

Transactions

American Laryngological Association

SIXTY-NINTH VOLUME

“DOCENDO DISCIMUS”

TRANSACTIONS

OF THE

SIXTY-NINTH ANNUAL MEETING

OF THE

American **Laryngological Association**

1948

HELD AT THE HOMESTEAD

HOT SPRINGS, VA.

APRIL 14th and 15th, 1948

PUBLISHED BY THE ASSOCIATION

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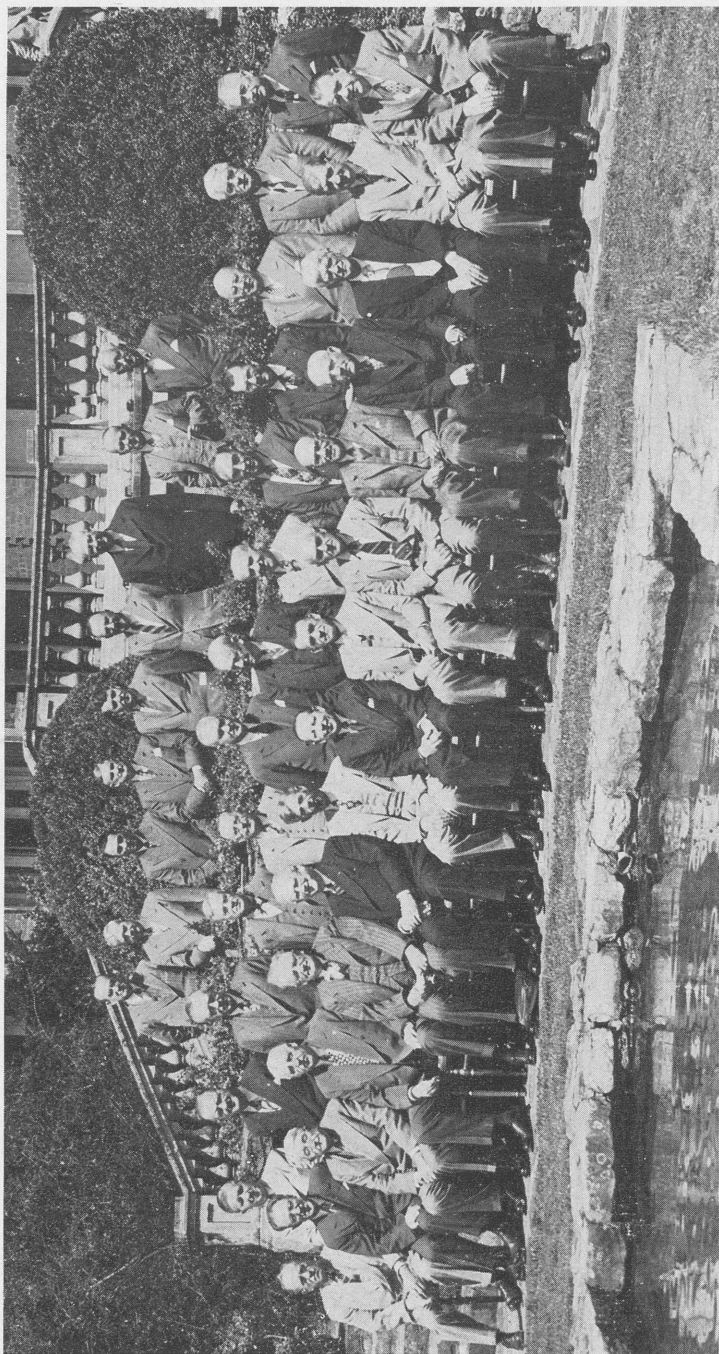
1948

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THOSE IN ATTENDANCE AT THE SIXTY-NINTH ANNUAL MEETING

Rear Row: Jackson, Whalen, Hilding, New, Eagle, Broyles, H. L. Williams, Jr., Woodward, Salinger
Middle Row: Boies, Schall, Maxwell, Seydell, Looper, Canfield, Lyman, Theobald, Lukens, Engler, McMahon, VanAlvea
Front Row: Beatty, Figi, Shurly, Harkness, Tucker, Shea, Hill, Proetz, Clerf, Lillie, Berry, Foster, Cody, Robb, Cumming

OFFICERS FOR 1947-48

PRESIDENT

ARTHUR W. PROETZ, M.D.

FIRST VICE-PRESIDENT

JOHN J. SHEA, M.D.

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TREASURER

FREDERICK T. HILL, M.D.

LIBRARIAN AND HISTORIAN

HARRY P. SCHENCK, M.D.

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JOHN D. KERNAN, M.D.

CHARLES J. IMPERATORI, M.D.

HAROLD I. LILLIE, M.D.

FRANK R. SPENCER, M.D.

TRANSACTIONS OF THE SIXTY-NINTH ANNUAL
MEETING

OF THE

American Laryngological Association

HELD AT

THE HOMESTEAD, HOT SPRINGS, VA.

APRIL 14TH AND 15TH, 1948

The Sixty-ninth Annual Meeting of the American Laryngological Association was called to order by the President, Dr. Arthur W. Proetz at The Homestead, Hot Springs, Va., at 10 A.M. on April 14th, 1948.

REGISTRATION

The following Fellows were in attendance:

ASHLEY, REA E.

CODY, CLAUDE C.

BALLENGER, HOWARD C.

CROWE, SAMUEL J.

BEATTY, HUGH G.

CUNNING, DANIEL S.

BERRY, GORDON

EAGLE, WATT W.

BOIES, LAWRENCE R.

ENGLER, C. W.

BROYLES, EDWIN N.

FIGI, FREDERICK A.

CAMPBELL, EDWARD H.

FOSTER, JOHN H.

CANFIELD, NORTON

FURSTENBERG, ALBERT C.

CLERF, LOUIS H.

GALLOWAY, THOMAS C.

GOODYEAR, HENRY M.	McCASKEY, CARL H.
GROVE, WM. E.	McMAHON, BERNARD J.
HARKNESS, GORDON F.	McNALLY, WM. J.
HICKEY, HAROLD L.	MOSHER, HARRIS P.
HILDING, ANDERSON	NEW, GORDON B.
HILL, FREDERICK T.	PROETZ, ARTHUR W.
HOOPLE, GORDON D.	ROBB, JAMES M.
HOUSER, KARL M.	SALINGER, SAMUEL
HUNT, WESLEY M.	SCHALL, LeROY A.
JACKSON, CHEVALIER L.	SEYDELL, ERNEST M.
JARVIS, DeFOREST C.	SHEA, JOHN J.
JONES, MARVIN F.	SHURLY, BURT R.
KELLY, JOSEPH D.	SIMPSON, W. LIKELY
KERNAN, JOHN D.	TAYLOR, H. MARSHALL
LIERLE, DEAN M.	THEOBALD, WALTER H.
LILLIE, HAROLD I.	TOBEY, HAROLD G.
LOOPER, EDWARD A.	TUCKER, GABRIEL
LUKENS, ROBERT M.	VAN ALYEA, OLIVER E.
LYMAN, HARRY W.	WEILLE, FRANCIS L.
MARTIN, ROBERT C.	WOODWARD, FLETCHER D.

PRESIDENT'S ADDRESS

ARTHUR W. PROETZ, M.D.

SAINT LOUIS, MISSOURI

Once in so often a thoughtful man takes himself into some quiet corner and asks himself two questions: "What am I doing here?" and "How?" Thus, by gentle disillusionment and readjustment, does he maintain a perspective toward his work and his fellow creatures—a perspective which will reconcile his energies with his talents, his ambitions with his limitations and his hopes with his disappointments.

We are here today in this quiet corner of Virginia not by accident, but by design. The time seems ripe to meet again more intimately than was possible during the past few years; to renew old friendships, to talk things over and to lay plans for the future.

In the social and political struggles now raging it is not our province to participate. But there *are* things which concern us mightily, which clamor for our attention whether we like it or not.

"The Association shall have for its purposes" according to the Constitution,

"To advance and promote . . .

"To encourage and raise . . .

"To publish . . .

"To honor . . ."

All of these relate to what might be called our intrinsic functions; but the world may look to us, as a "Senior" Society, for guidance in a larger sense, based upon that seniority and the weight of experience which should go with it.

The scientific record of the American Laryngological Association for seventy years, stands proudly on a shelf now five feet long. These are our laurels. We must not rest on them, how ever comfortably we sometimes lean.

The status of the otolaryngologist is changing. Progress in therapeutics has so simplified some of our toughest problems that unless we apply our talents to fresh ones we will soon be unable to justify our existence. There is of course no abstract reason why otolaryngology should survive beyond its era of usefulness. Possibly it is the ultimate measure of success for a specialty to solve all its special problems and wipe itself out . . .

Meanwhile what to do?

In the hour-glass are two heaps of sand: the sands of yesterday and the sands of tomorrow. Between them flows a tenuous stream of great activity where the grains are passing so rapidly that they are gone before they can be brought into focus, to be clearly seen. But every grain is like every other grain. Yesterday's pile grows only larger; we know exactly how it will look some moments, or hours, or years, from now.

Not so with the stuff of life. At that hiatus which we call "today" trained observers—scientists and philosophers—are watching the passing show. They scrutinize the grains as they fall. They anticipate what is coming; analyze what is past. Out of their knowledge they forge the tools with which we shove and beat and twist each grain until we crush it or shape it to our whim. It glows, or sparkles, or it fizzles. Sometimes it explodes and cracks the glass.

Life today and hope for tomorrow rest squarely upon these specialists; their training, judgment, discrimination, motives.

In every field, and therefore in ours, the Senior Society is presumably made up of such fellows. It should be the crucible for their combined experience. Out of it should come technical progress and something more: guidance and leadership in the philosophy of medicine and human relations.

How to assume such guidance—and, in contemporary language, to "implement" it—is a delicate matter.

Resolutions? We file them, feel righteous for a moment and go on to the next order of business.

Committees? They report self-consciously—and are continued. The result is artificial, and formal, and therefore unmoving.

In this sophisticated century all propaganda, good or bad, is suspect and we are no great adepts at it. The classroom is our stamping ground and there we are at home. Here we may lead a student into habits of thought which will protect him in his middle age from cynicism and compromise no matter what may be the medical high-jinks of the day.

To this end a society should have a plan, consciously developed; one that progresses from here to there, and one that works.

In the selection of candidates for Fellowship in this Association the Council has always looked beyond mere scholarship or popularity for the added quality of balance, so that we are well equipped.

Planned deliberation of matters not strictly technical, on our programs, could form a useful prototype for the conferences with our students which they so sorely need and so eagerly seek at this time. In the midst of intensive basic courses and clinical teaching they now find themselves almost completely without that counsel and worldly wisdom once supplied by a paternal preceptor.

During this epoch of stormy convalescence when, to doctor and layman alike, the expedient of the moment is more compelling than the demands of an uncertain future, somebody had better be taking the long view.

The fairy tales abound with stupid people who, being granted three magical wishes, invariably fumble the whole business and lose them all. Riches, fame and happiness are held out to them—always with a condition. You or I, or for that matter anybody beyond the third grade, could side-step such conditions with his eyes shut. But not they! The treasure within grasp, they trifle by the wayside, they unlock the wrong door, or speak that forbidden word and—SLAM!—they are back in Hooverville.

In the past decade two such magical windfalls have descended upon us: the sulfonamides and the antibiotics. True to type, however, there are conditions: we must not abuse them, we must not squander them,

for little by little, like the ass's skin, they shrink. We are warned to beware, or in the great hour of need they will have vanished.

We know the conditions by heart and are guarding our priceless gifts; we are using our heads and avoiding the pitfalls which we know have been laid for us, are we not? Not we. Every sniffle and sore throat receives its dash of penicillin, every itch and earache its "sulfa" pill.

Magical, fashionable drugs! We feed them, inject them and rub them in. We hide them in ice-cream and chewing gum. (See, they even work under water!)

This in the face of undisputed evidence that through our folly we are producing resistant strains of organisms. Scientists, doctors, manufacturers and even the public know it; only the first and the last seem to be showing any concern.

If we are under the delusion that, in the light of clinical experience, the warnings are overdrawn, we have only to read the literature. To pick an example, Barber records that in 1946, 14.1% of *Staphylococcus pyogenes* derived from actual lesions and examined by her were penicillin resistant. By 1947 this figure had advanced to 38%—almost three times. Her cases include many conditions important to us; extradural abscess, osteomyelitis, respiratory tract infections and otitis media. "It is obvious" she states "that the main cause for this increase . . . is the widespread use of penicillin . . . although the patient yielding such an organism may not himself have ever had any."

Here is work for us. Only the weight of medical opinion can curb such things.

And in wider fields . . .

Clerf, with his wry-lipped astuteness, has remarked that we may be coming to an age in which a medical consultation could degenerate into a series of tests to be recorded by punching a card. The card could speedily be "processed" by some Infrarational Business Machine which would flick out the diagnosis, the approved treatment and the prognosis within a fraction of one per cent.

To carry the idea farther: The tabloid magazines could teach the layman how to perform the tests—(they are already telling him how to compensate for psychosomatic variables)—then he could feed his own card into the machine together with some nickels, thus completely eliminating the doctor with his tiresome advice and his unspeakable waiting room.

A hypochondriac could set himself back a considerable handful of nickels, for the machine would be expensive, what with spiraling labor costs, the federal tax, the lights and music, and all. Then he could complain once more of the "high cost of medical care."

A fantastic flight of imagination? Perhaps. I have indulged in it only to point out that there is no part which does not have its germinal *anlage* in the present-day philosophy of some of our more respected medical centers. The high cost of medical care can be, at times, the high cost of redundant medical machinery and hospitalization.

It is not always the physician who is to blame. Hospital management comes in for a large share. The patient also clamors, insurance blanks in hand, and the weary doctor capitulates just as he reaches for the penicillin.

This is no blanket indictment of group practice and laboratory medicine. Nothing could be farther from my thought. I have observed, however, that even the best human thinking when it yields to popular fancy can be diverted with such momentum that it may travel an amazing distance on the bias before it wakes up to find itself ridiculous.

A few weeks ago when the British Medical Association held a plebiscite and voted 9 to 1 against participating in the new Socialized Medicine Plan, a spokesman for the Labor Government characterized the action as a strike "to organize and exploit an outburst of mass hysteria among doctors," to the detriment of the patient!

I do not imply that the headaches of medical practice are all nasal headaches to be diagnosed and cured by the American Laryngological

Association. Nevertheless all these problems and many more like them are partly ours and we know that great movements come through small groups who start and set the pace of popular trends.

This, I opine, is some of what we should be doing.

Others will judge how we succeed.

TONSILLECTOMY IN THE UNITED STATES*

LAWRENCE R. BOIES, M.D.

MINNEAPOLIS, MINNESOTA

This should be a worn-out subject. This is the eighty-third paper on some aspect of the tonsil problem to be presented before this Association in its sixty-nine meetings. However, there is evidence that a problem concerning the tonsil still exists. In the five years 1942 to 1946 inclusive, there has been an average of over one hundred papers per year concerning tonsils listed in the *Quarterly Cumulative Index*, and this in spite of the war years when medical literature was less voluminous than in ordinary times. In addition, lay publications have aired and emphasized the negative side of the problem. The *Woman's Home Companion* in July, 1947, publicized the tonsil business under the title "Unnecessary Operations." A few months later the *Reader's Digest* summarized this article for wider lay consumption. The following is quoted from that summary:

"More tonsils are yanked out of the American anatomy than any other organ. The well-known pediatrician, Dr. Harry Bakwin, is the latest to lash out at the unwise but widespread practice of removing tonsils indiscriminately. He points out that while tonsillectomy is considered a relatively safe minor operation, it sometimes results in death and oftener in serious complications such as pneumonia or lung abscess.

"The absurdity of indiscriminate tonsil operations was demonstrated some years ago in a study of a thousand eleven-year-old children conducted by the American Child Health Association. Three-fifths already had their tonsils removed. The other four hundred were examined by a group of doctors who decided that half of them needed tonsil operations. Those who passed were examined by a second group of doctors. Again, half were chosen for tonsil operations. After the process was repeated for the third time only sixty-five children were left with their tonsils intact. Some of these no doubt would have been marked for an operation if a fourth doctor had been called in.

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"The directors of this classic survey concluded that tonsil operations depended not so much on the child's health as on the vagaries of a peculiar doctor."

My purpose in this presentation is to raise a dissenting voice to the contention that the tonsil and its surgical treatment need be a problem. When tonsil surgery is performed by one adequately trained and with good technic, it is a highly satisfactory procedure in terms of the end results. At this point I would state, however, that in a majority of cases, I have not found it to be a simple minor surgical procedure, but that it is not followed by any serious morbidity and should never result in a mortality.

In the writings, both medical and lay, on this subject, there seem to be three aspects of tonsillectomy which constitute the problem part of the matter. These are: (1) the indications for tonsillectomy, (2) the adequacy of the operation and (3) the morbidity and even the mortality connected with tonsil surgery.

THE INDICATIONS FOR TONSILLECTOMY

I have personally performed tonsillectomy on slightly more than 3,000 persons. Practically all of the children on whom I operate are referred by pediatricians who are acquainted with the whole picture of the particular child's health. Most of my adult cases are under the care of an internist or are cases in which there is no doubt about their needs because of very obvious local disease or they have repeated attacks of tonsillitis. Out of this experience I have simplified the indications into three categories as follows:

1. Repeated attacks of tonsillitis.
2. Hypertrophy to the extent of obstruction.
3. Evidence of persistent chronic infection.

There should be little argument concerning the first and second indications. Simple tonsillar hypertrophy is common to early childhood and in itself is not a need for tonsillectomy. When the hypertrophy is excessive, the child is invariably a mouth breather even though the

adenoid may not be obstructive. Also, he eats poorly. In a majority of these instances, a considerable weight gain is experienced in these cases after the tonsils and adenoids are removed.

The third category is one in which there is often the element of chance that tonsillectomy will not relieve a condition in which a focus of infection is thought to be a factor in the morbidity. However, the chance seems worth taking.

There are many aspects of this third category concerning which the pros and cons could be discussed. Not infrequently cases are encountered in which the family or the doctor are interested in tonsillectomy to prevent head colds. When the tonsils and adenoids are obstructive or obviously diseased, the benefits from tonsillectomy and adenoidectomy in the prevention of colds not of virus origin are in most cases very obvious. When the tonsils and adenoids are within normal limits and removal is advised as a prophylactic measure, the end results are often disappointing. Some physicians have expressed a belief that removal of tonsils and adenoids have rendered a child more susceptible to head colds. It is possible that a protective function of the tonsils and adenoid does play a role in this respect. However, the fact is often overlooked that the early school age is usually the child's most susceptible period in the matter of respiratory infections, a period in which he may be developing some immunity and that the removal of his tonsils and adenoids in a preschool period may have little to do with the relatively increased frequency of upper respiratory tract infections after he is exposed to the infections so prevalent in the early years of school life.

THE ADEQUACY OF TONSILLECTOMY

Much has been written about incomplete tonsillectomy and the importance of tonsil remnants which are often referred to as "stumps" or "tags."

Many of you will remember an editorial in the *Journal of the American Medical Association* in 1928¹ that had apparently been inspired by two contemporary reports, one² from data which had indicated that one-third of all operations performed since 1924 among American urban

population was done for the removal of tonsils and adenoids, and the other the studies of Rhoads and Dick³ describing the high incidence of postoperative tonsillar remains and the patent bacterial content of these remnants. Rhoads and Dick had found fairly large pieces of tonsillar tissue remaining in the throats of 290 of 403 nurses (73 per cent) entering training at the Presbyterian and Cook County Hospitals in Chicago. When some of these pieces of tonsil tissue were removed and their bacterial content studied quantitatively, it was found that a larger count of pathogenic bacteria could be found in a gram of tonsillar remnants than in an equal amount of unoperated tonsillar tissue.

Now, nearly nineteen years later in the same *Journal of the American Medical Association* for June 21, 1947, there appears under "Current Comment"⁴ the following statement in part:

"Tonsillectomy is the most frequent surgical procedure in the United States. . . . The results of the analysis of the rate of recurrences of tonsillar growth after tonsillectomy indicate that tonsillectomy should command all the respect of a major operation. The incomplete removal of tonsillar tissue and the subsequent formation of infected tonsil stumps are rather common. The Spuths recently reported the presence of tonsil stumps in 80 per cent of 285 tonsillectomized patients. Instead of being relieved through tonsillectomy of their foci of infection, many patients continue to absorb bacteria and toxins from the tonsillar tissue left in the throat and are often systemically much worse off than prior to their operation. While there is drainage of infections and toxic material from the external surface of the tonsils before tonsillectomy so that only a part of such harmful matter is absorbed into the organism, this route of escape becomes obliterated after tonsillectomy, for the infectious and toxic material contained in the usual tonsil stumps are covered by scar tissue. It is resorbed in its entirety through the lymphatics and veins and may cause, in addition to fatigue, heart palpitations, vague muscular pain, secondary infectious and allergic complications in remote organs (polyarthritis, nephritis, endocarditis, asthma, chronic phlebitis). Sometimes the diagnosis of tonsil stumps cannot be made by an inspection of the tonsillar region because the retained tonsil tissue may be buried beneath a thick layer of scar tissue. However, as inflamed tissues, the presence of which is usual with tonsil stumps, are sensitive to pressure, tonsil stumps can be detected with reliability by exerting

pressure either directly on the tonsillar scar tissue or externally from the angle of the lower jaw in the direction of the tonsillar fossa. Tonsils when functioning normally are protective organs and should not be removed. Definitely infected tonsils and especially tonsil stumps should be radically removed under general anesthesia and with adequate surgical technic and skill."

This high incidence of pieces of tonsillar tissue remaining in tonsillar fossae (in three-fourths of the cases) seems almost unbelievable. It is probable that in some instances the observer confused simple lymphoid nodules with an actual tonsil remnant.

It has been my observation that in a majority of instances of complete removal of the faucial tonsil with capsule intact and in which the plicae are thoroughly removed, small lymphoid nodules grow into the fossae from the membrane lining and fossae. This is particularly true in children. The follicles are no different than the lymphoid nodules seen on the posterior pharyngeal wall. The younger the patient the greater the tendency of these nodules to appear. Also, they appear in greater abundance in the patient who suffers from an allergic rhinitis.

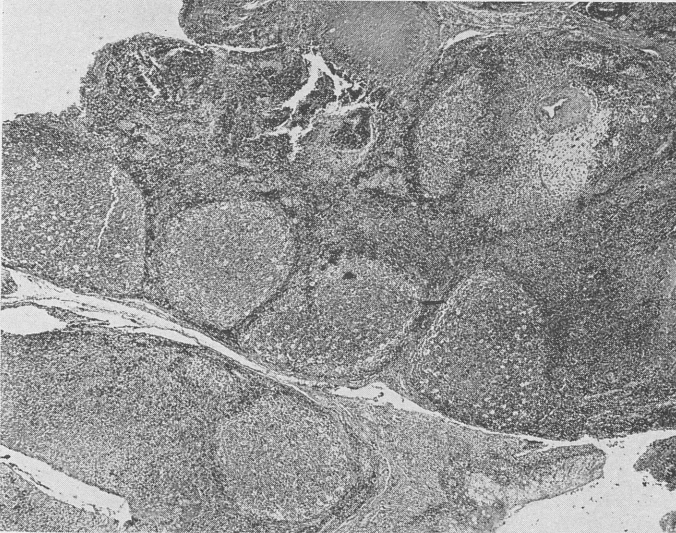


Fig. 1.—Low power magnification of a portion of a *plica triangularis* showing numerous lymph follicles. The plica was thoroughly removed after the tonsil had been enucleated with a guillotine.

Lymphoid structures with germinal centers can be found throughout the respiratory tract and in the digestive tract in "Peyer's patches" and in the appendix. Presumably, their role is a protective one when not diseased. There is some recent evidence that lymphoid tissue is the site of antibody formation.⁵

A tonsil cleanly enucleated cannot grow back. As part of this clean enucleation it is advisable to thoroughly remove the plicae and the prominent immediately adjacent projection of the lingual tonsil. If the well-developed plica triangularis is left, the lymphoid follicles often

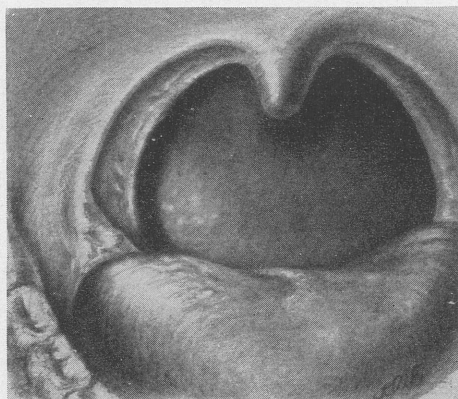


Fig. 2.—The usual picture of a remaining *plica triangularis* in the right fossa. This is a drawing of an adult whose tonsils were removed in early youth. The presence of this plica had caused no morbidity.

present in it (Fig. 1) tend to hypertrophy and sometimes fill the lower portion of the fossa. This is more likely to occur in small children. In my opinion the hypertrophied lymphoid tissue of a remaining plica triangularis (Fig. 2) does not have the pathologic potentialities found in the remnants of the main tonsil mass with its crypts which branch.

Leshin and Pearlman⁶ in a study of the incidence of these follicles found them to be present in 66.6 per cent of sections from the plicae

triangularis, in 30 per cent of the plicae supratonsillaris and in 47.7 per cent of the retrotonsillar plicae.

Many otolaryngologists use some form of guillotine for tonsil surgery in children. I prefer it for the large, soft tonsils of children but agree with Kelly⁷ in preference of a dissection technic for the recessed or submerged tonsils which may be more or less adherent. Whenever a guillotine is used, it is important to inspect the capsule and plical attachments as soon as the tonsil has been enucleated. One occasionally

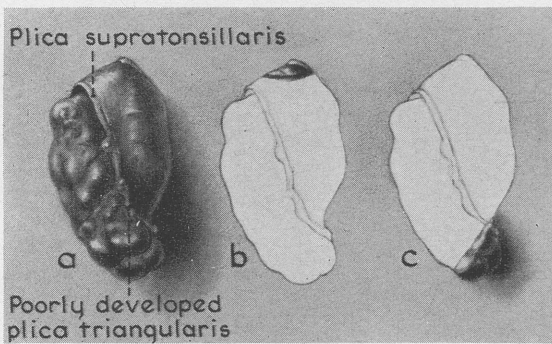


Fig. 3.—(a) A large soft tonsil cleanly and completely enucleated. The plica supratonsillaris is intact. In this type of tonsil development, the plica triangularis is usually poorly developed.

Occasionally guillotine enucleation misses a piece of tonsil at the upper pole (b) or cuts across a portion of the lower pole (c). The tonsil should be carefully inspected as soon as it has been enucleated in a search for a defect of this type.

finds one of the following defects in what he assumes is a complete enucleation:

A portion of the plica supratonsillaris is missing.

There is a defect at the upper pole with a small piece of tonsil tissue missing. It can be found by a careful inspection of the fossa at the site of the defect (Fig. 3b).

The lower pole has been cut across (Fig. 3c).

A projecting enlargement bulging the capsule such as is often seen

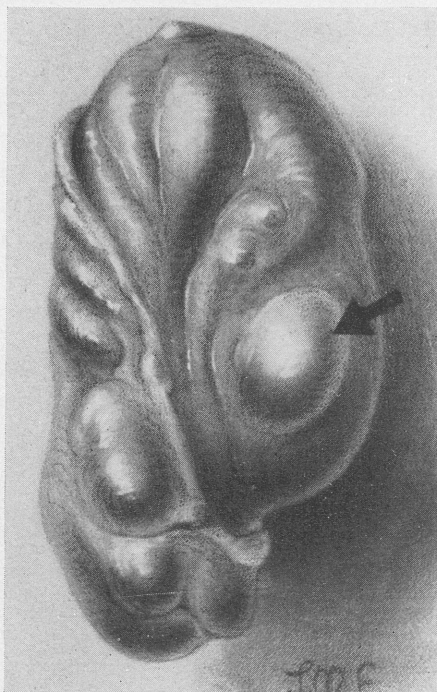


Fig. 4.—A large soft tonsil cleanly enucleated with the guillotine. The enlargement (indicated by the arrow) which bulges the capsule may be cut across by the guillotine.

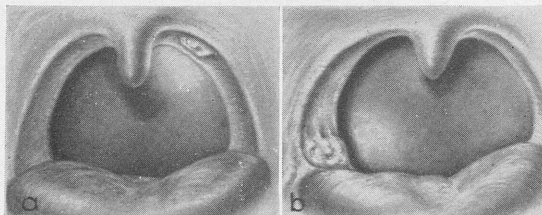


Fig. 5.—Completely healed fossae, with hypertrophy of the remnants, in instances of tonsil pieces left at (a) the left upper pole and (b) at the base in guillotine tonsillectomy.

in the large, soft tonsils of children is sometimes cut across by the guillotine and a piece is left in the fossa (Fig. 4).

Figure 5 depicts the completely healed fossae with hypertrophy of the remnants in instances of tonsil pieces left at (a) left upper pole and (b) at the base in guillotine tonsillectomy.

Because the two surveys referred to in the *Journal of the American Medical Association* indicated that tonsil remnants existed in approximately three-fourths of the individuals examined, I made a personal study of a group of patients of all ages passing consecutively under my observation. A critical inspection was made of the fossae of tonsillectomized patients to determine in what percentage tonsil remnants existed. Also, I determined in which cases the tonsillectomy had been done by specialists and in which the general practitioner had performed the operation. When the latter fact could not be reliably determined the case was not included in this series. Four hundred and thirty tonsillectomized patients were studied. The following data were obtained:

<i>Number of tonsillectomies</i>		<i>Tonsil</i>		<i>Lymphoid</i>		
		<i>Per cent remnants</i>		<i>Per cent ingrowths</i>	<i>Per cent</i>	
Performed by specialists	164	38.1	14	8.5	41	25
Performed by general practitioners	266	61.9	121	45.5	76	28.5
Total	430	100.0	135	31.4	117	27.2

The figures which I have determined from personal observation do not compare with those of Rhoads and Dick or the Spuths. In my series, there were actual tonsil remnants in 135 of the 430 cases, or in 31.4 as against the 73 to 80 per cent of their series. I have suggested that in some instances the observer may have confused simple lymphoid follicles with an actual tonsil remnant. But even if I add the instances of lymphoid ingrowths to the cases of actual tonsil remnants and call all of these tonsil "stumps" or "tags," I get only 58.6 per cent.

The last paper on an aspect of the tonsil problem presented before this association was one by Campbell⁸ in 1939. His paper on the "Local Results of Tonsillectomy" reported observations on 621 cases of ton-

sillectomized young women between eighteen and twenty-one years of age. When what he considered to be tonsil remnants were found they were classified as infected or noninfected. If the remnants appeared to be simply noninflamed lymphoid tissue, without observable crypts containing exudate or without signs of being partially obscured by scar tissue, they were classed as noninfected. If the remnants showed signs of chronic inflammation or if there were crypts containing exudative material or if they were imbedded and partially covered by fibrous scar tissue, they were classified as infected. Campbell reported that 77.3 per cent of the 621 students showed tonsillar remnants in the fossae. Of these, 66.6 per cent were placed in the class of noninfected remnants and 10.6 per cent in the class of infected remnants. I suspect that his "noninfected" remnants correspond to what I consider to be lymphoid ingrowths, which in my opinion are not tonsil remnants. In Campbell's series, data on only 153 of his cases was obtained as to whether the tonsil surgery had been performed by one specially trained or by the general practitioner. He found 74.7 per cent of the cases operated on by specialists showed tonsil remnants but did not give any figures on this 74.7 per cent as to how many of the remnants were of the noninfected or simple lymphoid type. If I apply his percentage of 66.6 per cent of noninfected remnants of his total of 621 cases to the 153 cases done by specialists, which he studied, the percentage of infected remnants, "tags" or "stumps," approximates 8 per cent and corresponds to the remnants left by specialists in my series.

The report of the Spaths⁹ which was the basis for the comment in the *Journal of the American Medical Association* in July, 1947, stated that three-fourths of a series of 285 new patients who had had tonsillectomy exhibited infected tonsil stumps and lingual tonsils. They claim that:

"The majority of people with infected tonsil stumps have very few complaints referable to their throats. The infection seems to affect the entire body. They usually complain of feeling tired, loss of weight, joint pains, shooting pains down the arms and legs, and frequently, bouts of fever. Others may have skin affections and eczemas. In some patients chronic sinusitis or chronic middle ear disease is noted."

They apparently subscribe to the claims of Otto Meyer¹⁰ who finds tenderness in the submaxillary region, over the tonsil stump and along

the course of the jugular vein significant of tonsil stump infection. These writers speak of the removal of the tonsil stump under general anesthesia and including *the whole lingual tonsil*. I wonder if those who recommend removing the lingual tonsil are convinced that the actual whole of the lingual tonsil is removed. This would require removal of a segment of each side of the tongue extending to the median glosso-epiglottic fold in the midline. In my experience, this is a procedure which from practical considerations should be avoided.

I have asked Dr. C. B. Sputh, Jr., for a statement as to how much of the lingual tonsil they actually remove. He replied, "By careful dissection, we carry a line of cleavage underneath the lingual tonsil from the faucial tonsil. This is then snared off, taking care that we do not remove any of the musculature or mucous membrane of the tongue."

I believe that this describes the removal of a portion of the lingual tonsil and is comparable to the amount I remove in instances where there is a hypertrophied prolongation of the lingual tonsil.

In my experience the ordinary lingual tonsil is not a common cause of morbidity and I only occasionally encounter an infected tonsil stump accounting for symptoms and signs such as the Spuths or Meyer describe.

MORBIDITY AND MORTALITY FROM TONSILLECTOMY

The bugbear of tonsillectomy is postoperative hemorrhage. It is my observation that this is practically needless if an extra few minutes is given to careful hemostasis at the time of tonsil removal. This hemostasis should be performed according to the best standards of surgical technic, using tie and suture with a minimum of tissue inclusion or of tissue trauma. This will avoid all but an extremely small incidence of postoperative hemorrhage. Bleeding several days postoperatively, particularly at the end of the first postoperative week, occurs in a small percentage of cases as the membrane lining the fossa separates. As a rule, this offers no great difficulties.

The instances of lower respiratory tract pathology following tonsillectomy are probably dependent largely on the aspiration of blood, tonsillar debris, and possibly fragments of adenoid tissue getting into the trachea during the operation. I make it a routine practice to do a direct laryngoscopy at the close of every operation under general anes-

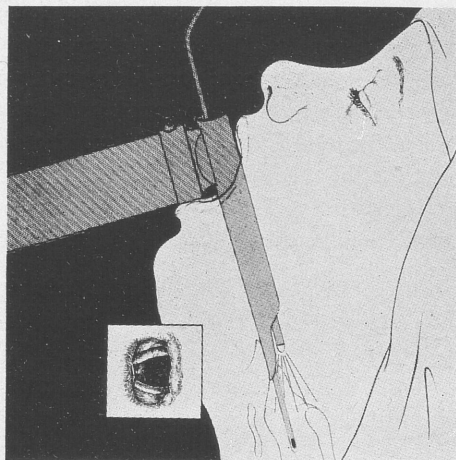


Fig. 6.—Aspiration of the trachea with a laryngeal suction tube through the Flagg laryngoscope is routinely performed in all cases of tonsillectomy done under general anesthesia.

thetia and aspirate the trachea with a laryngeal suction tip (Fig. 6). One can introduce this easily down to the carina, and if it is gently done no trauma results. Not infrequently I find it helpful to aspirate mucus from the trachea as soon as the child is anesthetized and before starting the surgery.

When large children or adults are done under general anesthesia, an intratracheal tube provides smoother anesthesia, and it is part of the technic to aspirate the trachea with a catheter inserted through the tube.

I have heard anesthesiologists remark that statistics indicate that the tonsil anesthetic is the most hazardous of general anesthetics. That hazard and fatal hemorrhage apparently are the important contributors to the mortality associated with the operation. Recently, within a twelve-month period I have been asked to express an opinion as to the possible fault in five cases of death associated with the operation for tonsillectomy and adenoidectomy. Three of these seemed to be anesthetic deaths and two could be blamed to hemorrhage.

A carefully administered general anesthetic by one adequately trained, when the surgical technic is performed by an experienced oper-

ator and hemorrhage has been controlled, is in my experience a safe procedure.

In a discussion of morbidity associated with tonsil and adenoid surgery in this day, one must consider the possible relationship between the surgical wound created by the operation and a greater susceptibility to poliomyelitis, particularly the bulbar form.

The data accumulated to date suggested that there is no causal relationship. However, to be convincing and according to the proper factors in statistical analysis it seems necessary to introduce the factors of a certain age group and the number of tonsil and adenoid operations performed in the area being studied. It is my personal belief that when the facts are all in we will not regard the tonsil and adenoid operation as predisposing to poliomyelitis. However, regardless of whatever is eventually proved regarding this relationship, there should be no more basis for performing tonsil and adenoid surgery during an epidemic of poliomyelitis than there would be during an epidemic of respiratory infection or any other type of infectious disease.

SUMMARY

In the minds of the laity and for some of the medical profession, tonsil surgery is a cause for concern on three counts: (1) the indications, (2) the probable inadequacy of the operation and (3) the associated morbidity and even mortality.

In the hands of the trained laryngologist, none of these three aspects of the tonsil operation need be a cause for concern.

The faucial tonsil which is cleanly enucleated with complete removal of the plicae does not grow back. Lymphoid ingrowths from the lining of the fossa are common. These are often erroneously called tonsil remnants. Ordinarily, these lymphoid ingrowths are not a source of any significant pathology.

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DISCUSSION

BURT R. SHURLY: M.D. Mr. President, we are indeed in a new era of change and chaos, and the medical profession has certainly been affected by it and in a very considerable degree.

I believe this paper is timely and important, and that we should come back a few years and reconsider the scientific truths in connection with this operation. Having done through fifty years more than 20,000 so-called tonsil operations, beginning with the old Matthews' tonsillitome and removing as much as possible in the office, we have progressed through the years and now consider tonsillectomy as the proper procedure. During that period we operated many hundreds of children and adults with chloroform anesthesia and nobody died. But under the modern changes which have taken place, we have gone into the use of local anesthesia and ether. In the 20,000 we had no poliomyelitis develop, and I feel that the essayist has stated properly that the danger is not as great as it has been outlined. The public are certainly full of fear for operative procedures and we are feeling the effects of that.

The Sluder operation came into vogue and I have felt that it has been highly successful for children and have used it in hundreds of cases.

What is the psychology of the public? It is a fear of operative procedures which has developed in a large way and the medical profession must certainly combat that idea. I feel that we should get together in trying to prevent that alarm so that the benefits from tonsillectomy may continue in the future. The public are against it and that has developed from the psychology outlined by the public health officer who has a chance to advertise his ideas while we keep ours in the dark.

JOHN D. KERNAN, M.D.: Mr. Chairman, Dr. Boies need not apologize for his selection of a subject. I have looked over the program for the next two days and I cannot see a more important paper on the whole program.

When you consider that in spite of the opposition of the public, about two million tonsillectomies are done in the United States every year, you will realize that it is still an important operation.

I am going to raise the question as to why tonsillectomies are done at all? I think that the function of the tonsil has not been sufficiently investigated. Although I have not read all these hundreds of papers, I have never seen it stated in any one paper what the function of the tonsil is. It stands to reason that it must have some very important function in young children.

We have all had the experience of taking out tonsils and then in a year seeing the whole throat plastered with lymphoid tissue. The only reason for the reoccurrence of that lymphoid tissue must be a physiological demand in the child's biochemistry; certainly the function of the tonsil should be a subject for the most intensive research.

We do not know what we do to a child when we take out the tonsils. We do not know what the changes in the blood are. We do not know what the changes in the whole metabolism are. Of course we have to consider it. I have no doubt if it is thoroughly investigated there will be found to be a good reason for tonsillectomies. If there is not a good reason for tonsillectomies, the otolaryngologists are going to lose that important operation and most of us will be reduced to the practice of medicine.

HENRY M. GOODYEAR, M. D.: Mr President, I would like to add to the indications for tonsillectomy, given in this very interesting paper of Dr. Boies, mouth breathing, frequent attacks of otitis media and enlarged glands of the neck, which were not mentioned.

I feel it is particularly important that we consider a tonsil and adenoid operation in cases which give a history of repeated attacks of otitis media. We must also give greater consideration to the removal of tonsils in adults where there is a history of deafness.

When we speak of tonsillectomies in children we are usually including an adenoidectomy. I feel that a tonsil and adenoid operation, or either one, is as difficult as an appendectomy, and we try to teach our students that that is the case. We do not permit our temporary interns on the service at the Children's Hospital or at the Cincinnati General Hospital to do tonsillectomies. It is our aim to discourage tonsillectomies and adenoidectomies by general practitioners. The operation is difficult even for a man who does tonsil operations every day, and unless you are intent upon your operation, you will find that you have inadvertently disturbed a pillar or left a piece of tonsil. I know of no operation which requires greater care.

Many tonsil operations are done today with the head in the extended position. I mention this only to condemn it, unless the man who uses this position is willing to flex the head and examine the adenoid area before he feels that his operation is completed. There is no excuse for a laryngologist today doing a blind operation on the adenoids. It is an important and a difficult operation and it requires all the respect and care that you would use in an appendectomy, tonsillectomy or an operation elsewhere.

We have done a series of cases with the head in the extended position and in the modified Trendelenburg's position and as far as recovery is concerned the results are the same.

While we are using the modified Trendelenburg's position, we retract the soft palate and by the way, there is only one good retractor that I know. This is now being made by Storz of St. Louis. It is surprising how many of these cases show a large piece of adenoid tissue remaining, even after we feel we have done a careful operation.

I know of one clinic where this operation is done with the head in the extended position who report that 75 per cent of the adenoid operations are incomplete. I

am sure they are incomplete if they have been done in the extended position and the nasopharynx has not been examined by direct vision.

During this examination bleeding points are seized with a hemostat; it is not necessary that they be ligated but the field should be absolutely dry and clean before the patient leaves the table. The adenoidectomy is done before starting the tonsil operation.

For the tonsil operation, we teach that of dissection. We have given up the ring operations. In these operations an Allis forceps is applied to the posterior pillar which is drawn toward the mid-line, and it is surprising how much mucous membrane you expose by so doing. While this tension is being applied, a small rake retractor such as is used in eye operations retracts the tonsil laterally and the membrane is split close to the tonsil with a new No. 15 Bard Parker blade on a long handle. The tonsil is then elevated by the same eye retractor exposing the plica triangularis for resection with a flag knife. The tonsil is then dissected under tension and very few ligations are ever necessary.

I know of no way of keeping islands of granulation tissue from recurring in the tonsil fossa and it is very discouraging sometimes to see patients upon whom you have operated, and find large islands of granulations present. These children are helped by increasing their iodine intake.

JAMES M. ROBB, M.D.: Mr. President, it has been my good fortune to have done quite a few tonsillectomies and adenoidectomies. I have a feeling that the most poorly done operative procedure of all surgery is an adenoidectomy. I would like to mention a word about the value of tonsillar tissue. Some years ago I obtained thirteen rats from the Department of Health of Detroit and dissected them. I could find no lymphoid tissue in the nose and throat of this rodent. It is a creature which lives under the most unhygienic circumstances—it lives under old houses and in filth of the alleys of our cities and what it eats must always be somewhat dirty—so that I would feel that if nature were going to protect the body with lymphoid tissue in the nose and throat, it should protect the rat.

JOHN D. KERNAN, M.D.: I would like to advance the pig as a rival of the rat in living under unhygienic conditions. The pigs that I have seen around our country tramp around in the most vile pig-pens and they all have tonsils.

L. R. BOIES, M.D.: (closing) I wish to thank the discussers.

I believe that if Dr. Goodyear will reflect a little on the three general categories that I have mentioned, into which fit all the indications for tonsillectomy, he will find that the child with neck glands and the one who is subject to repeated attacks of otitis media and the one who mouth breathes, if these disorders are due to tonsil and adenoid diseases, will either have had repeated attacks of tonsillitis, or have hypertrophy to the extent of obstruction, or have evidence of persistent chronic infection in the tonsils or adenoids.

I am sure, Dr. Robb, that the rat has a tonsil. I believe all mammals have tonsil structures. In fact, most vertebrates that are not mammalian have collections of lymphoid tissue corresponding in arrangement to the tonsil and adenoid structures in higher forms of life. However, I will investigate the rat.

LERMOYEZ'S SYNDROME—AN ALLEGIC DISEASE

WATT W. EAGLE, M.D.*

DURHAM, N. C.

When an individual has an attack of nausea and vomiting, associated with vertigo, usually with sudden onset, followed by tinnitus and deafness, which usually are progressive, Meniere's disease is the probable diagnosis.

When an individual has an onset of tinnitus and deafness, followed by vertigo, associated with nausea and vomiting, *then* has immediate improvement of the tinnitus and deafness, we probably shall make a diagnosis of Lermoyez's syndrome. These symptoms may be unilateral or bilateral in both syndromes, but are more often unilateral.

A brief review of the chronological history of Meniere's syndrome is in order to appropriately present Lermoyez's syndrome for analysis. In both diseases we encounter the same symptoms but their appearance is reverse to each other, and the prognosis is entirely reversed. Meniere's disease may cause more or less permanent disability, while Lermoyez's syndrome produces only a temporary disability.

Meniere in 1861 reported the now famous case of the young lady with an acute infection that he had treated over a short period of time and who had complained of nausea, vomiting and vertigo, with severe deafness and tinnitus. He, it is now thought, erroneously reported the pathological picture in that case in that he described a hemorrhage into the labyrinth. For many years this one report was accepted as providing the true causation of Meniere's syndrome. However, investigators, not agreeing with the hemorrhage into the labyrinth etiology, began to search for other causes. Eventually numerous theories appeared and only recently has there been a unified opinion. Anatomical and pathological material has never been plentiful, and the interpretations on the studies made had been quite varied. The fact that the symptoms sometimes appear in young people caused some observers to believe that blood dyscrasias, such as anaemias and leukemias, were responsible. This theory presumed that only the middle-aged to elderly individuals would provide the arteriosclerotic and atheromatous conditions which could conceivably cause hemorrhages into the labyrinths.

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During a later period theories of the cause of Meniere's disease presented the specific or general infections and, then, in more recent decades the vascular anomaly theory appeared. The very recent literature abounds with articles describing hydrops of the endolymphatic system as being the cause of Meniere's disease, and there has been much research work by numerous workers, including Dederling, Lindsey, Fowler, Hallpike and Cairns—and many others, to substantiate this opinion, which is now the accepted one.

During the interim from the older theories to the two latter theories just mentioned—those of vascular anomalies and hydrops of the endolymph sac—there appeared the allergic causation of Meniere's disease and the water and sodium electrolytic metabolism studies by Furstenburg. In 1923 Duke¹ reported in full two cases of allergic etiology of Meniere's disease and mentioned three other cases which presented typical symptoms of the disease. These symptoms in his cases could be produced in typical attacks by the ingestion of foods to which the individual was sensitive and, then, the attacks could be controlled by adrenalin. Allergy vaccine therapy was found to produce attacks. Unfortunately Duke, as an allergist and not an otologist, had very little to say about the hearing impairment in these cases, merely stating that in one case the patient had one-fifth depression of hearing. There was no statement as to whether the impairment of hearing progressed or regressed. Duke's report of allergic basis of Meniere's disease was followed in a few years by numerous other reports—in part, those of Rowe and Richet in 1930, by Proetz² and by Vaughan in 1931, by Levy and Vogel in 1932, and in 1933 by Yandell³ and also Malone.

Proetz in 1931 reported an individual who was extremely sensitive to dairy products, especially butter, who had a very positive reaction to these allergens when eaten but was not reactive to skin tests. This patient demonstrated a visible patch of edema—allergic in type—in the nasopharynx. This case had symptoms of Meniere's disease over a five-year duration previous to the examination, and he was followed for a period of five additional years. Deafness persisted in this case, indicating a true Meniere's disease. The symptoms were intermittent, a phase of the disease which was also reported by Crowe in later years. This intermittency seems to depend on exposure to, or the use of, allergens, both in Meniere's disease and in Lermoyez's syndrome.

Yandell and Malone reported orris root as the primary allergen

in cases of Meniere's disease. Various other authors found various allergens to be responsible for symptoms. Excellent reviews of the role of allergy affecting the inner ear have been presented by Dean⁴ and by Hansell.⁵ It is quite noticeable and, probably of considerable significance, that bacterial allergy is scarcely mentioned by any of the writers. Foods seem to have played the predominant role in the allergic production of the symptoms.

In 1941 Atkinson⁶ stated that Meniere's disease may be due to more than one cause; in other words, the origin is not always from the same basic condition. From his observations he was able to state that allergy and angiospasm were the two factors which could cause Meniere's disease. He thought that histamine as an allergen was an instigator of many allergic cases of Meniere's disease. He also stated that the histamine test would always be positive in such cases. On the other hand, he found that histamine tests were uniformly negative in the vasospasm cases. Angiospasm cases could be relieved of their symptoms by the use of amylnitrite pearls and acetylcholine, while adrenalin was utilized in producing symptoms, which then could be relieved by nicotinic acid. Atkinson found tinnitus and deafness more pronounced in the vasospasm cases than in the allergic cases, and these symptoms always became worse after the attack of vertigo. In the allergic group, however, he found the opposite—that the hearing impairment and tinnitus improved following the attack of vertigo. His findings, therefore, in the allergic group, coincide with Lermoyez's syndrome in that the appearance of the vertigo was the signal for the improvement of the deafness and tinnitus. Atkinson pointed out that vasospasm occurs sometimes in young people, producing Meniere's disease, and also that allergic origin of the disease may also occur in young people.

The history of Lermoyez's syndrome is rather brief. In 1927 the late Marcel Lermoyez⁷ proposed again the idea of vasospasm, which he had fostered for many years, as the cause of deafness and tinnitus which were followed by vertigo, nausea and vomiting, with sometimes sweating, with all of these symptoms subsiding after the vertiginous attack. He also noted that these attacks may be recurrent. Lermoyez was influenced by the observations of Kobrak as he followed a case of Raynaud's disease, which presented the vasospasm and vasodilation in sequence, and he attempted to correlate what happened in Raynaud's disease to what may happen in the internal auditory artery. Kobrak

had noted these spasm sequences but thought them due to angioneurotic seizures.

Kerekes⁸ reviewed all the literature from Meniere's first report up to his own report in 1934. He reviewed the hemorrhage into the labyrinth idea, which Meniere had proposed and was some years later substantiated by Swabach, but only in cases occurring in blood dyscrasias—the anaemias and leukemias. Alexander dissented, however, and reported that he found hemorrhage into the labyrinth in no single instance in any of his cases. Manasse had found hemorrhage into the labyrinth in one case who had vertigo associated with apoplexy. Quinke theorized on local edema producing the pathology in the labyrinth, which theory most closely approximates the hydrops of the labyrinth theory in the very recent years.

Lermoyez was of the opinion that vaso-constriction involving the internal auditory artery caused the hypoacuity of hearing and tinnitus and that when the vertigo appeared it was a sign that circulation had been restored in the artery. With the restoration of the circulation to normal in the artery, all symptoms disappeared and the patient enjoyed normal health again. He pointed out the possibility of spasm in the cochlear artery or vestibular artery individually. Lermoyez is well remembered for his remark relative to the fact that an attack of vertigo is necessary in the disease he described, due to vasospasm, for the patient to regain normal hearing. He compares the episode to a storm clearing the sky. Throughout the dissertations of Lermoyez and Kerekes there is no mention of allergy as a possible or probable cause of the vasospasm. They were not impressed by any allergic manifestations prior to the symptoms. Kerekes did state, however, that he thought the disease may be due to fundamentally different causes, as Atkinson also stated later, which naturally would permit allergy to be one of these causes. Kerekes reported four cases which he considered to present typical symptoms of Lermoyez's syndrome, all of which he thought to be the result of angiospasm. Portmann⁹ in 1929 summarized all the literature relative to vasomotor affections of the internal ear and queried as to whether vasospasm was due to hyperactivity of the sympathetic nervous system or whether due to hyperfunction of the adrenal glands.

Let us now return to the remarks of Miles Atkinson. It was stated by him that Meniere's disease could be due to allergy or vasospasm. To repeat further, he found that the hearing impairment and tinnitus were less severe, and then improved or completely disappeared

following the attack of vertigo when the disease was due to allergy. These statements coincide exactly with Lermoyez's recordings, which were affirmed by Kerekes and cited by Nasiell¹⁰; but none of these writers named allergy as a basic factor. Is an allergic state the cause of angiospasm? Most authorities of allergy and angiology think not.

We wish to attempt to prove that Lermoyez's syndrome is an allergic disease and that it coincides exactly with the sequence of symptoms and events as described by Atkinson, which he claimed produced Meniere's disease, but only when allergic in origin. This is in contrast to the vasospasm origin of symptoms by Lermoyez. Could this reversal in sequence of symptoms as described by Meniere, then, truly be a Meniere's disease? Does not Meniere's disease demand the deafness and tinnitus to follow the vertigo? Would it not seem more logical that Atkinson is describing Lermoyez's syndrome, provided Lermoyez had based his symptoms on an allergic origin? We should like to believe that vasospasm as described by Lermoyez and Kerekes was due to an allergic origin. We do not wish to classify our cases under the title of Meniere's disease, because their symptoms appeared in reverse to Meniere's disease as we have always known Meniere's disease. Vasospasm of non-allergic origin may easily be an additional or secondary factor following allergy in the promotion of Lermoyez's syndrome, we readily admit, for we have observed some patients who had definite emotional and vasomotor instability. It is to be assumed that the internal auditory artery can be affected quite as well as any other artery elsewhere in the body, and it is not assuming too much to state that this artery, bilateral, may be involved.

Over a period of nine years, our observations in eleven cases, which we believe to have had symptoms characteristic of Lermoyez's syndrome in the clinical presentation of symptoms, have lead us to the opinion that Lermoyez's syndrome is entirely due to, or associated with, an allergic disease. All eleven cases are essentially well or have been well for a long period of time following their episodes of the symptoms and none have had any permanent symptoms which have incapacitated them, so far as known.

Our most important reason in claiming allergy as a cause of Lermoyez's syndrome was the consistent finding of urticaria—frequently the giant type of urticaria—which definitely is an allergic disease. Allergy studies have verified an existing allergic state in some of the patients, while in others who did not have allergy studies we assumed from the

history of urticaria given by the patient that the disease was allergic in origin.

We wish to present briefly some of the eleven cases we have encountered demonstrating Lermoyez's syndrome. There have been instances of recurrence of the disease, and there have been instances of bilateral involvement of the ears. We have been able to follow some of the cases quite satisfactorily, but there have also been some cases that have not been checked often enough. This is due to the fact that some of the patients resided a long distance from our hospital and only the initial examination could be made. The diagnosis thus depended on the history given by the patient, together with our findings at the time of the examination. Requests of patients to write in later developments as to whether they have had recurrence of symptoms or not have not been too satisfactorily complied with.

