears edematous with vacuolated areas present at times between the cells. The basement membrane in the red type tends

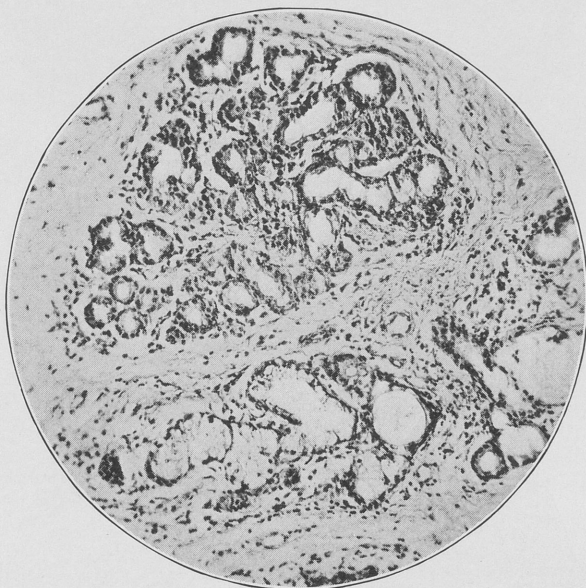


FIG. 12.—Photomicrograph of maxillary sinus mucous membrane removed at a secondary operation upon an asthmatic patient. $\times 122$. It shows dilated serous glands and very active mucous glands.

to be thicker than in the pale, and may be enormously thickened. The tunica propria in the pale membrane is exceedingly edematous, whereas in the red membrane it consists of moderately loose connective tissue. Both types are rich in glands but those in the red membrane are chiefly serous and undilated, while the pale membrane shows very active mucous as well as dilated serous acini. The red membrane is richer in infiltrating cells. In both types there is a rich vascular supply but in the red type the vessels

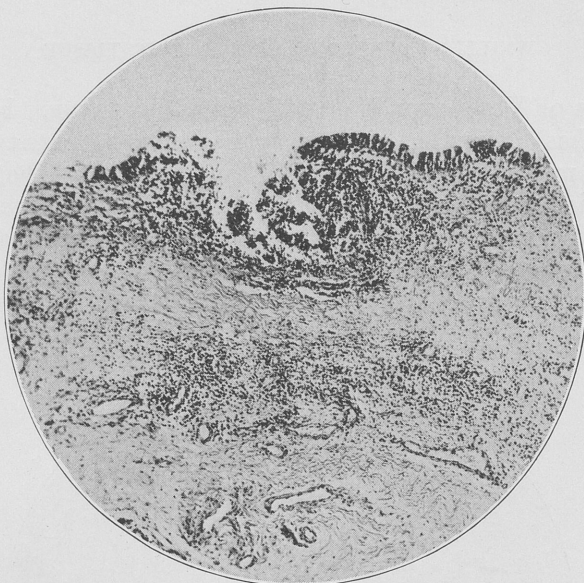


FIG. 13.—Same tissue as in Fig. 12, but from the region of the canine fossa. $\times 60$.
It shows a large subepithelial lymphoid nodule.

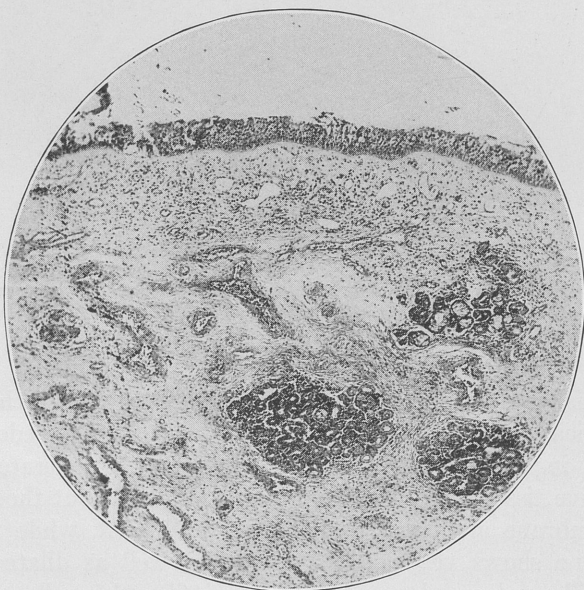


FIG. 14.—Photomicrograph of mucous membrane from the anterior tip of the middle turbinate in a patient having vasomotor rhinitis. $\times 35$. This mucous membrane is of the "red" type. See text for contrast with the "pale" type.

are relatively much richer than in the pale. In the specimens examined the thickness of the two types of membrane was the same.

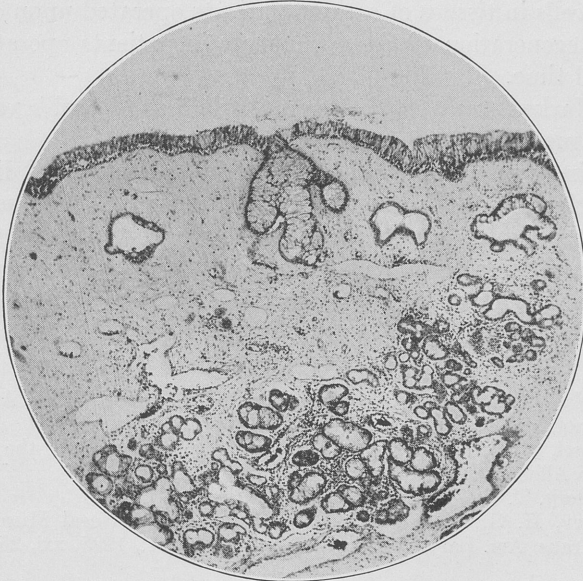


FIG. 15.—Photomicrograph of mucous membrane from the anterior tip of the middle turbinate in a patient having vasomotor rhinitis of the "pale" type. $\times 35$. See text for contrast with the "red" type. See also Fig. 14.

SUMMARY. One hundred and sixty specimens of tissue were removed from the nose and accessory sinuses in 26 patients having asthma or vasomotor rhinitis or both and were examined grossly and microscopically.

The most interesting gross finding was the presence of occasional pus pockets in sinus membranes.

The microscopic examination of the tissue sections showed:

- (a) Metaplasia at times of the type of epithelium.
- (b) Possibly metaplasia of individual epithelial cells.
- (c) Thickening of the basement membrane.
- (d) Edema or fibrosis of the tunica propria in two-thirds of the specimens.
- (e) Very active mucous glands; dilated serous glands.

(f) Increased vascularity; no special smooth muscle hypertrophy.

(g) Prominence but not predominance of eosinophiles as infiltrating cells in tissues of asthma patients operated upon once.

(h) Regeneration of tissue elements in patients operated upon a second time.

(i) Marked contrast between "pale" and "red" vasomotor mucous membrane.

A point worthy of emphasis is that the thickened lining, so frequently reported in the antrums of patients having asthma or vasomotor rhinitis, on the basis of the present study offers about one chance in nine of harboring pus pockets, and therefore of itself being a focus of infection.

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ALLERGIC CONDITIONS OF THE NOSE AND THROAT. CLINICAL CONSIDERATION

HILL HASTINGS, M.D.

It seems unnecessary to spend much time in describing the well-known changes in the mucous membrane of the nose and throat that result from allergic reactions. It is, also, unnecessary to attempt any detailed classification of our cases, nor to go into the statistics. Our experience is likely a duplication of the experience of most of you who have given time and thought to this subject. It is sufficient to say that hayfever and asthma, alike, are due to extrinsic or intrinsic irritants. Under the first heading we have pollens or dust, as, for instance, horse dandruff. Under the second heading we have foods and bacteria. The clinical manifestations are either seasonal or perennial. This brief classification can be enlarged, but it seems rather useless to do so.

I shall attempt to briefly outline some of our experiences in hayfever and asthma patients.

1. *History-taking.* Our experience is that unusual care is necessary in taking histories of these patients. They come to us first, as a rule, looking for relief. Many receive prompt surgical attention, but unfortunately not the desired relief. Most of us think we take good histories. I am persuaded that we do not. The explanation is simple, it takes much time and thought. Also, possibly it requires detective traits of character that some are born with and others acquire, if so minded. I find that this lack of extreme care in history-taking applies also to the allergic specialist. He is apt to take a fair history and depend on his skin tests for positive diagnoses.

Some one aptly said that we specialists should be ever mindful of the fact that we are first of all doctors. We should be narrow only because of our fitness for work in a special field of medicine. So acting, the consideration of operative indications and operative procedures should be held in abeyance and especially so in allergic conditions of the nose and throat. This method of approach in history-taking is self-evident but needs to be emphasized.

To emphasize the value of unusual detective ability in taking histories I feel it worth while to narrate an experience related to me by William Allen Pusey, dermatologist of Chicago, with whom I was discussing this point. He had a patient with recurring attacks of dermatitis of the hands. The attacks would occur usually on Monday. Every possible skin test was made, diets of all sorts were tried, for it seemed that the Sunday diet indiscretions must be the cause. The attacks could be cured but always recurred. There was no getting at the cause of the trouble until Doctor Pusey turned his patient over to one of his assistants who had had good luck in the study of some other patients. After a long time his assistant stated he had found the cause of the attacks. The cause was found to be the colored ink in the photogravure section of the Sunday paper, which he demonstrated several times. The patient, a gentleman of considerable intelligence and importance in the community, says that this assistant practically made him expose every detail of his daily living; and was indefatigable in his search. It finally developed that during his usual summer camping trip in the North woods he had been free of the dermatitis until the middle of a certain week. On going over his life in the North woods it was found there was nothing out of the ordinary except that the Sunday paper did not arrive on time. This was seized upon by the assistant as a possible clue and promptly solved the problem.

In history-taking we have found of late years that food sensitization could be guessed when we elicit a history from the mothers that the child developed so-called eczema when, as a baby he or she was taken off the breast. Naturally in such a case the search narrowed down to very few articles of food.

The absence of symptoms when the patient is at the beach or on a sea voyage, of course, is important as indicative of pollen or dust sensitization. The rather persistent perennial hayfever or asthma, in spite of diet changes, prompts one to eliminate foods and pollens and to consider dusts, such as feathers (beds and pillows); dandruff (horse, dog or cat, etc.); cosmetics, face powders, chewing gum, and last and I think of less consequence, bacterial foci, such as chronic tonsil infection and chronic paranasal sinus infection. By the way, in routine examination of our hayfever patients the

antra and orbits nearly always are more or less dark to transillumination and to the roentgen-ray, which we think is due to the chronic edematous condition of the nasal mucosa and of the paranasal sinuses. It does not necessarily prove chronic sinus infection, in our opinion.

The "hayfever," "asthma" child frequently starts as an eczematous baby. Unfortunately and too frequently the history shows these succeeding steps in treatments—removal of tonsils and adenoid, trimming of turbinates, removal of polyps, and still later, after secondary infections of the paranasal sinuses have occurred, radical operative procedures of various kinds; the latter part of the history pertaining mostly to adult life. The result in most cases, the history shows, is poor or fair and in many of the cases the history of repeated radical sinus operations shows a continuation of the asthma and, added to this, a severe neuralgic condition of the head, which we feel is a result of too radical nasal surgery.

I do not mean to be hypercritical and am not free of sin myself but this kind of history is all too commonly met with for us, as rhinologists, to be happy in reviewing the past histories of allergic patients.

2. *Physical Signs.* As to the physical signs of allergic reactions, it seems unnecessary to take the time to cover this part of the subject. Harold Tobey, three or four years ago, did this most completely, as have many others. I would like to emphasize a few things: The history having been taken as thoroughly as is humanly possible, and that does not mean always adequately, the examination requires much painstaking care. The physical signs will vary, of course, with the stage of the disease. If seen early in childhood an edematous condition of the mucosa is, of course, the one physical sign that stands out in all cases. At a later stage of the disease, polypoid degeneration of the middle turbinates and a similar condition of the ethmoids is usually met with. Infection of the paranasal sinuses I have learned to regard as purely secondary, and in itself is rarely the cause of asthma. Patients upon whom nasal surgery has been performed frequently show the polypoid replacement, even to such an extent that one would doubt that any real surgery had been performed. Some are inclined to think that this is

the result of inadequate surgery. While this may be true in some cases my experience is to the contrary. In some cases the examination discloses that there has been a very radical exenteration of the paranasal sinuses with a surgical result that evidently has been quite pleasing to the surgeon, inasmuch as there has been no polypoid reformation, but on the contrary a very tight scarring of the exenterated ethmoid region. However thorough the work has been and however beautiful the result, I have found many of these patient to have become confirmed sufferers from intense neuralgia without any apparent hope for a cure. Also, the asthma has persisted in most of these patients. I do not mean to criticize relief of the chronic suppuration of the sinuses by surgery. The added burden to the asthmatic of such chronic suppuration should be removed if possible. It is a question, however, whether drainage alone is not the better plan. I do not wish to discuss this surgical question, but it comes forward in reporting the results of my examination of asthma patients covering a period of a good many years. I feel that surgical conservatism still has a strong supporting leg to stand upon.

Coming from Southern California where so many allergic patients are sent on account of the climate it may be that I see too many that are unsuccessfully operated upon and am a bit pessimistic.

3. *Treatment.* As to treatment our rule is to send our patients with allergic nasal disease to a confrère, who has made this his life work, for the skin testing and treatment with the injections of the proper allergens. After a variable period of time of such treatment, conservative operative measures are taken, such as removal of polyps, and of polypoid portions of turbinates, drainage of antra, usually by the intranasal window method, less frequently, by the Caldwell-Luc method, and partial ethmoidectomy.

It is surprising what changes are at times seen in the diseased nose after competent treatment of the allergic condition. I was about to operate upon one patient but delayed to see what relief could be obtained by a study and treatment by Dr. Piness, on account of possible allergy. This was the more desired because the patient during the past years had undergone many intranasal sinus operations and one external frontal operation before coming to California, and with only temporary relief. For a year I had

treated her nose conservatively and without result. The regrowth of polypoid tissue was sufficient to warrant more nasal surgery. However, after three months' treatment by injections for pollen and food allergy there was such a marked subsidence of the nasal polypoid regrowth that my assistant thought I had done a bilateral ethmoidectomy. That was five years ago. She has had allergen treatments off and on and has seen me for an occasional "cold." My last examination note of March 1, 1930, is as follows:

"March, 1928, was treated by Dr. Millsbaugh on account of rheumatism. Patient returned today for observation. One year ago had a cold for two weeks which caused a headache and nasal discharge; cleared up after about three or four weeks. Since then nose has been in good condition. Seldom douches nose. Does not soil a handkerchief—a little postnasal clear mucus two or three times a day. Good breathing. No cough. Nasal examination shows, as two years ago, the appearance of a very satisfactory result without operating; a little sticky mucopus, apparently from the anterior ethmoid region from right, a little from left. The boggy edematous masses that filled the upper part of each side of the nose, when seen four years ago and which subsided after three years of injections for allergic condition, have not recurred. Except for a cold, a year ago, she has been entirely free of hayfever and she never has trouble sleeping, whereas up to 1924 had to prop up on three pillows at night."

In closing one is tempted to speculate on the future possibilities. The discovery of allergic reactions is after all, comparatively recent. While our attention is focussed on the allergic reactions commonly known as hayfever and asthma, tissues in other parts of the body are similarly affected. We must recognize that there may be other conditions, the etiology of which is unsuspected, which may be found due to allergic reactions. For example, many observers have found a chronic antrum infection in patients suffering from nephrosis. My assistant, Dr. S. D. Wimmer, has been working on this subject at our Children's Hospital and has found that simple irrigations of the antra in these children produced rather rapid, though temporary, improvement. His washings did not produce much pus. At times there was slight stringy mucus but often no cloudiness in the returned solution. Always,

however, centrifugalizing showed a high cell count. The cultures showed staphylococci. In a few cases there was edema of the face and soft palate which rapidly subsided, after antrum irrigation, that is, within a few hours. This experiment was repeated with the same result. It is not unlikely that the toxin found in the antral secretion had an allergic effect. I should say that Dr. Wimmer has embodied his studies in a thesis for his postgraduate degree from the University of Pennsylvania, and makes no claim for a proof of allergy in nephrosis. His work, however, shows a thick antral mucosa, without much pus, in the nephrosis cases studied, and suggests an allergic reaction as a cause of periodic edema of the soft palate.

