Transactions

American Laryngological Association

NINETIETH VOLUME

"DOCENDO DISCIMUS"

TRANSACTIONS

OF THE

NINETIETH ANNUAL MEETING

OF THE

American Laryngological Association

1969

HELD AT

THE ROOSEVELT HOTEL

NEW ORLEANS, LOUISIANA

MARCH 23 AND 24, 1969

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First Row: Brown, Knight, A. Miller, Boies, Holinger, Galloway, Woodman, Hill, Murtagh, Davison, Lathrop, Schall, Clerf, Theobald, LeJeune, Salinger, Van Alyea, McCall, Kirchner. *Second Row:* Lewis, Fitz-Hugh, McCabe, Norris, Sisson, Priest, Cocke, Harris, DeWeese, Daly, Goldman, Bordley, Walsh, Trible, Fearon, Hoople, Atkins, Cracovaner, Weille, Lederer, Simonton, Schenck, Andrews, Lewy, Reed, Suehs. *Third Row:* Friedberg, Bryce, Harrill, Barretto, Ogura, Tabb, Skolnik, Hilding, Hart, Goodale, Richardson, Pratt, Henry, Ferguson, Dickinson, Farrior, Chandler, Work, Keim, Arnold, Hilger. *Fourth row:* Cardwell, Ausband, Tolan, Cody, Eagle, Baxter, Munoz-MacCormick, Montreuil, Tremble, Baker, D. Miller, Saunders, Alexander, Weaver, Silcox, Putney, Hanckel, Alfaro, Jones.

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TRANSACTIONS OF THE
NINETIETH ANNUAL MEETING
OF THE

American Laryngological Association

HELD AT
THE ROOSEVELT HOTEL
NEW ORLEANS, LOUISIANA
MARCH 23 AND 24, 1969

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The Ninetieth Annual Meeting of the American Laryngological Association was called to order by the President Dr. Francis W. Davison, at the Roosevelt Hotel, New Orleans, Louisiana on March 23, 1969, at 8:15 A.M.

REGISTRATION

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Kirchner, John A.
Knight, John S.
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Lewis, John S.
Lewy, Robert B.
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McCall, Julius W.
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Miller, Daniel
Montreuil, Fernand
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Norris, Charles M.
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O'Neil, James J.
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Schall, LeRoy A.
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Sisson, George A.
Skolnik, Emanuel M.
Suehs, Oliver W.
Tabb, Harold G.
Theobald, Walter H.
Tolan, John F.
Tremble, G. Edward
Trible, William MacL.
Van Alyea, Oliver E.
Walsh, Theodore E.
Weaver, Delmar F.
Weille, Francis L.
Williams, Henry L.
Woodman, DeGraaf
Work, Walter P.

SCIENTIFIC SESSION

SUNDAY MORNING, MARCH 23, 1969

The first Scientific Session of the Ninetieth Annual Meeting of the American Laryngological Association, Inc., held at the Roosevelt Hotel, New Orleans, Louisiana, convened in the Grand Ballroom on Sunday morning, March 23, 1969, at nine-five o'clock, President Davison presiding.

PRESIDENT DAVISON: Lyman Richards was a wonderful person. He lived with enthusiasm, as you know. Everything he did he did with great enthusiasm. He loved his family, he loved medicine and he loved the American Laryngological Association and for the last ten years before his death was its very active and efficient Secretary.

I have asked Dr. Charles Ferguson to give us some highlights of Dr. Lyman Richards' career.

§

LYMAN GILDER RICHARDS, M.D. — AN APPRECIATION

CHARLES FOSS FERGUSON, M.D.

BOSTON, MASSACHUSETTS

Today our President planned to honor the man in person who had so skillfully guided this Association through the past eight years, our ever genial, always efficient and thoroughly dedicated Secretary, Lyman Gilder Richards. It pleased him tremendously to accept this high honor, but it was, however, with easily understandable reservations, realizing he was soon to face major cardiac surgery. At the same time, well-disguised apprehensions and vague misgivings in no way diminished his true appreciation, since this Association was the closest to his heart. All of us remember at the time of his retirement as Secretary last year his witty, even casual, comments on his health. Very few had any inkling of the stoic effort he continually manifested as he conducted those meetings during

the days at Hollywood Beach, constantly striving not to divulge his increasing physical incapacities. He even passed off his approaching major surgery as simply a "valve-grinding job."

As we are once again meeting together, today should be a sad one for us, since he is not here in person to accept this richly deserved honor. Yet we know undue sadness would not be keeping faith with such an indomitable spirit who constantly radiated such happiness. All who really knew him feel that in spirit he is still with us. Such an exuberant personality, so ever buoyant, and so full of the true "joie de vivre," can never die as long as there is one person left to remember him. His vitality and always youthful enthusiasm will continue to be felt among us for a long time to come. Those who knew him well are also certain that he would fervently wish to be remembered as the harbinger of joy and good fellowship, and definitely not be mourned with long faces or any solemn ceremonies. Always a striver for perfection, he was constantly annoyed at reading glaring errors and inaccurate, misleading statements in published obituaries of his friends and colleagues. For this reason, years before his death he prepared his own obituary, always to be ready for that inevitable day. It was an accurate compilation of facts he earnestly, but skeptically, hoped the news media would not distort. Funerals and memorial services always depressed him, and he believed such ceremonies should be appropriately upgraded. He felt this so strongly that he personally wrote and arranged his entire funeral service in order to spare his family and friends unnecessary hardship and grief. As one might expect, that service was cheerful and forward-looking, a virtual symphony composed in a major key, full of hope and expectation, conveying no minor key somberness signifying defeat or death. It truly exemplified his ever-radiant spirit and was a fitting last tribute to a completely fulfilled life of rich accomplishment.

Lyman was born in Marion, Massachusetts in 1893, the son of George Lyman Richards, M.D., a nationally recognized otolaryngologist and a former President of The Triological Society, an honor which was also later accorded to his distinguished son. In 1912 he was graduated from Phillips Exeter Academy, and in 1915 he received his A. B. degree cum laude from Harvard College, completing the entire academic course in three years. He was also elected a member of the Phi Beta Kappa Society. Harvard awarded him the M. D. degree also cum laude in 1919, and he attained membership in Alpha Omega Alpha. After a surgical internship and residency at St. Luke's Hospital in New York City, he served as resident at

the Massachusetts Eye and Ear Infirmary. He was one of the earliest Diplomates of the American Board of Otolaryngology, receiving certification in 1926 following postgraduate courses in Philadelphia with Chevalier Jackson, Louis Clerf and Gabriel Tucker, Senior.

My first personal contact with him was in 1935 when I began my career at The Children's Hospital in Boston where he was Chief of Otolaryngology. Since this was a relatively small service in comparison to those of medicine and surgery, it had always been regarded as an "orphan," poorly equipped and in dire financial straits. Not to be daunted after having accepted this new challenge, he generously gave much of the equipment for the throat and bronchoscopic operating rooms in memory of his mother, "Mary L. Richards, a lover of children," as the bronze cabinet still reads. He also paid out of his own pocket the first few residents whom he justifiably considered essential to the proper functioning of a developing and active department. After my residency was completed in 1938, for three years we shared suburban offices in Wellesley Hills. During the early war years, although he was at that time Chief at the Peter Bent Brigham Hospital, his patriotic concern and natural restlessness could be contained no longer, and he joined the Navy Medical Corps as Lieutenant Commander. His first assignment was that of Marine Recruiting Officer in Springfield, Massachusetts where it was quickly discovered he had been "hiding his light under a bushel." It was then arranged that he be transferred to the South Pacific. While in transit, as the only medical officer on shipboard, he performed an appendectomy. As one would suspect, even though his days of abdominal surgery were far behind him, it was most successful. At Pearl Harbor, where he remained until the cessation of hostilities, he was happily in more familiar "otolaryngologic surroundings."

Following his return to private practice, he became bronchoscopist to the Massachusetts Eye and Ear Infirmary under Dr. Schall who had succeeded Dr. Mosher. Bronchoesophagology had always been one of Lyman's greatest interests. Later, at a time when most men considered retirement, he looked around for more challenging oportunities, and became otolaryngologist to Massachusetts Institute of Technology, working regularly in the clinic until his 70th year, and justly feeling he still had valuable services to offer his fellow men.

Lyman contributed greatly to all of the organizations to which he belonged, serving for ten years as Editor of the Triological So-

ciety, and in 1948 as its President. For four years he was Secretary of the American Broncho-Esophagological Association and President in 1939. In 1964 at our San Francisco meeting he was the Guest of Honor, and the masterful address he delivered on that occasion will long be remembered. From 1960 to 1968 he served as a most dedicated and efficient Secretary of the American Laryngological Association, and in 1966 received its coveted Newcomb Award, an honor which pleased him intensely. Only confinement to a hospital bed and the absolute insistence of his surgeon kept him from attending in person that meeting in San Juan which he had planned so carefully, and to which he had looked forward so long. His acceptance speech, however, was conveyed to the dinner by a very realistic transcription from his hospital bed, and his familiar voice brought a fine personal touch to the occasion, even though we had to forego his customary musical renditions. Very reluctantly, but deferring to his better judgment, last year he saw the handwriting on the wall and offered his resignation as Secretary. We still remember his very poignant remarks on relinquishing this dearly beloved portfolio, a step he honestly dreaded to take, but felt was in the best interest of the Association.

Although he published several papers in the otolaryngologic field, especially on laryngotracheo bronchitis, his greatest contribution was his text book "Otolaryngology in General Practice." Thirty years have elapsed since this was offered to the profession, but even today it still contains countless up-to-date "words of wisdom," including valuable suggestions for the management of common problems still encountered by the general practitioner, as well as sage advice as to when he has reached his limits and referral to a specialist becomes desirable. My personally autographed copy inscribed, "To Fergie with kindest regards from his Wellesley Partner," will always be a prized possession.

His writings never failed to bear the hallmark of a master of the English language and the reading of them has always been a source of great delight. His "Half Century of Triological Transactions," given at the 1954 meeting in Boston, was a literary masterpiece, the fitting culmination of a monumental task of devoted research, always challenging to his everinquiring mind. From this memorable monograph two quotations are especially worth recalling.

In apologizing for the omission of individual names he writes, "Granted that some stars have shone with greater brilliance in the otolaryngological firmament than others, nevertheless so many work-

ers long since forgotten have done so much to illumine the darkness, that it would do them grave injustice to single out only planets of the first magnitude." And the following delightful paragraph reminiscent of another medical literary genius, Sir William Osler, "Whatever miraculous discoveries may be ahead, the otolaryngologist will, I am sure, do well to continue to place himself beneath the sheltering wings of the guardian angels of the doctor, namely, 'Common Sense,' 'Surgical Judgment' and the 'Study of the Individual'." (Page 437, Triological Transactions, 1954.)

It would be impossible to describe the innumerable interests of such a versatile and always intensely curious soul, so infinitely did they involve so many fields of human knowledge. Music was especially dear to him. He was a member of the Wellesley Community Orchestra. We all recall his enjoyment, and ours too, when at times he borrowed the violin from one of the performers during the cocktail hour, and either played a solo or merely went along with the rest of the orchestra, often amusingly replacing the conductor. Years ago after office hours which he sometimes purposely hurried along, he would have time to practice on his violin, much to the consternation of other medical tenants who still had patients. When vibrant chords from his saxophone filled the corridors, doors of distant offices would be thrown open and disonant, much harsher sounds from human vocal cords would mingle with those of the saxophone or violin. With a characteristic twinkle in his eye, at the same time feigning utmost disdain, he would exclaim, "The ingrates! It just goes to prove what you get for nothing is never appreciated. After all, I was only trying to entertain them."

His flair for the dramatic was fulfilled on many occasions when he wrote the script and skillfully directed in rehearsal most clever skits, modelled after then popular radio or television shows. These were presented at the Triological and Laryngological Association dinners, and also at the 1957 International Congress in Washington. Many members and their always-willing spouses gamely participated. One of the most memorable was that of Allen's Alley, the popular radio program 20 to 30 years ago, produced in 1948 when he was President of the Triological Society. Later on at Dr. Schall's retirement dinner, the assembled multitude adjourned to an adjacent theatre where "This is Your Life" was presented in truly professional and theatrical style.

Always a devoted family man, as the years went by he looked forward with even increased interest in the activities and accom-

plishments of his two sons, his daughter and his seven grandchildren. Fortunately, only a week before he went to the hospital for his final illness, a birthday brought the entire family together for a happy reunion. Always young in heart and spirit, as well as in body, he taught Sunday School for years, thoroughly enjoying the close contact with the younger generation. Indicating his appeal to "all sorts and conditions of men," for some time he conducted a course in conversational French at the New England Old Age Center. For physical activity he was a golfer, a squash player, and an avid skier, frequently enjoying the mountains of New Hampshire. When this undertaking seemed a little beyond his reach, he could be found on the hills of Wellesley, and even three months before his death at age 75, still suffering from severe aortic stenosis, he managed a ski trail which he constructed in his own backyard, where in more clement weather he delighted in the cheerful labors of his garden. Confirming his eternal youth, I should like to quote from his Guest of Honor address before The American Broncho-Esophagological Association in 1964. "It is inevitable that with the passage of time one's active participation in the work of such an Association as this must progressively diminish. I hope, however, that I still retain a keen interest in this Society's accomplishments, particularly those of the younger men as they continue so ably to fill the gaps left by those like myself who move on. My interest in these younger successors reminds me of a colorful scene in that theatrical gem, "Teahouse of the August Moon." The American captain developing a new island community feels that only the importation of a young woman is needed to round out its fulfillment, and so sends a delegation of young Okinawans to a neighboring island to bring back the beautiful "Lotus Blossom." Noting that this young group is being joined by a venerable and white-bearded gentleman desiring to assist in this mission, the captain seeks an explanation for this incongruity from his genial interpreter, Sakini. With a broad grin displaying his pearly teeth, Sakini replies, 'Man old, Boss, but not dead yet'." This truly typified Lyman himself. (Transactions American Broncho-Esophagological Association, 1964, page 20).

Mingled in the person of Lyman Richards was that rare but fortunate combination of human attributes which together produced a sympathetic physician and a skilled surgeon, an enthusiastic teacher and philosopher, a talented author, an accomplished musician, a clever dramatist, a sports enthusiast, and more recently a family historian, truly a man of universal tastes and catholic interests. Who could ask for more? He was always blessed with boundless

energy, constant, youthful enthusiasm, a genial nature, and an inborn natural curiosity. Above all, his extraordinary courage was never more manifest than his last months of necessarily limited physical activity.

This was a man one had to know personally in order to appreciate his many endearing qualities. Those of us who were privileged to know him well over many years loved and respected him. Now we sorely miss him, especially at these meetings he always enjoyed so thoroughly. He was a soul richly endowed with countless natural talents which he fortunately was able to mobilize and to utilize so effectively.

Now speaking for all of us, and for the Association he so devotedly served, we are happy to grant to him that age-old approbation which he has truly earned, "Well done, thou good and faithful servant."

INTRODUCTION OF GUEST OF HONOR, HENRY L. WILLIAMS, M.D.

PRESIDENT FRANCIS W. DAVISON

It is a great pleasure for me to introduce next a young man who for about 30 years has studied nasal physiology and mucosal misbehavior. He has read widely and thought deeply. I don't know anyone better informed about the problems of Otorhinolaryngology than Dr. Henry L. Williams, our Honored Guest.

§

REMARKS OF THE GUEST OF HONOR

H. L. WILLIAMS, M.D.: I find considerable difficulty in attempting to rephrase Dr. Lyman Richards. In spite of the eulogy that has just been given, I remember him best for, I think, the only good paper that I have read on post nasal drip. I think this is really a remarkable production and I include it amongst the gems of otolaryngologic literature that it has been my pleasure to read. So often we are merely subjected to the more dramatic aspects of our specialization and sometimes these things which we see so often that we are really ignorant about are not emphasized to the point one wishes they were.

This present paper is one which I presented in the hope that we might have greater interest in Rhinology, which I believe has failed because of possible lack of appreciation for the physiology and I think this ignorance has interfered with the understanding and also with the practice of Otolaryngology.

I believe that the fact that "rhinology" has been dropped from the title of some of our specialties is based on this lack of understanding.

THE NOSE AS FORM AND FUNCTION

HENRY L. WILLIAMS, M.D.
W. ST. PAUL, MINN.

Nasal form as indicative of an important function as part of the respiratory organ system, is at best thought mildly amusing; at worst it is called an absurdity.

In the less sophisticated era in which modern rhinology began, there was no doubt in the mind of the rhinologist that the human nose played a role in respiratory control. Just from looking at it, it was plain to him that the nose is a series of valves and it is reasonable to suppose that a valvular structure at the beginning of the airway must have a regulatory function. It was soon suggested, however, that such an intuitive belief is naive and unworthy of a true "naturalist," as scientists called themselves in those days. As a scientist, a rhinologist must subject each hypothesis to "the vigorous indigation of efficients" so as to separate the factual from the merely fanciful, if he is to escape castigation as "teleologist" or "arm-chair scientist." This doctrine was so new, so authoritarian and so seemingly correct, that these terms were accepted by the rhinologist almost without resistance, as being justly opprobrious. It has been recognized only recently that trying to construct a physiologic whole by addition of a "shambles of measurable but disconnected units" may destroy highly relevant data, for as Weiss[1] stated, it is not that the whole is *more* than the sum of its parts but that it is so different.

The early skeptics (and a good many later ones) threw out the functional baby with the teleologically tainted bath water when for the ensuing century rhinologists failed to secure data that could support a respiratory control function for the nose.

Although it is often stated that past diseases in "the upper respiratory tract" are the cause of a large number of disorders for

From the Otology, Rhinology and Laryngology Service, U.S. Veterans Administration Hospital, Minneapolis, Minnesota.

which the patient consults the internist, that present, active, acute or chronic, disease or functional disorder, in that part of the upper respiratory tract constituted by the nose and paranasal sinuses, can have a significant impact on the general health of the organism is often denied. In spite of its elaborate anatomic construction and the demonstration of extensive reflex connections, not only between the nose and the lung, but also between it and the entire external integument, the generative and abdominal organs, the endocrines and even the psyche, much mention of nasal function is cleverly avoided in our schools.

It was not until Ogura[2] pointed out that to measure a possible influence of the nose on respiration, it is, in general, at least necessary to correlate nasal airway resistance with the mechanics of pulmonary function, that valid support for the proposition that the nose is part of a regulatory mechanism for respiration was secured.

The present consensus seems to be, as Thompson[3] put it, "Still, all the while, like warp and woof, mechanism and teleology are woven together, and we must not cleave to the one or despise the other."

DISCUSSION

In considering nasal physiology it seems reasonable, on the basis of both function and convenience, to divide it into three parts: 1) those of air conditioning, 2) of resistance to invasion by pathogenic microorganisms, and 3) of its activity as a factor in control of respiration. All three of these parameters are interesting and important, but the last seems to have caused the most uncertainty, doubt and recriminative discussion among rhinologists and will be the one considered here.

It may be instructive to note that at first, rhinosinal disease was not a matter of great interest to the rhinologist. It seemed so obvious to him that obstruction to normal ease of passage of inspired or expired air must have a pernicious effect on the general health of the entire organism that "nasal stenosis" was considered the most common and most important pathergy. Kyle[4] described two varieties of nasal stenosis, 1) "anatomic" stenosis, and 2) "reactive vasomotor" stenosis. He thought that the latter had a causal relationship to asthma and other diseases of the lung. Burnett[5] remarked, "Removal of the turbinated bodies in a state of hypertrophy is principally indicated for the relief of nasal stenosis and its immediate results; habitual mouth breathing, rhinorrhea and excessive accumulation of

nasal mucus; as well as for more remote results such as serous otitis media, obstruction of the lachrymal duct, nasopharyngitis, laryngeal hyperemia, laryngitis and secondary lung disease. Removal of the turbinated tissues is also indicated for the relief or removal of various reflex neurotic conditions, directly or indirectly traceable to these structures when in a state of relative hypertrophy."

This simple ingenuous advice led to a destructive surgery which only rarely gave relief and often added more severe complaints to those the procedures were designed to relieve.

Poor surgical results caused some prominent rhinologists to doubt the validity of the hypotheses which were our guidelines, and they began to question the propriety of such surgery. Skeptics, however, said it was not the stirrings of conscience in the rhinologist, but an increasing tendency for the patient to take flight from the rhinologist's chair as soon as he heard the word "operation" that caused the former to abandon many of these misguided practices. Actually, leading rhinologists protested that too many rhinologic operations were being done for inadequate indications. They said that the indication for nasal surgery should be disturbance of nasal respiratory function, and its objective, restoration of such function to normal. Some rhinologists, however, derided the suggestion that there might be a "physiologic surgery," asking how disturbance of a function that is not well understood can serve as a surgical indication, and what operation can restore a nose to a normal that has not been ascertained.

These and similar questions stimulated a number of rhinologists to do research on nasal respiratory function. The first may have been Paulsen,[6] who studied particularly the pathways of inspired and expired air through the nasal chambers. His findings, in general, have been repeatedly confirmed by subsequent investigators.

Courtade[7] attempted to make judgments as to the openness of one or the other nasal passage by having the patient expire on a cold mirror held just inferior to the nares, the nasal conductance being estimated relative to the areas of condensation of the water vapor in the exhaled air on its surface.

Franke[8] demonstrated "swirls" in respiratory flow and also devised a method which he believed related inspiratory pressure to air flow. He placed an oval tube, about 8 cm in length, with a radius of about 9 mm and a cross section of about 200 mm^2 in each nares. A lateral opening with an attached tubule was made in each of the

tubes of the model nose and led to a manometer, by which inspiratory and expiratory pressures were measured while the patient breathed. Franke reported that in symmetrical noses under normal conditions, when breathing through one or the other side of his "nose model" alone, the pressure was about twice as great on this single side as when breathing through both sides together.

A year later Kayser[9] used a rhinometric technique in which the concept of "driving pressure" was considered. By placing an olive connected to a manometer in the nostril of that side of the nose which was not being used to measure flow, the nasal chamber then acted as a transducer of pressure from the pharynx. He thus measured the pressure difference between the pharynx and ambient atmosphere through one nasal passage while measuring the air flow it produced through the other.

These two techniques, or modifications of them are called, respectively, anterior and posterior rhinometry. Until recently these were the only techniques used in attempts to find an "absolute" quantitative measurement of normal ranges of the driving pressure air flow relationship in the nose that might be related to respiration in general. Unfortunately, the data obtained were applied to establishing indications for "lumen enlarging" operations on the nose. When it began to be noticed that the pre-operative opinion as to the presence or absence of "nasal stenosis" formed on the basis of such rhinometry did not always coincide with the patient's complaints, and further that the expected symptomatic improvement from a given lumen enlarging technique frequently did not occur, protests were again heard.

At this time Goodale,[10] using slight modifications of the methods of Franke[8] and Kayser[9] undertook an intensive investigation of nasal respiratory physiology. His paper, published in 1896, deservedly received the Boylston Medical Prize of Harvard University.

Goodale stated, "Physiologically considered, the subject of nasal respiration resolves itself naturally into the following divisions: 1) The action of the nasal mucous membrane upon respired air with regard to heat and moisture; 2) Chemical changes effected in the air current by the nasal mucous membrane; 3) The normal alterations in intranasal air pressure during the respiratory act."

In regard to the first category, humidification and temperature control, Goodale's experiments were so sound that his report is superior to many later observations on this facet of nasal physiology.

He found that the nose contributed only about two-thirds of the moisture necessary to saturate inspired air of average relative humidity. This can be contrasted with the statement of some later authors that saturation of the inspired air for proper gas exchange in the pulmonary alveoli is probably the principal nasal function. It was not until Negus[11] pointed out that the primate nose has regressed functionally from that of fur bearing mammals that it was realized that "air conditioning" is a subsidiary and a relatively less important nasal function, a sort of physiologic afterthought. Goodale did not elaborate on the second division, and it will not be considered further.

The third category relates to changes in intranasal air pressure and resistance in relation to flow during respiration and has been the subject of discussion since the dawn of modern rhinology. Goodale concluded, "Inasmuch as the manner of breathing of each individual is dependent upon a natural or acquired habit peculiar to the person in question, absolute figures denoting respiratory alterations in air pressure are out of the question, even under wholly physiological conditions. In any case we must be satisfied with an approximate mean. Experiments instituted, therefore, for the purpose of comparing abnormal with normal pressure changes, must inevitably include considerable error, owing to the fallacy involved in attempting to obtain absolute results from relative and variable factors."

It would seem reasonable to infer from these statements that Goodale opposed the clinical use of rhinometry in attempts to establish normal or abnormal function for a given nose, either prior to or following surgical manipulation, or at any other time. He saw that the data secured by measurement of the pressures and flows in an isolated nasal chamber could not, in themselves, relate to overall respiratory function nor establish respiratory dysfunction as being caused by the nose. His opinions were sound when uttered and remain so to this very day.

Rhinometry at that time appears to have suffered from overeagerness of the rhinometrist to apply a "scientific" method of measuring nasal function that would tell him what sort of surgical manipulation should be done and upon what part of the nasal chamber it should be practiced. He was unwilling to consider rhinometry as an adjunctive examination to be combined with the more important history, physical examination and, above all, previous clinical experience, to arrive at a well reasoned conclusion such as is encompassed by the term "clinical judgment."