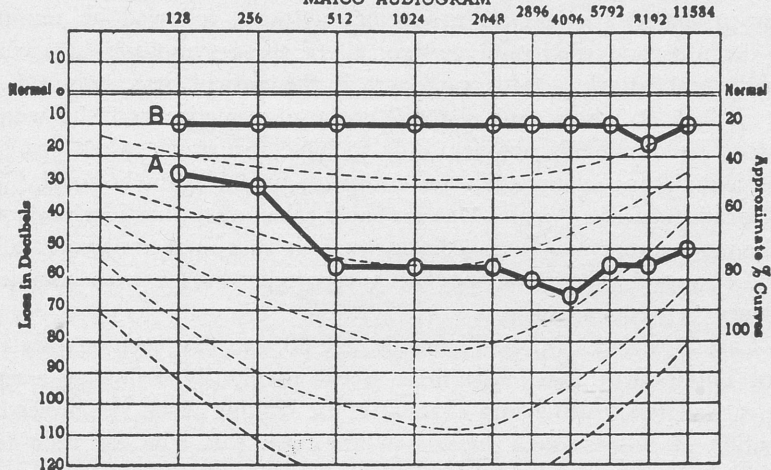
The eleven cases in our series, which admittedly is a small number, show that all of them belong to the white race. The age grouping was of especial interest in that 1 patient was in his twenties, 5 in their thirties, 3 in their forties, and 2 in their fifties. The oldest patient was 55 and the youngest 28. As to sex, 7 were female, and there were 4 males. The ages of the patients found in our series of Lermoyez's syndrome is of significance because Meniere's disease is expected to occur more often in the fifties and sixties, and not predominantly in the thirties and forties, as we found in our group. The blood serology in all cases was negative.

Case 1 (Fig. 1) V. F. E., age 39, a physician's wife, was our first patient to have what could be considered Lermoyez's syndrome. The past history was entirely negative for allergy and otherwise the patient's personal history had been essentially negative up to the present illness. In September, 1938, she had onset of a recurrent swelling, resembling angioneuroedema, on the right lower leg, over the shin. At that time there was much emotional strain and vasomotor instability because of her meticulous caring for a young child less than a year of age. After several weeks the condition subsided and then in November, 1938, she had pain in the left side and left chest. She had been carrying the baby on her left hip, and due to the size of the baby and her own small stature, she was constantly quite fatigued. She got along fairly well up to July, 1939, when she had giant urticaria involving the entire abdomen and thighs, and in November, 1939, there was swelling of both

V.F.E., 40, #17937

LERMOYEZ SYNDROME

MAICO AUDIOGRAM



A = Audiogram Right Ear 4/2/40

B = " " " 9/19/40

Left X Db. loss Whisp. Voice Air L. _____ R. _____ Bone L. _____ R. _____
 Right O Av. Db. Loss 256-2048 L. _____ R. _____ $\times 0.8$ App. % Loss L. _____ R. _____

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lips, the lower lip following the upper lip. Then for the first time she noticed tinnitus and impaired hearing in the right ear and she soon began to have dizzy sensations, and the most severe dizziness, which approximated vertigo, along with nausea, occurred in December, 1939. Hearing tests and vestibular tests unfortunately were not done at this time. She then had recurrent swellings of the body involving the right hand and arm, and the right side of the face occasionally felt numb. The dizzy sensations and nausea recurred, approximating vertigo. An audiogram was made on April 2, 1940, which showed marked loss of hearing of nerve type in the right ear. The hearing had again become much impaired on that date. On that same date she was found sensitive to lipstick and other cosmetics, but all other allergy studies were negative. She apparently had become sensitive to her cosmetics about the time of onset of symptoms the year before, with the recurrent attacks of symptoms as given. By changing her cosmetics, all of the symptoms

disappeared and there has been no recurrence to date of any of her symptoms of tinnitus, deafness or vertigo.

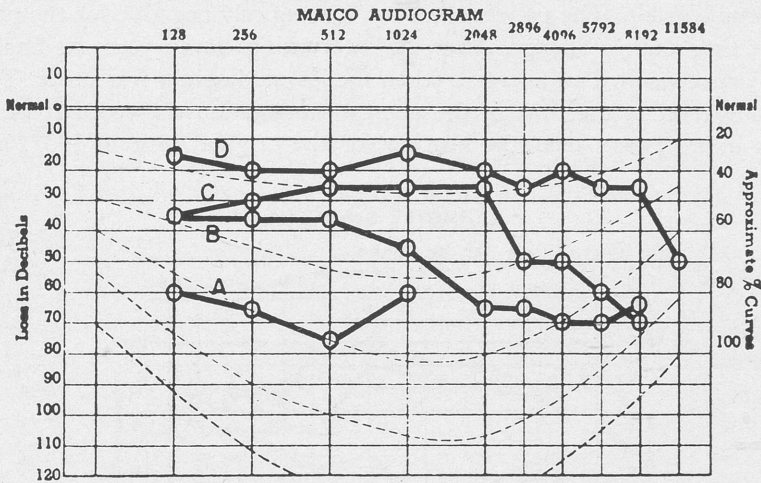
This case is an example of sensitivity to orris root or some of the other products in cosmetics producing urticaria, followed by tinnitus and deafness and then mild vertigo, all of these symptoms appearing in recurrent attacks. With each attack the patient was required to stay in bed, at which time she did not use cosmetics and she would improve or completely recover, only to have recurrence when she became ambulatory again when the cosmetics would again be used. The audiogram made on April 2 was probably taken at a time just previous to the most severe nausea and dizziness. This case further demonstrates that Lermoyez's syndrome may be a recurrent disease with complete recovery between attacks.

Case 2 (Fig. 2) M. G. F., female, age 38, was first seen on July 12, 1946. This patient stated that three weeks previously, following a ride in a car, she was sitting in a chair at home reading when she noticed a sensation of fullness and tinnitus in the right ear. The ear then felt "dead" and she noticed marked deafness in the right ear. Immediately thereafter she became nauseated and vomited, and she continued on numerous occasions to vomit over the next few days. She had difficulty in walking. She had a positive history of asthma for the past five years, and in February, 1945, she had a severe attack of urticaria. The general history for the past was otherwise negative and she stated that her blood pressure had always been normal. The otolaryngological examination revealed perfectly normal ear drums and quite patent eustachian tubes, with the remainder of the examination entirely normal, except for the right ear where hearing was almost totally lost. Hearing in the left ear was normal. The general physical examination, including a neurological examination and laboratory studies, revealed no abnormalities. On subsequent examinations, the first examination only three days after the first visit, the next one month later and the last six weeks later, the hearing in the right ear was found to show continuous improvement and the tinnitus was decreasing. The graph showing these audiograms, for the right ear only, demonstrates how much improvement in hearing this patient actually had. This patient decided to live in Florida and following the last audiogram immediately went to that state and has not been heard from since.

This patient exhibits a solitary but severe attack of Lermoyez's syndrome. She had a history of asthma and urticaria in the past but

M.G.F., 38, #B83699

LERMOYEZ SYNDROME



A = 7/12/'46

C = 8/16/'46

B = 7/15/'46

D = 8/25/'46

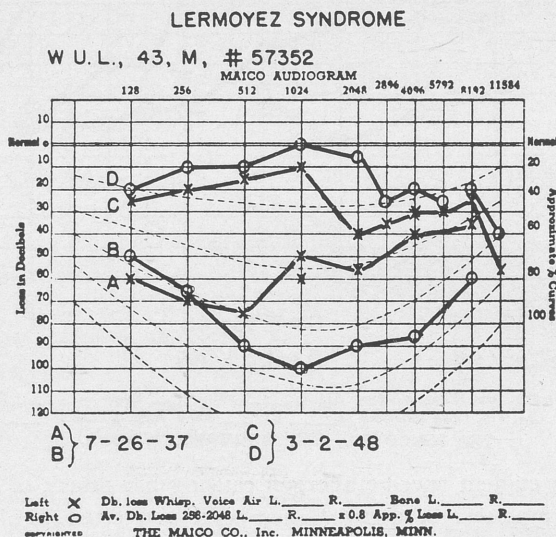
Left X Db. loss Whisp. Voice Air L. _____ R. _____ Bone L. _____ R. _____
 Right O Av. Db. Loss 256-2048 L. _____ R. _____ x 0.8 App. % Loss L. _____ R. _____

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there was no evident specific allergen causing her attack of Lermoyez's syndrome symptoms. Allergy studies were requested but, because of the distance to the hospital and inability to obtain appointment, were not done. Her recovery of hearing was progressing well during the six weeks' period of follow-up, but even so, her hearing recovery was not so rapid as Lermoyez always described. He expected recovery of normal hearing a few hours after the vertiginous attack.

Case 3 (Fig. 3) W. U. L., male, age 37. This patient was first seen in 1937. He had a partial blindness, bilateral, since childhood, and in more recent years had been associated with the State Blind Commission in his occupation. When first seen he had marked deafness, bilateral, and had a positive history of being sensitive to various allergens. This patient was seen on numerous occasions, at least annually, following the original visit, and each time he would be aware of recurrent tinnitus and impaired hearing, which in each case was followed by vertigo, nausea and vomiting, with all of the symptoms receding after

the vertigo. It was not until some years after he was under observation that we recognized the condition and classified him in the Lermoyez's syndrome group. The patient's history was entirely negative for the past other than for his allergy. After the intervening eleven years he finally this year consulted an allergist, when the last audiogram was made. The hearing level at the time of the allergy consultation showed a tremendous improvement over the hearing for the original visit in 1937, but



he states that his hearing is entirely normal between attacks. The first audiogram on the graph probably was made just before an attack of vertigo, and the last audiogram apparently was made just after an attack of vertigo, but before the hearing had completely returned to normal. Presumably his symptoms had been bilateral during all of his attacks. He was found sensitive to a few allergens when skin tested and is now only beginning his allergy therapy. We do not know what the future incidence of attacks, if any, will be. The otolaryngological and general examinations in the patient were entirely negative otherwise.

The three preceding cases were taken from our total of eleven cases and they present the various phases of what we consider Lermoyez's syndrome.

SUMMARY AND ANALYSIS

The reversal of the sequence of symptoms in Meniere's syndrome and Lermoyez's syndrome has been presented—namely, in Meniere's syndrome the tinnitus and deafness FOLLOW a vertiginous attack and become status quo or progressively worse, while in Lermoyez's syndrome the tinnitus and deafness PRECEDE the vertigo and diminish or completely subside immediately after the vertigo becomes established. Therefore, there exists a contrasting prognosis for the two syndromes.

Lermoyez attributed his syndrome to a vasospasm of the internal auditory artery without any suggestion of an allergic origin. We have observed eleven patients who demonstrated the typical symptoms of Lermoyez's syndrome, but in each instance we were able to find an allergic origin, mainly that of urticaria—usually the giant type—sometimes prior to the onset of symptoms. We believe the syndrome to be allergic in origin and urticaria probably to be the necessary etiological factor.

Atkinson credited a portion of his patients exhibiting Meniere's symptoms to an allergic etiology. Those patients reported in his dissertation coincided precisely in the appearance of their symptoms to Lermoyez's syndrome. We do not think that his patients should have been classified under the title of Meniere's syndrome merely because the group of symptoms was the same as occurs in Meniere's syndrome, since the symptoms appeared in reverse order. It seems no more justifiable to classify his allergic patients under the title of Meniere's disease than it would be to classify a typhoidal or streptococcal infection under the title of diphtheria, simply because all of these diseases produce fever, headache and numerous other similar symptoms.

It would seem correct, therefore, to make a diagnosis of Meniere's *syndrome* (as Lermoyez preferred to term it) only when the deafness and tinnitus *follow* vertigo, and likewise it would seem correct to make a diagnosis of Lermoyez's syndrome in all cases where the deafness and tinnitus are alleviated following the attack of vertigo, with eventual total recovery. We grant that true Meniere's syndrome may be allergic in origin, as has been shown by Duke, Proetz and others, or that Meniere's syndrome may be due to vasospasm, as stated by Atkinson. Likewise, we may also state that Lermoyez's syndrome is due to allergy, as we have attempted to prove, or it may be due to vasospasm as advocated by Lermoyez, Kerekés, and others. But, when we then use the terms,

Meniere's syndrome and Lermoyez's syndrome, we should be utilizing two terms which really signify a separate pattern of symptoms and a definite and different prognosis for each. This would clarify the confusion that exists at the present time and would cause many cases which are now classified under Meniere's syndrome to be rightfully designated under Lermoyez's syndrome.

CONCLUSIONS

1. From observations of eleven patients, all of whom had definite allergic manifestations, usually urticaria, and who exhibited symptoms of Lermoyez's syndrome, we believe that Lermoyez's syndrome is definitely an allergic disease.

2. We recognize Lermoyez's syndrome as a separate clinical entity in contrast to Meniere's syndrome and believe that patients who recover normal health after vertigo has cleared their tinnitus and deafness should be classified as having Lermoyez's syndrome.

3. On account of the excellent prognosis of Lermoyez's syndrome, compared to Meniere's syndrome, it is essential that Lermoyez's syndrome be recognized when it exists.

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DISCUSSION

FRANCIS L. WEILLE, M. D.: I cannot let this opportunity pass without mentioning one point to keep us in perspective. At a recent staff meeting at the Massachusetts Eye, Ear and Throat Infirmary, Dr. Francis Rackemann was present. He was at that time Chief of the Massachusetts General Hospital Allergy Clinic and stated that he had never seen a case of vertigo in which allergy was important in his experience. Even though the otolaryngologist does see occasional patients in whom the problem of allergy seems important in relation to vertigo, such patients are not frequent in proportion to other manifestations of the allergic state.

MARVIN F. JONES, M.D.: I would like to refer Dr. Weille to a paper published in the *Annals* some years ago in which I reported a series of cases, about eight in number. The title of the paper was "Allergy of the Ear."

DR. HENRY L. WILLIAMS, JR, M.D.: Mr. President, this paper of Dr. Eagle has left me somewhat confused. I think, however, that possibly some of the confusion results from a misunderstanding and possibly a slight misquotation of Meniere's original description of the syndrome that has come to be called by his name.

Meniere, instead of reporting a single case in which the patient died after exposure to cold, reported a series of cases—I believe there were 11—in which he very clearly described the syndrome; that is, deafness, usually the first symptom although it sometimes appears years before the attack, tinnitus and the initial attack of vertigo followed by remissions and recurrences. Meniere described deafness as continuous and progressive to the extent of complete loss of hearing. This description has been corrected by Dr. Crowe, who showed that there is a very marked tendency toward remission, not only in the attacks of vertigo but as to the degree of hearing loss.

Meniere wished to cap his description of the new syndrome with a pathologic picture. Accordingly he inserted the report of a case to which Dr. Eagle referred, a case which he had reported thirteen years previously and happened to recall at the time he wrote his paper. McKenzie, who dug this matter out, stated that whatever the case might represent it was certainly not Meniere's disease. It is therefore incorrect to report that Meniere stated that this disorder was due to hemorrhage into the labyrinth. I believe that one would be less confused if the syndromes which Meniere actually described were given as the basis of discussion. There is nothing in Meniere's description which would outlaw the hypothesis of vascular spasm as a cause of this disorder. Therefore, the attempted distinction between Meniere's disease and Lermoyez's syndrome on the hypothetic basis that the latter is caused by vascular spasm is not valid.

As far as the patient's sensitivity to his histamine is concerned, I think histamine as an allergen has been thoroughly discredited by the work of Farmer in New York and other men who have investigated this subject. I do not believe that testing with histamine is useful as far as the differential diagnosis of Meniere's disease is concerned.

At the Mayo Clinic we see about 250 persons a year who have what we designate as "Meniere's disease" or "Meniere's syndrome." As I reviewed the group of patients seen over a two-year period I found but one case which I thought might possibly fall into the classification of Lermoyez's syndrome. The patient was a woman past the menopause, who apparently also had otosclerosis. The history of the development of loss of hearing and the audiogram were rather typical of otosclerosis. However, she gave a history of attacks of vertigo with nausea, vomiting and nystagmus which occurred after the menopause and stated that her hearing was improved during the attack of vertigo. Her symptoms of vertigo were relieved completely by the use of stilbestrol. Whether stilbestrol caused either a vasospasm or a collection of extracellular fluid in the inner ear to diminish is a matter for speculation. It would seem probable, however, that such a case is extremely unusual.

One might also call attention to the fact that frequently many so-called allergic symptoms and signs such as asthma, angioneurotic edema, urticaria and the like, as well as Meniere's disease, are not an antigen-antibody type of reaction but are the so-called physical allergy type of reaction originally described by Duke. A great deal of work has been done by Ottfried Mueller and others on the "physiologic pathology" of "physical allergy," a condition which may or may not be called an "allergy," depending on the particular definition of allergy which the discussor wishes to adopt. This work tends to indicate, however, that Meniere's disease is associated with arteriolar spasm and capillary dysfunction. Therefore, the presence of vascular spasm would not distinguish Meniere's disease from Lermoyez's syndrome. As a matter of fact, I do not feel that enough evidence has been produced by either Lermoyez or Dr. Eagle to establish the existence of a separate syndrome. Certainly, several of the cases which Dr. Eagle has presented do not meet one of the diagnostic criteria which he established for Lermoyez's syndrome in the early part of his paper; namely, that the hearing is returned to normal by the attack of vertigo. Dr. Eagle, however, has presented an extremely interesting group of cases which will stimulate speculation as to the exact mechanism by which these functional disorders of the labyrinth are caused, and will undoubtedly lead to more defensible hypotheses than have yet been produced.

W. W. EAGLE, M.D. (Closing): Mr. Chairman, I am delighted that no one asked for a discussion of the labyrinthine function in these cases. Those studies have not been too well accomplished, but we do hope in the future to make some very accurate studies of the labyrinth on patients exhibiting Lermoyez's syndrome.

It is hoped by this discussion today to prove that Lermoyez's syndrome is a disease in its own entity and that it is to be distinguished from Meniere's disease. It should be less confusing in the future in following patients to know that they exhibit either definite Meniere's syndrome or definite Lermoyez's syndrome.

It is our impression that Lermoyez's syndrome is an allergic disease in origin, and this fact alone may be helpful in the future in considering a diagnosis.

I wish to thank the discussers.

THE VOLUME OF THE BRONCHIAL TREE
AT VARIOUS LEVELS
AND ITS POSSIBLE PHYSIOLOGIC SIGNIFICANCE*

A. C. HILDING, M.D., AND DAVID HILDING (*by invitation*)

DULUTH, MINNESOTA

We consider the nose to be an efficient air cleanser, and so it is. It removes the dust and bacteria which are in suspension, and at the same time humidifies the air. Despite this, some particles do get through to the alveoli as is indicated by the disease of silicosis, and by the black spots one sees on the plura of the lungs in any adult subject at necropsy. This brings up the question as to whether or not there is any other air cleanser lower in the tract. Proetz¹ states that there is, but does not enlarge upon the subject. A laryngectomized person gets along very well without a nose. Nature seems to have a safeguard, or substitute, against the loss of most other organs. What is the subglottic substitute for the nose?

One of us had occasion a few years ago to make some preliminary measurements on the bronchial tree of a dog. These incomplete measurements gave the impression that there might be a constriction of the total volume of the respiratory tract a short distance below the bifurcation. We tried to look up the volumetric figures of the human bronchial tree in the literature but were unable to find anything except on the tiny microscopic branches (Fig. 1) (Miller,² Best and Taylor³). A number of internists and physiologists, and anatomists were asked if they knew what the measurements might be. None could give any specific information, but all were of the opinion that the volume of the tract increased progressively with the branching in much the same way as does the circulatory system.

Proetz, in his book on "Physiology of the Nose," shows some figures of experiments in which dust-laden air was passed through glass tubes

* From the Department of Ophthalmology and Otolaryngology, University of Minnesota.

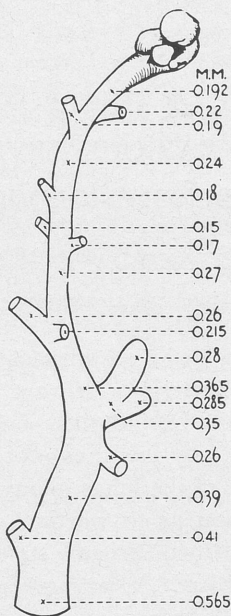


Fig. 1.—Diagram of a reconstruction of a bronchiolus and the structures between taken from W. S. Miller. According to these measurements, there is a ninefold increase in the total volume of the air passages between the bronchiolus and the alveolar duct. Beyond the latter, there is a still greater increase in volume.

of various forms. He found, that if there was constriction in such a tube, the dust would be deposited just beyond the constriction. It also settled just beyond any bends or turns in the tube. The possibility occurred to us that if there were a constriction or isthmus in the bronchial tree, it might be purposeful. It might act to deposit such dust as the air entering the trachea might carry with it, upon the lining mucous blanket, which is, of course, carried away by ciliary action.

Experiments, similar to those described by Proetz, were done by passing cigarette smoke through glass tubing in which had been blown round, or fusiform bulbs, and other irregular forms, as well as the

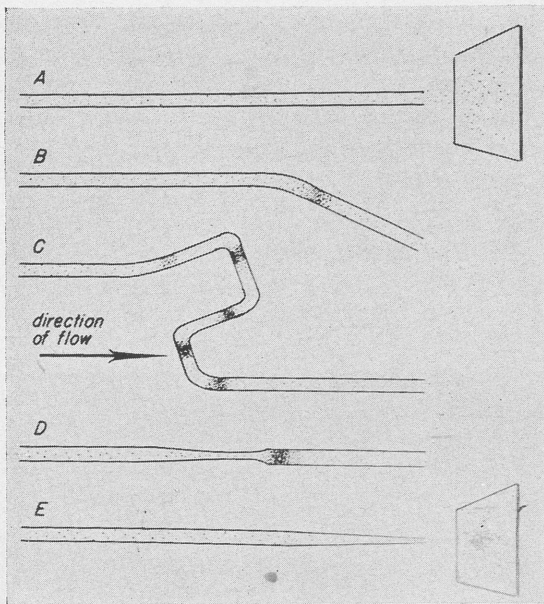


Fig. 2.—Illustration taken from Proetz showing points of deposition in different shaped tubes.

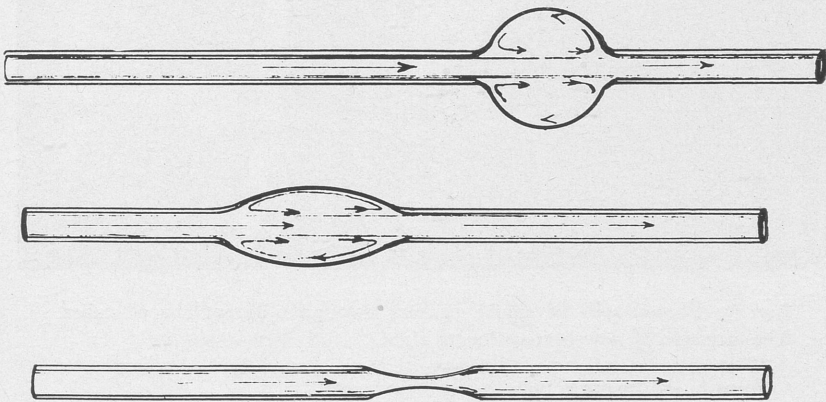


Fig. 3.—Diagram of reverse eddies formed in bulbous enlargements on glass tubing through which cigarette smoke was drawn. The eddy, in the case of the constricted tube, formed in the obtuse angle just at the end of the constriction. This eddy also flowed in a reverse direction but was so tiny that it was difficult to see. Actually these eddies are rings, the circumference of which revolves as indicated. Suspended matter tends to deposit from these eddies upon the adjacent glass wall.

constriction which he described (Fig. 2). It was found that when an air stream laden with cigarette smoke was passed through a bulbous enlargement on a glass tube that reverse eddies formed in the bulb (Fig. 3). The major portion of the stream, however, flowed smoothly through the center of the bulb and did not enter the eddies at all. The eddies rotated rather slowly and did not join the central stream to any extent. It seemed apparent that only a very small portion of the air passing in the stream ever got into these eddies, and that which did enter the eddies tended to remain there. A deposit formed in the bulb, but, judging by simple inspection, it seemed to include only a very

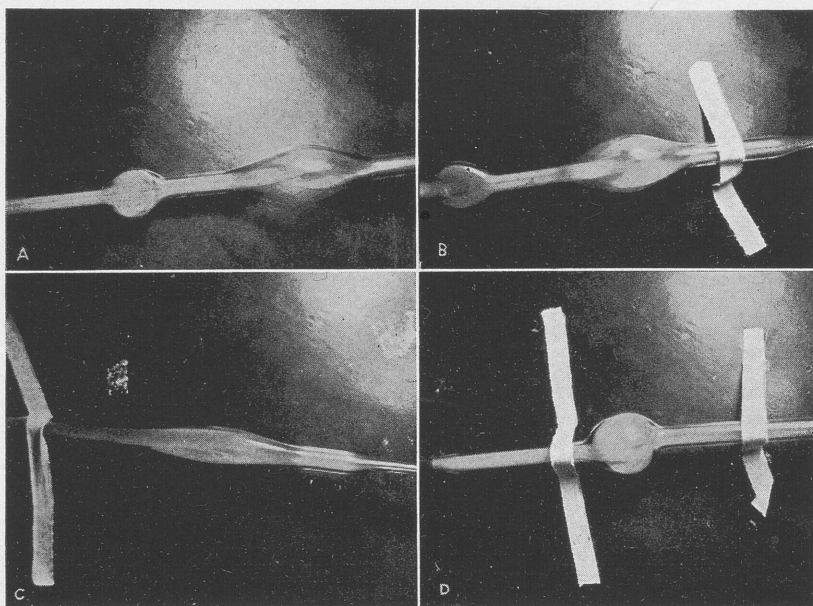


Fig. 4.—Photographs of smoke passing through different shaped tubes. The direction of flow is from left to right.

A. The reverse eddies are well formed in the round, and just beginning to form in the fusiform bulb.

B. The smoke is beginning to clear from the central portion of the round bulb throwing the eddy into relief. The eddy in the fusiform bulb is not completely formed, and is enveloping the central stream like a sleeve.

C. In this long, narrow curved enlargement, a larger eddy forms below, and a smaller, very thin one above.

D. Burbling in a distorted bulb. The smooth flow of the central stream seen in the other forms is broken up in this one.

small percentage of total suspension. Most of the suspended material passed through the tube in the main stream of the air flow and never got into the eddies at all. The action was similar in the fusiform enlargements, and in the enlargements just beyond the constricted areas (Fig. 4). Reverse eddies formed in both instances but only a small portion of the air entered the eddies. Irregular shapes produced burbling and spiral motions. It seemed apparent that eddies and irregularities in air-flow would not serve very well as air cleansers. Nevertheless, it was decided to make some measurements of the bronchial tree of man.

Material. The material used consisted of a few specimens of lungs, or portions of lungs, removed at necropsy. One was the entire tract from a stillborn infant, and four were from adult males whose ages were, respectively: 34, 44, 80, and 83. The bronchial tree of the left upper and left lower lobes of the newborn were measured; the left lower lobe of the 34-year-old male, the right middle and right lower lobe of the 44-year-old male, and the left lower lobe of the 83-year-old male. The entire tract of the 80-year-old male was obtained but the data was discarded because it was found to be unsatisfactory. Fifty-seven bronchi and bronchioles were measured in the left lung of the newborn, and in the adults, from 96 to 276 in each lobe.

Technic. It was intended, at first, to make corrosion specimens by filling the respiratory tract with a lightweight resin, and then dissolving away the tissue. Thus far, we have been unable to obtain the resin. An attempt was then made to fill the entire respiratory tract of the 80-year-old male with gelatin and section the gelatin at intervals of 1 centimeter. This method proved to be impractical, and the specimen was discarded. In the remaining specimens, the entire bronchial tree was laid open by slitting it posteriorly. It took some little practice to cut the lung tissue in such a way as to avoid cutting across the bronchi. When a lobe has been dissected out in this manner, the lung tissue is cut into thin slices which lie in antero-posterior planes. If any considerable portion of lung remains intact, then some branches have been missed. Each branch was followed as far as possible with probe and scissors. Despite great care some may well have been missed. Measurements were made at points just beyond each division. The bronchi were grouped according to the number of divisions through which they had gone.

The measurements were made, by means of a small millimeter rule, across the slit bronchi as they lay flattened out in the form of bands or ribbons. As the measurements were made, the figures were recorded in a diagram similar to that used for family trees. About 650 measurements were made. These measurements corresponded to the circumference of the bronchi during full distension. The cross-sectional areas were calculated from the circumferences.

Sources of Error. This method is subject to a number of sources of error. The trachea and primary bronchi are not round, and, using the measurements as obtained, the figures for cross-sectional area would represent perfectly round tubes. Another source of error is the distensibility of the membranous bronchi. When they have been slit open, it is very easy to stretch the band of mucosa in such a way as to make the circumference seem larger than it truly is. Therefore, the measurements of the trachea and bronchi, which have cartilaginous arches, or rings, in their walls, yielded values which were greater than the actual cross-sectional area during life. On the other hand, it is not likely that the membranous tubes in life are fully distended. Therefore, these values are also probably considerably exaggerated since in each case the measurement would be that of maximum distension. (The criticism of casts made from heavy Wood's metal is that the delicate membranous tubes become greatly distended.) During the respiratory cycle the membranous bronchi and bronchioles vary greatly in diameter, whereas the variations in the bronchi and trachea, which have rings, is comparatively slight during the respiratory cycle. At best, the figures obtained are only approximations.

Another source of error was the irregularity of branching. In some regions it seemed dichotomous, but often monopodous, or a mixture of dichotomous and monopodous. Occasionally the branching was found to be trichotomous. This irregularity in branching made it difficult to classify the tubes into groups which would represent comparative progression along the tract. At first it was thought that all branches should be measured at regular intervals at a given number of centimeters below the bifurcation. This, however, did not prove to be feasible. Some would branch two or three times before others had branched at all. Some would go through three or four divisions, whereas others would go through eight or ten.

The figures of the cross-sectional areas of the various bronchi were added together, in order to construct a single tube. In so doing, it was necessary to use some of the cross-sectional areas at several different levels more than once. For instance, if one bronchus coming off at a right angle high in a lobe went through just two or three divisions to reach the minimum where measurements were possible, and the other branches going deeper into the lung went through many divisions, in order to indicate the full volume of the tract it was necessary to include the measurements of the short branches, together with the longer branches, at each successive lower level.

Measurements were made after each branching or division, and each branch was followed out as far as it was possible to measure by this method. The measurements were all grouped according to the order of branching. For instance, all measurements just beyond the fifth division in all the different branches were grouped together, and the cross-sectional area of each tube calculated. In order to give a composite picture of the volume of the entire tract, all of the cross-sectional areas of all the tubes at each level were added together. From these values composite diameters were calculated and a single composite tube diagrammed. Also, all of the measurements which were made were added together to give composite figures of the circumference.

Results. It was found practically at the outset that there was no such constriction as the preliminary measurements on the dog seemed to indicate. There were indeed many minor irregularities, such as the formation of a sort of hood at the beginning of a bifurcation where the cross-sectional area was greater than either just above or below this point. There were also many bends and turns, but, as far as could be determined, there was nothing that would indicate the use of eddies for the purpose of cleansing the air.

It also became apparent early that there was no marked progressive uniform increase in the total volume between the trachea and the small bronchi as seems to be the general impression. In most instances there was a small increase, about 50% to 100%, and there was a corresponding small increase in the calculated composite diameter, up to 40%. There was some slight irregularity, probably due to error in the method of measurement, but generally the composite diameters were surprisingly consistent and uniform.

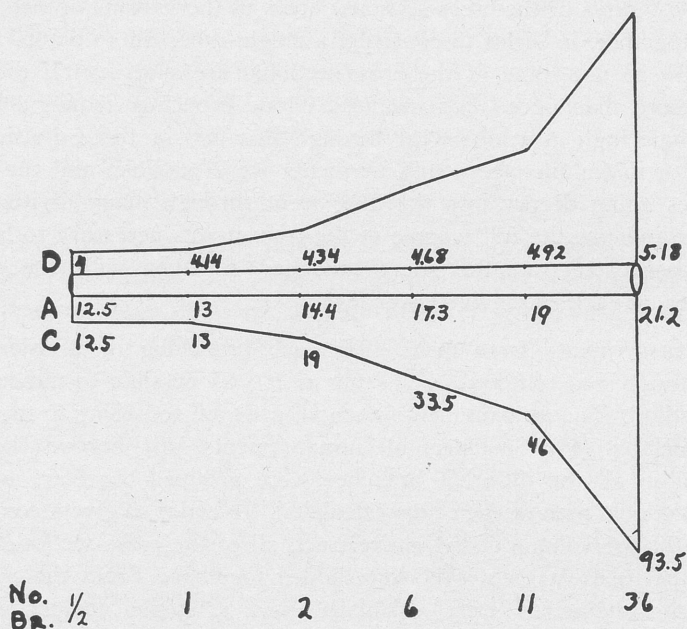


Fig. 5.—Composite measurements at six successive levels of the bronchial tree from the left lung of a stillborn infant. The bronchial tree was slit posteriorly, and the circumferences of the trachea and all bronchi down to an average size of 0.83 mm. in diameter, were measured. The comparative composite volumes of the tracheo-bronchial tree at the six different levels are shown constructed into the form of a single tube. The tracheal end is toward the left. Figures opposite "D" represent the calculated diameters of the composite single tube, and those opposite "A", the summation of the calculated cross-sectional areas of the bronchial tree at the six different levels. There is a rather small increase in total volume, as indicated by the increase in diameter from 4 mm. to 5.18 mm. The figures opposite "C" represent the summation of the measurements of circumferences. The area between the diverging lines represents diagrammatically the actual area of the mucous membrane lining the tracheo-bronchial tree. The figures below (No. Br) indicate the number of bronchi measured at each level. The reason that the trachea is given as $\frac{1}{2}$, is that only the left lung was measured. It is assumed that half the air passing through the trachea goes to the left lung. The calculated diameter of a tube of half the volume of the trachea measured in this instance would be 4 mm. The actual diameter of the trachea was 5.76 mm. (calculated from the measurement of the circumference.)

D—Calculated diameters of the composite single tube having the same volume at the various levels as the bronchial tree.

A—Summations of the calculated cross sectional areas of the bronchial tree at the various levels.

C—Summations of all measured circumferences at the six various levels.

No. Br—Number of bronchi measured.

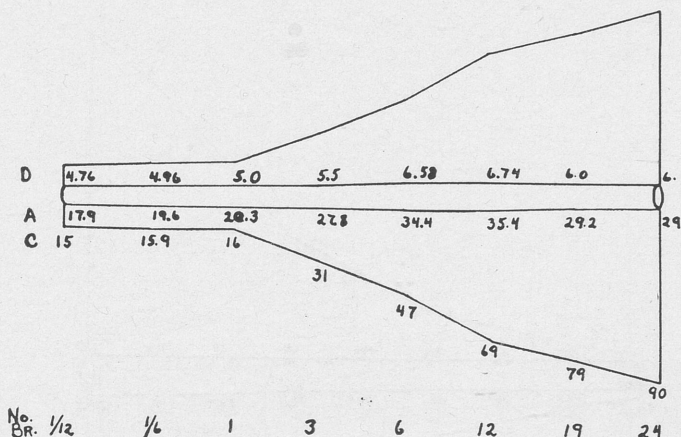


Fig. 6.—Composite measurements at eight successive levels of the bronchial tree of the middle lobe of the right lung from a 44-year-old man. From the measurements obtained, it was estimated that one-sixth of the air passing through the right bronchus, and one-twelfth of that passing through the trachea entered the right middle lobe. The diagram shows the size of a composite single tube having the volume of the bronchial tree at the various levels, including fractions of the trachea, and right bronchus.

D—Calculated diameters of the composite single tube.

A—Summation of the calculated cross sectional areas of the bronchial tree at the various levels.

C—Summations of all measured circumferences at the various levels.

No. Br.—Number of bronchi measured at the various levels.

As in the case of the infant, (Fig. 5), there is comparatively small increase in total volume of the tract from the trachea to the smallest bronchi measured. In this case, the average size of the smallest bronchi measured was 1.2 mm. The total area of the lining mucus membrane, as represented by the diverging lines, shows a marked progressive increase at the various levels.

When a single tube is constructed from these calculated diameters representing the total cross-sectional area of the bronchial tree at different levels from the trachea to the small bronchi, it is found to have a surprisingly uniform caliber (Figs. 5 to 9). A tube constructed from the composite circumferences, on the other hand, opens out like a funnel, and would represent the area of ciliated mucosa with which the air must come in contact. This great widening is due, of course, to the fact that the total circumference of many small tubes is much greater than that of a single tube of equal volume. The number of tubes in the

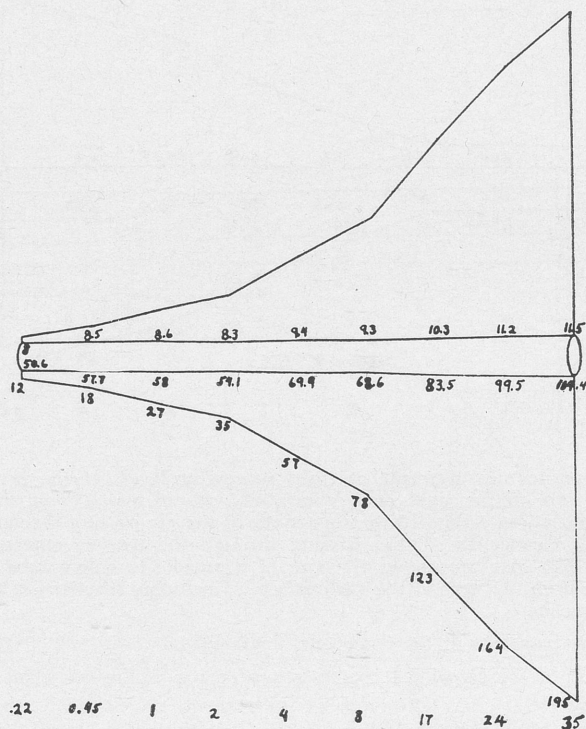


Fig. 7.—Composite measurements at nine successive levels of the bronchial tree of the lower lobe of the right lung from a man 44 years old. From the measurements obtained, it was estimated that 22 per cent of the air passing through the trachea, and 45 per cent of that passing through the right bronchus, entered the right lower lobe. The size of the composite tube shown is calculated at the first two levels on the basis of these per percentages. The diagram of the composite tube indicates the total volume at the various levels. Again, as in the case of the middle lobe, and of the left lung of the infant, there is a small progressive increase in volume at the successive lower levels as indicated by the increase in diameter from 8 to 10.5 mm. The average diameter of the smaller bronchi measured in this specimen was 1.76 mm.

D—Calculated diameters of the composite single tube of the same volume as the bronchial tree.

A—Summations of the calculated cross sectional areas of the bronchial tree at the various levels.

C—Summations of all measured circumferences at the nine various levels.

No. Br.—Number of bronchi measured.

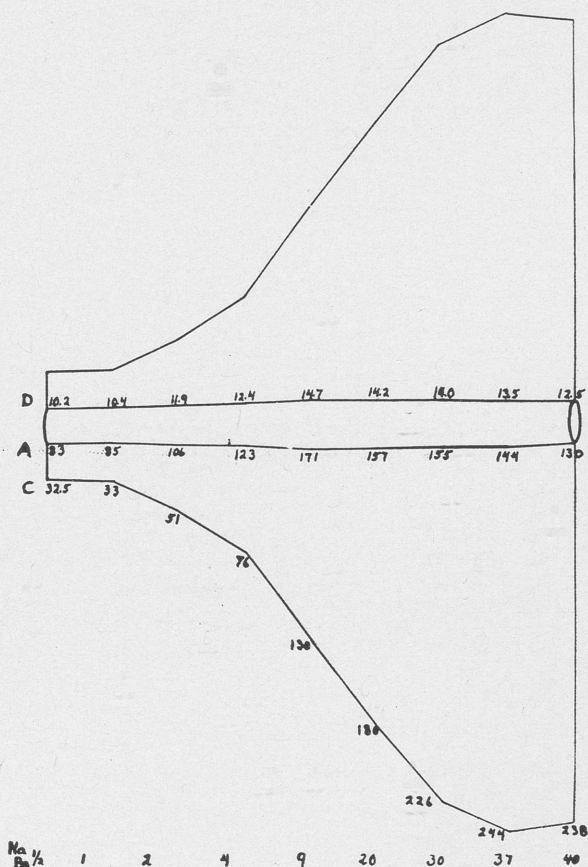


Fig. 8.—Composite measurements at nine successive levels of the bronchial tree of the left lower lobe from a man 83 years old. The left-hand figures represent the left bronchus. No measurement of the trachea was obtained in this case. There was considerable broncho-pneumonia in this lung, and many of the membranous bronchi were dilated and full of purulent secretion. This may account for the increased volume in the middle of the composite tube shown in the diagram as compared with the two ends. It may also account for the greater composite circumference at the eighth level as compared with the ninth. On the other hand, it may be that some bronchi at the ninth level were missed. The diameter of the composite tube in this case increased from 10.2 mm. at the right bronchus to 12.5 mm. at the level of the smallest bronchi measured (in this instance, 1.74 mm.)

D—Calculated diameters of the composite single tube having the same volume at the various levels as the bronchial tree.

A—Summations of the calculated cross sectional areas of the bronchial tree at the various levels.

C—Summations of all measured circumferences at the nine various levels.

No. Br.—Number of bronchi measured.

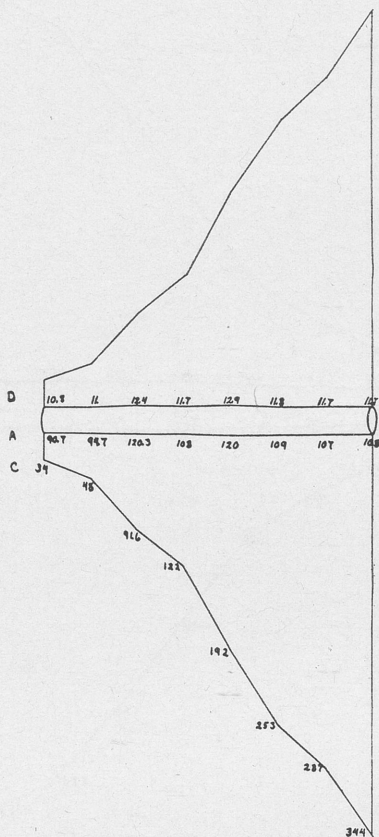


Fig. 9.—Composite measurements at eight successive levels of the bronchial tree of the left lower lobe from a man 34 years of age. The left-hand figures represent the left lower lobe bronchus.

The progressive increase in volume was quite small in this case as indicated by the increase in cross sectional area from 90.7 to 108 sq. mm. The increase in the area of the mucous membrane, on the other hand, was very striking. The total of the added circumferences increased ten fold between the first and the eight levels. The specimen was a good one, and it was possible to measure the bronchi down to an average diameter of 1.12 mm.

D—Calculated diameters of the composite single tube having the same volume at the various levels as the bronchial tree.

A—Summations of the calculated cross sectional areas of the bronchial tree at the various levels.

C—Summations of all measured circumferences at the eight successive levels.

No. Br.—Number of bronchi measured.

baby increased from one (the trachea) to 35 small measurable tubes in the periphery of the left lung. Single lobes were measured in the adult specimens. The increase in these varied from one to 35 to one to 96.

Discussion. The first conception of an isthmus beyond which eddies form for the purpose of depositing suspended matter proved to be entirely erroneous. In any case, eddies produced in irregular tubes do not form an efficient device for removing suspended dust particles. The air in the eddies rotates but tends to remain in the eddies, and does not join the main stream. Suspended matter in the main stream does not become deposited. In fact, one gets the impression that in small tubes, through which an air stream is passing, the air current tends to streamline its own channel, filling in irregularities with rather slow moving eddies of comparatively stagnant air.

The principle of air eddies probably is used in the upper part of the nose to bring air into the olfactory fissure, but it is not important in the cleansing of air. On the other hand, impingement is probably much more effective, and it occurs more or less all through the tract at the irregularities, turns, and branchings. The conception that the total composite volume of the respiratory tract increases uniformly and progressively in volume, as it does in the blood vessels, also seems to be inaccurate. The figures for the blood vessels given by Glasser⁴ indicate that the volume of the arterial tree increases four fold in passing from the aorta to the primary branches, which are about forty in number. The volume increases approximately six fold to what he calls the main arterial branches. (The volume of the arterioles is about 150 times that of the aorta). This is more than the measurements found in the bronchial tree, where the increase from the larynx to the smallest tubes which we measured, is only from 50 to 100 per cent.

The conditions in the two systems are, however, very different. In the blood vessels, a rather heavy fluid is passing under pressure at a fairly high velocity. Friction increases very rapidly as the diameter of the contained vessels is reduced, and is very great indeed in the microscopic vessels. If there were no increase in total volume, the work of the heart would be increased tremendously. Moreover, the flow in the vascular system is all in one direction, and there are no stagnant pools of blood in the arterial tree. The material passing in the bronchial tree is a gas; and the friction factor is very much less. This is not only be-

cause of the difference in nature of the two materials—the blood and air—but also because the velocity of the latter is comparatively low. In ordinary quiet respiration the flow down the trachea is calculated to be about one meter per second, and perhaps half that rate in the smaller tubes. There is another factor which is entirely different than in the circulatory system; namely, the air does not pass continuously through the tubes, but flows first in one direction, and then comes to a stop, and then flows back again. This means that at the end of inspiration the bronchial tree is left full of fresh air, and at the end of expiration is left full of alveolar air. In other words, the volume of the tracheo-bronchial tree, which is about 150cc, is dead space. We take in about 500cc of air with each breath in quiet respiration, but the first 150cc which passes into alveoli is not fresh air. It is the last 150cc to have been expelled at the last previous expiration. Therefore, we get 350cc of fresh air with each inspiration. If the volume of the respiratory tract were increased six fold then the dead space would be 900cc, and in order to get 350cc of fresh air, we would have to inhale 1250cc with each breath. This would seem to be both uneconomical and unnecessary. Moreover, if the increase in size were progressive and uniform from the trachea to the terminal bronchioles, the increase in volume would probably be much more than six fold. Such an increase in volume would require a larger chest cavity.

On the other hand, somewhere between the smallest tubes which we have measured and the alveoli there is a very large increase in volume. Miller⁵ has shown this in his book in an illustration of a reconstruction of the fine air passages between the bronchiolus and the alveolus. There are other data (Best and Taylor³) indicating that there is a substantial increase in total volume in these small air tubes of less than 0.5mm. in diameter. This would mean that there is a very large number of these small tubes which together would form an effective filter. One trachea among these specimens was very carefully measured, and the cross-sectional area found to be about 340 square mm. If an area of 340 square mm. is divided into 1 mm. tubes, 430 tubes will be required. If the tubes are 0.5mm. in diameter, it will take 1730 of them to make up a cross-sectional area of 340 square mms. If the tubes are 0.35mm. in diameter, which is about the size of the terminal bronchioles, it will take 3700 to make up a total cross-sectional area of 340 square mms.—this without allowing for any increase in any total volume at all below the

trachea. And, we have found that the volume does about double. In addition, according to Miller's measurements, there is a volumetric increase of five to ten fold in the passages less than 0.5mm. in diameter before the alveolar ducts are reached. The greatest increase in volume of the whole tract apparently takes place between the alveolar ducts and the alveoli. The ciliated mucous membrane extends down through the whole tree as far as the respiratory bronchioles. Here, where the alveoli begin to appear, it gradually disappears. The terminal bronchioles, however, are well ciliated. One illustration in Miller's book pictures a bronchiole 0.35mm. in diameter, and the cilia are very well formed. Considering bronchioles 0.35mm. in diameter, and making calculations from the data which we have, there would be at least from 15 to 40,000 of them—and possibly many more. These would constitute a very efficient filter since each of the bronchioles are lined with a blanket of mucous. Assuming that there are 25,000 such tubes, their total cross-sectional area would be 2,250 sq. mm., and their composite circumference about 26 meters. That would mean that the 500cc of tidal air, which we pass back and forth in quiet respiration, would be divided up into 25,000 small jets and would be distributed over a mucous blanket 26 meters in width. The greatest possible distance between a dust particle and the mucous blanket would be one-sixth of a mm. The rate of flow would be about 15 centimeters per second. Such an arrangement would furnish a very effective filter, and a last chance depository for dust and suspended matter before the cilia cease. When the respiratory bronchioles are reached, the epithelium changes to a non-ciliated form. May we suggest for this arrangement of bronchioles the name "bronchiolar filter."

The protection of the delicate alveolar lining from damage by inspired air is impressive—first, the efficient cleansing, warming, and humidifying action of the nose; then further cleansing and warming of the air as it flows down the bronchial tree; final cleansing in the bronchiolar filter where it passes over several meters of ciliated mucosa. There is further protection in the rapidly progressive reduction in velocity of flow in the small passages from one meter in the trachea, to probably less than one centimeter per second as it enters the alveoli. In addition to these measures, 350cc of fresh air in quiet breathing is preceded by 150cc of air which has already been in the alveoli, and is then diluted in some 2000cc of residual air. It is possible that this bronchiolar filter might be capable of furnishing both heat and moisture for inspired

air in the absence of nasal functions. That would depend upon the volume of blood flow and the production of respiratory mucus.

Summary. There is a small progressive increase in the volume of the tracheobronchial tree from the larynx to the bronchi measuring 1.5mm. in diameter. It is from 50 to 100 per cent. In this portion of the tree there does not seem to be any very effective air-cleansing device except the direct impingement of the air on the mucous blanket lining the tree. From fragmentary data available, there seems to be very considerable expansion in the volume of the tract in tubes less than 1.5mm. in diameter. Before the cilia cease the tree has divided into bronchioles which number at least somewhere between 15 and 40,000 tubes, each of which is lined with ciliated epithelium and a sticky mucous blanket. The composite circumference of these tubes measure many meters. This, the bronchiolar filter, is an effective, final device to protect the alveoli from dust and the fumes that might gain access to the trachea. One infers that the cilia move off the mucous blanket as is the case elsewhere in the tract. One might also infer that this bronchiolar filter would be even more effective in the matter of dust removal and humidification, if sufficient secretion is produced, than the nose. It does not, of course, protect the trachea and larger bronchi from improperly prepared air.

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DISCUSSION

H. I. LILLIE, M.D.: It was implied that perhaps the bronchial tree could take the place of the nose in conditioning the air. It is not mentioned how moisture and warmth can be obtained. In breathing through a tracheal tube the air must be more or less conditioned from the standpoint of warmth and moisture before it goes into the trachea if the patient is to be comfortable. In other words, if this mechanism takes the place of the nose, how does it do it?

THOMAS C. GALLOWAY, M.D.: We hear a lot of talk of the danger of narrowing our thinking in subjects related to our specialty. This investigation of an allied field is very important and broadening.

There is a problem I believe which is related. Will Dr. Hilding give us a visualization of what can happen in the area of widening of the bronchioles to accumulation of secretion in such a condition as bulbar poliomyelitis? Here the usual mechanism of cough, tussive squeeze and respiratory movement have failed.

PRESIDENT PROETZ: Is there any further discussion or are there any questions?

This is such a fundamental type of paper that it is difficult to discuss without having had an opportunity of digesting it. To my mind it is one of those things which will go into the literature and be referred to for years. The information which it stimulates will pyramid the bronchial tree.

Will Dr. Hilding please close.

A. C. HILDING, M.D. (Closing): I want to thank the discussers for their remarks.

In reply to Dr. Lillie's question, how does this protect the trachea, it does not. I think he perhaps missed the statement that the trachea and terminal bronchi are left unprotected. I believe after the tracheotomy that for a while the trachea secretes considerable mucus and crusts are apt to form and there has to be special protection. However, the trachea and the primary bronchi are not the prime things to be protected. It is the delicate lining of the alveolus where the oxygen exchange takes place. The protection of that lining is truly impressive in the normal. We have first, the function of the nose, then the air flowing down through this tree, depositing material as it goes, then going through all of these thousands of bronchioles and being further conditioned. So far as the cleaning is concerned the fact is that only 350 cc. of fresh air gets in. That is spearheaded by 150 cc. of air which has already been in and is well conditioned. Then on top of that there are 2,000 cc. of residual air. If I can understand it correctly, the fresh air actually never gets in contact with the alveolar wall at all. There is an interphase between the residual air and the new air coming in and an exchange takes place by diffusion. There is a marked protection there. I do not know about the warming and the moistening in this bronchiolar filter. It is stated that there are not many mucous glands. I have not examined them microscopically myself. You would not need many glands in 25,000 tubes to equal what you have in the nose.

As far as Dr. Galloway's question is concerned, I cannot say a great deal except in respiratory paralysis the cilia are not paralyzed. The cilia act without any nervous control at all. If there is a tracheotomy tube in it has to be cleaned. The cilia bring mucus up to that point. If you work with the trachea you see how extremely little it takes to serve the function of the mucous blanket and the cilia moving in the wavy material. I think I cited before the instance of one ox

trachea that I nailed out and used all one afternoon for some tests. The blanket moved off fast. I measured it and it was about 35 mm. a minute and that continued for two or three hours. At the end of that time there was so little mucus at the top end of the trachea one could scarcely pick it up. In other words, the mucous blanket is vanishingly thin. They say you can pound a piece of gold the size of a pea so thin it will cover an acre. I think the same thing is true of the respiratory mucus.

NERVE DISTRIBUTIONS OF CLINICAL SIGNIFICANCE IN LARYNGOLOGY

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AND

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ANN ARBOR, MICHIGAN

An uncommon manifestation of interest can hardly be expected to well up among clinical men over a complex anatomical subject of this character, yet it is hoped that it will serve in a measure as a basis for accurate thinking in some of the practical aspects of laryngology. Specifically the text treats of certain respiratory reflexes and the nerve pathways along which they travel. An attempt is made to describe various nerve circuits composed of peripheral, nuclear, and association routes and to explain by reason of their position, relationships and function, some of the phenomena observed in clinical practice.

While much of this subject may be only of academic interest, it is nevertheless axiomatic that the basic sciences lay the foundation of medicine. From a knowledge of them one may travel in any direction. A veritable panorama of opportunity is exposed to the individual who is grounded in the fundamental principles of some phase of science; the opportunity for intelligent observation, research, the development of therapeutic facts, and the ability to put these facts into discriminate practical usage. It may not be too much to expect therefore that this anatomical study of some of the reflexes of respiration may serve as a common core about which a better understanding of respiratory symptomatology and a greater knowledge of rational methods of treatment may be enveloped.

I. The neuron paths from cortex to cord concerned with increasing respiration.

The frontal cortex is connected with the preoptic area and rostral part of the hypothalamus, (1) either directly by cortico-hypothalamic

fibers (Mettler) or (2) by two neuron arcs (cortico-thalamic and thalamo-hypothalamic tracts) with synapses in the dorsal thalamus.

A. From the caudo-ventral preoptic and anterior hypothalamic transition area (and from the hypothalamic area farther caudalward in the region of the dorso-medial hypothalamic nucleus, in our own material) fibers are contributed to the dorsal longitudinal fasciculus. These fibers run in the periventricular area from the hypothalamic region caudalward to the brain stem, so that they are involved in lesions affecting the central gray. They terminate, among other regions, in the motor nucleus of VII, in the dorsal efferent nucleus of the vagus (not illustrated), in nucleus ambiguus and in the medial reticular gray of midbrain, pons and medulla. They end within the respiratory center (medial reticular gray) of the medulla and it is altogether probable that they reach the pneumotaxic center in the pons.

In the respiratory center of the medulla they come into relationship with the descending pathways of the ventrolateral reticulo-spinal tracts which distribute to the neurons supplying the diaphragm and the other muscles concerned with inspiration. Ordinarily the expiratory center serves to inhibit the inspiratory center periodically. However, in hysterical coughing or in excitement, expirations may be forced; then those connections of the expiratory centers through the ventrolateral reticulo-spinal tract with the motor neurons supplying the muscles of expiration (abdominal muscles and internal intercostals, etc.) and the fascicles passing to nucleus ambiguus (through which are innervated the abductor and the posterior cricothyroid muscles of the larynx) are important in expiration.

Stimulation of (1) the preoptico-hypothalamic transition area, or (2) the periventricular region of the midbrain through which the dorsal longitudinal fasciculus runs, produces an increase in respiratory rate and amplitude.

B. Other descending paths from the hypothalamus are concerned in increasing the respiratory tract. From the ventral part of the preoptic area and the anterior and dorsal regions of the hypothalamus, preoptico-hypothalamo-tegmental fascicles (preoptico-hypothal-tegmento-reticular tract) pass through the lateral hypothalamic area into the tegmentum of the midbrain near the region of emergence of the oculomotor roots.