I recall one patient of ours eighteen years ago, who would not submit to a tonsillectomy but would come in periodically for removal of a large cheesy mass from one tonsil, claiming it always cured an aching wrist. Several times I saw the rapid subsidence of the pain. Since then many observers have noted the connection between tonsil foci and rheumatism and neuritis, indicating in some cases an allergic reaction rather than the establishment of secondary foci.

From the clinical point of view one is justified in feeling that we can be of real help to the laboratory men if we study our patients more thoroughly and with a thoughtful breadth of view. As a consequence I believe, that there will be less nasal surgery done on hayfever and asthma patients.

GENERAL PRINCIPLES OF TREATMENT OF ALLERGIC DISEASE

FROM THE RHINOLOGIST'S STANDPOINT

HAROLD G. TOBEY, M.D.

BEFORE taking up the general principles of the treatment of allergy, it will be well for the purpose of this paper to define the field of discussion.

1. It is essential to confine the discussion to the treatment of the nasal and bronchial manifestations of the phenomenon of allergy, namely, allergic or atopic coryza and asthma. Other manifestations, such as urticaria, angioneurotic edema, migraine, abdominal conditions, etc., need not concern us.

2. It is necessary to recognize that while the development of the study of allergy has added greatly to our knowledge of asthma and the vasomotor disturbances of the nasal mucous membrane, it has been by no means universally admitted to be the sole cause or, in some quarters, to be the principal cause of these conditions. By some, asthma is considered a vasomotor neurosis and by others a toxemia.

Rackemann states that the proportion of patients whose asthma is definitely dependent upon some particular foreign substance is between 40 and 45 per cent. Walker gives 48 per cent, Larson and Beel 44 per cent.

Positive skin reactions are found in much larger numbers, variously quoted as from 45 to 95 per cent.

Skin tests are useful in two ways, to confirm a diagnosis as suggested by the history and to point out other possibilities.

3. The study of allergy has advanced so rapidly and so much special data has been collected that it has become a specialty in itself. Every city at the present time has one or more physicians who devote their entire time to its study and practice. This is rightly so, as a whole-hearted coöperation of the internist, allergist and rhinologist is required for the successful treatment of these conditions.

The undoubted success of widely divergent methods of treatment should teach us to keep an open mind and to be careful not to ignore any lead, no matter how insignificant it may seem to be.

Treatment may be specific, nonspecific and symptomatic.

The treatment of illness caused by hypersensitiveness to foreign proteins may be directed along several lines, namely, avoidance or removal of the specific cause; avoidance or removal of contributory causes; specific protein treatment; nonspecific protein treatment; and symptomatic treatment.

Avoidance of the cause is often simple, if the nature and distribution of the substance responsible for the reaction is known. Duke mentions a patient, sensitive to egg, who had symptoms occasionally when egg had been seemingly entirely eliminated from the diet. It was eventually found that she was also sensitive to hen meat, but could tolerate rooster meat. Those sensitive to the products of vegetable life sometimes react to traces of the substance in milk.

Pollen can usually be avoided by a change in geographical situation. In mild cases relief may be obtained by avoiding trips to the country and by confining the patient to the city.

The products of vegetation other than pollen can usually be avoided easily. Most prominent among these is orris root, widely used in the manufacture of perfumes, flavoring extracts and toilet articles of all sorts, including tooth paste, face cream and soap.

The avoidance of foods which may cause symptoms depends upon how common the food may be. Those foods commonly used in cooking, such as milk, egg, wheat and sugar, are very difficult to avoid, especially if the patient is sensitive to the cooked forms, and it is only to these foods that attempts at desensitization should be made. Other less commonly used foods can best be avoided.

Animal danders are more easily avoided. Only in exceptional cases should an attempt be made to desensitize the patient.

Drugs are also easily avoided.

House dust as a cause of asthma and nasal disturbances has been emphasized by Cooke. Relief can often be obtained by the removal of certain articles of furniture from the house.

Duke mentions certain contributing causes, the removal of

which frequently relieves the patient when it is impossible to discover the specific substance primarily responsible for the reaction. These contributing causes are chiefly physical in character such as light, heat, cold, mechanical and chemical irritants and functional activity which frequently influence the symptoms of reaction and often determine the site of their localization. He also raises the question of specific hypersensitiveness to these physical factors.

SPECIFIC PROTEIN THERAPY. The outstanding example of specific therapy is, of course, the treatment with pollen extracts. Highly successful, although somewhat technical, want of time and space does not permit of its discussion at this time. It is well to point out that the treatment is now in most cases carried out by the allergist and not by the rhinologist. There is some disagreement among the allergists as to the system of treatment, whether preseasonal or coseasonal treatment, with long or short intervals, result in most benefit to the patient.

Specific therapy with proteins other than pollen is not so satisfactory as that with pollen. Here, again, it is usually easier and more satisfactory to avoid the cause.

Infants who show an intolerance of egg or milk may have their tolerance increased by the feeding of minute quantities of the substance in increasing doses. In general it is well to treat for food sensitiveness with food in the form in which it makes the patient ill.

NONSPECIFIC PROTEIN THERAPY. Treatment with nonspecific proteins has a very definite place, particularly in those cases where a specific foreign protein as a cause cannot be demonstrated. Numerous agents have been employed for this purpose, such as typhoid and other bacilli, all types of vaccines, both autogenous and stock, tuberculin, milk, peptone, etc.

The benefit seems to result from the inflammatory reaction which is set up in the tissues and the degree of benefit proportional to the severity of the reaction.

SYMPTOMATIC TREATMENT. Adrenalin is by far the most important drug in the symptomatic treatment of asthma. Patients develop a marked tolerance to the drug, so that its use can be continued over periods of years with no evident deleterious effects.

Peculiarly enough, the internal or subcutaneous administration has no effect upon the nasal mucous membrane, but marked temporary relief may be obtained by local application to the nose. However, in severe cases even this local application is of no benefit.

Numerous other drugs have been employed with more or less success. These include cocain, stramonium leaves burned for inhalation of the fumes, the potassium and sodium salts of iodine, atropine, thyroid extract and pituitary extract.

The internist and the allergist are, perhaps, not sufficiently alive to the benefits which may accrue from bronchoscopy, both as to diagnosis and treatment. Many patients suffering from chronic asthma with superimposed infection of the bronchial tree, and perhaps, small areas of bronchiectasis are greatly relieved by the periodic aspiration of the thick tenacious mucus and pus. Other pathology is found frequently enough to justify Dr. Jackson's dictum that "All who wheeze have not asthma."

Coming to the more immediate field of endeavor of the rhinologist, namely, the nasal manifestations of allergy, all that has been said hitherto is applicable.

Vasomotor rhinitis taken, by itself, as an entity, without relation to asthma, should properly be treated by the rhinologist aided by the internist and the allergist.

To the rhinologist primarily, but in conjunction with the internist and the allergist, falls the responsibility for the evaluation of local conservative treatment and surgery in the nose and its accessory sinuses.

The typical allergic vasomotor rhinitis is recognized by the usual symptoms of nasal obstruction, watery discharge from the nose and sneezing, with the pale, edematous mucous membrane which frequently is markedly polypoid.

Confronted with such a condition I conceive it the duty of the rhinologist to put aside all thoughts of surgery, or even other local therapy, except for the temporary relief of the patient, until every step has been taken to discover the exciting cause. This necessitates an extremely careful and minute history as to the time and mode of onset, whether seasonal or perennial, continuous or intermittent. The circumstances under which the attacks occur as well as the occupation of the patient is important. The

family history should be gone over carefully, not only to establish the presence of hay fever and asthma, but also of urticaria, eczema, migraine and possibly epilepsy. A positive family history is presumptive evidence of allergy.

In seasonal cases the history usually narrows the possibilities within a small scope. In perennial cases a distinct lead is arrived at much less frequently. Hypersensitiveness to foods as a cause of vasomotor rhinitis, while not unknown, seems to be relatively infrequent.

The possibilities of endocrine disturbances should be constantly kept in mind. Disturbances of the ovarian and pituitary functions seem to be the most frequent cause of these conditions.

Mrs. H., presented all the signs and symptoms of a severe allergic vasomotor rhinitis which had persisted for two years. The history brought out the fact that the symptoms began coincidentally with irregular menstruation following an hysterectomy. Following the administration of ovarian extract the relief was prompt and complete.

Having established the probable allergic nature of the condition, we should proceed with the skin tests to confirm the diagnosis and to suggest other possibilities. Positive skin tests must not be considered conclusive unless supported by therapeutic results.

Failure to establish any specific protein hypersensitiveness necessitates the study of the body chemistry with special reference to the endocrine and to the calcium content of the blood. If one is so fortunate as to be near a special clinic for this purpose, a great deal of time and labor is saved and unsatisfactory empirical experimentation with the various extracts can be avoided.

During this time the possibility of a bacterial origin is being considered. An autogenous vaccine can be prepared although it is probable that a stock vaccine will prove to be equally as efficient.

Attention to the general hygiene of the patient is very important. By this we mean a well-regulated life with regular exercise and avoidance of fatigue.

Dean, Stucky, Jarvis and others have pointed out the importance of diet in diseases of the nose and throat. If these factors

are present they unquestionably add to the difficulty of treating an allergic rhinitis.

It is only when all of these methods of approach have been studied and have failed that surgery should be considered and then only to the end that any possible focus of infection may be eliminated and that all demonstrable foci are eradicated. This may consist of the removal of the middle turbinate or much more radical measures by which all the sinuses are opened, ventilated and drained.

Although marked improvement may follow the more radical procedures, even to a return of the normal color to the mucous membrane, it is well to be skeptical of a permanent cure from surgery alone.

Miss I. C., had for five years presented a typical allergic vasomotor disturbance. All efforts to discover the cause were without avail. Radical exenteration of both ethmoids, which were completely broken down and filled with mucopus and polypoid tissue, together with an intranasal opening of both antra, resulted in complete relief for seven months. At that time, following what was presumably an acute respiratory infection, all the symptoms have returned even to the pallor and edema of the lower turbinates. Thus far over a period of one month she has derived no benefit from treatment.

On the other hand, local treatment, such as argyrol tampons, cauterization, minor and major surgical procedures have a distinct place in the treatment of the vasomotor disturbances which present the reddened, congested mucous membrane. Since these cases, as a rule, rarely prove to be due to allergy any discussion of them would be out of place at this time. However, it is interesting to cite Alexander Francis in an article entitled "Asthma, a Vasomotor Neurosis."¹ He states that asthma is a vasomotor neurosis and can be relieved by stabilizing the vasomotor system. This is accomplished by administering a shock to the system by cauterizing the mucous membrane, usually in the nose. A most interesting observation is that the more normal the nasal mucosa the greater the probability of success. The presence of nasal polypi and low blood-pressure definitely interferes with the success of the procedure.

¹ Practitioner, 1929, exxiii, 68.

In a few cases surgery offers the only hope of relief after all other treatment has failed.

Mrs. C., has had practically complete nasal obstruction for twenty years. Nasal polypi had been removed innumerable times. Every effort had been made by the internists and allergists without avail. Vaccines had failed and the endocrine studies were negative. Radical surgery was resorted to with tremendous relief to the patient. The breathing is normal, taste has returned and for the first time in all these years she has a full night's rest. The presence of innumerable eosinophiles establishes the probable allergic nature of the condition. How permanent this relief will be only time will tell.

THE TREATMENT OF NASAL PATHOLOGY IN RELATION TO ASTHMA. By introducing this much-vexed question we would seem to be digressing from the main subject of the discussion, that is, the general principles of the treatment of allergy. Yet, for the rhinologist, the relation of nasal pathology to asthma is of extreme interest and importance. It is of interest to note that the importance of the subject has increased greatly by our knowledge of the etiology of asthma as advanced by the study of allergy. For years, the cure of asthma, by means of intranasal treatment and surgery has been the "happy hunting ground" of the rhinologist. Now the allergist rises up to dispute the grounds for such assertions and is gathering a mass of evidence to support his views. Some, not all, will agree that at the present time the burden of proof as to the cure of the asthmatic patient lies with the internist and allergist and that the rhinologist plays a secondary rôle, in contradistinction to the part played by him in the treatment of vasomotor rhinitis. Now, it may well be, that the possibilities offered by the development of our knowledge of allergy has brought from retirement many asthmatic patients who had previously been thought to have been cured by intranasal therapy.

We need not dwell upon the general principles of intranasal treatment but rather for what purpose and to what end they shall be applied.

Three hypotheses are admissible:

1. The nasal pathology is *per se* the cause of the asthma.
2. The nasal pathology is due to the same cause as the asthma and may be considered a part of the asthma.

3. The nasal pathology results in a greater permeability to the toxins which cause the asthma.

1. If we hold to the first hypothesis, that nasal pathology is the cause of asthma, we are led into innumerable operations upon the nasal chambers for conditions which otherwise would be considered of little importance. Such procedure would include the correction of deviations of the nasal septum, the removal of septal spurs, middle or lower turbinotomy to remove points of contact, cauterization of the nasal mucous membrane, and from a partial ethmoidectomy to radical operation upon all of the sinuses.

Such an extreme view can hardly be maintained in view of the data obtained from "follow-up" cases.

The incidence of upper respiratory infection in conjunction with asthma is variously reported to be from 40 per cent to 90 per cent. Rachemann found that 14 per cent of his patients with asthma also had vasomotor rhinitis.

Definite reports of actual cures by nasal treatments and surgery are extremely meager. Rachemann reports 5 per cent of cures in 1074 cases which can be attributed to surgery alone.

Dundas Grant, in 1927, reports "great improvement" in 39 per cent, "some improvement" in 42 per cent, and no improvement in 19 per cent.

Lintz states that over 30 per cent of asthmatic patients are operated on without benefit. Piness, in 1925, found that 49 per cent of his 834 patients had had previous operations on the nose and throat which had not been successful. Elsewhere it has been shown, in a large series of cases, that the eradication of foci of infection from the upper respiratory tract had little bearing upon the ultimate outcome of asthma.

2. The frequency with which vasomotor rhinitis is coincident with asthma, the similar etiology and pathology, particularly the eosinophilia, makes our second hypothesis more tenable. With this conception, radical surgery is the last procedure to be considered and then only for the same indications which would hold good if asthma were not present.

3. The third hypothesis also lends itself to rational treatment. Infections of the upper respiratory tract might well be included under this head.

At least all three groups, the internist, the allergist and the rhinologist can well agree upon the last two hypotheses and work in harmony to the greater benefit of the patient.

The first hypothesis is arbitrary and a bone of contention. Except in a very few cases can it result with benefit to the patient.

DISCUSSION ON THE PAPERS OF DRs. KOLMER, WEILLE, HASTINGS AND TOBEY

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

We have listened this afternoon, it seems to me, to the most appealing things that I have heard in a long time. We have got right down to brass tacks in what Dr. Kolmer has told us. It is exceedingly simple. We have learned about the "gens." About the "gins" we already knew before the Volstead era. Then we have learned that these "gens" could not "gen" unless there was an antibody to "gen" on; so all that is necessary for us to do is to get rid of the antibodies and then all will be well. So it is evident we have a very definite object toward which to aim our artillery and from which to acquire knowledge and truth. We should be better able to do that, Dr. Kolmer, if you could only tell us how we get to be that way, possessed of these deplorable antibodies.