As a result of Goodale's paper, conservatism in nasal surgery received a tremendous impetus. Holmes[12] was able to state: "When for any reason the middle turbinate and lower wall of the ethmoid have to be sacrificed, there results a deformity which practically produces all of the ill effects of mouth breathing. The inspired air is not sufficiently warmed or moistened and there almost always follows a chronic dry pharyngitis and chronic laryngitis. Frequently the trachea and bronchi suffer in like manner as the upper respiratory organs. These patients almost always suffer from orbital as well as nasal disturbances whenever they are subjected to the influence of cold or dry air, or to air contaminated with irritating dust or gases."

It is unfortunate that the teaching of physiologically oriented rhinologists such as Goodale and Holmes was not taken more to heart. In a short time their words were forgotten and it was again recommended that the first step in ethmoidectomy, merely as a matter of convenience, should be the removal of the middle turbinate. This was the recommendation of Yankauer,[13] although he recognized that this procedure is not without undesirable after effects. He said, "The conservation of function is of special importance . . . for the middle meatus is the main respiratory channel through which the greatest part of the inspiratory air passes. The demands made upon this region are greater than upon all the rest of the nose together. To remove the entire diseased organ may be one way of terminating the disease, but it is not the way to cure the patient," and again, "In the large majority of the cases the healing of the wound is completed in six to eight weeks, and further treatment is unnecessary. But the function of the parts has not yet been completely restored. . . . During this time the nose is sensitive to sudden changes in atmospheric conditions so that attacks of acute inflammation . . . of the mucous membrane are apt to occur. These attacks continue until the nose has experienced *at least* [italics not in original] one complete cycle of such changes, i.e., until one year has passed. During these attacks the mucous membrane becomes swollen, edematous and greyish in appearance, sometimes distinctly polypoid." Although in these paragraphs Yankauer was limiting his observations largely to the effect of destroying the tempering and humidifying power of the middle turbinate, he tacitly admits that meddlesome removal of structures having an important respiratory function will result in iatrogenic inflammatory symptoms which might have an unfavorable impact on the health of the patient.

Goodale's paper resulted in virtual abandonment of interest in rhinometric assessment of nasal respiratory function in North America for a long time. It is not likely that this was Goodale's intention. If one is to judge from his paper, his intellectual stature would forbid him to adopt an anti-experimental position. Opposition to rhinometry was attributed to him, however, so that whenever a rhinologist evinced interest in a trial of rhinometry, the general flock of rhinologists rallied to the attack, bleating the rallying cry of Goodale!! Goodale!! This may have been due to the fact that such rhinologists found themselves relaxed in the comfortable environment of old fashioned, destructive, lumen enlarging surgical procedures (a rhinologic unthink that is still supported by some rhinologists). Goodale was used as a stalking horse by which such individuals justified and distracted attention from their nefarious activities. The attitude of American rhinology, in general, had become "don't annoy me with evidence; my mind is made up."

Tonndorf's paper[14] explains one of the reasons why rhinometry had not been successful in obtaining values of the resistance-flow relationship that could pronounce one range of resistance or one range of conductance or the relationship between them, normal or abnormal. He started on this study, he said, because his father had called his attention to clinical opinions that did not fit well with his own observations as to the behavior of nasal resistance and its relation to the flow rate of respired air. He had found that in septal deflections the narrowed side might permit a better passage of air than the apparently more open side; or that after resection of an apparently blocking enlargement of turbinate or septum, the patient might have more respiratory obstruction than before, although upon inspection the nasal chamber appeared wider. Also, that in several forms of atrophic rhinitis a large cavity may exist in the nose as a result of the disease which is associated with increased resistance and reduced conductance.

By his experiments on a cast of the nose through which he passed his own respiratory air, much as Goodale had done with his parallel glass tubes, he was able to demonstrate that these seeming inconsistencies resulted from the difference between laminar, unstable and turbulent flow.

Previous rhinometrists had assumed that nasal resistance is a simple function of the air pressure and of the flow rate as is expressed in Ohms law in an equivalent electrical circuit: $R=\frac{\triangle P}{i}$, where $\triangle P$

is the potential difference in volts, i the current in ampheres and R the resistance in Ohms. While as Mead[15] showed, equivalent electrical circuits are useful in estimating pressure-resistance-flow relationship in the lower airway where flow may be laminar, Tonndorf found their use in the nose produces invalid data for aerodynamic reasons.

At velocities below Mach 1, as Tonndorf pointed out, air behaves like an incompressible fluid and the laws of hydrodynamics apply. For the ideal case of air streaming frictionless through a tube of constant diameter, the pressure is the same everywhere along the tube. If the diameter of the tube is variable, however, and the presence of friction is assumed, as in the human nose, pressure changes occur according to Bernouille's law, which states that with fluid of constant volume flowing through a tube of irregular diameter, the pressure is greatest where the cross section is greatest and the velocity least. This may be stated mathematically in the equation $Pst \times \frac{1}{2} \varrho \bar{V}^2 = K$, a constant, where Pst is the static pressure, ϱ the coefficient of density, $\bar{V}$ the mean linear velocity and K a constant. The expression $\frac{1}{2} \varrho \bar{V}^2$ is also the aerodynamic pressure.

Hyatt and Wilcox[16] state that the pressures along a stream of gas flowing through a non-uniform conduit system may be analyzed by considering two properties of the flow, its frictional properties and its inertial properties. At any moment there are two superimposed pressure drops along the system related to these properties. A frictional pressure drop will exist related to the physical properties of the gas and to the flow geometry of the airways. An inertial pressure drop will also exist related to the density of the flow and to the acceleration of the flow. The acceleration of flow is of two varieties, local and convective. Local acceleration is the rate of change of velocity of the gas particles that occurs with time, such as in alternation of flow. Convective acceleration is the rate of change of velocity of the particles that occurs with the distance traveled by the gas particles, such as would occur with converging or diverging flow boundaries. This is the part of the pressure-velocity relationship that is considered under Bernouille's law. In both cases, following Newton's second law, the rate of change of velocity must be accompanied by a force pressure gradient. Hyatt and Wilcox find the transient pressure drop associated with local acceleration negligible in the lower airway, but state that the pressure drop associated with convective acceleration has not been thoroughly studied and may not be negligible.

In addition to the negligible factor of local acceleration (which may not be negligible in the nose) and that of convective acceleration which at the "ostium internum," in that part of the airway formed by the nasal vestibules and nasal chambers which has the form of parallel Venturi tubes, is probably considerable, the factor of dynamic internal molecular friction, according to Tonndorf, plays the major role in causing resistance to air flow. Reynolds[17] had stated, "that although the theoretical calculations of Sir Gabriel Stokes seemed to agree so closely with the experimental determinations of the flow of fluid through pipes as seemingly to prove the truth of the assumption involved, this was the result of comparing the flow of water through uniform small tubes and at slow velocities." Reynolds found these results to vary, both theoretically and practically, with water moving at higher velocity through larger tubes. He said, "In 1883, I succeeded in proving . . . that when water is caused by pressure to flow through a uniform smooth pipe, the motion of the water is *direct*, i.e., parallel to the sides of the pipe, *or sinuous*, i.e., crossing and recrossing the pipe, according as $\overline{V}$, the mean velocity of the water, as measured by dividing Q, the discharge, by $\triangle$ the area of the section of pipe, is below or above a certain value given by ϱ/μ K/D, where D is the diameter of the pipe, ϱ the density of the water, μ the viscosity and K a numerical constant, the value of which according to my experiments, and, as I was able to show, to all the experiments by Poiseuille and by Darcy, is for pipes of circular section between 1900 and 2000, or in other words, steady direct motion in round tubes is stable or unstable according as $\varrho \frac{D\overline{V}}{\mu} > 1900$ or < 2000, the number K being thus a criterion of the possible maintenance of sinuous or eddying motion."

He pointed out that the existence of a sudden change in the law of motion of fluids between solid surfaces when $D\overline{V}\,\mu/\varrho$ K proved the dependence of the manner of motion of the fluid on a relation between the product of the dimensions of the pipe multiplied by the velocity of the fluid, and the product of the molecular dimensions multiplied by the molecular velocities which determine the value of μ for the fluid, also that the equations of motion for viscous fluid contained evidence of this relation.

Tonndorf calculated the Reynolds number (Nre) for a normal human nose with respirations of 16 per minute at approximately 1000. He stated, "This means that at the lowest flow rates the airflow is still laminary but being close to the borderline — 1160

(Rohrer:Recrit for the nose) — probably not very stable." This theoretical result was confirmed by subsequent experiments. He observed that eddying made its appearance under a variety of conditions: A) when the breathing rate was only slightly accelerated, B) when the cross-sectional diameter was altered in some section of the passageway, either 1) by narrowing the lumen, e.g., by causing swelling of the middle turbinate, or 2) by widening the lumen, e.g., as in an atrophic nose, and C) each time that the direction of flow was reversed, even during quiet respiration. Tonndorf found the path of inspired and expired air to be similar to that originally described by Paulsen and by Franke. It is only recently that Masing,[18,19] using a transparent nose model and the colored water technique of Reynolds, has been able to produce a three dimensional schema of the paths of air flow through the nasal chambers which is better than Tonndorf's. His findings strongly support the assumption that the pathway of both inspiratory and expiratory air flow is extremely complicated and is not actually comparable to the drawings found in books on nasal physiology such as that of Proetz. It seemed evident to him that the lower area of the nasal cavity must be as important as the upper in aerodynamic function.

Tonndorf supported the statement that in patients with definite, marked atrophy of the luminal walls, objective measurement demonstrated increased resistance and, therefore, that they actually have the obstructed nose they complain of. It is not necessary to evoke the hypotheses of occluding crusts or nasal neurosis to explain the complaint. Tonndorf also demonstrated the very great importance of streamlining to nasal respiratory function of which Thompson[3] said, "In hydrodynamics we try to determine the resistance encountered by bodies of various shapes moving through various fluids at various speeds; and in so doing we learn the enormous, the paramount importance of streamlining. There would be no need for streamlining in a "perfect fluid," but in air or water it makes all the difference in the world. Streamlining implies a shape round which the medium streams so smoothly that resistance is at last practically nil; there only remains the slight skin friction which can be reduced or minimized in various ways. But the least imperfection in the streamlining leads to whirls and pockets of dead water or dead air which means large resistance and waste of energy."

It can be realized from this quotation and from the findings of Tonndorf in regard to nasal conductance that one of the reasons for iatrogenic symptoms following nasal surgery is ignorance of the

hydrodynamics of nasal flow, and meddlesome interference with or failure to restore the streamlined contours of the nasal lumina.

Uddströmer,[20] Tonndorf,[14] and particularly van Dishoeck[21] reported that even if conductance through one nasal chamber is restricted, if breathing is done through both sides of the nose at once, the volume of inspired air may be normal with normal mechanical efficiency of respiration. Ogura's findings explain how a unilateral rhinoscopically observed nasal respiratory obstruction can have an adverse effect on the cardio-pulmonary organ system by reflex constriction of bronchial smooth muscle in the lower respiratory tract. The illogicality of drawing far reaching conclusions as to increased work of respiration by measuring the pressure changes caused by nasal deformity on the occluded side of the nose alone is easily demonstrated by the use of such a device as the mask flow-meter with which pressure-flow determinations may be made on the unimpeded nose free from the influence of rebreathed CO_2. Such measurement usually indicates a steady conductance within a so-called normal range.

From the standpoint of cybernetic theory, the diurnal and day-to-day changes in nasal resistance produced by reflexes from the respiratory "centers" to erectile tissues and nasal respiratory muscles may occur, so that normally the alveolar ventilation may remain constant as long as the work load on the organism remains the same. Therefore, nasal resistance is of interest or importance in normal and abnormal physiology only as it influences the minute volume of respiration, the alveolar ventilation and eventually the work of respiration.

It would seem to be a reasonable deduction from the evidence reviewed that improved alveolar ventilation is not always achieved by surgical procedures which merely increase luminal cross section, but depends much more on maintaining or restoring the smooth rounded contours of the vestibule, the limen nasi, and the turbinates, and avoiding interference with the function of erectile tissue or dilating muscle. This is what is meant by the term "physiologic surgery," as was recently pointed out by Anderson.[22]

It would also seem reasonable to suppose from the information available that a nose can be classified as functionally pathologic, only when under given circumstances it does not allow the passage of sufficient air to prevent hypercapnia, hypoxia and respiratory acidosis (hypoventilation) without an expenditure of energy that is

excessive for the general state of activity of the individual as compared to that of another who has a "normally" functioning nose.

The conclusion that bilateral nasal respiratory obstruction, both when noticeable even with quiet breathing or when present only at increased rates of respiration, can have morbid repercussions on the organism as a whole, however, seems difficult to escape.

Objection has been raised to this hypothesis by the statement that if nasal respiratory obstruction is present even at rest, all the patient needs to do to achieve adequate alveolar ventilation is to breathe through the mouth. There is evidence, however, that this oft repeated statement is a misleading half truth; that, while as an emergency measure, to compensate for an extraordinary demand for a short period, mouth breathing is effective, for respiration over a long period, mouth breathing is unphysiologic and inefficient, causing an increased expenditure of energy for a given alveolar ventilation.

Hellman[23] stated that the physiologic superiority of nasal over mouth breathing lay in the slower, deeper respiration associated with the former. He suggested that slower, deeper breathing gives more and needed time for mass transfer and diffusion of the respiratory gases in the pulmonary alveoli. It has also been suggested by Williams et al.[24] that deeper breathing dilates the more peripheral alveoli better and allows distribution of surfactant in them, both processes tending to prevent micro-atelectasis.

Davies, Haldane and Priestly[25] found that nasal blockage can cause a considerable degree of hypercapnia and hypoxia before mouth breathing will be resorted to.

Lüscher[26] found that blockage of the upper respiratory tract by hypertrophied tonsils and adenoids caused a decrease in the alkali reserve of the blood. This change could be duplicated by artificial occlusion of the nasal airways.

We also have the report of Noonan[27] subsequently supported by reports from several pediatric groups[28,29] that upper airway obstruction caused hypoxia, hypercarbia, pulmonary hypertension and edema, and reversible cor pulmonale even though these children were free to breathe through their mouths.

Comroe et al.[30] stated that a normal resting subject does not experience any respiratory symptoms until airway resistance is increased threefold or more, but that patients with weakened or

fatigued respiratory muscles often suffer severe distress with the addition of even small external resistances. Ogura and his associates[31] have recently found that in dogs occlusion of the nasal airways, in spite of forced feeding, will produce a wasting illness and death.

The available evidence suggests that the nose has an important respiratory control function that cannot be completely compensated for by mouth breathing. Increased resistance to air flow through the combined nasal airways might, therefore, have a deleterious effect on the general health of the individual through hypoxia and other less well understood physiologic mechanisms. The early rhinologists were correct in making this assumption and may be said to have looked at the nose in the proper way. As Szent-Györgyi[32] stated, form and function are two sides of the same coin. If something cannot be deduced about function from form, it means that form is not being looked at correctly.

CONCLUSION

It does not seem too imaginative to suggest that a control mechanism to regulate breathing so as to secure the maximum mechanical efficiency of respiration, makes use of the series of valvular structures which constitute the nasal portion of the upper respiratory tract. This is supported by the fact that according to van Dishoeck, in mouth breathing the musculature of the pharynx, palate and tongue act involuntarily to make up for the absent nasal resistance. Also according to Murtagh and Campbell[33] the glottic chink, which in normal nasal respiration, is estimated to supply only seven percent of the resistance of the upper airway, in mouth breathing may supply four or five times this degree of resistance. One might deduce from these observations that the nose, the pharyngeal muscles and the glottis are effector devices, a series of mechanical negative feedback controls for inspiratory and expiratory air flow. This would be generally consistent with the cybernetics of blood flow and other vital functions.

It is apparent that understanding of what can and what cannot be demonstrated by rhinomanometry has been hindered by poor communication between those interested in the physical laws of hydrodynamics and aerodynamics, and those interested in how and why respiratory air preferably passes through the nasal chambers.

Reynolds[17] stated that the story as to how certain mysteries of fluid motion, which have resisted all attempts to penetrate them, are

at last explained by the simplest means and in the most obvious manner is closely analogous to the situation present in Poe's story "The Purloined Letter." In this story the Parisian police were completely baffled by the simple plan of destroying all *curiosity* as to the contents of a letter hanging in plain sight. Reynolds said that lack of curiosity is, indeed, no new story in science. This lack seems to have been "endemic" in the rhinologist for a very long time.

Although it has been heard repeatedly that from the standpoint of physics air may be considered an incompressible fluid of low viscosity, I think rhinology may be pardoned for its failure to realize what a major role the internal friction of the air plays in producing increased resistance and decreased conductance in the nasal chambers. Since turbulent fluid flow presented difficulties to even Galileo and Newton, we need not wonder that the pioneer rhinologic surgeons ignored (and even that most of the present generation still ignore) the fact of its presence in planning surgical procedures to correct what they sometimes incorrectly termed "nasal stenosis."

It is clear that the disregard in which rhinology is held among some otolaryngologists at the present time does not descend from a primary failure of our rhinologic ancestors to develop the concept of the "telos" or final purpose of the nose as an organ of the respiratory system, but arose from their lack of knowledge of the aerodynamic laws bearing on the driving pressure-resistance-conductance relationship in the nasal chambers, and of the correlation of nasal conductance with alveolar ventilation.

The hypothesis that the nose is part of a negative feedback servomechanism to match the impedance of the respiratory tract to the demands of alveolar ventilation is admittedly of the imaginative variety. It has, however, received considerable support, sometimes serendipitously, from pulmonary and cardiovascular physiology and the pediatric clinic, as well as from the rhinologic research laboratory.

Sufficient evidence has accumulated to make it seem reasonable to state that the failure of rhinology to procure symptomatic relief in some patients with objective evidence or subjective complaint of "nasal stenosis" has lain in its lack of understanding of the fact, stated by Tonndorf, that we must consider turbulence as the physical factor most influencing resistance in the nose. This being the case, it becomes obvious that by disturbance of streamlining, many of the surgical and semi-surgical methods for "improving nasal conductance" have been incorrectly designed to accomplish their osten-

sible purpose, and often have had the opposite effect. It is suggested that subconsciously this threat to overturn ingrained medical and surgical behavior has furnished a basis for the highly emotional content of some of the opposition to rhinometry.

Rhinometry in North America at the present time is at about the stage of development of audiology in the 1920's and electronystagmography ten years ago. It has a long way to go, but the future seems to promise important developments if rhinometry is given an opportunity to produce advances in knowledge of the relationship of nasal to pulmonary and respiratory physiology which can only be gained by accurate measurement with improved mechanisms and more elegant techniques.

At the present it is unwise to attempt to elevate these speculative and working hypotheses, prematurely, to the status of "established fact." The rhinometric data available, owing to inadequacy of equipment and technique, still falls short of justifying such an attempt.

However, "physiologic nasal surgery" does seem adequately justified in observation of the satisfaction obtained by patients and by the increasing good experiences as to long term results of rhinologic surgeons who base their techniques, whether consciously or not, on the fact that the respired air in obeying Reynolds' "criterion" of hydrodynamics behaves like a viscous fluid.

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PRESIDENTIAL ADDRESS

In Defense of Clinical Research

Each one of you has contributed something worthwhile to the solution of our mutual problems, else you would not have been invited to be a Fellow of this Association.

Clinical research may be defined as that type of investigation based on the study of a group of patients having the same disease syndrome, stating in detail what they had, what was done, and what happened one, two and more, and especially five years later.

Much valid and important work of this type has been done by members of this Association without the help of computers, electronmicroscopes and five-figure grants of taxpayers' money.

Many of the more important investigations have not been supported by taxpayers' money and this in my opinion is distinctly to the credit of the men who have expended their own time and money to explore some facets of our unsolved problems. Grants are essential for extensive research that requires expensive equipment. Acquiring a grant might be likened to marrying a rich widow. Nice if you can do it but then the widow may turn out to be very demanding. I won't carry the analogy any further.

My real purpose is to demonstrate that a man with an idea and the tenacity of purpose to pursue it can make significant contributions despite a limited budget.

In an attempt to substantiate this point I reviewed our Transactions for the past 20 years, selecting two or three *clinical studies* from each program, and noted whether or not it was supported by outside funds.

Admittedly personal bias entered into the selection of these papers. I was influenced in favor of the papers that helped me treat patients more successfully. Other equally important papers would have been chosen by someone else.

The 50 papers selected demonstrated that very fine and clinically useful investigative work can be done without large financial backing. Only three of the 50 papers had a by-line indicating that

the authors had outside financial support for their studies. Thinking and judgment are more important than complex and expensive equipment.

At this point I am reminded of Walter Cannon's[1] story about an overseas biological chemist visiting the old Harvard Medical School. He found Cannon and associates crowded into small, dark, ill-adapted laboratory rooms. After listening to apologies and complaints, he eased them by remarking "I've never noticed that the nature of the cage determined the singing of the bird."

In his 1960 Presidential Address, Dr. McNally[2] devoted considerable time to the subject of research and pointed out very clearly what constitutes ethical research. I think all investigators would do well to reread Bill McNally's Presidential Address.

May I offer some additional references? "The Way of an Investigator," the delightful autobiography of Walter B. Cannon[1] will be enjoyed by all men who are inspired to expend time and effort in working on unsolved problems.

Beveridge[3] in his book, "The Art of Scientific Investigation," recounts many of the great advances in medicine, most of which were made by *one* man and not by a team of technicians working a 40-hour week, less coffee breaks.

Another book worth every man's time is "Controlled Clinical Trials" edited by Professor A. Bradford Hill.[4] In this book there is a chapter written by D. D. Reid entitled "The Patient As His Own Control."

I think the patient can serve as his own control if the physician has an accurate record of the signs and symptoms for a definite period of time before and after initiation of a treatment program. Selection of a reliable test period will depend on whether the condition is acute or chronic.

The insistence on the importance of controls leads me to quote Cannon's[1] story of the biologist who was reported to have baptized one of his identical twins and kept the other as a control. The outcome of this experiment was never reported, so possibly the father did not like the result.

An important paper of the 1949 meeting was H. L. Williams'[5] discussion of "Lethal Granulomatous Ulceration Involving the Midline Facial Tissues." In addition to an extensive report of the litera-

ture, he presented three detailed case reports. This leads me to remark that three cases carefully studied can be more informative than 300 cases abstracted by a junior investigator.

For those who have a large enough series of cases to permit statistical analysis I recommend Bradford Hill's 1966 edition of "Principles of Medical Statistics."[6] I used to be intimidated by mention of the chi-square test. Much valid clinical information has been established without it

I doubt that many of you have recently read the Constitution of the American Laryngological Association so I think it is not amiss to quote briefly Article II which reads as follows: "This Association shall have for its purposes 1) The advancement and promotion of the study of the science of medicine and surgery, devoting special attention to the upper respiratory tract and to engage in and to encourage research with respect thereto. 2) The elevation of standards of the fundamental study and teaching of Rhinology and Laryngology in the undergraduate and postgraduate medical schools of America. 3) The publishing annually of the proceedings of this Association. 4) The honoring of those who by their contributions in the field of Rhinology and Laryngology have aided in its advancement."

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STOP THE MEDICINE

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(Manuscript not available for publication)

DIAGNOSTIC RADIOLOGY FOR THE RHINOLARYNGOLOGIST

GEORGE E. FUNKHOUSER, M.D.
(by invitation)
DANVILLE, PA.

(Slide Presentation)

DIPLOPHONIA

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LOS ANGELES, CALIF.
(by invitation)

JAY W. SANDERS, PH.D. RONALD GOLDMAN, PH.D.

NASHVILLE, TENN.
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GAINESVILLE, FLA.
(by invitation)

Diplophonia is the simultaneous production by the voice of two separate tones. In diplophonia, or diplothongia as it is sometimes called, two sources of tone operate at the same time. The tones are usually similar in quality and loudness, but distinctly different in pitch. This problem can be distressing to the speaker and amusing to the listener.[1] Diplophonia, a relatively uncommon phenomenon, is generally regarded as a symptom of laryngeal pathology. We recently observed and intensively studied an 18-year-old girl who possessed the ability to produce diplophonia in the absence of a pathologic lesion. The objective of this paper is to discuss the differential diagnosis of diplophonia and present an analysis of the findings in this unusual case.

Diplophonia has been attributed to simultaneous vibration of the vocal cords and the ventricular bands or the tissues of the pharynx. Double voice or alternating voice is nearly always present in the early stages of dysphonia plicae ventricularis,[2] a pathologic entity due to phonation with the ventricular bands. This sometimes

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occurs when the cord has been removed because of cancer and in patients with congenitally absent or rudimentary vocal cords.[3] Upon examination of the larynx in dysphonia plicae ventricularis with accompanying diplophonia, the cords are seen to commence to vibrate and emit a short sound before they are damped by the overriding closure of the false cords, which also begin to vibrate. Jackson and Jackson[2] described two cases in which there was alternating production of sounds. The low notes were produced by phonation with the ventricular bands, whereas the high notes were produced by the true vocal cords. This type of diplophonic voice is in reality an alternation of high and low pitches producing a tremolo that may give the impression of diplophonia. Tumor and other lesions that hold the cords apart can cause the ventricular bands vicariously to assume the function of phonation. According to Lederer,[4] diplophonia may result from tubercular invasion of the false vocal cords, which brings them near the midline. Luchsinger and Arnold[5] indicate that it may occur as a result of bilateral vocal nodules dividing the glottis into anterior and posterior segments and forming it into an "hourglass shape."