With or without a synapse in this region the system proceeds caudalward (probably as a chain of neurons) to end in the motor nucleus of VII and the medial reticular gray of the medulla, within which is the medullar respiratory center. This is a part of the temperature regulating path.

It is probable that these fascicles also send fibers to the pontine pneumotaxic center. The pathways from these various areas by which inspiration is increased are comparable to those just described.

II. The neuron path from cortex to centers in medulla and cord inhibitory over respiration.

(1) Cortico-preoptic fibers from the postorbital gyrus to the dorsal part of the preoptic area—(2) Then preoptico-tegmental tract from the dorsal preoptic area to midbrain tegmental gray in the region lateral to the oculomotor nucleus (There may be only partial synapse in this region). (3) From tegmental gray as a neuron chain to the pneumotaxic center of the pons and the expiratory field in the respiratory center of the medulla-inhibiting inspiration and producing long, drawn out expiration. (This is a tegmento-reticular tract). (4) From the pneumotaxic center of the pons to the expiratory and inspiratory field of the medulla, also a tegmento-reticular system. (5) From the expiratory field of the medulla oblongata to the inspiratory field inhibiting inspirations and so slowing respiration.

Stimulation of this neuron chain (1) in the postorbital area (see recent work of McCulloch and his colleagues) or (2) in the preoptic area (Ransom, Magoun and Kabat), or (3) in the tegmentum of the midbrain (various observers and our own experiments) results in a slowing of respiration. Recent removal of the postorbital region in the monkey in our own laboratory resulted in respiration in which the inspiratory phase was greatly prolonged and was followed by a short expiration accompanied by a sound, something like a hiccough.

There is a huge body of anatomical and experimental data dealing with respirations. No attempt will be made here to review the literature since a wealth of information is readily available in current physiologic texts. Only certain modifications of respiration which come to the attention of the laryngologists have been selected for discussion such as hiccoughing, sneezing, coughing, or conditions of dyspnea, apnea, or hyper-apnea. It is of course well known that these respiratory manifesta-

tions may be due either to some pathological change in the nose, nasopharynx, pharynx, larynx and the nerves innervating these areas or primarily to an involvement of the central nervous system. It is with the latter that I wish to give particular emphasis.

Experimentally we are able to trace well established neuron paths from the cerebral cortex to the centers in the medulla and cord which influence the rate of respiration. In the early stages of meningitis, and particularly frontal lobe abscess we frequently observe slow sighing respirations. The rate may be decreased to 8 or 10 a minute. This has been attributed to an irritation of the vagus nerves which inhibit respiration and the pathological change has been assumed to be a purulent exudate on the brain stem in the region where the vagus nerves make their exit from the medulla. It has been stated also that increased intracranial pressure was occasionally the cause of irritation and that this likewise was exerted in and about the brain stem.

Autopsy findings in some of our cases of meningitis, particularly those arising from nasal accessory sinus disease were not consistent with this theory. It was frequently observed that respirations were slowed appreciably before there was evidence of increased intracranial pressure. Moreover, postmortem studies often revealed the presence of a thick exudate on the inferior surface of the cerebral hemispheres with little or no pathological change about the medulla and pons. This was invariably a disillusionment to me when during the course of the disease we had ascribed the respiratory changes (first slow due to vagi stimulation and then rapid due to paralysis of the nerves) to inflammatory changes in the posterior cranial fossa.

Our recent studies on the brain of the monkey bring a fresh point of view and a new approach to the cause of respiratory changes in meningitis, secondary to nasal accessory sinus disease. A more rational explanation for an early decrease in respiratory rate can be found in an irritation of the postorbital gyrus. This structure is superficially placed under the inferior surface of the cerebral hemispheres and is accessible to an invasion of the meninges and brain from ethmoidal and sphenoidal sinus infections. It is the seat of a round cell infiltration in meningitis in this region and when irritated causes a definite slowing of the respiratory rate. By the same token, stimulation of the preoptico-hypothalamic transition area, a region slightly caudalward to the postorbital gyrus,

produces an increase in respiratory rate and amplitude, a change usually observed in the terminal stages of meningitis or brain abscess. This structure is also superficially placed and accessible to an inflammation of the meninges and brain which has become extensive and deep-seated. The implications therefore are quite obvious. These are the regions capable of governing the rate of respiration and because of their vulnerability to intracranial infections from the nasal accessory sinuses are in all probability responsible for the respiratory phenomena observed in meningitis and brain abscess arising from these sources. This explanation is more conformable to rationalistic principles than that which attributes the changes in the rate of respiration to a direct attack upon the vagus nerves at their exit from the brain stem and it must be added that when this condition prevails we may observe most of the classical symptoms of meningitis except that sign upon which we usually place much reliance, namely the stiff neck.

Hiccough.—An example of a respiratory variation which may be due to peripheral or central lesions is the well known hiccough. The hiccough is an inspiratory reflex of clonic type induced by sudden contraction of the diaphragm with the glottis partly or wholly closed. The sensory side of the arc in hiccoughs may be provided by the sensory fibers in the phrenic, the afferent fibers of the vagus or by other sensory neurons. In the diagram (Fig. A) the sensory fibers of the vagus and phrenic are shown. The sensory fibers of the vagus terminate in the nuclear gray of fasciculus solitarius. From this nuclear gray intercolated neurons project the impulses on the inspiratory field of the respiratory center. From this field, neurons send their neuraxes to the cervical levels of the cord (C III to C VI) where they terminate around the motor neurons which provide the efferent fibers of the phrenic nerve. The descending neuraxes from the respiratory field to the spinal cord constitute the ventrolateral reticulo-spinal tract. The sensory fibers which come in over the phrenic nerve send neuraxes into the ventral horn region to terminate directly around the neurons giving rise to the phrenic nerve.

This reflex may persist for only a short time (as in the intoxicated person) and have no clinical significance. It may be set up by peripheral stimulation of the sensory side of the arc as in aortic aneurism or pleurisy of certain types, and may be sufficiently severe and long standing to

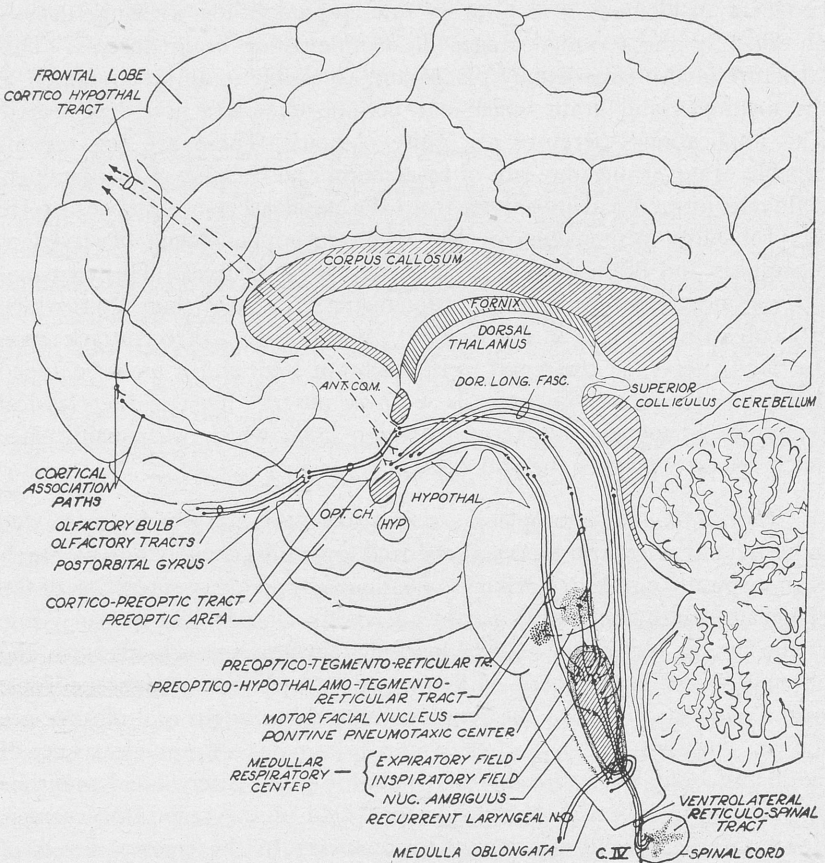


Figure A

warrant freezing, crushing, or section of the phrenic nerve. It may appear in tabes dorsalis where the dorsal root ganglia are irritated at cervical levels. In this case it will appear as a transitory symptom due to irritation of the ventral horn neurons of the cord at the level of the fourth cervical vertebra. This phase is usually followed rather promptly by a degeneration of the neurons supplying phrenic fibers to the diaphragm with a resultant paralysis of the muscle fibers on the side of the involvement. It is to be noted that occasionally unilateral paralysis of the diaphragm causes difficulty in breathing (sometimes with recurrent attacks of cyanosis). Persistent hiccoughs are present in some cases of

lethargic encephalitis, indeed are said at times to be the only symptoms of this condition and are thought to be the result of a myoclonus of the diaphragmatic muscles. They are observed occasionally in an epidemic form (epidemic singultus) in which the pathological changes do not appear to be known. They may appear also in certain psychogenic states and a recognition of this fact is exceedingly important. There are definite central nerve pathways for the transmission of emotional impulses and I shall deal more specifically with them in considering the mechanisms of coughing. The point I wish to emphasize is that underlying so simple a modification of respiration as hiccoughs there may be a wide range of pathologic conditions.

Coughing.—Coughing provides another illustration of a behavior pattern which may be due to peripheral irritation of some part of the

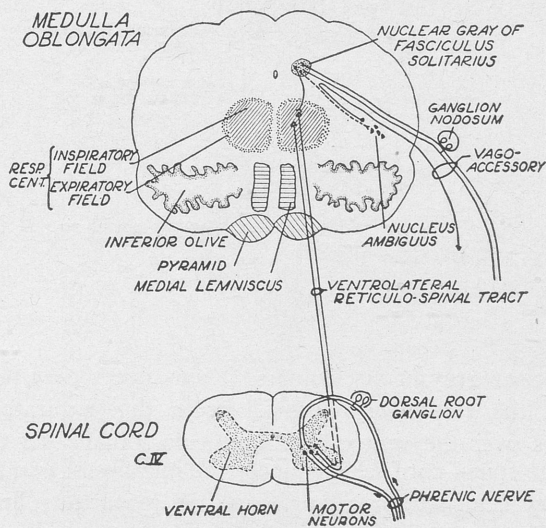


Figure B

respiratory mechanism or to involvement of the central nervous system. Coughing is an *expiratory* reflex frequently associated with difficulty in breathing. The reflex pattern is illustrated in Fig B. The sensory fibers of the vagus nerve may be stimulated by irritating particles in the larynx. Such sensory fibers have their cells of origin in the nodose ganglion and their neuraxes terminate in the nucleus gray of fasciculus solitarius.

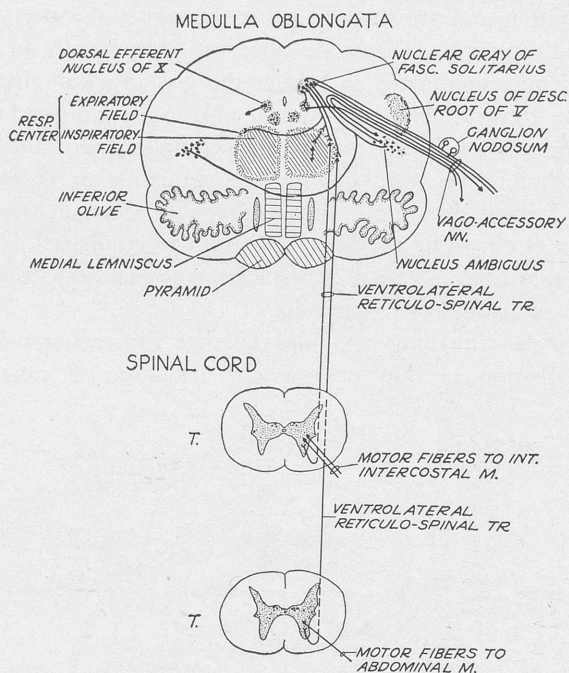


Figure C

From the nuclear gray of fasciculus solitarius fibers pass to the expiratory field of the respiratory center. From this expiratory field the impulses pass over the ventro-lateral reticulo-spinal tract to the motor neurons of the spinal cord which supply the muscles of expiration. From this expiratory field neurons also pass to the inspiratory field to inhibit inspiration. From the nuclear gray to the fasciculus solitarius crossed and uncrossed fibers pass to the nucleus ambiguus of the same and the opposite side. This nucleus provides fibers of the bulbar accessory nerve. Through this nerve abduction occurs in the larynx and the way is open for expiration. If the particle should have been drawn beyond the larynx into the trachea or bronchi contraction of the smooth muscles of the bronchi may aid in expelling it. This smooth muscle is innervated by a two-neuron chain with the preganglionic neuron in the dorsal efferent nucleus of the vagus. Such preganglionic neurons are indicated in Fig. C.

They synapse with postganglionic neurons at the hilus of the lungs which terminate in the circular muscle of the bronchi.

On the efferent side of the arc coughing may result from pressure on the recurrent laryngeal nerve as in aortic aneurism, from irritation of sensory ganglion cells, as in tabes dorsalis or from irritating lesions in the nose and throat, or in children, in the mouth as an accompaniment of dentition. Involvement of efferent fibers of the recurrent or superior laryngeal nerve may likewise produce coughing or interference with breathing.

Pertinent to this subject and worthy of more sympathetic understanding perhaps than has been accorded it, is that type of reflex cough-

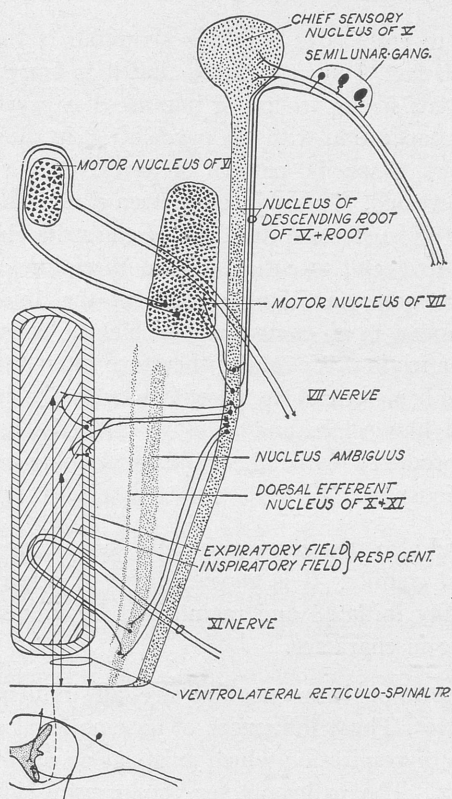


Figure 1

ing which must be attributed to etiological factors of psychogenic origin. It should be recognized that there is a definite central mechanism (outline in Fig. I) for the transmission of emotional impulses.

We have all had the experience many times of consulting with the mother who tells us that her child has a chronic cough. He coughs day and night. He may sleep for a time when first put to bed but awakens after midnight much to the distress of the parents and coughs more or less for the remainder of the early morning hours. In the vast majority of these cases we look in vain for an organic etiological factor responsible for this symptom. There are no abnormalities in the larynx, the pharynx presents no pathological changes, the cough cannot be attributed to anything within the nose and radiographs of the chest are entirely negative.

I should like to emphasize that this symptom is frequently on an emotional basis. In fact, I am convinced that it is more often than not an expression of some strong feeling or impulse of a psychogenic nature. Working in close cooperation with the psychiatrist in the care of a number of these patients, a specific pattern of parent-child relationship has been frequently revealed. There is an extreme dependence upon the mother. A feeling of frustration develops because the child is receiving too little paternal love and attention. Rigid discipline, repression and the enforcement of vigorous rules of conduct rob the child of his security and leave him stranded in an environment which he considers devoid of parental love and affection. Coughing therefore often fulfills the child's purpose, namely, to bring one of the parents, usually the mother to dispell his fears and give him solace and comfort. But the cough is seldom voluntary. It is produced while the child is sleeping or at least in a state of semiconsciousness.

I am convinced also that some asthmatic patients are the victims of a similar emotional conflict. It is another form of respiratory variation which is occasionally initiated and maintained by influences which are largely psychogenic in character.

Whether we realize it or not we are all influenced by factors extrinsic to ourselves. These influences of an emotional nature may act upon a mechanism of respiration which exists as an anatomical entity in the cerebral cortex. The pathways for the transmission of emotional impulses can be clearly defined and are capable of giving rise to a great

variety of respiratory changes among which we may find the cough of the insecure child or the emotional release of the asthmatic.

Sneezing.—Sneezing is a modified respiratory reflex in which a *long inspiration* is followed by a short sharp expiration. The reflex pattern is indicated in Fig. C. Irritating substances may set up nervous impulses in the peripheral fibers of the V nerve. These nerve fibers have their cells of origin in the semilunar ganglion and the neuraxes terminate in the chief sensory nucleus of V (for tactile, especially discriminating tactile) and in the nucleus of the descending root of V for pain, temperature and general tactile. From this nucleus connections are made with the motor nucleus of the VII nerve, with the respiratory fields and with the nucleus ambiguus. The connections with the motor nucleus of VII from which motor fibers pass to the muscles of the nose complete the reflex arc, the sensory side of which is provided by the root fibers of V and the motor fibers by the root of VII. If any part of this reflex arc is destroyed (as for example, the peripheral fibers of the trigeminal nerve) the reflex arc will not function and the nasal openings will not widen with the passage of air into the nostrils.

The nucleus of the descending root of V is connected with the inspiratory portion of the respiratory center. From this inspiratory field connections are made through the ventral lateral reticulo-spinal tract to the motor centers of the cord which innervate inspiration. Thus the long inspiration is produced which precedes sneezing. The muscles of expiration are stretched as the muscles of inspiration contract. From these stretched muscles sensory impulses are relayed through a series of neuron arcs to the expiratory field of the medulla. The connections from the expiratory to the inspiratory field serve as a pathway for inhibition of the inspiration. Where the expiration is violent as in sneezing, connections are made from the expiratory field through the motor centers of the cord innervating muscles of expiration. This connection is made through the ventral lateral reticulo-spinal tract.

A phenomenon in this connection which has excited my interest and curiosity for a number of years is that observed in the individual who has experienced a successful submucous resection of the septum for the relief of nasal obstruction but continues to complain bitterly of an inability to breathe through the nose. The nasal cavities are wide open. There is a large volume of air traversing the nasal chambers with each

respiration but the patient vigorously defends his contention that he still can't breathe adequately.

We might conclude and I suspect properly in some cases that this is an hysterical manifestation. Yet I doubt seriously that this explanation will suffice to explain the factors which give rise to this complaint in the majority of patients. There is perhaps a more rational reason in an evaluation of organic factors which have altered the respiratory reflex arc described for the act of sneezing. For example, it has been demonstrated frequently in our laboratory of physiology that one may regulate the rate of respirations within certain limits by stimulation of the nasal mucous membrane. The respiratory rate may be altered within a range of from 16 to 24 respirations per minute in a tracheotomized dog by blowing air from a bellows through the nasal cavities. The experiment clearly indicates that one may bring an extraneous influence into play which governs the rate of respiration to a limited extent.

Scientists familiar with this field tell us therefore that something has altered the reflex respiratory arc in those patients with wide open noses who complain of impaired breathing after the submucous resection of the septum—that we have injured or in some manner altered the sensory side of the arc provided by the fibers of the trigeminal nerve. Although the nerve supply to the nasal mucosa is widespread and a considerable part of it outside the field of the submucous operation, there may occur nevertheless sufficient damage to the sensory distribution to alter the pathways which furnish the impulses of respiration. A number of my patients during the past thirty years have complained of a loss of the sense of smell after the submucous resection of the septum. While this has usually been temporary there have been instances when the sense of smell remained permanently impaired. In several cases it has seemed plausible to explain this disturbance to injury of the middle turbinate or the mucous membrane of the septum adjacent to this structure; there have been patient in whom adhesions between the middle turbinate and septum were obviously the offending factor but in the vast majority of my patients the mucous membrane of the nose appeared normal and entirely devoid of any pathological change which would imply damage to the end organs or fibers of the olfactory nerves.

If it is possible to impair the sense of smell without subsequent evidence of any gross pathological changes in the mucous membrane of

the nose, is it not therefore feasible to expect that the submucous resection of the septum might do similar damage to the end organs of the trigeminal distribution or some of its important fibers. The point to be emphasized is that in some patients who complain of difficult breathing in the presence of wide open nasal cavities after the submucous resection of the septum there is perhaps a definite organic basis for this phenomenon arising from a disturbance in the sensory innervations of the nasal mucous membrane. Perhaps too in some cases the olfactory sense plays a part because we know that it is directly connected with the central emotional system which exerts an important influence upon the respiratory centers in the brain stem. As further proof of this theory which ascribes this phenomenon to some change in the nasal-respiratory reflex arc is the fact that these patients usually recover spontaneously when sufficient time has elapsed to permit regeneration of the fibers of the trigeminal nerves.

CONCLUSIONS

As the title indicates an effort has been made to give due consideration to the peripheral and central pathways over which the impulses of respiration are transmitted. Special emphasis is given the central tracts in describing certain common respiratory variations.

2. The absence of pathological changes about the brain stem in individuals who had died of meningitis gave rise to doubt that direct stimulation of the vagus nerves and later paralysis of them were responsible for the respective slowing and acceleration of respiration. The inquiry into a more rational explanation for these changes arose particularly from autopsy findings in meningitis, secondary to nasal accessory sinus disease where the exudate was largely confined to the surfaces of the brain. Laboratory experimentation on monkeys demonstrates well established areas in the cerebral cortex which when stimulated either slow or accelerate respiration. Autopsy material obtained from patients who died of the type of meningitis above described demonstrated inflammatory involvement of these regions.

3. While the text probes only superficially into the emotional factors responsible for respiratory variations such as hiccough, sneezing, coughing, and asthmatic breathing, it treats specifically of the central nervous apparatus along which emotional impulses are carried. A

knowledge of these pathways and a better understanding of human relationships may widen our knowledge of the psychology of cough and asthma.

4. In discussing the reflex arc of sneezing, an effort is made to offer an explanation of an organic basis for the occasional complaint of nasal obstruction after a submucous resection of the septum in a patient who has wide open nasal cavities. Although the picture is complicated a disturbance of the pathways of the reflex arc is described which may properly explain this phenomenon.

5. And finally this initial inquiry into the nervous mechanism of respiration and its common variations leads one to the conviction that further important information may be obtained from a more thorough survey of this subject. The one generalization that may be derived from this discussion is that the whole mechanism of respiration is a complicated one governed by a reflex arc and an equally important central nervous system component susceptible to emotional stimulation. In looking for the cause of respiratory changes we must remember not to confine our efforts to an investigation of organs supplied by the peripheral nerves. A knowledge of the central nervous system mechanism and its connections with the peripheral nerve distributions may perhaps help us as laryngologists to think rather than feel our way toward a better understanding of respiratory abnormalities.

DISCUSSION

NORTON CANFIELD, M.D.: Mr. President, I enjoyed this presentation extremely because it may explain two cases which I have had under observation during this past year, about which I would like to ask Dr. Furstenberg. These children were both under one year of age when first seen. They came into the hospital with signs and symptoms of respiratory croup associated with laryngeal infection. Both of these cases required prompt tracheotomy, and the course of the infection was normal as with other cases of croup. However, the tracheotomy tubes in these cases could not be permanently removed because of laryngeal spasm. Respiration under those circumstances with the tube out was labored and it required replacement. The condition in one patient continued for a period of more than six months before the laryngeal spasm subsided enough to permit removal of the tube. The first child was tracheotomized at the age of three months and the second at the age of two months. Inspection of the larynx in these cases, after the acute infection had subsided, showed it to be perfectly normal except

for the spasm when the tube was out. When the tube was replaced the larynx would then function normally and the patient could breathe. Under deep anesthesia in one case the same phenomenon occurred, that is, the larynx would go into spasm when the tube was removed but the spasm would be relieved when the tracheotomy tube was replaced.

During the last four months in the case of the first child, the child breathed perfectly through the larynx with the tracheotomy tube plugged and would do so for many weeks at a time. Immediately the tube was removed the larynx would then go into spasm.

With the second child we had the tube plugged for approximately one month, so that the tube does not function as such, but the larynx goes into spasm as soon as the tube is removed.

I would like to ask Dr. Furstenberg, therefore, if the neurological work which he has done can explain this reflex path, if it is such, and if so, if he has any suggestions for interrupting this reflex path so that we can remove these tracheotomy tubes at an earlier date.

MARVIN F. JONES, M.D.: Gentlemen, this presentation of Dr. Furstenberg is like welcoming an old sweetheart to me. Some of the older men of the society may recall my early and continued interest in similar subjects.

There is only one phase of his presentation that I have even an opportunity or time to discuss and that is the sensation of breathing and the break of the reflex arc that produces that sensation.

It is not only following the septum operation that such imagined difficulty in breathing occurs but it is also in certain cases where, to speak vaguely, you could drive a horse through the nose without touching the side, there being atrophy of tissue present. These patients will breathe through their mouth and in spite of the fact that they have ample room will tell you that they are unable to breathe through their nose. You can also substantiate that fact by holding the ordinary head mirror under the nose and asking them to breathe through the nose and then you will see an equal distribution of the clouding on the mirror. The break in the reflex arc that Dr. Furstenberg described should be classed probably as a traumatic break. In this I believe there has been a degenerated break in the end organ.

We find in these patients the history that they never have had colds recently but that they used to have frequent colds. It has been a guess on my part that these patients have early been affected by a vasomotor rhinitis and the subsequent changes have caused the pathology whereby their normal secretions and their normal sensations are not present. You can test that out by putting an applicator in the nose and they have less reflex sensations than the normal individual. It also happens that the membrane in these patients is thin, and if you have x-rays taken of the sinuses you find this also extends to the sinus-like

membranes. Your x-rays will come back darker than normal, more penetration than normal and very often by x-ray men will be called negative. These membranes also act to cause reservoirs in the sinuses and do not eliminate themselves. In other words, it is a non-functioning membrane and a non-functioning end organ that causes the lack of sensation, producing the reflex which they understand as breathing. Therefore, they say they are unable to breathe.

I submit that as possible substantiation for the proposal of Dr. Furstenberg.

CHEVALIER L. JACKSON, M.D.: Not during the reading of the paper, but during the discussion, I have been reminded of a patient (who had had a laryngectomy done by someone else) who experienced great difficulty in breathing as his only complaint. He seemed to be mentally and physically well enough adjusted to the condition of being without a larynx. Incidentally, this man was a doctor of medicine. After spending several months in Florida and trying to follow the advice of his physicians, he still suffered great distress, feeling that he could not get his breath. We had careful x-ray examinations carried out, and arranged consultations with the cardiologist and the internist, but all studies were negative. We found nothing organic to account for this sensation of difficulty in breathing.

I wonder if this patient was another example of either psychosomatic or reflex origin of symptoms.

JAMES M. ROBB, M.D.: Dr. Furstenberg always pleases me with his presentation. His slides are good and his description accurate. I was interested in the tearing element which comes in association with sneezing. As you know, the parasympathetic element of the tear sac arises from the nervus intermedius and passes over to the Gasserian ganglion and then over the second division of the fifth, and from here over the zygomatica-temporal branch to the lachrymal gland. This could be another nerve distribution of clinical significance in laryngology.

H. I. LILLIE, M.D.: As usual, Dr. Furstenberg has presented something that makes us think.

I should like to ask him to explain how, in sneezing bouts, pressure on the upper lip many times prevents the sneeze.

I have observed that some patients, who have had zinc ionization done, complained of not being able to breathe even when the nose showed no objective evidence of obstruction. Apparently, complete atrophy of the mucous membrane and loss of function existed, but still these patients complained that they were unable to breathe.

It occurred to me, as this paper was being presented that atrophy and loss of sensation of adequate breathing within the nose might have resulted from permanent anesthetization of the mucous membrane as the result of local anesthesia. I don't know whether that would be possible or not, but in view of the fact

that it has occurred in patients in whom the nerves have been destroyed by zinc ionization, it might be a possibility.

LOUIS H. CLERF, M.D.: I should like to know what the mechanism is that causes sneezing when going, for example, from this dimly lighted room into the bright sunlight when I often will sneeze several times.

PRESIDENT PROETZ: Gentlemen, to pile Pelion on Ossa or vice versa, I have a doctor-patient-friend whom I can make sneeze by touching the hairline on one side. There is one small area, less than the size of a thumb nail, that will set off the sneezing. I have reported this at one time. May I include that area in connection with this question?

Dr. Furstenberg, will you please take on this coterie of critics?

A. C. FURSTENBERG, M.D. (Closing): I must say, gentlemen, that I am overwhelmed by all of these questions. I am not capable of answering them, although perhaps further study will give us the answers at some future time.

Dr. Canfield and Dr. Jackson have described some interesting cases. Perhaps we have a definite explanation for the occurrence of laryngeal spasm as a result of irritation of the mucous membrane of the trachea, especially the mucous membrane close to the larynx. There is, of course, a definite reflex arc through the recurrent laryngeal nerves, to the medulla. Thus it should be possible for some irritation in the trachea to produce a spasm of the larynx.

Dr. Jones has been good enough to verify the observation, that after a successful, or what appears to be a successful submucous resection of the septum, the patient may occasionally complain of difficulty in breathing. In some cases perhaps this is the result of a degeneration of the end organs of the trigeminal nerve. Enough disturbance may occur in the end organs to cut off the reflex arc and give the patient the sensation of difficulty in breathing. I am rather inclined to think that this is a temporary situation because many of these patients come back several months later completely relieved from this symptom. Possibly enough time has elapsed to permit regeneration of the end organs of the trigeminal nerves.

Dr. Lillie wants to know why pressure on the upper lip may stop sneezing. Here again I think it may be possible to stimulate the reflex arc by pain sufficiently to inhibit inspiration and thus terminate the attack of sneezing. By expirations sufficient stimulation of the pathways to the excitatory center would inhibit the inspiratory center and possibly stop your attack of sneezing.

THE ETIOLOGY, TREATMENT AND PREVENTION OF CHRONIC SINUS INFECTION*

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The ultimate object of all researches on the common cold has been to find a drug, a vaccine or some means to protect against repeated infection with the common cold virus. All such studies so far have been a failure.¹ It has been proved that the common cold is due to a virus, which is an anaerobe that grows and produces clinical symptoms only after it has entered a living cell. Also that the virus must be in contact with a cell for at least 30 minutes before it can gain entrance to the cell and begin to multiply and produce clinical symptoms. This suggests that the common cold virus, which is an intracellular parasite, does not possess all the enzyme systems essential for growth and reproduction, but is dependent on the host cells for these necessary enzymes. It also offers a possible explanation for the fact that viral diseases do not respond like bacterial infections to known chemotherapeutic agents and antibiotics. Most bacteria and spirochetes possess all the enzyme systems essential for growth and reproduction, and their destruction by these therapeutic agents is believed to be due to interference with their enzyme systems or metabolic processes.²

Pyogenic bacteria are present in the nasopharynx from infancy to old age. Their first appearance in infants soon after birth often is not associated with an inflammatory reaction, or any evidence of infection. There is a growing impression that these pyogenic bacteria, the streptococcus, staphylococcus, pneumococcus and *H. influenzae*, are in themselves powerless to initiate infection.³ The carrier rate of these organisms varies from time to time and year to year. When cultures show widespread distribution of pathogenic organisms in a community, acute upper respiratory infections are usually severe, and ear, sinus and lung complications are common. On the other hand during the summer and

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early fall, when cultures usually show the distribution of pathogenic organisms is relatively limited, the majority of acute upper respiratory infections are mild, complications much less frequent and the usual diagnosis is the common cold. Repeated infections with the common cold virus seem to facilitate the distribution and virulence of bacteria in the nasopharynx, and prepare the soil for widespread outbreaks of respiratory disease in the community.

In a sense the ear, sinus and lung complications of upper respiratory infections are secondary to the chain of events set in motion by the common cold virus. This virus, being airborne, enters the nose, is carried to the nasopharynx by the mucus and cilia of the nasal mucous membrane, finds a lodging place in the crypts and folds of lymphoid tissue, penetrates the lining epithelial cells, breaking down cell membranes and protective barriers, and thus providing a portal of entry for the bacteria that are always present in this location. A cellulitis ensues, the number and virulence of the bacteria increases, the resulting edema causes obstruction in the nasal passages or eustachian tubes that interferes with drainage, and an acute sinusitis or otitis media results. If the outlet of any air-containing cavity becomes occluded by edema or a fibrin plug the air within the cavity is soon absorbed, and as the air pressure within the cavity becomes less than the atmospheric pressure on the surface of the body, the blood and lymph vessels in the cavity become increasingly engorged and often rupture. This adds to the obstruction of the only outlet through which drainage can occur. When the eustachian tubes are thus occluded, the resulting changes in the middle ear are called aerotitis; when a sinus orifice is occluded an aerosinusitis ensues, and when a terminal bronchus is occluded it is reasonable to assume an aeropneumonitis follows. If bacteria happen to be present in the area thus shut off from the outside world, an abscess is formed. This is probably the genesis of bronchiectasis. In the ear and sinuses it is possible to establish drainage by surgical measures. In the lung the only way to establish drainage is to visualize the area through a bronchoscope and with the aid of vasoconstrictors applied locally and point suction, remove the obstructing plug. By employing these mechanical measures to insure drainage early in the course of the infection, before extensive cellular destruction and granulations have formed, and at the same time giving adequate doses of sulfonamides or antibiotics, it is possible to

prevent chronic ear and sinus infections, and it should be possible to prevent chronic suppurative bronchiectasis.

Lymphoid tissue throughout the body, and especially that in the upper air passages, is extremely resistant to infection with the common pathogenic organisms. The chief function of lymphoid tissue in the upper air passages is to filter out bacteria and manufacture antibodies. It protects the body against bacterial invasion, but affords no protection against viral infection. Histologic examination of infected tonsils and adenoids, removed at operation, rarely shows an abscess in lymphoid tissue, but we always find a localized or widespread destruction of epithelium lining the crypts. The portal of entry for the bacterial infection is probably prepared by a preceding viral infection, just as a mechanical or chemical injury of skin epithelium makes conditions favorable for infection by the bacteria that happen to be in that locality, and a cellulitis ensues.

The discovery of a vaccine or drug that would protect against viral infections would be ideal, but until that day comes the otolaryngologist must use all the measures at his command to prevent the common cold and its complications. It often happens that marked improvement in the frequency of upper respiratory infections and their complications follows operative removal of tonsils and adenoids, especially in children. In many of these children however, the relative freedom from colds and ear or sinus infection lasts for only a few months or a year after the operation. In these cases examination usually shows recurrence of lymphoid tissue which again affords lodging places for the virus. It is not the size, but the location and contour of these lymphoid nodules that is important. Nasopharyngoscopic examination on a large group of patients in whom the operation has been a failure, showed nodules of lymphoid tissue in and around the orifice of the eustachian tubes, in the choanae near the posterior end of middle turbinates and on the posterior and lateral surface of the vomer—all locations inaccessible to operative removal. Proof that these nodules are often the cause of recurrent symptoms is afforded by histologic demonstration that they have crypts, and by the marked clinical improvement that follows their treatment with a radium applicator. The action of irradiation, in the dosage used, is to stop mitosis in the cells, which have absorbed the rays. After adequate irradiation no new lymphocytes are formed to replace those lost by the normal dissolution process. The nodule of lymphoid tissue gradually shrinks, the crypts

become flattened out or obliterated, and no favorable places remain in the nasopharynx in which the virus can lodge and remain undisturbed by the normal flow of mucus for a long enough time to allow it to enter cells.

We do not mean to suggest that the nasopharynx is the only portal of entry of the common cold virus, but certainly the first symptoms after experimental inoculation of the nasal passages of chimpanzees or human volunteers with the common cold virus is hyperaemia and irritation in the nasopharynx, not in the nose or pharynx. These findings suggest that it is possible for the otolaryngologist to do more to prevent recurring viral infections and their complications than either the pediatrician or internist, whose therapy is limited to vitamins, nasal drops and antibacterial drugs. These measures usually lead to recovery of the acute condition, but in no way protect against a recurrence of a virus infection. The mucous membrane of the ears and accessory nasal sinuses has a remarkable ability to recover after a single or an occasional infection. It is the oft-repeated, acute bacterial infections that eventually lead to chronic suppuration, and necessitates radical surgical operations. These repeated acute bacterial infections often are initiated by a preceding viral infection in the upper air passages. For this reason it is important that the otolaryngologist should do everything he can to lessen the frequency of colds and to prevent recurrence of pyogenic complications. The only known way to accomplish this, even in part, is to examine the nasal passages and nasopharynx after the acute infection has subsided and, if hyperplastic or chronically infected nodules of lymphoid tissue are seen in the nasopharynx, in the posterior part of the nose or around the eustachian tubes, remove them surgically, or at least change their contour with surgical operation, irradiation, or both.

It is not possible to remove all lymphoid tissue in the nasopharynx at surgical operation, and not safe to give the heavy doses of irradiation that would be necessary to remove permanently all lymphoid tissue in the nasopharynx. Years of experience have shown that small doses are effective in reducing the size and contour of lymphoid nodules without injury to other cells. The safe way to use a nasopharyngeal applicator is to be conservative and satisfied to relieve symptoms as they arise and not try to obtain a permanent cure with one or more large doses. It is not possible always to cure a patient permanently with

one operation, such as removal of tonsils and adenoids, or with one course of antibiotics. The same reasoning holds true for irradiation. Irradiation is a therapeutic measure that is of value in selected cases when used conservatively and safely in conjunction with surgery, antibiotics or other measures.

Our discussion of acute sinus infection will be limited to treatment of the frontal sinuses. The naso-frontal duct is small and subject to anatomical variations that often make it difficult to catheterize and irrigate the frontal sinuses. Drainage is essential in order to relieve pain and prevent complications, such as osteomyelitis, orbital abscess, brain abscess or meningitis. If, in the presence of an acute infection, the mucous membrane lining the naso-frontal duct is injured in trying to irrigate or start drainage by the nasal route, often the end result is granulations, scar tissue and a chronic sinusitis that necessitates surgical operations. As in acute mastoid infections we cannot always depend on the administration of antibiotics to cure an acute frontal sinus infection, unless there is also free drainage of pus. The obstruction to drainage in acute frontal sinusitis is not due solely to edema of the mucous membrane at the nasal end of the duct that may be relieved by local application of vasoconstrictor drugs and heat. From our experience in opening the frontal sinus through a small incision in the eyebrow in 88 cases of acute sinusitis, we have learned that the chief cause of obstruction is thick, fibrinous plugs of muco-purulent secretions on the sinus cavity. We must remember that the first changes in the sinus following obstruction of the duct are absorption of air in the cavity, congestion of blood vessels and edema of the lining mucous membrane—an aero-sinusitis. If bacteria are present, an abscess results. For prompt relief of pain, prevention of complications and avoidance of chronic sinusitis, free drainage is necessary and it should not be delayed.

For these reasons we have drained the frontal sinus through an external incision in all patients coming to the hospital with severe pain, tenderness on light percussion over the affected frontal sinus and changes on transillumination and x-ray. If the pain is very severe we promptly give penicillin intramuscularly and proceed immediately with the operation. In those with pain less severe we give both penicillin and sulfamerazine and use heat and vasoconstrictors locally. In 16 of 104 patients this conservative treatment was followed by recovery without external

drainage. In the remaining 88 patients the infected frontal sinus was opened through an incision about $\frac{1}{2}$ inch in length, which is made through the unshaven eyebrow just above the inner canthus of the eye. The patient is anesthetized with sodium pentothal and, with the eyes protected with a sterile, thin, flat, circular piece of cotton moistened with saline and covered with vaseline, the eyebrow and surrounding skin is washed with soap and water, then ether and zephiran. This method of preparation avoids the necessity for shaving the eyebrow, which when removed is very conspicuous and embarrassing to the patient for several weeks after he has recovered from the infection.

The incision through the skin and periosteum is just long enough to permit an opening $\frac{1}{8}$ inch in diameter to be made in the underlying bone. Great care is taken to avoid traumatizing the soft tissue and periosteum with retractors or clamps. The opening in the bone is made with a perforator and burr. The exposed mucous membrane of the frontal sinus often balloons out into the incision, due to the pressure of pus and gases in the sinus. The bulging mucous membrane is incised with a knife and the pus removed with a small suction tube, no larger than an eustachian catheter. The mucous membrane lining the sinus should not be removed or injured. No gauze or curettes are used in the sinus, and no effort is made to probe or enlarge the naso-frontal duct. If uninjured, the mucous membrane will soon become normal as it does in the nose or pharynx after an acute infection, but if scraped or wiped with gauze, granulations and scar tissue are likely to prevent complete recovery.

After the pus has been removed from the sinus with suction, the cavity is filled with penicillin (1000 units per cc. in saline), or with a tyrothricin suspension (1 cc. of the stock solution in 25 cc. of distilled water). A short piece of soft rubber tubing is put in the sinus to keep the skin edges apart. This is removed 24 hours later and through this opening the sinus is gently irrigated, usually with warm saline solution. No drain is replaced, since this procedure reinfects the cavity with skin organisms. To prevent closure of the skin incision, the edges are lightly painted with carbolic acid once a day, care being taken to prevent any of the acid from getting into the sinus. The sinus is irrigated with saline once a day, and usually after 3 or 4 days it begins to flow through the naso-frontal duct and out through the nose. The skin incision is then approximated and as a rule no further treatment of

any kind is necessary. We have employed this technic for several years and, if the cases are carefully selected, the results have been most satisfactory.⁴ We have had no complicating osteomyelitis or cellulitis; on the contrary the drainage prevents both acute complications and development of a chronic sinusitis. If the acute infection that brings the patient to the hospital is superimposed on a long-standing chronic infection, the external incision is used to obtain drainage, relieve pain and to identify the infecting organisms, but as a rule more extensive surgical procedures are necessary to cure the patient.

SUMMARY

Acute frontal sinusitis is a painful and potentially dangerous malady. To relieve pain, lessen the danger of osteomyelitis and other complications, we advocate external incision and drainage in selected cases. This is in addition to the administration of antibiotics. The chief advantage of external drainage is that it makes unnecessary intranasal instrumentation, which not only is very painful, but damages the acutely infected, edematous mucous membrane of the naso-frontal duct. The less the infected mucosa in this area is handled, the more likely it is to return to normal when the acute process subsides. Nothing should be done while trying to tide the patient over the acute period, that will damage the infected, edematous mucous membrane of the naso-frontal duct, since this may lead to the formation of granulations and scar tissue that permanently impairs drainage, and to the development of a chronic sinusitis. It is far easier to prevent a chronic sinus infection than it is to cure it.

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ASSOCIATED SYMPTOMATOLOGY OF DISEASES OF THE EPIPHARYNX

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Symptomatology in relation to the epipharynx has been a challenge to the acumen and thoroughness of the otolaryngologist since the days of Tornwaldt, when he first published his monograph "Bursa Pharyngea" in 1885. Despite earlier publications and many recent articles on the nasopharynx, the importance of the subject is apt to be more or less neglected. Too often in routine examination a critical survey of the epipharynx is omitted or considered of little importance in patients with postnasal discharge, recurrent pharyngitis, cough, headache, persistent fever, bleeding, hoarseness or asthma. In many instances the onset of recurring rhinitis (frequent colds) has its origin in the vault of the epipharynx (nasopharyngitis). The patient often states, "I have a burning sensation back of my palate." Deafness due to tubal insufficiency from lymphoid hyperplasia throughout the vault is to be included in this group. Much of the recent literature is directed mainly to the subject from the standpoint of diagnosis and treatment of tubal obstruction and various forms of hearing impairment by irradiation.

It is the purpose of this report to present a study of 150 cases observed over a period of two years, from the standpoint of a wide variety of symptoms and complaints, which have their source solely in the epipharynx. It must be borne in mind that this group of patients was studied carefully to eliminate all other factors which might otherwise produce the symptoms referred to, such as accessory sinus disease, hyperplastic turbinal changes, malignancy, or systemic disorders. For example, if a sinus conditions producing postnasal discharge was present, the case was not included in this study. Even though pathology of the epipharynx was found, it was assumed that this condition was secondary to disease in the nose and, therefore, not included in this analysis.

A number of patients, in whom evidence of an allergic disturbance was noted in either the history or upon examination, were not considered in this report. For a similar reason, cases complaining of that present-day, popular, and much abused term, "postnasal drip," where the discomfort and irritation is self-inflicted due to the habit of hawking or rasping, likewise are not included, since no gross pathology was present in the epipharynx. This is the same group where responses from such a symptom varied from that of apprehension or an esthetic annoyance to the extreme of psychologic upheaval.

The age of the patients studied in this group ranged from 5 to 75 years, with the greatest percentage occurring between the ages of 40 and 45, or 16.6 per cent. There were 58 males and 92 females. The most frequent complaint was postnasal discharge, thickened in consistency, and yellowish in color, sometimes foul in taste and occasionally noted as a mucoid, inspissated clump in the mesopharynx. The latter is often indicative of a true pharyngeal bursa. Next in order of frequency was the history of repeated sore throats or chronic pharyngitis in which cervical adenitis was sometimes present, accompanied by low grade temperature. The occasional sore throat which appears in the morning and subsides during the day is an example of the typical description given by the patient. Frequently, one symptom may be intimately related to another, occurring in association, but the immediate complaint is the one recorded in this classification. This is illustrated in patients in whom the cardinal complaint is postnasal discharge, while the associated history might include sore throat or cough or hoarseness.

Ten patients complained of frequent colds which seemed to have had their origin in the epipharynx, the history of onset being described as a burning sensation above the palate. There were nine cases of epipharyngitis with fever. Bleeding, in the form of bloody expectoration or cough, was observed in six cases. Three of these patients were suspected of having pulmonary tuberculosis and two of them had received sanitarium treatment, although no acid-fast bacilli had been discovered. One case of postnasal hemorrhage was severe enough to have previously required postnasal packing and one blood transfusion. The bleeding point was discovered by direct nasopharyngeal examination. Bleeding granulation tissue was found in the midline of the vault. Histologic study was negative for a neoplasm. This patient was treated by means of an electrocautery employed locally, supplemented with rutin and

ascorbic acid parenterally. In a recent re-check after a period of eight months, the nasal vault appears smooth and there has been no recurrence of the bleeding episode.

One patient, suspected of pulmonary tuberculosis because of blood-stained sputum, had a small, bloody area of granulation tissue in the vault. Bronchoscopy had been performed and all secretions were reported negative. She had a positive Rumpel-Leeds test, indicating increased capillary fragility, suggesting a probable vitamin C deficiency because she never had eaten citrus fruits. Silver nitrate (5%) applied locally was supplemented by large doses of ascorbic acid. She now has bloody sputum at infrequent intervals, and then only following a cold which apparently originates in the epipharynx.

In 16 patients in this series, cough was a major symptom—non-productive and paroxysmal in character—sometimes described as a “tickling sensation” and not relieved by the usual sedatives. Lymphoid hyperplasia with hyperemic and thickened lateral bands behind the posterior pillars, was the predominant finding in these cases. There was prompt relief following local treatment to this area. Hoarseness or laryngitis was present in 10 cases, all of them secondary to postnasal discharge. There were 10 cases with a history of frequent colds which apparently had their origin in the nasopharynx. It is my belief that many of our so-called head colds begin as a nasopharyngitis.

Epipharyngitis, referred to as a soreness back of the palate or a burning sensation in this area, and accompanied by slight fever, was observed in 9 cases. Asthma was relieved in one patient in whom all other forms of treatment, including allergenic therapy, had failed. A large, bulging, closed pharyngeal bursa, with a pinpoint opening allowing the escape of purulent secretion, was discovered. In a similar case of a closed bursa with a bulging vault, the patient complained of impaired nasal breathing. The sac contained a thickened, jelly-like mucus. Removal of the sac gave complete relief of symptoms in both instances.

Intermittent otalgia with fullness and hearing impairment was the primary complaint in 11 cases. With the exception of two adults, these were all children under 16 years of age. Tonsils and adenoids had been previously removed. The tympanic membrane picture was varied, but

gave evidence of serous exudate or inflammation. In a few of the cases myringotomy and inflation with recovery of fluid confirmed the ear findings. The presence of massive lymphoid tissue or adenoid growth in the children required surgery or radium, or both.

It is noteworthy that in eight of the reported cases, there was a history of persistent fever for a period of five months or more, and in one case, of 18 months' duration. The temperature ranged from 99 to 100 degrees. Three of these patients were given the benefit of surgery, while the others were treated conservatively. The systemic symptoms were constant fatigue, neuralgia or arthralgia, and sometimes a loss of weight was reported. The diagnosis was based upon the fact that no specific or local cause other than the epipharyngeal pathology could be found to account for the fever. All of the patients were studied exhaustively by an internist for the cause of obscure fever. It should be stated that following the first examination, which required slight manipulation of the infected epipharyngeal tissue, such as probing, a flareup of the temperature or exacerbation of the systemic symptoms sometimes occurred. Occasionally cervical adenitis was observed to have followed such a procedure. It is also significant that in this particular group the sedimentation rate and white cell count were only slightly elevated, if at all. Furthermore, sulfonamide and penicillin therapy had been administered without any influence upon the course of the temperature. Final diagnosis was definitely established when the temperature returned to normal and the symptoms disappeared following treatment of the infected epipharyngeal tissue. In this connection, I should like to emphasize the importance of a thorough study of the epipharynx in all individuals who have a low-grade fever of unknown origin.

One case of tuberculosis of the nasopharynx is reported, not as a symptom *per se* of the disease, however, because pulmonary involvement was a known factor. The so-called "postnasal drip" complaint led to the diagnosis of the primary source. This patient had a superficial ulcerating lesion, ovoid in shape, in the vault of the epipharynx, covered with a grayish exudate. Tubercle bacilli were recovered by smear examination. It is advisable, therefore, to bear in mind investigation of the epipharynx in cases of pulmonary tuberculosis. The healing effects of streptomycin in tuberculous lesions of the larynx and oropharynx have been dramatic. Its application to lesions of the nasopharynx have been reported equally as effective.

One patient with the cardinal complaint of occipital headaches is reported. In contrast to sphenoidal headaches, which are more generalized over the occiput, the pain is more localized. The diagnosis was based upon the presence of a deep median crypt and relief of the headaches which followed conservative treatment. In such instances there is believed to be an extension of the notochord remnant upward and backward against the basilar portion of the occipital bone. It may even penetrate the basilar fibrocartilage. Such extension is to be suspected in occipital headaches, as emphasized by Kully, who reported 12 similar cases in 1934.

Crusting was present in two cases. The mucous membrane was smooth and dry. A shallow bursa with a large opening was observed, with crusts adherent to the lower lip of the sac.

The presence of a bursa does not necessarily mean that it is pathologic. Its presence has been noted without clinical symptoms. To be pathologic the sac must become infected. This is apt to ensue from trauma, such as may occur in scraping of adenoid tissue. It is therefore possible to find a pharyngeal bursa after removal of adenoids. The incomplete removal or the presence of adenoid remnants is a frequent cause of lymphoid hyperplasia and subsequent formation of retention cysts or abscess or chronic inflammation. During the period of involution of adenoid tissue, cysts or abscesses may develop within any of the adenoid clefts, more often in the median recess. They may, therefore, be considered as congenital or acquired.

One should be constantly alert to the possibility of malignancy in the nasopharynx. There is none reported in this series. In one case, however, the first evidence of malignancy was discovered through biopsy of an enlarged cervical lymph node. This clue led to the nasopharyngeal examination from which diagnosis of anaplastic carcinoma was confirmed. These malignancies, occur most commonly between the ages of 40 and 60. The most frequent types encountered are carcinoma or lymphosarcoma. In 40 per cent of the cases reported by Jens Nielsen of Copenhagen, cervical gland enlargement was the first symptom observed.

Examination: Diseases of the nose and sinuses and allergy must be eliminated before all evidence can be placed against the nasopharynx

as a primary focus. The history alone is often indicative of the diagnosis. The sudden appearance of a mucoid clump into the mouth is the typical description given by the patient when a pouch is present. Inspection may be accomplished by use of the nasopharyngoscope, posterior rhinoscopy with the Michel mirror, by anterior rhinoscopy, or by direct nasopharyngoscopy. The last-named method is best accomplished by means of a palate retractor or the Yankauer speculum. This, to me, is the instrument of choice, and most serviceable because it provides a direct view of the vault and allows for instrumentation. Probing with a cotton-tipped applicator will reveal the depth and limitations of a pouch, as well as the overgrowth of lymphoid tissue. When the mere touch of an applicator provokes bleeding, it is evidence that inflammation or granulation tissue is present. In many instances the epipharynx is revealed by rhinoscopic inspection. When the turbinates are shrunk, and the septum is not obstructing, it is easy to observe the vault through one side of the nose, while probing this field through the other side with a small cotton-tipped applicator. For the study of neoplasms, choanal fibromas or polyps, the mirror gives adequate exposure. For lesions around the eustachian tube orifice and in and about Rosenmüller's fossa, the nasopharyngoscope is the most serviceable.

The bacteriologic study of the epipharynx was not particularly informative except in cases of persistent fever. The predominant organisms recovered by culture were non-hemolytic streptococcus, streptococcus viridans, hemolytic staphylococcus aureus, and diphtheroids. In the patients with fever, however, hemolytic streptococcus was the predominant organism, while in persons with closed bursas, cultures often proved sterile.

In this series of cases, diagnosed and treated, examination of the postnasal space revealed a wide variety of findings. The pharyngeal bursa has an opening into the sac which varies in size from that of a pinpoint to a vertical slit, or a large elliptical opening. It may give the impression of a mere cul-de-sac. The bursa pharyngea is not a closed sac lined with endothelium, nor is it of mesodermic origin. Therefore, in the strictest sense it is not a true bursa, and only long-continued use of the term has justified this terminology. Sometimes the opening is completely closed, forming a retention cyst or an abscess. The content may be purulent, cystic in character, or contain a jelly-like mucus. When

infected, the walls are reddened and there may be lymphoid tissue or granulation tissue within the sac.

The more common type encountered is the lymphoid hyperplasia which enlarges as a result of repeated inflammation, and thereby forms pockets, linear furrows or retention cysts. The contents of the cyst may be purulent, thus it is frequently termed an abscess. The hyperplasia may be of sufficient size to be termed an adenoid growth. In some cases there is merely an infected median recess. Thus no constant finding in the epipharynx is typical of one particular symptom or another. They are all intimately related, and the significance of findings should be given conscientious evaluation as to the cause of symptoms.

Histologic studies carried out in a number of the cases reported, consistently reveal chronic inflammation, and at no time did we find normal respiratory epithelium. The surface covering showed transitional epithelium with squamous metaplasia, and the stroma gave evidence of chronic inflammation by the presence of round cell infiltration and proliferation of fibroblasts. Some sections evidenced greater round cell infiltration than others, indicative of more intense chronic inflammation.

Treatment in these cases consisted of surgery, irradiation and local therapy. In treating the nasopharynx, surgical removal of hypertrophied lymphoid tissue is indicated, if present. In the cases reported, radium was employed in addition to surgery, because we were concerned with a regrowth of lymphoid tissue following previous adenoidectomy.

Local treatment consisted of the application of 5% silver nitrate through the Yankauer speculum. It is possible at times to make this application rhinoscopically when space permits. The use of a small cotton-tipped applicator is recommended so as to avoid an excessive amount of solution which might run down into the pharynx. More intensive caustic materials are not advised because of probable subsequent destruction of mucus glands or possible scar tissue formation. However, electrocauterization was employed in two cases where bleeding was the most prominent symptom, and trichloroacetic acid was applied in two others of this series.