Those of us who are normal can be bombarded by all kinds of pollens and all kinds of foods and do not have any trouble. The fellow who has the antibody is the sufferer. I think you told us the antibodies are usually inherited, but they can be acquired. Our experience in clinical life tells us we can acquire a sensitiveness, which means we can acquire antibodies. I am pretty serious in the matter. It does seem to me that we have there a very definite point of attack, to learn all we can clinically and from the laboratory and every other way, about how we acquire these antibodies.

I was disappointed, however, when you had gone on a little further and told us about the antibodies being in the cells and then you told us that the muscle fibers were the tissue that really harbored the trouble-makers. The smooth muscle fibers lie in the greater depths. Therefore, the exciting matter must get through the outer or epithelial cells and down into the tissues before it can reach the smooth muscle fibers. There is no doubt that having reached this sensitive structure, trouble would result.

Years ago I was interested in the production of polyps. I also had a wonderful theory and wrote a very learned paper on the subject of "Asthma, Its Intranasal Cause and its Surgical Treatment." We found in certain cases we did get a lot of relief and sometimes cure, by merely breaking up the vicious circle by getting rid of the nasal pathology.

I formerly believed, in fact I have been foolish enough to make the statement, I think, before this Association, that I could tell the person who is going to be or is susceptible to allergy by the character of the mucous membrane. If I could get a section under the microscope, I could tell the difference between it and the normal fellow who had his structures built as they should be.

I tried in my mind to assimilate and to coördinate your experience in attributing so much to smooth muscle fibers. I think this may receive our immediate commendation by what is already known to us clinically, because we can get such tremendous contraction of the nasal tissues by the use of adrenalin. That must mean that those muscle fibers, although few in number, are extremely active—hyper-sensitive.

I do not think we are on the right track when we consider what Dr. Tobey was saying, how one may repeatedly remove great masses of tissue, and then later, when one finds and can remove the real allergic cause, local pathology is changed. No more polyp formation.

We have all had our amusing cases and I do not want to take up time on them, except one case of asthma discovered forty years ago as due to feathers. I took out polyp after polyp and still he had his troubles. One day he went up into the country and was perfectly happy, then when he came back home, he got asthma again. I said, "It is something you are sleeping on which makes the symptoms. You are sleeping on some pillows made out of mother's old feather bed." He was. When he slept on hair pillows he was all right. For five years he was entirely free from polyps and asthma. Then he came to say he was worse again. I said, "You have been out and slept on those same pillows again."

He said, "No, I have slept in an entirely different house." He had been out to his mother-in-law's. I said, "If you look into it I think you will find some of those pillows there." He did, and was cured again.

DR. ARTHUR WALTER PROETZ, St. Louis, Mo.:

These two radiographs indicate the behavior of the antral mucosa during an allergic attack. The patient whose antral membrane had previously been normal in thickness, suffered an attack of

asthma after sleeping on a goose-feather pillow. Within a few hours the membrane became thickened almost to obliterating the sinus cavity.

After this case attracted our attention, we began to observe other cases with similar reactions in lesser degrees, and we found also that this unusual thickening subsided in an equally short time to an apparently normal condition.

There are two types of membrane, as Dr. Tobey says, and these are easily demonstrable with these radiographs. In one type the membrane is vacillating, one day it is thick, the next day it is thin. The contours are concave, as shown in the second figure, but this membrane may be perfectly normal tomorrow. The other type is not nearly so thick, possibly only 2 or 3 mm., if that much, is quite smooth in outline, and does not rise and fall with the patient's attack. It is exactly the same after an asthmatic attack as it was before. I think that second type is probably a true hyperplasia and if any operative procedure is to succeed, it is probably in that type and certainly not in this other one. (See illustrations on pages 125, 126, 127, 128.)

I would like to ask Dr. Kolmer, and the gentlemen who have discussed this question, since there is such a thing as sensitizing a patient to bacterial allergens, whether by any chance the repeated puncture irrigation of children's and infant's antra might lead to sensitizing these children to the bacterial allergens present in the antrum.

DR. JAMES A. BABBITT, Philadelphia:

I think these presentations of Dr. Tobey and the ones recently like that of Dr. Dean have impressed upon us the mistake of prematurely operating on these allergic cases.

Dr. Hastings reminded me of a case that I wanted to allude to in this discussion. This was a case of nephrosis with rather a marked allergic or vasomotor rhinitis condition. X-ray and other studies indicated ethmoid and antrum trouble. This case had been studied a long time in the pediatric ward of the University Hospital, and at the wish of the department we operated upon it and the child recovered. Four months later this child returned with the same amount of edema and the same rather critical condition. X-rays and studies indicated that further operative work should be done, but accidentally an abdominal condition set in which might require abdominal surgery. This stalled for the time any interference with the nose, and while simple measures were continued, nothing else was done. In three or four weeks this child seemed well again, the swelling disappeared and he left the hospital without further nasal operation.

DR. FREDERICK T. HILL, Waterville, Maine:

In a case where the mother during pregnancy has been subjected to an abnormal diet, so that we have a case of prenatal sensitization, as for cows' milk; when the child afterward has been desensitized, I am wondering whether later in life that child is especially susceptible to allergy.

DR. KOLMER (in closing):

Will Dr. Weille kindly state in his closing discussion whether he has made any histological studies of sections of tissue taken from the nose during an acute attack? I gained the impression that the pathology passed in review represents what might be designated as the results of repeated allergic attacks, and I am very much hoping that Dr. Weille has been able to study sections of tissue taken from the nose during the height of a hayfever attack. I have had the opportunity of making only a few myself, and only a few sections of skin taken from positive skin reactions, in which a great deal of vasodilatation was found, with considerable serous exudation containing some eosinophil cells that has led me to the view that the essential tissue reaction in acute allergy at least is vasodilatation, presumably due to vasomotor paralysis followed by serous and some cellular exudation.

Dr. Hastings brought up a very important point relative to hayfever patients yielding a large number of skin reactions as to which are to be chosen for the preparation of the extract for preseasonal desensitization. Patients react to many pollens that they can easily avoid and which do not require any particular consideration. Some pollens are insect-borne and need not be used for desensitization. Others are very heavy and are not distributed but for short distances from the plants. All that patients need do in the case of these is to avoid the plants. In other words, the allergist must be somewhat of a botanist and must know which pollens may be the more important. By all odds, the most important are those that are wind-borne, which are carried by currents of air over long distances. They are the pollens that the patient cannot avoid and they are the ones to which we should give special emphasis in choosing the extract for desensitization.

I was also glad to have Dr. Hastings and others bring up this question of avoiding foods. Our only hope of helping an individual subject to food allergy is to teach him or her which foods he or she must avoid. My experience with desensitization in food allergy has been mostly disappointing, and, as Dr. Hastings, Dr. Tobey and others have stated, we must not only teach our patients to avoid the foods as such, but also to avoid those foods into which they may enter in preparation.

The most distressing cases are those that have an extreme allergy to the stable foods like wheat, eggs and milk that enter so largely into the composition of other foods. I have 3 patients in Philadelphia who would not dare eat in a restaurant or even in the homes of their friends. They are confined entirely to taking their food in their own homes where these extreme precautions must be observed in avoiding all possible contact. The marble cake that was referred to was very illuminating. I have had experiences along the same line where a patient unsuspectingly had run into the very thing that should be avoided, but under the circumstances did not create any suspicion. Sometimes in asthma due to the effluvia from the hairs of the lower animals, we have patients that will react to the rabbit, guinea-pig, hog, etc., and they will say, "Well, Doctor, we do not have those animals around the house. How could it be possible that any part of my asthma is due to those things?" They overlook the possibility of these hairs being in the mattress and also in the old-fashioned hair-stuffed chairs in the house. I commonly advise patients with that type of asthma to do away with the mattress and to substitute, say, the Ostermoor or some other type of mattress.

In other words, the longer we study allergy, the more interesting it becomes from the standpoint of the possibilities of contact with things that may elicit the attack. It takes hours of time, infinite patience to teach patients the necessity of avoiding these things and to help them toward avoiding them. I have several patients whose rooms are as bare as the monk's cell; everything is taken out except the very bare necessities. The present tendency in allergy, as far as allergic asthma is concerned, is to instal into the bedrooms of some of these patients an air-filtering apparatus so that the air that they take has been largely filtered in order to give them at least relief during the hours of rest.

I think it was Dr. Hastings who raised the important question of the frequency with which preseasonal desensitization should be repeated. My practice at present is to give three preseasonal courses in succession. As I see it at the present time, there is no escape from the necessity of going through at least two or three successive years of pre-seasonal desensitization, and those that suffer a good deal during the hayfever season are quite willing to go through a second, a third and even a fourth year for the relief that they may obtain.

I think Dr. Hastings also asked me a question about the history of serum allergy or anaphylaxis. The credit for discovering allergy to serum is largely due, I believe, to Dr. Theobald Smith who, when he was at the head of the Massachusetts Department of Health, had

been using guinea-pigs for testing the strength of diphtheria antitoxin which required that they be given doses of horse serum antitoxin. Many of these pigs survived and Dr. Smith thought that they could be used again later on. He began using them again and giving them doses of antitoxin and a certain number of them developed acute convulsions and died. A less astute student and observer than Dr. Smith might have allowed those deaths to go by without inquiry, but not he. He wondered why they died. He described their death.

We in immunology referred to this as the Theobald Smith phenomenon, which was then subsequently studied by Dr. Rosenau and Dr. Anderson in this country and by Dr. Otto in Germany, and the mechanism finally unraveled as being an example of serum allergy or anaphylaxis; but we in America can take great pride in the knowledge that the development of our knowledge of allergy, as far as serum is concerned at least, was largely the result of our own American students, Smith, Anderson and Rosenau.

I think Dr. Hastings also brought up a very important point of possible allergic reactions from foci of chronic infection. For example, can a streptococcus growing in a nasal accessory sinus, tonsil or abscessed tooth produce iritis or arthritis due to the transportation of a toxin to the parts, followed by the development of allergic sensitization?

I think there is a good deal to support the view today that it might be an allergic reaction. Dr. Swift, in New York City, entertains this opinion in relation to the etiology of rheumatic arthritis. No one can give a positive answer to the question, but recent investigations make it quite probable that bacterial allergy may be the fundamental mechanism in the production of some of these conditions.

Dr. Swain asked the question of how allergy is acquired, and as I intimated when I first had the floor, I do not know how that question can be answered. Some persons become allergic; others do not. Some persons when they are given an injection of serum acquire an allergy to the serum; others do not. Why the difference, students of allergy have not yet unraveled. The present tendency is to the view that those who acquire allergic sensitiveness have inherited something that determines that state, but just the exact nature of that inheritable factor is not known.

You have very properly questioned the correctness of my view that the involuntary muscle of the body is the principal allergic tissue on the basis of the fact that the involuntary muscle of a mucous membrane is below the surface. I can only answer, though, that I believe the exciting agent can be absorbed to the depth of reaching the invol-

untary muscle just as a solution of adrenalin can be rapidly absorbed to constitute the physiological antidote to the allergic reaction, the allergic reaction causing a relaxation of involuntary muscle and adrenalin constituting its physiological antidote in causing a contraction of this muscle; and, I believe, therefore, that it is possible in both instances for this absorption to take place. This is only a personal opinion, but my clinical and laboratory investigations have lent a great deal of support to the view.

The question was asked whether children could acquire sensitization to bacterial protein by the repeated puncture of the antrum. On this point I have no experience and, therefore, can give no answer.

Dr. Hill asked the question as to the possible prenatal transmission of allergic sensitization. This is a very important subject, but again I regret being unable to give an answer. It may occur, but as far as I know, while it has had some attention, it has not had enough study to permit giving a satisfactory answer.

DR. WEILLE (in closing):

In reply to Dr. Kolmer's question as to whether I have removed any specimens of tissue at the height of a hayfever attack, I may answer that I have not done this. However, I have removed tissue at the height of a vasomotor rhinitis attack from a patient with pale mucous membrane; I have also removed the anterior tip of the middle turbinate from a patient who was in a subsiding stage of a vasomotor attack. In one very dramatic case with asthma, I operated on the patient after she had had asthmatic attacks all night and was having one when she took the ether anesthesia.

Memorials



MARCEL LERMOYEZ, M.D.

MARCEL LERMOYEZ

1858-1929

PROFESSOR LERMOYEZ was born in Cambrai, in Flanders, July 24, 1858. He possessed a trace of Spanish blood, which was suggested in his complexion and temperament. His father, a civil engineer of repute, was a man of high scientific and literary culture, who educated his son with earnest care but died when Marcel was fifteen, at which time his uncle, Leblanc, a man of erudition and a member of the French Institute, adopted him and awakened in the youth a love of archæology and belles-lettres. Lermoyez recognized the debt which he owed to the sentiment of his family toward the love of erudition. He had received a priceless intellectual heritage, namely, respect for learning, the passion for things of the spirit, and he was proud to be in his turn the standard bearer, through the exercise of his own high calling, of a tradition transmitted to him from generation to generation.

His medical studies began after a complete and refined preparation in the humanities, the sciences as well as the classics. He was a master, also, of the English and German languages. In time he became a constant and able writer, a brilliant lecturer and conversationalist, an able clinician and a physician of eminence.

Many years ago our late fellow-member, Dr. de Roaldes, between whom Lermoyez had existed a long-continued, intimate friendship, spoke of him in terms of high appreciation, regarding him, in company with Moure, Luc and Lubat-Barbon, as one of the most important laryngologists of France. As with de Roaldes, so was it with all.

As hospital intern he was under the leadership of Professor Gugenheim among others, and he followed the latter to the Lariboisière, where at that time Gugenheim was the only consultant in laryngology in the hospitals of Paris. In the course of this service Lermoyez undertook the study of laryngology. Passionately

fond of music and Professor Gugenheim being physician to the Conservatory, he undertook the study of the physiology of the voice, studied the mechanism of singing, and, in 1886, entitled his thesis, "Experimental Study upon Phonation."

At the very outset he undertook to specialize in laryngology, when the profession in general regarded the study of that organ, so essential through the ages to human speech and to the greatest refinements of music, as the most immaterial of the arts. Lermoyez conceded to Garcia, the professor of singing, the credit of the invention of the laryngoscope. Already inspired through the influence of Leblanc, his artistic tendencies had been developed and he was possessed of a large fund of general learning, for his mind was capable of understanding all forms of science.

Personally he was a youth of small figure, animated countenance, brilliant eyes, and quick but precise of speech. Becoming acquainted with Professor Leon Abbe, whose daughter he married, that great surgeon directed and supported him throughout his early medical career.

In order to pursue his specialty it became necessary to secure a hospital appointment. This he did in 1891. In 1892 he assumed direction of the *Annales de l'oreille et du larynx*. A year later, he became one of the founders of the *Presse médicale*, from that time being regarded as an undisputed authority. Many visits to foreign clinics and courses added to his education. His writings embraced a wide variety of topics, both medical and surgical, and he made many advances during the early history of laryngology and pathology. As an author he excelled to a degree which renders his methods, as described in his memoirs, worthy of study and imitation.

An accomplished linguist and an enthusiastic traveler, he represented France in most of the societies and congresses of Europe. Thus he was secretary-general of the Section of Otology of the International Medical Congress of 1900; of the International Congress of Otology in 1904; representative of France at the Congresses at London, Vienna and Budapest. He was honorary member of most of the otological societies of Europe, as well as of our own.

The life of Lermoyez was full of the substantial elements which

contribute to a distinguished and useful career. It has been set forth with unusual clarity and affection in his various published memoirs. References to them are given in the hope that some may care to learn more particularly of this remarkable confrère, through the stimulus of his story gaining encouragement toward the higher things of professional life, for, in addition to his rare personal characteristics, he was a clinician of strong and wholesome mind, thoroughly medical and thoroughly French, as demonstrated in his practical work and in his writings, a perfectly equipped otorhinolaryngologist, at the same time a skilled technician, as well as a master of the advanced nonsurgical methods of the day.