The division of the cords into two separately vibrating structures has also been observed and the larynx photographed during this process by one of the authors (P.M.). Male adolescents often have difficulty controlling their voices. The loss of control is usually evidenced by instantaneous, unpredictable and involuntary changes in pitch. Sometimes double resonance or momentary diplophonia is concomitant with the loss of control of the voice.

Negus[6] attributed the alterations in the voices of adolescent males to the sudden enlargement of the larynx that occurs at puberty and specifically to the lack of proper control of the increasingly massive muscles of the larynx. The rare congenital anomaly of congenital duplication of the vocal cords is a well known cause of hoarseness and could conceivably produce diplophonia, although Frank and Malev[7] make no mention of it in their paper on double vocal cord.

Another pathologic cause of double voice is paralysis of a vocal cord. Luchsinger and Arnold[5] describe diplophonia as the "third stage" of "flutter voice," which is due to the passive flopping movements of a flaccid, paralyzed vocal cord. The diplophonia results from the different tension of the two cords.

Transient states of diplophonia are often caused by strands or chunks of mucus which collect on the vocal folds during phonation.

Two tones can be heard momentarily as a result of the asymmetrical loading of one of the vibrating edges.[1]

The case to be detailed apparently represents physiologically controlled diplophonia rather than a pathologic variety. One of Van Riper and Irwin's patients discovered "the trick of double phonation" and deliberately used it for entertainment of friends.[1] She was able to sing, using both tones, though the interval between high and low tones was fairly fixed. To our knowledge the highly controlled types of diplophonia possessed by our patients has not been previously reported. Not only is it novel, but it presents us with the opportunity to obtain some new insight into the anatomy and physiology of phonation.

CASE REPORT

This 17-year-old Negro girl began to produce a double voice at the age of six. Quickly observing that it amused her playmates, she began cultivating this talent and began singing duets around age 12.

At the time she was initially seen by us, she had a strong primary alto voice and was singing in the choir that accompanied the local symphony. Her primary concern, as well as her mother's, was whether singing in the dual voice would damage her normal singing voice. The patient had been in excellent health and had always been a "chubby" little girl. There was some question of endocrine dysfunction because of her obesity and hirsutism. This had been extensively evaluated, however, and no abnormality had been delineated, except a slightly low basal metabolism rate. She had been treated with desiccated thyroid and "medication" had been necessary to induce menses. A sibling was also learning to sing in a double voice. She lacked accurate control of her voice, however, and was not as accomplished as the patient.

Physical examination of the ears, nose and pharynx was normal. The larynx appeared normal during single voice phonation. During double voice production the false cords were normal, and both tones appeared to come from the true vocal folds. The unusual nature of the ability of the patient to produce the two tones with apparently normal vocal cords stimulated us to study her case intensively, with cinefluorography, regular and high speed photography of the larynx and analysis of sonograms of her single and double voices.

Moving pictures of the larynx confirmed the clinical impression of an anatomically normal larynx and that the two tones were pro-

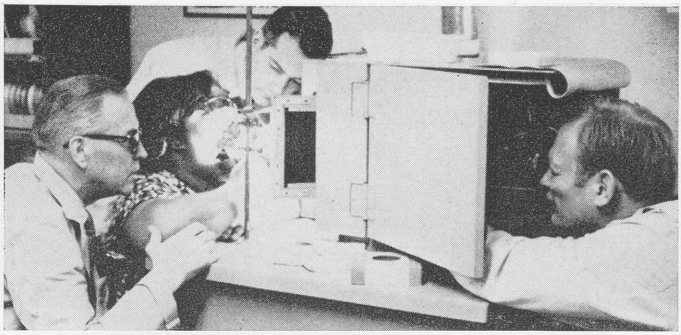

Fig. 1.—Photograph of patient in laryngeal laboratory in position for photographing the larynx.

duced by the true vocal cords (Fig. 1). High speed photography (1000 frames per second) demonstrated the asynchronous movements of the two vocal cords during the production of dual voice. The patient appeared to possess control of the degree of tension of the two vocal cords, thereby allowing the airflow through the glottis to affect them differently, producing the two tones. The tones were varied by changes in the tension of one or both vocal cords.

Cinefluorography demonstrated increase in the activity of the true vocal cords during diplophonic production. Excursion of the false cords was diminished, and indeed, there was an even wider opening between the false cords during the production of dual voice as compared to single voice. The only other noticeable difference was an increased excursion of the larynx upward or cephalad upon simultaneous emission of the two tones. Sonographic analysis of the high speed films during normal and diplophonic voice emission provided additional information. Graphic representation of the vocal fold movement, glottal width and glottal area during normal or single voice phonation is illustrated in Figure 2. The vocal fold movements under conditions of normal phonation are essentially in phase and the regularity is reflected in the curves for glottal width and glottal area. The pattern seen in this figure indicates an essentially normal voice.

An entirely different pattern is observed during diplophonic phonation. Examination of the curves representing the vocal fold movements clearly demonstrates the vocal cords vibrating at different frequencies (Fig. 3). The upper curve of vocal fold movement

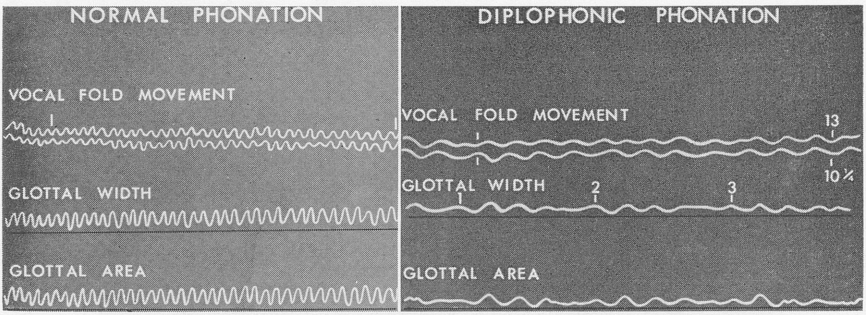

Fig. 2.—Graphic representation of the vocal fold movement, glottal width and glottal area during normal single voice phonation. The vocal fold movements under normal phonation conditions are essentially in phase and the regularity is reflected in the curves for glottal width and glottal area.

Fig. 3.—Sonographic pattern of the patient's voice during dual voice production. The curves representing the vocal fold movement clearly demonstrate the vocal cords vibrating at different frequencies. See text for further explanation.

shows a greater number of oscillations, which are also of smaller amplitude than are those of the lower curve. The combined motions create patterns of glottal width and glottal area, showing three or four well-defined instants of glottal narrowing, followed by a period of relatively little change in glottal width and area. This pattern of glottal area variation would be expected to create the auditory impression of two tones, one corresponding to the frequency of the relatively faster changes, and the other (a much lower pitched sound) corresponding to the total wave form or fundamental shown between the numbers 1 and 2 and between 2 and 3 (Fig. 3).

Analysis of the vocal fold movements in this section indicates a ratio between the curves of approximately 13 : 10¼. When two sine waves having vibrational frequencies in this ratio are combined, they produce the curves shown in Figure 4. The two sine waves are shown independently and in combination. When the glottal area measurements during diplophonic phonation (from the previous graph) are compared with the sine waves having the same ratio of frequencies, the similarities between the curves are impressive.

DISCUSSION

From very careful analysis of the various examinations, we can conclude that the patient has a normal larynx and that her vocal cords vibrate at different frequencies when she produces a double

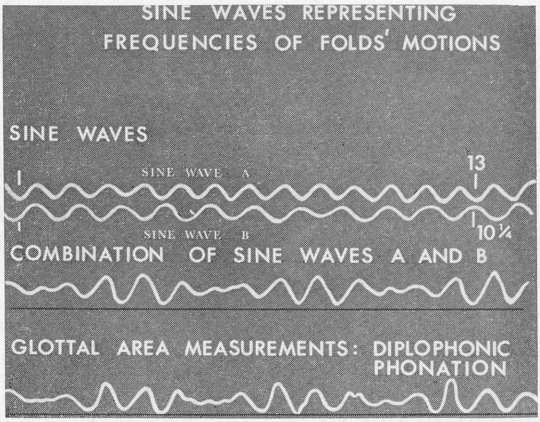

Fig. 4.—Comparison of sine waves emitted by vocal folds shows 13 to 10¼ frequency ratio. When combined and compared with glottal area measurements, the similarities between the curves are impressive.

voice. The tones are probably a perceptive phenomenon, resulting from the effects of short trains of three or four regular impulses separated from each other by brief periods of continuous airflow during which there is little or no pressure change. The trains of three or four pulses probably give the impression of the higher pitched component of the sound, and the total wave pattern creates the impression of a much lower pitch.

Since the patient can introduce the double tone at will, has significant control over the respective pitch of the separate tones, and since there appears to be no laryngeal abnormality, it can be concluded that the double voice results from differential contraction in the muscles of the vocal cords. Differential contractions would cause different degrees of elasticity in the two vocal cords. Since the air pressure against the vocal folds and the airflow through the glottis affect both folds equally, it can be assumed that the differences in frequency of vibration result from the controllable variations in elasticity that occur in one or both vocal cords.

From the analysis of the observations in this case, it appears that a much greater voluntary control of the individual intrinsic laryngeal muscles is possible, at least by some persons, than was previously considered possible. This control appeared to extend to selective, independent control of the two thyroarytenoid muscles in this particular case.

SUMMARY

Diplophonia, the simultaneous production of two different voices, is a rather unusual, infrequent disorder, usually due to laryngeal pathology.

The differential diagnosis of diplophonia includes dysphonia plicae ventricularis, the simultaneous vibration of several laryngeal and pharyngeal structures, dual voice production, secondary to bilateral vocal cord nodules, adolescent voice changes and loading of the vocal folds with strands or chunks of mucus.

An unusual case of physiologic diplophonia was presented, which was intensively studied by means of cinefluorography, regular and high speed laryngeal photography and sonographic analysis. The unusual ability of this patient selectively and independently to control the elasticity or tension of vocal cords enabled her to produce alterations in the vibratory pattern perceived by the listener as diplophonia.

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DISCUSSION

GODFREY E. ARNOLD, M.D.: Double phonation or diplophonia of this type is extremely rare. I know only a few publications in the European literature which describe this exceptional ability. The first was a presentation of a Hungarian singer before the Berlin Medical Society by T. S. Flatau (Berl. klin. Wschr. 47: No. 49, 1910). This person could sing with two voices that followed the melody with the constant interval of an octave plus a fifth, the second harmonic (12 tones apart such as c^1 and g^2). The double voice could not be produced except with almost closed mouth and it disappeared with any attempt at endoscopy or laryngoscopy.

The same singer was later studied radiologically by M. Scheier (Berl. klin. Wschr. 49: No. 18, 1912), the pioneer in radiologic phonetics. Although impressive changes in laryngeal positioning were seen during double phonation, the explanations offered by the assembly of famous voice physiologists remained speculative. The chief point was the *lowered* epiglottis during double voice, similar to ventriloquism.

A case of *triple phonation* was described by D. Weiss (Archives Néerland. Phonét. Expér. 11: 137, 1935). A member of a touring chorus, age 26, could produce two separate and independent tones by labial double whistling, employing all intervals from a second to an octave. Moreover, and this was so exceptional, he could sing (or hum) a third independent voice by laryngeal phonation. The unique performance was recorded on a phonograph that is still available from the Phonogram Archives, Vienna Academy of Sciences.

Double whistling in counterpart is an exceptional musical ability that has been observed occasionally; the phonetician Paget and his daughter could do it, performing quartets. Simultaneous *whistling and singing* has also been recorded, such as by the phonetician Moser who could perform fugues with his lips and larynx. Simultaneous *double-whistling plus singing* was reported by Weiss. *Double singing in fixed intervals* was heard in the first case above. *Double singing in counterpart,* as demonstrated by Dr. Ward, is a most unique phenomenon, however.

All these types of double or triple phonation, in unison, parallel, or counterpart, represent exceptional musical ability and neuromuscular control. These abilities presuppose extraordinary development of the cortical psychomotor areas for the voluntary control of these adjustments.

The completely new feature in Ward's case is the exploration of the double voice with modern techniques. High speed motion pictures have revealed the mechanism of a double voice. While the girl produced normal single voice, her glottis closed normally and nothing unusual was seen. Whenever we heard her double voice, however, the glottis became much wider, as one would expect in a case of severe hoarseness. I must conclude that the audible double voice is a psychoauditory phenomenon in the listener's ears. It may be produced by aerodynamic principles in this unusually wide glottis. The acoustic laws applicable to the physics of wind instruments may provide the mathematical explanation of this baffling observation.

I would advise caution regarding the different vibratory ratios or patterns of the two cords during double phonation. As far as we understand it, the vocal cords vibrate silently and do not by themselves produce sound as do elastic strings. If two vocal cords really vibrate differently, as with unilateral polyps, infiltrations, etc., marked hoarseness or pathologic diplophonia is heard. It seems inconceivable to me at present that in this double voice, one tone would emanate from the right cord and the other from the left, singing bilaterally as a pianist plays with two independent hands. I rather suspect that the second one is something like a harmonic, summation tone, or difference tone, originating within this peculiarly shaped glottis according to some psychoacoustic principle.

For the sake of deepening our knowledge of human phonation I hope that this unique case will be studied further, including the methods of acoustic phonetics, musical acoustics, psychoacoustics, cinefluorography, etc.

Finally, I hope that this charming girl will be given the opportunity of obtaining formal musical education so that she may develop her unusual musical and laryngeal talent to become a truly great artist. In our time of expanding temporal bone bank research, it also seems acceptable that a phenomenal individual of this type could be invited to bequeath the brain and larynx for eventual study in a neurologic and laryngologic institute, just as some great musicians and scientists have done in the past.

Thank you for showing us a phenomenon that I can readily hear and see, but do not comprehend.

DR. WARD (closing): This girl also has the ability to produce a third voice.

Since our interest in this one case we have had another situation brought to our attention: Apparently the lamas in Tibet learn to phonate with a dual tone and in order to become the Dalai Lama you have to be able to chant with a dual voice. However, since their plight is currently in question and they are kind of being chased out of their native land, I am afraid this talent may be lost.

UPPER AIRWAY PROBLEMS IN SEVERELY BURNED PATIENTS

GEORGE F. REED, M.D.

HERBERT L. CAMP, M.D.
(by invitation)

SYRACUSE, N. Y.

Coincident with significant improvements in the management of shock, fluid management and sepsis in the severely burned patient, respiratory tract damage has emerged as the principal killer in burned patients.[1-4] Despite the recognized nature of this aspect of the severely burned patient, and despite extensive clinical studies and laboratory research, there remains considerable confusion and disagreement regarding the assessment and management of burned patients with respiratory tract damage.

When studying the literature on this subject, one is immediately struck by the considerable disagreement regarding indications for and against tracheotomy in these patients, and the lack of knowledge and understanding relative to the role and importance of the larynx in burn injuries to the respiratory tract.

Not infrequently, the otolaryngologist may be called upon to help in the management of these patients, especially regarding tracheotomy and problems of tracheobronchial toilet. Having been faced with this challenge on a number of occasions and finding very little reference to this problem in the otolaryngologic literature, we decided to undertake a search of the general medical literature to formulate what we feel is a wise and well-founded plan of action when confronted with the responsibility of managing respiratory problems in the severely burned patient. This paper is the result of

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This study was supported in part by the National Institute of Neurological Diseases and Stroke, Grant No. TO1 NB05276.

this search, and its purpose is to assemble the important material which we have culled from the literature, to evolve a plan of treatment and to bring this information to the attention of other otolaryngologists.

The Offending Agent. To arrive at a sensible plan of assessment of the problem and management of the upper respiratory tract damage, it is important to have an understanding of what is the actual offending agent that brings about the respiratory tract pathology. At first glance, one would tend to think in terms of a "fire" or respiratory tract "burn" when faced with a patient with extensive body burns. The term "tracheobronchial burn" is used extensively in the literature. Experimental and clinical studies, however, demonstrate that damage to the respiratory tract in survivors of conflagrations is nearly always caused by the effects of smoke or "products of incomplete combustion" rather than heat.[3,5] Actual thermal burns of the respiratory tract are usually limited to the anterior portion of the nose and mouth due to very efficient heat exchange in the upper respiratory tract.[3,6] Heat sufficient to produce thermal burns of the pharynx and larynx produces acute obstructive laryngeal edema incompatible with life before actual thermal burns can be produced in the tracheobronchial tree. Even the larynx is usually spared from heat sufficient to cause laryngeal thermal burns, except in already unconscious patients since, as Taylor and Gumerick point out, "No alert human being will allow flame to reach his face if he is conscious."[7] Accordingly, our thinking in terms of damage to the laryngotracheobronchial system should be in terms of the effects of chemical irritation and destruction of tissue by smoke and chemical products of incomplete combustion rather than the effects of actual heat.

This concept has been demonstrated in animals by Moritz, Stone and co-workers.[3,6] These investigators conducted experiments with anesthetized dogs wherein the respiratory tract was exposed to oven heated air, flame and steam. They found that it was necessary to introduce the heated air, flame or steam directly to the trachea by an insulated translaryngeal cannula to produce any effect upon the tracheobronchial tree. Any of these agents applied directly to the upper respiratory tract caused immediate laryngeal and pharyngeal edema sufficient to kill the animal instantaneously before any damage could be done to the tracheobronchial tree.

Stone and his co-workers,[3] using rats, also found it necessary to use an insulated tube to bypass the larynx in order to study the

effects of direct heat on the tracheobronchial mucosa. In their investigations, they compared the variables of heat, humidity and smoke using the mortality rate during the ten days after exposure as the criteria of severity of damage. As expected, mortality was directly proportional to the amount of heat exposure. The mortality rate increased with increased humidity, but more important was the finding that exposure to smoke increased the mortality more than humidity. A heavy concentration of smoke in the inhaled air was almost as important regarding mortality as was the actual temperature to which the animal was exposed.

Pathology. Pathologic findings in Stone's animal experiments[3] demonstrated extensive destruction of bronchial mucosa, dilatation of large and small bronchi, hemorrhagic edema of the peribronchial connective tissue, generalized hyperemia and hemorrhagic edema of the peripheral bronchi and alteration in the alveolar capillary membrane.

By and large, the autopsy findings in patients dying of pulmonary "burns" conformed to the experimental findings of animals exposed to heat and smoke inhalation.[3,7-11] These consisted of intense tracheitis, bronchitis, and necrosis of the tracheobronchial epithelium with diffuse hemorrhagic and focal membranous reaction in the tracheobronchial mucosa. Pulmonary changes are manifested by one or more of the following: 1) pulmonary edema, 2) atelectasis, 3) intra-alveolar hemorrhage, and 4) disruption of the alveolar-capillary membrane. The amount of damage is, of course, dependent upon the severity and length of exposure.

Unfortunately, in the autopsy reports available in the literature, very little is reported regarding the histopathologic changes in the larynx of the patients succumbing to burns. In the few cases we were able to find, the laryngeal histopathologic changes were described as edema and necrosis of the surface epithelium overlying an intact basement membrane.[8,9,12]

Although we could not find this stated definitely, we have concluded, by inference, that in most victims of severe burns arriving at the hospital, our treatment philosophy must be directed at the pathologic changes in the entire tracheobronchial tree rather than the larynx or upper respiratory tract, since severe thermal or smoke damage to the larynx results in laryngeal obstruction and death at the scene of the conflagration.

History, Symptoms and Physical Signs. Although the presence of damage to the respiratory tract in the burn patient may be quite

obvious when first seen, this is frequently not the case. Lethal damage to the respiratory tract may have taken place with only minimal clues to direct one's attention to this area in addition to other more obvious, but possibly less lethal, burned areas of the body skin. It is of utmost importance to obtain a careful history as to the nature of the fire in which the burns were sustained. It has been shown that the greatest majority of significant respiratory tract damage in burned patients occurs when the burn was a result of flame-type injury in an enclosed space, such as a burning room or building.[2,3] This again reflects the importance of the influence of smoke and products of incomplete combustion in the respiratory tract damage. This concept is further confirmed by the fact that in the southern parts of the United States and in England respiratory tract involvement in burned patients is relatively rare.[13] This is because the burns are most often caused by the ignition of clothing by space heaters and unguarded coal fires, wherein the victim usually runs about in relatively clear air rather than inhaling the hot, smoky atmosphere of a burning room or building. Therefore, we must ascertain the situation in which the patient received his burns, not only whether the burn was incurred in a closed place, but also the nature of the combustible material contributing to the smoke in the area. Cope[5] has found that persons burned in enclosed spaces stood a 47 percent chance of suffering respiratory damage. He also noted that respiratory burns occurred twice as frequently in patients who had associated second and third degree burns of the respiratory area of the face. From his 18-year study of burn patients at the Massachusetts General Hospital, Cope found that 88 percent of the patients with pulmonary burns were found to have a combination of deep burns about the respiratory area of the face and a history of having been burned in an enclosed space.

Symptoms and signs to indicate the presence of pulmonary damage may be very obvious or very deceiving, depending upon the severity of respiratory involvement and the state of consciousness and cooperation of the victim. It is also important to realize that both symptoms and signs may be minimal on admission, only to become manifest a few hours later. The significant points of importance relative to history, symptoms and signs in patients with mild and severe respiratory tract involvement are presented in Table I.

Assessment of these findings indicates that combinations of the more severe signs and symptoms of each provide very important

TABLE I

POINTS OF IMPORTANCE IN INITIAL EVALUATION OF BURNED PATIENTS WITH RESPIRATORY INVOLVEMENT

CLINICAL ASPECTS	MILD INVOLVEMENT	SEVERE INVOLVEMENT
Historically		
Burn Agent	Flame	Flame
Environment	Enclosed	Enclosed
Presenting Complaints		
Sore Throat	Variable	Variable
Cough	Frequent	Always
Hoarseness	Infrequent	Variable
Dyspnea	Infrequent	Always
General Evaluation		
State of Consciousness	Alert	Lethargic to Comatose
Patient Cooperation	Good	Bad
General Orientation	Good	Bad
General Appearance		
Color	Normal	Cyanotic or cherry red
Respiratory Effort	Normal to slightly labored	Labored
Cough	Mild	Severe
Physical Examination		
Body Surface Burned	Usually involves respiratory area of face	Involves respiratory area of face
Nasal Vibrissae	Frequently singed	Always singed
Oropharynx	Injected	Injected
Chest	Normal to decreased breath sounds	Decreased breath sounds with rales
Ancillary Studies		
Chest X-ray	Normal	Normal or with signs of consolidation and atelectasis or pulmonary edema
Sputum	Normal	Soot flecked

prognostic implications. Cope and his co-workers have found that labored respirations, stridor or cyanosis on admission are observed in 50 percent of fatal cases. It must be remembered, however, that cyanosis may be masked by the cherry red color of blood induced by carbon monoxide poisoning. The presence of auscultative changes in the lungs on admission is also a very dire prognostic finding, since physical findings in the chest and x-ray changes in potential survivors usually do not occur until after a number of hours, or even days, following the burn. Another very important point to remember in respect to the signs of respiratory burns is that stupor and confusion are often encountered, and must be recognized as being

due to hypoxia from respiratory damage rather than being attributed to a shock or other causes.

Clinical Management. From the otolaryngologist's viewpoint, it is probably best to approach the management of airway problems by utilizing the three stages proposed by Stone,[1,3] i.e., 1) pulmonary insufficiency, 2) pulmonary edema, and 3) pneumonia.

The first and most severe stage is that of pulmonary insufficiency manifested by signs of marked airway obstruction (cyanosis, retraction, dyspnea, and diminished to absent breath sounds). It should be recognized that the prognosis is almost completely hopeless in this situation. Although many of the signs and symptoms point to upper airway or laryngeal obstruction, in most cases they are really due to pathology in the entire respiratory tract rather than the larynx. Further, if the main pathology is, indeed, in the tracheobronchial tree, tracheotomy may not only be futile but may be fatal. The barrier to ventilation which appears to be of laryngeal origin is primarily due to severe bronchospasm and/or extensive alveolar damage. In this stage of the disease process, tracheotomy should only be done if definite laryngeal obstruction can be demonstrated. In this regard, it is important to attempt an assessment of the laryngeal airway, either by direct or indirect laryngoscopy, as soon as the patient is admitted and before swelling of the head and neck or oropharynx with trismus obviates the opportunity to do so. Although there is considerable disagreement in the literature regarding tracheotomy in this stage of respiratory burns,[1,3,4,12,14-16] the general consensus of opinion appears to be that unless there is *obviously demonstrated* laryngeal obstruction, tracheotomy should be deferred due to the dire consequences, i.e., increased shock, introduction of sepsis, and premature induction of pulmonary edema. Keeping in mind the high probability of bronchospasm in this stage in the absence of definitely demonstrated laryngeal obstruction,[1,3] tracheotomy should be deferred pending the results of a large dose of corticosteroid in an attempt to break the bronchospasm. If, after steroid administration, the patient is still in respiratory distress, tracheotomy may be considered. It must be considered cautiously, however, remembering the importance of sepsis from the operative procedure and, more importantly, by recognizing the danger of premature induction of pulmonary edema as a result of lowering intrabronchial pressure.[1,3,15]

Stone has shown that in his series of 27 cases, death ensued immediately following the insertion of the tracheotomy cannula in

four patients, presumably due to decrease of intrabronchial pressure with resultant massive pulmonary edema. It must then be recognized that if a tracheotomy is performed during this stage of respiratory insufficiency, either because of definite or suspected laryngeal obstruction, a cuffed tracheotomy tube must be utilized and the patient immediately put on positive pressure ventilation. A word of caution is also necessary here to warn that most positive pressure ventilating machines are grossly contaminated and may quickly produce a fulminating pneumonia by inoculating the damaged tracheobronchial tree with bacterial organisms. Every effort should be made to maintain sterility of the ventilatory machine by gas sterilization at relatively frequent intervals. In general, it might be said that when a patient is encountered in the stage of pulmonary insufficiency, the prognosis is almost uniformly fatal and, with the exception of positively demonstrated laryngeal obstruction, tracheotomy more often does harm than good. In view of the grave prognosis and difficulty in determining the exact cause of ventilary insufficiency, the primary treatment consists of the administration of 40 percent oxygen at a 100 percent water vapor concentration via face mask or endotracheal tube.