Radium was applied to those cases which did not respond to conservative local management. The manner of application is essentially the Crowe technic, using the 50 mg. monel metal radium applicator, at

intervals of one month, for two and sometimes three treatments. The duration of the exposure is fifteen minutes. A note of caution should be sounded concerning the indiscriminate use of radium for treatment to the epipharynx. One should inquire always as to whether or not the patient has had previous radium therapy. Certainly, it should not be applied when no discernible pathology is present in the nasopharynx, nor should it be employed solely on the basis of aural findings. Furthermore, lymphoid tissue can regenerate following such radium therapy, and it is possible to have metaplastic changes take place. How are we to know in what percentage of cases the stimulating effects may bring about neoplastic formation in years to come? These are questions which concern us for the future, and further observations and studies are necessary before we can evaluate the true permanent effects of irradiation therapy.

SUMMARY

1. A review of 150 cases of epipharyngeal pathology in the order of frequency of associated symptoms, is presented.

2. In 40 cases the most prominent symptom was postnasal discharge. Next in order of frequency were 35 cases of recurrent sore throat. The other conditions presented in order of frequency were: Cough, earache, recurrent colds, hoarseness and bronchitis, epipharyngitis, fever, bleeding, crusting, and one case each of occipital headache, asthma and tuberculosis.

3. It is the intent of this paper to point out that more attention should be directed to routine examination of the epipharynx, particularly when dealing with symptoms referred to in this discussion.

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DISCUSSION

FREDERICK T. HILL, M.D.: Mr. President, this very excellent paper of Dr. Theobald should serve to stimulate greater interest in the careful examination of the nasopharynx, which is so often overlooked. I am glad he emphasized the importance of direct examination.

I think we should consider direct examination of the nasopharynx just about as we do direct examination of the larynx. We should not be satisfied with a mirror examination or the nasopharyngoscope any more than we would with a mirror examination of the larynx of any case which required more accurate study.

Electrocoagulation can be nicely used in attacking lesions in the fossa by using the Yankauer speculum as the indifferent electrode.

In considering acute epipharyngeal infections, it is well to keep in mind that sometimes, particularly in the cases who have been tonsillectomized, diphtheria may be manifest only on examination of the nasopharynx. We have seen not a few cases of nasopharyngeal diphtheria which would only be recognized by direct examination and culture.

Again I want to congratulate Dr. Theobald on a very excellent paper.

THOMAS C. GALLOWAY, M.D.: The late Dr. Profant of Santa Barbara told of a man who came to him with disabling arthritis. Dr. Profant found an infected pharyngeal bursa which was removed and the man cured. This man had a list of fifteen of the leading otolaryngologists of the country whom he had consulted without success. He was so impressed on the lack of information on this subject that he proposed to endow an institute for its study.

I have been listening all day to find an answer to the question as to the importance of lymphoid follicles in the nasopharynx. Dr. Boies touched upon that. I gather that he thought these follicles were rather unimportant. He quoted Dr. Rhoads' and Dick's investigation of infection on a quantitative basis determined on the number of colonies of bacteria in the tonsil stumps. Relatively the greater concentration was in the fragments.

I removed many of those stumps at the County Hospital for Dr. Rhoads. Later on I removed a good many lymphoid follicles from the nasopharynx and

lateral lymph bands. Dr. Rhoads did not accumulate enough information for a statistical report but it was definitely his impression that those contained highly important infection.

No one has mentioned a method of instrumentation which I think many of us are using with considerable satisfaction, and that is the use under direct inspection preferably by the Love retractor of a fine punch forcep to remove lymphoid follicles and lateral bands, leaving no large continuous denuded areas but islands of epithelium so there will be no scarring. I think it should be pointed out that the pharyngeal bursa is likely to enclose infection with only a small perforation through a diaphragm so that one cannot hope to cure such a lesion by irradiation but must rather unroof or eradicate such a lesion.

SAMUEL SALINGER, M.D.: Dr. Theobald mentioned the various methods of examining the nasopharynx. I would like to point out the fact that in children, as you all know, it is extremely difficult to use the post-nasal mirror. It is also quite a trial to use the nasopharyngoscope.

I have found the greatest value in anterior rhinoscopy through shrinking the turbinates. Through many years of practice, I have been able through the wider side—there is usually a wide side and a narrow side—to be able to see the nasopharynx and visualize the adenoids when the patient phonates. It takes just a little patience and practice to develop this technic, and it also requires a light which will penetrate. This cannot always be done with a head mirror unless one knows the exact focal distance of the mirror. I have been trained for many years, formerly through Dr. Freer, my preceptor, in using the old Kirstein headlight, which throws a very powerful light of parallel rays. This light will penetrate to a depth of 10 or 12 inches which makes it easy to illuminate the bottom of a narrow channel such as the inferior meatus.

W. H. THEOBALD, M.D. (Closing): I wish to express my thanks to Dr. Hill, Dr. Galloway and Dr. Salinger. I also appreciate the words of Dr. Hill substantiating the method of examination.

The greatest gratification I have had are the results obtained in those cases of chronic obscure fever and bleeding which were studied for weeks and months at a time by the internist without finding any cause for fever, until we found the source of infection in the epipharynx.

The direct examination after all is the only means by which we can evaluate the furrows and crypts, the depth of the lymphoid hyperplasia or the presence of a bursa. It is comparable to looking at an oat field where you see a little green patch but you do not see the weed patches until you get up in a plane and look down on the field.

The use of punch forceps after adenoidectomy is a very smart procedure.

It is my plan now to inspect the epipharynx after adenoidectomy, preferably with the Love retractor.

As Dr. Salinger mentioned, I think I brought out that the rhinoscopic examination of this field when there are no obstructions, gives a very good view of the epipharynx.

COMPLICATIONS FOLLOWING IRRADIATION OF THE THYROID GLAND*

R. M. LUKENS, M.D.

PHILADELPHIA, PENNA.

X-ray irradiation of the larynx for carcinoma is justified in cases of inoperable cancer and as after-treatment in those cases in which there is a question of remaining concealed cancer cells.

However, when x-ray is resorted to in treatment of other conditions in close proximity to the larynx and trachea (notably lesions of the thyroid gland), much consideration must be given to measuring the possibilities of damage to the underlying larynx and trachea.

"Among roentgenologists, irradiation of the thyroid gland for exophthalmic goitre, hyperthyroidism, etc., is considered definitely indicated; however, the dosage must be controlled and filtration properly applied. Over-dosage must be strictly guarded against. The object of the treatment is for physiologic effect only and any dosage or length of irradiation, which will produce organic change must be guarded against. Prolonged or intensive irradiation of the thyroid gland for goitre is unjustified.

"For surgical diseases of the thyroid, such as benign tumors, cysts, etc., x-ray treatment is not indicated."¹

When indiscriminate use of irradiation is made in trying to cure the diseased thyroid gland, damage can be done to the underlying larynx and trachea. The damage done to these organs is permanent and will require a lifetime of treatment. In addition, the patient is constantly in danger of death due to asphyxia. In the cases herein reported, obstruction to the airway was due, not only to pathologic narrowing of the larynx and trachea, but also was due to altered secretion accumulating at the point of stenosis. Indeed, one patient lost her life because

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crusts became wedged in the narrowed airway during the night and help could not be obtained quickly enough to save her life.

Cartilages of the larynx and trachea are susceptible to intensive irradiation. Jackson² has said in reference to irradiation for the cure of laryngeal papilloma that "in using radium and the Roentgen ray, the slightest overdosage will end in disastrous perichondritis, necrosis, stenosis, or sloughing."

Clerf³ has shown that extensive irradiation of the thyroid gland for exophthalmic goitre produced telangiectasis of the skin over the front of the neck with subcutaneous changes in which the blood vessels were degenerated rendering the subcutaneous tissues avascular and destroying the muscles in the region of the trachea. An immediate tracheotomy for emergency resulted in sloughing of the tracheotomy wound due to necrosis and gangrene of the devitalized tissues.

In 1927, Clerf read a paper before this association on Laryngeal Complications of Irradiation in which he reported in addition to four cases of papilloma of the larynx treated by irradiation, two cases of exophthalmic goitre treated by irradiation. Both of the goitre cases required emergency tracheotomy on admission. The tracheotomy wounds, due to the devitalized pretracheal tissues, sloughed. In the one case, death ensued and in the other case, healing finally was slowly accomplished after a stormy convalescence in which infection and necrosis had to be combatted.

Changes taking place in the neck and the trachea are not at once apparent. X-rays usually are of little or no help, as they generally are negative. Change may start as a tracheo-bronchitis which does not clear up but progresses to definite localized lesions. The degenerative process is slow and may not give alarming symptoms until several years later and the damage done is permanent and progressive.

Epithelium desquamates and gland functions either are changed or destroyed resulting in altered tracheal secretion, which is not moved readily by ciliary action and adheres to eroded and granulomatous surfaces. Crusts constantly form and become attached to the damaged areas. Proliferation of granulation tissue, and in some cases definite hemangiomatous lesions produce stenosis. The dyspnea due to the stenosis is markedly increased by the thick secretions and crusts collecting about the stenosed area.

Case 1—E. G., white female, age 48. Patient received very intensive x-ray treatment for nearly one year for exophthalmic goitre eight years before admission to the hospital. Following this treatment, her breathing gradually became difficult and the dyspnea steadily increased until at the present time, she is unable to lie down at night. Three years ago, a bronchoscopy was performed at another hospital which revealed stenosis of the upper trachea. A second bronchoscopy was done by another doctor, but he was unable to pass the bronchoscope because of marked obstruction in the trachea. The patient refused tracheotomy, which was advised at that time. Since then, she has developed a cough with expectoration of blood and pus, which varied in quantity, some days as much as a quarter of a cup full. At times, the blood is bright red, and other times, it is dark and clotted.

She was admitted to the Bronchoscopic Clinic of the Jefferson Hospital seven years after the irradiation of the trachea.

Examination showed very marked inspiratory dyspnea with an inspiratory stridor. The nose, mouth, and throat were negative. Mirror laryngoscopy: larynx was negative except that there appeared to be a narrowing of the trachea about the second ring. The neck showed marked telangiectasis, particularly on the right side. X-ray showed no distinct evidence of any mass compressing the trachea. Wasserman and Kahn tests were negative.

Diagnostic bronchoscopy: At about the level of the second or third tracheal ring, there was found a marked narrowing of the tracheal airway, the result of a large granulomatous mass, which was attached to the anterior wall of the trachea. An inspissated mass of secretion was found adherent to this. Following removal of the mass of secretion, the lumen could be visualized. This appeared crescentic. No attempt was made to investigate the trachea beyond the point of stenosis. It is not certain whether stenosis is the result of inflammatory tissue or whether there are extensive cicatricial changes. Thought inadvisable to pass the bronchoscope through the stenosed lumen because of development of postendoscopic swelling of the mucosa which would have necessitated a tracheotomy.

The following day, a tracheotomy was performed for relief of dyspnea.

Two days later, a second bronchoscopy was done, at which time

it was seen that the lumen of the trachea was not greater than eight mm. at the point of stenosis. The granulation tissue causing the obstruction was removed with great improvement in breathing. Laboratory report on granulation tissue: fibroangioma of trachea.

The stenotic area was dilated with round tracheal dilators after the granulations stopped forming, and the patient was discharged to care of family physician definitely improved.

Eight months later, the mucosa of the trachea showed a granular and inflamed area which had appeared just below the previously healed portion of the trachea since her discharge from the hospital. In the meantime, the patient had been expectorating blood-tinged sputum at irregular intervals. These granulations were removed by bronchoscopy.

One and a half years later, she was re-admitted, complaining of loss of seven or eight pounds in weight, and aching pains in both ears and across the chest. During the past three months, she had had choking attacks followed by hemoptysis. On one occasion, she stated that she brought up a pint of blood.

Bronchoscopy revealed a narrowed oval-shaped lumen immediately beyond the larynx with granulations at the narrowest portion on the anterior and right lateral walls. The stenosis reduced the lumen of the trachea to about one-third of its width. Auscultation of the chest elicited whistling sounds transmitted along the bronchi and into both upper lobes.

Four years later, a small granuloma was removed from below the stenosis. Laboratory report: inflammatory tissue. A tracheotomy was necessary. There was relatively little bleeding during the tracheotomy.

Two and one-half years later, a letter was received from her family physician stating that the patient was suffering with pain in the larynx and tracheal region and hemoptysis followed by severe pain. Patient needed repeated doses of Pantopon for relief. There is a question of malignancy developing in the stenotic area.

Case 2—I. B., female, age 31. Six years before admission in 1937 to the Jefferson Hospital, patient received deep roentgen therapy for the thyroid gland. Three years later, she had an attack of aphonia following what she thought was an acute upper respiratory infection. Shortly after this, she developed inspiratory dyspnea, which has persisted and become more marked so that patient has become unable to

do hard work or exert herself. She developed cough and wheezing with expectoration of fetid, muco-purulent material with occasional greenish lumps. She has been under care of many physicians who gave various diagnoses. She had two bronchoscopies at another hospital in 1936 at which time autogenous vaccine was made and given without improvement. A diagnosis of tracheobronchitis was made. X-ray of chest in July, 1937, was said to be negative. Wasserman negative.

Patient was admitted to Jefferson on December 3, 1937 with distressing inspiratory dyspnea and irritative cough. The mucosa of the larynx was moderately congested and large dark crusts were observed in the trachea, probably three cm. below cord level by mirror-laryngoscopy. There was moderate telangiectasis with some scarring in front of the neck, apparently the result of irradiation.

The day after admission, a tracheoscopy was performed revealing a marked narrowing of the trachea, the lumen of which was not greater than one cm. in its transverse diameter. The narrowing began at the third tracheal ring and extended downward for about one cm. There was a granular ulceration of the mucosa with slight bleeding on the posterior wall. Marked crusting was present.

Patient was treated by medicated oil intralaryngeal instillations with considerable improvement.

Several readmissions to the hospital were necessary over a period of 10 years to remove the crusts from the trachea. Repeated attacks of marked dyspnea due to crusting. It was necessary to instill salt solution to remove the crusts. Bronchoscopy was necessary on several occasions to remove the crusts from the narrowed trachea.

A bronchoscopy was performed one year ago to remove crusts, and it revealed a proliferating mass on the left wall of the trachea, which appeared to be either malignant or granulation tissue. Laboratory examination reported it to be hyperplastic epithelium.

Oil instillations were discontinued and normal saline solution was substituted to avoid lipid pneumonia.

In January, 1948, patient awakened at 1 P.M. and was aphonic. She awakened her sister-in-law and pointed to her throat. Patient died before a conveyance could be obtained to take her to a hospital.

This case exhibits atrophic changes in the mucosa due to irradiation. Ciliary action of the epithelium evidently was destroyed and destruction of the epithelium resulting in ulceration and excessive granulation formation, produced stenosis. Secretion became excessive and stagnant, resulting in crusting. The big problem was controlling the formation of crusts. Instillations of saline solution helped, but bronchoscopic removal was necessary at irregular intervals. In the meantime, the patient was in constant danger of asphyxia on several occasions of an emergency nature, and finally death.

Case 3—G.M., white female, age 40. Admitted to hospital in 1939 complaining of burns to neck following extensive x-ray therapy in 1931. She had a sense of something in the throat when swallowing and on speaking for a time. In 1931 diagnosis of goitre was made for which extensive x-ray therapy was given by a nose and throat specialist. Treatments were given once a month for a year. Recently scars as a result of the treatment in front of the neck have been troubling patient and she is becoming throat conscious (senses something in the throat). No weight loss; no pain; voice not affected.

Physical examination: The patient was well developed and nourished; there was no hoarseness; no wheezing. Larynx was negative. Neck showed a rectangular area of telangiectasis three inches by four inches in size on the front of the neck. It extended slightly more to the right and exhibited more thickening and induration on the right side. There were present a number of firm areas which suggest keratosis. It was difficult to palpate structures beneath the skin in this location. There were excrescences on the affected area.

Seven years after the irradiation, bronchoscopy showed a narrowing of the trachea due to an extra-tracheal lesion which was encroaching on the tracheal lumen of its anterior and right tracheal walls. There was a narrowing at a point about 3 or 4 cm. below the level of the larynx, a crowding inward of the anterior and right lateral wall, not sufficient to interfere with an adequate tracheal lumen. The mucosa of the entire trachea was found moderately congested, no ulceration was observed and remainder of the air passage appeared normal.

Esophagoscopy showed nothing abnormal. Patient was discharged from the hospital in November, 1939.

October, 1947—Patient was seen at her doctor's office. She is

having slight increase of dyspnea and apparently also some difficulty with crusts accumulating in the trachea. There is some breaking down of the superficial epithelial layers of the front of the neck. General condition is satisfactory.

Case 4—I.C., white female, age 43. Patient complained of hoarseness for past two years with occasional shortness of breath. She had a toxic thyroid removed twelve years before admission. Following this, a long course of irradiation therapy was received. Biopsy of gland, left side: question of malignancy.

Two years after receiving a year of irradiation therapy, patient became hoarse and dyspneic. This persisted and seven years later, she had two growths removed, one from each vocal chord. Following this, voice was clear for two years. However, hoarseness gradually returned.

Physical examination: the voice was moderately hoarse. There was no dyspnea. Larynx: the motility of the larynx was good. Vocal cords were thickened, granular and red suggesting telangiectasis in their anterior halves. There were no ulcerations present. Neck: there was marked telangiectasis of skin, with considerable thickening over the trachea.

Patient was discharged definitely improved.

One month later, there was a large quantity of very thick tenacious secretion adhering to the posterior commissure which was dislodged by instillation of normal saline solution. Right vocal cord now shows hemorrhage throughout its entire extent. Left vocal cord is quite red and exhibits crusting. Instillations of normal saline solution were required to remove crusting and secretion.

Patient readmitted in December, 1946. She was aphonic and moderately dyspneic. These symptoms developed following a recent respiratory infection. There was marked crusting on both vocal cords. Following removal of crusting by saline solution, a small elevation was observed on each vocal cord at the anterior third, the right being more anterior than the nodule on the left cord.

December, 1946—These elevations were removed by direct laryngoscopy. Laboratory report on biopsy: hyperplastic epithelium.

For the past year, intralaryngeal instillations of normal saline solution have been required to rid the trachea of thick stagnant secretions which hamper respiration.

At the present time, there is still some hoarseness. Examination of the larynx reveals inflammatory thickening of the right vocal cord but no evidence of nodulation. There is no impairment of motility and the airway is adequate. The neck exhibits telangiectasis with subcutaneous fibrosis so that the entire front of the neck is firm and it is impossible to determine the exact site of the trachea.

This is another incident of extensive irradiation therapy.

Case 5—R. P., white female, age 31. Patient was in good health until five years before admission to Jefferson Hospital in September, 1944, when she began to have palpitation of the heart and nervousness. A diagnosis of hyperthyroidism was made. One year later, her thyroid gland was treated by radiation. This was followed by telangiectasis on the anterior aspect of the neck, shortness of breath, swelling of ankles, and it became necessary for her to sleep with her head elevated. Two years before admission to Jefferson, she became hoarse. Condition became worse with a feeling of tightness in throat and dysphagia. She had frequent sore throats. In past few weeks, hoarseness and dysphagia increased with a loss of weight of seven pounds. Patient has been taking iodine, but none during past several months. Symptoms of hyperthyroidism have persisted.

Mirror laryngoscopy shows marked degree of stenosis of the larynx, due to a proliferating lesion which is subglottic, with complete obliteration of the anterior third of the glottis. There also is some change in the posterior commissure. As a result, the airway is oval in shape and occupies about 25% of the normal glottis. Motility of two sides of the larynx is quite satisfactory. There is marked telangiectasis of the front of the neck. X-ray shows no evidence of retrosternal thyroid. Wasserman and Kahn are negative.

Shortness of breath gradually is becoming more marked, and patient has not responded to medical treatment at home. Chest expansion is equal, impairment of percussion note both lower lobes anteriorly and posteriorly, and wheezing breath sounds throughout.

A tracheotomy was performed and the tissues were found to be more or less avascular. As soon as the trachea was opened, the patient coughed up a large clump of thick secretion. X-ray examination was negative and did not show anything to suggest stenosis but after instillation of iodized oil, X-ray showed a short, narrow constriction of the tracheal air column immediately below the thyroid cartilage.

Under direct laryngoscopy an incision was made with laryngeal knife along the left margin of the web practically to the anterior commissure. Laryngoscopic dilatation was done using No. 20 triangular bougies through narrowed laryngeal lumen, four days later and the airway became definitely larger than formerly.

December, 1944—Patient discharged to return to her physician.

When seen one and a half months later, patient's voice was good. Laryngeal airway seemed ample. Left vocal cord remained intensely red, and the right cord is almost normal. Part of web anteriorly has reformed, particularly subglottically.

A second incision by direct laryngoscopy was carried along the left vocal cord extending to practically the anterior commissure, and a complete cork was used in the tracheal cannula.

Two months later, the cannula was removed because of satisfactory breathing through the larynx.

Seven months after decannulation, patient again began to have difficulty with breathing and particularly with phonating. She had to clear her throat a good deal and had difficulty in getting rid of thick sticky secretion. Her general condition was satisfactory.

Mirror laryngoscopy showed a marked redness of the mucosa and the presence of thick secretion adhering to the vocal cords, ventricular bands, and epiglottis. Instillations of salt solution were necessary to remove the thick and adherent secretions. The front of the neck now exhibits slightly more induration of the region of the telangiectatic areas, and treatment consists of intratracheal instillations of salt solution and 5 c.c. of ephedrine sulfate instilled into the larynx daily.

SUMMARY

Symptoms exhibited by the five cases recorded here are: hoarse-sensation, sensation of lump in the throat, dyspnea, wheezing, dysphagia, expectoration, loss of weight, and pain in chest.

Physical findings are: congestion of laryngeal mucosa, telangiectasis and scarring of neck, stenosis of larynx, inflammation of laryngeal mucosa, telangiectasis of vocal cords, stenosis of trachea, granulomatous lesions in tracheal walls, crowding inward of tracheal wall, viscid adherent tracheal secretions, and crusting. Cicatrices are not common. Only a suggestion of cicatricial tissue was present in one case. Tracheotomy

was required in three cases. The time between termination of irradiation and appearance of first symptoms of tracheal disease was one year (2 cases), three years (2 cases) and seven years (1 case).

Cases under continuous treatment for relief of symptoms: one year (1 case), three years (1 case), nine years (1 case), ten years (1 case) and eleven years (1 case).

CONCLUSION

While the irradiation is not aimed at the larynx or trachea, these organs will suffer from excessive or extensive irradiation of the thyroid gland. Not only may the tissues of the larynx and trachea be altered by the rays, but the overlying tissues will be damaged by changes in the blood vessels and lowered resistance to infection so that operative procedures (tracheotomy) become hazardous. Not only is healing of the operative field retarded or prevented by the devitalized tissues, but resistance to infection is lowered, resulting in some cases in necrosis and gangrene of the operative field.

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DISCUSSION

LOUIS H. CLERF, M.D.: As Dr. Lukens stated, 21 years ago I reported before this Association a number of cases of untoward effects of excessive irradiation carried out for benign lesions of the larynx.

Since then I have seen additional cases and I found to my surprise, that my associate Dr. Putney presented several cases in 1938. In the last two months I have seen two additional long standing cases. In one, a woman 47 years of age, intensive irradiation was started in 1924 and continued a long period of time for a benign thyroid lesion. She developed slight hoarseness, but about two years ago her difficulties increased. When I saw her about seven weeks ago she had the usual evidences, marked telangiectasis of the neck, with firm induration of all tissue, a lack of all anatomical markings and telangiectasis of both vocal cords. This appeared as a vascular tumor on one cord with telangiectatic changes of the opposite cord.

In another case, a woman 58 years of age, irradiation therapy was started 12 years ago for a benign lesion of the thyroid gland. About 18 months ago she began to have symptoms referable to her pharynx with dysphagia. I found an extensive post-cricoidal carcinoma.

Obviously women do get carcinoma in this location if they had no irradiation therapy, but I am of the opinion that the carcinoma in this case was unquestionably related to the irradiation because irradiation does in time produce certain degenerative changes.

All of us will agree that irradiation has a place in therapy but I do not believe that the average competent roentgenologist will continue it indefinitely for a benign condition. If it does not relieve after a definite dosage it should be discontinued.

Too many men who are not familiar with irradiation use a very powerful drug, concerning which they know little or nothing and those are the people that get into difficulty. May I say that when these patients come to us we too get into difficulty if we try to relieve them of their disability.

SAMUEL SALINGER, M.D.: Several years ago, in reporting my experiences with irradiation for carcinoma of the larynx I took occasion to note several cases in which extensive changes had taken place in the larynx, including sloughing of cartilages, etc.

More recently I reported a case of a young man in whom the development of the larynx had been arrested through irradiation, who developed marked atrophy of the neck, to such an extent that we had to do a complete grafting of the front of the neck in order to replace the atrophic tissues.

The thing which surprises me about Dr. Lukens' report is that at this present time when irradiation therapy is supposed to be safe we still find cases in which you have this irreparable damage. I am wondering whether among the many cases of carcinoma of the larynx treated by deep therapy there may not be a number which will later manifest these delayed changes.

It seems to me that this matter is something that requires more careful follow-up. I know that there are a number of radiation experts who treat carcinoma of the larynx without consultation on the part of a competent laryngologist and without adequate follow-up, and this report of Dr. Lukens leads me to believe that among these groups in which a high percentage of cures is reported there are very likely to be a certain number in which delayed damage to the cartilages has not been noted.

R. M. LUKENS, M.D. (Closing): I wish to thank you gentlemen for discussing my paper.

Regarding the cure of disease of the thyroid gland by x-ray, I have had no experience. Some roentgenologists believe that x-ray therapy is definitely indicated in disease of the thyroid gland, but that physiologic reaction is the limit to be reached and not tissue alteration. The disease for which irradiation had been used too vigorously often persisted after permanent damage had been done to the thyroid and adjacent organs.

MUCOCELES, PYOCELES AND CYSTIC DEGENERATIVE LESIONS OF THE FRONTAL AND ETHMOIDAL SINUSES*

H. I. LILLIE, M.D. AND CLIFFORD F. LAKE, M.D. (*By Invitation*)

Mucocele, pyocele and cystic degenerative lesions of the lining membranes of the frontal and ethmoidal sinuses have been encountered as surgical problems since they were first described in 1725 by Nicolai of Strasbourg. Many reports of cases of these lesions have appeared in the literature. Surgical procedures designed to control these lesions have been advocated on an empiric basis because the underlying etiologic factor is not well understood even at the present time. If the pathologic basis for these lesions were understood there should be no uncertainty about what type of surgical procedure could be expected to control the disease process. Inferentially, if such lesions could be recognized in the very early stages of their development, surgical procedures could be better applied. It is evident, because the results of surgical treatment frequently are unsatisfactory, that the problem is not settled.

In the critical analysis of the material here presented an attempt will be made to present relevant observations that are important in the management of such pathologic conditions.

MATERIAL

We reviewed a series of cases of the clinical syndrome generally accepted by rhinologists as due to mucocele, pyocele and cystic degeneration of the lining membranes of the frontal and ethmoidal sinuses, in order to ascertain if any deductions could be made relative to (1) the cause of the pathologic lesion, (2) the cause of failure of surgical treatment and (3) the choice of surgical procedure. In 22 cases, operation had been performed before the patients came to the Mayo Clinic. In 10 cases in which the patients had not been operated on before they came to the clinic, secondary operations were performed at the clinic. Such pathologic conditions as subperiosteal abscess, meningioma, osteoma,

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orbital tumors and neoplasms which cause the deformities usually associated with mucocele, pyocele and cystic degeneration lesions have not been included. Such lesions may coexist, however.

If the intranasal examination disclosed definite evidence of suppurative sinusitis, the case was excluded. The reason for excluding such a case was that the probability was great that the lesion in the frontal sinuses which caused the external symptoms and signs was due to abscess formation with retention, or to inadequate drainage and was not due to a mucocele, a pyocele or a cystic degeneration lesion. The objection may be raised that such a distinction is too finely drawn. If an intranasal operation had been performed previously for relief of suppurative frontal sinusitis and if the intranasal examination did not reveal any evidence of purulent discharge or inflammation and, in addition, if the nasofrontal duct could not be probed, the case was included on the basis that the lesion in the frontal sinus was a closed empyema with an inflammatory capsule.

Cases in which there was no particular evidence of localized inflammation in the nose but in which a fluctuant mass was encountered in the region of the middle meatus but not in the orbit were included. Such findings were considered an indication that the expanding mass in the frontal and ethmoidal sinuses had extended toward the interior of the nose instead of externally into the orbit.

Another group of cases that were included were those in which draining fistulas in the orbit had persisted after an external exploratory procedure and the intranasal examination did not disclose evidence suggestive of suppurative disease of the paranasal sinuses.

It may be said that the cause of cystic tumors which arise in the frontal sinus is not known. There is considerable evidence to suggest that trauma, either caused by direct force or by inflammation, may be a factor in causing cystic degeneration of the mucous membrane.

Pathologically there is much evidence to suggest that this type of lesion is due to cystic degeneration of the mucous membrane. The walls of the cyst may contain ciliated epithelium which suggests that the origin of the lesion is in the ducts of the glands. Cuboidal or columnar epithelium may suggest that the lesion originates in the alveolar portion of a gland. If these facts are true, one would expect such lesions to

arise in the mucous membrane closely associated with the outlet of the sinus because secreting glands in the sinuses are usually situated in the immediate vicinity of the nasofrontal duct. Seldom, however, have operations been carried out early enough to confirm this assumption. Although exploratory operations have revealed cystic lesions that did not fill completely the cavity of the frontal sinus, the origin cannot be determined definitely.

The cystic mass may contain mucus, a thin fluid which usually is straw colored or greenish in color, or an opalescent milky fluid which somewhat resembles pus. Cultures of the fluid do not produce bacteria. After the fluid of the cyst has been removed, an amorphous mass may remain. Examination of this mass which is suggestive of cholesteatoma, may reveal cholesterol crystals and deposits of calcium and hemosiderin.

The walls of the cysts are usually very thin and the removal of the cysts is difficult because their walls are very friable. It is more difficult to remove the cyst from the periorbital portion of the sinus than it is to remove the cyst from other portions of the sinus.

If infection of the cyst has occurred, an abscess may be present and the wall of the cyst may be thick enough to be called a capsule. It is this type of lesion which properly may be considered to be a pyoceles. Such lesions are more readily removed than are thin-walled cysts. Examination of the walls may reveal little or no evidence of epithelial lining but will reveal fibrous tissue.

If the mass presenting in the orbit has been aspirated or drained previously and if such a procedure has resulted in a draining fistula, infection of the cyst may have taken place. Such infection was observed in several cases.

If the cystic mass fills the sinus completely, as it sometimes does, the origin and nature of the lesion may not be recognized.

Closed empyema resulting from closure of the nasofrontal ducts either as a result of inflammatory reaction or scar tissue caused by intranasal operation does not have these general pathologic characteristics but resembles encapsulated abscesses.

SYMPTOMS AND SIGNS

The generally accepted symptoms and signs of cystlike lesions of the frontal and ethmoidal sinuses are about as follows. The history may be depended on to reveal that pain of varying degree has been present at some time in the course of the development of the lesion. The onset may occur after some injury, a factor frequently overlooked, or it may follow an infection of the upper part of the respiratory tract, or there may be no apparent antecedent cause. The pain gradually increases in severity and is intensified by conditions which cause congestion of the nasal mucous membrane. Usually the pain is more pronounced when the patient is in the upright position or when he stoops or strains. If relief of pain is associated with a discharge into the nose, in the type of lesion under consideration, it can be inferred that spontaneous rupture has occurred or that the position of the lesion has changed so that escape of the accumulated secretions within the sinuses may have occurred when obstruction to the duct was relieved. Recurrence of pain which gradually increases in severity is a common symptom under these circumstances if the cyst re-forms.

The pain may or may not be relieved by acetylsalicylic acid and similar drugs. Occasionally sedatives are necessary. Tenderness may be present over the frontal sinus. The tenderness increases on pressure. As the pressure within the sinus increases, resorption of the bone of the walls of the sinus gradually takes place in any direction, but most frequently occurs in the floor of the frontal sinus. Diplopia may be noticed at this stage. As more of the bone of the floor of the sinus is resorbed the pain becomes less troublesome. A mass may be noticed over the eyeball. The mass slowly increases in size and causes the eyeball to be displaced downward and outward as a general rule. If the displacement of the eyeball is directly forward or toward the nasal aspect of the orbit, tumors within the orbit may be considered to be the cause of the symptoms. Disturbance of vision other than diplopia occurs when the tumor mass occurs posteriorly to the globe.

Diplopia may occur in the early stages. It may persist throughout the course of the disease. Diplopia can be considered a sign if elicited on change of direction of the eyes.

The deformity resulting from the protrusion of the eyeball can

be extreme or so slight as to be hardly noticeable. Patients are more likely to seek relief from the embarrassing deformity of the orbit and diplopia than for other signs and symptoms.

In the early stages of the increase in size of the cystic mass within the frontal sinus there may be no external signs of a lesion. Its presence can be suspected only on the basis of information revealed in the history and on the basis of experience.

Palpation of the tumor mass in the orbit always yields important information. The surface is smooth and the mass feels like a soft rubber ball. The mass does not disappear unless it ruptures into the nose. The edge of the mass can be felt to be a margin of bone and at times the defect in the bone can be outlined if the mass is small. If the lesion is situated deep in the orbit, it is not always palpable and cannot be outlined by palpation.

It is highly improbable that roentgenologic examination will reveal the presence of such a lesion but, since other types of tumors must be considered in the differential diagnosis, roentgenologic examination always should be performed. Such examination will furnish important anatomic information.

DIFFERENTIAL DIAGNOSIS

In the differential diagnosis, orbital tumors must first be considered. Sometimes the diagnosis is difficult if the cystic lesions are situated deep in the cone of the orbit and arise from the ethmoidal cells. However, in cases of orbital tumor the history usually will reveal that pain or headache was of minor importance in the early stages, whereas an expanding lesion within the sinus will cause pain until pressure is relieved by extension into the orbit. In the presence of either lesion, the clinical course is afebrile and there is no suggestive evidence of inflammation. Trauma well may be a factor in the development of either type of lesion but the importance of trauma is most often overlooked in the history. This fact was apparent in our study.

If the mass in the orbit does not seem to be associated with the bone of the floor of the frontal sinus or is not fixed in position, an orbital tumor must be considered as the cause of the deformity. The lesions under consideration are fluctuant on palpation. This also may be true of certain other orbital tumors. If the proptosis of the eyeball is down-

ward and inward, the lesion probably is an orbital tumor unless roentgenologic examination reveals that the frontal sinus extends beyond the zygoma. If pressure on the mass in the orbit causes the mass to disappear by releasing a purulent discharge into the nose, it may be concluded that the empyema is associated with subperiosteal abscess and that the mass is not cystic. If an intranasal operation has been carried out previously, it can at least be inferred that intranasal drainage has not been adequate, no matter what the lesion in the orbit proved to be.

In addition to diplopia, disturbances of vision and alterations of the visual fields may occur with any intraorbital lesion but most frequently are associated with lesions which are situated deep in the orbital cone.

Aneurysms occur in the orbit and may need to be considered in the differential diagnosis although the symptoms and signs of the two lesions are quite different. The essential difference is that in cases of aneurysm a discrete mass is not palpable but a pulsation frequently is discernible. Positive proof of the presence of aneurysm is the detection of a bruit.

Thrombosis of the cavernous sinus, if aseptic, is a remote cause of proptosis. The presence of the inflammatory type of thrombosis can at once be ruled out because of the lack of evidence of severe illness.

Roentgenographic examination may be very helpful if the contents of the mass are relatively opaque to the roentgen rays. In cysts or pure mucocoeles confined within the frontal sinus, the results of roentgenologic examination may be disappointing. Such examination may disclose downward displacement of, or breaks in the integrity of, the floor of the frontal sinus.

Lastly, aspiration of the mass may be very revealing but a creamy opalescent type of content must not at once be considered to be purulent unless organisms can be demonstrated by bacterial culture. If the mass in the orbit has been drained previously, secondary infection may have occurred and may confuse the diagnosis.

TREATMENT

The treatment of the cystic lesions under consideration is essentially surgical. The type of operation to be employed must be adapted to the needs of the individual patient. Certain general principles for the

surgical management may be mentioned from the experience gained in the study of this group of cases.

If the lesion is encountered before external manifestations are present in the orbit or interior of the nose, an external operation and complete dissection of the cyst without interference with the nasofrontal duct may produce a favorable result. This statement is made because of our experience in such cases. In 1 case, after the removal of the cystic mass was accomplished, the nasofrontal duct was enlarged. Subsequently, suppurative sinusitis developed after the patient had had an infection of the upper part of the respiratory tract. Three subsequent operations were necessary before the disease was controlled. In another case a mucocele confined to the bregma cell was not recognized at the time of the original operation and a subsequent operation was performed. In other cases the nasofrontal duct was not interfered with but the external wound was closed. In these cases the result was good.

If the manifestation of a lesion is entirely intranasal, a two-stage operation may be indicated. The first stage consists of the routine extensive removal of the intranasal manifestation of the lesion. Such an operation may result in permanent control of the pathologic process. If it does not, an external operation and removal of the cystic sac can be carried out at a later date.

If the lesion is both orbital and intranasal, a two-stage operation may produce a similar result. If the lesion is of this type, following Goodyear's suggestion, that is, manually forcing any depressed edges of bone in the orbit back into their normal position to relieve the deformity, may be effective. In 8 of 13 cases in which the lesion was of this type, the first stage of the operation proved to be effective.

In cases in which the cyst is entirely orbital and the nasofrontal duct is patent, an external operation and dissection of the cystic mass and noninterference with the nasofrontal duct rightfully may be expected to control the disease. Such a cystic mass may be encountered in any portion of the frontal sinus or even may fill the sinus completely. If the nasofrontal duct is not patent in cases in which this type of lesion is present, the surgeon may elect or not to fashion the duct into the nose. In 6 of 8 cases of this type in which the primary operation was performed without enlarging the nasofrontal duct, the results were satisfactory.

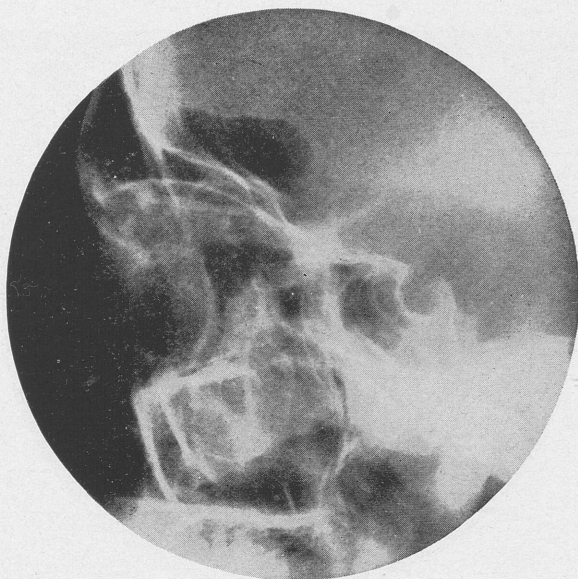


Fig. 1.—Preoperative roentgenogram showing defect in bone over temporal region due to resorption of bone from effect of pressure from a large pyocele.

In cases of closed empyema or pyocele in which there is a history of sinusitis, it would seem important that the opening of the nasofrontal duct into the nose should be enlarged. This procedure produced satisfactory results in 30 of 34 cases of this type. Experience in this type of case suggests that the result will be better if the sac of the pyocele (fig. 1 and 2) is removed completely. This is particularly true in cases in which secondary operations have been necessary.

In cases in which a second operation is necessary, the proptosis is more difficult to relieve owing to the presence of scarring in the orbit. In 6 cases of this group, draining fistulas resulting from incision of the fluctuant mass were encountered. In 12 of 22 cases in which operations were performed, the results were considered satisfactory from the point of view of the patients. From the cosmetic point of view of the surgeon, the result may not be entirely satisfactory because the proptosis was not completely relieved. This accounts for the discrepancy in deciding whether the result is satisfactory or not.

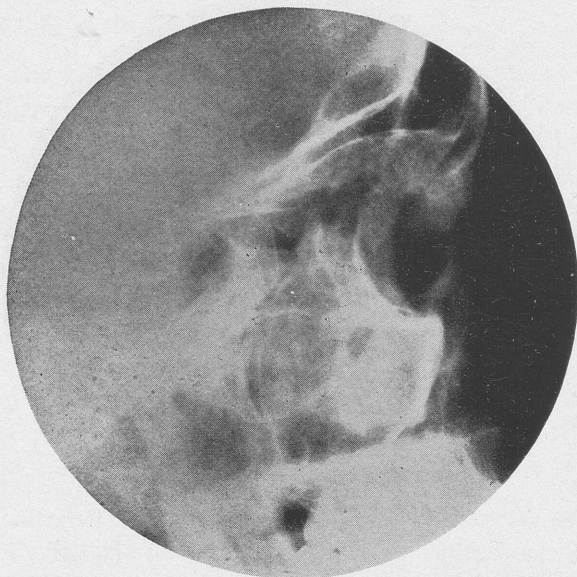


Fig. 2.—Postoperative roentgenogram showing repair of defect in bone over temporal region one year after removal of large pyocele.

If any portion of the cystic sac is left in the cavity, recurrence may take place whether the nasofrontal duct is patent or not. Such a recurrence occurred in several cases in which the cystic sac was not removed completely. It was these experiences which caused us to remove every vestige of the sac in subsequent cases. New cystic tumors, unrelated to previous ones, may occur. Such tumors were encountered in 3 cases. It cannot be said that the original operation was not successful because another cyst developed after a lapse of several years. The occurrence of the new cyst was due to the tendency of the mucous membrane to undergo cystic degeneration. Complete removal of the lining membrane would not have prevented recurrence, in our opinion.

It was interesting to observe that the dura had been exposed by the disease in 6 cases. The exposure of the dura did not prove to be of any significance. In 1 case in which a secondary operation was necessary to remove a newly formed cyst, the defect over the dura was found to have undergone spontaneous repair (figs. 3, 4 and 5).

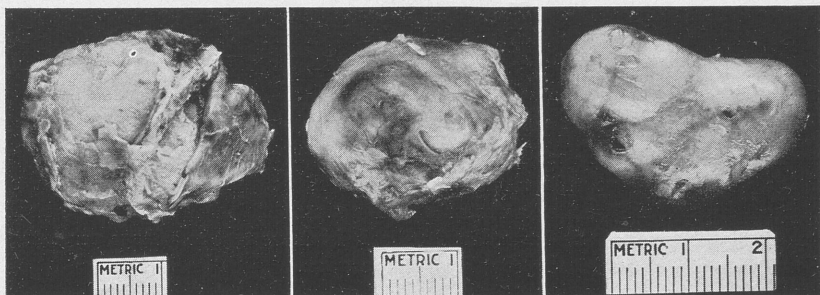


Fig. 3.—Mucocoele from frontal sinus.

Fig. 4.—Pyocoele from frontal sinus.

Fig. 5.—Pyocoele from frontal sinus.

In 2 cases, blindness had occurred preoperatively. In another case, postoperative blindness developed as a result of thrombosis of the central artery of the retina. No adequate explanation for this incident has been given.

SUMMARY AND CONCLUSIONS

This report is based on a study of the symptoms and signs of cystic type lesions of the frontal and ethmoidal sinuses. It is apparent from this study that the importance of an accurate history frequently has been overlooked. The symptoms and signs are characteristic and more accurate preoperative determination of the nature of the lesion may be possible if these signs and symptoms are considered. The pathologic characteristics of this type of lesion are not uniform. The cause of these lesions has not been determined. Surgical interference must be adapted to the individual requirements of the pathologic process encountered. A routine procedure should not be used in all cases. Earlier recognition of such lesions may prevent the application of unnecessarily extensive surgical procedures. It is extremely important that the cystic mass be removed intact. If any portion of the cyst is not removed, recurrence of the tumor is likely. It is most difficult to remove the portion of the cyst which may have become attached to the periorbita. It is not always necessary to enlarge the nasofrontal duct.

DISCUSSION

HENRY M. GOODYEAR, M.D.: Mr. Chairman, I am not only grateful to Dr. Lillie for this very beautiful presentation, but also for his kind reference to my interest in this particular lesion.

Dr. Lillie has emphasized the importance of resecting the lining membrane in toto in these lesions. He does not recommend it in all cases but he has emphasized it. We do not remove this membrane because of its pathological condition or changes, but because we fear a recurrence of a closed cyst. The membrane itself, if it remained, would do no harm. On that basis I believe that in a large percentage of these cases we can greatly simplify the operation.

In 22 of the 44 cases reported by Dr. Lillie there had been a previous operation and, of course, those are the difficult cases to cure and they are the cases which require the most radical surgery.

If we do completely resect a mucocele and do a radical operation, as the wound heals, of course, the nasofrontal passage is going to be filled with a fibrosed mass which will remain quiet until we have a secondary infection.

We have had mucocèles which we have operated upon and so dissected which have recurred five or ten years later, with a recurrence of infection of the frontal ethmoid area.

Ten days ago while at the meeting of the Ohio State Society, I incidentally met a young man whom I recognized and he told me that I had operated upon him some years ago for a mucocele in the frontal sinus. He had a diplopia and at that operation nothing was done but to remove a small area from the floor of the extension of this mucocele into the anterior ethmoid area and the large tumor mass in the orbit was pressed back in position with the finger. This man told me that he had gone through two years of very active service in the war without any recurrence or trouble with this sinus.

Two weeks ago we showed at our clinic a woman on whom I had done a similar operation, two years ago, who had a very extensive mucocele. She had a large frontal sinus and most of the frontal bone over the frontal area, for perhaps an area in width of 2 or 3 inches was gone and she had been incised twice previously for removal of fluid in this cyst, but nothing had been done in her nose, fortunately, so that I had the first opportunity, because the success in these cases lies with the first operator. If too much is done, and the epithelially lined duct, which nature has made for us, is not preserved the operation will not succeed.

W. LIKELY SIMPSON, M.D.: In discussing Dr. Lillie's very important presentation, it seems to me that to discuss the prognosis of these cases, especially those factors which if observed improve the prognosis, would be time well spent.

First, I would like to compare the feasibility of removing the diseased bone and mucous membrane of the accessory sinuses with that of the mastoid cells. It is not always possible to remove all of the diseased septa and the mucous membrane of the mastoid cells, especially in the petrous portion of the temporal bone,

and often some of the infra-labyrinthine cells are inaccessible with due regard to the safety to the surrounding structures, while it is practically always possible to remove all diseased bone and mucous membrane of the sinuses. It does seem to me that obtaining a dry ear after a mastoid operation depends almost entirely upon the thoroughness of removal of the diseased bone and mucous membrane and very rarely depends upon the pathology in the eustachian tube.

A moist or suppurating mastoid cavity is usually indicative of cells which have not been exenterated and the same principles can be applied to operative results on the accessory sinuses.

The operative procedures upon the ethmoid, sphenoid and frontal sinuses which have been very satisfactory to me and if followed give a better prognosis are:

1. The external approach.
2. The removal of all the ethmoid cells with their mucous membrane leaving the middle turbinate. The nasofrontal duct is entirely removed with the cells. Skin or mucous grafts have not been helpful to me.
3. Removal of all mucous membrane of the sphenoid when diseased.
4. Complete exenteration of the frontal sinus. Removal of the entire floor of the cavity. Visualization of all parts of all sinuses is basic. Often the entire frontal sinus lumen can be visualized only by the aid of mirrors.

After all septa of all the sinuses have been removed with forceps, currettes, rongeurs, etc., the remaining ridges are removed flush with the surrounding bone with cutting and polishing burs with marked improvement in the healing time and postoperative course.

The prognosis depends upon the thoroughness with which the exenteration of the bony septa and mucous membrane of both the ethmoids and frontal is accomplished along with careful non-traumatic surgery.

H. I. LILLIE, M.D. (Closing): Surgical problems are usually subject to differences of opinion. I still insist that I do not, in the type of case under discussion, care to remove all the lining membrane of the frontal sinus. It is not necessary. I do not always open the ethmoids extensively and purposefully make a large defect in the nose unless it is necessary to remove the disease process completely. In that respect Dr. Simpson and I do not agree, so far as these cystic lesions are concerned. The operative procedure necessary in suppurative sinus disease is another question. He may be correct. What might be considered to be an adequate operation in one individual would not be an adequate operation in another, and therefore I have no routine method of operating on any lesion. I adapt my surgical procedure to the apparent needs of the case in hand.

ALIPHATIC VERSUS AROMATIC AMINES AS VASOCONSTRICTORS

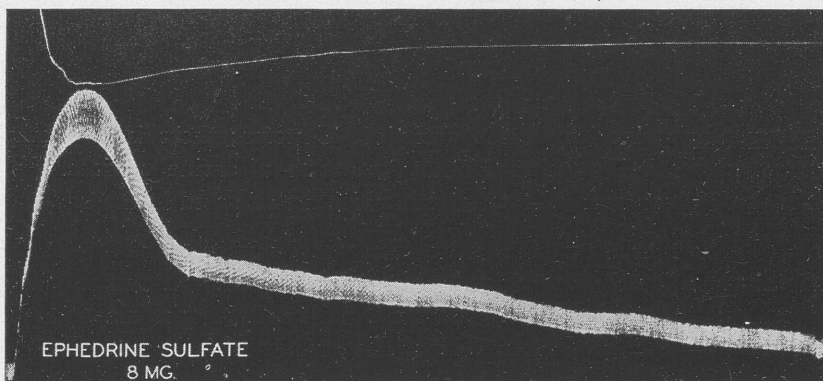
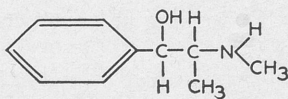
K. K. CHEN, Ph.D., M.D.

INDIANAPOLIS, INDIANA

The treatment of any disease in its best form is directed to the cause. This principle unquestionably also holds true with the affections of the nose and accessory sinuses, characterized by turgescence of mucous membrane. Unfortunately, etiological treatment of these conditions is either unyielding or not available. We, therefore, have to resort to palliative measures. According to Proetz,¹ Jelinek used cocaine for the first time on the nose in 1884, the same year Koller² discovered its local anesthetic action in ophthalmology. Proetz also mentioned that Velich and Königstein first applied an epinephrine solution to the nose in 1897, the same year Abel announced his attempts to isolate epinephrine in pure form.³ For over a quarter of a century, these two products, cocaine and epinephrine, were the only vasoconstrictors to give temporary relief to sufferers of rhinitis. The comfort to the patient, facilitation of nasal drainage, and reduction of anoxemia, as a result of such palliative therapy, cannot be underestimated. It was gradually recognized that cocaine produces addiction upon repeated use, and that occasionally the amount of cocaine absorbed from the nasal mucosa causes toxic manifestations and death. By injection, epinephrine is a perfectly useful drug in the management of allergic diseases, and in combination with local anesthetics for surgical purposes; but upon direct application to the nose, it is too short acting and induces secondary congestion which is often worse than before treatment.

It was thus not surprising that after Alice H. Cook, Fetterolf, and Sponsler⁴ demonstrated the vasoconstrictor action of ephedrine on the human nose, cocaine and epinephrine were promptly abandoned. Their results indicated that the action of ephedrine when applied locally to the nose lasted for about $3\frac{1}{4}$ hours—with no signs of irritation. Equally convincing are the animal data. Figure 1 shows the blood pressure

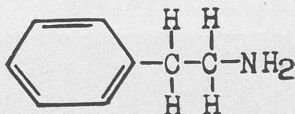
* From the Lilly Research Laboratories, Indianapolis 6, Indiana.



tracing of a pithed dog following an intravenous injection of ephedrine. During the rise of blood pressure, the nasal volume diminished, indicating the contraction of mucous membrane of the nose and accessory sinuses—recorded by the Jackson technique.⁵ Complete return of blood pressure in this experiment occurred in 57 minutes. In human subjects, a dose of 60 mg. given by mouth caused circulatory changes for more than 2 hours.⁶ This long duration of action, as well as effectiveness by mouth, is due to the fact that the enzyme amine oxidase, which is abundantly present in the body, does not destroy ephedrine.⁷ The drug is slowly excreted in the urine. Evidence is accumulating to show that the methyl group attached to the α -C-atom as the amino group protects the compound from being attacked by amine oxidase.^{8,9}

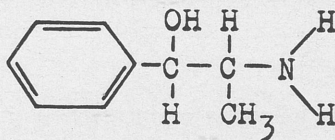
Ephedrine is a relatively simple substance in the opinion of organic chemists. It has a benzene ring and a side chain of 3 C-atoms, with a methylamino group attached to the middle C-atom (Figure 1). It therefore belongs to the aromatic amine series. Among the older aromatic amines which can be readily prepared synthetically is β -phenylethylamine. Investigations in animals soon revealed that β -phenylethylamine has a short action, and is not effective by mouth.¹⁰ Clinical work by Fenton and Larsell¹¹ fully substantiates the laboratory data, and rules

it out as a suitable drug. By looking at its structural formula, we can understand the reason why:

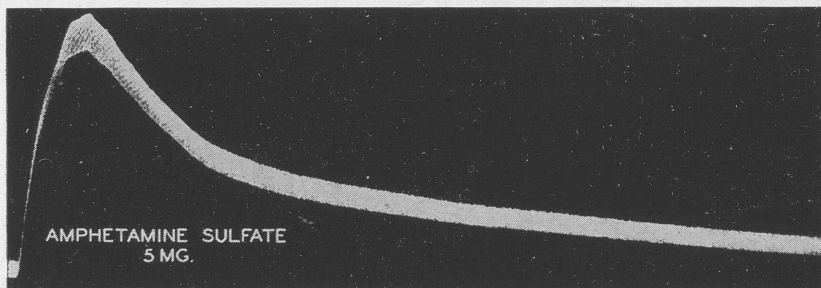
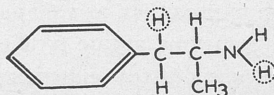


β -phenylethylamine has a side chain of 2 C-atoms; there is no methyl group on the α -C-atom to inhibit the action of amine oxidase.^{12,13,14} It is thus, in contrast with ephedrine, quickly eliminated in the body.