Retaining through life this charming individuality, his declining years were cheered by the universal love of the pupils, patients and friends of a lifetime, and by the respect and admiration of his colleagues everywhere. The end of his serene declining days was hastened by the loss of his splendid son, who, seriously wounded while in service during the war, never recovered.

LIST OF OBITUARIES OF DR. MARCEL LERMOYEZ

Ann. des maladies de l'oreille du larynx du nez: June, 1929, xlviii, No. 6, frontispiece.

Arch. internat. de laryng., 1929, xxxv, 131.

Bull. de l'Acad. de méd., 1929, ci, 180.

Bull. et mém. Soc. méd. d. hôp. de Paris, 1929, liii, 218.

Bull. et mém. Soc. méd. d. hôp. de Paris, December 30, 1929, p. 548 (unbound).

Grandes figures médicales contemporaines, No. 4, p. 7.

Jour. Laryngol. and Otol., 1929, xlv, 360.

Paris méd. (annexe), 1929, ii, 10, 11.



CHARLES WILLIAMSON RICHARDSON, M.D.

CHARLES WILLIAMSON RICHARDSON

1861-1929

CHARLES WILLIAMSON RICHARDSON was born in the city of Washington, D. C., August 22, 1861. He was the son of Charles F. E. Richardson, Hookstown, Baltimore County, Md., a merchant. His mother, Charlotte Ann Richardson, of Washington, was, like the father, of Scotch-American parentage. Dr. Richardson's early life was spent in Washington, where he attended private and preparatory school, and where he spent three years at Columbian (now the George Washington) University. He studied medicine at the George Washington University and at the University of Pennsylvania, receiving the degree of M.D. in 1884, under the preceptorship of Dr. Z. T. Sower.

After a service of one year as intern, at the University of Pennsylvania Hospital, he began the study of laryngology at Vienna under Professors Schroetter, Stoerk and Chiari. Returning to Washington, he entered upon the practice of laryngology and became actively associated with the dispensaries of the George Washington University Hospital, the Providence Hospital, the Episcopal Eye, Ear, Nose and Throat Hospital and the Children's Hospital, later becoming attending laryngologist to these institutions and to the Foundling Asylum Hospital.

For many years Dr. Richardson was professor of otolaryngology in the medical department of George Washington University, becoming emeritus in 1924. He was admitted to the American Laryngological Association in 1902, and was also a fellow of the American Otological Society, the American Climatological and Clinical Association, fellow of the American Medical Association, president of the Medical Society of the District of Columbia, of the American Rhinological, Laryngological and Otological Society, and of the Climatological and Clinical Association, vice-president of the American Medical Association and for three terms one of

its trustees. He was elected president of "The Fossils" in 1927, and in June of the same year received the honorary degree of Sc.D. from the University of Pennsylvania, having already been awarded it, in 1921, by the George Washington University.

His philanthropic interests were of a general civic nature. His chief diversion was yachting, and he was a ruling elder of the Presbyterian Church. In politics Republican, he was in Washington a member of the Cosmos, the Army and Navy, the Chevy Chase Clubs, and of the Duxbury Yacht Club in Massachusetts.

Dr. Richardson conspicuously applied his natural talents and the rich experience acquired through many years of unusual activity and wide opportunities of observation to the uplift of humanity and to the alleviation of its sufferings. Devoted to his profession and to its highest aims, he justly merits the unstinted esteem accorded him as physician, scientist, investigator, author, educator, churchman and occupant of the many positions of honor and trust he so zealously and ably filled.

During the World War he was an officer in the Medical Corps of the U. S. Army, later rising to the rank of colonel, and about ten years ago was on the staff of the Surgeon-General of the Army, directing the work in behalf of the deaf. Untiring in his services during the War, on October 25, 1917, he reported for duty, Section of Otolaryngology, Division of Surgery of the Head, at the Surgeon-General's Office, Major Harris P. Mosher having been ordered over seas. He then attended the Congress of Clinical Surgeons in Chicago and presented a paper on "The Activities of the Section of Otolaryngology" as part of the symposium of the specialties in the Surgeon-General's office. Later he prepared a card diagnostic sheet for use in base hospitals and cantonments, and reported to Major King with regard to the reconstruction of the defects in hearing and speech. Continuing his activities throughout the World War, he finally culminated them in the publication of the report of the committee, Division of Medical Sciences, National Research Council, on the "Physical Causes of Deafness." Part I related to the medical study. The editing of this work, made up of contributions furnished by various members of the committee, reflected great credit upon Dr. Richardson, as well as upon the others.

Dr. Richardson's last contribution to medical literature was his address as president of the American Laryngological Association at its meeting in Atlantic City in 1929. This contained many advanced ideas upon subjects urgently demanding attention, and brought them impressively before the meeting in a manner to challenge attention and to command practical results. The dignity with which he presided will long be remembered. The occasion itself was a fitting culmination to a career which, as set forth by the record already given and by the experience of those about him, has placed him among the foremost of the progressive, devoted members of our profession and of the true-hearted, unselfish patriots of our Country.

A few weeks after the meeting of the American Laryngological Association in 1929, Dr. Richardson left home to attend the annual convention of the American Medical Association at Portland, Oregon, but, taken ill soon after his return, he underwent an operation at Boston which appeared to have been successful. Within a month, however, and while convalescing, pneumonia supervened and on the 25th of August he died.

He is survived by his widow, Mrs. Amy Small Richardson, a daughter, Mrs. William Dwight Chandler, five grandchildren, and two sisters, Mrs. William D. Searles, of Washington, and Mrs. Arthur Oswald, of Mountain Lake Park, Md.

At a Joint Memorial Service in memory of Dr. Richardson, held by the New York Avenue Presbyterian Church, the George Washington University and the Medical Society of the District of Columbia, on Sunday afternoon, December 8, 1929, addresses were made by Professor George Edmund deSchweinitz, of the University of Pennsylvania, who presided, and by Cloyd Heck Marvin, President of the George Washington University; Josiah Harmar Penniman, President and Provost of the University of Pennsylvania; John A. Foote, President of the Medical Society of the District of Columbia and Dean of the Medical School of Georgetown University; Major-General Merritte Weber Ireland, Surgeon-General, United States Army, President American College of Surgeons; Miss Enfield Joiner, Superintendent of Defects of Hearing and Speech in World War Hospital, No. 11, Cape May; John Campbell Merriam, President of Carnegie

Institute of Washington, National Research Council; Hon. Ray Lyman Wilbur, Secretary of the Interior, former President of the American Medical Association; the Hon. James Montgomery Beck, former President of the Society of Fossils and by Rev. Joseph Sizoo, D.D., pastor of the New York Avenue Presbyterian Church.

This meeting was remarkable in bringing together, as it did, a group of men of the highest standing, each one of whom had been actively and intimately associated in a department of work distinct from that of the others, often under circumstances of historic importance. No higher tribute could be paid than has been given in the testimonials of these loyal friends, exemplifying, as they do, the ever-present attributes of the true physician as well as those of the man himself. They are herewith given as well-deserving permanent record in our archives.

Following the invocation by the Rev. Dr. Radcliffe, the chairman, Dr. deSchweinitz, in his introductory remarks, paid tribute to Dr. Richardson as one who, true to the obligations of manifold duty, had passed through the gateway of high endeavor in the fields of notable achievement; one who lived his life abundantly in the dignity of professional labor, in the forum of medical instruction, in the duties of administrative work and in the obligations inherent in the medical military service. His friendship was a precious gift; his genial hospitality, with her who knew and loved him best, remains a treasured memory. He served his country, his profession, his university, his fellow-man, faithfully and well.

Dr. Cloyd Heck Marvin said:

In the death of Dr. Richardson the University, as well as all who knew him, has lost a good, great friend. As student, professor, alumni leader and trustee, he allied himself intimately with the progress and fame of the George Washington University. His fine work while a student distinguished him as one destined to serve mankind through a high order of scholarship. We cannot think of his leadership in the classroom or among the alumni without appreciation. As a trustee, his sincere and generous contributions leave the University his debtor. Becoming a scholar in pioneer fields he used the ordered knowledge that was his to bring relief to those afflicted. Diligent in his profession, he dealt honorably with all men. Charity for divergent views and a tolerance toward the beliefs of others, tempered his convictions. Unnumbered generations will be his debtors.

The chairman then read the citation made by President Penniman of the University of Pennsylvania, when he bestowed the honorary degree of Doctor of Science upon Dr. Richardson:

Son of Columbian, now George Washington, University, and of the University of Pennsylvania, from which you received the degree of Doctor of Medicine in 1884, after which you pursued further medical studies in Vienna. You have been honored by the degree of Doctor of Science by George Washington University, and are a Fellow of the Royal Society of London. For many years you were a professor in the medical school of George Washington University. To the advancement of all knowledge you have contributed many papers, especially in the field of otolaryngology. Your career has been a notable one in public, as well as in professional life. You were appointed to the National Research Council to represent the American Medical Association's permanent committee of the Deaf Child, Throat and Ear Section. As early as 1908 you became a member of the Medical Reserve Corps of the U. S. Army. When this country entered the World War you gave up your private practice and devoted your entire time to the service of the country. With other distinguished physicians and surgeons, you aided in making possible the medical organization that trained the army of doctors, nurses and hospital assistants who cared for the sick and wounded in France, as well as in the camps at home. You saw that the work would be long and difficult. To you was committed the task of directing the reconstruction in the Division of Defects in Hearing and Speech. You were notably successful in this colossal task. This long record of service has been characterized by the devotion of all your powers of thought and skill in medicine and surgery to the alleviation of suffering and the correction of physical deficiencies and defects. As fellow and trustee of the American Medical Association, your interest has been not only in the subjects in which you have made such a distinguished record, but in the general advancement of the science and art of medicine. You have held important offices in numerous medical organizations, you have given freely of your time and skill to hospitals. You have shown great administrative ability, which has made effective the work of committees and associations of which you have been a member. You have made contributions of thought and knowledge which are being applied in civil life, especially in the public schools.

The chairman then read a message, coming from a very great man, the oldest living surgeon in the United States, and in his day the most distinguished.

The passing of Dr. Charles W. Richardson is a distinct loss to the medical profession, especially in the realm of laryngology, and in the District of Columbia. He was a national figure, widely known, always highly esteemed as a man and among the most eminent in his own department of medicine. In Washington he was a worthy representative of medicine.

I happily counted him as one of my warmest friends for years.
(Signed) W. W. KEEN (ninety-seven years of age, 1929.)

Dr. John A. Foote said:

When we review the eminence of Dr. Richardson as physician, surgeon and contributor to surgical knowledge, we can explain his success as the result of a splendid education, combined with unusual intellectual and physical endowment, and an almost preternatural industry, all intelligently contributed to the attainment of his purpose. We can easily explain his rare qualities of heart and mind as the direct result of adherence to the admirable precepts laid down for his guidance by his father, in his earlier days.

Among the many sides manifested in his professional career and character, let us recall his activities, long ago, when epidemics of diphtheria terrorized the hearts of those who had young children, affecting rich and poor alike. In 1885 a modest New York medical genius, Joseph J. O'Dwyer, perfected intubation for the relief of laryngeal obstruction in these cases; this great contribution to medical science was at first received with scepticism by many; but Dr. Richardson eagerly sought the inventor and mastered the technique of intubation to a degree which caused him to be summoned from far and wide, often with no remuneration. This work, performed so willingly and so unselfishly, would alone be sufficient to distinguish him as a benefactor.

He was one of the first in Washington to use diphtheria antitoxin and to teach the value and the technique of its administration.

As an example of strong moral courage, Dr. Richardson, several years ago, noticed that, after certain operations upon the throat which he had performed, an occasional patient developed abscess of the lung. He analyzed and published his observation, in 1912, as an undesirable sequel to tonsillectomy. Similar conditions were promptly recorded from other parts of the world, and investigations were begun tending to the prevention of this serious accident, a direct result of the courage of one who was his own most consistent and severe critic.

His interest in organized medicine as represented by the Medical Society of the District of Columbia never lessened. He was foremost in every important enterprise inaugurated by the society over a period of many years; always present at its meetings, unless with adequate excuse, and at one time its president. In the interests of public health, campaigns for pure food, clean water, pasteurized milk, medical licensure and the many educational campaigns waged by the society to improve the health of the community, Dr. Richardson was foremost. In the campaign for the erection of a suitable building to house the Medical Society of the District of Columbia, indifference and lethargy were overcome by Dr. Richardson, who, stimulating others, personally raised one-fourth of the necessary fund.

If Dr. Richardson could have added to his life all the years of life he saved for others, through his own exertions and through his teachings to other men, he would indeed have attained an earthly immortality.

Surgeon-General Ireland, U. S. A.:

In looking backward we may often see the causes and effects of events and actions, not clearly revealed to us at the time of their occurrence. It has now become quite clear that, in 1908, was taken a great step forward in the organization of the Medical Corps of the Army, which, though it attracted no great attention at the time, was destined to make possible the remarkable success of the medical service of the national army in the greatest war in the annals of the human race. It secured, also, for our wounded the best professional care that had ever been given to the victims of war.

This step was the creation, by the Act of April 23, 1908, of the Medical Reserve Corps. It broke down the barrier which had always before existed between the army surgeon and his professional brethren in civil life, and brought to these as a class the realization that they had, as members of the medical profession, a definite patriotic responsibility, which was to do their part in the medical service of the army or navy in time of war.

This act for the first time authorized the enrollment of the civilian doctor in the army in time of peace under a reserve commission. The rank of these reserve commissions was an humble one, being in the grade of first lieutenant.

The Surgeon-General felt obliged to accept this rather than arouse antagonism in the Army and in Congress by asking for any higher grades at that time. So much greater is the credit due to the dis-

tinguished men in all professions for being willing to accept these subordinate grades in time of peace, with the expectation, but without any guaranty, that they would receive higher and more appropriate grade in the volunteer army with which we expected at that time to wage future war.

The first commissions given in the Reserve Corps, 170 in number, were dated July 5, 1908, and on this list may be found the foremost of the medical profession of our country at that time. On it was the name of Charles W. Richardson, of Washington. On either side of him were Harvey Cushing and Richard Cabot, of Boston; Alexander Lambert, of New York; Arthur Bevan, Frank Billings and Murphy, of Chicago. George deSchweinitz, of Philadelphia; Mitchell, of Washington; with Finney, Thayer, Matas and Crile, but a few of the great names to be found on that noble list. Thus our friend began his military affiliation in most distinguished company. When the World War came, Dr. Richardson at once offered his whole time and abilities to the service of his country. His prominence at once won him immediate recognition and he was brought into the office of the Surgeon-General to take charge of the subsection of otolaryngology. Thus it fell to him to be the representative of his specialty in the Army, and to see that the best men in that branch were brought into military service and were given a rank appropriate to their age, experience and ability, and were assigned to the hospitals where their services were most needed.

The importance of his work is obvious; he did also much staff work for his chief in addressing important meetings at medical centers in all parts of the country, in inspections of the hospitals and in other medico-military activities.

On May 20, 1918, he was promoted to the grade of Lieutenant-Colonel, national army.

In September, 1918, he was placed in charge of the division of physical reconstruction, which brought him even wider interests and responsibilities. One of these, which greatly interested him, was General Hospital No. 11, at Cape May, New Jersey, which was especially devoted to the care and, if possible, the cure of soldiers suffering from the defects of speech and hearing.

Like others, he longed for the wonderful experience of service in France, and was fortunate in securing the powerful influence of General Gorgas, who recommended him for duty as an inspector in his specialty in the American Army in France. But through the fickleness of fortune and the uncertainties of war, Col. Richardson was destined to complete his service without the great adventure of service abroad.