The second stage to be expected and anticipated is that of pulmonary edema. If this is not induced by premature tracheotomy, it seldom develops before the institution of fluid therapy, usually at least eight to ten hours following the burn. The treatment of choice during this stage is that of positive pressure ventilation administered through a nasal endotracheal tube with sufficient sedation for the patient to tolerate the tube.[1,3] Again, one must remember the importance of attempts at maintaining sterility of the ventilating device. In patients with pre-existing cardiac disease, fluids should be used cautiously and administration of diuretics and/or digitalis preparations may be of benefit in controlling the tendency to pulmonary edema from the cardiac viewpoint, but these agents, in the absence of pre-existing cardiac disease, do not alter the course of pulmonary edema on the basis of the pulmonary changes resulting from the burns. If a previous tracheotomy has been done and the patient has survived to this stage of pulmonary edema, positive pressure ventilation can be administered via the cuffed tracheotomy tube. Since the time period requiring forced ventilation in this stage of the disease is relatively short, an endotracheal tube is preferred for this purpose, thus obviating the increased dangers of sepsis from the tracheotomy.

TABLE II

SUMMARY OF IMPORTANT ASPECTS OF THREE STAGES OF RESPIRATORY INVOLVEMENT IN SEVERELY BURNED PATIENTS

Stages	*Pulmonary Insufficiency*	*Pulmonary Edema*	*Pneumonia*
Time of Onset	0-8 Hour Post-Burn	8-36 Hours Post-Burn	2-6 Days Post-Burn
Symptoms	Dyspnea, sternal retractions, and decreased breath sounds	Dyspnea, frothy sputum and rales	Dyspnea, fever, and areas of pulmonary consolidation
Pathophysiology	Marked bronchospasm	Transudation of fluid into the bronchioles and alveoli	Bacterial invasion of lung parenchyma
Treatment	Intravenous steroids 40% O_2 at 100% humidification by mask	Intratracheal intubation with continuous intermittent positive pressure ventilation	Large doses of antibiotics; tracheotomy to assist tracheobronchial toilet
Prognosis	Grave 80% Mortality	Poor 65% Mortality	Fair 30% Mortality

The third stage, one which all surviving patients pass through, is that of bacterial pneumonia. This is almost always associated with massive bronchial pulmonary secretions. The onset of this stage may be as early as the second day following the burn, but is usually encountered between the fourth and seventh day. It is during this stage that tracheotomy plays its most significant role in the management of respiratory involvement in the severely burned patient. At this time, the control of the large amounts of bronchiopulmonary secretions is difficult by any other means than tracheotomy. Accordingly, tracheotomy for a short period of time is very desirable for adequate tracheobronchial toilet. It must be remembered, however, that the tracheotomy is useful for this purpose only, and that the cannula must be removed as quickly as possible to minimize further bacterial contamination associated with the tracheotomy wound and to avoid other complications of tracheotomy in the damaged tracheobronchial tree, such as tracheal erosion, tracheitis, etc. The pneumonic process itself is managed by massive doses of appropriate antibiotics. It should be pointed out that prophylactic antibiotics in the early stages of "pulmonary burns" have not been demonstrated to be of any value in preventing this stage of the disease process; they may be detrimental by changing the bacterial flora towards more antibiotic resistant organisms. The important aspects of these three stages are depicted in Table II.

SUMMARY

A review of the literature relative to the problems concerning the respiratory tract in the severely burned patient reveals a paucity of information on this subject in the otolaryngologic literature. In view of the fact that otolaryngologists may be called upon to assist in the management of the airway in these patients, the general literature has been reviewed and condensed to point out the factors of importance in the history, symptoms, signs and management of patients during the various stages of involvement with this type of injury.

This review has pointed out the extreme paucity of information relative to the role of the larynx in the severely burned patient, indicating a need for additional clinical and laboratory investigations to properly assess the importance of laryngeal changes in the management of these patients.

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DISCUSSION

THOMAS C. GALLOWAY, M.D.: So far as I know, the first important study of burn injury of the respiratory tract was by Dr. George Livingston. This was presented before the Chicago Laryngological Society but unfortunately not published. This was before the Coconut Grove report. He described three children who slept on a tenement house floor where a wave of flame swept over them.

I bronchoscoped these children within two or three hours while Dr. Livingston did tracheostomies. We found in the pharynx and trachea red thick mucosa looking like a first degree burn and not unlike that of a very acute laryngotracheo bronchitis. We ascribed the injury to a blast of flame and it did not seem that a chemical damage could be so severe.

If Dr. Reed's thermograms were taken under duplicate conditions we may conclude that it was chemical as there was not yet obstruction or infection to account for the picture. These children had also the cherry red skin and blood findings of carbon monoxide poisoning which must not be overlooked in such circumstances. This caused the death of two of these children.

I question some of the statements about tracheotomy and pulmonary edema. With respiratory obstruction as seen in tracheobronchitis and polio, for instance it seemed due to increased negative intrathoracic pressure and increased capillary permeability associated with hypoxia and increased carbon dioxide tension and possibly infection, with some cardiac insufficiency. If a tracheotomy is done early as it should be if it is to be done, it is hard for me to see any increased danger of pulmonary edema from it. I still think the edema, increased exudate, atelectasis with infection and cardiac failure are markedly increased by added obstruction that usually occurs with tracheostomy. I would treat these cases as I would a very severe laryngotracheo bronchitis.

I have seen two other cases with similar findings. In one where there was an explosion of a gas tank in an auto accident with the severe findings chiefly in the pharynx and hypopharynx.

The paper was valuable. I hope we hear more about thermograms.

JEROME A. HILGER, M.D.: I had the misfortune of having experience with burns of the upper respiratory tract in World War II. The Spitfires that saved Britain had a gas line running through the cockpit and the German streams of machine gun fire had a tracer every fifth shell and as the gas line would be ruptured the cockpit would fill with gas fumes under pressure and the tracer bullet would ignite producing a flash burn.

Flash burns are interesting. They are somewhat different from the usual burn. It was notable in these men who had to wear silk gloves to protect their hands that when they were brought in after a flash burn in the cockpit, if they had a hole in their glove they had a third degree where the hole was but they had no burn at all on the rest of their hands, showing the transient quality of the flash.

It was said in Britain in those days, and I accepted it, that no one with flash burns severe enough to cause airway obstruction survived. This we disproved, how-

ever, in Africa because there were flash burns of comparable types from other sources brought in that were so severe that when I did a tracheotomy the thyroid was cooked and there was quite a bloodless field. It was notable that the larynx was literally burned from the flash heat, also the mouth and, to a degree, the interior of the nose. Some of those patients (and perhaps unfortunately, I am not the one to judge that ethical problem) survived and so the British were disproved. One can survive a flash fire that burns the larynx sufficiently to require tracheotomy. I agree with Dr. Galloway that if one is going to do a service for them it requires rather prompt tracheotomy. These were the product of intense heat in the upper airway and were not the product of any chemical irritant with delayed action.

DANIEL C. BAKER, JR., M.D.: This problem of fires and flash burns and the like is one that is ever present. The most recent one happened in New York City at four-thirty in the afternoon when the streets were crowded with people and offices were filled. I believe the count of the individuals that died was at least ten. It all happened from flash burns from the third floor of a building right near Saks Fifth Avenue.

I mention it because one of my neighbors, an architect, age 41, died in the fire and he had his coat in his hands, presumably trying to put out a blaze when he died.

I would suggest a lot of information could probably be gained by getting information from the coroners and the people who performed the autopsies as to what injuries did take place in the laryngeal tracheobronchial tree.

DR. REED (closing): These opinions that we have put down in this paper of necessity have been culled from the literature in general and are not the basis of any direct work that we have been able to do.

I am delighted that Dr. Galloway bronchoscoped the five patients he spoke about. Almost nowhere in the literature could we find any descriptions of not only the tracheobronchial tree but the larynx. The people who handle these problems don't look at the larynx. I think this is something that needs to be done to know where we stand with some of these severe burns.

We didn't mention this in our short presentation, but the business of the cherry red color of carbon monoxide poisoning is very important. You may be looking for cyanosis and think the patient can't have any airway obstruction because he is so rosy pink from the carbon monoxide.

I still would have to stand by avoidance of the early tracheotomy. As far as we have been able to determine, especially in the dearth of information about the larynx, although the patient looks and sounds as though he had a laryngeal obstruction and this is what is causing his airway problem, the problem nearly always is diffuse chemical tracheobronchitis and destruction of the alveolar membrane (that is the important part) and the tracheotomy doesn't do any good. It not only doesn't do any good but it introduces infection and other problems, especially the loss of intrabronchial pressure with edema.

Dr. Hilger, I am afraid I can't really speak about flash burns in those old-fashioned airplanes because I don't think we carried our research into the literature as far as that primeval war. It is interesting, though, that this is a very unusual situation which, of course, would be difficult to document, and I will just have to take your word for it that those larynges were cooked and that you were able to save them with tracheotomy. Our experimental investigators couldn't do it. They had to bypass the larynx to keep from killing the animal.

One other rather interesting little comment I would like to make is the studies of the Cocoanut Grove fire, which was probably one of the worst in the country and which was very well documented by Cope and many others in Boston. There were three survivors who had received a full exposure in that fire and those survivors had no damage to the tracheobronchial tree and all three of them had done nothing more than hold moist handkerchiefs in front of their nose and mouth. If you ever do get caught in one of these horrible situations, the very simple expedient of putting a moist handkerchief in front of your nose and mouth will save you from death by pulmonary burns.

CHRONIC LARYNGEAL STENOSIS

DANIEL C. BAKER, JR., M.D.

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The subject of chronic laryngeal stenosis has become increasingly important. In this presentation, it is my purpose to give a working classification of the causes and the management of this condition. Three excellent papers on the repair of chronic laryngeal stenosis were presented at the last meeting of the American Laryngological Association by Dedo and Sooy,[1] Bryce et al.,[2] and Montgomery.[3] Much of our literature in recent years has been directed to the early and late management of injuries to the larynx and trachea. Holinger and Johnston,[4] Fitzhugh et al.,[5] Ogura and Powers,[6] and Bryce and Lawson[7] have made important contributions to this subject.

Chronic laryngeal stenosis is a disabling disorder. Not only is there the problem of communication due to an inadequate voice, but there is also the inconvenience and responsibilities of care of the tracheostomy tube. In addition, there is the psychological effect on a growing child who must mingle with other children at home, at play and at school. In the case of an adult, there is sometimes a serious mental disturbance when faced with a future of wearing a tube, and one or more surgical procedures. If all goes well with the management of the particular problem, the resultant voice is frequently far from satisfactory. In many instances the laryngologist is unable to assure the patient of more than a usable voice. Further surgical procedures to improve the voice do not always obtain their objective, and in some instances the problem can be aggravated.

The management of a patient with chronic laryngeal stenosis is dependent upon the etiology, the age of the patient, and the changes that are present at the time of observation. Another important factor

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is the experience and skill of the laryngologist in charge of the patient. The surgical treatment of this disabling disorder is dependent upon the ingenuity and creative ability of the laryngeal surgeon. No two cases of chronic laryngeal stenosis are identical. Procedures have to be modified to fit the individual problem. The objectives of the surgeon are the establishment of a good airway without the use of a tracheostomy tube and a satisfactory voice. The careful planning of the operative procedure is most important.

There are many causes of chronic laryngeal stenosis. The condition can be present at birth and may occur in more than one member of the family and probably transmitted through the genes. Baker and Savetsky[8] reported three cases of congenital partial atresia of the larynx. The condition was present at birth in the mother and in two of her offspring.

Acquired chronic laryngeal stenosis can occur at any age. In children, two long-standing causes are still with us, namely, the poorly placed tracheostomy opening and a poorly fitting tube. Failure to recognize these two factors can cause subglottic stenosis. In adults, conservation surgery for carcinoma of the larynx may be followed by stenosis of the remaining portion of the larynx. Measures must be employed to prevent possible stenosis of the anterior portion of the larynx.

Chronic granulomatous lesions present their own problems. Laryngeal tuberculosis is seldom seen today, but it is always a possible cause of stenosis. The condition can be brought under prompt control with medications. If stenosis develops after the curative phase, surgical correction is possible. Syphilis of the larynx in my present day experience is rare. I have not seen a gumma of the larynx in 20 years. This is probably due to the fact that few patients have passed through this generation without receiving penicillin or some other antibiotic for some respiratory infection or other illness. In my practice, I have seen patients with chronic laryngeal stenosis due to a granulomatous process and the diagnosis was never adequately established. If the condition is active, it may rule out the possibility of surgical repair. One granulomatous lesion that lends itself to surgery is rhinoscleroma. The disease is slowly growing, and large rounded masses which may appear in the larynx and trachea can be removed by laryngofissure with the re-establishment of the airway.

Table I lists many causes for laryngeal stenosis. The list is not complete and the measures employed for the management of the

TABLE I

ETIOLOGY OF CHRONIC LARYNGEAL STENOSIS	MANAGEMENT OF CHRONIC LARYNGEAL STENOSIS
A. Congenital	A. Congenital
1. Congenital laryngeal stridor (Abnormality of aditus)	1. Avoid external surgery unless absolutely necessary a) Tracheostomy—instruction to parents
2. Partial atresia—web +	2. Limited internal procedures a) Preserve the mucosa in the anterior commissure Incision of web—with bouginage
3. Cysts	b) Cysts best opened so as not to involve the vocal cords
4. Cystic hygroma	c) Never use cautery on the vocal cords
5. Tumors—hemangioma	d) Don't be afraid to seek advice from colleagues
B. Acquired	B. Acquired
1. Trauma a. External—fracture, dislocation contusions, ecchymosis	1. Trauma—external a. Early recognition; education of those in emergency room b. Planning the necessary emergency procedure that will ensure a good result, i.e., good laryngeal function c. In chronic stenosis following injury careful study of the problem—use of polytomography and laryngography d. Skin grafting e. Partial epiglottectomy—value in mirror laryngoscopy (postoperative)

2. Post-surgical
   a. Tracheostomy, cricothyrotomy
   b. Laryngofissure
   c. Vertical partial laryngectomy
   d. Thyroid surgery, bilateral abductor paralysis

3. Radiotherapy

4. Trauma—Internal
   (a) Intubation

   (b) Direct laryngoscopy
   Healing process—denudation of anterior commissure
   Use of cautery

   (c) Corrosive burns

2. Post-surgical
   a. Tracheostomy-cricothyrotomy (check position of tracheostomy tube); lower tube to proper position; select proper size of tube
   b. To avoid stenosis in external surgery. Laryngofissure or vertical partial laryngectomy—use of skin graft if the anterior commissure has been compromised—or McNaught keel
   c. Bilateral vocal cord paralysis—lateral fixation of vocal cord—selection of proper procedure to secure a good functional result. Experience of the surgeon

3. Radiotherapy
   Edema—corticosteroids
   Perichondritis—if rapid process, establish drainage
   Fistula—explore for necrotic cartilage
   Simulated vocal cord paralysis—dissolution arytenoid cartilages. Treatment—lateral fixation of cord

4. Trauma—Internal
   (a) Tracheostomy
   Patience—allow child to grow
   Avoid external surgery on larynx
   Remove granulation tissue

   (b) Direct laryngoscopy—web formation
   How to avoid—preserve the anterior commissure—early postoperative observation. Repeated dilatation until epithelization takes place.
   Surgical approach: McNaught keel; approach of Haslinger, Frazer, Pennington

   (c) Corrosive burns
   Children—postpone definitive surgery until growth has taken place

TABLE I (Continued)

5. Chronic Inflammatory Disease	5. Chronic Inflammatory Disease
(a) Chronic granulomatous diseases	(a) Chronic granulomatous diseases
Tuberculosis	Tuberculosis—primary, secondary
	Antibiotics, Streptomycin
	Surgery after healing has taken place
Syphilis	Syphilis—less and less tertiary ? on the increase
Rhinoscleroma	Rhinoscleroma—removal of obstructing masses by external surgery—slow progression
(b) Perichondritis	(b) Drainage. Explore for necrotic cartilage
(c) Non-Specific	(c) Radiotherapy—same dose as for infection
Progressive granulomata—after direct laryngoscopic removal of contact ulcer, or pacchyderma	
(d) Incomplete resolution or organization of changes of acute infections	(d) In infants and children—no operative interference on the larynx unless absolutely necessary. Train parents to change tracheostomy tube. Allow time to elapse. Almost 100% of children can be decanulated before the age of 4 years.
Persistent subglottic stenosis in infants and children	
Failure at decanulation	
6. Tumors	6. Tumors
(a) Benign	(a) Multiple papillomata—avoid trauma to the mucous membrane. Avoid prolonged procedures. Other tumors are managed according to the location and presenting problem
Multiple papillomata	
Chondroma	
Neurogenic tumors	
Neurilemmoma	
Neurofibroma	
Rhabdomyoma, leimyoma	
Cysts—laryngocele	Complete excision where possible
Mixed tumors	
(b) Malignant	(b) Radical surgery
Chondrosarcoma	X-ray therapy
Rhabdomyosarcoma	Combined therapy
Leiomyosarcoma	
Carcinoma	
Pseudosarcoma	

particular problem is not presented in depth. A paper could be evolved from any one of the listed conditions. Progress has been made in our knowledge and therapy of chronic laryngeal stenosis. It can be expected that further progress will be made, perhaps in the field of transplantation, not of the entire larynx, but a portion of it.

SUMMARY

A classification as to the etiology and the management of chronic laryngeal stenosis has been presented.

Experience has shown that definitive laryngeal surgery on infants and children should be given considerable thought. A conservative approach with allowance for growth of the larynx is of paramount importance.

The development of new plastic prostheses and the ingenuity and creative ability of the laryngeal surgeon has given us a fresh outlook on the management of this disabling disorder.

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DISCUSSION

Herbert Harris, M.D.: I think Dr. Baker has thoroughly covered a very broad subject and I have little comment to make.

Unfortunately, I guess I should say "unfortunately," I can't disagree with him on very many things he has mentioned. Of course, he has given a general outline of many different procedures, all of which, I am sure, he doesn't follow himself.

There are two or three areas, though, that I wish to speak specifically about. One is that I have long since learned to not dilate the larynx because I think it is too troublesome, it takes too much time and I think there are other methods that suffice better.

There was one thing I was happy to see him mention and that was skin grafting. Several years ago, I published a paper on grafting of acute injuries to prevent contraction and I am not sure that it went over very well, though we have extended our experience now into quite a number of cases and our successes are still 100 percent with stents remaining for a short period of time.

Now, there is one other thing that I wish to also mention and that is the use of acrylic molds. I try to follow surgical principles whenever I do surgery and I think all of us should. One of the first things I learned in medical school was never to place a cast over a bony prominence without it being well padded. If anyone can tell me how you can put an acrylic mold in the larynx without producing a pressure necrosis I would like to see that demonstrated.

I am not sure that I should say more because there is so much to be said about this paper that perhaps I should sit down and allow someone else to talk.

In expressing an opinion, I do so from having had the experience in a fairly large number of acute injuries of the larynx and chronic stenosis from injury.

There is one thing in closing that I do wish to say. I heartily agree with Dr. Baker in not entering the larynx of the very young as an external approach. I feel that in the cases that we have done, our results have not been good. I cannot say that the larynx didn't develop, but our successes were not that which we expected.

WALTER P. WORK, M.D.: It might be of interest to point out, particularly in laryngeal trauma, the interest that is being developed for determining how these injuries are produced. At our Highway Safety Institute at the University of Michigan, sponsored by the big three of the motor car industry, the members of this Institute have taken a special interest in laryngeal work. One of our residents, fortunately, has been tied in with this and as a sidelight to the actual injury itself, has found that in dogs if the injury is directly anterior to posterior instead of a glancing blow that these dogs often die. They die apparently from hemorrhage within the brain stem. The trauma is forceful enough to transmit energy through the cervical vertebral skull. This is an observation that may be able to be carried over into the human. We may be losing some of the patients in automobile accidents by this mechanism. We are not sure yet, but I thought I would point out this observation.

G. ARNOLD HENRY, M.D.: In traumatic cases, and I am speaking of adults, there is frequently tremendous hypertrophic overhealing, not only of the soft tissues but the arytenoids and other laryngeal cartilages.

In these cases as well as inserting a proper stent, one has frequently to remove a large portion of this hypertrophic tissue. One may remove one or even both arytenoids when they are grossly swollen and semi-immobile. Like the lung fish who has a primitive larynx without cartilage, a muscle nerve segment too weak to move following injury, may now function reasonably well.

Last December I was asked to admit a patient, 60 years of age, because of shortness of breath. Eighteen months previously he had 6000 rads of radiotherapy for cancer of the epiglottis. After performing a tracheotomy, liberal pieces of hypertrophic granular tissue were removed from the arytenoid and pharyngeal wall area. The epiglottis was clean. To our surprise, this tissue came back labelled necrosis following radiotherapy.

A week later with this in mind, I removed a great deal more of this hypertrophic tissue around the arytenoid and laryngeal glottis. This came back negative for cancer as well.

I saw the patient two weeks ago. One vocal cord did not move but this had been true before his irradiation. The other cord had almost normal range of movement and the larynx had resumed almost a normal appearance.

I think we have to remember that sometimes tremendously hypertrophied tissues are more than a weakened or even an intact muscle nerve segment can move.

ARTHUR J. CRACOVANER, M.D.: I want to make a comment on one statement, that of "careful planning." I find in cases of traumatic stenosis, in spite of the laryngograms and pharyngograms and direct laryngoscopy, it is difficult to anticipate what you are going to find when you open the larynx. Frequently, when you do open it, you have to improvise, you have to make up your mind what you are going to do at that time and I find the thing that has helped me most in this improvisation is knowing the history and development of the treatment of chronic stenosis. If you go way back and follow the course and the various methods of treating laryngeal stenosis, you can come up with an idea at operation that is appropriate, so I think that "careful planning" is difficult in these patients.

JOSEPH H. OGURA, M.D.: I would like to limit the remarks to traumatic stenosis of the larynx in the chronic phase.

I believe that most of the difficulties that you have here can be with relationship to how do you rehabilitate this in the very severe form?

Now, those which are at the glottic and supraglottic areas are, in my hands, one of the most difficult to anticipate where the pathology is, so how far can you get down to the larynx and be able to resect it and rehabilitate it?

Quite commonly the supraglottic lesions are involved with the pharyngeal injury in combination and so the resection must entail a horizontal resection. Skin grafts in this type, of course, will not work.

Likewise, in the subglottic forms it has been my experience that the direct surgical approach by a thyrotracheal anastomosis has been the best. Now, some of these failed but the failure rate based on a single operative procedure with a subglottic resection is small, compared to high failure rate with skin grafts and repeated dilatation.

DR. BAKER (closing): To Dr. Harris. I agree with him that dilatation in chronic laryngeal stenosis is of little value. It is also a complete waste of time. But I do emphasize the point that we are faced with, namely, that when we do a direct laryngoscopy for removal of a benign anterior commissive lesion, the patient may or may not come back to you. It could be your own case or it could be from another laryngologist and the patient presents himself with a web, with a worse voice than that he started out with. There should be something that one might do to prevent such a web formation and I feel that the way to do this is to observe your patient early after a direct laryngoscopy and if healing is satisfactory, leave the patient alone. If there is any question about possible web formation, it is necessary to keep those areas separated or else the patient will develop a web and then you will really be in trouble.

THE USE OF DOUBLE TRANSVERSE INCISIONS IN MAJOR HEAD AND NECK SURGERY

J. R. Chandler, M.D.

Virgil A. Ponzoli, M.D.
(by invitation)

Miami, Fla.

While no one incision is suitable for all types of major head and neck surgery,[1] the incision used must fulfill several criteria. Adequate exposure must be provided in order to permit visualization of all vital structures and to allow adequate resection of involved tissues. They should be planned in order to allow any necessary shifts of skin and other structures for closure and primary reconstruction procedures when some skin must be sacrificed or used for mucosal surfacing. Maximal blood supply to all flaps should be preserved in order to minimize the likelihood of flap necrosis. They should be arranged in an optimal fashion in order to provide maximal protection to the underlying vital structures, viz., the carotid artery, its bifurcation, the internal carotid artery and the external carotid artery and all of its major branches. Possible linear scar contractures should not interfere with motion of the head and neck; and finally, they must be cosmetically acceptable.

With the increasing use of pre-operative radiation therapy, either as a planned combined procedure or as frequently occurs in many patients in whom salvage is attempted following radiation failure, avoidance of flap necrosis and maximal protection for the carotid artery and its branches has assumed great importance.[2]

For more than 12 years we have found that the modified "Y" type incision with a serpentine vertical limb as described by Fraser[3]

From the Division of Otolaryngology, Department of Surgery, University of Miami School of Medicine, Miami, Florida.