Newer aromatic amines were then provided by chemists. Of the numerous variants, propardrine has the most comparable action to ephedrine. This is understandable, because it differs from ephedrine by only one methyl group on the amino-N, as shown below:

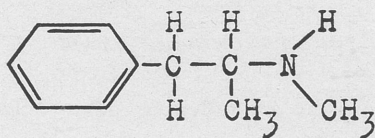


Another widely known product is amphetamine or benzedrine. In Figure 2, it can be noted that it has a long duration of action; and it is effective by mouth—due to the protective action of the methyl group on



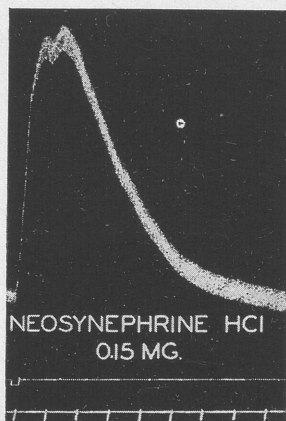
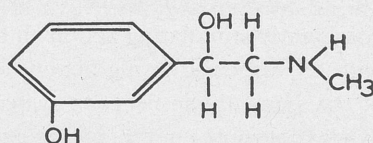
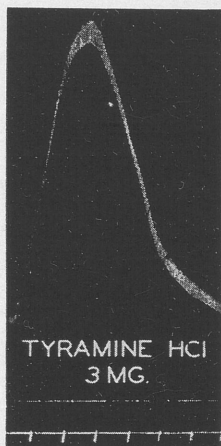
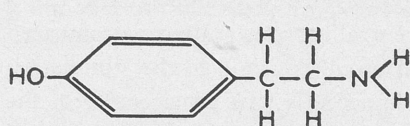
the α -C-atom from amine oxidase. Unfortunately, amphetamine has a dominantly stimulating action on the central nervous system, as compared with its vasoconstricting action, which owes its origin to the stimulation of the sympathetic nervous system. Apparently, the replacement of the hydroxy group on the β -C-atom by a H-atom results in a compound having a special affinity for the brain and spinal cord (Figure 2). During World War II, the stimulating action of amphetamine on the central nervous system certainly justified its purpose, for General Grant¹⁵ of the American Air Force said, "Benzedrine is the most satisfactory drug now available for temporarily postponing sleep when the desire for sleep endangers the security of a mission." On the other hand, amphetamine is becoming less and less desirable in otolaryngology in view of the central stimulation, addition liabilities,¹⁶ and relatively high toxicity.^{17,18,19,20,21}

The Germans during World War II used pervitin, known in this country as desoxyephedrine, for alerting purposes in their air corps. If the following formula is examined, the close similarity of its structure to that of amphetamine is obvious:



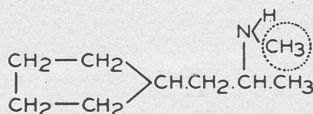
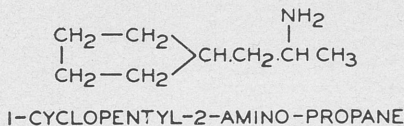
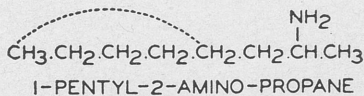
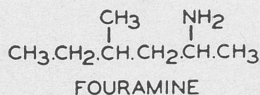
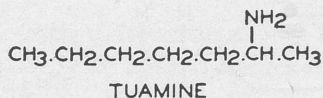
The disappearance of the hydroxy group on the β -C-atom again confers a marked degree of central stimulation.

In search for newer vasoconstrictors, the analogues of epinephrine were not forgotten. The two phenolic hydroxy groups add instability and bring about rapid destruction by phenolase and other enzymatic oxidation systems.^{22,23,24} Even a single phenolic hydroxy group, as in the case of tyramine and neo-synephrine (Figure 3), cuts short the vasoconstricting action, and nullifies the systemic effects after oral administration. Paredrine belongs to the same class. It thus appears clear that in spite of the numerous aromatic amines synthesized, there are very few which can be truly called good vasoconstrictors.



Chemical studies were extended to other fields by synthesis.^{25,26} It was soon observed that the benzene ring is unnecessary; in fact, amines of the straight chain series have the same type of vasoconstriction. These are known chemically as aliphatic amines. Here again, a methyl group on the α -C-atom confers a prolonged action since it protects the compound from oxidation by amine oxidase.²⁷ One of the potent compounds, tuamine, has a length of 7 C-atoms. Its clinical possibilities were explored by Proetz.^{1,28} If the methyl group at the end is transferred to C₄, the resulting compound, fouramine, is 13% more active than tuamine. Neither product is effective by mouth, nor stimulates the central nervous system in therapeutic doses.

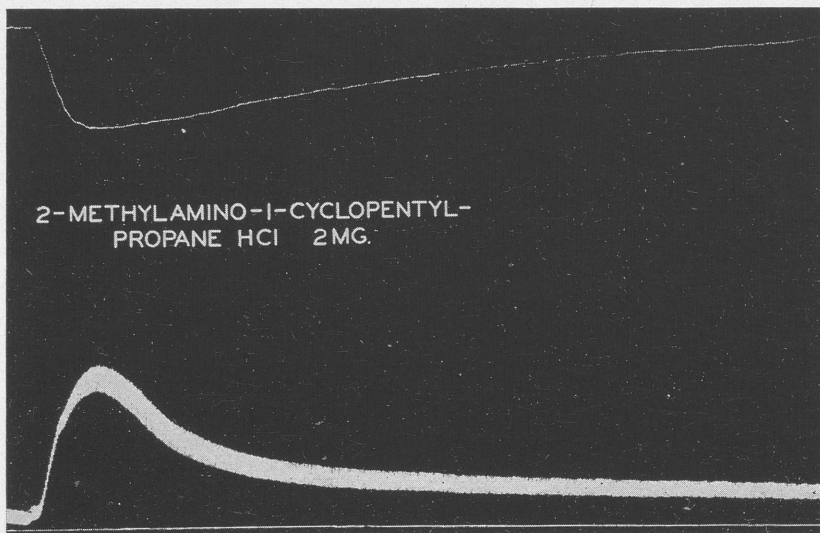
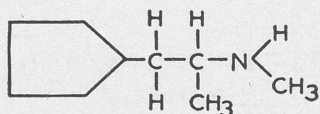
Compounds of shorter or longer chains are invariably less active. Thus, one of 8 C-atoms has only 1/6 the activity of tuamine. For sake of clearness, the following formulas are listed:



I-CYCLOPENTYL-2-METHYLAMINO-PROPANE (NO.02040)

If the terminal C-atom joins the fifth C-atom to form a ring, as indicated, the product approaches fouramine in activity as measured by the blood pressure response. Further, if a methyl group is introduced to the amino group, the resulting substance, 1-cyclopentyl-2-methylamino-propane, becomes 56% more active than fouramine. Judged by peak of action, it is more potent than ephedrine. As yet, no simple name has been assigned to the compound, and for brevity it is known to us by its serial number 02040.

A sample experiment on blood pressure is shown in Figure 4. No. 02040 has the same duration of action as ephedrine. When given by mouth, it is more easily absorbed than ephedrine.²⁹ A rise of blood pressure is detectable in human subjects with an oral dose of 50 mg. of the hydrochloride. It does not cause immediate inhibition of the ciliary



movements of the rabbit's isolate trachea, as studied by the Proetz technique.^{30,31} Table 1 shows its comparison with ephedrine, tuamine, and saline control.

Compound No. 02040 has a very slight stimulating action on the central nervous system. The activity curves of rats indicate that it is 1/40 as active as amphetamine recorded by the modification³² of the spring-suspended cage technique originally designed by Schulte and associates.³³ Ephedrine studied under the same conditions is 1/10 as active as amphetamine on the central nervous system. It appears from our laboratory data that No. 02040 is better all-around than other vasoconstrictors of either the aromatic or aliphatic series.

Compound No. 02040 dilates the rabbit's pupil when instilled in the eye. This is to be expected, because any sympathomimetic amine stimulates the endings of radial dilator fibers. Mydriasis also occurs in

Table 1. Action of Vasoconstrictors on the Ciliary
Activity of the Isolated Rabbit's Trachea

Drug	Concen- tration	Number of Specimens Studied	Mean* Duration of Ciliary Activity	
	percent		hours	minutes
Ephedrine Sulfate	0.5	6	5	38
	1.0	6	3	53
	2.0	6	2	53
Tuamine Sulfate	0.5	6	3	19
	1.0	6	3	54
	2.0	6	1	19
1-Cyclopentyl-2- methylamino-propane Hydrochloride	0.5	6	3	43
	1.0	6	2	36
	2.0	6	1	24
Sodium Chloride	0.9	12	17	48

* This is the geometrical mean. According to statistical principles, the geometrical mean, which is the antilogarithm of the average of logarithms of individual readings, is more reliable than the arithmetic mean.

man if a 5% solution of the hydrochloride is dropped into the eye, with no cycloplegia. There is a racial difference of response, however, the pupil of the Caucasian dilates, that of the negro is refractory, and that of the Chinese reacts slightly—exactly similar to cocaine or ephedrine, as far as the selective action on the human eye is concerned.³⁴

For over 20 years, vasoconstrictors have been directly applied to the human nose in a great variety of pharmaceutical forms. There is a trend that they are used altogether too frequently. Certain patients actually spray their own noses every few minutes in spite of the physician's warning. This sort of self-medication defeats the very purpose of modern therapy. In the first place, all the long-acting vasoconstrictors show tachyphylaxis, namely, they produce very little response if they are repeated

before the effects of a previous dose wear off. Secondly, incessant administration of these nose preparations results in absorption of the active ingredients with the occurrence of side reactions due to central stimulation, such as, nervousness, a jittery feeling, and insomnia. Thirdly, too frequent medication with vasoconstrictors may constitute a predisposing factor of middle ear complications. Such dangers have been emphasized by Kully.³⁵

In 1926-27, Gaarde and Maytum^{36,37} treated ragweed hay fever patients with ephedrine by mouth, and they observed a decrease of nasal secretion. They also stated that ephedrine as a nasal spray was less efficacious, and the relief was of shorter duration. Since their publications, I have personally confirmed their results from season to season—namely, by oral use of the drug only. In other words, vasoconstriction of the nose can be accomplished by the systemic effect of ephedrine. This may apply to other drugs, including No. 02040. I submit this proposition to you for your consideration, because I believe that if these drugs are prescribed by mouth twice or three times a day, better results may be forthcoming in otolaryngology.

During the last 2 years, there has been increasing evidence that allergic rhinitis favorably responds to antihistaminic drugs. The question arises as to which type of drug is the more effective—vasoconstrictors or antihistaminics. The answer will not be available until some carefully controlled comparisons have been made. From a pharmacologic point of view, there may be a synergism between the two—one by sympathetic stimulation, and the other by neutralization of histamine, a product of the allergic reaction. It is not impossible to visualize that simultaneous administration of both types of drugs, and by mouth, will give rise to better results in such conditions, than with either alone. The correction of wakefulness, which is inherent to some vasoconstrictors, by the sedative action of antihistaminics, would favor such a combination. A preliminary report³⁸ seems to substantiate this contention.

Acknowledgment. The author is deeply indebted to Mr. E. E. Swanson, Dr. Francis G. Henderson, and Dr. Paul N. Harris for assistance in the preparation of this manuscript. Thanks are also due to Miss Marian H. Ellaby and Mr. Harold M. Worth for the preparation of the illustrations.

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DISCUSSION

ERNEST M. SEYDELL, M.D.: I should like to ask one question: what about the habit producing effects of ephedrine by mouth in comparison to its use when taken by nose?

MARVIN F. JONES, M.D.: Will you add to that the dosage?

BERNARD J. McMAHON, M.D.: May I ask Dr. Chen whether or not the barbiturates used in conjunction with ephedrine by mouth would have an antagonistic or synergistic action on the latter?

THOMAS C. GALLOWAY, M.D.: Does the continued use of vasoconstrictors produce the same kind of rebound swelling or turgescence if taken by mouth?

OLIVER E. VAN ALYEA, M.D.: I wonder if Dr. Chen would care to discuss the dependency action that one of the sympathomimetic drugs seems to have upon the others. He mentioned several of these, yet I don't think he mentioned prinine hydrochloride for which there seems to be more of a dependency. It is more difficult to be relieved from the habit than it is from the others. Why? Also, would he care to talk about or give us his ideas as to whether or not he thinks there is any

justification for the Federal Government label required on nose drops of the vasoconstricting type which requires a warning for those individuals suffering from heart disease, thyroid disorders, diabetes mellitus and hypertension? The warning states that these individuals should not use vasoconstricting drugs without careful supervision. Do you think that label is justified?

K. K. CHEN, M. D.: (Closing) Thank you for the discussion! I will make my replies brief.

There is no evidence that ephedrine is a habit-forming drug by either local application or oral administration. Occasionally, however, a few individuals with a constitutional inferiority may develop psychic dependence on ephedrine, just as they do with any other drug or agent. This must not be mistaken as habit-formation, because it involves no tolerance and physical dependence.

Regarding the dose of ephedrine by mouth, I would say that for adults it should be 25 mg. (gr. 3/8) twice or three times a day; for children over ten years of age, 12 mg. (gr. 1/5); and for children older than five years, 8 mg. (gr. 1/8).

In answer to the question about the possible damage to the blood by antihistaminic agents, I will say this. Any new drug before being placed on the market must be approved by the Food and Drug Administration, based on the pharmacologic and clinical data submitted by the manufacturer. An application for such an approval almost always includes the results of chronic toxicity studies; and blood examination is usually part of the experiments. This unquestionably holds true with antihistaminic drugs. As far as our laboratory is concerned, we have not noticed any change in blood cells and chemical constituents following the administration of "Histadyl" (Thenylpyramine, Lilly).

The combination of a barbiturate with ephedrine is a good medication at bedtime, because the hypnotic action of the former neutralizes the stimulating action of the later. The combination of an antihistaminic drug with ephedrine accomplishes the same purpose, since all the antihistaminic drugs now on the market are known to have various degrees of sedative action. No barbiturate needs to be added in this case.

Privine is chemically 2-(7-naphthyl-methyl) imidazoline. It is neither an aromatic nor an aliphatic amine. This accounts for my not mentioning it in my presentation. Unless you gentlemen publish your results, and make a plea to the Food and Drug Administration for a special label, "Warning, may be habit-forming," the drug will remain at large—free for self-medication among the laity.

OBSERVATIONS BASED ON AN EXPERIENCE OF TWENTY-EIGHT YEARS WITH LARYNGEAL TUBERCULOSIS

EDWARD ANDERSON LOOPER, M.D.

BALTIMORE, MARYLAND

and

I. B. LYON, M.D. (*By Invitation*)

STATE SANATORIUM, MARYLAND

Laryngeal tuberculosis is the most common chronic disease of the larynx. The importance of this condition is not always appreciated by the average laryngologist unless he is associated with a sanatorium for the treatment of tuberculosis. This paper is of a practical nature, representing an opinion based on twenty-eight years' experience in the study of laryngo-pulmonary diseases.

In 1928, we¹ made a study of all cases admitted to the Maryland State Sanatorium from August, 1923 to February, 1928. In this group 3,227 patients suffering from tuberculosis were treated. Of these, 500 or 15.5 per cent of the group showed laryngeal involvement.

We thought it might be of interest to make a recent survey of cases to compare the earlier results with our present findings. Therefore, we have analyzed the patients entering the sanatorium over a ten year period, namely, from January 1, 1938 to January 1, 1948.

During this period 5,238 patients were admitted to the Maryland State Sanatorium. The larynx was examined in 2,770 cases; it was not examined in 2,468 patients because of an absence of symptoms, a short stay in the hospital, and various other reasons. Of the 2,770 patients examined, tuberculosis involvement of the larynx was found in 214 cases, an incidence of 7.7 per cent. This compares more favorably than in the previous report¹, where in the period between 1923 and 1928 the complication was found in 15.5 per cent of all patients.

The decrease in the manifestation of this serious complication is encouraging and is evidently not confined to this locality alone, for Myerson³ reported an incidence of 10.6 per cent in a study of 10,657 cases in

1944. This improved condition cannot be explained by the type of pulmonary cases admitted to the sanatorium because in the period from 1923 to 1928 less than 50 per cent of the patients had advanced lesions, whereas in the past few years more than 50 per cent of the admissions showed far advanced lung lesions. Education has undoubtedly played an important part in reducing the incidence of this complication. In teaching medical students and other members of the medical profession as well, it is advisable to stress the importance of having a laryngeal examination made as soon as a patient is found to have pulmonary tuberculosis.

Since laryngeal complications are always secondary to pulmonary involvement, many cases showing beginning pathologic changes can be recognized and treated in the early stages. These cases are easy to cure if detected before ulcerative changes develop. Furthermore, tuberculosis changes often develop within the silent area of the larynx and do not present subjective symptoms.

The intra-arytenoid space, arytenoids, vocal processes, and false cords may have advanced pathologic changes before hoarseness, pain, difficulty in swallowing, or other subjective symptoms occur. An early examination of the larynx with a laryngeal mirror frequently reveals beginning pathologic changes which can be arrested, thus preventing serious complications. Consequently, prophylaxis here is one of the most valuable factors in contributing to improvement in the pulmonary disease, because no patient can recover if he has dysphagia, hoarseness and the cough which inevitably develops if the local laryngeal lesion is unrecognized and untreated.

The number of deaths from tuberculosis has been reduced greatly in the past ten years. For a number of years tuberculosis was the second greatest cause of death but the mortality rate for the past few years has been less than fifty per 100,000. The lessening of the mortality rate no doubt coincides with the reduced number of laryngeal complications. This no doubt shows that by education and early diagnosis many of the patients are able to build up better resistance to tuberculosis, therefore complications are not so common. Improved general treatment, proper diet, and recognition by the medical profession of the handicaps imposed by possible complications are all factors which favorably influence the results in these cases.

Pulmonary collapse in selected cases of pulmonary tuberculosis has been one of the greatest advances in the treatment of this disease. It has also reduced laryngeal complications to a considerable degree. Many be-

lieve that infected sputum brought up in contact with the laryngeal structures is the most important etiologic factor in causing the development of laryngeal lesions. Sputum coughed up and lying in contact with the moving mucosal structures of the posterior pharyngeal wall invades the mucosa, with resulting pathologic changes. Accordingly, if the lung is collapsed and the supply of infected exudate is reduced, the laryngeal involvement becomes less, and certainly the severe cases are prevented. Collapse therapy, therefore has had a most helpful effect in reducing laryngeal complications.

Diagnosis. The diagnosis of laryngeal tuberculosis is not difficult to those associated with tuberculosis sanatoria. However, to the average laryngologist the problem may at times be difficult.

It is now generally admitted that laryngeal tuberculosis is secondary to some pulmonary infection; consequently, sanatorium patients who develop voice changes, readily suggest laryngeal complications. In routine office work, however, the possibility of not making a diagnosis in such cases is more difficult. Hoarseness is usually the first and the most common symptom. This may range from a weakness or huskiness of the voice to complete aphonia, depending upon the pathologic condition. As destructive changes develop, the symptoms are aggravated and there may be dysphonia.

Pain referred to the ear is one of the earliest subjective symptoms. This is a reflex otalgia referred from the superior laryngeal nerve through branches of the vagus.

A cough is usually present. Exudate that is brought up irritates the superficial sensory nerves even though ulcerative areas have not developed. In more destructive ulceration, the act of coughing may be intense and exasperating.

Pain on swallowing is most often seen in lesions of the epiglottis and the arytenoids. Even emollient solutions such as milk may cause intense pain on swallowing if there is much destruction of the mucosa of these structures.

Some patients with edema or infiltration in the glottic airway may develop dyspnea. In a few cases a tracheotomy may be necessary to relieve the symptoms. In our experience, however, a tracheotomy has been required in only two cases.

Predisposition. Men were affected much more frequently than women. In the group reported here 163 or 76 per cent were men, and 51

or 25 per cent were women. Evidently the reason males appeared more susceptible is the use of tobacco and alcohol and exposure to more strenuous occupations than females.

The onset of laryngeal involvement is most frequently between the ages of thirty and fifty in men and between twenty and forty in females; however it may occur in the extremes of life. Our youngest patient was eighteen years of age and the oldest sixty-eight.

Occupation. The relation of occupation to this complication is shown in Table 2. It is interesting to note that although there were a number of voice users in our group, such as ministers and singers who were affected with pulmonary tuberculosis, not one of them showed laryngeal lesions. In other words, the use of the voice seemed to play no part in the development of the complication.

Table 1. Age Distribution

Age	Males	Females	Total
10-20	3	2	5
20-30	17	18	35
30-40	48	18	66
40-50	51	10	61
50-60	35	1	36
60-70	9	2	11
Total	163	51	214

Table 2. Relation Of Occupation To Laryngeal Tuberculosis

Group	Occupation	Number of Cases
1.	Physicians, Dentists, Nurses, etc.	5
2.	Students, Teachers	0
3.	Salesmen, Saleswomen	5
4.	Dusty Occupations	45
5.	Open-Air Occupations	78
6.	Clerks, Stenographers	27
7.	Housewives	31
8.	Sedentary Occupations	18
9.	Voice Users	0
10.	No Occupations	2
	Total	214

Laborers, motormen, farmers and other workers engaged in outdoor occupations showed a greater number of complications than the group of painters, printers, barbers or those who were engaged in some dusty or irritating occupation. This would offer evidence that laryngeal tuberculosis is not a primary lesion but is secondary to the pulmonary condition.

Differential Diagnosis. It is at times difficult to differentiate between cases of catarrhal laryngitis and early tuberculosis. As a rule, in patients with catarrh there is some infection found in the sinuses, teeth or tonsils which produces a uniform redness of the mucosa of the pharynx and larynx, and the inflammation clears up when the infected areas are given attention; in tuberculosis there is usually some irregularity in the laryngeal injection and infiltration.

Syphilis has not been common in this series and when present is not difficult to differentiate from tuberculosis since the serologic test for syphilis and general symptoms confirm the diagnosis.

Carcinoma often presents a serious problem in diagnosis. Male patients who are over forty-five years of age and are being treated for pulmonary tuberculosis frequently develop chronic lesions on the vocal cords which require considerable observation and study to determine their true nature. In many cases it is necessary to resort to biopsy for a final diagnosis.

Regardless of the number of patients a surgeon has seen, one can never be too sure about the diagnosis. In classes held at the Looper Clinic two patients were presented with extensive pathologic changes in the larynx. The diagnosis of tuberculosis was made in one and of cancer in the other. However, when biopsies were made, it was found that both diagnoses were incorrect. This emphasizes the importance of biopsy in questionable cases.

Pathology. The anatomic construction of the posterior laryngeal wall makes it susceptible to invasion by tubercle bacilli. The loose folds of mucous membrane which are in almost constant motion become covered with tubercle-laden sputum. The epithelium is easily irritated and eroded, so that organisms can enter the underlying tissue and tubercles develop. The nature and extent of involvement depend upon the patient's resistance and course of treatment.

The one characteristic feature of the pathologic changes in laryngeal tuberculosis is the lack of uniformity of the lesion or its location in the

larynx. The lesion may be inflammatory, infiltrative, edematous, or of the chronic tuberculomatous types.

However, it is unnecessary in this type of report to go into the detailed pathology of this subject. It has been ably described in many textbooks, especially by Myerson² in his excellent book.

Table 3 shows the location of the lesions found in our 214 cases. As illustrated in this table, lesions of the vocal cords were most frequent, with seventy-one cases. Involvement of the vocal cords and arytenoids was second, with twenty-six cases. Many patients were admitted in a critical condition with obvious widespread involvement of the larynx, and proper and thorough examination could not be made.

Table 3. Location Of Lesions

<i>Group</i>	<i>Location Of Lesions</i>	<i>Number of Cases</i>
1.	Vocal Cords	71
2.	Vocal Cords and Arytenoids	26
3.	Epiglottis, Arytenoids and Aryteno- Epiglottidean Folds	17
4.	Arytenoids Only	17
5.	Epiglottis and Vocal Cords	17
6.	Epiglottis Only	14
7.	Widespread Laryngeal Involvement	11
8.	Location Not Stated	11
9.	Epiglottis, Vocal Cords and Arytenoids	9
10.	Tongue, Without Laryngeal Lesion	9
11.	Tongue, Tonsils With Laryngeal Lesion	5
12.	Posterior Wall and Interarytenoid Sulcus	3
13.	Palate, Without Laryngeal Lesion	2
14.	Tonsils, Without Laryngeal Lesion	2
Total		214

The sputum examination is shown in Table 4. In the moderately advanced group, the sputum was positive for tubercle bacilli in 85.7 per cent and negative in 14.3 per cent. In the far advanced group, 99 per cent had positive sputum, and in only 1 per cent was it negative.

Table 4. Sputum Examinations

<i>Extent of Pulmonary Lesion</i>	<i>Positive</i>	<i>Negative</i>	<i>Total</i>
Minimal		2	2
Moderately Advanced	12	2	14
Far Advanced	196	2	198
Total	208	6	214

Prophylaxis. Many cases of laryngeal tuberculosis can be prevented; undoubtedly, our most promising work can be accomplished along this line. It is the duty of laryngologists to instruct the family physician in the earlier recognition of these cases. He should be advised of the importance of routine laryngeal examination in every case of pulmonary tuberculosis. Since so much good has been accomplished by educational measures in the recognition and treatment of pulmonary tuberculosis, the same methods should be applied with reference to the complications. Everyone should know that this serious disease is not as hopeless as is generally represented, but that most cases can be cured if recognized in the early stages.

All possible conservative measures should be directed to the correction of pathologic conditions in the upper respiratory tract. Infected sinuses, diseased teeth, and focal infections in the tonsils should be given every possible attention.

It is well known that constant absorption of toxins from these structures seriously affects a normal person: in a patient suffering from tuberculosis, the effects are more dangerous because the system is already overloaded with septic material and any extra handicap may be that is necessary to overcome the chances of recovery.

Furthermore, irritating purulent secretion has a deleterious action on normal laryngeal mucosa and may be sufficient to injure the epithelium so that invasion by tubercle bacilli is inevitable. Serious consideration should therefore be given to all factors that may have a bearing on the prevention of this complication.

Treatment. The treatment of laryngeal tuberculosis should be considered under three divisions, namely, general treatment, vocal rest, and treatment of the local lesion.

The general treatment of the pulmonary disease is the most important of all because, as Dworetzky³ has pointed out, regardless of the type of laryngeal lesion, slight or extensive, there will be little improve-

ment if the patient has a rapidly progressive caseous type of lesion with much exudate. Therefore, every effort should be made to arrest the lung lesion. Rest, good food, and the usual hygienic measures of institutional treatment should be applied. Pulmonary collapse has been effective in controlling many cases and has certainly been one of the outstanding factors in arresting and preventing the extension of the laryngeal lesion.

All patients should be treated in a sanatorium, if possible, because a carefully regulated plan of general treatment of the pulmonary lesion is most necessary. It is unwise to have patients with active tuberculosis travel long distances to physicians' offices and wait in hot, crowded rooms for treatment. Under such conditions coughing is increased and the strain of travel lowers the patient's general resistance. These unfavorable features are eliminated in sanatorium treatment and the cases can be carefully followed by both the clinician and the laryngologist, between whom there should be the closest cooperation.

At the Maryland State Sanatorium, if the patient's temperature is over 99.4°F. and laryngeal complications are found, he is put to bed and kept on vocal rest. As soon as the resistance is built up somewhat and the temperature begins to subside, cauterizations are started and continued at monthly intervals until the patient is cured.

Vocal Rest. In all patients in whom tuberculous invasion of the larynx has been discovered, absolute rest of the voice should be instituted. There is little to be gained in telling the patient not to talk; he should be instructed to write everything. Such instructions impress him with the seriousness of his condition and are also important as a curative measure. Unquestionably, many cases have been cured by silence alone, as has been frequently pointed out, so that whatever other form of treatment may be supplemented, vocal rest should be carried out diligently.

Local Lesion. For active treatment of the local lesion we have found the judicious use of electric cautery most beneficial and now employ it in preference to all other methods of treatment. The results have been most gratifying.

The technic of cauterization is simple, the results are sure, and complications are negligible. Cauterization can be carried out easily in an office or at the bedside without inconvenience to the patient. Local anesthesia is employed and the indirect method is advisable; however, the direct method may be used when desired. Early lesions clear up after two or three treatments, and advanced cases will show definite improvement after a few cauterizations. Even in severe and hopeless cases the

cautery is of great value as a palliative measure to relieve pain and coughing.

In only three cases was it necessary to inject or resect the superior laryngeal nerve. These patients received temporary relief from intractable pain but eventually died.

Fourteen patients were treated by sulfanilamide powder insufflation which was used for the relief of pain in cases of widespread laryngeal destruction. Relief was definitely obtained in six cases, the pain being reduced and the exudate diminished. Two cases were unimproved, and six patients died.

Table 5 shows:

1. Only two cases of laryngeal tuberculosis occurred with minimal lung involvement. One of these was cauterized and improved; the other did not require cauterization and improved.

2. In the fourteen patients with moderately advanced pulmonary disease, the throat was cauterized in ten or 72 per cent of the cases, and not cauterized in four or 28 per cent of the cases.

- (a) Patients whose throats were cauterized; Seven patients or 50 per cent were improved, and three patients or 21.4 per cent were unimproved. There were no deaths in this group.

- (b) Patients whose throats were not cauterized; Four or 28.6 per cent were improved under ordinary sanatorium care, including vocal rest or absolute silence. There were no unimproved or fatal cases in this group.

3. Of the 198 patients with far advanced lung involvement, the throats were cauterized in 115 or 58.1 per cent, and were not cauterized in eighty-three or 41.9 per cent.

- (a) Patients whose throats were cauterized; Sixty-nine or 34.8 per cent were improved and healed; twelve or 6.1 per cent were unimproved, twenty-nine or 14.6 per cent died. In this last group the cauterization was done more for a psychic reason or temporarily to relieve severe pain associated with swallowing, particularly in patients with involvement of the epiglottis. The outlook of this group was absolutely hopeless on admission.

- (b) Patients whose throats were not cauterized; Thirteen were improved and healed under ordinary sanatorium care, including vocal rest or absolute silence; twenty-one or 10.6 per cent were unimproved, forty-eight or 24.3 per cent died. Three per cent of the far

advanced cases left the institution before a follow-up report could be made on the laryngeal lesions.

The rather large percentage of unimproved patients and deaths in the group whose throats were not cauterized can easily be explained by the fact that the majority of patients with far advanced disease are toxic. In them the disease runs an active course, there is a septic temperature, and cautery treatment is contraindicated.

Of the forty-eight who died without having the throat cauterized, the majority were in the sanatorium for only brief periods of time. The comparative results of those whose throats were cauterized with those whose throats were not cauterized are rather striking. It was definitely noted in far advanced cases, where the prognosis was always grave, that the cautery exercised a favorable influence on the lungs and on the general condition in many patients.

The laryngeal lesion is essentially a cellular infiltration. This infiltration is avascular, with a tendency to rapid destruction of the central cells. The beneficial action of the cautery is attributable more to the revitalization of the tissues by induced hyperemia than to destructive action of the heat. The development of granulation tissue and the formation of new blood vessels following the sloughing of the cauterization area account for the good results. The great advantage of using electrocautery is that its action is limited to the diseased area.

Since electrocautery will in almost every instance accomplish good results it should be considered the best method for the treatment of laryngeal tuberculosis. The treatment should be undertaken only in patients in whom there is no fever or just a slight elevation of temperature, except in patients with urgent symptoms that demand intervention.

Every case should be studied carefully for indiscriminate cauterization will not accomplish results. It is most important that there be the closest cooperation between the internist and the laryngologist when treating these cases. A patient who is admitted with fulminating general symptoms and elevated temperature should be kept in bed and the general resistance built up before cauterizations are started, for if there is not sufficient resistance to overcome some of the general effects one cannot expect the laryngeal lesion to be cured by any form of treatment.

However, we feel that in any reasonably early case chances of cure are good. The prognosis of laryngeal tuberculosis should be more promising and the percentage of the complicated cases grow less

as times goes on. We should be able to get these patients in time for effective treatment, for this will also help to reduce the general mortality of tuberculosis.

In the past few years there has been a definite reduction in the number of laryngeal lesions requiring cauterization. This is evidently the result of education and earlier recognition of these cases, better general treatment, collapse lung therapy, and a lessened incidence in the number of these cases. Our latest report showed only 7.7 per cent with laryngeal complications.

The use of streptomycin and the sulfonilamides have proved so satisfactory that their importance cannot be overemphasized in the treatment and prevention of this complication and no doubt as their general use is more employed, the incidence will be further reduced. Such treatment has undoubtedly played a part in the reduction of cases requiring cauterization.

In the earlier work at the sanatorium, it was not unusual to cauterize from twenty to twenty-five patients per month. At the present time, the average runs from two to five cases during the same period. This is very encouraging as it shows that these serious laryngeal complications are under control and there is great promise of continued improvement.

Table 5

Patients Treated Between January 1938 and January 1948

<i>Throats Cauterized</i>					
<i>Extent of Pulmonary Lesion</i>	<i>Improved & Healed</i>	<i>Unim- proved</i>	<i>Died</i>	<i>No Report</i>	<i>Total</i>
Minimal	1				1
Moderately					
Advanced	7	3			10
Far					
Advanced	69	12	29	5	115

Throats Not Cauterized

Minimal	1				1
Moderately					
Advanced	4				4
Far					
Advanced	13	21	48	1	83
					<hr/>
					Total 214

SUMMARY

1. During the past twenty-five years much progress has been made in education, diagnosis and the treatment of tuberculosis.

As laryngeal tuberculosis is always secondary to lung involvement, the subject should be treated as a laryngo-pulmonary infection. No laryngeal complication can be cured if the patient has a soft exudative lung lesion, with little resistance to overcome the general infection; therefore, every effort should first be directed to the treatment of the general disease.

2. Pulmonary collapse has been a marked factor in reducing laryngeal complications as well as its beneficial affects on the lung lesion. These procedures lessen the chances of infected sputum being brought up and deposited on the laryngeal mucosa to cause contact invasion of tubercle bacilli.

3. Infected sputum is the most common cause of laryngeal tuberculosis, but organisms may enter by way of the lymph and the blood streams.

4. A thorough examination of the larynx should be made in all cases of tuberculosis, as soon as pulmonary lesion has been discovered.

5. Slight congestion, dryness and tickling sensations of the larynx should promptly receive attention, as such symptoms often indicate early invasion of the larynx.

6. Laryngeal tuberculosis occurs at all ages, but the majority of cases are found between 20 and 50 years of age with males predominating.

7. Laryngeal tuberculosis has no relation to occupation.

8. All pathologic conditions in the upper respiratory tract, such as deviated septa, sinusitis, hypertrophied turbinates and infected tonsils should be corrected.

9. Non-tuberculous laryngitis, colds, tonsillitis and excessive coughs in tuberculous patients should receive prompt attention, as conditions leading to superficial loss of epithelium open the way to infection by the sputum.

10. The frequency of involvement of various parts of the larynx is directly proportional to exposure, irritation and functional activity.

11. The use of drugs in the treatment of laryngeal tuberculosis has in the past been very unsatisfactory. Caustics and irritants have been discarded and the use of chaulmugra oil and sunlight is of doubtful value. However, since the discovery and use of the sulphonamides and streptomycin in particular, a new era in the treatment of laryngeal tuberculosis is developing. As the use of streptomycin becomes more general, the reports of good results continue to increase and it is encouraging to look to future progress in this field.

12. In selected cases, we still continue to use the electric cautery as our results in the past have been very satisfactory. In recent years, there has been a very noticeable reduction in patients requiring cauterization.

13. Of all our patients with laryngeal lesions with moderate lung involvement treated by electrocautery, 50% were improved and healed, and 58.1% with far advanced lung involvement were improved and healed.

14. Cautery treatment in many patients exercised a favorable influence on the lungs and on the general condition. No bad results were encountered.

15. Cauterization in all patients has been done under local anesthesia by the indirect method at about monthly intervals.

16. Results obtained by electric cautery treatment indicate without doubt that tuberculosis of the larynx is curable, if started reasonably early.

17. In all suspicious cases we advise vocal rest or absolute silence. While active laryngeal tuberculosis does not have any tendency toward spontaneous healing, it is surprising with what rapidity some incipient cases with slight infiltration will clear up as the result of absolute silence. Therefore regardless of whatever method of treatment is added, silence is imperative in all cases.

18. We still feel that the electric cautery continues to be the best method of treatment of laryngeal tuberculosis at the present time.

19. The revitalization of the tissues by induced hyperemia and the development of granulation tissue with formation of new blood vessels account for the good results obtained by the cautery.

20. Elevation of temperature (high degrees), marked asthenia and high blood pressure are contraindications in using the cautery, although in patients in whom serious symptoms (severe pain, difficulties of swallowing) demanded alleviation, we did use the cautery to bring temporary relief.

21. In using the electrocautery, one thing should be particularly and strongly emphasized and this is the condition of the lungs. No laryngologist should undertake electrocautery treatment without cooperating with or being guided by a clinician. Indiscriminate use of the cautery in unsuitable cases will do more harm than good and the method of treatment itself will be brought into disrepute.

22. It is encouraging to note that the number of cases requiring cauterization in the past few years have been steadily diminishing. When I first started at the State Sanatorium twenty-eight years ago, from twenty to thirty cases per month required this treatment, while today only four or five per month are cauterized.

23. This is probably due to the smaller number of cases found.

24. In our recent survey, we found an incidence of only 7.7% of laryngeal complications, compared with 15.5% of our cases in 1928. Dworetzky (4) found an incidence of 25.6% in 1914. This shows how much less these complications have become. This holds much promise for the future and we can be optimistic for continued progress in preventing these complications.

DISCUSSION

BURT R. SHURLY, M.D.: Mr. President, it is certainly a joy to have lived long enough to be able to report and see the tremendous improvement in the treatment of laryngeal tuberculosis over the older days.

I have been doing this work for fifty years, and our difficulties were great. We had in our sanatorium 15 per cent of laryngeal cases. This apparently has dropped in recent years to about 8 per cent. We went through the various therapeutic hopes of the earlier days with lactic acid, with formalin and with the galvano cautery applications, which persisted as the best treatment up to the discovery of streptomycin.

At the present time we have treated in our institution more than 100 cases of pulmonary tuberculosis and laryngeal tuberculosis with uniform beneficial results.

It is a joy to practice medicine. We can really do something for the patient and have absolute confidence in ourselves that we are accomplishing a grand and glorious result.

Streptomycin is a drug that has great possibilities and some dangers. When I left Detroit, we had 35 cases taking streptomycin, one gram a day injected round the clock every three hours. We have been through the higher dosage and found that the dangers of higher dosage were quite possible and quite prevalent, and the beginning dizziness, vertigo or a small, slight effect on the auditory nerve must be met by immediate discontinuing of the drug. We found that our auditory cases cleared up but the history over the country shows some very bad damages from streptomycin and that should be guarded against and the dosage should never be over 1 gram a day.

You can see this laryngeal pathology diminish from day to day, or week to week, and cases with aphonia and pain and much ulceration fade away and in our experience the voice has returned. Those cases reported from the Mayo Clinic, a very wonderful report we had, confirm exactly our experience that this is the greatest discovery of the time for laryngeal tuberculosis. I hope that it will be given greater and greater scientific observation and report in this Society.

LOUIS H. CLERF, M.D.: I have been impressed by the fact that phthysiologists generally are optimistic about eradicating tuberculosis but I think they missed opportunities to further their ends. I emphasize particularly the need for phthysiologists to recognize, as Dr. Looper pointed out, that tuberculosis of the larynx may be present with an apparently normal chest. We can accomplish much in the eradication of tuberculosis by the early recognition of cases. I have been impressed with this because I have discovered many cases of pulmonary tuberculosis, previously unrecognized, on the basis of examining a larynx or throat of a patient who complains of indefinite symptoms.

Phthysiologists also fail to recognize that every patient in a sanatorium should have a routine examination of the nose, throat and larynx in addition to routine chest studies. A competent laryngologist should be on the staff and he should examine every larynx routinely, complaints or no complaints. If one waits for patients to complain before examinations are made advanced disease often will be found.

I would suggest to those who write textbooks on disease of the larynx that they realize what the statistics of Dr. Looper indicated. Anatomical predilection of tuberculosis has erroneously crept into text books and has been perpetuated. As Dr. Looper pointed out tuberculosis may attack any part of the larynx, not merely the posterior half, yet textbooks will give you that impression.

FREDERICK A. FIGI, M.D.: This is an extremely valuable and interesting report on a very extensive experience with laryngeal tuberculosis which Dr. Looper has given us. I am sorry, however, that he did not go into the antibiotic therapy of this condition to a greater extent, for this in our experience bears great possibilities.

I have a few slides which I would like to present to show the results of such treatment.

(Slide) On the left is shown the tuberculosis larynx of a housewife 32 years of age. This was a papillary hypertrophic type of laryngeal tuberculosis. The

condition had produced symptoms for a year. This was treated by means of streptomycin, giving a gram in divided dosage every 24 hours parenterally, also from $\frac{1}{2}$ to 1 gram daily by nebulization. We feel that the administration of streptomycin by nebulization is of distinct advantage in this connection. The photograph on the right shows the result at the end of six weeks of treatment. Without further therapy, this patient went on to complete recovery and at the time last observed, five months following the initiation of treatment, she was a perfectly normal individual in every respect as far as could be ascertained by physical examination, roentgenographic study of the chest and detailed laryngeal study. Her larynx was perfectly normal in appearance.

(Slide) This is an extensive tuberculous laryngitis that was encountered in a male, 50 years of age, who had been known to have pulmonary tuberculosis for some years. The laryngeal involvement had been present for more than a year. He had had such extreme dysphagia that at times he had gone for a day or two without taking any nourishment. Merely the drinking of a glass of water was accompanied by extreme pain and he was in a marked state of emaciation at the time he came under observation. In addition, his pulmonary lesion was well advanced.

(Slide) This is another photograph taken at the time of the initial examination.

(Slide) And this is the result following six weeks of treatment with streptomycin. In this instance we gave in addition some penicillin with the nebulized streptomycin. This patient showed very little response as regards the pulmonary condition at this time. However, after six months the pulmonary condition was very markedly improved and when the patient was observed a year subsequently his pulmonary process was entirely quiescent and his laryngeal condition looked perfectly normal. The possibilities of these antibiotics in the treatment of laryngeal tuberculosis far exceed the results obtainable with the older methods of treatment.

HAROLD M. HICKEY, M.D.: Mr. Chairman, I have enjoyed Dr. Looper's presentation very much.

My experience with laryngeal tuberculosis has not been quite so extensive as Dr. Looper's but it does go back to a period immediately after the First World War when it was my good fortune to take care of a good many ex-servicemen who developed tuberculosis. Many of them came from the East and Middle West out to Colorado and we saw them in all stages. We too have noticed a very marked decrease in the number of laryngeal cases in recent years. Out of those having pulmonary lesions, where before it was a rather easy matter to get cases of laryngeal tuberculosis for demonstration to our students, now in our institutions which still have a large tuberculous population, such as Fitzsimmons' General Hospital, we do not often see cases that are well advanced. We do see a fair number of minimal lesions. The use of streptomycin of course has decreased the number of all laryngeal cases.

Dr. Looper mentioned the fact that the care of various sources of focal infection is important in the proper hygiene of a tuberculous patient. I am sure that is true. There is one observation, however, made in those earlier years which

I think might be of value, and that is, in quite a few patients the history of development of laryngeal symptoms, which were later shown to be due to laryngeal tuberculosis, followed the performance of a tonsillectomy on an active or even a quiescent case; this occurred in enough patients so I am sure it was not just an accident. So we made a hard and fast rule not to do a tonsillectomy even under local anesthesia on a patient until he reached the arrested stage.

Then in the matter of vocal rest, this we found to be of a great deal of importance and still feel the same way about it; also the general activity of the body, particularly of the arms, which of course brings into play the pectoral muscles, we feel is of considerable importance, because the fixing of the pectoral muscles has a very definite effect on the function of the glottis. It is not enough just to give a patient a pencil and pad but his general body activity must be restricted as well.

FREDERICK T. HILL, M.D.: No one has called attention to the importance of the consideration of non-tuberculous lesions in the larynx.

Dr. Green some years ago presented a very interesting paper on that phase of the subject. With the decrease in laryngeal symptoms, just as Dr. Looper has said, there has been less call for routine throat examinations.

Interesting enough, I, too, for 28 years have consulted in state sanatoria. We used to see every patient. Now they are only interested in the bronchoscopic examination in the thoracoplasty case.

In point, just a short time ago I was asked to see a patient because of indefinite symptoms in regard to the throat, fullness, soreness, some difficulty in swallowing were present. This was a young man with a very active pulmonary tuberculosis. He had a very marked lingual thyroid. Taking care of the lingual thyroid surgically so relieved his irritation of the throat that his collapse therapy was much more effective afterwards.

I enjoyed Dr. Looper's paper very much.

DANIEL S. CUNNING, M.D.: Mr. Chairman, I would like to ask Dr. Looper and all those present, if they have seen any cases of primary tuberculosis of the larynx.

I examined this particular patient last September and noted a small growth on the middle of his right vocal cord which I suspected to be an early carcinoma. I advised a complete and detailed physical examination such as, chest x-rays, serology, etc. His medical picture proved negative. I then performed a biopsy and it was diagnosed as "tuberculosis." I felt this report to be positively accurate as it was re-examined by three excellent pathologists who confirmed the former findings. The patient was then re-examined by an outstanding medical man who reported, as before, that his chest was negative. Since this time I have been closely observing the patient and after periodic examinations his larynx remains normal. I would be interested to know if any of the gentlemen present have seen a condition of this type.

E. A. LOOPER, M.D. (Closing): I have nothing further to add, except I have never seen a case of primary tuberculosis in the larynx.

MONILIASIS OF THE LARYNX: REPORT OF TWO CASES*

CLAUDE C. CODY, M.D.

HOUSTON, TEXAS

The monilia and blastomyces are classified by Henrici in a transitional group between the true molds and yeasts. Their structural characteristics and cultural reactions seem to vary at different stages of growth so that at one period they may appear to be a mold and at another a yeast. The same organism may be put in varying genera by different mycologists. These variations in classification and identification make the differential diagnosis between moniliasis and blastomycosis rather uncertain and a terminology rich in synonyms adds further to the confusion of nomenclature. However, the specific identifications of these mycotic infections of the larynx are not so important from a practical standpoint, as the clinical pattern of symptoms, physical findings and treatment is much the same for both of them. The importance of the diagnosis of moniliasis of the larynx resides in its differentiation from diseases with a far higher incidence, such as cancer, tuberculosis, syphilis and acute simple laryngitis, rather than in distinguishing it from blastomycosis.

Thrush, or the monilial infection of the mouth, is of relatively common occurrence. Bronchomoniliasis which so often simulates chronic pulmonary tuberculosis, is being recognized with increasing frequency. But moniliasis of the larynx is rarely referred to in the literature. Two cases of monilial infection of the larynx are here reported. In one, an infiltrative granuloma mimics a cancer, and the other may be classified as an acute or subacute laryngitis.

Case I. Mrs. T. D. H., age 48, was referred by Dr. J. Edward Hodges with the complaint of hoarseness. The onset of hoarseness occurred about eight months previously and during the past three months has become progressively worse. Pain in the left ear was first noted four months ago and recently has been increasingly persistent and uncomfortable. Pain on swallowing has been worse during the past month. The previous history referable to the presenting complaint was that a

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diagnosis of chronic pulmonary tuberculosis had been made four years ago, but Dr. Hodges did not agree with this. A persistent cough developed one year ago from chronic bronchitis.

The examination of the larynx demonstrated one tumor on the left vocal cord, another on the inter-arytenoidal mucosa, and no membrane. The left vocal cord was fixed in abduction and a warty tumor, slightly pink occupied its posterior third with a small extension to the inter-arytenoidal mucosa. The tumor on the inter-arytenoidal mucosa was apparently a pedunculated papilloma. The persistent hoarseness, referred pain to the ear, dysphagia, cough and a tumor on a fixed vocal cord as shown in Figure 1, suggested a differential diagnosis between cancer, tuberculosis and syphilis. The radiologist Dr. C. P. Harris, reporting on the x-ray of the chest of which Figure 2 is a copy, stated that both lungs showed broncho-vascular mottling, no tuberculosis, no malignancy, and findings indicating a monilial infection. The clinical pathologist, Dr. C. B. Sanders, reported that the serological tests for syphilis were negative

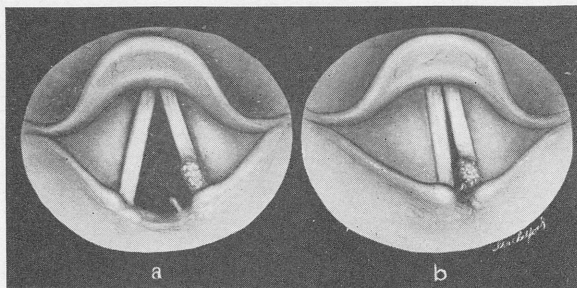


Fig. 1.—Schema of the Larynx. (A) Inspiration (B) Phonation

and that the sputum examination was negative for *B. tuberculosis*, but showed a profuse amount of *monilia albicans*, as illustrated in Figure 3. Figure 3. *Monilia albicans*. Two weeks growth. x-

On the basis of these reports a preliminary diagnosis of cancer of the larynx was made and a biopsy performed for pathological examination. The pathological report on the specimen, Figure 4, was chronic, inflammatory tissue, non-specific with no evidence of malignancy. A second biopsy was done five days after the first to confirm the pathologist report. The report on the second specimen was the same as on the first.

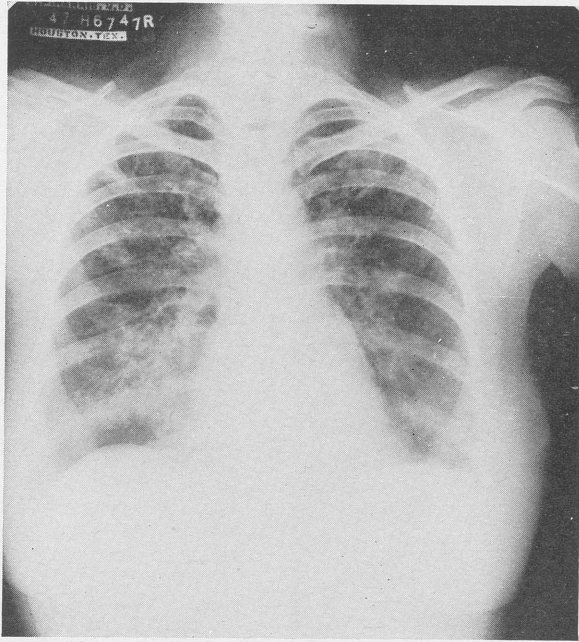


Fig. 2.—Bronchomoniliasis

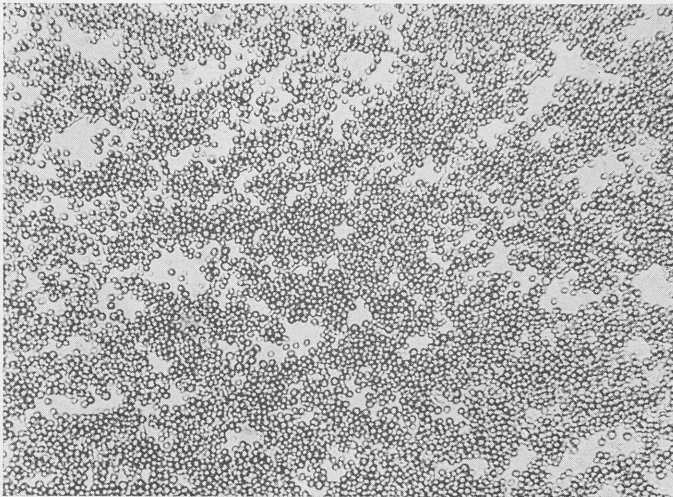


Fig. 3.—*Monilia albicans*. Two weeks growth. x-

The post-operative reaction of the larynx after the second biopsy was quite severe and the infection was uncontrolled by topical treatment. Since streptomycin is effective in tuberculous granuloma of the larynx, it was selected for the treatment of the monilial infiltrative granuloma. Intramuscular injections of streptomycin 1 gram per diem were administered for 3 days. On the fifth day after starting streptomycin, the infection was greatly improved, the left vocal cord was freely mobile, only

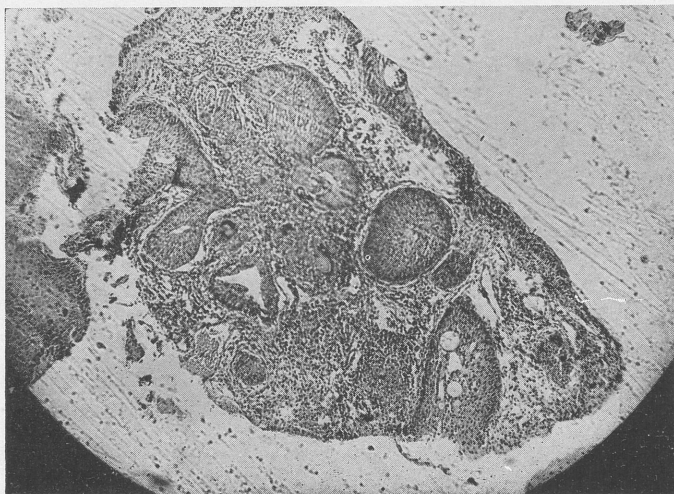


Fig. 4.—Microphotograph of monilial granuloma of the larynx.

a small remnant of the granuloma or "tumor" remained on the left vocal cord and the granuloma on the interarytenoidal mucosa had disappeared. A second series of streptomycin injections was begun on the sixth day and continued for 3 days. On the tenth day, the vocal cords were freely mobile and healed with no evidence of granuloma, but still hyperaemic and thick. Monilia being present in the sputum, potassium iodide was prescribed in increasing dosage. The voice was clear 6 weeks later.

Case II. Mr. J. L. C., age 54, has a complaint of malaise and hoarseness. The onset occurred two weeks previously without fever. The voice was husky, tired rapidly and became hoarse on talking for a short time. He has had a cough which he attributed to cigarettes and recently an increasing amount of sputum. The examination of the larynx demonstrated an acute laryngitis without any membrane. The vocal cords were

mobile, met in the mid-line on phonation and their mucosae were hyperaemic and thickened in small patches.

The acute laryngitis was treated systemically and with topical application. The laryngitis under this therapy rapidly became worse. The patches of hyperaemic and thickened mucosa extended and coalesced with each other, so that the vocal cords took on a red velvety appearance. The x-ray was reported by Dr. Harris to be negative for chronic pulmonary tuberculosis. Dr. Sanders reported the serological tests for syphilis to be negative. The sputum examination was negative for *B. tuberculosis*, but showed a large amount of *monilia albicans*. After the previous experience with monilia, it was not regarded as merely a concomitant finding. Intramuscular injections of streptomycin gram 1.5 per diem was administered as in Case I. The acute laryngitis on the tenth day was much improved; but a few tiny patches of hyperaemic and thickened mucosa remained on the vocal cords, and monilia persisted in the sputum. Potassium iodide in increasing amounts was exhibited but was discontinued on account of the patient's poor tolerance. *Liquor Iodii Compositi* (Lugol's Solution) in an increasing dosage was effective in clearing the small red patches of thickened mucosa from the vocal cords and monilia from the sputum. The hoarseness and cough were relieved.

No conclusions, of course, can be based on two cases, but the results here reported may be summarized as follows:

1. Moniliasis of the larynx may occur as an acute or chronic infection.
2. A monilial infiltrative granuloma of the larynx is rare, and it may mimic cancer.
3. Streptomycin controls an acute monilial infection of the larynx and causes the rapid disappearance of a granuloma, but in the dosage here reported does not clear monilia from the sputum.
4. *Liquor Iodii Compositi* (Lugol's Solution) is effective in treating moniliasis and clearing monilia from the sputum.
5. The incidence of acute laryngitis due to monilia is unknown, but like bronchomoniliasis it may be recognized with increasing frequency.
6. Monilia in the sputum does not necessarily demonstrate it to be the invading organism.

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DISCUSSION

JOHN J. SHEA, M.D.: I desire to report two patients who suffered from moniliasis of the upper and lower respiratory tract, one which lived and the other died.

The initial lesion may be a granuloma and as such will simulate a tuberculous or leucitic lesion. Like Dr. Cody's first case the diagnosis was made by a tip-off from the roentgenologist after a study of the chest.

Six kodachrome slides of the progress of the lesion were demonstrated.

Comment: The three drugs of value in the treatment of moniliasis are iodides, thymol and sodium benzoate.

A seventh slide was shown of the pulmonary field showing an extensive moniliasis of the lungs, with the comment that when this patient entered the hospital his lesions were so advanced and his general condition so poor, that in spite of medical help he expired on the third day after admission.

THOMAS C. GALLOWAY, M.D.: I would like to ask whether the monilia are primarily pathogenic or saprophytes, whether such cases as these depend upon some other systemic effect which allows a rather normally harmless organism to become harmful.

C. C. CODY, M.D. (Closing): The literature on that question is rather clear. Many types of monilia are not pathogenic. *Monilia albicans* in connection with some pyogenic type of infection may become of itself an invading organism; but, as in otomycosis, the mycologists dispute whether a pure infection of a fungus is pathogenic. This is an academic question because in the larynx, ear or lung, pure infection of any one organism are the exception rather than the rule. I doubt that *monilia albicans* by itself would be pathogenic for the reason that it is a very common fungus with clinical manifestations quite rare.