Miss Enfield Joiner:

When our country entered the World War, Col. Richardson went into active service with a very definite purpose. He had heard that the English government and the other governments of the Allies had made an irremedial mistake in discharging their war-deafened soldiers on their return from the front. Having once reached home, these soldiers had refused to leave again to accept the rehabilitation opportunities their government had provided. Col. Richardson was determined that the deafened American soldier should not become the victim of such a short-sighted policy.

The first step in carrying out his plan was to have a bill passed in Congress, keeping the disabled soldier under the authority of the Surgeon-General. He accomplished this practically singlehanded, working against heavy odds. The next step was to have lip reading for the deafened and re-education in speech for the aphasic, defined as treatment. He then selected the officers and teachers of his unit, assigned to U. S. A. General Hospital, Cape May, N. J. At a time when other rehabilitating services were beginning to organize, the section of Defects of Hearing and Speech were ready to function.

Dr. Richardson did not seem to display any of the qualities that might be called "diplomatic." Clear in his thinking, he was decided in his conclusions, direct in speech, fearless but constructive in criticism. He seemed to accomplish things by being always so remarkably right. In July, 1918, Col. Richardson brought together in the hospital lobby a group of disheartened deafened soldiers and a half dozen teachers of lip-reading and corrective speech; it was wonderful to see the dawn of hope on the faces of those men as they watched him, who was bringing to them what he himself called "a light in the darkness."

From the beginning the work was a spectacular success, a blessing seemed, nay, did rest upon it. The spirit of the commanding officer inspired the unit, a spirit of optimism and of readiness to give all and to do one's best. He gloried in the rapid progress of the men; his attitude toward them was one of friendliness, kindness and even tenderness.

Neither from an economic nor a spiritual viewpoint can one appraise the result of such work. In one instance the government profited to a large extent. When the Fourth Liberty Loan was being raised some rehabilitated men were sent out to plead the cause of the government; two expert lip-readers sold \$80,000 worth of bonds in one evening. After their discharge many of the men refused further aid. All over

our country today are ex-soldiers who understand lip-reading in spite of their deafness, and others who have been trained to correct their war-afflicted speech defects. Their rehabilitation was the result of the vision, the labors and the sacrifices of this great man and of his accomplished and self-sacrificing associates.

The result at Cape May gave an impetus to the teaching of lip-reading throughout the country and even abroad. What such knowledge will mean to the many deafened is already being abundantly proved.

Dr. John Campbell Merriam said:

One of the questions most attractive to Dr. Richardson in his later years concerned the development of research on the physical causes of deafness.

Largely through the personal efforts of Dr. Richardson and Dr. Percival Hall, the National Research Council obtained from the Laura Spelman Rockefeller Memorial such funds as were necessary for this work. The study in its first phases covered the present conditions and methods applied in schools of the deaf; the second phase was devoted specifically to the study of the physical conditions out of which deafness arises.

The early grants were apportioned to various subdivisions of the survey, relating to administrative, psychological and teaching problems connected with the promotion of the teaching of the deaf.

In the second study, consideration was given to questions of heredity, stage or age at which deafness developed and the discoverable specific causes of deafness. Examination was made of about 3000 deaf persons in institutions throughout the country. The research brought together an important body of data already published, and has come into use in many directions as the foundation for intensive studies upon similar problems. Through the peculiarly favorable relations which Dr. Richardson maintained toward the specific study of different individuals, through his relation to a large group of able students in the medical field and through a wide range of scientific researches touching the problem of deafness, he was extraordinarily qualified for the work.

In building the complicated structure of knowledge and in planning its application to meet the widening range of human needs, there is no function more important than that of the man who, from long experience in practice and administration, applies himself to the bringing together for the study of the fundamental problems the agencies

which have come under his observation. Such was this contribution of Dr. Richardson to the advancement of research on problems relating to the deaf.

Secretary Wilbur said:

Medicine is proud of her many honors and looks back upon a long series of leaders in the various fields who have by their combined efforts built up the great structure which is now serving mankind.

The American Medical Association belongs to the medical profession of the country. For forty-three years Dr. Richardson was an active member and for nine years a trustee. He accepted no task lightly. To be a member meant for him to be a worker for the common good.

In 1889 he presented his first paper before the newly created section on laryngology and otology. The most striking of his long series of scientific papers was that on the "Physical Causes of Deafness," part of a report of a committee of the National Research Council. His leadership as a young man is shown by his appointment, with Dr. Osler and Dr. Matas, as a member of the Association Medal Committee in 1900. But his interests were broader than those of medicine alone. He felt that the general public must learn something of the great discoveries and great opportunities associated with personal and public health. As trustee he was instrumental in inaugurating the new *Journal of Hygiene*, which is extending its influence in the prevention of disease and the promotion of health.

His wide knowledge of Washington, and its ways, made him a most effective supporter of the legislation on caustic materials passed by Congress. He saw in it the saving of children from damaged eyes and strictured throats, doubtless to his own deep satisfaction.

In his trusteeship of the American Medical Association he was keen, conscientious, persistent and courteous. Kind and thorough, he made an admirable member of the board.

Honored in every organization in the country pertaining to his chosen field, busy with the activities of good citizenship, it was as practising physician that I like best to think of him, and, speaking for the National Association, it is just that he should be known as one of the very best.

Mr. Beck said:

May I bring to this occasion a tribute from the boyhood friends of Dr. Richardson. Therefore, speaking from an entirely different activity from any that have preceded, it would have been most

gratifying to him that a place should be found upon this program for a group of boyhood friends who would testify to his generous spirit and to the great sympathy that he always felt for the ties of youth. Among the friends of Dr. Richardson's youth was a group of boys who, fifty years ago, found their greatest pleasure in editing, printing and publishing juvenile periodicals. Always successful in what he undertook, Dr. Richardson's periodical, called *Our American Youth*, was among the best, and it was as a contributor to its columns that I first gained the privilege of his friendship. Twenty-five years later, a group of these young men, desiring to renew the associations of boyhood, formed a society which they jocosely called "The Fossils." Our friend was an active member and a past president. Each year the Fossils hold an annual dinner to recall the memories of boyhood. Thus, we have tried to keep alive what Shakespeare has called "the consonancy of youth." Beloved by all his associates in this organization, he took the most friendly interest in their welfare. He was eminently a man of enthusiasm which rises above the commonplaces of the world to view with a divine exaltation the nobler elements of human life, an enthusiasm which never lessened with the passing years. In that spirit he lived and worked, meeting the difficulties and problems of life with a courageous smile.

Rev. Dr. Joseph Sizoo said:

I know of no finer and simpler interpretation of the life of Dr. Richardson than the word loyalty. His was an unfaltering life into which there came, out of the past, three great influences, three remarkable personalities. With the biographies of those three forebears we are not so much concerned as with the dominant elements of their character, which may have deeply affected the character of Dr. Richardson. First in this group was the figure of Daniel Boone, the symbol of physical endurance and courage; it was but natural that Dr. Richardson should have been possessed of great physical endurance and stamina. Always he lived a vigorous life, rising early both winter and summer, year after year. He gained ample exercise through briskly walking in the open air, thus each morning coming to his task fresh and prepared. When honor and prestige came, he never permitted it to dissipate his physical wellbeing.

The love of his profession might well have been traced to another ancestor, Sir John Richardson, born toward the end of the eighteenth century and one of England's foremost surgeons; a man of intrepid courage, for he was associated with Sir John Franklin, the Arctic

explorer. As a surgeon he was identified with the great hospital in Edinburgh, physician to the British fleet, head of the Melville Hospital in London, Director-General of the Medical Department of the British navy and inspector of hospitals in Great Britain.

As a student at the University of Pennsylvania, Dr. Richardson met the leading men of the day, and, above all, Dr. William Osler, whose first intern in an American hospital he became. Later in London he met Sir Frederick Treves, and, finally, that company of remarkable teachers in laryngology in Vienna.

These two aspects of his life, however, did not fully explain it. There stood behind him a third personality, Alexander Campbell, a Scotch divine, of Montross, famed as a preacher, who, besides his ability to work, preached to seamen whose ships were riding in the harbor, addressing them in English, German, Italian or French, as the necessity might arise. He was an humble man, known for the simplicity of his life and his unfaltering faith. The soul of that man is reflected in the religious life of Dr. Richardson, who, a devout man, never failed to interpret his religion in terms of good will, kindness and rich service. Pasteur once said to a company of doctors: "Happy is he who carries with him a God," to which Moralis added: "You touch God when you lay your hand on the human body." Dr. Richardson practised the latter and felt the former. To the interests of his church Dr. Richardson gave many an hour and sacrificed much. Crowded as were his days with professional work, his religious duties were never neglected. Unfaltering to the profession to which he had dedicated his life, he was unwavering in his faith.

Among his published articles we find the following important contributions:

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Some Remarks on the Operative Methods in the Treatment of Adenoid Growths, *Jour. Am. Med. Assn.*, November 18, 1893.

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A Case of Perichondritis of the Thyroid Cartilage, *Ann. Otol. Rhinol. and Laryngol.*, May, 1897.

Diseases of the Antrum, *Virginia Med. Semi-Monthly*, December, 1897.

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Report of 2 Cases of Cerebral Abscess, with Recovery, *Trans. Am. Otol. Soc.*, 1903.

A Case of Multiple Recurring Papillomata of the Larynx. *Fraenkel Fest. No.*, *Ann. Otol., Rhinol. and Laryngol.*, December, 1906.

Constitutional Conditions Affecting Nasal Catarrh, *Month. Cyclop. and Med. Bull.*, October, 1902.

A Case of Rarefying Osteitis of the Mastoid Bone, *Ann. Otol., Rhinol. and Laryngol.*, September, 1911.

The Report of a Case of Sinus Thrombosis, Excision of the Internal Jugular, with Recovery, *Trans. Am. Otol. Soc.*, 1912.

Complications of the Operation for Removal of Tonsils, *Proc. Am. Laryngol. Assn.*, May, 1912.

Tonsillectomy, with Consideration of its Complications, *Wash. Med. Ann.*, May, 1912, xi, No. 2.

Some Observations on Intra-nasal Surgery, *Trans. Am. Laryngol. Rhinol. and Otol. Soc.*, 1913.

Laryngitis Submucosa Subglottica Acuta, *The Laryngoscope*, July, 1914.

Vasomotor Disturbances of the Upper Air Tract, *The Laryngoscope*, August, 1911.

Congenital Atresia of the Postnasal Orifice, *The Lancet*, August 15, 1914.

Tonsillectomy in the Adult. Are We Justified in Doing so Many Indiscriminate Tonsillectomies for Remote Infections? *The Laryngoscope*, May, 1915.

Abscess of the Lung Following Operation on the Tonsils and Upper Air Tract, *The Laryngoscope*, May 9, 1916.

Malaria Simulating Diseases of the Mastoid and Frontal Sinuses, *Ann. Otol., Rhinol. and Laryngol.*, September, 1916; *Trans. Am. Otol. Soc.*, 1916.

Lymph Angioma of the Larynx, *The Laryngoscope*, January, 1918.

Syphilitic Necrosis of the Intermaxillary Bone, *Ann. Otol., Rhinol. and Laryngol.*, June, 1918.

Ear Protectors, *The Laryngoscope*, July, 1918,

Reconstruction of Defects of Hearing and Speech, *Ann. Am. Acad. Pol. and Soc. Sci.*, No. 1245, November, 1918.

The Unpleasant End-results of Well Performed Tonsillectomies, *Ann. Otol., Rhinol. and Laryngol.*, September, 1922.

The American Medical Association, *Atlantic Med. Journal*. Read before Gen. Session of the Med. Soc. of the State of Pennsylvania, October 2, 1923.

Problem of the Near Deaf and the Deaf, *Jour. Am. Med. Assn.*, 1925, lxxxiv, 1709.

Physical Causes of Deafness. Report of Com., Div. Medical Sciences, Nat. Research Council, *Arch. Otolaryngol.*, 1928, vii, 415.

President's Address (*Am. Laryngol. Assn.*, May 20, 1929), *Ann. Otol., Rhinol. and Laryngol.*, No. 2, p. 493.

Dr. Richardson also contributed articles of value to various standard works including "Diseases of the Eye, Ear, Nose and Throat," Posey and Wright, 1903; *The Reference Handbook of the Medical Sciences*, etc.

Executive Sessions
1930

FIRST EXECUTIVE SESSION

THURSDAY, MAY 22, 1930

THE meeting convened at 12.45 P.M., Thursday, May 22, 1930, President Lewis A. Coffin in the Chair.

Dr. Francis P. Emerson, of Boston, and Mr. V. E. Negus, of London, were elected Corresponding Fellows of the Association.

Dr. Robert F. Ridpath, of Philadelphia, was elected an Active Fellow of the Association.

The following Active Members of the Association were elected on the Nominating Committee: Dr. Henry L. Swain, Dr. J. Gordon Wilson, Dr. Arthur W. Proetz, Dr. Joseph H. Bryan and Dr. Norval H. Pierce.

Dr. Francis R. Packard and Arthur W. Proetz were appointed by the President as members of the Auditing Committee.

ANNUAL BUSINESS MEETING

FRIDAY, MAY 23, 1930

THE Annual Business Meeting of the Association was called to order by the President, Dr. Lewis A. Coffin, at 10.10 A.M., Friday, May 23, 1930, in the New Ocean House, Swampscott, Mass.

REPORT OF THE SECRETARY

At the Fifty-first Annual Congress of the American Laryngological Association in Atlantic City, N. J., fifty-six Fellows were registered. Dr. Charles W. Richardson presided and, as a guest of the Association, Prof. R. Adams Dutcher from State College took part in the scientific discussion.

Since the Annual Meeting of 1929 the Association has lost by death its President, Dr. Charles W. Richardson.

There are at present two Honorary Fellows, twenty Corresponding Fellows, eleven Emeritus Fellows, ninety-one Active Fellows and nineteen Associate Fellows.

The Annual Meeting of the Council was held at the University Club, New York City, the following members being in attendance: Drs. Coffin, Bryan, Fetterolf, Chamberlin, Mackenty and Coates.

The resignation of Dr. D. J. G. Wishart, of Toronto, was accepted with regret.

The Council recommended for Active Fellowship Dr. Robert F. Ridpath, of Philadelphia, and elected the following Associate Fellows: Dr. Edward Hastings Campbell, Philadelphia, Pa.; Dr. Frederick A. Figi, Rochester, Minn.; Dr. French K. Hansel, St. Louis, Mo.; Dr. Karl Musser Houser, Philadelphia, Pa.; Dr. Ernest M. Seydell, Wichita, Kansas.

The New Ocean House at Swampscott was selected as the place for the Fifty-second meeting on May 22, 23 and 24, following the meeting of the American Otological Society.

The Council also approved of a symposium on allergy for the current meeting.

The tentative plans that were being made for a combined meeting with the British Otolaryngologists in the fall of 1930 were

abandoned owing to advice received from Sir St. Clair Thomson that but a few of our British colleagues would be available for the meeting.

The Second Meeting of the Council was held at the New Ocean House, Swampscott, Mass., May 21, 1930, there being present Drs. Coffin, Bryan, Jackson, Mackenty, Chamberlin, Fetterolf, Fenton and Coates.

The resignation of Dr. Otto J. Stein of Chicago was accepted with regret.

Dr. Francis P. Emerson and Mr. V. E. Negus of London, England, were recommended to the Association for election to Corresponding Fellowship.

The following Fellows were elected as a new Candidates' Nominating Committee in accordance with a resolution passed by the Association at the Atlantic City meeting in 1929: Drs. Harris P. Mosher, Robert C. Lynch and Frank R. Spencer.

The Secretary was authorized to have printed a hundred extra copies of the *Transactions*, the entire 1929 edition having been exhausted with numerous unfilled orders.

Dr. Charles J. Imperatori was reelected Abstract Editor.