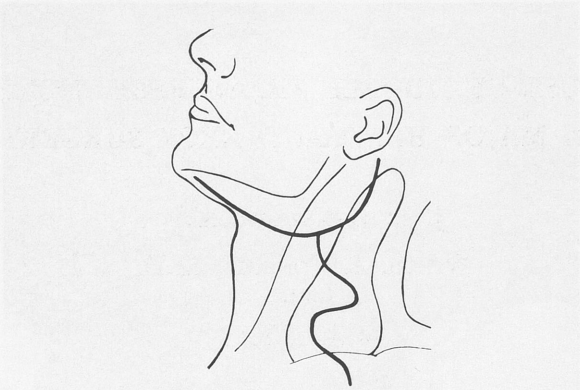

Fig. 1.—Fraser incision. The serpentine curve of the vertical incision minimizes unsightly and disabling linear scar contractures.

has proven very satisfactory (Fig. 1). The extension of the inferior limb for several centimeters below the midpoint of the clavicle provides excellent exposure to the posterior triangle and good access to the opposite side of the neck as necessary for a composite wide field laryngectomy. With increasing amounts of planned radiation therapy being given even to those patients in whom primary reliance is placed upon surgical excision, the increased incidence of postoperative complications has been evident (Fig. 2). Because of several instances of carotid artery exposure, necrosis, rupture, hemorrhage and exsanguination, the use of pedicled muscle flaps, dermal grafts, lateral displacement of the carotid artery, and more recently, fascia lata grafts have been used. In spite of this, flap necroses, secondary breakdown of tissues, infection, and dissolution of tissue, particularly with infection due to Pseudomonas aeruginosa, have resulted in increasing concern for protection of the carotid artery and the exploration of all possible avenues to decrease this formidable hazard. A vertical midline incision has been strongly advocated for a variety of good reasons[4] but seems contraindicated on the basis of probable very limited exposure to the lateral areas of the neck and because of the almost inevitable objectionable scar contracture.

While the use of parallel transverse incisions for neck dissection was originally proposed solely for its cosmetic advantages it was noted that better wound healing was also a feature.[5] More recently they have been used in a small series of previously irradiated patients in order to avoid the complications noted above.[6]

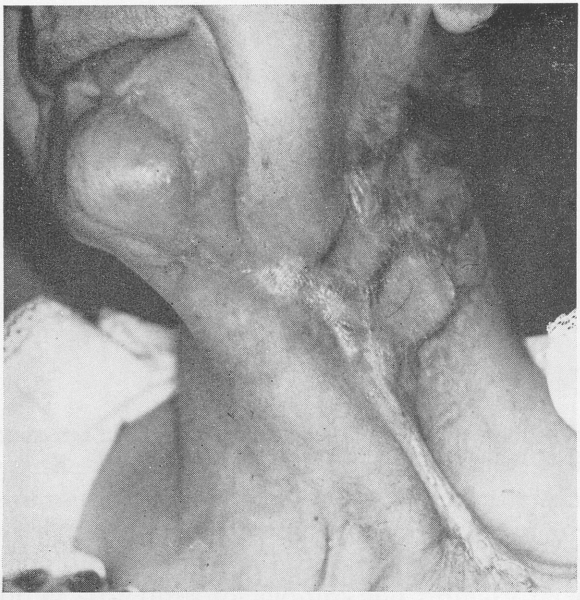

Fig. 2.—This patient received 6000 rads prior to a composite neck and jaw resection. A Fraser incision was utilized. Flap necrosis, infection and scar contraction resulted in this cosmetically objectionable and functionally disabling result.

It was decided that the double transverse neck incisions should be used in a series of selected patients in order to evaluate its possible advantages and disadvantages. In a 15 month period from October, 1967 to January, 1969, the double transverse incisions were used for a selected series of patients requiring radical neck dissections, either alone or in combination with a laryngectomy or other composite resection of various primary lesions. A total of 31 such patients operated upon on the private and staff services at Jackson Memorial Hospital and the Veterans Administration Hospital in Miami are included in this study. In addition, double transverse incisions were used for a variety of other procedures including three laryngectomies and two laryngectomies with bilateral neck dissections. These latter cases are not included in this report. In order to make some valid judgment as to the advantages and disadvantages of the double transverse incisions, a number of parameters were analyzed by comparing them with an unselected group of 31 similar patients operated upon in the period just prior to the study.

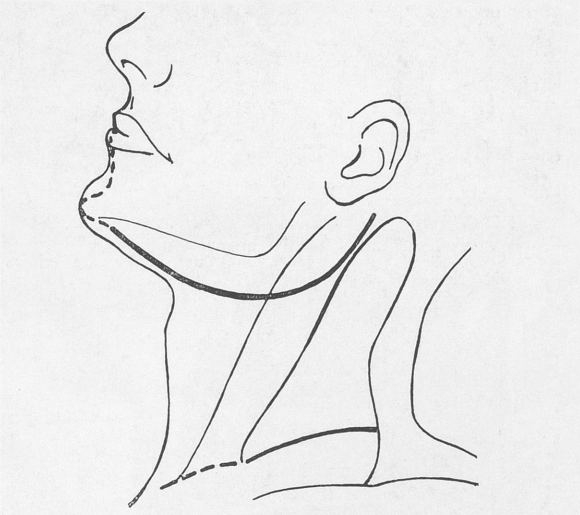

Fig. 3.—Double transverse incision. Dotted lines show optional extension through the upper lip or into the suprasternal notch.

PROCEDURE

The arrangement of the incisions and operative technique was essentially the same for all of the patients in this group. The upper incision passes two cms beneath and parallel to the mandible from the midpoint of the chin to the mastoid tip. In composite resections of the mandible and other intraoral structures, it may be carried through the lower lip if desired. The lower incision is shorter and is approximately eight cms long. It is placed so that it is two cms above and parallel to the clavicle beginning about one cm anterior to the trapezius muscle and extending to just above the head of the clavicle anteriorly. In composite resections which include the larynx, the inferior incision extends into the suprasternal notch where an ellipse of skin is removed for the stoma. Superior and inferior skin flaps are elevated and the bipedicled central skin flap including platysma muscle is also developed from the trapezius muscle to the strap muscles or even well beyond them for a laryngectomy (Fig. 3).

Dissection begins uniformly in the posterior triangle at the level of the clavicle and the entire posterior triangle and lower portion of the anterior triangle dissected up to well beneath the bipedicled skin flap. The contents of the neck, including the larynx in appropriate

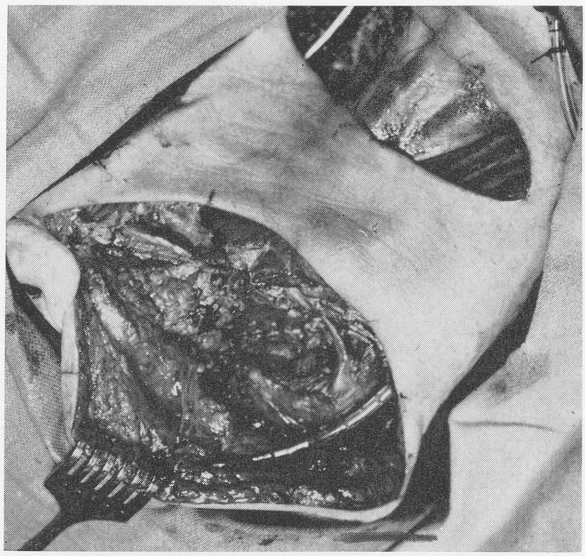

Fig. 4.—The completed dissection with suction tubes in place. The patient's chin is to the left and at the top of the picture.

instances, are then passed beneath the bipedicled skin flap and brought into the upper skin incision. Following this, the operative procedure is then completed, and includes in all instances dissection of the submental triangle as well as the inferior portion of the parotid gland and the entire contents of the submaxillary triangle (Fig. 4).

It should be noted that as originally described by MacFee, the "neck dissection" did not include the internal jugular vein or the sternocleidomastoid muscle.[5]

CASE MATERIAL

It can be seen that the two groups are quite comparable in most respects (Table I). The various surgical procedures performed in the two groups are also quite similar (Table II). While a consideration of the possible advantages of combined irradiation and surgery in the control of certain malignant neoplasms of the head and neck is not the subject of this report, the relationship of pre-operative radiation therapy to the frequency and severity of postoperative complications requires that this factor be considered. In the study

TABLE I

CASE MATERIAL

	Average Age	*Color*	*Sex*	*Site of Primary Lesion*
Study Group N=31	60	29 White 2 Black	24 Male 7 Female	Larynx 14 Tongue 2 Tonsil 3 Pharynx 7 Other 6
Control Group N=31	57	29 White 2 Black	22 Male 9 Female	Larynx 13 Tongue 5 Tonsil 3 Pharynx 4 Other 6

TABLE II

SURGICAL PROCEDURES PERFORMED

	Radical Neck Dissection Only	*Radical Neck Dissection And Total Laryngectomy*	*Radical Neck Dissection And Other Composite Resections*
Study Group N=31	9/31=29%	14/31=45%	8/31=26%
Control Group N=31	8/31=26%	17/31=55%	6/31=19%

TABLE III

PATIENTS RECEIVING PRIOR IRRADIATION THERAPY

	Planned Combined Low Dose (less than 3500 Rads)	*Planned Combined High Dose* (more than 4000 Rads)	*Curative* (more than 5000 Rads)	*None*
Study Group N=31	6/31=19%	9/31=29%	13/31=42%	3/31=10%
Control Group	4/31=13%	6/31=19%	14/31=45%	7/31=23%

group only three patients had not received pre-operative radiation therapy (Table III). This contrasts significantly with the control group, operated upon in the period immediately preceding this study, and which contained seven patients who had received no radiation therapy. While a controlled study to evaluate the effect

of pre-operative administration of low as compared to high doses of ionizing radiation in the control of the primary lesion is underway in this institution, there was consderable variation in the number of rads given to patients prior to surgery. It can be seen that the majority of patients operated upon were treated with radiation therapy for cure and were operated upon for their residual primary lesion or metastases in an attempt at salvage. A lesser number of patients received either low doses varying from a minimum of 2000 rads to 3500 rads or a high dose amounting to more than 4000 rads up to as much as 6000 rads in some instances. The former group of patients were operated upon one to two weeks following completion of therapy while the latter group were operated upon four to six weeks later. Those having received "curative" amounts were, of course, operated upon at varying intervals following completion of their therapy.

Some attempt was made to quantify the anticipated slightly increased difficulty in elevating the flaps and securing exposure by evaluating the time required for surgery in the two groups. A possible influence on the amount of blood loss was also made by comparing the amounts of blood replaced in the operating room. It had been our initial impression during the course of this study that there was increased difficulty in elevating the middle flap to its proper level and extent in order to secure adequate exposure. It was also our feeling that even with proper preparation of the flaps that the exposure in certain areas was somewhat less than optimum. This was particularly true beneath the anterior and posterior aspects of the middle flap and in conducting the dissection of the opposite side of the larynx. In some individuals with long necks, even though thin, the increased width of the flap was annoying. On occasions, the control of irksome bleeding from the undersurface of the middle flap was difficult, whether at its midpoint or its lateral or medial extent. It was further felt that unwise and too extensive retraction on the ends of the bipedicled middle flap with retractors or particularly with Penrose drains or gauze sponges, in some instances may have resulted in some trauma to the flap. As a general rule, patients with thin necks were relatively easy to operate upon even though the bipedicled flap was a bit wider. Thick and short necked men were somewhat more easy to operate upon in this regard because of better exposure and a more narrow middle flap.

In all instances the ratio of length to width of less than 1.5 to 1 for the bipedicled central flap resulted in a strong highly vascular

TABLE IV

AVERAGE OPERATING TIME AND BLOOD REPLACEMENT

		Hours Operating Time	*Units of Blood Administered in Operating Room*
Study Group N=31	RND Only	4.8	3.0
	RND & Laryngectomy	7.6	4.0
	RND & Other Composite Resection	7.7	3.7
Control Group N=31	RND Only	5.4	2.7
	RND & Laryngectomy	8.7	5.6
	RND & Other Composite Resection	7.7	6.2

integumentary covering for mucosal suture lines and the carotid artery.

Two relatively short transverse incisions provided excellent access to the larynx for isolated laryngectomies and healed rapidly and well in the three cases in which it was employed. The lower incision is directly through the site of the future stoma and the shorter superior incision is at the level of the hyoid bone. Such incisions are easily adaptable to the subsequent use of double transverse lateral neck incisions in the event that later neck dissection is necessary.

In each instance of simultaneous bilateral neck dissection and laryngectomy, there was extensive necrosis of skin and subcutaneous tissues in the midline. Though both patients had received previous radiation therapy, it seemed that the length of the flap from trapezius to trapezius and mastoid to mastoid across the midline, does not provide sufficient blood supply to maintain the integrity of its central portion. Split thickness skin grafts were necessary for final healing in each patient. For such cases, the wide superiorly based apron flap seems much more practical. Such an incision extends from mastoid to mastoid, curving inferiorly in a wide U to the level of the stoma. This flap provides excellent exposure and has great vitality.[7]

It was found that there was no significant difference in the operating time nor in the amount of blood replaced during surgery (Table IV); in fact, it seemed that contrary to our expectations, the

TABLE V

POSTOPERATIVE COMPLICATIONS

	Infection	*Wound Dehiscence*	*Fistula*	*Carotid Artery Hemorrhage*	*Mortality*
Study Group N=31	9/31=29%	9/31=29%	12/31=38% 12/22 Composite =55%	1/31=3%	3=9%
Control Group	13/31=42%	12/31=38%	12/31=38% 12/24 Composite =50%	1/31=3%	2=6%

operating time and blood replacement was slightly less in the study group. Thus, our observations seem to be at variance with those of MacFee, who feels that "performance of neck dissection through transverse incisions is more difficult."[8]

RESULTS AND DISCUSSION

The five postoperative complications with which we are concerned are tabulated in Table V. While it was difficult to separate in most instances wound infections from wound dehiscences and some subsequent fistula formations, an attempt was made to do so. For a period of approximately nine months during this evaluation, a concurrent study was in progress which dealt with the usefulness of pre- and postoperative antibiotic therapy with intensive intravenous antibiotic therapy being given in the operating room as compared to no postoperative antibiotic therapy. It was found that the incidence of infection in the latter group was significantly higher so that this study was discontinued. It was our feeling that this possibly accounted for the significant difference in wound infections in the two groups. There were wound infections in 42 percent of the control group, which included the antibiotic period alluded to as compared to a significantly lower rate of wound infection of 29 percent in the study group. It was not felt that the incisions themselves were responsible for the decrease of the wound infection rate. There were less wound disruptions in the study group with only nine of 31 patients developing any degree of wound dehiscence. In the control group, which included those not receiving antibiotics in the postoperative period in some instances, the wound dehiscence rate was 38 percent. It is difficult to be certain about this point, but it is our impression that the double transverse incisions do tend to insure

the integrity of the skin flaps better than other incisions previously employed.

In the study group, 12 of 22 patients in whom a composite resection was carried out, developed fistulas of varying size. This compared to 12 of 24 patients undergoing composite resections in the control group. It can be seen that these are approximately the same. In the patients with double transverse incisions, evidence of beginning fistula formation made itself known with the usual signs of fever and some redness or tenderness along either the upper or lower incision, or occasionally beneath the central portion of the bipedicled flap. Such signs call for the removal of several sutures and the establishment of immediate drainage. In several instances, adequate drainage was established through the upper incision. In most instances, the medial aspect of the bipedicled flap became elevated by saliva and infected material which dissected its way beneath the flap so that drainage was required through the inferior incision. Following this, the upper incision usually healed quite promptly, except in those patients with loss of tissue and permanent fistula formation. Counter drainage, which may be established through a stab wound, may avoid this problem. In one patient, who received only 2000 rads of ortho-voltage radiation, Pseudomonas infection resulted in a very rapid sloughing of most of the central portion of the bipedicled flap and exposure of the carotid artery. A large fistula resulted, though with intensive nursing care and local attention, the carotid artery which had been otherwise unprotected, granulated over. This patient is undergoing reconstructive surgery at the present time. It was also felt this patient's crippling emphysema and marked tissue hypoxia contributed to his skin necrosis, infection and persistent large fistula.

One patient in each group developed bleeding from the carotid artery. Both patients had the common carotid artery and its bifurcation protected with a large cuff of levator scapulae muscle. In each instance, infection of the wound occurred and necrosis of the overlying muscle and carotid artery were found at autopsy. The patient in the study group survived with ligation of the common carotid artery at which time it was found that the muscle had become necrotic along with a small pinpoint area of the internal carotid artery just above the bifurcation. In the control group, one woman developed infection and fistula formation with exposed necrotic muscle overlying the carotid bulb and just as preparations were being made to debride the area, and probably ligate the artery, she expired with massive bleeding from rupture of the artery. After

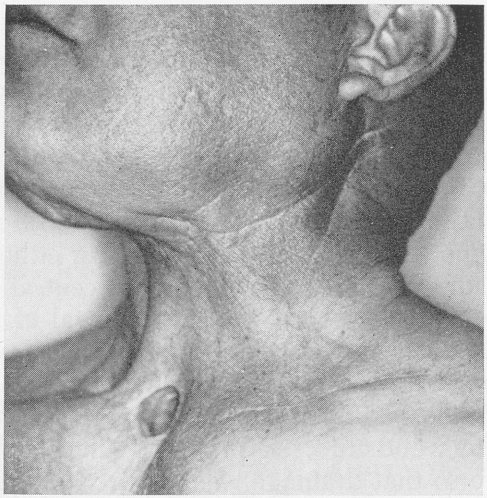

Fig. 5.—One year following second neck dissection. A right neck dissection and laryngectomy following 6000 rads radiation therapy had been performed three months earlier. Note short previously thick neck and near absence of visible scars.

these two incidents, which followed closely, we abandoned the use of the levator muscle protective cuff, evaluated the dermal graft briefly and are now using fascia lata grafts in all patients requiring composite resections who have received previous radiation therapy.

Three patients in the study group died as a result of their surgery. One woman died seven days following radical neck dissection and composite resection of a large infiltrative alveolar ridge tumor requiring mandibular resection and a large forehead flap. Post mortem examination failed to reveal any definite cause of death and it was felt that she died as a result of a probable myocardial arrhythmia.

Another patient in the study group died one month following surgery as a result of pneumonia and respiratory failure. This patient was operated upon for residual metastatic disease in his neck with a hypopharyngeal primary lesion apparently controlled by radiation therapy. At the time of surgery, extensive infiltrative neoplastic disease was found in his neck and the vagus and phrenic nerves were sacrificed. Marginal pulmonary reserve and this additional handicap resulted in his demise.

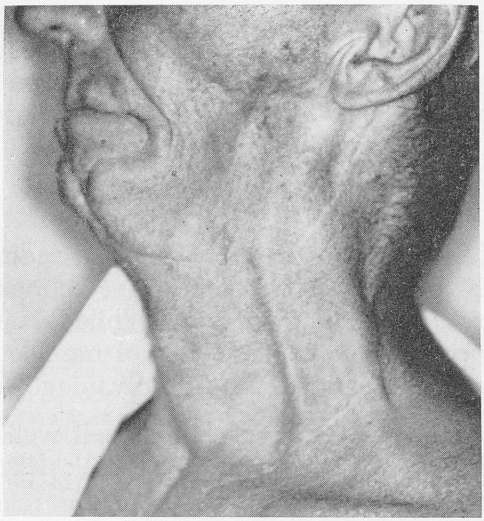

Fig. 6.—One year following salvage surgery for a large residual lesion of the lower alveolus and buccal mucosa. A large area of cheek skin was also sacrificed in a through-and-through excision of the primary lesion.

Another 56-year-old man in the study group expired two days following surgery as a result of probable anoxia and myocardial arrhythmia secondary to severe emphysema and decreased pulmonary ventilation. This patient also had extensive residual metastatic disease in his neck following radiation therapy for a pyriform sinus primary. Unresectable tumor was found, although during the attempted resection, the vagus and phrenic nerves were likewise resected and this was felt to contribute to his death. In none of these three patients was there any infection, dehiscence, fistula formation or other local complication at the time of death. On the other hand, the two deaths in the control group were a direct result of complications in the neck. One of these patients expired as the result of bleeding from the carotid artery as described above and another died from bleeding from the superior thyroid artery one month after a composite resection, extensive wound infection, necrosis and a large fistula formation.

This suggests that two of 31 patients in the control group died directly as a result of local complications secondary to the surgical procedure, while none of the three deaths in the study group could be attributed to such causes.

With the exceptions noted above, the cosmetic appearance of the neck was vastly superior in all instances (Figs. 5 and 6). Late contracture of the denervated platysma muscle occurred in some instances but seemed to be less than with conventional incisions. No limitation of motion of the neck as a result of scar contracture was noted.

CONCLUSIONS AND SUMMARY

The use of double transverse incisions for radical neck dissections alone and with a variety of composite resections has been evaluated in a selected group of 31 patients. While this is a small series, it has been compared to a similar group of unselected consecutive patients, and its use, advantages and disadvantages discussed.

The double transverse incision lends itself well to most extensive surgical procedures requiring radical neck dissections with or without resection of the primary lesion.

It is not applicable to those patients requiring a simultaneous bilateral neck dissection nor in those with large metastatic deposits in the upper posterior triangle of the neck.

It does seem to offer some security to the carotid artery though other additional means for its protection in previously irradiated patients seems indicated and is recommended in composite resections.

While dissection beneath the bipedicled flap is a bit more tedious and the control of some bleeding annoying, exposure is adequate and sufficient and closure is facilitated.

The cosmetic and functional results of this incision are superior to all others.

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DISCUSSION

JOHN F. DALY, M.D.: I have had the opportunity of observing Dr. William Mc Fee develop this approach for neck dissections at the Manhattan V.A. Hospital where he has been Chief of Surgery for the past ten years. This approach was worked out by the group who worked under Dr. Simkin at the New York Skin and Cancer Hospital in the early twenties. It is an old technique and its modern version lends itself very well to the use of the Bakamjian flap, i.e., the delto-pectoral flap for the reconstruction of the pharynx. The Bakamjian delto-pectoral flap is an inverted tube flap with the epithelium turned in on itself to form the lining of the tube. The tube is then slipped under the transverse flap through the lower incision of the double transverse incisions and brought into place in the pharynx and sutured to the upper end. The flap can be brought up as high as the nasopharynx which gives the surgeon great latitude in resecting the pharynx.

The transverse flap between the two incisions acts to cover and support the inverted tube flap.

Dr. McFee's approach to a neck dissection was designed not only for better cosmetic results, but also to conserve such structures as the sterno-mastoid, the jugular vein and the accessory nerve. I realize that there is a definite difference of opinion as to whether one should preserve these structures. Dr. McFee's results were good. The flap is a very useful one.

THOMAS C. GALLOWAY, M.D.: I would like to know why a double flap? Why not three or four? When treating a large bronchial cyst, this gives a most desirable cosmetic effect.

DR. CHANDLER (closing): Dr. Daly pointed out one important use for the double transverse incision is with the Bakamjian flap.

Some little practice with the technique of a formal radical neck dissection through this type of incision, I think, is helpful to someone attempting the Bakamjian flap for the first time. It does introduce some technical problems. Residents don't like to use it for the first several times because it does not offer the exposure that the traditional Fraser or the other classic incisions do.

In answer to the other question about the multiple incisions, we evaluated the double transverse incision primarily from a protective standpoint. We wanted a strong covering for the carotid artery. That was our objective and I think if we had more incision lines we would obviate that great advantage.

I can't emphasize too much that I wouldn't depend entirely upon the transverse skin incision, although it certainly is nice to cover up the carotid artery. Even when a fistula is present at least the carotid artery is covered with the strong, vital flap and healing is much more prompt than when the vertical line breaks open and exposes the carotid artery. Certainly one feels a good deal more comfortable not looking at the carotid artery every day as the dressing is changed.

THE ZENKER'S (HYPOPHARYNGEAL) DIVERTICULUM

PAUL H. HOLINGER, M.D.

JOYCE A. SCHILD, M.D.
(by invitation)

CHICAGO, ILL.

A Zenker's diverticulum is a well known and well documented clinical entity. Many forms of therapy have been described; one type of surgical treatment, the endoscopic diverticulotomy, with an evaluation of over 100 cases reported by Dohlman,[1] and the report of a smaller series by Holinger and Johnston in 1962,[2] has invariably led to controversy. A re-evaluation of this and other procedures in our total experience of 217 cases is presented herein.

SYMPTOMS AND FINDINGS

In this series there were 146 males and 71 females, the youngest being 34 years of age, the oldest 91. There were seven in the 30 to 40 year age group, 30 between 40 and 50 years of age, 43 between 50 and 60 years, 69 between 60 and 70 years, 49 between 70 and 80, 17 between 80 and 90 and two over 90 years of age. There was no essential difference in age distribution between men and women (Table I).

From the Department of Otolaryngology, University of Illinois College of Medicine, and Presbyterian-St. Luke's Hospital, Chicago.