RHINOSCLEROMA APPARENTLY CURED WITH STREPTOMYCIN*

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Rhinoscleroma is a chronic granulomatous process involving the nose, upper lip, mouth and upper air passages. It is believed by many to be an infection but it is only slightly, if at all, contagious. An organism of the genus, *Klebsiella*, may be isolated from such patients. The chronic progressive clinical disease of rhinoscleroma has not been reproduced in animals but the organisms are always present in the tissue of patients with this clinical manifestation. The organisms are usually seen in the large phagocytes and are easily grown in an artificial culture medium. Organisms with similar cultural features are frequently isolated from the respiratory tract of normal persons.

The disease was once thought to exist only in Central and South-eastern Europe but it now occurs anywhere and the incidence seems to be increasing in South and Central America. According to Cunning and Guerry,¹ a total of 102 cases had been reported in the United States or Canada by 1942; 16 of the patients were native-born.

Clinically, there is first an insidious growth of hard smooth nodules in the anterior nares. This growth spreads gradually until, in many cases, protruding rounded, smooth masses with intact skin surface close the nostrils. From the anterior part of the nose the lesion spreads backward

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into the pharynx, larynx or trachea or even into the bronchi. It may extend forward onto the upper lip or involve the cheeks, gums, antrum, lacrimal ducts, soft and hard palate and the tonsils. Because of the presence of these masses function is impaired, thus giving rise to such symptoms as foul nasal discharge, nasal obstruction, deafness, tinnitus, hoarseness and difficult breathing. Tracheotomy is often necessary. The disease is limited to the mucous membrane and skin, ulceration is the exception and not the rule and constitutional symptoms are rare.

Up to the present no satisfactory method of treatment has been available. Therapy with roentgen rays or radium is, at best, palliative and must be repeated as recurrence of the disease dictates. Sulfanilamide and its derivatives seem to control the secondary infection but they have no effect on the scleromatous tissue.

Recently a patient with rhinoscleroma has been treated at the clinic with streptomycin.

REPORT OF CASE

A white woman aged 32, of Russian descent, born in Siberia and reared in the Far East, was first seen at the clinic on September 4, 1946. Her chief complaint was bilateral nasal obstruction for fifteen years with hoarseness, a sense of constriction in her throat and slight difficulty in swallowing for one year. A diagnosis of rhinoscleroma had been made in 1938 in Shanghai, China, where she lived. Treatment with autogenous vaccine apparently arrested the disease process except for a short period in 1941 when there was reactivation. In 1945 the case was reported² as one of rhinoscleroma which had been arrested by autogenous vaccine.

On examination at the clinic both nostrils were found to be almost completely obstructed with granulomatous tissue. There was scarring of the pharynx, with a small opening into the nasopharynx. The posterior surface of the epiglottis and the left side of the larynx were involved with granulomatous masses. Tomographs of the larynx showed almost complete obstruction of the airway at the level of the glottis. This was so marked that tracheotomy followed by skin grafting of the larynx was considered.

With the aid of suspension laryngoscopy, biopsy was performed on the larynx September 13, 1946. On microscopic examination a diagnosis of chronic granuloma, probably rhinoscleroma, was made. Culture of the granulomatous material showed large numbers of Klebsiella.

Previous workers³⁻⁶ have shown that organisms of the *Klebsiella* genus are sensitive to streptomycin. It was expected therefore that if the *Klebsiella* strain commonly found in such clinical conditions were etiologically related to the disease process, streptomycin might have therapeutic value. For these reasons the organism isolated from this patient was tested for sensitivity to the drug. It was found that growth was inhibited by 0.3 unit per milliliter of medium. Accordingly on September 27, 1946, treatment with streptomycin was begun. The patient received 250 mg. of streptomycin intramuscularly every three hours. After the first week, that is, after 12.75 gm. of streptomycin had been administered, the antibiotic was discontinued because of joint pains, cutaneous rash, malaise and fever. After twelve days' rest it was possible to continue the administration of streptomycin for forty-three consecutive days or until a total of 97.25 gm. had been given. Treatment with the antibiotic was discontinued on November 26, 1946, because of dizziness and slight nausea.

The patient did amazingly well. After 18 gm. of streptomycin had been given, she stated that she felt 50 per cent better than before treatment, that the condition of her nose was better and that the laryngeal obstruction was less obvious.

After 38 gm. of streptomycin had been administered, the patient stated that there was only one third as much mucopurulent material and crusting in her nose as there had been before treatment. After 53 gm. had been used, tomographs of the larynx showed a good airway, and suspension examination of the larynx showed some scarring anteriorly and on the left side but no evidence of active disease was present. The results of culture of tissue removed from the nostrils were negative for *Klebsiella*. After 95 gm. of streptomycin had been given, again the results of culture of tissue from both nostrils were negative for *Klebsiella* and clinically the patient was cured.

The patient suffered from varied toxic manifestations ascribable to the streptomycin during the course of treatment. The dermatitis, fever and arthralgia subsided rapidly after administration of streptomycin was discontinued and did not occur when treatment with the antibiotic agent was resumed. Just before the completion of treatment, symptoms of reduction in the vestibular function of the eighth cranial nerve developed, as manifested by a disturbance in equilibrium and slight nausea. The nausea subsided soon after the use of streptomycin was stopped. The

disturbance in equilibrium persisted for several weeks but the patient was able to compensate for this reduction in vestibular function. At the time of the patient's dismissal, there was no apparent disturbance in equilibrium.

On March 19, 1947, approximately four months after the administration of streptomycin had been discontinued, suspension examination of the larynx revealed no activity and again the results of culture of material from the nostrils were negative for *Klebsiella*.

On July 14, 1947, approximately eight months after the cessation of treatment with streptomycin, examination of the larynx showed no evidence of the disease other than the residual scarring, and the results of culture of tissue removed from the larynx were negative for *Klebsiella*. The presence of large numbers of these organisms in the issue before treatment and their rapid disappearance, with clinical recovery, under treatment with streptomycin suggest that they may have been causally related, at least in part, to the pathologic process.

On July 25, 1947, the cicatricial stenosis of both nostrils was corrected by dissecting out the scarring produced by the disease and relining the nostrils with shaved grafts placed over soft rubber molds. The grafts were taken from the abdomen. Later, perforated vernetite obturators replaced the molds in either nostril and the patient was advised that they should be worn from time to time, possibly for six months.

The patient was dismissed from our care with no evidence of active rhinoscleroma September 16, 1947, approximately ten months after treatment with streptomycin had been completed (fig. 1).

This patient with rhinoscleroma is the first to be reported as probably cured with streptomycin, as far as we know. At the time of the original examination, since the airway was partially obstructed, it was thought that tracheotomy would be necessary; as much of the granulomatous and scar tissue in the larynx and trachea as possible would then be removed and replaced with a skin graft and later with an obturator in order to maintain an opening. The use of streptomycin cleared up the granulomas and also eliminated *Klebsiella* from tissue taken subsequently for biopsy. This evidence adds to the general impression that *Klebsiella* is an etiologic factor in the disease. The tomographs show very strikingly the increase in the diameter of the airway following the treatment.

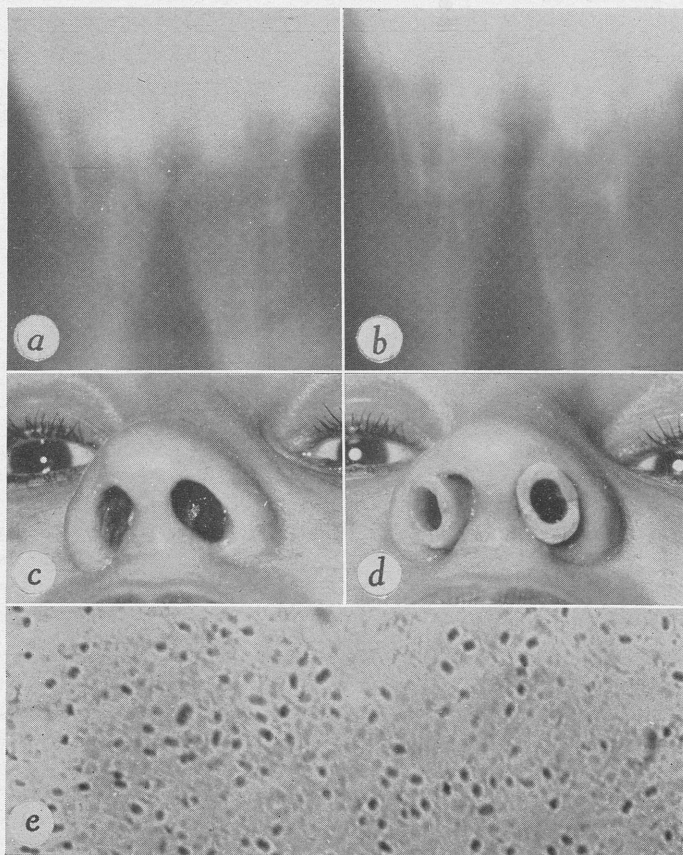


Fig. 1.—(a) Tomograph made before treatment, September 4, 1946. Note the small airway at the glottis (b). After treatment, November 1, 1946. Note the increase in the size of the airway. (c) Nostrils after treatment. The skin graft can be seen in the right nostril. (d) The vernonite obturators are in place. These should be worn from time to time, possibly for six months, in order to maintain the airway. (e) *Klebsiella* organism.

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DISCUSSION

DANIEL S. CUNNING, M.D.: At this meeting, one year ago, I reported two cases of scleroma in native born brothers, one of which has since died, having choked to death.

Dr. Blaisdell of Syracuse, New York, discussed that paper and reported a case of his which he treated with streptomycin and from all appearances, apparently cured. Due to Blaisdell's experience I was anxious to start treatment on this native born patient of mine. About the 1st of May we started him on 1.5 grams of streptomycin daily and continued this treatment for a period of nine months. The boy's general condition improved such as, added weight, but so far as the local lesions in his nose and larynx were concerned there was no change whatsoever.

HENRY L. WILLIAMS, JR., M.D.: I should like to report some work which one of my associates, Dr. K. M. Simonton, has done on a condition which possibly is related to rhinoscleroma; in other words, ozena. In this condition he has found routinely on culture the presence of *Klebsiella* sometimes associated with *Pseudomonas*. Whether or not these organisms are causative in ozena has of course been a matter of debate for a number of years, but as a result of this finding Dr. Simonton arranged to have his patients receive streptomycin therapy. The patients were given a gram of nebulized streptomycin daily in divided doses every two hours. The streptomycin was atomized with a flow of oxygen of 5 liters a minute. This produced very marked abatement of symptoms for the first few weeks, but at the end of six weeks these organisms, *Klebsiella* particularly, had developed such resistance to streptomycin that the initial improvement was lost and the patients again began to develop the original symptoms. For this reason, the fact that no permanent improvement had been secured, Dr. Simonton has not reported these cases. However, I thought they would be of interest here because of the fact that *Klebsiella* in particular rapidly develops such marked resistance to streptomycin that the drug soon becomes ineffective against the organism. It well may be that the relatively large doses which Dr. New used and the fact that he gave them parenterally instead of by nebulization resulted in the good effect which he achieved before a streptomycin-resistant strain could develop as opposed to the ineffective results which Dr. Simonton achieved in a possibly related condition. On the other hand, Dr. New may be incorrect in ascribing *Klebsiella* as

the etiologic agent in rhinoscleroma. Another organism less resistant to streptomycin may be the actual causative organism.

HENRY M. GOODYEAR, M.D.: I would like to ask Dr. Williams if he has tried streptomycin hypodermically. I know that some cases do apparently respond to parenteral injection of streptomycin who have atrophic rhinitis.

DR. WILLIAMS: That we have not tried.

G. B. NEW, M.D. (Closing): I would like to ask Dr. Cuning whether the organisms of *Klebsiella* were found in the patient he refers to and, if so, were they tested as to their sensitivity to streptomycin before the treatment was given. I think it is very important to test the stain of organisms to streptomycin if we expect to help that particular patient. I would like also to ask him how much streptomycin was given. We knew of the work of Doctor Simonton, and this aided us in determining the use of large doses to get rid of these organisms.

CANCER OF THE LARYNX*

A STATISTICAL STUDY

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JOHN J. AYASH, M.D. (*By Invitation*)

MINOT, NORTH DAKOTA

This is a report of 500 consecutive cases of cancer of the larynx seen at the Massachusetts Eye and Ear Infirmary from 1930 to 1945 inclusive, and treated either at the Infirmary or in the Radiological Department of the Massachusetts General Hospital. Included in the report are 46 cases who were seen, treated and followed as private patients.

Of the total of 500 cases, there were 28 who were seen for diagnosis only and who were treated elsewhere; 23 who refused treatment; and 32 who were treated but could not be traced. Therefore 418 cases of cancer of the larynx were followed either from their hospital records and by correspondence with the patient, with their families, their family physician or with their town or city clerks.

Cancer of the larynx is a disease of males. In this series 92% were male patients and only 8% female. The average age of the male patient was 60 years while that of the female patients was 40 years. The youngest patient was a young man of 20 years.

Type Of Lesion

Carcinoma Grade 1	35 or 7%
Carcinoma Grade 2	210 or 42%
Carcinoma Grade 3	140 or 28%
Carcinoma Grade 4	25 or 5%
Ungraded	55 or 11%
Clinical Carcinoma	25 or 5%
Micellaneous	10 or 2%

* This study was financed by a grant from "The American Cancer Society—Massachusetts Division." From the Department of Otolaryngology, Massachusetts Eye and Ear Infirmary, Boston, Mass.

TRACHEOTOMY

Tracheotomy was required in 80 cases, 25 of these were intrinsic cancer and 55 were extrinsic. In 46 cases tracheotomy was urgent and was done preliminary to treatment while in 34 cases tracheotomy was necessary either during or soon after external irradiation.

INTRINSIC AND EXTRINSIC CANCER

The terms intrinsic and extrinsic cancer are not standardized and convey a different meaning to each observer. Until these terms are standardized it would be better to describe the lesion as involving an anatomical structure, such as the true vocal cord, the laryngeal ventricle, the false cord, etc., rather than by the use of an indefinite term. Our own interpretation of the term "intrinsic" is that of an involvement of the lumen of the larynx from the laryngeal surface of the aryepiglottic fold down, while that of the extrinsic cancer is that of involvement of extra laryngeal structures, such as the pyriform sinus, the mouth of the esophagus, or perforation of the thyroid cartilages.

TREATMENT

With our present day knowledge of cancer we have but two methods of treatment that have stood the test of time, namely, surgery to remove the cancer, and irradiation to destroy it—or a combination of the two. It is the selection of the type of treatment for the individual case, and each case must be individualized, that calls for experience and wise judgment. In this series the records show that the type of treatment selected to have been—

External Irradiation	246 case
Radium with other treatment	14 cases
Surgery or surgery with irradiation	158 cases

COMMENTS OF THE GROUP TREATED BY EXTERNAL IRRADIATION

The treatment of cancer of the larynx by external irradiation has not been uniform during the past 15 years. Prior to 1936, the amount of irradiation was usually less than 5000r and was divided over a period of two or three months. During the past five or six years the technic of therapy as practiced by the radiologists of the Massachusetts General Hospital is to give a total dose of at least 6000r and more often that of

7000r if tolerated by the patient, and additional radiation for extension of the disease and cervical metastasis. The period is four weeks with a daily dosage of 300r to one lateral field the following day. In contrast is that of Cutler who gives from 6000r to 6500r to one portal in 18 days, giving two treatments daily.

With our present method of treatment as carried out at this hospital one would expect to see a higher percentage of cures in the latter years compared to the patients treated during the period of 1930 to 1940. By dividing our series into three groups of five years each and analyzing the results of each group the results of external irradiation are disappointing.

CASES TREATED WITH EXTERNAL IRRADIATION

Year	Cases	Dead	5 Yr. Cure	Less Than 5 Years
1930-1935	24	18-75%	6-25%	
1935-1940	75	58-77%	17-22.6%	
1941-1945	147	104-70%	5-3%	38-27%

If we take the patients seen and treated during the years of 1930 to 1940 and therefore observed for five years or more we find that of 99 cases of laryngeal cancer 76 or 76% are dead and the 23 or 23% have a five-year cure of their disease. Since many of the patients seen in the period of 1941 to 1945 have not been under observation or treatment for 5 years, it is shocking to note that of 147 patients 104 or 70% are already dead.

It is also interesting to note that the number of cases treated by external irradiation during 1935-1940 is three times the number so treated during 1930-1935; and that during 1941-1945 twice the number were so treated than during 1935-1940. About 1935 when the first supra-voltage x-ray was installed at the Huntington Memorial Hospital for Cancer Research, it was predicted that supra-voltage irradiation would replace surgery for cancer of the larynx. We shared in that enthusiasm and consequently referred many patients with cancer of the larynx, ideal for surgery, for treatment by irradiation. When the Huntington Hospital closed in 1940, the patients and their records were transferred to the Tumor Clinic of the Massachusetts General Hospital, and have been accessible for this study. With the further development of supra-radiation to the 3 million volt machine, our interests in the selection of pa-

tients for irradiation has continued, as we have been anxious to determine its value on other than the hopeless case. From our experience we are now of the opinion that:

1. In the so-called hopeless case of cancer of the larynx, external irradiation is not palliative and often hastens the demise of the patient.
2. In circumscribed local lesions of the vocal cord, without fixation, in a small series of cases 46% 5-year cures have been obtained.
3. Regression of the lesion does not signify a cure; the patient must be kept under close observation for at least five years.
4. The so-called trial period of irradiation is a fallacy. A full curative dose of external irradiation is necessary to determine radio sensitivity.

RADIATION RESULTS ACCORDING TO SITE OF LESION AND EXTENSION

Site	No. of Cases	Dead	5 Yr. Cure	Less Than 5 Yrs.
Laryngeal	72	66%	17%	19%
Epiglottic	38	71%	19%	20%
Subglottic	15	67%	13%	20%
Hypopharynx	66	79%	4%	17%
Tongue	11	91%	0	9%
Metastasis to neck	111	85%	4%	11%

When the disease is limited to the intrinsic larynx particularly the vocal cords or the supraglottic structures, the chances of a cure by external irradiation are best. As the lesion extends beyond the larynx the prognosis grows definitely worse. It is almost nil when the extension reaches the tongue and 85 per cent of our patients with glandular involvement are dead. Actually the percentage would be higher than 85 per cent when the final result of those under observation less than 5 years is ascertained.

The time to cure cancer is the first time that you attempt to do anything about it whether by surgery or by irradiation. Your surgery should be radical through normal tissue to remove the disease. The problem is not how little you can remove, but how much.

With radiation the dosage should be to the skin tolerance; when the first series of adequate irradiation fails to produce a regression of the lesion subsequent treatment is of little if any avail.

RELATION BETWEEN GRADE AND RADIATION RESULT

Grade	No. of Cases	Dead	5-Yr. Cure	Less Than 5 Yrs.
Grade 1 & 2	80	61%	19%	20%
Grade 3 & 4	104	72%	8%	20%

The general opinion among many surgeons regarding choice of treatment of carcinoma of the larynx depends upon the grade and histological picture of the tumor. Carcinoma grade 1 and 2 are generally considered radio-resistant and grade 3 and 4 radio-sensitive. Jackson and Jackson² state; "Tumors of grade 1 and 2 that used to be considered radio-resistant are apt to be more radio-curable than the less differentiated tumors of higher grades. These latter tumors of grade 3 and 4 carry a poor prognosis with any method of treatment." According to Cutler¹ radio-sensitivity depends upon the gross and microscopic picture and the site of the lesion. The choice of proper treatment should not depend upon grading alone. Cancer of one cord is radio-sensitive only if treated early before infiltration and fixation. Cancer arising from false cords and epiglottis is radio-sensitive. Any cancer that infiltrates and fixes these structures is radio-resistant.

In this series, the results obtained did not depend upon the grading of the tumor alone, but depended to a greater extent on the extension of the disease. The results of external irradiation were better than two to one in carcinoma of grades 1 and 2 than in grades 3 and 4.

CORDAL CANCER—IDEAL FOR SURGERY—TREATED BY IRRADIATION

No. of Cases	Dead	5-Yr. Cure	Less Than 5 Yrs.
11	3-27%	5-46%	3-27%

These 11 cases were considered as ideal for surgery in that the lesion was localized without fixation of the cord. Irradiation gave a 46 per cent five-year cure. With the final result of the 3 cases that are still under observation this percentage without a doubt will be better.

SURGERY

The usual procedure when a patient presents himself with malignant disease at the Massachusetts Eye and Ear Infirmary is to admit the patient to the Hospital and after a thorough study, a biopsy is taken and upon obtaining the microscopic report, the patient is referred to the

Tumor Clinic where the patient is examined by a group, including an Otolaryngologist, a Surgeon, and a Radiologist. There the case is discussed and the treatment decided. Previous to 1940, when surgery was recommended the patient was handled by the Surgeon on Service. As a consequence too many operators without tumor clinic experience were doing cancer surgery. Since 1940 all tumor surgery is done by one of us (LAS) or done under our direction.

The ideal case of laryngeal cancer for thyrotomy or laryngofissure is a localized lesion of the anterior third or the middle of a vocal cord, without involvement of the anterior commissure or extension to the vocal process, preferably of low grade, and without fixation of the cord.

LARYNGEAL CANCER TREATED BY LARYNGOFISSURE

Years	Cases	Dead	5-Yr. Cure	Less Than 5 Yrs.
1930-1940	31	13 or 42%	18 or 58%	
1941-1945	28	8 or 18%	2 or 8%	18 or 74%

From an analysis of our records only 39 cases were ideal for laryngofissure. Eighteen cases should not have had laryngofissure because of cord fixation, extension to the arytenoid or by reason of subglottic extension. Of these eighteen only one was cured by surgery, two had laryngectomy after laryngofissure failure; four have been under observation less than five years; three cured by irradiation and eight are dead. Out of the twenty cases that died after laryngofissure there were two that were operative mortalities,—one from pulmonary hemorrhage from an old tuberculosis lesion on the eighth post-operative day and the second was a post-operative lung abscess (1934). Eight should have had a laryngectomy instead of a laryngofissure. Ten were apparently well chosen cases that developed recurrences.

A review of our failures with laryngofissure reveals that the recurrences have mostly occurred in those cases in which the cancer had reached the anterior commissure, had crossed to the other cord or had reached the vocal process. For the anterior commissure lesion, we have followed the Jackson anterior commissure technic; but as with all cases of laryngofissure, we have used the diathermic scalpel and snare for incision and removal of the soft parts of the lumen of the larynx. The cut surface is also dessicated by electro-coagulation.

We still use the nasal feeding tube for 24 to 48 hours, and from experience advise its insertion before rather than after the operation

when the swallowing reflex is abolished by the anesthetization of the larynx.

LARYNGECTOMY

1930-1940	44	16-36%	28-64%	
1940-1945	53	14-26%	2-4%	37-70%

This group of laryngectomized patients can be further broken down into those who were treated only by surgery and those in whom the extension of the disease made the operative removal questionable and therefore they received external irradiation after their surgery.

Laryngectomy	63	14-22%	19-30%	30-48%
Laryngectomy and X-ray	34	16-47%	11-33%	7-20%

Our over-all statistics of laryngectomy of the five-year or longer group show a survival rate of 64 per cent, while that of New³ is 60.2 per cent.

The psychology of a surgeon dealing with cancer should be radical. The problem is how much can be removed and not how little. When a patient is faced with the loss of his larynx, he is not interested any longer in conservative surgery. The routes of extension of cancer of the larynx have been well demonstrated by Taylor and Nathanson⁴; and the importance of removing the pre-epiglottic space has been emphasized by Bisi.⁵

In the operation of total laryngectomy, we have always used the "T" incision or the "U" incision of Gluck-Soerensen. The sterno-hyoid muscles, and at times the anterior belly of the omo-hyoids are removed with the hyoid bone or the body of the hyoid bone.

The pre-tracheal gland on the crico-thyroid membrane is always examined microscopically for cancer. The pre-tracheal muscles are removed since they are rich in lymphatics and are in close relation to the pre-tracheal gland. We have found metastatic cancer in the sterno-hyoid muscle when on gross inspection, it could not be detected. The thyroid hyoid muscle is removed with the larynx.

The carotid sheaths are always exposed and examined for glands, and should glands be found a radical neck dissection is done along with the laryngectomy. In cases of laryngeal cancer with unilateral cervical metastasis both Sylvestre⁶ and Brunschwig⁷ have demonstrated not

only their operability but an encouraging curability by radical neck dissection and laryngectomy.

We have experimented with closure without drainage, and have obtained a surprising number of primary closures. The fistules, since the advent of the sulfonamides and the antibiotics, are not a problem.

In this series of 97 laryngectomies there were four operative deaths—one was from lung abscess, one periesophageal abscess, two were cardiac deaths, one on the eighteenth day, the other suddenly on the third day. Four patients died suddenly, either at home or on the street. One patient (female) died from pneumonia 3 years after the operation, a diabetic survived ten years. Ten cases died of extrinsic disease and although 12 were by all standards good risks, they were frank failures.

SURGERY AND IRRADIATION

When external irradiation fails to control the disease, surgery can still be done although the healing may be delayed. In this series laryngofissure was done in four cases and laryngectomy in 8 after external irradiation failed to control the disease. Two of the laryngectomies were done on patients for recurrences, occurring after three years of apparent cure by irradiation.

LARYNGECTOMY AFTER X-RAY FAILURE

Cases	Dead	5-Yr. Cure	Less Than 5 Yrs.
8	2	2	4

METASTASIS

Of 19 cases with distant metastasis, the following organs were involved—lung 11, brain 3, spine 2, and one each of knee, hip, jaw, prostate and skin of chest. In one patient after 7 years with the larynx free of disease, the lung and thigh showed metastasis. In another, after laryngofissure in 1935, laryngeal recurrence appeared in 1941, and after treatment by x-ray there was regression of the growth. Metastasis in the lung developed in 1943 and was treated by lobectomy; the patient died of post-operative empyema.

RESULTS OF SURGERY ON GRADE 3 AND 4 CARCINOMA

	Cases	Dead	5-Yr. Cure	Less Than 5 Yrs.
Laryngofissure	4	3	0	1
Laryngectomy	7	3	1	3
Laryngectomy and X-ray	11	4	4	3

It is difficult to draw conclusions from this small series. However, it is evident that surgery on well chosen cases of high grade malignancy has some chance of success. Since high grade malignant disease is difficult to cure by any method of treatment, we feel that combined treatment—surgery and external irradiation—offers the patient the best chance of life.

The surgeon's duty to his patient does not end with the discharge of the patient. At least a 5-year follow-up is necessary to detect early recurrence. The laryngectomized patient should be rehabilitated by the development of a voice. We have followed the work of McCall⁸ in pre-operative training, and by the establishment of a daily speech class.

Many of our patients have developed good esophageal voice within two weeks following their laryngectomy, while others require months. Many of our patients have come from other cities and many have been operated from one to four years without any attempt being made in rehabilitation by either an esophageal voice or the artificial larynx. In our clinic in the past year over 100 patients have been taught to speak with the so-called esophageal voice.

SUMMARY

A total of 418 cases of carcinoma of the larynx were studied, their treatment classified and evaluated. Of these 418 cases, 246 were treated by external irradiation. Twelve patients underwent surgery after external irradiation failed to control the disease.

The site of the lesion and the extension of the disease are of more importance in the proper choice of treatment than the grading of the tumor. Carcinoma of grade 1 and 2 are more radio-curable and surgery curable than are the more anaplastic types of growth.

Limited lesions of grade 3 and 4 are best treated by surgery followed by post-operative external irradiation. Laryngofissure is the operation of choice for early cancer limited to one cord without fixation. Extension of the growth to the anterior tip of the opposite cord is still amenable to laryngofissure by wider dissection. Any fixation or posterior extension to the arytenoid even without fixation contraindicates laryngofissure.

Early unfixed cordal lesions are 46 per cent radio-curable by concentrated external irradiation. With the extension of the disease to the subglottic space and with cord fixation laryngectomy is the treatment

of choice and not external irradiation. Borderline cases, cases which some observers call extrinsic, can be successfully treated by surgery and irradiation.

In a group of poorly selected cases a 58 per cent five-year cure was obtained by laryngofissure. Total laryngectomy in a series of cases operated between 1930 and 1940, and therefore followed over 5 years resulted in a 64 per cent survival.

With the patient under close observation, a full course of irradiation is justifiable with the distinct understanding that unless the tumor shows evidence of regression, surgery will be instituted.

Early diagnosis and repeated biopsies of suspicious lesions until the diagnosis is absolutely established can never be overemphasized as any delay will make the disease less and less favorable for cure by any of our methods of treatment.

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DISCUSSION

LOUIS H. CLERF, M.D.: This is a very fine analysis of a large group of cases of carcinoma of the larynx done by one whom we all know is honest, and further, it is an analysis of a large group of cases that were treated by irradiation.

I will venture to say that there are few radiologists who have had such an excellent opportunity to demonstrate what can be done and where results have been so well supervised.

In this group a large number of the lesions irradiated were considered by Dr. Schall to be suitable for conservative laryngeal surgery. He says he expresses disappointment at the results, and I too must confess that the results are very disappointing.

I was interested in his comments on grading. Recently I voiced the same sentiments with regard to grading, in that the majority of the patients with cancer of the larynx belonged to the II and III groups, which we put in the intermediate group. A relatively small group are Grade I the low grade, and Grade IV or the undifferentiated group.

Furthermore, it is of interest to observe that as we progressed in the field of radiation therapy the results seem to become progressively worse. From 1941 to 1945 I believe there were 147 cases treated and there are already 70 per cent dead, and the end is not yet.

I subscribe to Dr. Schall's views with regard to the indication for thyrotomy. When one elects thyrotomy, one ought to be in a position to tell the patient that he has a 90 per cent chance of passing the five-year mark without recurrence. One must therefore be very circumspect in selecting the cases that are suitable for conservative surgery. There is no need of cutting too thin, for if one does the five-year non-recurrence rate will be very low.

I still believe that laryngectomy offers more to the patient than does any other plan of therapy. While, as I stated in Atlantic City, there are probably some larynges that are sacrificed, on the other hand, there will be more people alive after five years. That really is important from the patient's standpoint.

With regard to the technic of laryngectomy there is no point in conserving structures that are attached to the larynx. They are of no value and unhappily they may harbor cancer cells.

Since 1942, we have been doing block dissection and laryngectomy at one sitting, whenever it is necessary in our estimation, and I am pleased with the end results. I am sure that when we report our next five-year group it will look a lot better than the last one I reported.

JOHN D. KERNAN, M.D.: I really don't believe in this discussion at all because I am too old. The first laryngectomies I saw were done by Dr. Mackenty. That is thirty-five years ago. Then the complications were many and dangerous. Now one can say that all the old objections to laryngectomies have been overcome, the danger of lung complications, the danger of sloughing. Mackenty used to welcome sloughing in the neck because, as he said, a lot of cancer might slough away that he did not get. Healing took many weeks, now the wound heals promptly. It is not unusual to have it perfectly clean within a week, the tube out and the patient leaving the hospital in ten days or two weeks.

Few patients learned to talk in those days. Then it was necessary to invent a talking machine attached to a tube in the neck and teach them to talk that way. Now, as Dr. Schall said, everyone learns to talk. I think you can promise that. They are not all taught up in Massachusetts. We taught an insurance salesman to talk. If one learns enough to sell insurance as a talker that is enough.

Yet for all these successes I cannot be persuaded that it is a pleasure to have a laryngectomy. A laryngectomy is not a cure. It leaves a partially crippled man; so we should look for something better.

I know that x-ray works in certain cases, the very cases which are amenable to the lesser operations, local excision under suspension, or excision through the laryngofissure route. I think there are some cases (drawing on the board) if

this were one vocal cord and that were the other, and you had a lesion show that way, there are certain cases that run along under the mucous membrane that way (indicating) and they run along under a perfectly normal-looking surface. They are called intramucosal or carcinoma in situ. Those cases you might operate under suspension cutting out a loop like that (indicating) and you might leave a bit of cancer in each end. Even with a laryngofissure that might be done. Everyone is anxious to preserve function and is proud when the patient has a voice. You are likely to keep away from the arytenoid, for if you get a mobile arytenoid you will get a good voice for you get a new vocal cord. Therefore you try to keep away from the arytenoid and leave a little cancer under a normal-looking surface. In that way you endanger the patient. That danger would be avoided if radio-therapy were used. They are said to be amenable to x-ray. I don't know whether that altogether is so or not.

The histological picture is very important (drawing on the board). To show you how influential the histological picture, the biological nature of the growth is, I will mention two or three cases.

I had a man and here was his tracheotomy, the carcinoma sprouting all around the tracheotomy opening like that (indicating), so that he had to have an incision like this and like that. The whole thyroid gland was taken out, and about everything in the neck. It took four hours to do; in spite of the extensive nature of the cancer, he is living 25 years from then. He taught himself to speak. He did not have to go to school.

Within a month I did a man who looked just the same, another one (drawing on board) who had a carcinoma like that and he was dead within two months. He looked just the same but the histological picture was very different.

I had still another man, this one more recently (drawing on the board), and this fellow had a carcinoma, a little one up in there (indicating), the anterior commissure, and we did a rather limited laryngectomy. I thought sure we would get a cure but he died in six months. He had a prompt recurrence. The appearance of the glands in the neck was almost like an explosion. We never felt any little ones. They appeared over night, they grew rapidly and within the week the neck was all filled out from the chin to the shoulder. Histologically the growth was very malignant, and when we got the larynx out and examined the whole thing we found a little patch of cancer way over there (indicating), the arytenoid region, under the mucous membrane which we had missed altogether, as grossly it did not show. I think the great danger in doing the laryngofissures is not to look everywhere after the larynx is open.

The indications which Dr. Schall has given, the mobile cord, growth in the middle, anterior commissure not involved, the subglottic space not involved and the arytenoid not involved, and the growth of low-grade malignancy are valid. If you stick to those you will get 90 per cent cured. If you don't, you are putting the patient in great danger.

Thank you very much.

CHEVALIER L. JACKSON, M.D.: I have immensely enjoyed Dr. Schall's very sound analysis of his material. There are only one or two little comments that I would like to make on it.

I think he has stated extremely well the indications for laryngofissure. It seems to me that even though we all recognize now that a laryngectomy is no longer to be considered a catastrophe, still a procedure which will cause so much less handicap as laryngofissure, having proven itself capable of 85 to 90 per cent of 5-year cures in properly selected cases, should not be cast aside. I believe that what those of us who have faith in this procedure should do is to insist more and more strongly on the necessity of following these indications. I am quite sure that too many laryngologists are inclined to do a laryngofissure because they think they can do that much of an operation, but are unwilling to undertake laryngectomy, or are still too much aghast at the predicament in which it is going to leave the patient. However, if we insist upon following the proper indications we still have in laryngofissure a valuable procedure which should not for a moment be done away with.

I would like to have Dr. Schall state to us once more what he considers to be the indications for irradiation today. He said, and I agree with him, that palliative treatment by x-ray in the inoperable, hopeless case is really not a palliation in most instances, but I have seen one or two surprisingly good results in such cases.

In reference to the kind of case Dr. Kernan mentioned, I have seen several of this kind also, and I think that one of the types of lesion unfavorable for laryngofissure is the lesion without a sharp line of demarcation. I feel that these are the cases which are treacherous if treated by laryngofissure. Some of them are not extensive enough to justify laryngectomy as the first method of treatment. However, I recently had a patient upon whom I had intended to do a laryngofissure, but on opening the larynx I found it so difficult to determine just how far this limited superficial lesion went, that I closed up without starting the intralaryngeal dissection. This particular patient is now receiving a course of protracted fractional irradiation. Our experience is just the same as that of Dr. Schall with regard to the relation of grading to prognosis of treatment. The Grade I and II cases are the ones that give the best results, whether by surgery or by irradiation. The Grade III and IV cases are the ones that are both less radio curable and surgically curable.

EDWIN N. BROYLES, M.D.: Mr. Chairman, I would like to ask Dr. Schall one question. He mentioned tracheotomy, preliminary tracheotomy, where the patient is dyspneic. It has been my general experience when a patient comes in with carcinoma of the larynx requiring tracheotomy, in everyone that I have seen, and I would like to have Dr. Schall's opinion and his experience in this, that the carcinoma has already gotten outside of the larynx, either through the cricoid membrane or elsewhere.

I congratulate him on this wonderful paper on carcinoma and its treatment.

L. A. SCHALL, M.D. (Closing): May I thank you all for listening to this paper and for the generous discussion!

To Dr. Clerf I want to say that the radiologists with whom I work are in complete agreement with me as to the treatment of cancer. There is no conflict. In fact, I have referred patients to the radiologists whom I thought should be treated by external radiation and they have sent them back and said, "Please do

your surgery because we feel this type of patient is not suitable for external irradiation." Our group of radiologists have gone over this paper and approved what I have had to say.

The more I see of cancer the less I know about it. At one time I felt that grading was very, very important, because if we accept the principle that the dividing cell is the most susceptible to external irradiation, then it is logical to suppose that the Grades III and IV would be most radio-sensitive. I have seen Grades III and IV stimulated by external radiation and grow wild, out of bounds, and I have seen Grades I and II melt away. I think grading is of importance as to prognosis because we know from experience Grades I and II give us our best results, no matter what type of treatment we use.

I do not propose, Dr. Jackson to cast aside laryngofissure in those cases that I cited my indications for. I have many patients on whom I have used the Jackson anterior commissure technique in which the lesion extended to the anterior cord around the other side (drawing on the board) and have had successful cures, but I have also had recurrences in that type of case, and most of my recurrences have been after that type of operation.

Indications for irradiation, if this is an anaplastic growth and if this patient has a good airway, and is in an occupation where his voice is of utmost importance such as a minister, attorney, school teacher, and if he will stay under my observation, I will try external irradiation. If that patient has had suitable treatment without regression of that growth then he must consent to surgery. You cannot do irradiation and lose track of your patient.

We have had too many tragedies in cases that I have seen in consultation being treated at other clinics, lost track of both by the radiologist and referring physician, coming in two and three years later beyond any hope of cure by any method.

Then too in irradiation we have surprises. I recall a patient at the Huntington Hospital, where on his first admission we did a tracheotomy and a few days later a gastrostomy, who had a Grade II lesion, that was completely cured for five years by irradiation. So don't cast aside irradiation but control it.

Laryngectomy after irradiation, I have operated in the height of the irradiation reaction. The healing has been delayed but I have not lost any patients.

About tracheotomy, Dr. Broyle's question, I cannot form any opinion as to whether it is already beyond the larynx except to say that a large percentage of our patients have had tracheotomy before operation and they are in this five-year group.

CARCINOMA OF THE TRACHEA

CLARENCE W. ENGLER, M.D.

CLEVELAND, OHIO

Any instance of carcinoma of the trachea seems worthy of report, in view of the rarity of the condition. This is all the more true now that better roentgenographic methods and bronchoscopy are generally available, for cancer of the trachea is no longer merely of academic interest as a pathologic curiosity, but is one of the malignancies of the upper respiratory tract that the practicing physician may be called upon to recognize and treat. Since the condition is so rarely encountered, only the collective experience of the entire profession can provide sufficient basis for formulating any conclusions that may add to our knowledge of this type of lesion.

The first case I have to present is definitely one of primary carcinoma of the trachea. The patient was first observed two years ago and was treated by roentgen irradiation. Recently he has had a recurrence of symptoms and of the carcinoma and is now undergoing another course of radiation therapy. In the second case, observed by my associate, Dr. S. C. Missal, the carcinoma was so far advanced when the patient was first observed that it could never be definitely determined, even at autopsy, that the lesion was primary in the trachea.

Case 1—A man, aged 47 years, was admitted to St. Vincent Charity Hospital for treatment of an illness which had begun eight weeks before with incessant tickling in the throat and continuous coughing. The patient complained also of shortness of breath and wheezing which had become progressively more severe. During the last week, the cough had been productive of small amounts of blood and frothy sputum.

Fluoroscopic and radiographic examination (Fig. 1 and Fig. 2) of the chest showed the presence of a sharply circumscribed spherical tumor, about 6 centimeters in diameter, in the upper mediastinum just to the right and behind the ascending arch of the aorta, lying in intimate relationship with the esophagus, which it compressed, and with the trachea at its bifurcation. The tumor showed no calcification and there was no similar tumor elsewhere. The lungs appeared clear and well

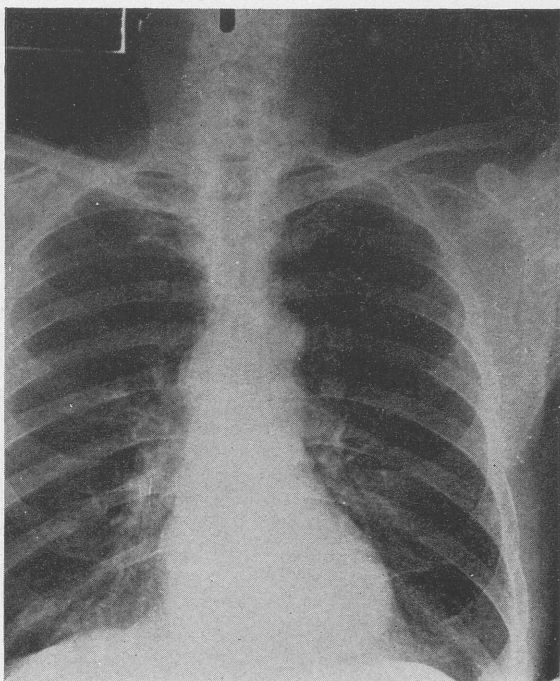


Fig. 1.—(Case 1.) April 23, 1946—Posterior-anterior view of the chest showing a sharply circumscribed spherical tumor approximately 6 cm. in diameter in the upper mediastinum and to the right of the arch of the aorta.

aerated, indicating absence of bronchial obstruction. The heart and great vessels appeared normal in size and shape. The roentgenologic diagnosis was right upper mediastinal tumor of undetermined origin.

Tracheoscopy disclosed a large fungating mass extending over the posterior wall of the trachea, most prominent at 5 o'clock. This was situated 25 centimeters from the gum line, just at the bifurcation. The tumor bled freely and was quite friable. Esophagoscopy examination showed nothing abnormal.

Histologic examination of tissue removed from the trachea showed that it was composed of necrotic tissue and fibrinopurulent exudate with associated tumor. The tumor was characterized by compact masses of moderately pleomorphic neoplastic epithelial cells displaying no architectural arrangement and no evidence of keratinization. Numerous

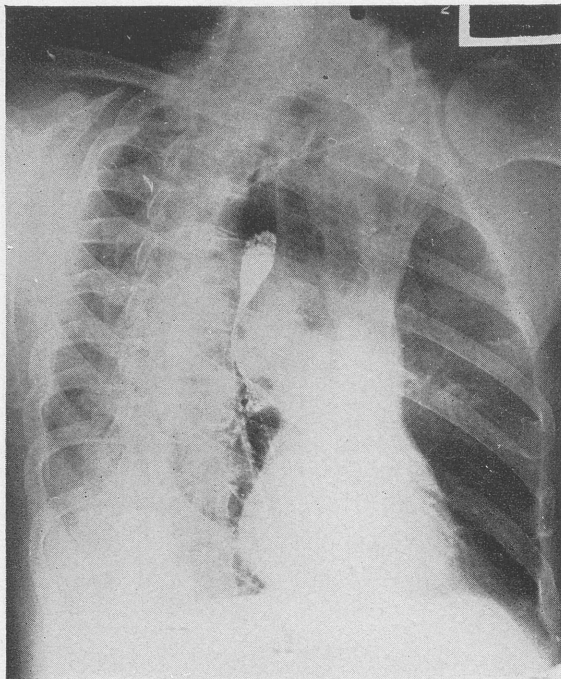


Fig. 2.—(Case 1) April 23, 1946—Left oblique view of the chest showing the upper mediastinal mass lying behind the arch of the aorta and compressing the esophagus.

mitotic figures were evident. The pathologic diagnosis was poorly differentiated squamous carcinoma (Fig. 3).

Roentgen therapy was instituted immediately, and the patient was discharged from the hospital five days after admission, to continue roentgen treatments as an out-patient. A week after his discharge, he became dyspneic, cyanotic and apprehensive, and re-entered the hospital in acute respiratory distress. His temperature at that time was 37.8 C.; pulse, 120; respirations, 30, and the blood pressure was 160 systolic, 100 diastolic. Examination of the chest showed dullness at the base of the left lung and decreased breath sounds, indicating atelectasis. The right lung was hyper-resonant.

Since I was out of town at the time this episode occurred, the patient was cared for by Dr. Missal. Because the tumor had demonstrated a tendency to bleed excessively, he wisely decided against per-

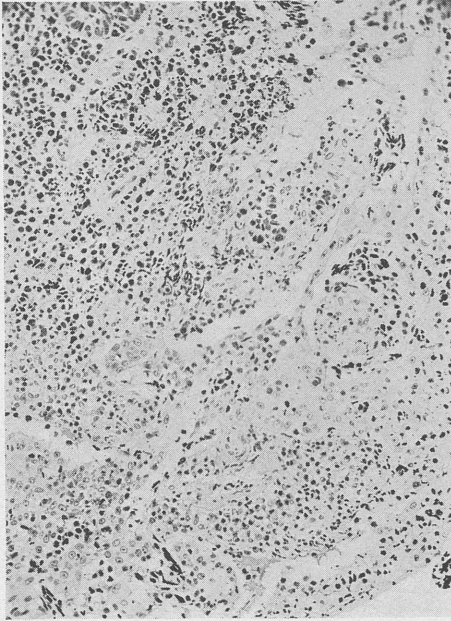


Fig. 3.—(Case 1) Section of the tracheal tumor with necrotic tissue and fibrino-purulent exudate. The tumor was characterized by compact masses of moderately pleomorphic neoplastic epithelial cells displaying no architectural arrangement and no evidence of keratinization, with numerous mitotic figures.

forming a bronchoscopy; instead he passed a catheter and instilled adrenalin and pontocaine to reduce the edema and loosen the secretions. Oxygen was administered through the tube. After this procedure, the patient coughed up a considerable quantity of thick, blood-tinged sputum. When the tube was removed, breathing improved and the left lung showed evidence of re-expansion. By the following day, a roentgenogram showed that both lungs were aerated equally and fully. After ten days, the patient was dismissed from the hospital.

The course of roentgen irradiation was administered in thirty-four days. This was given through six fields, each 6 to 8 centimeters in diameter, with the central ray directed toward the tumor by fluoroscopic localization. Two fields were irradiated daily through a filter of 0.5 millimeters of copper. Other factors were 200 kv, 1.0 millimeter copper half-value layer, 70 centimeters distance. The posterior field received

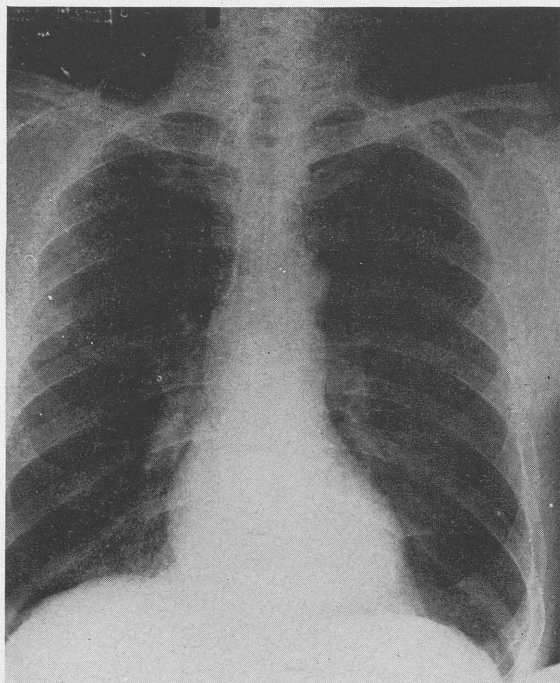


Fig. 4.—(Case 1) May 28, 1946—Postero-anterior view of the chest showing no evidence of tumor.

2800 r (in air) in twenty-five days and each of two anterior and posterior oblique fields received 2050 r (in air) in twenty days. The estimated quantity of radiation received by the tumor was 4300 r.

Roentgen treatment was completed on June 3, 1946. On May 28 (Fig. 4), a roentgenogram of the chest showed no evidence of the lesion. The patient experienced complete relief of symptoms and gained 15 pounds in weight during the course of radiation treatment. Within the next few weeks, he gained an additional 7 pounds. Repeated fluoroscopic examinations on June 28, October 15 and December 27, 1946, and on April 16, 1947, were all negative for evidence of tumor, and the patient remained in excellent health generally.

On February 13, 1948, the patient reported that he had had a recurrence of coughing and wheezing during the last week. He stated that his general health had been good and the weight gain which had

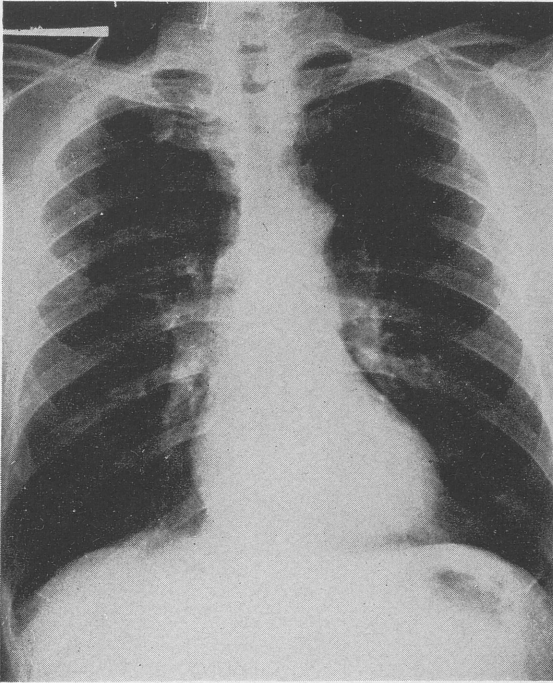


Fig. 5.—(Case 1) February 13, 1948—Posterior-anterior view of chest showing no change in anterior mediastinal shadows but apparently some widening of the mediastinum at the level of the seventh and eighth ribs posteriorly.

followed radiation treatment two years before had been maintained.

An anterior-posterior view of the chest showed that the anterior mediastinal shadows had not changed since the last examination, but there appeared to be slight widening of the mediastinum at the level of the seventh and eighth ribs posteriorly (Fig. 5). A left oblique view showed a slight depression of the esophagus in the posterior mediastinal region, but no distinct soft-tissue mass could be identified (Fig. 6). A right oblique view (Fig. 7) also showed no evidence of a tumor.

Bronchoscopy was performed on March 5, 1948. A mass was encountered just below the subglottic space on the anterior wall of the trachea. This measured 1.5 centimeters in diameter. A fungating mass also was present at the site of the original lesion. Vision into the right main bronchus was not obstructed, but the bifurcation was not evident

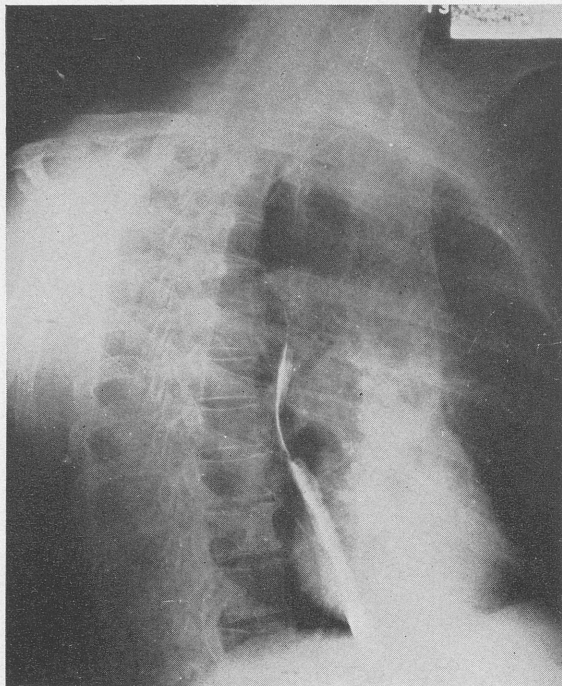


Fig. 6.—(Case 1) February 17, 1948—Left oblique view of the chest showing slight depression of the esophagus in the posterior mediastinal region, but no distinct soft tissue mass is identifiable.

and the orifice of the left main bronchus could not be seen. The tumor in the lower part of the trachea extended from the posterior wall upward laterally and seemed to extending also into the left main bronchus. Biopsy specimens from both tracheal lesions were removed for histologic study. The microscopic picture was identical in both and the same as that of the original growth.

The patient is now receiving a second course of roentgen treatment. It is planned to administer the radiation at 50 centimeters distance, through two portals (15 x 15 centimeters) to the lesion at the lower end of the trachea. Both anterior and posterior fields will receive 1500 to 2000 r (in air) according to the patient's tolerance. The plan of treatment of the upper tracheal lesion is to give 2000 to 3000 r (in air) anteriorly and posteriorly through a 4-centimeter field at 50 centimeters distance.

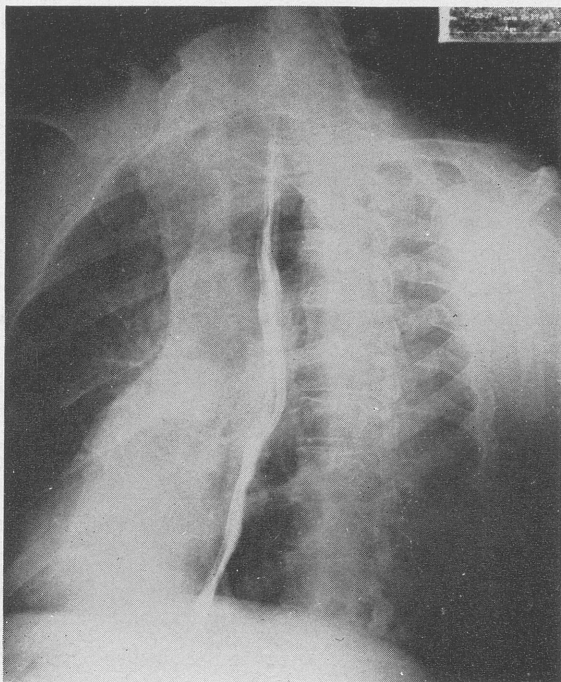


Fig. 7.—(Case 1) February 17, 1948—Right oblique view of the chest shows no evidence of tumor.

Comment—This patient's case history focuses attention on several facts that are interesting from the clinical standpoint. He was examined elsewhere a few days before I saw him and was told that a chest roentgenogram showed nothing abnormal. Furthermore, although his symptoms of dyspnea, wheezing and hemoptysis clearly indicated the need for a bronchoscopy, this was not done, apparently because the negative chest roentgenogram allayed any suspicion the examining physician may have had as to the seriousness of the condition.

It has been amply demonstrated that special radiologic methods must be used to reveal tumors of this type; the routine anterior-posterior roentgenogram of the thorax is not reliable for this purpose. Oblique and lateral views and use of a contrast medium in the esophagus and fluoroscopic examinations with the patient changing positions, coughing and swallowing, may be necessary. This has been stressed by Brown,

Weiss, Iglauer and Fine,¹ Tinney, Moersch and McDonald,² Rathbone³ and Boemke and Moritz.⁴ Rathbone's patient had had repeated roentgenologic examinations of the neck and chest over a five-year period which had been reported negative, while the dyspnea of which she complained became progressively more severe. The tumor was finally discovered radiologically by asking the patient to swallow while in the lateral position. In the case reported by Boemke and Moritz, the patient also had had symptoms for five years and repeated radiologic examinations of the chest revealed no evidence of tumor. Guttman⁵ and Penta⁶ have also reported cases in which patients with tracheal tumors had been undergoing treatment for asthma while their dyspnea and wheezing progressed to serious respiratory distress. These occurrences make it evident that despite all that has been said and written to show that bronchoscopy is indicated in most cases of respiratory difficulty, there is still need for reiteration and emphasis of this fact to the medical profession generally.