The Council recommends the following nominations for the Board of Governors of the American College of Surgeons: Dr. Joseph H. Bryan, Dr. Chevalier Jackson, Dr. Lee W. Dean.

A letter from the Secretary of the Congress of American Physicians and Surgeons notifies the Association that in the future, meetings of the Congress will be held at five-year intervals instead of every three years as has been the custom hitherto. Thus the next meeting of the Congress will be held in Washington, D. C., in 1933 instead of 1931.

Respectfully submitted,

GEORGE MORRISON COATES,

Secretary.

This report was accepted and ordered placed on file.

The Auditing Committee, Drs. Packard and Proetz, reported that they had examined the Treasurer's report and found it correct.

TREASURER'S REPORT TO MAY 10, 1930

There are the usual four funds upon which to report, the General Fund and the three Trust Funds.

1. THE GENERAL FUND

Before reading the figures of this year's report the Treasurer would like to call the attention of the Fellows to two rather interesting high spots.

1. This is the second year in which the reduction of the annual dues of Active Fellows from \$25.00 to \$20.00 has been in force. In spite of the diminution of 20 per cent in income from this source the unexpended balance in the treasury at the end of the current fiscal year is greater than ever before. It amounts to the safe and cheerful sum of \$3682.06, with, of course, all bills paid.

2. In listening to the ensuing figures, the Fellows will note that there has been received this year from the sale of *Transactions* \$713.50. This is an unprecedented sum. However, it is also an abnormal sum. The explanation is that the late appearance of the 1928 *Transactions* precluded their sale during their proper fiscal year and, therefore, the above-mentioned amount is derived from the sale of both the 1928 and the 1929 *Transactions*. In this connection the Fellows will be interested to learn that the issue of 1929 *Transactions* has been completely sold. The Secretary may mention this in his report, but he will not tell you that the increased sale of *Transactions* in recent years has been due largely to systematic and painstaking efforts on his part.

The figures:

Receipts

Balance in bank at last report	\$3180.54
Interest on bank deposit	65.94
1928 Dues:	
1 Active Fellow	20.00
1929 Dues:	
15 Active Fellows	300.00
1930 Dues:	
85 Active Fellows	\$1700.00
17 Associate Fellows	255.00
	<hr/>
	1955.00
Sale of <i>Transactions</i>	713.50
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Total	\$6234.98

Expenditures

Stenographer, 1929 Meeting	\$ 255.60
Stereopticon, 1929 Meeting	65.00
Dinner expenses, 1929 Meeting	30.00
Expenses, Dr. R. A. Dutcher	72.75
Secretary's expenses, printing, etc.	192.85
Treasurer's expenses, stationery, etc.	5.45
Treasurer's surety bond	42.50
Secretary's honorarium	300.00
Abstract Editor's honorarium	50.00
Treasurer's honorarium	100.00
Insurance on old volumes of <i>Transactions</i>	2.50
Flowers and night letter, Dr. Birkett's jubilee	15.60
Flowers for funeral of Dr. C. W. Richardson	16.00
Collection charge on Canadian check60
Printing, wrapping and mailing <i>Transactions</i>	1364.07
Refund of Active Fellow's dues	20.00
Refunds on <i>Transactions</i> not delivered	20.00
Total	\$2552.92
Balance in bank	\$3682.06

2. THE TRUST FUNDS

These three funds—the DeRoaldes, the Casselberry and the Johnston—are in excellent condition. Two of them have been drawn upon during the past year. From the Casselberry Fund was awarded the Casselberry Prize, the honorarium being divided between Dr. Edward H. Campbell, who is present with us this year as a newly elected Associate Fellow, and Drs. R. A. Kern and H. P. Schenck. From the Johnston Fund the Council of the Association appropriated the amount necessary to prepare and print the fifty-year Index to the *Transactions*. The DeRoaldes Fund alone of the three was untouched. The details:

(a) *The DeRoaldes Fund**Receipts*

Balance in bank at last report	\$800.12
Interest on deposit	32.00
Total	\$832.12

Expenditures

None

Balance in bank	\$832.12
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The original gift of Dr. DeRoaldes having been \$500.00, only the income of which may be used, there is at present in the Treasury of the Association \$332.12 of accumulated interest, more than sufficient wherewith to award medals for three years.

*(b) The Casselberry Fund**Receipts*

Balance in bank at last report	\$1348.70
Interest on \$6000 bonds	350.00
Interest on deposit	41.50
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Total	\$1740.20

Expenditures

Divided prize awarded to:

Dr. Edward H. Campbell	\$ 250.00
Drs. R. A. Kern and H. P. Schenck	250.00

Total	\$ 500.00
Balance in bank	\$1240.20

No essay has been submitted in competition this year, so that at the next annual meeting there will be in the Treasury of the Association money enough to award the Casselberry Prize three times over.

*(c) The Johnston Fund**Receipts*

Balance in bank at last report	\$506.56
Interest on \$5000 bonds	300.00
Interest on deposit	22.39
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Total	\$828.95

Expenditures

New York Academy of Medicine, indexing fifty years of <i>Transactions</i>	\$ 30.75
Wm. J. Dornan, printing fifty-year Index of <i>Transac- tions</i>	290.49
Total	\$321.24
Balance in bank	\$507.71

The income of this Fund, to be used unrestrictedly by the terms of the will of the farsighted founder, is an excellent financial "back log" for the Association. Already it has been of great usefulness, and undoubtedly it will be found to be of constant value as the years go on. There is at present the sum of \$507.71 at the disposal of the Association.

3. SUMMARY OF ASSETS AND LIABILITIES

Assets

General Fund:

Balance in bank	\$3682.06
Unpaid dues:	
One Active Fellow, 1929 . . . \$ 20.00	
Eight Active Fellows, 1930 . . 160.00	
Two Associate Fellows, 1930 . . 30.00	_____
	210.00
	_____ \$3892.06

De Roaldes Fund:

Original legacy	\$ 500.00
Unexpended income	332.12
	_____ 832.12

Casselberry Fund:

Original legacy	\$5000.00
Added principal	1000.20
Unexpended income	1240.00
	_____ 7240.20

Johnston Fund:

Original legacy	\$5000.00
Unexpended income	507.71
	_____ 5507.71

Total assets	\$17,472.09
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Liabilities

Unpaid bills	None
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One thought before closing! In 1926 the Treasurer, in his annual report, called attention to the increasing, unnecessarily increasing, surplus in the General Fund. To cope with this condition he suggested that the Association either pay the expenses of the members of the Council in attendance at the January meeting or vote a reduction in the annual dues. The matter was submitted to the Council for consideration and report, and that body recommended a reduction in the dues of the Active Fellows, which recommendation was later adopted.

The financial condition of the Association is now such that the Treasurer wishes to suggest that the matter of paying the expenses of the Councillors again be considered. No member of the Council begrudges the expenditure essential to the performance of his duties, but it would seem to be only fair, if finances would allow of it, that this charge, a charge incurred solely for the good of the Association, be met by the organization rather than by individuals.

To learn the approximate cost of such a plan the Treasurer has ascertained from the railroads the amount which the Association would have paid if this plan had been in force with the Council constituted as it was last year and is this year. The figures are as follows:

Had every member been present at the January 1929 Meeting the cost would have been \$405.82, and in 1930, \$401.38.

With the present balance in the Treasury, and with the Johnston Fund in the background, it is believed that the Association is now in a financial position to give thought to paying at least the railroad and Pullman fares of the Council members. The Treasurer suggests that this plan be considered and acted upon by the Association.

Respectfully submitted,

GEORGE FETTEROLF,
Treasurer.

This report was accepted and ordered placed on the records of the Association. After considerable discussion of that portion of the Treasurer's report recommending that traveling expenses of the Councillors in attendance at the Annual Meeting be paid by the Treasurer, Dr. Coakley moved that the railroad expenses and

hotel accommodations of those members of the Council attending the Annual Council meeting be paid out of the funds of the Association. This motion was seconded by Dr. Chamberlin and carried.

REPORT OF THE LIBRARIAN

The Librarian reports since the last meeting there have been added to the library fifty-two unbound pamphlets and one unbound volume. These include a number of reprints by Dr. Charles W. Richardson and contributed by Mrs. Richardson.

The Librarian was directed to ascertain whether there were any of the earlier *Transactions* in the Association library at the Library of the Surgeon-General U. S. A.

Colonel Garrison made a careful investigation and found there were no copies of the *Transactions* in the Association library.

Respectfully submitted,

JOSEPH H. BRYAN,
Librarian.

The Secretary reported, in the absence of the Librarian, that Dr. Bryan had asked him to inform the Association that he thinks that in a very short time the Association will have to find another depository for the Library as the Surgeon General's Library is becoming crowded and Dr. Bryan does not think that it will be possible for the Association to continue storing its manuscripts and books there.

This report was accepted and placed on file.

REPORT OF THE ABSTRACT EDITOR

The Abstract Editor has the honor to report that all the abstracted papers and discussions of the Fifty-first Congress of the American Laryngological Association were sent to the Editor-in-Chief of the *Archives of Otolaryngology*, August 5, 1929, and were published in that journal in the November issue, 1929.

Galley proofs were received the early part of October and sent to the following journals:

Dr. Logan Turner, Editor, *Journal of Laryngology and Otology*,
Edinburgh, Scotland.

Editor, *Clinica Oto Laringoratrira*, Rome, Italy.

Herr Prof. Dr. H. Hajek, Editor, *Monatsschrift für Ohrenheilkunde*, Vienna, Austria.

Dr. Thomas G. Atkinson, Editor, *The Ear, Nose and Throat Monthly*, Chicago, Illinois.

Prof. Fernand Le Maitre, Editor, *Archives Internationales de Laryngologie, Otologie, Rhinologie et Broncho-œsophagoscopie*, Paris, France.

Editor, *Acta Oto-laryngologica*, Hospital Sabbatsberg, Stockholm, Sweden.

These abstracts are read by those practising otolaryngology or the allied specialties, and to be of value should be published as soon as possible after the meeting of the Association. Of recent years they have appeared in the fall numbers of the American journals.

The honorarium of \$50.00 was received from the Treasurer and the necessary expenses paid.

Respectfully submitted,

CHARLES J. IMPERATORI,
Abstract Editor.

This report was read by the Secretary in the absence of Dr. Imperatori and was accepted and ordered placed on file.

ACTIVITIES OF THE AMERICAN SOCIETY FOR THE CONTROL OF CANCER FOR THE YEAR 1929

THE activities of the Society have been carried on for the past year with little change in the program. In October of 1929 Dr. Clarence Cook Little was selected as Managing Director of the Society and Dr. Raymond Voorhees Brokaw, who had been acting director, made Executive Secretary. The immediate affairs of the Society were placed in the hands of an Executive Committee of ten, which meets regularly once a month.

The distribution of literature to the medical profession and to the laity has been continued. Over 900,000 pieces have been sent out the past year, the great majority being to the general public. Many requests for information concerning the diagnosis of cancer, methods of and facilities for its treatment, the value of certain

reported "cures" and so on, have been received from physicians and the laity. These are answered and in specific instances the inquirer referred to some local authority on cancer.

The Canti film, showing the behavior of living tissues *in vitro* and the effect of radium upon normal and malignant cells, has been shown before many medical, scientific and educational groups in the United States, Canada, Porto Rico and Cuba. A film for the lay public, "This Great Peril," showing the danger of delay and of submitting to quacks for treatment, has been shown before several audiences.

The survey of the facilities for cancer treatment in Philadelphia, as part of the Philadelphia Hospital and Health Survey of 1929, has been carried on by the field representative of the Society and the report has been published. The crude cancer death-rate in 1928 in Philadelphia was found to be 125.2 per 100,000 of the population; no scarcity of beds for cancer patients exists; 4.2 per cent of the total admissions in forty-two hospitals were classed as early cancer; only nine hospitals possessed, in 1929, the complete physical equipment to handle all types of cancer cases and these nine institutions together owned 5134 milligrams of radium. Eight hospitals report research in progress, but it is organized in only two of these. Specific recommendations were made that all general hospitals with a cancer service adopt the standards set forth by the American Society for the Control of Cancer and affiliate with a Cancer Institute.

A survey of the facilities for cancer treatment and diagnosis in Delaware was completed by the field representative during the year.

A very intensive study of the Steiner Clinic in Atlanta, Georgia, for the treatment of cancer also has been made by the Society's field representative and a state-wide program with clinics over the state working in conjunction with the Steiner Clinic as the center, has been completed and actually inaugurated.

The organization of several of the states on the tripartite plan, which includes the State Medical Society, the State Health Department and the Society has continued.

Respectfully submitted,

D. BRYSON DELAVAN,
Chairman.

This report was read by the Secretary in the absence of Dr. Delavan and was accepted with thanks.

REPORT OF THE NOMINATING COMMITTEE

Dr. Henry L. Swain reported as the selection of the Nominating Committee the following names as Nominees for Directors of the Association for the ensuing year:

Dr. Francis R. Packard, Philadelphia, Pa.

Dr. Henry Hall Forbes, New York, N. Y.

Dr. James A. Babbitt, Philadelphia, Pa.

Dr. George M. Coates, Philadelphia, Pa.

Dr. George Fetterolf, Philadelphia, Pa.

Dr. Joseph H. Bryan, Washington, D. C.

Dr. Chevalier Jackson, Philadelphia, Pa.

Dr. D. Bryson Delavan, New York, N. Y.

Dr. Herbert S. Birkett, Montreal, Canada.

Dr. Lewis A. Coffin, New York, N. Y.

Additional nominations from the floor were Dr. Harris P. Mosher and Dr. John F. Barnhill. It was moved, seconded and carried that the nominations be closed. Dr. Coakley then moved that the Nominating Committee's report be accepted and placed on file and the Committee be discharged with thanks. This motion was seconded and carried.

REPORT OF THE AMERICAN BOARD OF OTOLARYNGOLOGY

Examinations have been held by the Board during the past year prior to the American Medical Association meeting in July, 1929, in Portland, Oregon, with about 60 candidates; and prior to the American Academy of Ophthalmology and Otolaryngology meeting in October, 1929, in Philadelphia, with about 120 candidates. Approximately 1400 candidates have been examined, and the percentage of failure ranges from 15 to 17. It is interesting to note that many candidates who become aware of deficiencies in their training at the examination have reappeared within three years and have secured their certificates with excellent grades.

Many hospital staffs and certain local societies have now set up the certificate of this Board as a prerequisite to membership;

their endeavor to foster improved standards in otolaryngology has been more active in the West than along the Atlantic seaboard, and merits the attention of teachers and clinicians in this Association.

Respectfully submitted,

HARRIS P. MOSHER,
ROSS H. SKILLERN.

This report was accepted and ordered placed on file.

REPORT OF THE BIOGRAPHER AND HISTORIAN

This report consisted of the Memorial of the late Dr. Charles W. Richardson which was read by the Secretary and will be found under the head of Memorials.

MISCELLANEOUS BUSINESS

The recommendation of the Council that Drs. Joseph H. Bryan, Chevalier Jackson and Lee W. Dean be nominated for membership on the Board of Governors of the American College of Surgeons for the term expiring 1933 was approved.

A resolution approving of the work of the U. S. Commission for the Celebration of the Two-hundredth Anniversary of the birth of George Washington was approved and the Secretary directed to notify the Commission of this action.

Dr. Chamberlin moved that the provision for the competitions for the Casselberry Prize be more widely advertised to the profession and that the Secretary be instructed that these provisions be published in the three leading American journals in this specialty as well as in the Journal of the American Medical Association. This motion was seconded by Dr. Barnhill and was carried.

FINAL EXECUTIVE SESSION

SATURDAY, MAY 24, 1930

THE final Executive Session convened at 11.30 A.M., First-Vice-President, Dr. William B. Chamberlin, in the Chair.