TABLE I

AGE DISTRIBUTION
Males 146, Females 71

Decade	*Number*
30 - 40	7
40 - 50	30
50 - 60	43
60 - 70	69
70 - 80	49
80 - 90	17
Over 90	2
	217

The patient complaints referable to a Zenker's diverticulum were described as 1) a choking sensation associated with food sticking in the throat, 2) difficulty in swallowing, noisy swallowing or gurgling on swallowing, 3) hoarseness, 4) lodgement of food and subsequent regurgitation particularly at night, 5) "throaty saliva," a feeling of something left over after swallowing, 6) a constant desire to clear the throat, and 7) coughing spells. Occasionally patients gave a history of repeated foreign body episodes with a final episode of a large bolus of meat or a bone that could not be dislodged and required endoscopic removal. A significant loss of weight of as much as 35 lbs was noted in 42 cases or approximately 20 percent of patients.

Examination of the neck by palpation sometimes disclosed a smooth, soft swelling under the left sternomastoid muscle, close to the midline, which on pressure gave rise to a bubbling sensation, followed by an immediate cough. On mirror examination of the larynx, the pyriform sinuses were usually filled with frothy saliva which, particularly in the older patients, flowed back and forth over the posterior commissure and arytenoids.

The lateral x-ray of the neck sometimes demonstrated an air pocket behind and below the level of the larynx, often with a fluid level (Fig. 1). The barium contrast film was classic with the round, smooth mass of varying size from a tiny protrusion posteriorly from

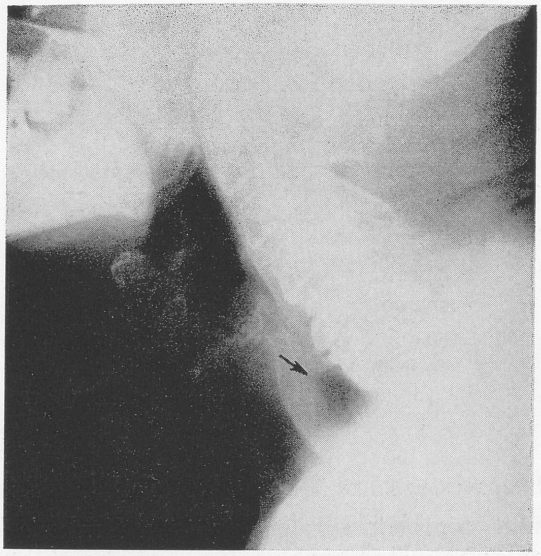

Fig. 1.—Fluid level in an air pocket in the retrotracheal area which proved to be a Zenker's diverticulum.

the hypopharynx just above the cricopharyngeus, to a pouch of 5 or 6 cm across and as much as 7 to 8 cm deep, with its upper border having a typical fluid level. In the largest sacs, an indentation produced by the aortic arch was evident. In one of the patients in this series, a midline raphe appeared to divide the sac. Esophagoscopy was only needed occasionally to confirm the diagnosis since the x-rays, showing the barium with its straight upper border and round lower contour at the level of the clavicle or just below it, were pathognomonic. However, a tight benign or malignant stricture of the cervical esophagus may, on rare occasions, simulate a Zenker's diverticulum. If the patient had had hemoptysis, a complicating carcinoma was considered in association with the diverticulum, and esophagoscopy was indicated.

It is of interest that in 17 cases or eight percent of the series, other esophageal disease than the Zenker's diverticulum was found. There were seven with hiatal hernias, three with traction diverticula in addition to the pulsion diverticulum, two with esophageal varices, two with achalasia and one with a Dysphagia Lusoria. Two patients had carcinoma of the esophagus in addition to their Zenker's diverticula. Sixteen of the patients had had one previous operation else-

TABLE II
THERAPY

Esophagoscopy only	18
2 stage diverticulectomy	2
Inversion and suspension	13
Single stage diverticulectomy	108
Diverticulotomy	37
Surgery contraindicated or died before surgery	11
Refused surgery	28
	217

where for their diverticula, three had had two previous operations and two had had three previous operations.

Pre-operatively, pathologic starvation was noted in extreme cases. Loss of weight up to 35 lbs, general debility and dehydration constituted the findings caused by inability to swallow solid and liquid food. In two of the patients, one 78 and one 91 years of age, in whom no surgery for the diverticulum was attempted, the patients' terminal condition when first seen was due to severe esophageal obstruction, and they died in spite of supportive measures. In two, only a gastrostomy was performed in hopes of preparing them for a diverticulectomy, but they did not respond sufficiently to accomplish this corrective procedure. Four patients had putrid lung abscesses due to aspiration of food and fluid, of whom one died before definitive treatment of the diverticulum could be done. A number had suppurative bronchitis, bronchiectasis, aspiration pneumonia and cardiac decompensation, requiring extensive medical management prior to the diverticulum operation. Six of these were considered incapable of withstanding surgery. In 12 cases, foreign bodies such as meat, bones, grapes, pins or a tooth pick became lodged in the sac and, following removal, studies led to the establishment of the diagnosis of the diverticulum; in two, the presence of the diverticulum had been known prior to the foreign body incident.

PROCEDURE

The management of a Zenker's diverticulum is surgical. However, of our own series of 217 patients, surgery was contraindicated in 11, and 28, or 12.9 percent refused surgery or were seen in consultation only (Table II). In 18 patients the only operative procedure

consisted of an esophagoscopy. This was admittedly not a definitive corrective procedure, except in six patients who had developed varying degrees of postoperative stenosis of the cervical esophagus following diverticulectomy elsewhere, requiring esophagoscopic dilatation of the stenosis, and in one patient who was referred because of a complete esophageal atresia following a diverticulectomy. Of the remaining 11 patients the esophagoscopy was done in six for the removal of foreign bodies lodged in the sac, some of whom subsequently had a more definitive operation elsewhere, and five who simply refused further surgery on being assured their dysphagia was not due to a malignancy.

METHOD

Probably the most conservative of the multiple types of surgical procedures is the inversion and suspension of the sac. This was done in 13, or 6.4 percent of the patients. When done with esophagoscopic assistance, the procedure was as follows: Under endotracheal anesthesia an incision was made anterior or posterior to the sternomastoid muscle to expose the esophagus, and the sac was identified by inserting an esophagoscope into it. After the sac was isolated and freed of adherent fibrous tissue, it was implanted into the wound, high up on the outer edge of the sternomastoid muscle, and the neck was closed with a soft rubber drain left in place.

A two stage operation, strongly advocated by Lahey,[3] was used only twice in this series. It consisted of resection of the sac 14 days following the identification and inversion, as described above, which constituted the first stage; after two weeks it was considered that the mediastinum would be sealed off. After the neck of the sac was again exposed, a forceps was placed across its esophageal mouth, and the sac was incised proximal to the forceps, suturing the esophageal wall with fine interrupted silk sutures.

A single stage diverticulectomy with esophagoscopic assistance was the operation most frequently used in this series and was performed on 108, or 50 percent of the patients. The technique was as follows: General anesthesia was most satisfactory since the procedure might be prolonged and the position to be maintained was uncomfortable for the patient. Intratracheal intubation insured an adequate airway whether or not the esophagoscope was used to assist in locating the sac in the retroesophageal space or to prevent stricture formation through too tight a closure of the esophagus. The choice of anesthetic agents depended upon individual preference, with nitrous oxide or pentathol and a muscle relaxant most commonly used. Since the sac lies to the left of the midline in almost every case, the incision was usually made on the left side along either the anterior or posterior border of the sternomastoid muscle from the level of the superior border of the thyroid cartilage to the insertion of the sternomastoid muscle. The platysma was cut and the anterior border of the sternomastoid muscle retracted laterally or medially to reveal the omohyoid muscle. This muscle may be divided at the tendon between the anterior and posterior bellies or, if the incision was made along the posterior border of the sternomastoid muscle, retracted medially to elevate the thyroid gland and the vessels leading to it. If the incision was made along the anterior border of the sternomastoid muscle, the lateral veins of the thyroid gland were ligated and the gland retracted medially to expose the inferior thyroid artery which, if necessary, may be double ligated and cut. Retraction now of the sternomastoid muscle and the contents of the carotid sheath laterally, and the thyroid gland and sternothyroid muscle medially, exposed the site of the diverticulum. The more direct approach along the posterior border of the sternomastoid muscle has been favored by some otolaryngologists for years and was again described by Cornell and Swan.[4] At this point the surgeon may be assisted in locating the sac by the insertion of an esophagoscope directly into the sac. The esophagoscopist can direct the sac laterally and bring it into the surgical field, demonstrating its distal extent as well as

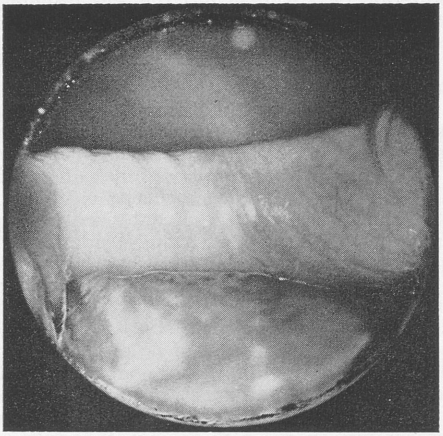

Fig. 2.—The cricopharyngeus as it is engaged in the slot of a Dohlman endoscope.

the neck at its attachment to the esophagus. The neck lies at the level of the cricoid cartilage and the sac can be seen descending posterior to the esophagus. The esophagoscope may be passed over a previously swallowed thread so that after the sac is identified, isolated and freed of its attaching fibrous tissues, the esophagoscope may be removed from the sac and more easily advanced into the subdiverticular esophagus if the string guide is firmly enough anchored in the intestine that tension can be applied to it.

Isolation of the sac and freeing it of the fibrous tissue strands that often cause it to adhere to the posterior wall of the esophagus is one of the most important steps of the operation. Every effort must be made to avoid tearing the sac, and yet the dissection must be carried up to the mucosal neck of the sac. The sac is resected from the esophagus, in the manner described for the second stage of the two stage operation, by placing a forceps on the edge of the neck and incising proximal to the forceps, suturing the esophageal wall with through-and-through fine silk interrupted sutures as the excision proceeds. Excision of the sac must be complete so that no pouch will be left to initiate the development of a recurrence; but excision must not narrow the esophageal lumen.[5] A second row of sutures may cover the first; with the esophagoscope in the esophagus it is impossible to make a closure which will constrict the esophagus. However, this complication must be borne in mind and prevented by bringing together only the edges of the cricopharyngeal and esophageal muscle fibers in the second suture line.

After resection of the sac the esophagoscopist inserts a feeding tube into the esophagus and removes the esophagoscope. The wound is closed leaving a soft rubber drain in place for three or four days, avoiding contact of the drain with the suture line. Feedings are continued by tube for one week and then the patient is given sips of liquids. After three days mechanically soft foods may be added, and only after a number of weeks is solid food permitted.

In 1958, Dohlman[1] re-described an endoscopic method of treating pulsion diverticula, whereby the cricopharyngeus muscle was cauterized and divided in the midline through a specially designed speculum. In nearly 100 of his cases operated by this method, no deaths occurred and no severe complications were encountered. Recurrence rate or the radiologic demonstration of a residual sac is comparatively high, but the ease of re-operation endoscopically is in sharp contrast to the difficulty of a second external approach through the scar of a previous diverticulectomy. The design of the

endoscope allows easy insertion; an anterior lip lifts the larynx and passes into the esophagus and a posterior beak slides into the sac bringing the cricopharyngeus muscle clearly into view across the central portion of the visual field (Fig. 2). The endoscope is inserted under general anesthesia with the airway maintained with an intratracheal tube. An alligator forceps with a sufficiently insulated shaft is passed through the endoscope, and the dividing wall, the cricopharyngeus, is grasped firmly between the blades in the midline for a distance of 2.5 to 3 cm. The active pole of a cautery current is attached to a post on the forceps and the indifferent pole is attached to a similar post on the handle of the endoscope. A strong coagulating current is applied until the tissue in the grasp is visibly cauterized. The forceps is then removed and a small spoon-like protecting insulator is inserted into the sac behind the dessicated tissue. Under direct vision, the cauterized portion of the pouch is then incised from anterior to posterior with a cutting blade using a mixed cutting and coagulating current. The blade cuts against the protecting insulator, dividing the partially coagulated cricopharyngeus in the midline. Bleeding at the cut ends of the tissue is minimal and is easily controlled by gelfoam which may be placed in the edges of the severed cricopharyngeus to complete the operation. The patient is maintained on intravenous fluids for 48 hours and if no residual discomfort is noted at that time, clear liquids are allowed by mouth. In 24 hours the diet is increased to include mechanically soft foods for another 48 hours, after which time a modified soft diet is permitted as tolerated. The patient is usually ready to leave the hospital swallowing easily and without discomfort on the fifth to seventh postoperative day. A general diet is permitted two weeks after the operation.[6]

In this comprehensive series, 37 patients or 17 percent were operated using the Dohlman technique.

POSTOPERATIVE COMPLICATIONS

The complication most feared in the surgical management of the Zenker's diverticulum is mediastinitis due to leakage. Meticulous care in avoiding spillage, accuracy in regard to suture placement, and adequate drainage are the points of importance in avoiding this complication. Should mediastinitis develop following either the external or endoscopic operation, prompt and thorough drainage is indicated. Four patients operated elsewhere were admitted with mediastinitis following perforation of the sac during an attempted esophagoscopy; in addition, two were seen with mediastinitis and esophagocutaneous fistula apparently due to leakage of an attempted diverticulectomy. All but one recovered, although the postoperative hospitalization of each was long and required repeated cervical or thoracic surgical procedures in addition to a gastrostomy.

Two patients developed a mediastinitis following diverticulotomies, one following a vomiting episode the evening of the operation, and the second in whom the cautery was admittedly extended too deeply; both recovered following immediate external mediastinal drainage and subsequent diverticulectomy. A third developed a mediastinitis due to rough, inaccurate intubation for anesthesia which was recognized by intrapharyngeal bleeding and cervical emphysema before the speculum was inserted; a mediastinotomy was performed at once to provide adequate drainage. The diverticulum was subsequently inverted and suspended two months later.

Left recurrent laryngeal nerve paralysis occurred in six diverticulectomies with spontaneous recovery in three. In two additional cases paralysis had been present following a previous diverticulectomy.

The development of an esophageal stenosis following diverticulectomy occurs not infrequently because of attempts to suture the opening at the neck of the sac in several layers. As mentioned above, this complication can be avoided if the suturing is done with an esophagoscope in the esophagus. In the six patients in whom strictures had occurred elsewhere, dysphagia was moderate or severe, and postoperative x-ray studies indicated the nature of the complication. Esophagoscopic or retrograde dilatations became necessary and were repeated until a normal lumen was re-established. In four patients in this series, one to three dilatations were necessary because of demonstrable stenosis.

There were nine deaths associated with the diverticula in this series; five patients described above had had no surgical corrective procedures other than palliative gastrostomies in two; three of the 108 died following diverticulectomies, one postoperatively due to congestive heart failure, diabetes and uremia, one of bronchopneumonia and pulmonary emboli, and one of extensive gastrointestinal bleeding from gastric and esophageal ulcers; and one of the six with mediastinitis following perforation elsewhere during attempted removal of a foreign body in the presence of an unknown Zenker's diverticulum.

Recurrences. As mentioned above, 21 of the patients in this series had had one or more previous operations elsewhere for Zenker's diverticula. Nineteen of our own patients developed recurrences which first became apparent as long as 26 years later. The recurrence rate, or more appropriately the demonstration of a residual sac, was highest following the endoscopic diverticulotomy. But it must be pointed out that while patients with these residual portions of the sac following the endoscopic diverticulotomy experience hesitation in swallowing some types of food, the sac does not become large enough to obstruct swallowing. Furthermore, it no longer overflows into the larynx and trachea but empties easily into the esophagus during a second swallow.

SUMMARY AND CONCLUSIONS

In a series of 217 patients with Zenker's (hypopharyngeal) diverticulum, there were 146 males and 71 females ranging in age

from 34 to 91 years of age with the largest number in the 60 to 70 year age group. The most serious associated findings were esophageal obstruction, foreign bodies, loss of weight and the bronchopulmonary pathology of aspiration, such as pneumonia and lung abscess due to overflow. The diagnosis was readily established by the esophagram. However, failure to demonstrate the sac, prior to attempted foreign body removal or leakage from incomplete diverticulectomy elsewhere, had resulted in the severe complications of mediastinitis and esophagocutaneous or esophagopleural fistula in six patients, one of whom died. They were first seen for management of mediastinitis, and the diverticula were demonstrated on subsequent study. Seventeen had other esophageal pathology in addition to the Zenker's diverticulum.

It is accepted that management of a Zenker's diverticulum is surgical; yet, of the 217 patients, surgery was contraindicated in 11 for medical reasons or they died of their disease before surgery could be attempted. Twenty-eight procrastinated and postponed or refused treatment. Twenty-one had had one to three operations previously for their diverticula. A variety of surgical procedures were used; in 18 patients the only operative procedure was esophagoscopy for various reasons: seven because postoperative stenosis or atresia of the cervical esophagus, following diverticulectomy elsewhere, required only esophagoscopy, and 11 refused further surgery for the diverticula, six of whom had had foreign bodies lodged in the sac.

Complications in association with this condition were numerous. Three of the 108 diverticulectomies died following surgery, one 68, one 78 and one 84 years of age. Two of the diverticulotomies developed mediastinitis, one following vomiting the evening of the operative day, and one in whom the cautery admittedly extended too deeply. Both recovered and later had diverticulectomies. Left recurrent laryngeal nerve paralysis occurred in six patients, three temporary and three permanent. Postoperative stenoses were seen in four (in addition to the seven operated elsewhere). The four of our own cases required one to three dilatations of the cervical esophagus. Twenty-one patients, or 10 percent of the series, had had one or more previous operations for their Zenker's diverticulum, and 19 of our own patients developed symptoms of recurrence in up to 26 years. Recurrence rate was highest following diverticulotomy, but the sac remained small, did not cause obstructive symptoms and emptied readily without overflow into the larynx. For these reasons

we now reserve the diverticulotomy for the older, severely debilitated patients and the more definitive diverticulectomy for younger patients.

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COMBINED EXTERNAL AND ENDOSCOPIC HYPOPHARYNGEAL DIVERTICULECTOMY

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Lawrence J. McStravog, M.D.

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(by invitation)

In hypopharyngeal diverticula, first described by Ludlow in 1767,[1] the evolution in surgical management began in 1830 when Bell[2] proposed emptying the diverticulum of its contents through a fistula. Niehans[3] is credited with first excising a diverticulum in 1884 on a patient with a concomitant goiter. Various methods of surgical treatment followed including invagination of the diverticulum and suturing of the esophageal walls, diverticulopexy, two-stage diverticulectomy and one-stage diverticulectomy.[4]

The first intimation of the combined approach was in 1896 when Professor Joseph Hearn[5] operated upon an almost completely obstructed sea captain who had refused an operation seven years previously; a large acorn bougie was passed by mouth after exposing the diverticulum-bearing area in the neck. This aided in identifying the sac which was the recipient of the bougie. The earliest record of the combined external and endoscopic treatment of hypopharyngeal diverticula was in 1915 by Gaub and Jackson[6] who operated upon two cases. The largest series was that of Shallow and Clerf.[7]

In the combined procedure the diverticulum can be more easily identified after passing the esophagoscope into the fundus. This is helpful in small diverticula where the walls blend subtly into the esophageal musculature. The pouch can be protruded into the neck

wound facilitating exposure. Passage of the esophagoscope into the subdiverticular esophagus helps locate the exact junction of the diverticulum and the esophageal wall so that excision of the sac will not be followed by esophageal stenosis.

The operation can be performed under local anesthesia, but general anesthesia is usually employed. Even in high risk patients it has been accomplished with minimal minor complications. The operation is essentially the same as the prevailing hypopharyngeal diverticulectomy with endoscopic assistance.

The type of incision is largely one of individual choice. Of the three types, 1) the typical goiter with the end of the incision turned slightly upward on the operative side to aid the approach to the pharyngoesophageal junction, 2) the transverse incision in the skin folds over the area of the diverticulum, and 3) the incision paralleling the anterior border of the sternomastoid muscle, I prefer the latter particularly in the larger diverticula. In this incision the cosmetic result is only slightly less desirable and several advantages have been found. One is not certain pre-operatively whether the dissection will be easy or difficult, and an enlarged deep portion of the thyroid gland may overlay the sac increasing the difficulties. When large and extending into the mediastinum, inflammation of the fundus may cause adherence to the pleura, predisposing to injury, as the entire sac is delivered externally (Fig. 1). Through an incision along the anterior border of the sternomastoid muscle these conditions can be more easily handled.

The side of the neck incision has received a great deal of attention; however, the approach can be made from either side since the diverticulum arises in the midline. The left side is usually selected since large diverticula tend to protrude slightly to the left in the depth of the neck. Another consideration is the fact that the recurrent laryngeal nerve can be identified somewhat easier on the left side.

Prior to operation a diagnostic esophagoscopy is performed to confirm the diagnosis of pulsion diverticulum of the hypopharynx and to rule out carcinoma. Cases of carcinoma in the fundus of the sac or in the cervical esophagus with a secondary diverticulum have occurred and one of the authors has observed one case of each. A lesser advantage to pre-operative esophagoscopy is cleansing the sac of its retained fluid and debris so that there will be less contamination of the operative field at the time of surgery. The esophag-

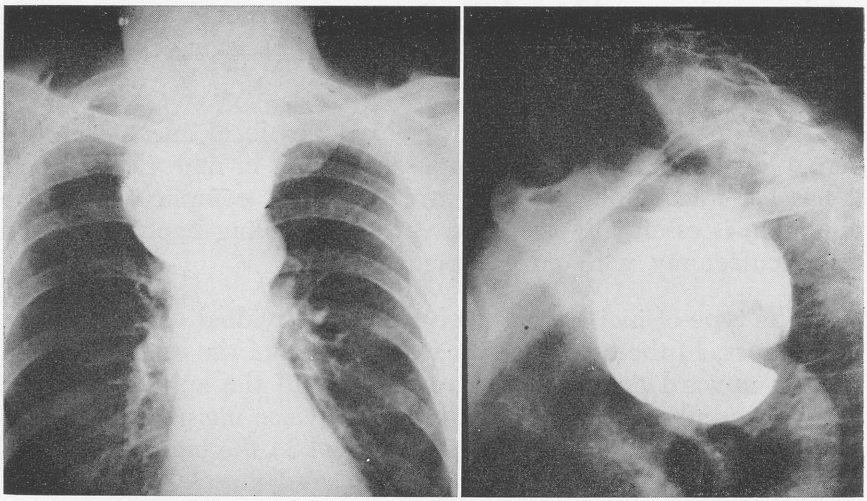

Fig. 1.—Large hypopharyngeal diverticulum extending into the mediastinum below the arch of the aorta.

oscope readily enters the diverticulum but only in rare cases can the opening to the esophagus be visualized. Twenty-four hours prior to surgical treatment the patient swallows a thread which becomes anchored in the gastrointestinal tract, and this enables the endoscopist to locate the esophageal opening at the time of operation.

After the external skin incision is made and the platysma muscle incised, the omohyoid muscle may obscure adequate exposure of the sac, and if so, it can be divided and resutured at the end of the operation if desired. The inferior thyroid artery is encountered in the lower portion of the wound and it is advisable to determine the position of the recurrent laryngeal nerve. Following the neck incision the endoscopist passes the esophagoscope into the sac itself, empties it of any retained material and manipulates the sac into the neck wound where the fundus is grasped by the operator. If desired the sac can be transilluminated from within for better identification. The esophagoscope is then removed and dissection of the muscle fibers of the cricopharyngeus from the sac down to the mucosa is begun. It is essential to free all of the muscle fibers from the neck of the sac and since it originates posteriorly directly in the midline at the junction of the pharynx and esophagus, it may be difficult to remove the fibers on the side opposite that exposed through the neck. As the muscle is separated from the mucosa, a plexus of veins

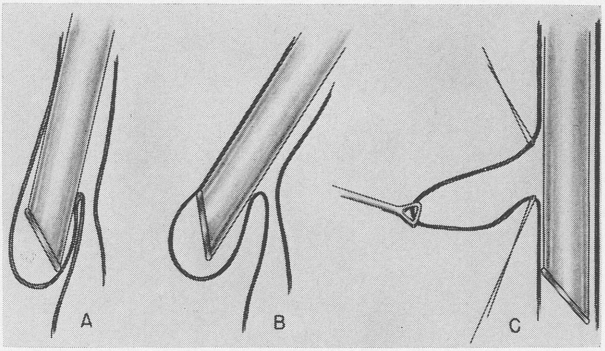

Fig. 2.—A) Esophagoscope introduced into the sac; B) Projection of the diverticulum outwards for easier manipulation; C) Esophagoscope in the esophageal lumen with temporary sutures marking the junction of the pouch and hypopharynx.

is encountered which commonly hinders complete visualization of the operative site. Unless these fibers are completely separated from the mucosa recurrence may be promoted. Dissection is particularly difficult in a patient with a short neck and can be complicated by adherence to the cricopharyngeal muscle, the mediastinum or the pleura. At times the sac may become so densely adherent to the esophageal wall that it appears to have fused with it. After stripping the muscle down to the mucosa, the esophagoscope is reintroduced and passed over the previously swallowed thread into the esophagus. The junction of the pouch and the esophageal wall is marked superiorly and inferiorly by temporary sutures. The sutures insure that the line of excision will be at the proper site (Fig. 2). The esophagoscope is then withdrawn so the surgeon can better deliver the diverticulum into the wound.