Although the present outlook in this case is certainly not good, the history so far would seem to indicate that it may not be entirely hopeless. This patient originally had a highly malignant lesion on the lower posterior wall of the trachea, yet his response to irradiation with roentgen rays enabled him to enjoy good health and to carry on his work and other normal activities for two years before there were any signs of recurrence. The prognosis must necessarily be guarded at this time, but there are a few reports in the recent literature which give some basis for the hope that radiation treatment of the recurrent lesion may also be productive of considerable benefit.

Brown, Weiss, Iglauer and Fine¹ reported a patient who had remained symptom-free three years after roentgen treatment for a recurrence of squamous-cell carcinoma of the trachea. Orso⁷ reported a secondary squamous-cell carcinoma of the trachea which occurred six months following an operation for a similar cancer of the larynx. After removal through the bronchoscope, the tracheal tumor was treated by roentgen irradiation and ten years later the patient had had no recurrence of the lesion. A patient with primary squamous-cell carcinoma of the trachea, reported by Olsen⁸ and also by Tinney, Moersch and McDonald,² was known to be alive nine years after treatment with diathermy and radium.

Numerous other cases of tracheal tumors, probably of less malignant type than these squamous-cell carcinomas—although in some there were recurrences, in which the patients survived one to several years following some form of radiation therapy have been reported by Cann,⁹ Engelstad,¹⁰ Pierson,¹¹ Fisher,¹² Dean¹³ and others.^{2,6} The evidence seems to indicate that all the patients whose treatment for tracheal tumor has been of significant benefit have received either roentgen or radium irradiation. In some of these cases, surgical removal by open operation or through the bronchoscope was also carried out.

Another interesting feature of the present case is that, in addition to the recurrent tumor at the original site on the lower posterior wall, there is also another tumor, of identical structure, higher in the trachea and on the anterior wall. This tumor is apparently entirely distinct from the original lesion which seems to be extending downward into the bronchus rather than upward. The question arises as to just how the new tumor should be classified. Is it an extension, a metastatic lesion or a new primary tumor? This occurrence is somewhat comparable to that in the case reported by Orso,⁷ who believed the tracheal lesion was an implant from the previous laryngeal operation. Watson¹⁴ also reported a patient with tracheal cancer who had had a laryngofissure and radium treatment fifteen years previously for a squamous-cell carcinoma of the larynx. The tracheal cancer developed on the posterior wall of the trachea where the tracheotomy tube came in contact with the mucosa, and was removed by an open operation. Watson was inclined to believe that this lesion was not directly related to the laryngeal cancer but that it was another primary malignancy resulting from chronic irritation of the mucosa by the tracheal tube.

Case 2—A woman, aged 66 years, was admitted to the accident room of St. Vincent Charity Hospital on June 14, 1947, because of extreme respiratory distress. She was sent immediately to surgery for a tracheotomy.

A midline incision was made and a portion of the third tracheal ring was removed. A No. 6 tracheotomy tube was inserted, but could not be passed because of a tracheal obstruction. The tracheotomy tube was removed and a 7-mm. bronchoscope revealed a tumor mass almost totally occluding the trachea. A smaller bronchoscope was then passed beyond this mass, and the patient began to breathe more easily. With the bronchoscope in place, the incision was opened downward, exposing

a large portion of the trachea. The tumor extended too far downward to make its total removal possible, however. Pieces of it were excised for histologic study and a long-stem No. 5 tracheotomy tube was inserted. After this procedure, the patient was able to breathe with less difficulty.

A posterior-anterior roentgenogram of the chest showed the tracheotomy tube in place and revealed a pronounced widening of the upper mediastinum (Fig. 8). There was no evidence of metastatic or parenchymatous lesions of the lungs. The heart shadow was of normal size,

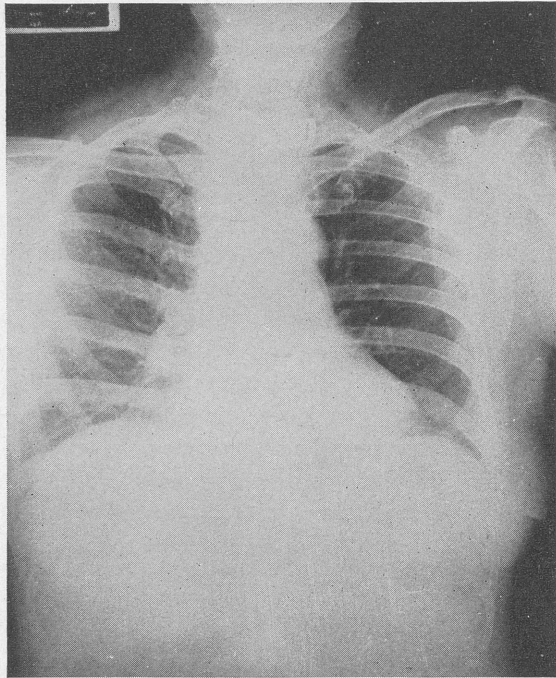


Fig. 8.—(Case 2) June 17, 1947—Posterior-anterior view of the chest showing the tracheotomy tube in place and marked widening of the upper mediastinum.

but it was apparently displaced somewhat to the left, possibly owing to scoliosis of the mid-dorsal spine or to the rotational position of the patient. A lateral view showed that the esophagus was distorted and greatly constricted at the site of the lesion in the trachea (Fig. 9). This observation suggested that the lesion might be primary in the esophagus.

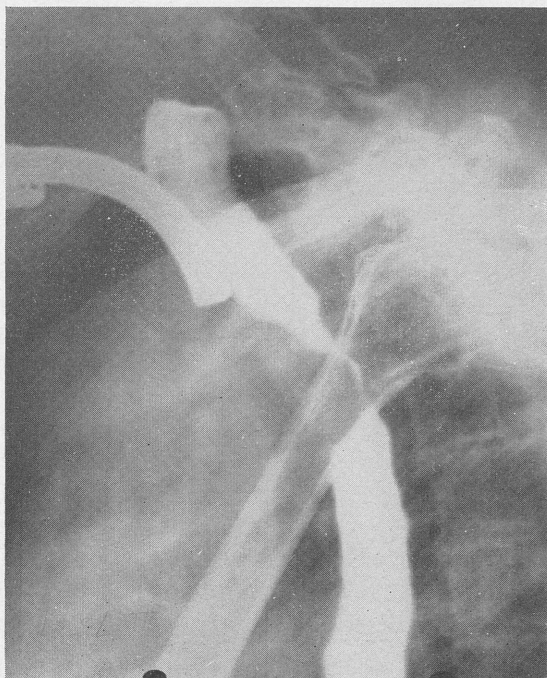


Fig. 9.—(Case 2) July 9, 1947—Lateral view of the chest showing distortion of the esophagus with marked constriction at the level of the lesion in the trachea.

Histologic examination of the tumor removed from the trachea showed that it was characterized by large, irregular, compact masses of neoplastic squamous epithelium, showing no keratinization. These cells were interspersed with a small amount of fibrous connective tissue stroma. The pathologic diagnosis was partially differentiated squamous-cell carcinoma.

Several bronchoscopic procedures were carried out, with removal of tissue and dilatation to provide a freer airway. Roentgen therapy also was instituted. This was administered through a single anterior field, and two anterior oblique fields, 10 centimeters in diameter. High-voltage radiation (200 kv), with a filter of 0.5 millimeters of copper, at 70 centimeters distance was used. The dosage received by the central thoracic field was 2500 r (in air) in thirteen days and the oblique fields each received approximately 1000 r (in air) during the following

twenty-nine days. Owing to the patient's poor condition and her poor tolerance of radiation, the dosage was inconsistent in both periodicity and quantity and was considered to be therapeutically ineffective.

Since this patient's condition was so critical at the time of admission and the disease so far advanced, it was to be expected that all treatment would be fruitless. Her course was steadily downward and she died on September 24, three months after she was first examined.

Autopsy showed a carcinoma of the trachea with direct invasion of the esophagus. The mucosa of both trachea and bronchi was thick, injected and covered with a foul, purulent exudate. The tracheal tumor was on the posterior surface and extended from just above the bifurcation upward for approximately 3 to 4 centimeters. The tumor was firmer than the surrounding tissue and showed ulceration. The portion of the trachea invaded by the tumor was adherent to the esophagus and similar ulcerated lesions were present on the mucosal surface of the esophagus at this same level, and 2.5 centimeters below. Histologic study of the tumor tissue from both the trachea and the esophagus showed the same structure as that found in the biopsy, that is, squamous-cell carcinoma without keratinization.

Comment—This case has little interest from the clinical point of view, since there was no hope of doing anything that would benefit the patient. It should be noted that at the same time this patient was first seen by the otolaryngologist, she was in extreme danger of immediate respiratory failure. In this respect this case of tracheal tumor is similar to others reported by Boemke and Moritz,⁴ Guttman,⁵ Cann,⁹ Fisher,¹² Dean,¹³ Harill,¹⁵ Robin and Mann¹⁶ and Calvet and Girou.¹⁷ Instances in which carcinoma of the trachea had reached a hopeless stage before the patient was examined were described by Brown, Weiss, Iglauer and Fine,¹ Fisher,¹² Dean,¹³ Miller,¹⁸ Keeney,¹⁹ Guerra Rosseau, Daumy Amat and Aguirre Medrano²⁰ and Muta and Ogata.²¹ That so many of the relatively few cases of tracheal tumor did not receive skilled medical attention until the patient had symptoms of respiratory collapse or until the malignant lesion was far advanced, or both, certainly indicates the need for persistent efforts to educate both the public and the medical profession regarding the significance of respiratory symptoms that should lead to the suspicion of a new growth in the respiratory tract.

From the pathologic standpoint, this case has a certain interest in illustrating that it may not be possible to determine definitely, when the lesion is far advanced, the site of the primary growth. Although the pathologist believed that this tumor originated in the trachea, with invasion of the esophagus, the lateral roentgenogram (Fig. 9) certainly was suggestive of a primary esophageal lesion.

Discussion—In preparing this paper, it was planned to make a thorough study of the literature that has appeared since Culp's²² excellent review, published in 1938, of cases reported through June, 1936, and to present a definite, up-to-date sum of the cases of primary carcinoma of the trachea. In attempting to do this, numerous difficulties were encountered that make such mathematical exactitude apparently impossible. The chief of these concerns the confusion of terms used by pathologists in describing tumors of the trachea and the disagreement among them as to whether certain lesions are benign or malignant. Until the pathologists agree more thoroughly on the nomenclature and criteria of malignancy of tracheal tumors, it would appear impossible for a clinician reviewing the reports to classify or count them exactly. Another difficulty in trying to determine the number of cases is that sometimes the same case is reported twice by two different authors, and there is even some discrepancy as to classification and enumeration of cases to be noted in successive reports by different authors from the same institution on the same clinical material. Furthermore, a few articles listed in the Index Medicus could not be obtained for review (Goto and Kyo,²³ Hinojar²⁴ and Khayutin²⁵). The Index Medicus for 1947 is not yet available, so there probably are articles that have appeared during the last year that could not be traced. It is possible also that some articles reporting supposedly benign tumors would contain cases that according to some classifications should be listed as malignant.

For these reasons, the number of cases to be added to the list since Culp's review can be only approximate. Of cases diagnosed as squamous-cell carcinoma, adenocarcinoma and carcinoma of high-grade malignancy, there have been approximately thirty-five to forty instances.^{1,2,8,12,13,14,16,18,19,20,21} In addition, there is a group of about twenty-five cases, with other pathologic designations, that some might classify as true cancer and others not.^{2,6,8-11,13,15,17,26-28} Since Culp listed 147 cases of primary carcinoma of the trachea found in the literature to June, 1936, the number of cases of this type so far reported is in the

neighborhood of 200. It should be remembered also that of the 147 cases listed by Culp as carcinoma, only eight-two had histologic verification. Practically all of the cases reported in the last decade have had histologic study by competent pathologists, but even so, numerous questions and confusions remain concerning their exact classification.

The type of lesion about which the most confusion exists is that variously designated as cylindroma, basal-cell carcinoma, mixed tumor of salivary type, cylindric epithelioma and adenoma. If these tumors are not of the same type, the reports about them certainly indicate striking similarities, and it would be hard to recognize any criteria for making definite distinctions among them. Some cases reported as low-grade adenocarcinoma apparently belong in the same general group. Some pathologists even report what appears to be the same type of lesion as sarcoma.

Eggston and Wolff,²⁹ in their monumental work on Histopathology of the Ear, Nose and Throat, list cylindroma as a benign tumor of the trachea and in discussing cylindroma generally have this to say: "A type of composite tumor of the seromucinous and salivary glands has been designated as cylindroma. This tumor is composed of anastomosing cords of broad masses of epithelial cells, surrounding irregular areas or gland-like structures which are filled with epithelial mucus. This should not be confused with the connective-tissue myxoid degeneration which occurs in the stroma. In this type of tumor the cells are suggestive of basal cells of the type of adenoid cystic epithelioma, . . .

"Cylindromas may occur in the lacrimal, seromucinous or salivary glands. The present authors see no justification for the continuation of this terminology, as it represents a type of tumor where there are masses of mucoid secretion surrounded by epithelial cells, a common finding in the mixed or composite tumors in the upper respiratory tract . . . It has been well agreed among students of the mixed tumors of the upper respiratory region that basilomas and cylindromas represent, biologically and clinically as well as from a microscopic standpoint, one and the same tumors and, therefore, it would be well if these terms were dropped." Eggston and Wolff suggest that it would simplify the situation if all these tumors were spoken of as adenomas of their respective glandular structures.

On the other hand, Tinney, Moersch and McDonald,² in reporting a series of twenty-seven cases of carcinoma of the trachea observed at the Mayo Clinic, classify cylindroma among the distinct groups of malignant tumor, although their clinical records show that in this group the survival rates far exceeded those for other types of cancer. The Mayo Clinic authors say that cylindroma is sometimes confused with adenoma but presents a different pathologic picture. It tends to produce mucus while adenoma does not. Use of mucicarmine stain may make the distinction. Adenoma tends to be more vascular. Mixed tumor also is sometimes confused with cylindroma but this has, in addition to epithelial elements, connective tissue which is undergoing myxomatous change.

Another detailed discussion of cylindroma has been presented by Boemke and Moritz.⁴ They noted that one school of pathologists has regarded these tumors as of mesenchymal origin, while another has claimed that they are epithelial growths, and still another that they are mixed tumors, consisting of both mesenchymal and epithelial elements. These authors offered evidence to prove that cylindromas are of epithelial origin. They emphasized that the clinical literature offers no real information concerning cylindroma of the respiratory organs, since, because of mistaken and confused histologic interpretations, many tumors of this type have been considered as carcinomas or endotheliomas and described as such. They concluded that although these tumors almost always are benign, they often show infiltration and eventually metastases may supervene.

This last statement, which the current reports seem to corroborate, furnishes the clue to the confusion regarding these growths. It would appear that this group of neoplasms, although essentially benign, may undergo malignant change eventually and hence must be considered as potentially, if not actually, malignant. Nevertheless, some distinction should be made between this group of questionable or low-grade malignancy and the more rapidly developing, poorly differentiated types of neoplasm. This distinction is essential, from the clinical standpoint, in evaluating the probable results of treatment.

Although the results of treatment will be least satisfactory in patients with tumors of high-grade malignancy, it should be emphasized that most patients with tracheal tumors of all kinds die as the result of

respiratory failure because of obstruction. Any tumor which grows large enough to obstruct the tracheal airway will have a lethal effect on the patient, no matter how benign it may be histologically. Since this is so, and since some of the benign tumors apparently are potentially malignant, this again emphasizes the need for bronchoscopic examination of all patients whose symptoms suggest a tracheal obstruction, and for prompt and radical removal of any tracheal tumor. If this were generally done, at least some cases of cancer of the trachea should be prevented and the results of treatment, even of tumors that are frankly malignant, should be improved. Since so many of the reported case of tracheal carcinoma have been so far advanced when the patients were first observed, it is extremely difficult to evaluate the efficacy of treatment in this condition. Results in a few cases, however, including the first case presented here, would seem to indicate that the chances for prolonging the survival period beyond that usually reported appear at least promising. This hope is strengthened by the fact, which has been promoted by all who have written on the subject, that in the majority of cases of carcinoma of the trachea, the metastases are only regional. The only exceptions noted in the recent reports reviewed were the patient reported by Keeney¹⁹ who had widespread generalized metastases even involving the skin and the one described by Fisher¹² who had metastasis to the bones of the arm. In some of the cases in which the tumors were of high-grade malignancy, death occurred from respiratory obstruction before any metastasis or extension outside the trachea had occurred.

The general facts and features in regard to tracheal carcinoma have been thoroughly discussed by other authors. Hence the details regarding the extreme rarity of tracheal carcinoma in relation to bronchial and other cancers, the dependence of symptoms on mechanical factors incident to the situation of the tumor, age and sex incidence, the predilection of tracheal cancers for the postero-lateral walls of the lower third, near the bifurcation and the history of early reports will not be reviewed here.

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DISCUSSION

EDWIN N. BROYLES, M.D.: I have enjoyed Dr. Engler's paper.

I have reported on different occasions two cases of carcinoma of the trachea, one in a patient that we fulgurated that lived in comparatively good health for eleven years. He came for fulguration at regular intervals every three or four months, kept his job, worked on, and, although the carcinoma was present at all times, took eleven years for that carcinoma to grow to such a point that we could not keep his tracheal lumen open.

Another case was in a young woman in the mid-trachea. The trachea seemed so filled with carcinoma that she could hardly breathe, I made one very bad mistake, as I thought that she was a hopeless case and therefore gave her a little x-ray treatment. She shut off and died. At autopsy there was only a thin outgrowth of carcinoma that filled up her trachea. If we had a laminagraph done to show the extension of that tumor, I am sure we could have saved that case. So therefore in cases of carcinoma of the trachea, especially those where you can't see how far they extend down but can only see the upper portion of that growth, it is very important to have a laminagraph to see the extent of the growth.

REPORT OF POST-OPERATIVE COURSE OF SUPERICHONDRAL TOTAL LARYNGECTOMIES*

EDWARD M. WALZL, M.D. (*By Invitation*) AND EDWIN N. BROYLES, M.D.

In 1938 Dr. Samuel J. Crowe and Dr. Edwin N. Broyles published the details of a one-stage operation for total laryngectomy in selected cases of cancer of the larynx. This technique was carefully planned so as to preserve as much as possible of the undiseased structural elements of the neck and to provide the best possible closure of the pharyngeal defect resulting from the removal of the larynx.

These authors emphasized that the cases suitable for this type of operation are only those in which the cancer has not invaded the cartilages, false cords, or ventricles, arytenoids or epiglottis, and has not extended into the muscles or glands of the neck.

A number of patients have been operated upon with this technique and it now seems advisable to review the results obtained, especially with regard to the post-operative course of the patients. Some modifications in technique of the original operation are also discussed.

SUMMARY OF OPERATIVE PROCEDURES AND MODIFICATIONS IN TECHNIQUE

It is important previous to operation to have a thorough medical check-up to remove any foci of infection in the upper air passages, especially in the teeth and gums.

The preliminary stages of the operation can be performed under local anaesthesia. During actual removal of the larynx, the patient is anesthetized with pentothal sodium.

The operative procedure used at present differs from the original description in making use of the mucous membrane on the dorsal surface of the epiglottis to obtain a complete mucous membrane closure of the pharyngeal defect. An outline of the stages of the operation follows: The patient is placed on the operating table with the foot of the table

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elevated. The patient's head is extended as much as possible. A sand bag under the shoulders may be necessary to obtain sufficient extension. After local infiltration of the skin and deep tissues of the front of the neck with procaine, a midline incision is made from the hyoid to about one centimeter above the sternum. The strap muscles are separated, and the thyroid is divided. At this point the patient is anesthetized with pentothal sodium. The inner tracheotomy tube is then inserted in an opening made in the trachea below the second ring. Oxygen is insufflated through this tube. The perichondrium is elevated from the thyroid cartilage divided. The thyro-hyoid membrane is then divided.

The incision through the thyro-hyoid membrane into the pharynx in the present technique is made directly through the base of the epiglottis leaving this structure for the time being attached to the base of the tongue. In the original description of the operation, the opening was made between the epiglottis and the base of the tongue. We feel that leaving the epiglottis attached to the base of the tongue at this stage is advantageous for several reasons. First a better view into the pharynx is afforded; and second, the mucous membrane on the dorsal surface of the epiglottis can be utilized later in closing the pharyngeal defect.

Under direct vision, the inferior constrictors are now freed from the larynx on each side, and the incision in the thyro-hyoid membrane extended down and across the posterior face of the cricoid. The larynx is then freed from the esophagus and removed. The stump of the trachea is anchored to the sterno-hyoid muscles. The mucous membrane on the upper surface of the epiglottis is incised and elevated, and the epiglottis removed. The mucous membrane from the posterior face of the cricoid is then sutured to the mucous membrane from the upper surface of the epiglottis and anchored to connective tissue at the upper angle of the wound. The remaining defects in the mucous membrane of the pharynx on each side are closed with inverting sutures. Each suture includes muscle which acts as support to the suture line. The wound is irrigated with tyrothricin and sulfathiazole in suspension. The strap muscles are plicated, and the skin closed with interrupted sutures. A small protective wick is placed in the lower part of the wound. The skin around the tracheotomy opening is inverted into the trachea with interrupted sutures.

In any operation of this magnitude, success depends on following

basic surgical principles. Traumatization of tissues with clamps or retractors, the use of high frequency cautery for hemostasis, or the leaving of pockets or dead space predispose to poor healing and wound infection.

POST-OPERATIVE COURSE OF LARYNGECTOMIZED PATIENTS

Post-operatively these patients require little care. They are permitted to drink fluids the day of operation and are usually up in a wheel-chair the second or third post-operative day. Supplementary intravenous fluids are given for the first two days, after which sufficient amounts of high caloric liquid diet can be taken by mouth. There is very little coughing, and little suctioning is required if care is taken to prevent blood from getting into the trachea at the time of operation. Since there is little coughing, a special nurse is usually not essential. The small protective drain is removed the day after operation. Penicillin is usually given prophylactically. However, in cases in which it was omitted, there were no more complications than in those in which it was given.

Chart 1

PATIENT	DIAGNOSIS	CHEMO- THER.	POST OPERATIVE COURSE						POST OPERATIVE DAY		
			SHOCK	COUGH	FEVER	WOUND INFE- CTION	FISTULA	NASAL FEED- ING	LIQUID DIET	OUT OF BED	DISCHARGED
H.K. AGE 63	SQUAMOUS CARC.	SULFA DIAZ.	0	SLIGHT	SLIGHT	0	NO	YES	2 ND.	8 TH.	16 TH.
O.P. AGE 49	SQUAMOUS CARC. LYMPHOS.	"	0	"	"	0	NO	NO	5 TH.	---	33 RD.
E.P. AGE 47	SQUAMOUS CARC.	SULFA IN WOUND	0	"	"	0	NO	NO	2 ND.	4 TH.	13 TH.
G.F. AGE 56	SQUAMOUS CARC.	SULFA DIAZ.	0	"	"	YES	YES	NO	2 ND.	8 TH.	24 TH.
A.H. AGE 62	SQUAMOUS CARC. DIABETES	SULFA THIAZ.	0	"	"	YES	YES	YES	9 TH.	5 TH.	25 TH.
J.G. AGE 61	EPIDERM. CARC. A.C.V.D.	"	0	MOD.	MOD.	0	NO	YES	2 ND.	4 TH.	16 TH.
J.W. AGE 56	EPIDERM. CARC.	NONE	SLIGHT	"	"	YES	YES	YES	2 ND.	4 TH.	37 TH.
C.W. AGE 69	SQUAMOUS CARC.	SULFA DIAZ.	0	SLIGHT	SLIGHT	"	"	"	3 RD.	3 RD.	44 TH.
T.S. AGE 77	SQUAMOUS CARC.	NONE	0	"	"	0	NO	NO	4 TH.	4 TH.	21 ST.
J.M. AGE 52	EPIDERM. CARC.	NONE	0	"	"	0	NO	NO	3 RD.	4 TH.	22 ND.

Chart 2

PATIENT	DIAGNOSIS	CHEMO- THER.	POST OPERATIVE COURSE						POST OPERATIVE DAY		
			SHOCK	COUGH	FEVER	WOUND INFE- CTION	FISTULA	NASAL FEED- ING	LIQUID DIET	OUT OF BED	DISCHARGED
L.M. AGE 68	SQUAMOUS CARC.	TYRO. PEN.	0	SLIGHT	SLIGHT	0	NO	NO	2 ND.	4 TH.	9 TH.
J.F. AGE 45	"	"	0	"	"	0	"	"	2 ND.	3 RD.	12 TH.
J.M. AGE 80	"	"	0	"	"	0	"	"	3 RD.	9 TH.	16 TH.
I.B. AGE 63	"	"	0	"	"	0	"	"	2 ND.	5 TH.	7 TH.
F.E. AGE 54	"	"	0	"	"	0	"	"	2 ND.	3 RD.	12 TH.
A.P. AGE 64	"	"	0	"	"	0	"	"	3 RD.	3 RD.	15 TH.
M.S. AGE 53	"	"	0	"	"	0	"	"	2 ND.	3 RD.	10 TH.

Chart 3

PATIENT	DIAGNOSIS	CHEMO- THER.	POST OPERATIVE COURSE						POST OPERATIVE DAY		
			SHOCK	COUGH	FEVER	WOUND INFE- CTION	FISTULA	NASAL FEED- ING	LIQUID DIET	OUT OF BED	DISCHARGED
L.R. AGE 61	SQUAMOUS CARC.	TYRO. SULFA	0	SLIGHT	SLIGHT	0	NO	NO	3 RD.	4 TH.	10 TH.
H.B. AGE 24	"	"	0	"	"	0	"	"	2 ND.	4 TH.	13 TH.
J.F. AGE 57	"	"	0	"	"	0	"	"	3 RD.	4 TH.	9 TH.
A.B. AGE 57	"	"	0	"	"	0	"	"	2 ND.	3 RD.	9 TH.
W.F. AGE 49	"	"	0	"	"	0	"	"	2 ND.	4 TH.	8 TH.
F.T. AGE 30	"	"	0	"	"	0	"	"	2 ND.	5 TH.	11 TH.
J.J. AGE 59	"	"	0	"	"	0	"	"	1 ST.	5 TH.	10 TH.
L.D. AGE 52	"	"	0	"	"	0	"	"	3 RD.	6 TH.	9 TH.
A.H. AGE 54	"	"	0	"	"	0	"	"	3 RD.	4 TH.	9 TH.
A.K. AGE 58	"	"	0	"	"	0	"	"	2 ND.	3 RD.	9 TH.

The post-operative course of the last twenty-eight consecutive total laryngectomies performed at The Johns Hopkins Hospital are summarized in Charts 1 and 2. These were all cases of squamous carcinoma. A number of these patients were elderly and feeble; a number also had serious ailments such as diabetes, arteriosclerosis, heart disease, etc. The operative mortality was zero. There were no cases of shock, post-operative hemorrhage, or pneumonia. In none was there more than a degree or so rise in temperature at any time. Of the cases summarized in Chart 1, there were four with some breakdown of the wound. One of these patients had received considerable irradiation therapy previous to operation, and one was a severe diabetic. In all of these four cases, the wound had healed before the patient left the hospital.

In the second series of cases (Chart 2), the wounds were irrigated with tyrothricin 1:100 during the operation, at the time of the closure of the pharynx. Comparison of the results in these cases with the previous group in which tyrothricin was not used (Chart 1) shows a great decrease in the length of the patient's stay in the hospital as well as a complete absence of wound infection and formation of fistulae.

In the cases shown in Chart 3 systemic penicillin was given in addition to the local use of tyrothricin. The results are essentially the same as those shown in Chart 2.

The longest stay of any of the patients in the last two groups post-operatively has been sixteen days; the shortest, seven days.

The main criticism which has been directed at the operation described in 1938 by Dr. Crowe and Dr. Broyles is that there is certain to be contamination of the wound by secretions from the mouth during the removal of the larynx. (Vasconcelos et al.). In order to circumvent this hazard, a technique was developed in which a heavy clamp is used to close off the pharynx before the larynx is removed. In this technique the larynx is dissected from below upwards, the hyoid is sectioned and after the larynx is freed from the inferior constrictors, a clamp is applied across the mucous membrane. After this, the larynx is removed. The mucous membrane is sutured before the clamp is released.

There are certain very obvious disadvantages to this procedure. First, there is danger of aspiration of blood into the trachea during the whole period of dissection of the larynx. Second, the hyoid must be sectioned. Third, it is impossible to preserve as much of the normal

mucosa of the pharynx as with the Crowe-Broyles technique. Fourth, the pharyngeal defect is pulled together under tension and is closed longitudinally instead of horizontally. Fifth, there is danger of leaving devitalized tissue in the suture line as a result of trauma from the clamp.

In reviewing the results obtained by the Crowe-Broyles technique when chemotherapy is employed, one must conclude that the danger of wound infection from opening into the pharynx is unimportant. We feel that the crushing of the mucous membrane by the clamp, and closure of the mucous membrane under tension is much more conducive to breakdown of the wound than is contamination of the wound from secretions from the mouth. We believe that the advantages of preservation of the anatomic structures of the neck which are gained by the modified Crowe-Broyles technique make this the operation of choice in selected cases for total laryngectomy.

SUMMARY

1. A modification of the Crowes-Broyles technique for total laryngectomy is described which permits complete mucous membrane closure of the pharyngeal defect after removal of the larynx.

2. The post-operative courses of the last twenty-eight consecutive total laryngectomies at the Johns Hopkins Hospital are reviewed. The effectiveness of prophylactic use of tyrothricin on prevention of wound infection is demonstrated.

3. It is concluded that the operation as described reduces to a minimum post-operative complications and greatly shortens the recovery period.

DISCUSSION

LOUIS H. CLERF, M.D.: I have been interested in superichondral technique for if one wishes to utilize the ribbon muscles of the neck and the perichondrium of a part of the larynx in closure of the wound, the question immediately arises: is not there a certain hazard in connection with this? That is what I am particularly interested in. That is the one thing I question about this approach. I would like to have the essayist re-emphasize that particular aspect.

LEROY A. SCHALL, M.D.: May I congratulate the essayists on their very excellent presentation? However, I would like to point out to them that the utilization of the mucous membrane of the epiglottis was first used to my knowledge by Dr. Harry A. Barnes about fifteen years ago and that I have used the

mucous membrane of the epiglottis in six cases to help close the pharyngotomy opening. Those six cases promptly developed recurrence and are all dead, so I no longer utilize that mucous membrane.

JAMES M. ROBB, M.D.: I should like to ask about the matter of recurrences.

E. M. WALZ, M.D.: (Closing) In regard to Dr. Clerf's remarks, I think it should be emphasized again that probably the success of treatment of laryngeal cancer depends upon choosing the procedure suitable for the specific case. Certainly, there are cases where laryngofissure can be performed with a good chance of success. There are also cases where the so-called narrow field laryngectomies can be done without jeopardizing the patient's chance of a cure and at the same time, getting the patient out of the hospital and back to work quickly with a good esophageal voice and thus minimizing the psychological trauma. Such cases should be studied extremely carefully, just as one carefully studies a case before deciding on a laryngofissure.

Certainly, in cases where there is even a suggestion of extension, where the tumor mass has gotten large or may have extended up into the pre-epiglottic space, the operation selected should be radical enough to remove all the growth. This may necessitate the removal of the hyoid bone, block dissection of the larynx and adjacent muscles and dissection of the glands of the neck. I should like to emphasize again, however, that if one is careful in selecting his cases that there are many in which the narrow field operation will give the best result without decreasing the chance of a cure.

I have avoided considering the percentage of cures since many of the cases have only been operated a relatively short time. However, of the ones that have been followed five years or more, I think there is a fairly low recurrence rate.

Dr. Broyles is much better capable of answering this than I am. My own impression is that the ones that have recurred have all recurred in the crico-thyroid membrane region, and those that have recurred were not really suitable for this type of operation. I have an impression that of those that have been followed five years, there is probably between 65 to 70% cure. Is that your impression, Dr. Broyles?

DR. BROYLES: Yes.

PRESENTATION OF INSTRUMENTS
SET OF BRONCHOSCOPIC TUBES WITH
OPTICAL TELESCOPES

EDWIN N. BROYLES, M.D.

BALTIMORE, MD.

DR. EDWIN N. BROYLES: Mr. Chairman, any means that the laryngologists and the bronchoscopists can use to enlarge their visual field will also aid them in diagnosis of diseases and treatment of patients, so I would like to present for your consideration a set of bronchoscopic tubes and with them an optical system of telescopes through which right angled views are obtained, retrograde views or direct magnification.

(Slide) This is a picture of the tube and telescope. The top telescope you can see is a right angle one which enables one to get a beautiful magnified view of the upper lobe bronchi, not only the main upper lobe bronchus but also the subdivision. The second telescope is one which gives a retrograde view. This can be used in the trachea or the larger primary bronchi where you are enabled to pass the scope beyond the lesion and get a view from the lower side as well as from the direct upper side. The third telescope is one that gives a direct view and its division magnified as you look down straightaway. Below that there is a set of a telescope connected with a biopsy forceps so in cases where we would have to be extremely careful or want to be delicate in the removal of tissue down in the lower lobes, this can be used in association with the telescope. These tubes come in 3, 4, 5, 6, 7, 8, 9 mm. in diameter. On the right are shown the little attachments through which compressed air is blown across the proximal end of the tube to prevent fogging of the operator's glasses.

I thank you very much. These are manufactured by the A.C.M.I. Co.

Memorials



FRANCESCO LASAGNA

(1882-1946)

Professor Francesco Lasagna, Corresponding Fellow, died on June 4, 1946, in Milan, Italy, after a prolonged illness. He was born in Parma, Italy, on July 21, 1882. His father, Desgratrias Lasagna, was a noted artist whose canvases hang in many European galleries. His mother was an Englishwoman, Marie Williams Lasagna, and born in London. Professor Lasagna spent his early life in Parma where he received his preliminary education at the Lyceum. Entering the University of Parma in 1900, he received his degree in medicine in 1906, graduating at the head of his class and with "Cum Laude." He at once became Assistant in Pathological Anatomy and Assistant in the Neuro-Pathological Clinic of the University of Parma. In 1907, he became Associate in Pathological Anatomy at the same institution. During the years 1906-1907 he performed 1120 autopsies and completed the histological preparations in each case. By 1908 when he began the study of Laryngology with Gradenigo, he had already published three important original papers. He was appointed Assistant to the Otorhinolaryngological Clinic of the University of Torino and Assistant to the Anatomo-Pathologic Institute in 1908, and Associate Professor of Otorhinolaryngology at the University of Torino in 1911. He was the first to give courses of instruction in otorhinolaryngology at the University of Torino. His clinical duties at this time were divided between the Parma Hospital and the Salso Hospital and he was appointed Director of the Otorhinolaryngological Ambulatorio of the Public Health Service of Italy.

In 1912, he became Assistant Professor of Anatomy at Torino, a position he held until 1919. In 1915, during World War I, he organized the otolaryngological facilities of the military department with headquarters in Parma. Later, he was assigned to numerous combat centers in the capacity of consultant in otolaryngology, serving first as a Captain and later as Major in the Medical Corps of the Italian Army. With the entrance of Italian troops into France for service on the Western Front, he was sent to France as a liaison officer and participated in many conferences dealing with military medicine. For his services in France he

was decorated with the Croix de Guerre, the Legion of Honor, and the Lavalier Corona Italia.

Upon his return to civilian life, he founded the Oto-Rhino-Laryngological Clinic at the University Hospital and expanded this until it now occupies the the entire west wing of the New Polyonic Clinic. As Professor of Otolaryngology, he also established a post-graduate school in this building. Here he developed facilities for research and teaching in audiometry, esophagology, bronchology, plastic and reconstructive surgery, fenestration surgery and physiology and clinical anatomy. Always interested in the basic science, especially histology and pathology, he fostered the establishment of excellent laboratory facilities. In 1942, he succeeded Professor Torrigiani as Director of the Otorhinolaryngological Clinic of the University of Milan. In addition to his military duties as Colonel and Director of the otolaryngological facilities of the Italian Army, he carried on his civilian duties and a heavy load of teaching until a train of misfortunes fell. During World War II he first saw his clinic destroyed by bombardment and soon thereafter his wife, the former Margaret Gaspar, of Budapest, Hungary, whom he married in 1921, died. A prolonged febrile illness reduced his activities until his death in Milan on June 4, 1946.

Of his published articles and monographs, more than 100 deal with anatomy, physical, pathology and pathological anatomy in the field of otolaryngology. His contributions to the literature on ozena, rhinoscleroma and the reproduction of scleroma in animals were classics. He was a prolific contributor to medico-legal publications and for a time became interested in the application of roentgen examination and therapy to his special field. His researches in stenosis of the esophagus and in pneumonography received general recognition, and he was founder and editor of the *Archivo Italiano della Mallattie-dell' Esofago, Trachea Bronchi*. Above all, he maintained a constant interest in balneology and was the founder and editor of the *Giornale Italiano di Idroclimatologia*. From 1913 to 1933, he was active in popularizing Italian thermal establishments, especially Salsomaggiore, and represented the Italian Government at the International Congress of Balneology in 1912. He organized the First Italian Congress of Otolaryngology held in 1912.

Professor Lasagna was a Fellow of the Medical Society of Parma, the Otolaryngological Society of Italy, the Italian Society of Medical

Hydrology, the Otolaryngological Society of Hungary, the American-Esophagological Association, and an Honorary Fellow of the Otorhinolaryngological Society of France, the Society Ospedali Parigi, the Otorhinolaryngological Society of Spain, the Otorhinolaryngological Society of Poland and the Magiara Otolaryngological Society. He was a corresponding Fellow of the American Laryngological Association and the American Otological Society. He served as vice-president of the Otorhinolaryngological Society of Italy from 1928 to 1930, secretary-general of the International Committee for the Control of Scleroma in 1932, president of the Italian-American Medical Association in 1930 and State Consultant for the Royal Thermal Institute of Salsomaggiore since 1920.

An inveterate traveller, he visited all of the capitals of Europe and represented his government at continental and international otolaryngological meetings. In 1911 he visited every important clinic in Europe and in 1930 visited the United States and Canada. During these trips he made an exhaustive study of the medical facilities in foreign countries. He was especially interested in developments in audiology, maxillo-facial surgery and bronchoesophagology. Following his sojourn at the Chevalier Jackson Clinic he began the publication of the *Archivo Broncoscopia Italiano*. Always concerned with fostering closer contacts between Italian and American otolaryngologists, he made every effort to strengthen the ties during his visit to the United States. While in New York he founded the Italo American Medical Association and donated a medical library to the Columbus Hospital of New York City.

Professor Lasagna was a patron of all the liberal arts. His official position in the Italian Red Cross and numerous tuberculosis and cancer organizations indicate his wide philanthropic interests. As a teacher, he was inspiring, paternalistic and ever ready to give encouragement or advice in purely scientific matters or in the affairs of everyday life. A great leader has passed from the stage of Italian Medicine.



FRANK B. KISTNER, M.D.

1874-1947

Dr. Frank B. Kistner was born in Indianapolis, Indiana, on December 7, 1874 and died at Astoria, Oregon, on September 25, 1947.

His preliminary education was obtained in the Indianapolis public schools, and he obtained his medical degree from the Medical School of the University of Indiana in 1898. Beginning practice in Heppner, Oregon, in 1899, he later engaged in post-graduate studies at the New York Eye and Ear Infirmary and at the Graduate School of Medicine of the University of Pennsylvania in 1910. Upon his return to Oregon he engaged in the practice of otolaryngology in Portland.

Dr. Kistner was Clinical Professor of Otolaryngology at the School of Medicine of the University of Oregon. He was a member of the staff at the Portland Eye, Ear, Nose and Throat Hospital and the Doernbecher Memorial Hospital for Children, and consultant at the Multnomah County Hospital, the Good Samaritan Hospital and St. Vincent's Hospital.

His society affiliations included the Portland City and County Medical Association, the Oregon State Medical Society, the Portland Academy of Medicine, the Oregon Academy of Ophthalmology and Otolaryngology, the American Medical Association, the American College of Surgeons, the American Academy of Ophthalmology and Otolaryngology, the American Otological Society and the American Laryngological Association.

Agriculture constituted his principal non-medical activity and to it he devoted much time and energy.

Surviving him are his wife, Mrs. Letitia Estelle Kistner, a son, Frank B. Kistner, Jr., and two daughters, Mrs. Lawrence Shaw, and Mrs. Richard Givens.

Dr. Kistner was for many years a most brilliant and outstanding teacher of clinical otolaryngology in the University of Oregon Medical School. He was an accomplished and daring operator upon the nasal accessory sinuses, and one of the earliest proponents of the focal infection theory from this source.



WARREN B. DAVIS, M.D.

1881-1947

Warren B. Davis, son of Luther A., and Mary Donnohue Davis, was born on the Cave Spring Stock Farm, Jessamine County, Kentucky, on September 6, 1881. He died in Philadelphia on July 7, 1947. His father was a great, great nephew of James Wilson, one of the signers of the Declaration of Independence, and Pennsylvania's first member of the United States Supreme Court. His mother was a great niece of Daniel Boone, and a direct descendant of Oliver Cromwell.

His early education was obtained in the public schools of Jessamine County, Kentucky, and he graduated with first honors in 1895. From 1898 to 1905 he took a special scientific course at Kentucky University, now Transylvania University.

He entered Jefferson Medical College in September, 1906, and was graduated with honors in 1910. He served as an intern at the Jefferson Medical College Hospital for eighteen months and at the completion of his internship was awarded the Corinna Borden Keen Research Fellowship. During the following year he carried out research work in the laboratories of Professor Ludwig Pick of the University of Berlin, Germany, and in the Friedrichstein Krankenhaus. The result of this work was published in a monograph entitled "Development and Anatomy of the Nasal Accessory Sinuses in Man." He entered private practice in Philadelphia in January 1913, at which time he was appointed Clinical Assistant in Surgery at the Jefferson Hospital.

In 1931, with J. Parsons Schaeffer, he was awarded the Gold Medal, Class II, for excellence of presentation of research illustrating the embryology, development and anatomy of the paranasal sinuses, at the Annual Meeting of the American Medical Association in Philadelphia.

In 1936, he was made Clinical Professor of Plastic and Reconstructive Surgery at Jefferson Medical College, and Maxillofacial Surgeon at Jefferson Hospital.

His professional career was outstanding for his work in harelip and cleft palate surgery, for which he received wide acclaim, both in this country and abroad. His contributions to the literature in this field are

of particular note. He was a founder and first editor of the Journal of Plastic and Reconstructive Surgery.

During World War I, Doctor Davis served on the War Department's Advisory Board of Pennsylvania, and later as a Captain in the Medical Corps of the U. S. Army, in charge of the School of Maxillofacial Surgery. In World War II, he served as a member of the National Faculty for Plastic and Maxillofacial Surgery.

He received an honorary degree of Doctor of Science from Georgetown College, Kentucky, in 1933. He was president of the Alumni Association of Jefferson Medical College in 1942, and from 1941 to 1947 was a member of the Pennsylvania State Board of Plastic Surgery.

Dr. Davis was Fellow of the American College of Surgeons, the American Laryngological Association, the American Academy of Ophthalmology, the American Society of Plastic Surgeons, the Association of Military Surgeons of the United States, the American Medical Association and the Academy of Surgery of Philadelphia. He was a past president of the American Society of Plastic and Reconstructive Surgery.

Elected a Fellow of the College of Physicians of Philadelphia in 1916, he took an active part in the activities of the Section on Otolaryngology, serving as its chairman in 1939 and 1940, and as a member of the Executive Committee from 1941 to 1946.

Jefferson Hospital was his principal workshop, and his colleagues will always remember his enormous capacity for work and his uncanny skill. A giant of a man, with commanding appearance, his unlimited energy was tirelessly exerted in his profession. As Clinical Professor of Oral Surgery at the Jefferson Medical College, he acquired international recognition as a leader in maxillofacial surgery. He was Consulting Otorhinolaryngologist to the St. Agnes Hospital, Assistant Surgeon to the Frankford Hospital, and Consulting Maxillofacial Surgeon to the Kensington Hospital for Women.

He was a generous and gracious host, whose dinners invariably included the youngest as well as the oldest of his colleagues and revealed his appreciation of the fine art of delightful living. To his juniors he was ever tolerant, sympathetic and helpful.

Doctor Davis is survived by his wife, the former Ada Springer, whom he married in 1913, and four sons, Doctor J. Wallace Davis, J. Leslie Davis, Warren S. Davis and Richard E. Davis.

GEORGE E. SHAMBAUGH, M.D.

1869-1947

Dr. George Elmer Shambaugh, one of our distinguished Emeritus Fellows, died on November 30, 1947, of carcinoma of the lung. He was born in Iowa on November 15, 1869 and received his M.D. degree from the University of Pennsylvania in 1895. He spent the next two years abroad studying in Berlin and Vienna and on his return became affiliated with the University of Chicago where he engaged in research and with Rush Medical College in the department of otolaryngology. His fine talent for teaching was soon recognized and he rapidly rose in rank until in 1916, on the retirement of E. Fletcher Ingals, he became head of the department, which post he held until 1935 when ill health forced him to retire.

Dr. Shambaugh's excellent contributions to the otologic literature soon gained him universal recognition and he became a member of all the major societies in otolaryngology attaining the presidency of the American Otological Society, the American Laryngological Association, the Chicago Laryngological and Otological Society and the Institute of Medicine, as well as the chairman of the Section on Otology and Laryngology of the American Medical Association. His original research in otology led to the publication of a paper on the circulation of the internal ear, which appeared almost simultaneously with that of Sieberman's on the same subject and which attracted world-wide recognition. As a result he was awarded the Baron de Lenval Prize for research and later was one of the few Americans to be accorded a fellowship in the International Collegium, an honorary society of the highest degree.

Dr. Shambaugh's interest in teaching and in advancing the standards in our specialty was evidenced by two outstanding activities. The first was his establishing a one year post-graduate course in otolaryngology at the University of Chicago and Rush Medical College, one of the first of its kind in the Middle West and forerunner of the present day Year's Basic Science Course. The second was his zeal and interest in the establishment of the American Board of Otolaryngology on behalf



of which he made strong appeals to the various specialty societies which eventually joined in the undertaking.

When the Archives of Otolaryngology was first established by the American Medical Association, Dr. Shambaugh became its first Editor, a post which he filled with great distinction up to the time of his retirement.

Dr. Shambaugh was the quiet serious type, careful and conservative in his utterances but strong and forceful in his convictions. He was modest and unassuming and gave one the impression of great inner strength. He was not a prolific writer but his contributions were all noteworthy for thoroughness and exactness, revealing depth of thought and understanding. There was never any mistaking where he stood on any proposition.

When ill health forced him to retire from active practice he continued his interest in medicine by constant reading and indulged his fondness for music by attending concerts and recitals. Up to the time when he became almost totally incapacitated he could be seen regularly at the Symphony concerts which he seemed to relish with deep satisfaction.

Dr. Shambaugh was married to Edith Capps in 1901 and their union was blessed with six children, three of whom are physicians, Dr. George E. Jr., who is known for his fine work in fenestration, Dr. Phillip, a successful surgeon and Dr. Benjamin who is just completing his internship at an eastern hospital.

It is unfortunate indeed that Dr. Shambaugh was forced to retire so long ago, thus depriving the younger generation of the pleasure of knowing him and enjoying his fine discussions. Those of us who knew him in the days when he was active remember him with deep affection and respect and will always cherish the fine example of the true scientific spirit which he always manifested.



ARTHUR WILLARD WATSON

(1858-1948)

Dr. Arthur W. Watson, Emeritus Fellow, elected in 1892, died on February 17, 1948, at Philadelphia, Pa., where he had lived throughout his professional life. He was born in Rochester, N. Y., September 3, 1858. His father, Rev. Benjamin Watson, D.D., was a prominent clergyman who lived in Philadelphia most of his active life excepting for a short interval spent in Rochester at the time of Dr. Watson's birth. His mother was the former Lucy A. Willard of Hartford, Connecticut. Dr. Watson's early life was spent in Philadelphia where he obtained his preliminary education at the Episcopal Academy. He was granted the degree of Doctor of Medicine by the School of Medicine of the University of Pennsylvania in 1880 and immediately embarked upon an internship at the Episcopal Hospital of Philadelphia. He began the study of laryngology with Dr. J. Solis-Cohen and was associated with him for many years. In 1885 he was appointed Professor of Diseases of the Nose and Throat at the Polyclinic Hospital, a position largely concerned with postgraduate teaching. The facilities of the Polyclinic Hospital were later merged with the Medico-Chirurgical Hospital and School of Medicine. When the latter was in turn merged with the Graduate School of Medicine of the University of Pennsylvania, Dr. Watson became Professor of Otolaryngology in the Graduate School until his retirement in 1926.

He was admitted to Fellowship in the American Laryngological Association in 1892 and served as First Vice-President in 1912. As a Fellow of the College of Physicians of Philadelphia, he served as Chairman of the Section on Otolaryngology in 1915. He was a staff member of the Mt. Sinai Hospital, the Jewish Hospital, the Philadelphia Home for Incurables and the Philadelphia Home for Consumptives.

Dr. Watson was a pioneer in septal surgery. He devised the Watson operation which preceded the current submucous resection. He also designed the Watson knife and the Watson snare.



GEORGE LORING TOBEY, M.D.,

1881-1947

Dr. George Loring Tobey was born in Lancaster, Mass., June 3, 1881, and died in Boston, May 20, 1947.

The son of a physician, he spent his early life in Lancaster, and graduated from the Clinton High School there. He was granted the degree of M.D. at Harvard in 1903 and immediately went to Berlin for postgraduate studies. Upon his return, he undertook courses at the Massachusetts Eye and Ear Infirmary.

From 1905 to 1928, Dr. Tobey was clinical assistant to the surgeon at Massachusetts General Hospital and instructor at the Harvard Medical School. He later became consulting otolaryngologist to the Massachusetts General Hospital and was affiliated with the Boston City, Brooks, North East Deaconess, Cambridge, Children's, Lynn, Union (Fall River), and Leonard Morse Hospitals.

During World War I, he served as Captain in the Medical Corps of the United States Army.

Dr. Tobey was elected a Fellow of this Society in 1937, and was a Fellow of the American Otological Society. He was a member of the American Laryngological, Rhinological and Otological Society, the American Academy of Ophthalmology and Otolaryngology, The North East Otological Society, the Pacific Coast Society of Ophthalmology and Otolaryngology, the American College of Surgeons and the American Medical Association.

His club memberships included the Algonquin, Harvard, St. Botolph, Country Club, Boston Art, New Riding, and Charles River Country Club. His hobbies were fishing and golf.

In 1907, Dr. Tobey was married to Miss Pauline Flanders, and his marriage was blessed with one daughter, Pauline F. Tobey.

Dr. Tobey was an important contributor to the literature of otolaryngology. Based upon careful and sound research, his reports marked significant advances in his chosen specialty.



HILL HASTINGS, M.D.

1874-1948

Dr. Hill Hastings of Los Angeles, California, died on February 14, 1948, aged 74 years, of bronchopneumonia. Born in Elizabethtown, Kentucky, on January 9, 1874, he spent his early life in Kentucky and obtained his preliminary education in Louisville. He graduated in Medicine at the University of Virginia in 1895.

After serving as an Assistant Surgeon, U. S. Public Health Service from 1897 to 1902, he became House Surgeon at the New York Eye and Ear Infirmary from 1903 to 1904. In 1904 he began the practice of otology in Los Angeles, and in 1907 became Professor of Otolaryngology at the University of Southern California, a position he held until 1912 when he was elected Professor Emeritus. Largely as the result of Dr. Hastings's efforts, the Eye and Ear Section of the Los Angeles County Medical Association was formed in 1905.

He was a staff member of the Good Samaritan Hospital, St. Vincent's Hospital and the Cedars of Lebanon Hospital, and consultant to the Los Angeles General Hospital and the Children's Hospital.

Dr. Hill Hastings served as Chairman of the Section on Laryngology, Otology and Rhinology of the American Medical Association in 1915-1916. He served as vice-president of the American Laryngological Association in 1927. He was a member and formerly president of the Los Angeles Society of Otolaryngology and of the American Laryngological, Rhinological and Otological Society; member of the Pacific Coast Oto-Ophthalmological Society; and former chairman of the Section on Otolaryngology of the California State Medical Society.

During World War I, he served as Captain, M. C., U. S. A.

Dr. Hastings married Mary Garvin Brown of Louisville, Ky., on October 3, 1907. His first wife died on May 25, 1910. He married Helen Newlin on February 3, 1915 and she survives him in addition to two married daughters, Mrs. Daniel E. Murphy and Mrs. Chas. Schriber, and two sons, Robert Pusey Hastings and Dr. Newlin Hastings.

As an Episcopalian he served as Vestryman of St. John's Church, Los Angeles. He was a member of the Los Angeles Country Club, Wilshire Country Club, Bel-Air Country Club and served as a member of the Board of Directors of the California Club and as a member of the Board of Directors of the University Club.

Always an ardent fisherman, Dr. Hastings spent many a vacation in the High Sierras and could recount many a tale of high adventure. His professional life spanned the transition from a budding specialty to an established science. He was instrumental in introducing and promoting the use of X-ray examination in his chosen field and stimulated advances in direct laryngoscopy. During his early years of practice, tonsillectomy and submucous resection were procedures in process of development. He was a prolific contributor to the literature during his teaching years. His passing breaks the link with the last of the original Los Angeles group.

Executive Sessions

1948

FIRST EXECUTIVE SESSION

APRIL 14, 1948

The First Executive Session of the Sixty-Ninth Annual Meeting of the American Laryngological Association at The Homestead, Hot Springs, Va., convened at 1:05 P.M., immediately following the first Scientific Session. The President, Dr. Arthur W. Proetz, presided.

DR. JOHN L. MYERS of Kansas City, Mo., and DR. FRANK R. SPENCER, of Boulder, Colo. were elected to Emeritus Fellowship.

DR. JAMES H. MAXWELL of Ann Arbor, Mich., DR. KENNETH A. PHELPS of Minneapolis, Minn., DR. EDWARD J. WHALEN of Hartford, Conn., and DR. HENRY L. WILLIAMS, 2nd, Rochester, Minn. were unanimously elected to Active Fellowship.

The Newcomb Award for 1948 was given to DR. GORDON BERRY.

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

DR. LE ROY A. SCHALL

DR. H. MARSHALL TAYLOR

DR. GORDON D. HOOPLE

DR. HUGH G. BEATTY

DR. JOHN J. SHEA

The meeting adjourned at 1:20 P.M.

Respectfully submitted,

LOUIS H. CLERF.

Secretary

ANNUAL BUSINESS MEETING

APRIL 15, 1948

The meeting convened at 12:15 P.M., Dr. Arthur W. Proetz, presiding.

The newly elected Fellow, Dr. James H. Maxwell, Dr. Kenneth A. Phelps, Dr. Edward J. Whalen and Dr. Henry L. Williams, 2nd., were introduced to the Association.

The report of the Secretary was accepted as printed.

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

The Nominating Committee recommended for election as Councilors:

DR. FREDERICK T. HILL	DR. HARRY P. SCHENCK
DR. HENRY B. ORTON	DR. CHARLES J. IMPERATORI
DR. ANDERSON C. HILDING	DR. HAROLD I. LILLIE
DR. LOUIS H. CLERF	DR. FRANCIS E. LEJEUNE
DR. GORDON F. HARKNESS	DR. ARTHUR W. PROETZ

There were no nominations from the floor.