A motion to reconsider the action of the preceding day in regard to the payment by the Association of the railroad and hotel bills of members of the Council attending the Annual Council Meeting was made by Dr. Barnhill and seconded by Dr. Fenton. This motion was carried. Dr. Barnhill then moved that the members of the Council be reimbursed for the actual railroad and pullman fare only to and from the Annual Meeting of the Council. This was seconded by Dr. Fenton and carried.

Of those nominated for Directors of the Association the following ten were elected by ballot and immediately organized with the following officers:

Dr. Francis R. Packard, President,
Dr. Harris P. Mosher, First Vice-President,
Dr. James A. Babbitt, Second Vice-President,
Dr. George M. Coates, Secretary,
Dr. George Fetterolf, Treasurer,
Dr. John F. Barnhill, Librarian,

and as additional members of the Council, Drs. Chevalier Jackson, D. Bryson Delavan, Herbert S. Birkett and Lewis A. Coffin.

After Dr. Francis R. Packard had been escorted to the Chair by Dr. Pierce and Dr. Fenton, Dr. Chamberlin presented him with the gavel of the Association and the meeting for the year was declared adjourned.

**List of Fellows
1930**

LIST OF FELLOWS

1930

Attendance during last five years shown by figures following each name.

Asterisk (*) indicates year paper was read.

Active Fellows

1917. ARROWSMITH, HUBERT, 11 Schermerhorn Street, Brooklyn, New York, 1921, '22, '24, '25, '26, '27, '29, '30.
1923. BABBITT, JAMES A., 1912 Spruce Street, Philadelphia, Pennsylvania, 1923, '24*, '25, '26, '27, '28, '29*, '30.
1929. BARLOW, ROY ALEXANDER, 403 Medical Arts Building, Rochester, New York, 1923, '24*, '28, '29*, '30*.
1915. BARNES, HARRY ALDRICH, 520 Commonwealth Avenue, Boston, Massachusetts, 1920, '24, '27*, '28, '30.
1908. BARNHILL, JOHN F., 408 Pennway Building, Indianapolis, Indiana, 1922, '24, '27, '28, '29, '30.
1928. BECK, JOSEPH C., 2551 North Clark Street, Chicago, Illinois, 1928, '30.
1921. BERRY, GORDON, 36 Pleasant Street, Worcester, Massachusetts, 1921*, '22, '23, '24, '25, '26, '27, '28*, '29*, '30.
1893. BIRKETT, HERBERT S., 1190 Mountain Street, Montreal, Canada, 1920, '21, '22*, '23, '25, '26, '27, '28*, '29, '30.
1925. BLAIR, VILRAY P., 400 Metropolitan Building, St. Louis, Missouri, 1922, '23*, '25*, '28*.
1891. BRYAN, JOSEPH H., 303 Stoneleigh Court, Washington, D. C., 1920*, '21, '22, '23, '24, '25, '26, '27*, '28, '29, '30.
1913. BUTLER, RALPH, 1930 Chestnut Street, Philadelphia, Pennsylvania, 1920*, '21, '23, '24, '27, '29.
1915. CANFIELD, R. BISHOP, Ann Arbor, Michigan, 1921, '22.
1927. CARMODY, THOMAS E., 806 Metropolitan Building, Denver, Colorado, 1924, '26*, '27, '28, '30.
1917. CHAMBERLIN, WILLIAM B., Osborn Building, Cleveland, Ohio, 1920, '21*, '22, '26, '27*, '28, '29, '30*.
1897. CLARK, J. PAYSON, 71 Marlborough Street, Boston, Massachusetts, 1920*, '21, '22, '23, '24*, '26, '28.
1929. CLERF, LOUIS H., 128 South Tenth Street, Philadelphia, Pennsylvania, 1925, '27*, '28, '29*, '30.

1902. COAKLEY, CORNELIUS G., 20 East Fifty-third Street, New York, 1920, '21, '22*, '23, '24, '25, '27, '29, '30.
1917. COATES, GEORGE MORRISON, 1721 Pine Street, Philadelphia, Pennsylvania, 1920, '21, '22, '23, '24, '25, '26, '27, '28, '29, '30*.
1905. COFFIN, LEWIS A., 114 East Fifty-fourth Street, New York, 1920, '21, '22, '23, '25, '26, '27, '28, '29, '30*.
1913. COFFIN, ROCKWELL A., 350 Commonwealth Avenue, Boston, Massachusetts, 1920, '21, '22*, '24, '30.
1913. DABNEY, VIRGINIUS, 1633 Connecticut Avenue, Washington, D. C., 1921*, '22, '24*, '25, '27*, '28*.
1918. DEAN, LEE WALLACE, School of Medicine, Washington University, St. Louis, Missouri, 1920*, '21*, '22, '23, '24, '25*, '26, '27*, '28, '29*.
1881. DELAVAN, D. BRYSON, 30 East Sixtieth Street, New York, 1920*, '21*, '22*, '23*, '25*, '26*, '27*, '28*, '29*.
1926. DENNIS, FRANK LOWNDES, Ferguson Building, Colorado Springs, Colorado, 1925*, '26, '27, '28, '30.
1919. FAULKNER, E. ROSS, 101 East Fifty-eighth Street, New York, 1920, '21, '22, '23*, '25, '27, '28, '30.
1926. FENTON, RALPH ALBERT, 806 Medical Arts Building, Tenth and Taylor Streets, Portland, Oregon, 1923, '25*, '26, '28, '30.
1914. FETTEROLF, GEORGE, 2010 Spruce Street, Philadelphia, Pennsylvania, 1920, '21, '22, '23*, '24, '25, '26, '27, '28, '29*, '30.
1922. FORBES, HENRY HALL, 40 East Sixty-first Street, New York, 1922, '25*, '27, '28, '29.
1897. FROTHINGHAM, RICHARD, 666 Madison Avenue, New York.
1922. GOLDSMITH, PERRY G., 84 Carlton Street, Toronto, Ontario, 1922*, '23*, '24, '26, '28.
1913. GOLDSTEIN, MAX A., 3858 Westminster Place, St. Louis, Missouri, 1920, '21, '22, '25*, '26, '27, '28, '29, '30.
1898. GOODALE, JOSEPH L., 258 Beacon Street, Boston, Massachusetts, 1920, '21, '22*, '23, '24, '26.
1905. GRAYSON, CHARLES P., 262 South Fifteenth Street, Philadelphia, Pennsylvania, 1921.
1906. GREENE, D. CROSBY, JR., 23 Bay State Road, Boston, Massachusetts, 1920*, '21*, '22, '23, '24*, '25, '26, '27.
1917. GREENE, JOSEPH B., 305 Haywood Building, Asheville, North Carolina, 1921, '22, '23*, '24, '26, '27, '28, '29*.

1905. HALSTED, THOMAS H., 906 Chimes Building, Syracuse, New York, 1920, '21, '23, '24, '25, '26.
1903. HARRIS, THOMAS J., 104 East Fortieth Street, New York, 1921, '22, '24*, '25, '26, '28, '29, '30.
1915. HASTINGS, HILL, 306 Medical Office Building, 1136 West Sixth Street, Los Angeles, California, 1921*, '24, '27*, '30*.
1919. HURD, LEE MAIDMENT, 39 East Fiftieth Street, New York, 1921, '23, '25, '26*, '28.
1925. IGLAUER, SAMUEL, 400 Pearl Market Bank Building, Cincinnati, Ohio, 1926, '27, '30.
1920. IMPERATORI, CHARLES JOHNSTONE, 17 East Thirty-eighth Street, New York, 1921*, '22, '24*, '25*, '26*, '27*, '28.
1904. INGERSOLL, JOHN M., Aladdin Building, Miami Beach, Florida, 1920, '21, '22, '23, '24, '25, '26, '27, '28, '29.
1907. JACKSON, CHEVALIER, 235 South Fifteenth Street, Philadelphia, Pennsylvania, 1921*, '22*, '23, '24*, '25*, '27*, '28, '29, '30.
1918. KENYON, ELMER L., 104 South Michigan Boulevard, Chicago, Illinois, 1922, '24*, '28.
1921. KERNAN, JOHN D., JR., 156 East Seventy-ninth Street, New York, 1921*, '22, '25, '26, '27, '29.
1929. KISTNER, FRANK B., 600 Mayer Building, Portland, Oregon, 1930.
1927. LARGE, SECORD HERBERT, 2323 Prospect Avenue, Cleveland, Ohio, 1924*, '25, '26.
1914. LEVY, ROBERT, Metropolitan Building, Denver, Colorado.
1918. LEWIS, FIELDING OTIS, 259 South Seventeenth Street, Philadelphia, Pennsylvania, 1921*, '22*, '23, '25*, '28, '29.
1925. LILLIE, HAROLD I., Mayo Clinic, Rochester, Minnesota, 1923*, '24*, '25, '26*, '28.
1911. LOCKARD, LORENZO B., Metropolitan Building, Denver, Colorado.
1915. LYNCH, ROBERT CLYDE, 632 Maison Blanche Building, New Orleans, Louisiana, 1920*, '21, '22, '23, '24*, '26, '28, '29*. *died June 1930*
1914. MACKENTY, JOHN E., 111 East Sixty-first Street, New York, 1920*, '21, '22*, '23*, '25*, '26*, '27*, '28*, '29, '30. *died Dec. 1931*
1928. MACPHERSON, DUNCAN, 114 East Fifty-fourth Street, New York, 1928, '29, '30.
1921. McCULLAGH, SAMUEL, 71 Park Avenue, New York, 1921*, '22, '23*, '25, '26*, '27, '28.
1927. MCGINNIS, EDWIN, 104 South Michigan Boulevard, Chicago, Illinois, 1924, '25*, '26, '27, '28, '29, '30*.

1913. McKIMMIE, OSCAR A. M., 1301 Massachusetts Avenue, N. W. Washington, D. C., 1921*, '22, '25, '28.
1929. McKINNEY, RICHMOND, 899 Madison Avenue, Memphis, Tennessee, 1923, '24*, '26*, '27, '29.
1904. MOSHER, HARRIS P., 828 Beacon Street, Boston, Massachusetts, 1920, '21*, '22, '23*, '24, '25, '26, '27, '28*, '29, '30.
1925. MULLIN, WILLIAM V., Cleveland Clinic, Euclid Avenue at Ninety-third Street, Cleveland, Ohio, 1923, '24*, '25, '26, '27, '28*, '30.
1914. MUNGER, CARL E., 81 North Main Street, Waterbury, Connecticut, 1921, '27.
1901. MYLES, ROBERT C., 1075 Park Avenue, New York, 1923, '25, '28.
1922. NEW, GORDON B., Mayo Clinic, Rochester, Minnesota, 1922, '24, '25*, '26*, '28*.
1926. NEWHART, HORACE, 527 Medical Arts Building, Minneapolis, Minn., 1923, '25*, '26, '28, '30.
1903. PACKARD, FRANCIS R., 302 South Nineteenth Street, Philadelphia, Pennsylvania, 1920, '21, '22, '23, '24, '25, '26, '29, '30.
1906. PIERCE, NORVAL H., Del Mar, San Diego Co., California, 1920*, '21, '22, '23, '24, '25, '26*, '27*, '28, '30.
1927. PROETZ, ARTHUR WALTER, Beaumont Building, St. Louis, Missouri, 1924, '25*, '26*, '27, '28, '29, '30*.
1908. RANDALL, B. ALEXANDER, 2320 Delancey Street, Philadelphia, Pennsylvania, 1920*, '21, '22, '23, '25, '28.
1903. RENNER, W. SCOTT, 341 Linwood Avenue, Buffalo, New York, 1920, '22, '24, '26, '28.
1884. RICE, CLARENCE C., 52 Park Avenue, New York.
1905. RICHARDS, GEORGE L., 124 Franklin Street, Fall River, Massachusetts, 1920, '21, '24, '27, '28, '30.
1930. RIDPATH, ROBERT F., Central Medical Building, Eighteenth and Chestnut Streets, Philadelphia, Pa., 1930.
1913. ROY, DUNBAR, Grand Opera House Block, Atlanta, Georgia, 1921*, '22, '23, '26, '28.
1921. SAUER, WILLIAM E., 537 North Grand Avenue, St. Louis, Missouri, 1921, '23, '25, '28.
1923. SEWALL, EDWARD CECIL, 3515 Pacific Avenue, San Francisco, California, 1924, '26*, '28*.
1907. SHAMBAUGH, GEORGE E., 122 South Michigan Boulevard, Chicago, Illinois, 1920*, '22, '23, '24, '26*, '27, '28, '29, '30.

1909. SHURLY, BURT R., 62 Adams Avenue W., Detroit, Michigan, 1920, '21*, '22, '24, '25*, '26, '27*, '28, '29*, '30.
1919. SKILLERN, ROSS H., 1811 Spruce Street, Philadelphia, Pennsylvania, 1920*, '21*, '22*, '23, '24, '25, '26, '27, '28, '29, '30*.
1908. SMITH HARMON, 150 East Sixty-second Street, New York, 1920, '21, '22*, '23, '25, '26, '28.
1928. SONNENSCHN, ROBERT, 180 North Michigan Avenue, Chicago, Illinois, 1928, '29*, '30.
1926. SPENCER, FRANK ROBERT, Physicians' Building, Boulder, Colo., 1923, '24*, '26, '27*, '28, '30.
1911. STOUT, GEORGE C., 2015 Walnut Street, Philadelphia, Pennsylvania, 1922, '23, '25, '27, '28.
1889. SWAIN, HENRY L., Colonial Building, 195 Church Street, New Haven, Connecticut, 1920, '21*, '22, '23, '24, '25, '26, '27, '28, '29, '30*.
1928. TAYLOR, HERMON MARSHALL, 111 West Adams Street, Jacksonville, Florida, 1924, '25*, '26, '27, '28.
1903. THEISEN, CLEMENT F., 172 Washington Avenue, Albany, New York, 1920*, '23*, '24*, '28*, '30.
1929. TUCKER, GABRIEL, 326 South Nineteenth Street, Philadelphia, Pennsylvania, 1925, '26*, '27*, '29, '30.
1892. WAGNER, HENRY L., 518 Sutter Street, San Francisco, California, '26.
1892. WATSON, ARTHUR W., 2013 Locust Street, Philadelphia, Pennsylvania, 1921, '23, '25, '27.
1928. WHERRY, WILLIAM P., 620 Bankers' Reserve Life Building, Omaha, Nebraska, 1924, '25*, '26, '27, '28, '29, '30.
1925. WHITE, FRANCIS W., 174 West Fifty-eighth Street, New York City, New York, 1922, '23*, '25, '26, '27, '28, '29.
1908. WILSON, J. GORDON, 5755 Kenwood Avenue, Chicago, Illinois, 1920, '21, '22, '24, '25, '26, '28, '29, '30.
1905. WOOD, GEORGE B., Suite 504, Physicians' Building, Northeast corner Twentieth and Chestnut Streets, Philadelphia, Pennsylvania, 1921, '22*, '23*, '25*, '28, '29.

Associate Fellows

1929. ALDEN, ARTHUR MAXWELL, 537 Frisco Building, St. Louis, Missouri, 1930.
1929. ARBUCKLE, MILLARD F., 1011 Beaumont Medical Building, St. Louis, Missouri, 1929.