In removing the sac neck, a cuff of approximately one-quarter inch is left so that the opening will remain tightly closed when sutured. Closure of the mucosal stump with an inversion suture in which the wound edges are brought together as the neck of the sac is incised has been helpful in large diverticula (Fig. 3). The risk of contamination from spillage into the wound has not been disturbing. After the mucosa has been sutured the remaining cricopharyngeus is brought together over the primary mucosal closure. In recent years some evidence has accumulated to suggest that these fibers should not be resutured, since a hyperplastic cricopharyngeal muscle might be the predisposing cause for the diverticulum and lead to a recurrence.

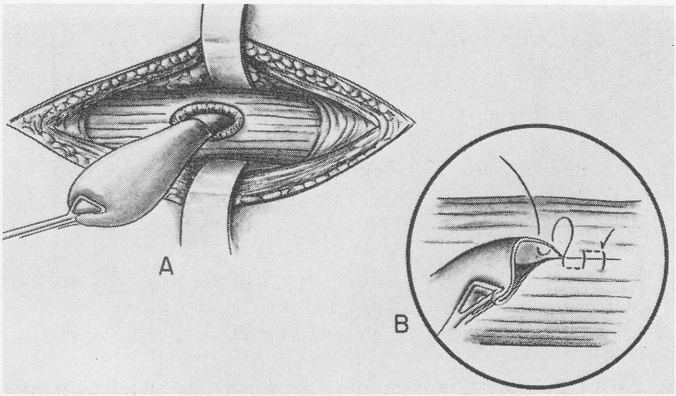

Fig. 3.—A) Cricopharyngeal muscle fibers dissected from neck of the sac with line of projected incision, leaving a cuff of one-quarter inch for closure; B) (*inset*) Inverting suture used for the mucosal closure.

The wound is closed after introduction of a drain, which is removed in 48 hours. At the end of the operation a feeding tube may be inserted and left in place for approximately one week. Two weeks postoperatively a swallowing function is performed, but no further esophagoscopy is done unless this study reveals an abnormality.

Of the complications, paralysis of the recurrent laryngeal nerve occurred temporarily in two cases, with full recovery later.

A pharyngocutaneous fistula may predispose to recurrence; this occured in one case in which the pharynx was inadvertently perforated at the time of passage of the esophagoscope. The tear was repaired but evidently the pharyngeal wall was weakened sufficiently to establish a fistula which closed spontaneously after approximately three weeks. Recurrence of a symptomatic diverticulum occurred in two years, requiring a second operation.

SUMMARY

The combined external and endoscopic hypopharyngeal diverticulectomy has the following advantages: 1) easier identification and manipulation of the sac, and 2) accurate localization of the junction of the diverticulum with the pharyngeal wall, thus minimizing the risk of stenosis.

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DISCUSSION

CHARLES M. NORRIS, M.D.: I would like to say just a word about one maneuver that we have found quite useful in the external operation as an alternative to using the esophagoscope. One of the problems in the esophagoscopically-guided operation is introduction of the scope into the subdiverticular esophagus, even with the patient under general anesthesia, and as an alternative we have found that it is useful to use a catheter of large caliber (about a 36 French) introduced over the previously swallowed string. This is cut with a rather long bevel at the lower end, and, with the degree of relaxation of the cricopharyngeus that it achieved with general anesthesia, it is usually quite easy to advance the catheter, with the tip of the bevel anteriorly, over the string and down through the cricopharyngeus into the esophagus where it is left during the remainder of the procedure.

This maneuver is helpful in three ways: (1) it prevents excessive removal of the wall of the hypopharynx, which might conceivably result in stenosis, (2) as compared to the esophagoscopic technique, it allows for a greater mobility, making it possible to draw the diverticulum further out into the wound to delineate the neck of the sac and transect it, and (3) in closure, access to the posterior wall of the hypopharynx is facilitated. A feeding tube is inserted through the catheter at the end of the operation.

If introduction of the catheter alone is difficult, another maneuver is used. The Jackson Plummer dilator, with a flexible metal tip, to which is added a small segment of rubber catheter as a safety measure, is advanced over the string and through the cricopharyngeus. The rubber catheter is then passed over this and the Jackson Plummer dilator removed, leaving the catheter in place.

DOUGLAS P. BRYCE, M.D.: I was very interested in Dr. Holinger's comments with regard to the Dohlman procedure. We have been fortunate in Toronto to have Dr. Dohlman with us and so a few years ago, when we started to gain some experience in this particular method, we had the advantage of having the master with us to show us his method.

The Swedes are a rather casual race, and would treat these patients as out patients. Dr. Dohlman does the procedure under local anesthetic and isn't concerned in any way whatsoever about mediastinitis nor does he use antibiotics routinely.

There are just two questions I wish to ask:

It is, I think, Dr. Dohlman's feeling and certainly ours that very often the cautery of the common wall must be carried out perhaps twice, maybe even three times, before a functional result has been obtained. I wondered how Dr. Holinger feels about this and whether he advises his patients that this may have to be done.

You see, the advantage of the Dohlman procedure is that it is a very simple, rapid, five-minute affair and a patient doesn't object to coming back for that sort of procedure once or twice.

The other thing was the anesthetic. Dr. Dohlman originally used a local anesthetic, and does so now. I have never quite had the courage to do this, but I wonder if Dr. Holinger uses a local anesthetic.

The third question is with regard to the cricopharyngeal muscle. I believe that in recurrence of large pharyngeal diverticuli the problem of hypertrophy of the cricopharyngeal muscle is a real one. I wonder if he has used myotomy of this muscle during his procedure in those large diverticuli which so frequently tend to recur.

HERBERT HARRIS, M.D.: I want to congratulate Dr. Holinger for having collected, as far as I know, the second largest series in the United States. Not having reviewed the literature in several years, I suspect that Dr. Lahey has the largest number of cases.

I think that Dr. Lathrop might bring us up to date on the most recent figures. At the time of Dr. Lahey's retirement he had something less than 400 cases and it is interesting to note that he would not allow a one-stage procedure to be done, at least this was my conclusion in his last report. I am sure all that are done at the Lahey Clinic now are probably done by the one-stage method.

I have not had the opportunity to have tried any diverticulotomies. It seems to me much easier and probably safer to do the procedure by the external route. We have had no real problem with the ones we have done. Certainly using the esophagoscope, when I started doing these cases a number of years ago was an advantage because I could find the diverticulum easier. Today I can find the diverticulum without this aid.

There is one thing that has been brought out, the use of an indwelling tube which to me is valuable. Now, I use a large feeding tube and insert it before doing the diverticulectomy and this gives me some idea as to how much esophagus I am removing. I can use my finders and feel the tube and leave an adequate amount of esophagus thus preventing strictures. We have had no problems nor has it been necessary to dilate the area post surgical.

Now, referring to the literature, I believe I printed an article ten or twelve years ago reviewing all of the different methods. One of them might be called the invagination method. I have only tried to do this once and it failed. I think it failed because I did not bring the muscle together well over the mucosa.

When you go back and study the two-stage method and study the results of Dr. Lahey, you will find that his mortality rate was, I believe, less than one percent, whereas prior to that time the mortality rate was as high as 20 percent with the one-stage method, but this was in the days before the antibiotics. Now we are in a new era and we can't compare the two.

Dr. Putney, the technique you have presented in your film is certainly similar to that which I have advocated.

ARTHUR J. CRACOVANER, M.D.: I have one comment I would like to make and that is the use of the feeding tube. It seems to me that a feeding tube acts as an irritant in the esophagus, especially along the suture line. There is a foreign body in the throat and the patient will usually want to swallow, will usually try to push that tube aside and, of course, the saliva goes into the esophagus as well.

If you did without a feeding tube for four or five days and asked the patient not to swallow but to expectorate the saliva that is in his throat, you put the esophagus at rest and you will be less likely to have a fistula. Of course, the patient is fed intravenously.

FRANCIS E. LEJEUNE, M.D.: I would like to report a little procedure that has been very helpful to us in doing the external operation.

At times it is difficult to find the diverticulum and dissect it out freely, so we have adopted this type of procedure.

An esophagoscope is introduced through the mouth and the diverticulum is aspirated and cleansed thoroughly. A gauze sponge is opened, extended, soaked thoroughly in merthiolate, squeezed out, and introduced and packed into the diverticulum. Prior to introducing the pack into the diverticulum, a black silk thread is attached to one end of the gauze sponge and brought out of the mouth so the pack can be removed at the appropriate time. This pack then serves several purposes, the main one being that it gives some body to the diverticulum sac, making it easier to palpate and establish its definite location, thereby making dissection easier. If there is any virtue to merthiolate, it will help in the sterilization of the tissues involved. When dissection is completed and prior to incising the neck of the diverticulum, the pack is removed through the mouth by pulling on the black thread. Following this, a #38 or 40 Hurst mercury bougie is introduced into the esophagus and this remains in place until the diverticulum is excised and sutured. With this technique, there is little or no chance of removing too much esophageal mucosa, thereby creating an esophageal stenosis. I have had to take care of several such stenoses created by the general surgeons who placed a bit too much tension on the diverticulum when incising the neck of the sac. Today, when the public has so much recourse to legal procedures, it behooves all to use all precautions to prevent postoperative complications.

W. Franklin Keim, M.D.: In the younger individuals with this condition there isn't much of a problem really in excising the diverticulum, but when a very elderly person comes in who has had angulation for a long period of time there may be even complete obstruction which may have lasted for four days to a week. These people have been dehydrated, their electrolytes have been out of kilter, very often their proteins are down and, as you all know, they are very poor risks. I would like to ask, how do you handle this type of problem in order to get the patient into condition for either the surgery or the diverticulotomy?

William M. Trible, M.D.: Since 1961, when Dr. Dohlman was in Washington, we have used the Dohlman procedure intermittently, doing about 15 cases, and some have been very, very poor risks. The most recent had been obstructed for a week with a temperature of 104, a man in his eighties. All the results have been very good with the Dohlman procedure and the simplicity is so much greater and the risk, I think, so minimal as to give this technique a high priority.

I really got up here to make one remark, a little off the subject:

The Dohlman instruments work very well with the webs which follow laryngectomy occasionally, if you have an anterior pouch and a severe web. We have done this in three patients, using the fulgeration of the Dohlman instrument. Since you have an anterior pouch rather than a posterior pouch, you usually can't slip the instrument in as well, but this works adequately and the swallowing of these post-laryngectomy patients with a pouch and web, is greatly improved.

Dr. Holinger (closing): Dr. Norris, with regard to the string, we have found that before pentothal anesthesia with relaxants, we had considerable difficulty in finding the subdiverticular esophagus. Since the advent of this combination, there has been no trouble at all and we have abandoned the use of the string, but I can imagine that there are instances in which this would be very helpful.

Dr. Bryce mentioned the Dohlman procedure and implied that treatment might be considered almost an outpatient operation! I think we are all a little hesitant to consider it as such and I never have. In regard to the anesthetic that we use, I repeat, it has been usually pentothal and a muscle relaxant. The procedures had to be repeated in a number of patients, and I mentioned this in the statistics. Professor Dohlman has stressed that it can be repeated, but nevertheless we have not found that patients are particularly willing or enthusiastic to have it done twice. Possibly we are less taciturn than the Swedes, but nevertheless our patients ordinarily do not like to have to come back for a second operation although sometimes they do; but they also have had to come back following some of the other operations for

TABLE I

SARCOMA OF THE NASAL CAVITY AND PARANASAL SINUSES (52 Cases)

Incidence Memorial Hospital	*Cases*
Cancer of the Nasal Cavity and Paranasal Sinuses	514
Sarcoma of the Nasal Cavity and Paranasal Sinuses	52
Incidence 52/514 cases – 10%	

the other hand, had unrelated nasal polyposis or surgical sinus pathology in 15 percent of cases. One case of chondrosarcoma in a 19-year-old male had a Caldwell-Luc procedure 12 years previously for fibrous dysplasia of the affected antrum.

Age. The age span varied from four years to 81 years, with the median age being 55 years. The youngest patient (a four-year-old male) presented with a myxosarcoma of the nasal cavity.

Sex. Seven of the 12 cases of chondrosarcoma and soft part sarcoma occurred in females. On the other hand, 22 (55%) of the 52 lymphosarcomas occurred in males (Table II).

TABLE II

SARCOMA OF THE NASAL CAVITY AND PARANASAL SINUSES

Median Age: 55 years	*Sex*		
Lymphosarcoma	Male	22 cases	(55%)
	Female	18 cases	(45%)
Soft Part and other Sarcomas	Male	5 cases	(42%)
	Female	7 cases	(58%)

Site of Origin. Anatomically, the nasal cavity and paranasal sinuses are intimately and complexly related with each other and adjacent structures. When sarcoma arises in this region it is prone to overgrowth of its natural boundaries at the time of diagnosis, and it is rarely confined to a single set of air cells. Thus, an accurate determination of the site of origin is difficult and sometimes impossible. When cancer arises from the floor or lateral wall of the maxil-

lary antrum it often presents as a bulging mass in the gingivobuccal sulcus or cheek. In such cases the site of origin may be fairly certain; nevertheless, cancer arising in the oral cavity, facial bones or orbit must be excluded. On the other hand, if the mesial wall of the antrum is involved, the primary site may be either nasal cavity, ethmoid cells, or antrum. Similarly, tumors filling the nasal cavity may cause confusion. Since such a degree of involvement is commonly observed, it is apparent that without surgical exploration the probable site of origin may be largely a matter of conjecture. Even at operation this point may be in doubt.

Despite the difficulties enumerated, we have attempted to classify our patients according to the probable site of the primary lesion (Table III). Although no site appears to be immune to the devel-

TABLE III

PATHOLOGY ACCORDING TO ANATOMICAL SITE

Site	*Lymphosarcoma*	*Soft part and bony Sarcoma*	*Total*
Nasal Cavity	18	6	24
Antrum	19	4	23
Ethmoid	3	1	4
Sphenoid	–	1	1
TOTAL	40	12	52

opment of cancer, some appear to be more susceptible than others. Twenty-four cases (46%) appear to arise in the nasal cavity, while the maxillary sinuses are a close second with 23 cases (44%) of the total. The ethmoid labyrinth was the primary site in four cases (8%). The sphenoid sinus gave rise to one case of chondrosarcoma. These findings are in sharp contrast to squamous carcinoma and adenocarcinoma of this region, where two-thirds of these cancers arise in the maxillary antrum, and 25 percent arise in the nasal cavity.[3]

Pathology. In our series there were 52 cases of sarcomas arising from the nasal cavity and paranasal sinuses. Of this group, 40 cases were diagnosed as lymphosarcomas, and 12 cases classified as soft part, cartilaginous or osteogenic sarcomas. Of the latter group there were four cases of spindle cell sarcoma, three cases of chondrosar-

TABLE IV

PATHOLOGY OF SOMATIC TUMORS

Type of Tumor	*Number of Cases*
Fibrosarcoma	5
Chondrosarcoma*	3
Myxosarcoma	2
Rhabdomyosarcoma	1
Osteogenic Sarcoma	1
TOTAL	12

* 1 case secondary to fibrous dysplasia of antrum

coma and one case each of myxosarcoma, rhabdomyosarcoma, fibrosarcoma and osteogenic sarcoma (Table IV). One case of chondrosarcoma, as already mentioned, developed at the site of previous surgery for fibrous dysplasia of the maxillary sinus. A similar case has been reported by Gross and Montgomery.[4]

In our experience, lymphosarcoma is the most common type of sarcomatous lesion in the nose and accessory sinuses. These may be 1) the small round cell, lymphoblastic sarcoma, 2) large round cell, reticulum cell sarcoma, or 3) an undifferentiated type called lympho-epithelioma or, by some, transitional cell tumors. All types of lymphosarcoma are more commonly found in lymphoid tissue of the pharynx and nasopharynx.

Fibrosarcoma is composed of spindle or round cells, and frequently has areas of calcification, myxoid degeneration or cartilaginous changes. These tumors, according to Eggston,[2] may be very vascular and contain embryonic angioblastic structures. Myxosarcoma is probably a variant of fibrosarcoma, and consists of very edematous cellular tumor masses that almost simulate nasal polyps. However, they have a great tendency to recur locally once removed, rapidly invading the surrounding structures and filling the nasal cavities. The choanal region is sometimes the site of chondromyxosarcomas in infants and children. Chondrosarcomas appear to arise from cartilaginous structures within the nasal cavity or ethmoids. Osteogenic sarcoma is rare, and our single case appeared to arise from the maxillary sinus. A single case of rhabdomyosarcoma in our

series arose from the nasal cavity in an adult and rapidly developed systemic metastases.

It is significant that in eight of 12 cases, i.e., two-thirds of the somatic soft part sarcomas, chondrosarcomas and the osteosarcoma, the initial pathology report was inaccurate or later changed to sarcoma after initial definitive treatment was completed and local recurrence was present. Lymphosarcoma was accurately diagnosed from the first in most cases. It is probable that if the initial diagnosis of fibrosarcoma, chondrosarcoma or a variant had been returned to the clinician, that aggressive surgical treatment may have salvaged cases otherwise treated.

Symptoms and Signs. Early symptoms and signs of cancer of the nasal cavity and paranasal sinuses are seldom characteristic, since they frequently are associated with chronic inflammatory conditions or nasal polyposis. The most common complaint was nasal obstruction lasting for a period of two months to four years before a definitive diagnosis was made. Local swelling, local pain, epistaxis and swelling of the cheek with proptosis was less common. Tearing frequently accompanied the nasal obstruction with blockage of the naso-lacrimal duct. These symptoms led to nasal polypectomies or minor sinus surgery in eight of the 12 somatic soft part sarcomas and those composed of cartilage and bone, before a definitive diagnosis was made.

Neck node metastases were present on admission in nine of the 40 cases of lymphosarcoma. Eight of these cases were in the homolateral neck, and one case was present in the contralateral neck.

Radiographic Findings. Obstruction to normal drainage channels is a natural sequence to both inflammatory conditions and cancer of the upper air passages. Cloudy air cells on x-ray are of limited value in differentiating between chronic allergic or inflammatory conditions and neoplasm. However, the finding of a mass or bone destruction is of great importance, since it is highly suggestive of neoplasm. In this series, bone destruction was diagnosed in nine of 11 soft part sarcomas and chondrosarcomas, and present in the one case of osteogenic sarcoma. Twenty-five percent of lymphosarcomas showed evidence of bone destruction on routine sinus films.

PROCEDURE AND RESULTS

Treatment. The treatment of sarcomas of the nasal cavity and paranasal sinuses is most difficult because of the proximity of local

vital structures and the advanced stage of the disease by the time the diagnosis is made. There is a rather sharp contrast in the management of the two basically different types of sarcomata.

Soft part sarcomas, chondrosarcomas and osteosarcomas present a surgical problem from the outset. None of these tumors are radiosensitive, and administration of radiotherapy prior to surgery is to delay curative treatment for six to eight weeks. Surgery must be most radical, and consists of subtotal resection of the maxilla, orbital exenteration, and removal of the contents of the nasal cavity, ethmoid and sphenoid sinuses. The defect is immediately skin grafted. The technique of subtotal maxillary resection has been described by Frazell[5] and Cocke and Braund,[6] the latter authors describing the rapid surgical resection utilizing Gigli saws. Preliminary ligation of the carotid arteries will reduce the blood loss about one-half, but complicates the management of cervical metastases by scarring the neck. If the frontal sinus is involved, the combined intracranial-extracranial approach of Smith et al.[7] is employed.

Lymphosarcoma was usually considered a radiation problem from the outset. Of the 40 cases admitted to our clinic, two cases were indeterminate, refusing the treatment offered. Of the 38 cases treated, 26 cases were irradiated and 12 cases were subjected to radical surgery. Two of the latter cases received postoperative radiotherapy as well. The nine cases with cervical metastases received radiotherapy to both the primary site and to the involved neck. The 26 cases that received radiation had external radiotherapy with orthovoltage techniques. One case had supplemental antrostomy with the insertion of intracavitary radium. Of the operated cases, 12 were subjected to resection of the maxilla and contents of the nasal cavity including nine cases requiring orbital exenteration (Table V).

TABLE V

NATURE INITIAL TREATMENT LYMPHOSARCOMAS (40 Cases)

Treatment	*Cases*
Indeterminate (patient refused treatment)	2
External Irradiation	25
External Irradiation and Intracavitary Radium	1
Radical Surgery only	10
Radical Surgery and Postoperative Irradiation	2

End Results. Of the 12 cases of soft part sarcomas, chondrosarcomas, and bony tumors, one case was indeterminate, having been seen in consultation only. Of the remaining 11 cases that were treated, three cases (27%) had survived at the 15-year follow-up period following radical surgery. The survivors consisted of one case each of myxosarcoma, fibrosarcoma and osteogenic sarcoma. None of the three cases of chondrosarcoma survived.

Among the 40 cases of lymphosarcoma, two cases were indeterminate. There were ten successfully treated cases among 38 determinate cases, giving a five-year salvage rate of 26 percent (Table VI).

TABLE VI

END RESULTS

LYMPHOSARCOMA NASAL CAVITY AND SINUSES

Determinate Cases	*Cases*
Determinate cases (status at five years)	38
Dead of cancer before 5 years	27
Postoperative death	1
Successful results	10 (26%)

Of the 12 operated cases there was one postoperative death. Of the 26 cases that were primarily irradiated, the salvage rate was 27 percent. Of the 12 cases that were subjected to radical surgery the salvage rate was 25 percent.

DISCUSSION

Although both the somatic soft part sarcomas, chondrosarcomas and lymphosarcomas were of common mesenchymal origin, the natural history and therapeutic problem of the latter group was markedly different. The soft part sarcomas were more common in females and were frequently associated with nasal polyposis and sinus disease. The lymphosarcoma was slightly more common in males and, preceding sinus disease, occurred in only about 15 percent of cases. The sarcoma group arose as commonly from within the nasal cavity as from the maxillary sinuses, whereas carcinoma in general originated from the maxillary sinuses in fully two-thirds of cases, and only 25 percent arose from the nasal cavity.

Initial false or misleading diagnoses are commonly made in association with soft part or chondrosarcomas. Adequate treatment

in this group is, therefore, frequently delayed. The presenting signs and symptoms were similar in all sarcomas with nasal obstruction, swelling of the cheek and gingivo-buccal sulcus, epistaxis, pain and lacrymation. The soft part sarcomas almost always showed evidence of bone destruction on x-ray, while only 25 percent of lymphosarcomas showed roentgen evidence of bone involvement. Neck node metastases were present in 22.5 percent of the lymphosarcomas.

Radical surgery was mandatory early in the soft part sarcomas, chondrosarcomas and osteogenic tumors. The surgery consisted of subtotal resection of the maxilla with orbital exenteration. The salvage rate at 15 years was three of 11 determinate cases (27%).

Of the 40 cases of lymphosarcoma, 26 were irradiated and 12 were treated by radical surgery. Two of the latter received postoperative radiotherapy. Of the 38 determinate cases in this group, the salvage rate was 26 percent.

SUMMARY

The origin, sex, age and natural history of 52 cases of the nasal cavity and paranasal sinuses are statistically analyzed. Although all cases were of common mesenchymal or mesodermal origin, a survey and contrast in the treatment of the 12 cases of soft part somatic sarcomas, chondrosarcomas and osteogenic sarcoma with the 40 cases of lymphosarcoma is elucidated.

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DISCUSSION

W. Franklin Keim, M.D.: Through his access to the large amount of material at Memorial, I am a little surprised that in the group of tumors of mesothelial origin, there were no solitary plasma cell myelomas. We have had three of these patients with this condition, two of whom have survived with either radiation or surgery, the other patient succumbing. We have also had a patient with hemangiosarcoma of the nasal cavity, who died in spite of treatment.

I don't know what the explanation of this would be. It is perhaps, again, a matter of histologic interpretation, but it would seem to me that, with this great experience that Dr. Lewis has surveyed, the solitary plasma cell myeloma perhaps should have been present.

Joseph H. Ogura, M.D.: I noticed in the somatic tumors that he had a survival rate of 27 percent, if I can remember accurately. I would like to ask Dr. Lewis, of these figures, what were the cases that survived? Were they fibrosarcomas? I would suspect that since he had five cases that perhaps two of these were fibrasarcomas.

Dr. Lewis (closing): First of all, it is true that I have not reported any cases of extramedullary plasmacytoma in this group. As I recall, the cases occurred in the nasopharynx region and I don't think they were classified as occurring in the nasal cavity or paranasal sinuses.

I am glad you asked that question about the type of cases that survive. Of the three cases that survived out of the 11 determinate cases, there was one case of fibrosarcoma, one case of myxosarcoma and one osteogenic sarcoma. None of our chondrosarcomas survived.

CANCER OF THE ORAL CAVITY

FIVE YEAR END RESULTS IN 237 PATIENTS

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This report includes 237 patients with histologically proven cancer of the oral cavity treated at the Washington Hospital Center from its opening in mid 1958 through December, 1963. Follow-up information is current through December, 1968; thus all patients were followed for five to ten years. There were no exclusions, and patients who refused or postponed suggested therapy, or were too ill for other than supportive measures, are included. The socio-economic status represented a cross-section of the greater Washington area; however, it is noted that only 16 percent were Negro compared with a 32 percent Negro incidence in pharynx and larynx carcinoma in the same interval.[1] Female patients comprised 26 percent. The youngest patient treated was a 16-year-old white girl who died of carcinoma of the tongue within one year of treatment. The oldest was a 94-year-old Negro male who died of heart disease, free of buccal mucosa carcinoma 16 months after treatment. The age distribution was 1% below 30, 3% 30 to 39, 18% 40 to 49, 33% 50 to 59, 23% 60 to 69, 15% 70 to 79, 6% 80 to 89, and 1% above 90 years. The starting time for survival in this study was the date of histologic diagnosis, in most cases within one month of the date treatment was instituted.