The Biographer and Historian read the names of the six Fellows whose deaths had occurred during the past year:

Active Fellows:

DR. GEORGE L. TOBEY, JR.	DR. WARREN B. DAVIS
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Emeritus Fellows:

DR. GEORGE E. SHAMBAUGH	DR. HILL HASTINGS
DR. FRANK B. KISTNER	DR. ARTHUR W. WATSON

Corresponding Fellows:

DR. FRANCESCO LASAGNA

The Membership rose and stood in silence in honor of the departed Fellows.

Dr. F. T. Hill reporting for the American Board of Otolaryngology stated that the examination was held in Chicago on October 8th to 10th, 1947. Seventy-one candidates were examined, 47 were passed, 18 conditioned and 6 failed. The next examination of the Board will be held in Chicago on June 16th to 19th, 1948 and on October 5th to 8th, 1948.

The report of the Memorial Fund was presented. The total gifts are \$1350.00. The report was unanimously accepted. Dr. Samuel Salinger and Dr. H. Marshall Taylor were recorded as Benefactors of the Memorial Fund and the recommendation of the Council that Dr. Gordon Berry be appointed Chairman and Dr. H. Marshall Taylor be appointed as the new member of the Committee were approved.

By unanimous vote the By-Laws, Article IX, Section 2, referring to Committees which states "The deRoaldes Fund Committee, consisting of five (5) Fellows appointed by the Council every third year," were changed as recommended unanimously by Council to read as follows: "The deRoaldes Fund Committee, a rotating Committee consisting of five (5) Fellows by the Council, one member of which shall be appointed annually to serve for five years."

It was regularly moved, seconded and passed that the dues for the ensuing year be placed at \$35.00, this sum being necessary in order to secure increased income for current expenses.

The Chairman announced that the Casselberry Award has not been made this year.

There being no further business, the meeting adjourned at 12:55 P.M.

Respectfully submitted,

LOUIS H. CLERF,

Secretary

REPORT OF THE SECRETARY

APRIL 14, 1948

The Sixty-Eighth Annual Meeting of the Association was held on April 23 and 24, 1947 at the Hotel Statler in St. Louis, Mo., under the presidency of Dr. Frank R. Spencer. The attendance was remarkably good; there were present 78 Fellows and 181 Guests. Fifteen scientific papers were presented and were liberally discussed.

Dr. Harris P. Mosher was elected to Honorary Fellowship. Dr. Frank B. Kistner was elected to Emeritus Fellowship. Drs. Watt W. Eagle, Clarence W. Engler, and Walter H. Theobald were elected to Active Fellowship.

The Newcomb Award was presented to Dr. Harris P. Mosher. The following officers were elected:

DR. ARTHUR W. PROETZ, *President*

DR. JOHN J. SHEA, *1st Vice-President*

DR. FREDERICK A. FIGI, *2nd Vice-President*

DR. LOUIS H. CLERF, *Secretary*

DR. FREDERICK T. HILL, *Treasurer*

DR. HARRY P. SCHENCK, *Librarian and Historian*

DR. JOHN D. KERNAN, JR., *1st Councilor*

DR. CHARLES J. IMPERATORI, *2nd Councilor*

DR. HAROLD I. LILLIE, *3rd Councilor*

DR. FRANK R. SPENCER, *4th Councilor*

The Association has lost through death in the past year 4 Emeritus Fellows, 1 Corresponding Fellow, and 2 Active Fellows.

The present membership is

Active Fellows	93
Emeritus Fellows	16
Corresponding Fellows	13
Honorary Fellows	4
Total	126

The Council at its Annual Meeting recommended the following candidates for election to Active Fellowship:

DR. JOHN H. MAXWELL, Ann Arbor, Mich.

DR. KENNETH A. PHELPS, Minneapolis, Minn.

DR. EDWARD J. WHALEN, Hartford, Conn.

DR. HENRY L. WILLIAMS, Rochester, Minn.

Dr. John L. Myers was recommended for Emeritus Fellowship.

Dr. Samuel Salinger was reappointed as Abstract Editor.

The Council voted unanimously to increase annual dues for Fellows from \$25.00 to \$30.00 per year. One dollar of this will be allocated to the unused income of the DeRoaldes Fund. The Council also voted that the registration fees for the Annual Meeting would be \$10.00. It was further voted that an admission fee of \$100.00 would be charged newly elected Fellows, this to include the annual dues and the registration fee for the first year.

The Council designated Dr. H. Marshall Taylor to serve on the Memorial Fund Committee to succeed Dr. William E. Grove whose term expires in 1948. Dr. Gordon Berry was designated to serve as Chairman for the Committee for 1948.

The American College of Surgeons has requested that all Fellows of the Association participate in the Cancer Archives Project of the College by reporting their five-year cancer cures.

The shortage of paper, increasing difficulties in the printing industry and delay in securing cuts for illustrations has increased the problems of publications of the Transactions. To expedite publication it is suggested that Fellows contemplating having their paper published in medical journals should request early publication if illustrations are used. By so doing the plates can be secured for the Transactions thus saving unnecessary expense in duplication and assuring an earlier issuance of the Transactions.

Respectfully submitted,

LOUIS H. CLERF,

Secretary

REPORT OF THE TREASURER

BALANCE SHEET

April 1, 1948

ASSETS

Cash in Bank.....	\$ 2,039.63	
Less Due Trust Funds.....	1,109.98	\$ 929.65

Trust Fund Investments:

Bonds:

\$1000 U. S. Treas. 2¾ 1948-51.....	\$ 1,101.20	
3000 U. S. Defense 2½—G—1953.....	3,000.00	
1000 U. S. Defense 2½—G—1945.....	1,000.00	
1000 U. S. Treas. 2¾ 1956-59.....	1,003.50	
5000 U. S. Defense 2½—G—1956.....	5,000.00	
1000 U. S. Defense 2½—G—1957.....	1,000.00	
2000 U. S. Defense 2½—G—1958.....	2,000.00	
1000 U. S. Treas. 2¾ 1958-63.....	1,101.20	
3000 U. S. Treas. 2½ 1946-69.....	3,000.00	
1000 U. S. Treas. 2½ 1967-72.....	1,000.00	
5000 U. S. Savings 2½ 1959 Series G.....	5,000.00	
1000 U. S. Savings 2½ 1960 Series G.....	1,000.00	\$25,205.90

Savings Deposits:

Waterville Savings Bank.....	\$ 2,725.53	
Due from General Cash.....	1,109.98	3,787.23 \$28,993.13

Total Assets		\$29,971.06
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LIABILITIES AND CAPITAL

Trust Funds

Casselberry Fund.....	\$ 5,000.00	
Johnston Fund.....	5,000.00	
De Roaldes Fund.....	3,000.00	
Newcomb Trust Fund.....	2,000.00	
Newcomb Prize Fund.....	500.00	
Memorial Funds.....	10,754.19	\$26,254.19

George E. Hourn Scholarship Fund.....	750.00
Reserve for Losses on Investment	48.28

Unused Income:

Casselberry Fund.....	379.59		
Johnston Fund.....	322.82		
De Roaldes Fund.....	39.04		
Newcomb Trust Fund.....	299.24		
Newcomb Prize Fund	(2.82)		
Memorial Funds.....	951.07	\$ 1,988.94	\$29,041.41

General Capital and Surplus	929.65
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Total Liabilities and Capital.....	\$29,971.06
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STATEMENT OF INCOME AND EXPENSE

FOR THE PERIOD APRIL 10, 1947 TO APRIL 1, 1948

Income:

Dues	\$2,325.00	
Registration Fees.....	650.00	
Income—Trust Funds.....	643.69	
Sale of Transactions.....	136.50	\$3,755.19

Expenditures:

Annual Meeting		
Notices-programs	\$110.11	
Dinner-cocktail party.....	665.95	
Reporting Service.....	134.02	
Guest Speaker-expense.....	134.97 (a)	
Projection Service.....	89.00	
Loud Speaker.....	50.00	
Photographer	23.00	
Officers' Reports.....	15.75	\$1,222.80
Allowances-Officers, Clerical Hire.....	450.00	
Transactions	1,679.16	
Council Meeting.....	441.05	
Stationery & Printing.....	300.61	
Postage	37.00	
Treasurer's Bond.....	75.00	
Gift—Auditing Accounts	52.01 (b)	
Transportation—Archives	87.05 (b)	
Amortization	48.28	
Miscellaneous	15.37	\$4,408.33

Excess of Expenditure over Income.....	\$ 653.14
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(a) Charged to Unused Income, Johnston Fund

(b) Charged to Unused Income, Newcomb Trust Fund

DISPOSITION OF TRUST FUND INCOME

Total Income on Trust Funds :.....		\$ 643.69
Casselberry Fund.....	\$ 123.31	
Johnston Fund.....	123.31	
De Roaldes Fund.....	73.98	
Newcomb Prize Fund	12.34	
Newcomb Trust Fund.....	49.33	
Memorial Funds.....	261.42	\$ 643.69

STATEMENT OF INCOME (TRUST FUNDS)

	Unused Income 4/10/47	Expended	Income 1947-48	Unused Income 4/1/48
Casselberry Fund	256.28		123.31	379.59
Johnston Fund	334.48	134.97	123.31	322.82
DeRoaldes Fund	34.94*		73.98	39.04
Newcomb Prize Fund	15.16*		12.34	2.82*
Newcomb Trust Fund	388.97	139.06	49.33	299.24
Memorial Funds	689.65		261.42	951.07

RECONCILIATION OF GENERAL CAPITAL AND SURPLUS

General Capital and Surplus April 10, 1947.....		\$1952.45
Income Prorated to unused income of Trust Funds.....	\$643.69	
Less: Expenses charged directly to unused income of Trust Funds.....	274.03	
	369.66	
Excess of Expenditure over Income.....	653.14	1022.80
General Capital and Surplus April 1, 1948.....		\$ 929.65

* Represents Overdraft

SECURITY TRANSACTIONS

Purchased

1000 U. S. Savings Bonds Series G 1959	\$1000.00
1000 U. S. Savings Bonds Series G 1960	1000.00

The following contributions have been received during the year :

Memorial Funds :

Dr. Fletcher D. Woodward	50.00	
Dr. Samuel Salinger	500.00	
Dr. O. E. Van Alyea	50.00	
Dr. H. M. Taylor	500.00	
Dr. Louis H. Clerf	100.00	
Dr. Bernard J. McMahon	50.00	
Dr. Gordon Berry	100.00	\$1350.00
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George E. Hourn Scholarship

Dr. George E. Hourn	\$ 250.00
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	\$1600.00
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Respectfully submitted,

FREDERICK T. HILL,

Treasurer.

April 1, 1948.

This is to certify that I have examined the books of the American Laryngological Association for the fiscal year 1947-48. The financial reports, as presented, correctly reflect the financial condition and operations of the Association, and the accounts are kept in accordance with sound accounting practice.

A. G. EUSTIS,

Treasurer, Colby College, Waterville, Maine.

MEMORIAL FUND

1948 ANNUAL REPORT TO THE ASSOCIATION

Since the annual report in April, 1947, there have been gifts from Drs. Berry, Clerf, Macpherson, McMahon, Salinger, Taylor, Van Alyea, and Woodward totaling \$1350. Last year those who had thus far given \$500 or over and were therefore called Benefactors, were Drs. Barnhill, Berry, Dean, Hill and Mrs. Hourn. This year Drs. Salinger and Taylor have each completed a gift of \$500 and it is therefore recommended by the committee that their names also be spread upon our records and listed in our Transactions as Benefactors of the Memorial Fund.

This Fund is designed for research in laryngology. The principal sum has now reached an amount sufficient to provide an annual income of about \$500, including the Hourn gift. By applying this as a part time salary it might furnish enough added financial help to permit some young laryngologist to devote extra time to research, under the supervision of one of our members. This would serve the double purpose of developing a scientific worker and making some constructive contribution to laryngology. This has been our goal.

It is urged that our Fellows bear this Fund in mind, and help the Committee discover and develop such a research worker. It is also urged that the Fellows give careful thought to adding to the principal of this Fund to the end that the yearly income will be more adequate. This is our own effort to foster and direct research. Let us strive to make it worthy of the American Laryngological Association.

Respectfully submitted,

WILLIAM E. GROVE '48

GORDON BERRY, *Chairman* '49

BERNARD J. MCMAHON '50

OLIVER E. VAN ALYEA '51

FLETCHER D. WOODWARD '52

FINAL EXECUTIVE SESSION

APRIL 15, 1948

The final Executive Session was called to order by President Proetz at 4:20 P.M. immediately following the Scientific Session. The Association unanimously elected as Councilors the Fellows recommended by the Nominating Committee in their report.

A short recess was declared while the new Councilors retired and elected the following officers:

FREDERICK T. HILL, M.D., *President*

HENRY B. ORTON, M.D., *First Vice-President*

ANDERSON C. HILDING, M.D., *Second Vice-President*

LOUIS H. CLERF, M.D., *Secretary*

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

HARRY P. SCHENCK, M.D., *Librarian and Historian*

CHARLES J. IMPERATORI, M.D., *First Councilor*

HAROLD I. LILLIE, M.D., *Second Councilor*

FRANCIS E. LEJEUNE, M.D., *Third Councilor*

ARTHUR W. PROETZ, M.D., *Fourth Councilor*

The newly elected President Dr. Frederick T. Hill was conducted to the rostrum and expressed his appreciation and adjourned the meeting.

Respectfully submitted,

LOUIS H. CLERF,

Secretary

List of Fellows

1948

LIST OF FELLOWS

Date Indicates Year Admitted to Active Fellowship

Attendance at meetings is shown by the figures following each name.

Asterisk (*) indicates year paper was read.

Active Fellows

1942. ASHLEY, REA E., 384 Post Street, San Francisco 8, California, 1946, '47, '48.
1936. BALLENGER, HOWARD C., 723 Elm Street, Winnetka, Illinois, 1936, '37, '38*, '40, '41, '42, '46*, '47, '48.
1944. BEATTY, HUGH G., 150 East Broad Street, Columbus 15, Ohio, 1944, '46*, '47, '48.
1921. BERRY, GORDON, 36 Pleasant Street, Worcester, Massachusetts, 1921*, '22, '23, '24, '25, '26, '27, 28*, '29*, '30, '31, '32, '33, '34, '35, '37, '38, '39, '40, '41*, '42, '44, '46, '47, '48.
1938. BLASINGAME, CHARLES DECATUR, 414 P. and S. Building, Memphis 3, Tennessee, 1938, '39, '41, '42, '44, '46*, '47.
1944. BOIES, LAWRENCE R., 1631 Medical Arts Building, Minneapolis 2, Minnesota, 1944, '46, '47, '48*.
1941. BOWERS, WESLEY C., 17 East 61st Street, New York City 21, 1941, '42, '44.
1932. BROWN, JOHN MACKENZIE, 1136 W. 6th Street, Los Angeles, 14, California, 1935*, '37, '40*, '41, '42, '44, '47*.
1940. BROYLES, EDWIN N., 1100 North Charles Street, Baltimore, Maryland, 1940, '41*, '42*, '46, '47, '48*.
1930. CAMPBELL, EDWARD HASTINGS, Spruce Street Medical Bldg., 19th and Spruce Streets, Philadelphia 3, Pennsylvania, 1930, '31*, '32*, '33, '35, '38, '39*, '40, '41, '47, '48.
1945. CAMPBELL, PAUL A., 700 North Michigan Avenue, Suite 314, Chicago, Illinois, 1946, '47.

1942. CANFIELD, NORTON, 789 Howard Avenue, New Haven, Connecticut, 1942, '46, '47, '48.
1925. CLERF, LOUIS H., 1530 Locust Street, Philadelphia 2, Pennsylvania, 1925, '27*, '28, '29*, '30*, '32, '33, '34, '35, '36*, '37, '38, '39, '40, '41*, '42*, '44*, '46, '47, '48.
1917. COATES, GEORGE MORRISON, 1721 Pine Street, Philadelphia 3, Pennsylvania, 1920, '21, '22, '23, '24, '25, '26, '27, '28, '29, '30, '31, '32, '33, '34, '35, '36, '37*, '38, '39, '40, '41, '42, '44, '46, '47.
1939. CODY, CLAUDE CARR, 1304 Walker Avenue, Houston, Texas, 1939, '41, '42, '44, '46*, '47, '48*.
1937. CROWE, SAMUEL JAMES, 4332 North Charles Street, Baltimore, 18, Maryland, 1937, '38*, '40, '42, '44*, '48*.
1941. CUNNING, DANIEL S., 121 East 60th Street, New York City 22, 1941, '42*, '44, '46, '47*, '48.
1941. DIXON, FRED W., 1027 Rose Building, Cleveland, Ohio, 1941, '42*, '44, '46, '47*.
1947. EAGLE, WATT WEEMS, Duke Hospital, Durham, N. C., 1948*.
1947. ENGLER, CLARENCE W., 2323 Prospect Avenue, Cleveland, Ohio, 1947, '48*.
1923. FENTON, RALPH ALBERT, 806 Medical Arts Building, Tenth and Taylor Streets, Portland, Oregon, 1923, '25*, '26, '28, '30, '32, '34, '35, '37*, '39, '40*, '42, '44, '46, '47*.
1930. FIGI, FREDERICK A., Mayo Clinic, Rochester, Minnesota, 1930, '32*, '34, '35, '38*, '42*, '47*, '48.
1932. FURSTENBERG, ALBERT CARL, 201 South Main Street, Ann Arbor, Michigan, 1933*, '36*, '38*, '46, '47, '48*.
1936. GALLOWAY, THOMAS CYRUS, 636 Church Street, Evanston, Illinois, 1936, '37, '38*, '39, '40, '41, '42*, '46*, '47, '48.
1940. GATEWOOD, E. TRIBLE, Professional Building, Richmond, Virginia, 1940, '41, '42*, '44, '47.
1940. GILL, WILLIAM D., 705 East Houston Street, San Antonio, Texas, 1940, '41*, '46.

1928. GITTINS, THOMAS ROY, 408 Davidson Building, Sioux City, Iowa, 1928, '31, '32*, '34, '36, '39*, '40, '42*, '47.
1940. GOODALE, ROBERT L., 330 Dartmouth Street, Boston, Massachusetts, 1940, '41, '42*, '44*, '46, '47.
1928. GOODYEAR, HENRY M., 556 Doctors' Building, Cincinnati 2, Ohio, 1928, '29, '30*, '31, '32, '33, '34*, '35, '36, '37, '38, '39, '40, '42, '44*, '46, '47, '48.
1934. GROVE, WILLIAM E., 324 East Wisconsin Avenue, Milwaukee, 2, Wisconsin, 1934, '35, '36*, '37, '38, '39, '40, '41, '42*, '44, '46, '47, '48.
1940. HANSEL, FRENCH K., 634 North Grand Avenue, St. Louis 3, Missouri, 1940, '42, '46, '47.
1933. HARKNESS, GORDON FOLLETTE, 2410 River Drive, Davenport, Iowa, 1934, '35, '36*, '40, '41, '44, '46, '47, '48.
1944. HAVENS, FRED Z., 102 Second Avenue S-W., Rochester, Minnesota, 1944, '46.
1942. HEATLEY, CLYDE A., 11 North Goodman Street, Rochester, N. Y., 1942, '46*.
1945. HICKEY, HAROLD L., 1612 Tremont Street, Denver 2, Colorado, 1946, '47, '48.
1943. HILDING, ANDERSON C., 324 West Superior Street, Duluth, Minnesota, 1944*, '46, '47, '48*.
1928. HILL, FREDERICK THAYER, Professional Building, Waterville, Maine, 1928, '29*, '30*, '31, '32, '33, '34, '35, '36, '37*, '38*, '39, '40, '41*, '42, '44*, '46, '47, '48.
1945. HOLINGER, PAUL H., 700 North Michigan Ave., Chicago 11, Illinois, 1946, '47.
1944. HOOPLE, GORDON D., Medical Building, Syracuse, New York, 1946, '47, '48.
1930. HOUSER, KARL MUSSER, 2035 Delancey Street, Philadelphia 3, Pennsylvania, 1930, '32*, '33, '34*, '37, '38, '39, '40, '41, '44, '48.
1939. HUNT, WESTLEY MARSHALL, 907 Fifth Avenue, New York City 21, 1939, '40*, '41, '42, '44, '46, '47, '48.

1920. IMPERATORI, CHARLES JOHNSTONE, Essex, New York, 1921*, '22, '24*, '25*, '26*, '27*, '28, '31*, '32, '33*, '35, '36, '37*, '38, '39*, '40*, '41*, '42*, '44*, '47*.
1938. JACKSON, CHEVALIER LAWRENCE, 255 South Seventeenth Street, Philadelphia 3, Pennsylvania, 1938, '39, '40, '41*, '42, '44*, '46, '47*, '48.
1928. JARVIS, DEFOREST CLINTON, Quarry Bank Building, Barre, Vermont, 1928, '30, '32*, '33, '34*, '35, '37*, '38, '39, '40, '41, '44, '46, '47, '48.
1939. JOHNSTON, WILLIAM HOWARD, 1515 State Street, Santa Barbara 7, California, 1939, '40, '41, '42, '44, '46, '47.
1944. JONES, MARVIN F., 121 E. 60th Street, New York City, 1944, '46, '47, '48.
1942. KELLY, JOSEPH D., 140 East Fifty-fourth Street, New York City 22, 1942, '44*, '47, '48.
1921. KERNAN, JOHN D., JR., 103 East Seventy-eighth Street, New York City 21, 1921*, '22, '25, '26, '27, '29, '31, '34*, '35*, '36, '37*, '39, '40*, '42*, '47, '48.
1942. KING, EDWARD D., 6777 Hollywood 28, California, 1942, '46, '47.
1936. LEJEUNE, FRANCIS ERNEST, Ochsner Clinic Building, Prytania and Aline Streets, New Orleans, Louisiana, 1937, '46*, '47.
1933. LIERLE, DEAN M., University Hospital, Iowa City, Iowa, 1933, '34, '35*, '37, '38, '39, '40*, '42*, '44*, '46, '47*, '48.
1922. LILLIE, HAROLD I., Mayo Clinic, Rochester, Minnesota, 1923*, '24*, '25, '26*, '28, '31*, '33, '36*, '37, '38, '40, '44, '46*, '47, '48*.
1935. LOOPER, EDWARD A., 104 West Madison Avenue, Baltimore 1, Maryland, 1935, '36, '37, '38*, '39, '41, '42, '44, '47, '48*.
1927. LUKENS, ROBERT M., 1308 Hunting Park Avenue, Philadelphia 40, Pennsylvania, 1927, '28, '31*, '32, '35, '36, '38, '39, '40*, '42, '46, '48*.
1939. LUPTON, IRVING MARTIN, 806 Medical Arts Building, Portland, Oregon, 1946.

1929. LYMAN, HARRY WEBSTER, 906 Carleton Building, St. Louis 1, Missouri, 1929, '31, '34, '35*, '36, '38, '39, '42, '44, '46, '47, '48.
1928. MACPHERSON, DUNCAN, 920 Fifth Avenue, New York City 21, 1928, '29, '30, '31, '33, '34*, '39, '40, '41, '44.
1941. MARTIN, ROBERT C., 384 Post Street, San Francisco 8, California, 1941, '44, '47, '48.
1948. MAXWELL, JAMES H., 201 S. Main Street, Ann Arbor, Michigan, 1948.
1939. McCASKEY, CARL HEBER, 608 Guaranty Building, Indianapolis 4, Indiana, 1939, '40, '41, '42*, '44, '46*, '47, '48.
1945. McLaurin, JOHN G., 1322 Medical Arts Building, Dallas 1, 1946.
1933. McMAHON, BERNARD J., Missouri Theatre Building, St. Louis 3, Missouri, 1934*, '35, '36, '38*, '41, '44, '46*, '47, '48.
1931. McNALLY, WILLIAM JAMES, 1509 Sherbrooke Street West, Montreal, Canada, 1931, '33, '34*, '39, '44, '47, '48.
1940. MORRISON, WILLIAM W., 140 E. 54th Street, New York City 22, 1940, '41*, '44, '47.
1927. MYERSON, MERVIN C., 416 North Bedford Drive, Beverly Hills, California, 1927, '28*, '29, '30, '31*, '32*, '33*, '34*, '35, '36, '37, '38, '39*, '40, '41*, '42, '44.
1937. NASH, CARLTON STEWART, Medical Arts Building, Rochester 7, New York, 1937, '38*, '40, '42, '44, '46, '47.
1922. NEW, GORDON B., Mayo Clinic, Rochester, Minnesota, 1922, '24, '25*, '26*, '28*, '31*, '33*, '35*, '37*, '39*, '41*, '46*, '48*.
1927. ORTON, HENRY BOYLAN, 224 Delavan Avenue, Newark 4, New Jersey, 1927, '28, '29, '30*, '31, '32, '33*, '35, '36, '37, '38*, '40, '41, '42, '44*, '46, '47*
1948. PHELPS, KENNETH A., 1137 Medical Arts Building, Minneapolis 2, Minnesota, 1948.
1924. PROETZ, ARTHUR WALTER, 1010 Beaumont Building, St. Louis 3, Missouri, 1924, '25*, '26*, '27, '28, '29, '30, '31, '32*, '33*, '34, '35*, '36, '37, '38*, '39*, '40, '41, '42*, '44, '46, '47*, '48*.

1934. RICHARDS, LYMAN GILDER, 520 Commonwealth Avenue, Boston, Massachusetts, 1934, '35, '36*, '37, '38, '39, '47.
1930. RIDPATH, ROBERT F., 1720 Spruce Street, Philadelphia 3, Pennsylvania, 1930, '31, '33, '37, '38*, '40, '41*, '44, '47.
1945. ROBB, JAMES M., 641 David Whitney Building, Detroit, Michigan, 1946, '48.
1938. SALINGER, SAMUEL, 25 E. Washington Street, Chicago, Illinois, 1938, '39, '40*, '42, '44*, '46, '47, '48.
1921. SAUER, WILLIAM E., 3720 Washington Boulevard, St. Louis 8, Missouri, 1921, '23, '25, '28, '31, '32, '35, '37, '39*, '41, '44, '47.
1932. SCHALL, LEROY ALLEN, 243 Charles Street, Boston 14, Massachusetts, 1934*, '35, '38, '40*, '41, '42, '44*, '46, '47, '48*.
1934. SCHENK, HARRY P., 326 South 19th Street, Philadelphia 3, Pennsylvania, 1934, '35, '36*, '37, '38, '39, '40, '41*, '44*, '46, '47.
1930. SEYDELL, ERNEST M., First National Bank Building, Wichita, Kansas, 1930, '31*, '33*, '34, '35, '37*, '38, '39, '40*, '41, '42, '46, '47, '48.
1928. SHEA, JOHN JOSEPH, 1018 Madison Avenue, Memphis, Tennessee, 1928, '29, '30*, '31, '32*, '33, '34, '35, '36, '37, '38, '40, '41*, '42, '46, '47*, '48.
1909. SHURLY, BURT R., 62 Adams Avenue W., Detroit, Michigan, 1920, '21*, '22, '24, '25*, '26, '27*, '28, '29*, '30, '31, '33, '34, '35, '36, '37, '38, '39, '40, '41, '42, '44, '46, '47, '48.
1939. SIMPSON, W. LIKELY, 512 Physicians' and Surgeons' Building, Memphis 3, Tennessee, 1939, '41, '44*, '46, '47*, '48.
1924. TAYLOR, HERMON MARSHALL, 111 West Adams Street, Jacksonville 5, Florida, 1924, '25*, '26, '27, '28, '31, '34, '35*, '36, '37, '38, '39*, '40, '41, '42, '44*, '46, '47, '48.
1947. THEOBALD, WALTER H., 207 N. Michigan Avenue, Chicago, Illinois, 1947, '48*.
1927. TOBEY, HAROLD G., 403 Commonwealth Avenue, Boston 15, Massachusetts, 1927, '29*, '30*, '31, '32, '35, '37, '39, '41, '46, '48.

1925. TUCKER, GABRIEL, 250 South Eighteenth Street, Philadelphia, Pennsylvania, 1925, '26*, '27*, '29, '30, '31, '32, '33, '34, '35*, '36, '37, '38, '39, '40*, '41*, '42, '46, '47, '48.
1943. VAN ALYEA, OLIVER E., 135 South La Salle Street, Chicago, Illinois, 1944, '46*, '47, '48.
1941. VIOLE, PIERRE P., 1930 Wilshire Boulevard, Los Angeles 5, California, 1944*, '46, '47.
1940. WEILLE, FRANCIS L., 247 Commonwealth Avenue, Boston 16, Massachusetts, 1940, '41, '42, '44, '46*, '47, '48.
1948. WHALEN, EDWARD J., 750 Main Street, Hartford, Connecticut, 1948.
1922. WHITE, FRANCIS W., 471 Park Avenue, New York City 22, 1922, '23*, '25, '26, '27, '28, '29, '31, '32, '37, '39, '40, '41, '42, '44, '46*.
1948. WILLIAMS, HENRY L. 2nd, Mayo Clinic, Rochester, Minnesota, 1948.
1939. WILLIAMS, HORACE JAMES, 5908 Greene Street, Germantown, Philadelphia, 1939, '40, '41, '42, '46, '47*.
1942. WISHART, D. E. STAUNTON, 170 St. George Street, Toronto, Canada, 1942, '44, '46, '47*.
1940. WOOD, V. VISSCHER, 3720 Washington Boulevard, St. Louis 8, Missouri, 1940, '41, '42*, '46, '47.
1935. WOODWARD, FLETCHER D., 104 East Market Street, Charlottesville, Virginia, 1935, '36*, '37, '38, '39*, '40, '42, '44, '47, '48.

Honorary Fellows

1939. (1907) JACKSON, CHEVALIER, 255 South Seventeenth Street, Philadelphia 3, Pennsylvania, 1939*, '40*.
1943. (1918) LEWIS, FIELDING O., Providence Road, Media, Pennsylvania.
1947. (1904) MOSHER, HARRIS P., 127 Front Street, Marblehead, Mass., 1947, '48.
1937. NAGER, F. B., Freiestrasse 3, Zurich, Switzerland.

Corresponding Fellows

- 1946. ALONSO, JUSTO M., Mercedes 1233, Montevideo, Uruguay.
- 1942. BATSON, OSCAR V., 133 South Thirty-sixth Street, Philadelphia 4, Pennsylvania, 1942*.
- 1938. BLAIR, VILRAY P., 508 North Grand Avenue, St. Louis 3, Missouri.
- 1940. COLLEDGE, LIONEL, 2 Upper Wimpole Street, London, W. 1, England.
- 1943. EGGSTON, ANDREW O., 653 Park Avenue, New York City, 1944.
- 1921. LE MAITRE, FERNAND, Avenue Victor Hugo, 120 (XVI), Paris, France.
- 1919. MCKERNON, JAMES F., New Canaan, Connecticut.
- 1930. NEGUS, VICTOR E., 149 Harley Street, London, W 1, England, 1938*.
- 1941. PATTERSON, NORMAN, Nobs Creek, Ashbridge Park, Berkhamsted, Herts, England.
- 1937. PORTMANN, GEORGES, 25 bis Cours de Verdun, Bordeaux, France, 1947.
- 1924. SCHAEFER, J. PARSONS, Jefferson Medical College, Philadelphia, Pennsylvania, 1934.
- 1896. SCHMIEGELOW, ERNST, 18 Norregade, Copenhagen, Denmark.
- 1936. VIALLE, JACQUES, 27 Rue de France, Nice (Alpes Maritimes), France.

Emeritus Fellows

- 1941. (1923) BARLOW, ROY A., Medical Arts Building, Winchester, Virginia.
- 1937. (1915) BARNES, HARRY ALDRICH, Pembroke, Massachusetts.
- 1944. (1913) BUTLER, RALPH, 1930 Chestnut Street, Philadelphia, Pennsylvania.
- 1946. (1933) FOSTER, JOHN H., Eagle Lake, Texas, '48.
- 1941. (1922) GOLDSMITH, PERRY G., 513 Medical Arts Building, Toronto, Canada.

1936. (1898) GOODALE, JOSEPH L., Argilla Road, Ipswich, Massachusetts.
1943. (1932) GRAHAM, HARRINGTON B., Bohemian Club, San Francisco 2, California.
1940. (1917) GREENE, JOSEPH B., 331 Haywood Building, Asheville, North Carolina.
1933. (1905) HALSTED, THOMAS H., 475 Fifth Avenue, New York City 17, N. Y.
1939. (1923) LARGE, SECORD HERBERT, 170 Eddy Road, Cleveland, Ohio.
1943. (1921) McCULLAGH, SAMUEL, 116 E. 63rd St., New York 21, N. Y.
1948. (1939) MYERS, JOHN LLEWELLYN, 1115 Grand Avenue, Kansas City, Missouri.
1941. (1903) PACKARD, FRANCIS R., 302 South Nineteenth Street, Philadelphia, Pennsylvania.
1942. (1931) POTTS, JOHN B., 1620 Medical Arts Building, Omaha, Nebraska.
- 1940.(1923) SEWALL, EDWARD C., 430 El Escarpado, Stanford University, California.
1948. (1923) SPENCER, FRANK R., Physicians Building, Boulder, Colorado.
1939. (1908) WILSON, J. GORDON, Old Bennington, Vermont.
1946. (1905) WOOD, GEORGE B., 329 Hathaway Lane, Wynnewood, Pennsylvania.

BENEFACTORS

DR. ARTHUR W. DEROALDES

DR. WILLIAM E. CASSELBERRY

DR. SAMUEL JOHNSTON

DR. CORNELIUS G. COAKLEY

DR. JAMES E. NEWCOMB

DR. JOHN F. BARNHILL

DR. LEE WALLACE DEAN

DR. GEORGE E. HOURN

BENEFACTORS OF THE MEMORIAL FUND

DR. JOHN F. BARNHILL

DR. AND MRS. LEE WALLACE DEAN

DR. GORDON BERRY

DR. FREDERICK T. HILL

DR. AND MRS. GEORGE E. HOURN

DR. SAMUEL SALINGER

DR. H. MARSHALL TAYLOR



Obverse of the deRoaldes medal

RECIPIENTS OF THE DE ROALDES AWARD

- 1928. DR. CHEVALIER JACKSON
- 1931. DR. D. BRYSON DELAVAN
- 1934. DR. HARRIS P. MOSHER
- 1937. DR. LEE WALLACE DEAN
- 1943. DR. RALPH A. FENTON

RECIPIENTS OF THE CASSELBERRY AWARD

1923. DR. GEORGE FETTEROLF AND DR. HERBERT FOX
1928. DR. RALPH A. FENTON AND DR. O. LARSELL
1929. DR. RICHARD A. KERN AND DR. HARRY P. SCHENCK
1929. DR. EDWARD H. CAMPBELL
1931. DR. ARTHUR W. PROETZ
1934. DR. ANDERSON C. HILDING
1936. DR. FRANCIS E. LE JEUNE: DR. JOEL J. PRESSMAN
1939. DR. H. MARSHALL TAYLOR: DR. BRIEN T. KING
1940. DR. FRENCH K. HANSEL
1941. DR. NOAH D. FABRICANT
1946. DR. PAUL H. HOLINGER

RECIPIENTS OF THE NEWCOMB AWARD

- 1941. DR. BURT R. SHURLY
- 1942. DR. FRANCIS R. PACKARD
- 1943. DR. GEORGE M. COATES
- 1944. DR. CHARLES J. IMPERATORI
- 1947. DR. HARRIS P. MOSHER
- 1948. DR. GORDON BERRY

PAST OFFICERS

Presidents

(Year of Election)

1879. Louis Elsberg	1908. A. Coolidge, Jr.
1880. J. Solis-Cohen	1909. J. E. Logan
1881. F. I. Knight	1910. D. Braden Kyle
1882. G. M. Lefferts	1911. James E. Newcomb
1883. F. H. Bosworth	1912. George A. Leland
1884. E. L. Shurly	1913. Thomas Hubbard
1885. Harrison Allen	1914. Alexander W. MacCoy
1886. E. Fletcher Ingals	1915. G. Hudson Makuen
1887. R. P. Lincoln	1916. Joseph L. Goodale
1888. E. C. Morgan	1917. Thomas H. Halsted
1889. J. N. Mackenzie	1918. Cornelius G. Coakley
1890. W. C. Glasgow	1919. Norval H. Pierce
1891. S. W. Langmaid	1920. Harris P. Mosher
1892. M. J. Asch	1921. Harmon Smith
1893. D. Bryson Delavan	1922. Emil Mayer
1894. J. O. Roe	1923. J. Payson Clark
1895. W. H. Daly	1924. Lee Wallace Dean
1896. C. H. Knight	1925. Greenfield Sluder
1897. T. R. French	1926. Chevalier Jackson
1898. W. E. Casselberry	1927. D. Bryson Delavan
1899. Samuel Johnston	1928. Charles W. Richardson
1900. H. L. Swain	1929. Lewis A. Coffin
1901. J. W. Farlow	1930. Francis R. Packard
1902. J. H. Bryan	1931. George E. Shambaugh
1903. J. H. Hartman	1932. George Fetterolf
1904. C. C. Rice	1933. George M. Coates
1905. J. W. Gleitsmann	1934. Dunbar Roy
1906. A. W. deRoaldes	1935. Burt R. Shurly
1907. H. S. Birkett	1936. William B. Chamberlin
	1937. John F. Barnhill

- | | |
|-------------------------|-------------------------------|
| 1938. George B. Wood | 1942-3. Charles J. Imperatori |
| 1939. James A. Babbitt | 1944-5. Harold I. Lillie |
| 1940. Gordon Berry | 1946. Frank R. Spencer |
| 1941. Thomas E. Carmody | 1947. Arthur W. Proetz |

Vice-Presidents

- | | |
|------------------------|--------------------|
| 1879. F. H. Davis | J. O. Roe |
| 1880. W. C. Glasgow | W. Porter |
| 1881. E. L. Shurly | E. F. Ingals |
| 1882. C. Seiler | S. Johnston |
| 1883. S. W. Langmaid | W. H. Daly |
| 1884. J. H. Hartman | G. W. Major |
| 1885. H. A. Johnson | J. N. Mackenzie |
| 1886. E. C. Morgan | S. W. Langmaid |
| 1887. J. N. Mackenzie | C. E. DeM. Sajous |
| 1888. W. C. Glasgow | C. E. Bean |
| 1889. E. Holden | J. H. Hartman |
| 1890. J. O. Roe | S. Johnston |
| 1891. M. J. Asch | J. C. Mulhall |
| 1892. S. Johnston | W. E. Casselberry |
| 1893. J. C. Mulhall | S. H. Chapman |
| 1894. C. C. Rice | A. W. de Roaldes |
| 1895. J. Wright | D. N. Rankin |
| 1896. T. M. Murray | H. S. Birkett |
| 1897. A. W. MacCoy | F. W. Hinkel |
| 1898. J. W. Farlow | M. R. Brown |
| 1899. T. A. De Blois | A. A. Bliss |
| 1900. H. L. Wagner | D. Braden Kyle |
| 1901. J. W. Gleitsmann | T. Melville Hardie |
| 1902. G. A. Leland | W. Peyre Porcher |
| 1903. J. H. Lowman | W. J. Freeman |
| 1904. Thomas Hubbard | C. W. Richardson |
| 1905. J. L. Goodale | A. B. Thrasher |
| 1906. G. H. Makuen | J. E. Rhodes |
| 1907. J. P. Clark | |

- | | |
|-----------------------------|---------------------|
| 1908. E. Mayer | F. R. Packard |
| 1909. C. G. Coakley | H. P. Mosher |
| 1910. Robert C. Myles | J. M. Ingersoll |
| 1911. F. C. Cobb | B. R. Shurly |
| 1912. A. W. Watson | W. Scott Renner |
| 1913. F. E. Hopkins | Geo. F. Shambaugh |
| 1914. Clement F. Theisen | Lewis A. Coffin |
| 1915. J. Gordon Wilson | Christian R. Holmes |
| 1916. Thomas H. Halsted | Greenfield Sluder |
| 1917. John Edwin Rhodes | D. Crosby Greene |
| 1918. G. E. Shambaugh | John R. Winslow |
| 1919. Francis R. Packard | Harmon Smith |
| 1920. Harmon Smith | W. B. Chamberlin |
| 1921. Dunbar Roy | Robert C. Lynch |
| 1922. George Fetterolf | Lorenzo B. Lockard |
| 1923. Hubert Arrowsmith | Joseph B. Greene |
| 1924. Ross H. Skillern | Gordon Berry |
| 1925. John E. Mackenty | Robert Levy |
| 1926. Lewis A. Coffin | William V. Mullin |
| 1927. Charles W. Richardson | Hill Hastings |
| 1928. Robert Clyde Lynch | Francis P. Emerson |
| 1929. William B. Chamberlin | Ralph Albert Fenton |
| 1930. Harris P. Mosher | James A. Babbitt |
| 1931. Joseph B. Greene | E. Ross Faulkner |
| 1932. Gordon Berry | Frank A. Spencer |
| 1933. E. Ross Faulkner | Thomas S. Carmody |
| 1934. Gordon B. New | Samuel McCullagh |
| 1935. Edward C. Sewall | H. Marshall Taylor |
| 1936. William P. Wherry | Harold I. Lillie |
| 1937. Frank R. Spencer | Bernard J. McMahon |
| 1938. Ralph A. Fenton | Frederick T. Hill |
| 1939. John H. Foster | Thomas R. Gittins |
| 1940. Charles T. Porter | Gordon F. Harkness |
| 1941. Arthur W. Proetz | Henry B. Orton |
| 1942-3. Harold I. Lillie | Dean M. Lierle |
| 1944-5. John J. Shea | Thomas C. Galloway |
| 1946. H. Marshall Taylor | C. Stewart Nash |
| 1947. John J. Shea | Frederick A. Figi |

Secretaries and Treasurers

1879. G. M. Lefferts	1895. H. L. Swain
1882. D. Bryson Delavan	1900. J. E. Newcomb
1889. C. H. Knight	1911. Harmon Smith

Secretaries

1912. Harmon Smith	1933. William V. Mullin
1918. D. Bryson Delavan	1935. James A. Babbitt
1919. J. M. Ingersoll	1939. Charles J. Imperatori
1920. George M. Coates	1942. Arthur W. Proetz
1947. Louis H. Clerf	

Treasurers

1912. J. Payson Clark	1933. James A. Babbitt
1923. George Fetterolf	1935. Charles J. Imperatori
1932. William V. Mullin	1939. Frederick T. Hill

Librarians

1879. F. H. Bosworth	1930. John F. Barnhill
1883. T. R. French	1934. Burt R. Shurly
1903. J. H. Bryan	1935. George M. Coates

Librarian and Historian

1936. George M. Coates	1944. Louis H. Clerf
1947. Harry P. Schenck	

PAST MEMBERS

Dates indicate original election to the Association

**Deceased*

Honorary Fellows

- *1908. Barnhill, John F., Miami Beach, Florida.
- *1893. Birkett, Herbert S., Montreal, Canada.
- *1878. Bosworth, Francke H., New York City.
- *1918. Dean, Lee Wallace, St. Louis, Mo.
- *1881. Delavan, D. Bryson, New York City.
- *1893. deRoaldes, Arthur W., New Orleans, La.
- *1890. De La Sota Y Lastra, Ramon, Seville, Spain.
- *1879. French, Thomas R., Brooklyn, N. Y.
- *1880. Garcia, Senior Manuel, London, England.
- *1903. Harris, Thos. J., New York City.
- *1878. Johnston, Samuel, Baltimore, Md.
- *1878. Lefferts, George Morewood, Katonah, N. Y.
- *1914. Levy, Robert, Denver, Colo.
- *1883. Mackenzie, John N., Baltimore, Md.
- *1881. Mackenzie, Sir Morell, London, England.
- *1910. Massei, Ferdinand, Naples, Italy.
- *1910. Moure, J. J. E., Bordeaux, France.
- *1918. Oliver, H. K., Boston, Mass.
- *1906. Pierce, Norval Harvey, San Diego, Calif.
- *1909. Semon, Sir Felix, Great Missenden, England.
- *1878. Solis-Cohen, J., Philadelphia, Pa.
- *1889. Swain, Henry L., New Haven, Conn.
- *1914. Thomson, Sir St. Clair, London, Eng.
- *1903. Tilley, Herbert, London, England.
- *1914. Wagner, Clinton, New York City.
- *1890. Wright, Jonathan, Pleasantville, N. Y.

Corresponding Fellows

- *1892. Browne, Lennox, London, England.
- *1901. Collier, Mayo, Kearsney Abbey, Kent, England.
- *1893. Desvernine, Carlos M., Havana, Cuba.
- *1930. Emerson, Francis P., Franklin, Mass.

- *1936. Fraser, John S., Edinburgh, Scotland.
- *1901. Grant, Sir James Dundas, London, England.
- *1887. Gouguenheim, A., Paris, France.
- *1894. Holden, Edgar, Newark, N. J.
- *1919. Kelly, Adam Brown, Helensburgh, Scotland.
- *1881. Labus, Carlo, Milan, Italy.
- *1931. LasSagna, Francesco, Parma, Italy.
- *1926. Law, Frederick M., New York, N. Y.
- *1902. Lermoyez, Marcel, Paris, France.
- *1897. Luc, H., Paris, France.
- *1896. MacDonald, Greville, Haslemere, Surrey, England.
- *1894. MacIntyre, John, Glasgow, Scotland.
- *1903. McBride, P., York, England.
- *1920. McKenzie, Dan, London, England.
- *1880. Meyer, Wilhelm, Copenhagen, Denmark.
- *1896. Mygind, Holger, Copenhagen, Denmark.
- *1919. Paterson, Donald Rose, Cardiff, Wales.
- *1919. Rogers, John, Jr., New York City.
- *1894. Sajous, C. E. DeM., Philadelphia, Pa.
- *1946. Segura, Eliseo, Buenos Aires, Argentina.
- *1940. Soto, E. Fernandez, Havana, Cuba.
- *1881. Thornton, Pugin, London, England.
- *1913. Turner, A. Logan, Edinburgh, Scotland.
- *1880. Whistler, W. McNeil, London, England.
- *1901. Wingrave, Wyatt, Lyme Regis, Dorset, England.
- *1894. Wolfenden, R. Norris, Kent, England.

Emeritus Fellows

- *1928. Beck, Joseph C., Chicago, Illinois.
- *1901. Brown, J. Price, Toronto, Canada.
- *1891. Bryan, Joseph H., Washington, D. C.
- *1897. Clark, J. Payson, Boston, Mass.
- *1899. Cobb, Frederick C., Bradenton, Florida.
- *1905. Coffin, Lewis A., New York City.
- *1893. Coolidge, Algernon, Boston, Mass.
- *1913. Dabney, Virginius, Washington, D. C.
- *1882. De Blois, Thomas Amory, Boston, Mass.
- *1892. Farlow, John W., Boston, Mass.

- *1922. Forbes, Henry H., New York City.
- *1905. Freer, Otto T., Chicago, Ill.
- *1880. Gleitsmann, Joseph W., New York City.
- *1906. Greene, D. Crosby, Jr., Boston, Mass.
- *1896. Hardie, Thomas Melville, Chicago, Ill.
- *1915. Hastings, Hill, Los Angeles, Calif.
- *1888. Hinkel, Frank Whitehill, Buffalo, New York.
- *1895. Hopkins, Frederick E., Springfield, Mass.
- *1927. Hubbard, Thomas, Toledo, Ohio.
- *1919. Hurd, Lee Maidment, Rowayton, Conn.
- *1904. Ingersoll, John Marvin, Miami, Fla.
- *1918. Kenyon, Elmer L., Chicago, Ill.
- *1929. Kistner, Frank B., Portland, Oregon.
- *1885. Knight, Charles H., New York City.
- *1894. Leland, George A., Boston, Mass.
- *1943. Lincoln, William R., Cleveland, Ohio.
- *1886. MacCoy, Alexander W., Philadelphia, Pa.
- *1896. Mayer, Emil, New York City.
- *1923. McKinney, Richmond, Memphis, Tenn.
- *1878. Porter, William, Ocean Springs, Miss.
- *1901. Myles, Robert C., New York City.
- *1923. Newhart, Horace, Minneapolis, Minn.
- *1903. Renner, W. Scott, Buffalo, N. Y.
- *1897. Rhodes, John Edwin, Chicago, Ill.
- *1884. Rice, Clarence C., New York City.
- *1905. Richards, George L., South Yarmouth, Mass.
- *1878. Robinson, Beverly, New York City.
- *1907. Shambaugh, George E., Chicago, Ill.
- *1908. Smith, Harmon, New York City.
- *1892. Wagner, Henry L., San Francisco, Calif.
- *1892. Watson, Arthur W., Philadelphia, Pa.

Active Fellows

- *1880. Allen, Harrison, Philadelphia, Pa.
- *1917. Arrowsmith, Hubert, Brooklyn, N. Y.
- *1879. Asch, Morris J., New York City.
- *1923. Babbitt, James A., Philadelphia, Pa.
- *1906. Ballenger, William L., Chicago, Ill.

- *1880. Bean, C. E., St. Paul, Minn.
- *1904. Berens, T. Passmore, New York City.
- *1924. Bigelow, Nolton, Providence, R. I.
- *1893. Bliss, Arthur Ames, Philadelphia, Pa.
- *1895. Boylan, J. E., Cincinnati, Ohio.
- *1892. Brown, Moreau R., Chicago, Ill.
- *1933. Buckley, Robert E., New York City.
- *1915. Canfield, R. Bishop, Ann Arbor, Mich.
- *1934. Carmack, John Walter, Indianapolis, Ind.
- *1924. Carmody, Thomas E., Denver, Colo.
- *1889. Casselberry, William E., Chicago, Ill.
- *1883. Chamberlain, C. W., Hartford, Conn.
- *1917. Chamberlin, William B., Cleveland, Ohio.
- *1896. Chappell, W. F., New York City.
- *1882. Chapman, S. Hartwell, New Haven, Conn.
- *1902. Coakley, Cornelius G., New York City.
- *1913. Coffin, Rockwell C., Boston, Mass.
- 1918. Cox, Gerard H., New York City.
- *1880. Cushing, E. W., Boston, Mass.
- *1878. Cutter, Ephraim, West Falmouth, Mass.
- *1880. Daly, W. H., Pittsburgh, Pa.
- *1878. Davis, F. H., Chicago, Ill.
- *1941. Davis, Warren B., Philadelphia, Pa.
- 1926. Dennis, Frank Lowndes, Colorado Springs, Col.
- *1901. Dickerman, E. T., Chicago, Ill.
- *1878. Donaldson, Frank, Baltimore, Md.
- *1935. Eves, Curtis C., Philadelphia, Pa.
- *1919. Faulkner, E. Ross, New York City.
- *1914. Fetterolf, George, Philadelphia, Pa.
- *1901. Freeman, Walter J., Philadelphia, Pa.
- *1917. Friedberg, Stanton A., Chicago, Ill.
- 1940. Fuchs, Valentine H., New Orleans, La.
- 1909. Getchell, Albert C., Worcester, Mass.
- *1897. Frothingham, Richard, New York City.
- *1907. Gibb, Joseph S., Philadelphia, Pa.
- *1878. Glasgow, William Carr, St. Louis, Mo.
- *1913. Goldstein, Max A., St. Louis, Mo.
- *1905. Grayson, Charles P., Philadelphia, Pa.
- *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.
- *1938. Hourn, George E., St Louis, Mo.
- *1901. Hyatt, Frank, Washington, D. C.
- *1925. Iglauer, Samuel, Cincinnati, Ohio.
- *1878. Ingals, E. Fletcher, Chicago, Ill.
- *1882. Ives, Frank L., New York City.
- *1880. Jarvis, William C., New York City.
- *1878. Johnson, Hosmer A., Chicago, Ill.
- *1879. Johnson, Woolsey, New York City.
- *1879. Kealhofer, R. H., St. Louis, Mo.
- *1901. King, Gordon, New Orleans, La.
- *1885. Knight, Charles H., New York City.
- *1878. Knight, Frederick Irving, Boston, Mass.
- *1898. Kyle, D. Braden, Philadelphia, Pa.
- *1880. Langmaid, Samuel W., Boston, Mass.
- *1878. Lincoln, Rufus P., New York City.
- *1911. Lockard, Lorenzo B., Denver, Colo.
- *1913. Leob, Hanau W., St. Louis, Mo.
- *1897. Logan, James E., Kansas City, Mo.
- *1888. Lowman, John H., Cleveland, Ohio.
- *1919. Lynah, Henry L., New York City.
- *1915. Lynch, Robert Clyde, New Orleans, La.
- *1879. McBurney, Charles, New York City.
- *1927. McGinnis, Edwin, Chicago, Ill.
- 1936. McGregor, Gregor, Toronto, Canada.
- *1913. McKimmie, O. A., Washington, D. C.
- *1885. McSherry, H. Clinton, Baltimore, Md.
- *1914. Mackenty, John E., New York City.
- *1898. Makuen, G. Hudson, Philadelphia, Pa.
- *1881. Major, G. W., Montreal, Canada.
- *1881. Morgan, E. C., Washington, D. C.
- *1886. Mulhall, J. C., St. Louis, Mo.
- *1925. Mullin, William V., Cleveland, Ohio.
- *1914. Munger, Carl E., Waterbury, Conn.
- *1892. Murray, T. Morris, Washington, D. C.
- *1881. Mynter, H., Buffalo, N. Y.
- *1893. Newcomb, James E., New York City.
- *1895. Nichols, J. E. H., New York City.
- *1894. Park, William H., New York City.
- *1892. Porcher, W. Peyre, Charleston, S. C.

- *1927. Porter, Charles T., Boston, Mass.
- *1908. Randall, B. Alexander, Philadelphia, Pa.
- *1882. Rankin, D. N., Allegheny, Pa.
- *1902. Richardson, Charles W., Washington, D. C.
- *1879. Roe, John O., Rochester, N. Y.
- *1881. Robertson, J. M., Detroit, Mich.
- *1913. Roy, Dunbar, Atlanta, Ga.
- *1878. Rumbold, T. F., St. Louis, Mo.
- *1879. Seiler, Carl, Philadelphia, Pa.
- *1893. Shields, Charles M., Richmond, Va.
- *1878. Shurly, E. L., Detroit, Mich.
- *1892. Simpson, William Kelly, New York City.
- *1919. Skillern, Ross H., Philadelphia, Pa.
- *1909. Sluder, Greenfield, St. Louis, Mo.
- *1879. Smith, Andrew H., Geneva, N. Y.
- *1932. Smyth, Duncan Campbell, Boston, Mass.
- *1928. Sonnenschein, Robert, Chicago, Ill.
- 1924. Stein, Otto J., Chicago, Ill.
- 1934. Stevenson, Walter, Quincy, Ill.
- *1911. Staut, George C., Philadelphia, Pa.
- *1879. Tauber, Bernhard, Cincinnati, Ohio.
- *1903. Theisen, Clement F., Albany, N. Y.
- *1899. Thorner, Max, Cincinnati, Ohio.
- *1892. Thrasher, Allen B., Cincinnati, Ohio.
- *1937. Tobey, George L. Jr., Boston, Mass.
- *1888. Van der Poel, S. O., New York City.
- *1938. Vail, Harris H., Cincinnati, Ohio.
- *1936. Voislowsky, Antonie P., New York City.
- *1933. Wanamaker, Allison T., Seattle, Wash.
- 1896. Ward, Marshall R., Pittsburgh, Pa.
- *1879. Ward, Whitfield, New York City.
- *1886. Westbrook, Benjamin F., Brooklyn, N. Y.
- *1924. Wherry, William P., Omaha, Neb.
- *1924. White, Leon E., Boston, Mass.
- *1922. Wishart, David J. G., Toronto, Ontario.
- *1896. Woolen, Green V., Indianapolis, Ind.