- 1930. CAMPBELL, EDWARD HASTINGS, 2117 Chestnut Street, Philadelphia, Pa., 1930.
- 1930. FIGI, FREDERICK A., Mayo Clinic, Rochester, Minn., 1930.
- 1928. GITTINS, THOMAS ROY, 401 Davidson Building, Sioux City, Iowa, 1928.
- 1928. GOODYEAR, HENRY M., 556 Doctors' Building, Cincinnati, Ohio, 1928, '29, '30*.
- 1930. HANSEL, FRENCH K., 1045 Missouri Building, St. Louis, Missouri, 1930.
- 1928. HILL, FREDERICK THAYER, Professional Building, Waterville, Maine, 1928, '29*, '30*.
- 1930. HOUSER, KARL MUSSER, 1826 Pine Street, Philadelphia, Pa., 1930.
- 1928. JARVIS, DE FOREST CLINTON, Room 7, Quarry Bank Building, Barre, Vermont, 1928, '30.
- 1925. KING, JAMES JOSEPH, 40 East Forty-first Street, New York, N. Y., 1925, '26*, '27, '28, '29*.
- 1927. LUKENS, ROBERT M., 1923 Spruce Street, Philadelphia, Pennsylvania, 1927, '28.
- 1929. LYMAN, HARRY WEBSTER, 700 Carleton Building, St. Louis, Missouri, 1929.
- 1927. MYERSON, MERVIN C., 198 Lincoln Place, Brooklyn, New York, 1927, '28*, '29, '30.
- 1927. ORTON, HENRY BOYLAN, 24 Commerce Street, Newark, New Jersey, 1927, '28, '29, '30*.
- 1927. PORTER, CHAS. T., 520 Commonwealth Ave., Boston, Mass., 1927, '28, '30.
- 1930. SEYDELL, ERNEST M., First National Bank Building, Wichita, Kansas, 1930.
- 1928. SHEA, JOHN JOSEPH, 1018 Madison Avenue, Memphis, Tennessee, 1928, '29, '30*.
- 1927. TOBEY, HAROLD G., 270 Commonwealth Avenue, Boston, Mass., 1927, '29*, '30*.

Honorary Fellows

- 1910. MOURE, E. J., 25 bis Cours de Verdun, Bordeaux, France.
- 1914. THOMSON, SIR ST. CLAIR, 64 Wimpole Street, W., London, England.

Corresponding Fellows

- 1901. COLLIER, MAYO, Kearsney Abbey, Kent, England.
- 1893. DESVERNINE, CARLOS M., 52 Cuba Street, Havana, Cuba.

- 1930. EMERSON, FRANCIS P., Franklin, Mass.
- 1901. GRANT, SIR JAMES DUNDAS, K.B.E., 148 Harley Street, London, W 1, England.
- 1919. KELLY, ADAM BROWN, 26 Blythswood Square, Glasgow, Scotland.
- 1926. LAW, FREDERICK M., 576 Fifth Avenue, New York City, New York.
- 1921. LE MAITRE, FERNAND, Avenue Victor Hugo, 120 (XVI), Paris, France.
- 1903. MCBRIDE, P., 20 South Drive, Harrowgate, England.
- 1919. MCKERNON, JAMES F., 636 Fifth Avenue, New York.
- 1896. MACDONALD, GREVILLE, Wildwood, Haslemere, England.
- 1920. MACKENZIE, DAN, 9 Weymouth Street, Portland Place, London, W 1, England.
- 1930. NEGUS, VICTOR E., 133 Harley Street, London, W 1, England.
- 1919. PATERSON, DONALD ROSE, 15 St. Andrew's Crescent, Cardiff, Wales.
- 1919. ROGERS, JOHN, JR., 177 East Seventy-first Street, New York.
- 1924. SCHAEFFER, J. PARSONS, Jefferson Medical College, Philadelphia.
- 1896. SCHMIEGELOW, ERNST, 18 Norregade, Copenhagen, Denmark.
- 1903. TILLEY, HERBERT, 72 Harley Street, Cavendish Square, London, W., England.
- 1913. TURNER, LOGAN, 27 Walker Street, Edinburgh, Scotland.
- 1901. WINGRAVE, WYATT, Lyme Regis, Dorset, England.

Emeritus Fellows

- 1914. BROWN, J. PRICE, 29 Edgewood Cres., Rosedale, Toronto, Canada.
- 1922. COBB, FREDERICK C., Riverview, Gloucester, Massachusetts.
- 1928. COOLIDGE, ALGERNON, 198 Beacon Street, Boston, Massachusetts.
- 1918. FARLOW, JOHN W., 127 Bay State Road, Boston, Massachusetts.
- 1924. FREER, OTTO T., 81 East Elm Street, Chicago, Illinois.
- 1927. HARDIE, T. MELVILLE, 30 N. Michigan Boulevard, Chicago, Illinois.
- 1918. HINKEL, F. WHITEHALL, 28 Norwood Avenue, Buffalo, New York.
- 1926. HOPKINS, FREDERICK E., 146 Chestnut Street, Springfield, Massachusetts.

1927. HUBBARD, THOMAS, 234 Michigan Street, Toledo, Ohio.
1912. LINCOLN, WILLIAM, Wade Park Manor, Cleveland, Ohio.
1929. MAYER, EMIL, 320 West Eighty-ninth Street, New York, N. Y.

Recipient of the De Roaldes Award

1928. DR. CHEVALIER JACKSON.

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.

Benefactors

ARTHUR W. DE ROALDES.
WILLIAM E. CASSELBERRY.
SAMUEL JOHNSTON.

PAST OFFICERS

Presidents

(Year of Election)

1879. Louis Elsberg	1905. J. W. Gleitsmann
1880. J. Solis-Cohen	1906. A. W. de Roaldes
1881. F. I. Knight	1907. H. S. Birkett
1882. G. M. Lefferts	1908. A. Coolidge, Jr.
1883. F. H. Bosworth	1909. J. E. Logan
1884. E. L. Shurly	1910. D. Braden Kyle
1885. Harrison Allen	1911. James E. Newcomb
1886. E. Fletcher Ingals	1912. George A. Leland
1887. R. P. Lincoln	1913. Thomas Hubbard
1888. E. C. Morgan	1914. Alexander W. MacCoy
1889. J. N. Mackenzie	1915. G. Hudson Makuen
1890. W. C. Glasgow	1916. Joseph L. Goodale
1891. S. W. Langmaid	1917. Thomas H. Halsted
1892. M. J. Asch	1918. Cornelius G. Coakley
1893. D. B. Delavan	1919. Norval H. Pierce
1894. J. O. Roe	1920. Harris P. Mosher
1895. W. H. Daly	1921. Harmon Smith
1896. C. H. Knight	1922. Emil Mayer
1897. T. R. French	1923. J. Payson Clark
1898. W. E. Casselberry	1924. Lee Wallace Dean
1899. Samuel Johnston	1925. Greenfield Sluder
1900. H. L. Swain	1926. Chevalier Jackson
1901. J. W. Farlow	1927. D. Bryson Delavan
1902. J. H. Bryan	1928. Charles W. Richardson
1903. J. H. Hartman	1929. Lewis A. Coffin
1904. C. C. Rice	

Vice-Presidents

1879. F. H. Davis	J. O. Roe
1880. W. C. Glasgow	W. Porter
1881. E. L. Shurly	E. F. Ingals
1882. C. Seiler	S. Johnston
1883. S. W. Langmaid	W. H. Daly
1884. J. H. Hartman	G. W. Major
1885. H. A. Johnson	J. N. Mackenzie
1886. E. C. Morgan	S. W. Langmaid
1887. J. N. Mackenzie	C. E. DeM. Sajous
1888. W. C. Glasgow	

1889.	E. Holden	C. E. Bean
1890.	J. O. Roe	J. H. Hartman
1891.	M. J. Asch	S. Johnston
1892.	S. Johnston	J. C. Mulhall
1893.	J. C. Mulhall	W. E. Casselberry
1894.	C. C. Rice	S. H. Chapman
1895.	J. Wright	A. W. de Roaldes
1896.	T. M. Murray	D. N. Rankin
1897.	A. W. MacCoy	H. S. Birkett
1898.	J. W. Farlow	F. W. Hinkel
1899.	T. A. De Blois	M. R. Brown
1900.	H. L. Wagner	A. A. Bliss
1901.	J. W. Gleitsmann	D. Braden Kyle
1902.	G. A. Leland	T. Melville Hardie
1903.	J. H. Lowman	W. Peyre Porcher
1904.	Thomas Hubbard	W. J. Freeman
1905.	J. L. Goodale	C. W. Richardson
1906.	G. H. Makuen	A. B. Thrasher
1907.	J. P. Clark	J. E. Rhodes
1908.	E. Mayer	F. R. Packard
1909.	C. G. Coakley	H. P. Mosher
1910.	Robert C. Myles	J. M. Ingersoll
1911.	F. C. Cobb	B. R. Shurly
1912.	A. W. Watson	W. Scott Renner
1913.	F. E. Hopkins	Geo. E. Shambaugh
1914.	Clement F. Thiesen	Lewis A. Coffin
1915.	J. Gordon Wilson	Christian R. Holmes
1916.	Thomas H. Halsted	Greenfield Sluder
1917.	John Edwin Rhodes	D. Crosby Greene
1918.	G. E. Shambaugh	John R. Winslow
1919.	Francis R. Packard	Harmon Smith
1920.	Harmon Smith	W. B. Chamberlin
1921.	Dunbar Roy	Robert C. Lynch
1922.	George Fetterolf	Lorenzo B. Lockard
1923.	Hubert Arrowsmith	Joseph B. Greene
1924.	Ross H. Skillern	Gordon Berry
1925.	John E. Mackenty	Robert Levy
1926.	Lewis A. Coffin	William V. Mullin
1927.	Charles W. Richardson	Hill Hastings
1928.	Robert Clyde Lynch	Francis P. Emerson
1929.	William B. Chamberlin	Ralph Albert Fenton

Secretaries and Treasurers

1879. G. M. Lefferts	1895. H. L. Swain
1882. D. B. Delavan	1900. J. E. Newcomb
1889. C. H. Knight	1911. Harmon Smith

Secretaries

1912. Harmon Smith	1919. J. M. Ingersoll
1918. D. B. Delavan	1920. George M. Coates

Treasurers

1912. J. Payson Clark	1923. George Fetterolf
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Librarians

1879. F. H. Bosworth	1903. J. H. Bryan
1883. T. R. French	1930. John F. Barnhill

PAST MEMBERS

DeceasedHonorary Fellows*

- *1915. Bosworth, Francke H., New York City.
- *1914. Cohen, J. Solis-, Philadelphia, Pennsylvania.
- *1893. De Roaldes, Arthur, New Orleans, La.
- *1914. De La Sota Y Lastra, Ramon, Seville, Spain.
- *1880. Garcia, Senor Manuel, London, England.
- *1928. French, Thomas R., Brooklyn, N. Y.
- *1924. Johnston, Samuel, Baltimore, Md.
- *1914. Lefferts, George Morewood, Katona, N. Y.
- *1922. MacKenzie, John N., Baltimore, Md.
- *1883. Mackenzie, Sir Morell, London, England.
- *1910. Massei, Ferdinand, Naples, Italy.
- *1879. Oliver, H. K., Boston, Mass.
- *1909. Semon, Sir Felix, Great Missenden, England.
- *1914. Wagner, Clinton, New York City.
- *1921. Wright, Jonathan, Pleasantville, N. Y.

Corresponding Fellows

- *1892. Browne, Lennox, London, England.
- *1887. Gouguenheim, A., Paris, France.
- *1894. Holden, Edgar, Newark, N. J.
- *1881. Labus, Carlo, Milan, Italy.
- *1902. Lermoye, Marcel, Paris, France.
- *1897. Luc, H., Paris, France.
- *1894. MacIntyre, John, Glasgow, Scotland.
- *1880. Meyer, Wilhelm, Copenhagen, Denmark.
- *1896. Mygind, Holger, Copenhagen, Denmark.
- *1894. Sajous, C. E. DeM., Philadelphia, Pa.
- *1881. Thornton, Pugin, London, England.
- *1880. Whistler, W. McNeil, London, England.
- *1894. Wolfenden, R. Norris, Kent, England.

Emeritus Fellows

- *1914. De Blois, Thomas Amory, Boston, Mass.
- *1914. Gleitsmann, Joseph W., New York City.
- *1912. Knight, Charles H., New York City.
- *1923. Leland, George A., Boston, Massachusetts.

- *1922. MacCoy, Alexander W., Philadelphia, Pa.
- *1914. Porter, William, Ocean Springs, Miss.
- *1924. Rhodes, John Edwin, Chicago, Ill.
- *1914. Robinson, Beverley, New York City.

Active Fellows

- *1880. Allen, Harrison, Philadelphia, Pa.
- *1878. Asch, Morris J., New York.
- *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.
- *1889. Casselberry, William E., Chicago, Ill.
- *1883. Chamberlain, C. W., Hartford, Conn.
- *1896. Chappell, W. F., New York.
- *1882. Chapman, S. Hartwell, New Haven, Conn.
- 1918. Cox, Gerard H., New York.
- 1880. Cushing, E. W., Boston, Mass.
- *1878. Cutter, Ephraim, West Falmouth, Mass.
- *1880. Daly, W. H., Pittsburgh, Pa.
- *1878. Davis, F. H., Chicago, Ill.
- *1901. Dickerman, E. T., Chicago, Ill.
- *1878. Donaldson, Frank, Baltimore, Md.
- *1901. Freeman, Walter J., Philadelphia, Pa.
- *1917. Friedberg, Stanton A., Chicago, Ill.
- 1901. Getchell, Albert C., Worcester, Mass.
- *1907. Gibb, Joseph S., Philadelphia, Pa.
- *1878. Glasgow, William Carr, St. Louis, Mo.
- *1878. Hartman, Jacob H., Baltimore, Md.
- *1879. Holden, Edgar, Newark, N. J.
- *1907. Holmes, Christian R., Cincinnati, Ohio.
- *1882. Hooper, Franklin H., Boston, Mass.
- *1893. Hope, George B., New York City.
- *1901. Hyatt, Frank, Washington, D. C.
- *1882. Ives, Frank L., New York City.
- *1878. Ingals, E. Fletcher, Chicago, Ill.
- *1880. Jarvis, William C., New York City.
- *1878. Johnson, Hosmer A., Chicago, Ill.
- *1879. Johnson, Woolsey, New York City.
- *1879. Kealhofer, R. H., St. Louis, Mo.
- *1901. King, Gordon, New Orleans, La.

- *1885. Knight, Charles H., New York City.
- *1878. Knight, Frederick Irving, Boston, Mass.
- *1898. Kyle, D. Braden, Philadelphia, Pa.
- *1880. Langmaid, Samuel W., Boston, Mass.
- *1878. Lincoln, Rufus P., New York City.
- *1913. Loeb, Hanau W., St. Louis, Missouri.
- *1897. Logan, James E., Kansas City, Missouri.
- *1888. Lowman, John H., Cleveland, Ohio.
- *1919. Lynah, Henry L., New York City.
- *1879. McBurney, Charles, New York City.
- *1885. McSherry, H. Clinton, Baltimore, Md.
- *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.
- *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.
- *1880. 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.
- *1878. Rumbold, T. F., St. Louis, Mo.
- *1879. Seiler, Carl, Philadelphia, Pa.
- *1893. Shields, Charles M., Richmond, Va.
- *1878. Shurley, E. L., Detroit, Mich.
- *1909. Sluder, Greenfield, St. Louis, Mo.
- *1879. Smith, Andrew H., Geneva, N. Y.
- *1892. Simpson, William Kelly, New York City.
- 1924. Stein, Otto J., Chicago, Ill.
- *1879. Tauber, Bernhard, Cincinnati, Ohio.
- *1899. Thorner, Max, Cincinnati, Ohio.
- *1892. Thrasher, Allen B., Cincinnati, Ohio.
- *1888. Van der Poel, S. O., New York City.
- 1896. Ward, Marshall R., Pittsburgh, Pa.
- *1879. Ward, Whitfield, New York City.
- *1886. Westbrook, Benjamin F., Brooklyn, N. Y.
- *1924. White, Leon E., Boston, Mass.
- 1922. Wishart, David J. G., Toronto, Ontario.
- *1896. Woolen, Green V., Indianapolis, Ind.