The oral cavity lesions were divided anatomically and staged clinically according to the February, 1967 report of the American Joint Committee,[2] without modification. The areas designated include 1) the *buccal mucosa*: the inner surface of the cheek and lips to the alveolar ridge and pterygomandibular raphe, 2) the *lower alveolar ridge*, 3) the *upper alveolar ridge*, 4) the *floor of the mouth*

From the Head and Neck Tumor Clinic, Washington Hospital Center.

posterior to the anterior tonsillar pillar base, 5) the *hard palate,* 6) the *soft palate*: the superior lateral margin is the pterygomandibular raphe, the inferior lateral margin includes the anterior surface of the anterior tonsillar pillar, 7) the *anterior two-thirds of the tongue*: from the line of circumvallate papillae to the root of the tongue at the junction of the floor of the mouth, and 8) the *base of the tongue*: from the circumvallate papillae to the base of the epiglottis. The vermillion surface of the lip is not included in the oral cavity. Areas commonly noted clinically as the retromolar trigone, the vallecula and the uvula must be reassigned according to presumed origin; similarly, lesions of the anterior tonsillar pillar are in the soft palate, and must be distinguished from tonsillar lesions which are in the lateral wall of the oropharynx.

Staging was retrospective and followed precisely the T-N-M categories of the American Joint Committee. Unlike the larynx and pharynx protocols[3,4] in which invasion of contiguous anatomic sites determines the "T" designation, in the oral cavity only measurement in greatest diameter is significant. Primary lesions were described as T_1 if 2 cm or less, T_2 from 2 to 4 cm and T_3 if over 4 cm. Cervical lymph nodes were N_0 if not palpable (or if palpable, metastasis not suspected), N_1 if movable homolateral nodes, N_2 if movable contralateral or bilateral nodes and N_3 if fixed on either side. Distant metastases were designated M_1. Stage I lesions were $T_1N_0M_0$, Stage II were $T_2N_0M_0$ and Stage III was $T_3N_0M_0$ or $T_{1\cdot2\cdot3}N_1M_0$. Stage IV was $T_{1\cdot2\cdot3}N_{2\cdot3}M_0$, or any TN with M_1. It should be noted that unlike the pharynx and larynx task force reports, $T_3N_0M_0$ lesions are in Stage III rather than Stage II. Each patient was classified according to his original clinical and radiologic appearance, and not redesignated by surgical findings, later recurrence or metastasis.

A histologic diagnosis of "carcinoma" was made in 233 patients and the bulk of this presentation concerns only these patients. The other four patients (two melanoma, rhabdomyosarcoma, and metastatic seminoma) died of cancer within two years. The six adenocarcinomas are not distinguished from the bulk of epidermoid, squamous, undifferentiated and anaplastic carcinoma.

RESULTS

End results are reported by the actuarial or "life-table-analysis" method illustrated in Table I. This method utilizes all information on patient depletion and results in a survival profile. In this study six patients were lost to follow-up and 20 died of other causes with-

TABLE I*

FIVE-YEAR SURVIVAL RATES OF 58 PATIENTS WITH CARCINOMA OF THE BASE OF TONGUE TREATED FROM 1958 THROUGH 1963 (ALL STAGES)

(1) *Time observed in months*	(2) *Total number patients*	(3) *Died tongue ca.*	(4) *Died other cause*†	(5) *Lost follow-up*†	(6) *Number at risk*‡	(7) *Proportion dying ca.*◈	(8) *Proportion living*§	(9) *Cumulative proportion surviving (%)*♮
0-6	58	5	2		57	.088	.912	91
7-12	51	15	1	1	50	.300	.700	64
13-24	34	10		1	33½	.299	.701	45
25-36	23	6			23	.261	.739	33
37-48	17				17	.000	1.000	33
49-60	17	1			17	.059	.941	31
60+	16				16	.000	1.000	31

* Similar tables prepared for each stage and site. † Without evidence of base of tongue carcinoma. ‡ Number in column 1 minus one-half of columns 3 and 4. ◈ Column 2 divided by column 5. § One minus column 6. ♮ Column 7 times preceding column 8, expressed to nearest whole percent.

out evidence of cancer before the sixtieth month (including four "operative" deaths within one month of definitive surgery). These are not considered oral cavity cancer deaths, nor are they indeterminate, but fit into the third and fourth columns of the Table. End results on samples too small are misleading; thus the stages are combined in reporting the buccal mucosa (17 cases), the alveolar ridges (11 cases and 3 cases) and the hard palate (10 cases). Series brevity also precludes subdividing Stages III and IV into component T-N-M divisions.

End Results Related to Anatomic Site. Buccal mucosa carcinoma occurred in 17 patients. Two were diagnosed at Stage I, three at Stage II, nine at Stage III and three at Stage IV. At six months 94 percent were alive, dropping to 81 percent at one year; only 34 percent survived the second, third and fourth years. Two patients died in the fifth year reducing the cumulative 60-month survival to 20 percent. The sole Stage I survivor received radiotherapy, and the Stage III survivor was operated.

Carcinoma of the lower alveolar ridge was present in 11 patients, of whom seven had Stage III disease. All survived six months, 88 percent one year, 76 percent two years and 62 percent the third through fifth years. The survivors were all treated surgically.

Only three patients had carcinoma arising in the upper alveolar ridge. One died in the second year and the other two in the fourth year, leaving no "cures."

Ten patients had hard palate carcinoma, of which eight were Stage II. At six months 90 percent survived, 60 percent at one year and 49 percent the second through fifth years. Surgery was the primary treatment modality in all survivors.

Table II shows the survival of the 56 floor of mouth patients. Of the 22 survivors, the primary treatment in 18 was surgical, and in four (one Stage I, three Stage II) radiotherapy.

TABLE II

SURVIVAL RELATED TO STAGE OF DISEASE IN 56 PATIENTS WITH FLOOR OF MOUTH CARCINOMA TREATED FROM 1958 THROUGH 1963

Survival time (months)	*Stage I* 5 pts. % surv.	*Stage II* 16 pts. (%)	*Stage III* 26 pts. (%)	*Stage IV* 9 pts. (%)	(*Total*) 56 pts. (%)
6	100	100	100	78	(96)
12	100	93	88	45	(84)
24	100	93	80	11	(74)
36	100	87	72	11	(68)
48	100	80	51	11	(56)
60	100	65	37	11	(46)

The 41 soft palate patients are illustrated in Table III. Only three of the ten survivors were operated upon primarily, the other seven receiving radiotherapy. Three of these seven irradiated patients required subsequent operation for recurrence or metastasis in order to survive five years.

TABLE III

SURVIVAL RELATED TO STAGE OF DISEASE IN 41 PATIENTS WITH SOFT PALATE CARCINOMA*

Survival time (months)	*Stage II* 14 pts. (%)	*Stage III* 16 pts. (%)	*Stage IV* 9 pts. (%)	(*Total*) 41 pts.* (%)
6	100	100	56	(90)
12	100	88	45	(83)
24	79	49	0	(51)
36	64	21	0	(35)
48	57	14	0	(31)
60	43	14	0	(26)

* There were only two patients in Stage I; both survived 5 years (100%).

The 37 anterior tongue carcinomas are profiled in Table IV. Of the 17 survivors, 16 had partial glossectomies, and only one Stage II patient was primarily irradiated.

TABLE IV

SURVIVAL RELATED TO STAGE OF DISEASE IN 37 PATIENTS WITH CARCINOMA OF THE ANTERIOR TWO-THIRDS OF TONGUE

Survival time (months)	*Stage I* 6 pts. (%)	*Stage II* 15 pts. (%)	*Stage III* 14 pts. (%)	*Stage IV* 2 pts. (%)	(*Total*) 37 pts. (%)
6	100	100	100	50	(97)
12	100	100	93	50	(95)
24	100	100	57	—*	(80)
36	100	92	36	—	(67)
48	100	92	22	—	(60)
60	100	92	22	—	(60)

* The surviving Stage IV patient died of other causes in the eighteenth month, apparently free of cancer.

Tables I and V show the 58 posterior tongue carcinomas. Of the 16 survivors, seven had glossectomies (two including the larynx), seven had primary irradiation and two had the combined modalities as planned treatment.

TABLE V

SURVIVAL RELATED TO STAGE OF DISEASE IN 58 PATIENTS WITH CARCINOMA OF THE BASE OF TONGUE*

Survival time (months)	*Stage II* 10 pts. (%)	*Stage III* 25 pts. (%)	*Stage IV* 21 pts. (%)
6	100	100	76
12	100	80	23
24	88	51	12
36	63	38	6
48	63	38	6
60	63	34	6

* There were only two Stage I patients, both surviving five years. The "total" column is given in Table I.

End Results Related to Stage. The oral cavity can conveniently be considered as a whole, increasing the cases in each category under discussion. The larger lesions involve adjacent sites. Accessibility is similar in all locations in contrast to the nasopharynx and hypopharynx. Five-year survival by site ranged from 20 to 60 percent (ignoring the zero-survival, three-case upper alveolar ridge); the survival by stage alone ranged from 5 to 90 percent (Table VI). It is apparent that the stage of the disease is far more important than the location. Those sites with poorer prognosis, such as the posterior

TABLE VI

SURVIVAL RELATED TO STAGE IN 233 PATIENTS WITH CARCINOMA OF THE ORAL CAVITY

Survival time (months)	*Stage I* 19 pts. (%)	*Stage II* 69 pts. (%)	*Stage III* 100 pts. (%)	*Stage IV* 45 pts. (%)	(*Total*) 233 pts. (%)
6	100	100	98	73	(94)
12	100	96	86	33	(80)
24	100	88	56	8	(60)
36	100	77	42	5	(50)
48	94	72	33	5	(44)
60	94	65	27	5	(40)

tongue (80% Stages III and IV), present with more advanced lesions than those with better prognosis such as the anterior tongue (43% Stages III and IV).

Treatment modalities can be compared by stage, first discarding the 26 patients indeterminate before five years and the ten patients who received only supportive therapy. Cure rates appear higher than the over-all experience but comparison is easier (Table VII).

TABLE VII

"ESTIMATED" FIVE-YEAR SURVIVAL RELATED TO TREATMENT AND STAGE IN 200 PATIENTS WITH ORAL CAVITY CARCINOMA*

Stage	*Surgery*	*Radiotherapy*†	*Combined*‡
I	92% of 14	100% of 4	
II	63% of 35	50% of 26	0% of 1
III	43% of 42	8% of 26	18% of 11
IV	6% of 17	6% of 17	0% of 7

* Indeterminate and supportive-therapy-only patients discarded, causing higher "estimated" figures for comparison. † Usually supervoltage 2 mev. ‡ Considered combined therapy if both modalities planned initially and completed within four months.

Unfortunately the distinction between palliative and curative attempts could not be made from the records, nor were those patients distinguished who refused or delayed operation. Nevertheless, results in moderately advanced lesions favor surgery over radiotherapy by a ratio of 63:50 in Stage II and 43:8 in Stage III. Choice of treatment seems less important in Stages I and IV. Pre-operative radiotherapy in a combined program was less effective than surgery alone in Stage III (18% survival), but these 11 patients had such varied treatment patterns that no conclusion should be inferred.

Recurrent disease was treated by surgery alone in 19 patients of whom six (32%) survived the fifth year. Supplementary radiotherapy was given to 35 patients of whom four (11%) survived. Both modalities were used to treat recurrent disease in 20 patients and seven (35%) survived. Eight patients died of their original cancer between the sixth and tenth year, and six were living with recurrent carcinoma over five years at the conclusion of this study

in December, 1968. These 14 patients are considered 60-month survivors in the Tables above but are not "cures." A second primary carcinoma developed at some time in 28 patients during the ten year interval studied, nine patients dying of their new disease. (Two of these patients succumbed to their fifth carcinoma.)

Related observations show a survival rate of "squamous carcinoma" biopsied patients nearly twice that of "epidermoid carcinoma" patients. Caucasian survival at five years was twice that of Negroes, and women outlived men by a ratio of 5:3.

SUMMARY AND CONCLUSIONS

1. The T-N-M system is easier to apply in the oral cavity than in the pharynx and larynx. There is little dispute about the greatest diameter of the primary lesion ("T"). The greatest dispute arises in deciding if small lymph nodes in the submaxillary region are metastatic or inflammatory. The most experienced opinion has proven most accurate.

2. Prognosis is more influenced by stage of disease than any other parameter. Early diagnosis is more important than site, histology or choice of treatment. Excluding the base of the tongue, the oral cavity is accessible to all physicians and dentists, and these cancers rarely present as metastasis. I have found little benefit from the use of exfoliative cytology or toluidine blue. All erythroplastic and most leukoplakic areas should be biopsied.

3. Treatment of most Stage I lesions, unless mutilating or refused, should be operative. The prognosis is similar and radiotherapy may still be used if a second primary lesion develops in a nearby less accessible, more mutilating area. Similar reasoning suggests surgery in Stages II and III, and here the prognosis is better than radiotherapy. In Stage IV, the prognosis is so poor that post-therapy comfort should be considered and "palliation" distinguished from "cure." The 19 patients in this series who had combined therapy (Table VII), did not out-survive those receiving surgery alone.

4. The continuing death rate in the fourth and fifth years (Table VI) suggests inadequate original treatment or multicentricity and casts doubt on studies with two and three year results in Stages II and III.

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DISCUSSION

Francis L. Lederer, M.D.: I cannot let the opportunity arise without commenting about the thoroughness of the work and the fact that these figures came about as a result of a great deal of effort on the part of Dr. Trible and his associates.

The group in Washington had their trials and tribulations just like other centers did as to who does what with which and to whom some years ago, and it is interesting to note that as time has gone on they have gotten not only additional experience but a great deal of credit from other professional groups.

I think the work, whether supported by the hospital itself, the community or the doctors, shows an effort to do a good job and I appreciated very much personally this type of presentation. I think it would be worth while to study it at leisure.

Dr. Trible (closing): I simply wish to thank Dr. Lederer for his kind remarks.

ONE HUNDRED LARYNGEAL CANCERS STUDIED BY SERIAL SECTION

JOHN A. KIRCHNER, M.D.

NEW HAVEN, CONN.

Serial section of the entire larynx as a method of studying the behavior of cancer within this organ was first reported by LeRoux Robert in 1936.[1] Although the value of this method is generally recognized, the process is so laborious and expensive that only small numbers of specimens have been prepared.[2-4]

Through the help of grants provided by the National Cancer Institute during the past four years it has been possible to prepare serial sections of nearly every larynx removed for cancer by members of the Yale New Haven staff in Otolaryngology. The present report is a summary of the experience with the first 100 specimens now available for study. During this period of time approximately 25 specimens were not made available for reasons of oversight or inconvenience. About three-quarters of the surgical specimens are from the author's private practice or from that of the chief resident. There were 91 total laryngectomies, nine hemilaryngectomies and three horizontal supraglottic laryngectomies in this series.

The larynx in each case was processed according to the technique described by Tucker.[5] Ninety-two specimens were sectioned in the coronal plane so as to be oriented in the same way as the pre-operative laryngograms. Three specimens were sectioned in the horizontal and five in the sagittal planes. Every fiftieth section was stained for the original examination. If this revealed a question on growth pattern or invasion of neighboring structures, or if the presence of residual tumor nests were being sought after radiotherapy, then sections between those already prepared were mounted and

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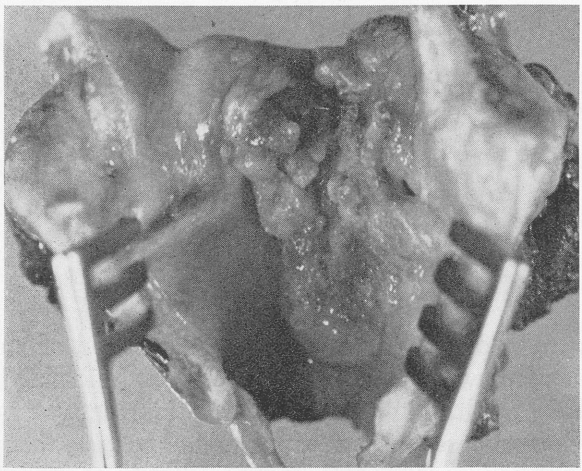

Fig. 1.—Posterior commissure cancer extending from upper edge of posterior laryngeal inlet to inferior edge of cricoid cartilage. Growth did not cross the ventricle, and is classified for this report as "posterior larynx."

stained. When necessary, each section was stained through certain portions of the larynx.

Among the objectives of the present study is a practical system of classification and staging of laryngeal cancer which will guide the laryngologist in planning appropriate treatment. Surgical or radiologic extirpation of laryngeal cancer is compatible with preservation of function only if there is available to the clinician a system of clinical evaluation which accurately reflects the tumor's biologic potential. Present systems of staging still exhibit certain shortcomings and are under continuing study by various groups to account for such clinical features as fixation of the involved tissues, and to recognize the importance of histologic differentiation. The importance of fixation in the biologic behavior of laryngeal cancer has been well documented statistically[6,7] and clinically.[8] The importance of the precise site of the lesion has been demonstrated by surgical[9-13] and histologic[14-16] observations.

Various characteristics of the primary lesion which influence the rate of cervical metastases have been studied and reported in detail by McGavran and his associates.[17] Effects of irradiation on laryngeal cancer have been described by Goldman[4] and indications for surgery, radiotherapy, or combinations based on clinical and

TABLE I

ONE HUNDRED LARYNX CANCER SPECIMENS

Glottic	31
Supraglottic	10
Epiglottic	10
Transglottic	19
Infraglottic	3
Pyriform Sinus	24
Anterior Larynx	2
Posterior Larynx	1

TABLE II

GLOTTIC CANCER WITHOUT FIXATION REQUIRING LARYNGECTOMY OR HEMILARYNGECTOMY (14)

Residual Cancer after Radiotherapy	8
Anterior Commissure invaded	3
Arytenoid involved	2
Hemoptysis during Radiotherapy	1

laboratory observations have been reported at length by LeRoux Robert.[18]

This report is part of a continuing study of the histologic correlates of such clinical features as fixation, location, staging, radio resistance and pre-operative radiographic appearance of the primary growth. Cervical metastatic rates have not been calculated for this report since many of the cases are too recent to be meaningful.

FINDINGS

Although all but three cases could be assigned to the usual categories of glottic, supraglottic, infraglottic and pyriform sinus, the interests of accuracy seemed better served by breaking off an "epiglottic" group from the supraglottic, and including a "transglottic" category. The latter term is one which was coined by McGavran and his associates to describe a growth extending within the deep tissues above and below the ventricle. It is a term which

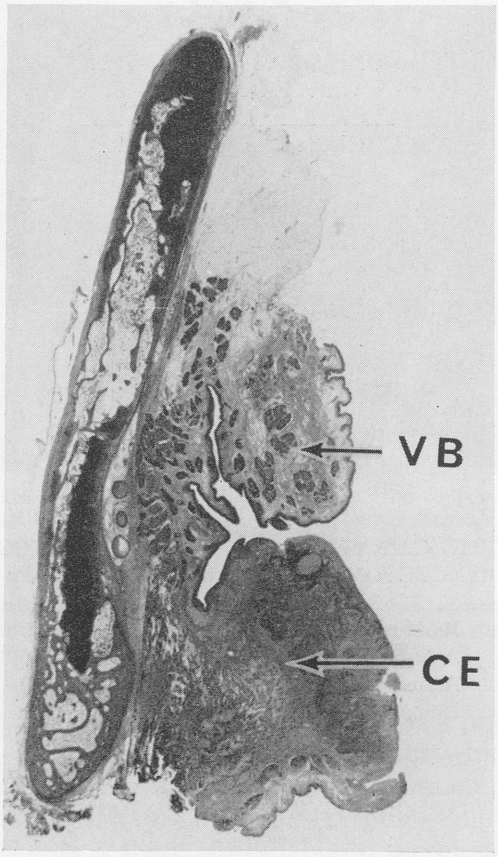

Fig. 2.—Glottic cancer with mobile cord. Removed by hemilaryngectomy. Growth is entirely superficial to the conus elasticus (CE), a characteristic of mobile cord lesions in this series.

is difficult to avoid as one studies full size sections of the cancerous larynx.

Two large growths in the anterior larynx and one in the posterior commissure resisted classification. The former probably started as epiglottic growths but involved the infraglottic larynx anteriorly without crossing the ventricle in the usual "transglottic" fashion. The posterior commissure tumor extended inferiorly to the lower edge of the cricoid cartilage, superiorly to both arytenoids, and still did not involve cords or ventricles (Fig. 1). These three are classified separately, two as anterior and one as posterior larynx (Table I).

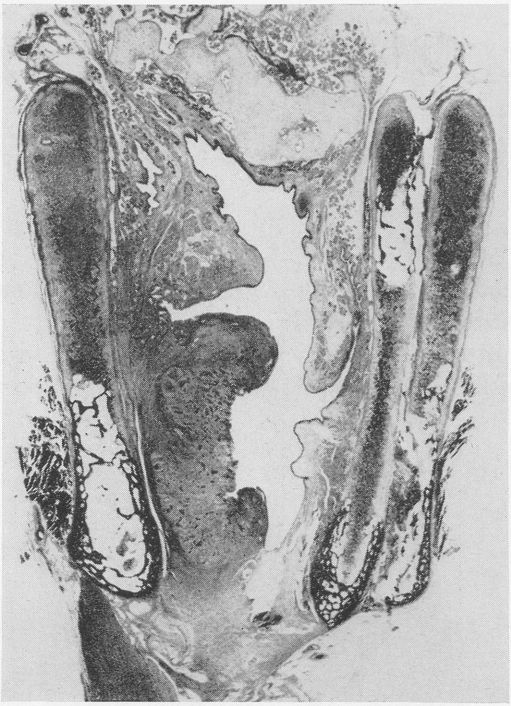

Fig. 3.—Glottic cancer with fixation of vocal cord. Cancer can be seen deep in the thyroarytenoid muscle adjacent to the thyroid cartilage. Infraglottic extension also contributes to fixation.

Glottic Lesions Without Fixation. Fourteen such cases came to operation, 11 total and three hemilaryngectomies. The indications for operation are given in Table II.

Serial sections showed that 12 of these 14 cancers were superficial to the conus elasticus (Fig. 2). One other case showed minimal invasion of the conus and one was histologically free of cancer after pre-operative radiotherapy.

TABLE III

GLOTTIC CANCER WITH FIXED VOCAL CORD (11)

Extension deep into thyroarytenoid muscle, to or near thyroid cartilage (two of these invaded arytenoid)	8
Invading thyroid cartilage	3

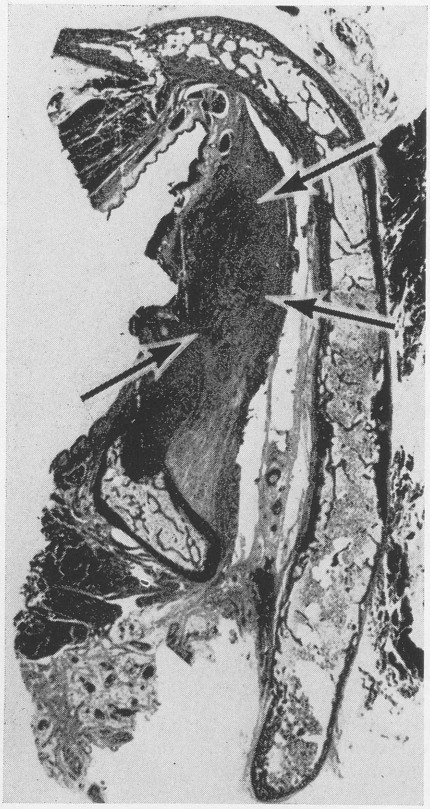

Fig. 4.—Horizontal section of glottic cancer with limitation of motion. Arrows indicate the extent of invasion into the muscle, which is less extensive than in the cases with fixation.

Glottic Cancer With Fixed Cord. Of 11 glottic cancers which had exhibited vocal cord fixation, eight showed deep invasion of the thyroarytenoid muscle to, or near, the thyroid cartilage (Fig. 3). Two of these eight invaded and partially destroyed the arytenoid cartilage. Three other cases showed invasion of the thyroid ala by the growth (Table III).

Glottic Cancer With Limited Mobility. Of six cases with limited mobility, four showed lesser degrees of thyroarytenoid muscle invasion than the the cases with fixation. In one case the arytenoid was surrounded by cancer, and in another the limitation was due to infraglottic extension (Fig. 4, Table IV).

TABLE IV

GLOTTIC CANCER WITH LIMITED MOBILITY (6)

Thyroarytenoid muscle invaded	2
Cancer in anterior cord only	2
Arytenoid embedded in cancer	1
Infraglottic extension	1

Infraglottic Cancer. Of three cases so classified, two showed extensive invasion of cartilage: the thyroid in one case, the cricoid in another (Fig. 5). The third case showed no residual cancer after 4500 r pre-operative radiation. This latter impression can only be an estimate, of course, even with serial sections (Table V).

TABLE V

INFRAGLOTTIC CANCER (3)

Invasion of thyroid cartilage	1
Invasion of cricoid cartilage	1
No residual cancer after 4500 r	1

Supraglottic. As reported by McGavran and his associates,[17] the supraglottic cancers tend to be less invasive than similar growths in other parts of the larynx. In only one of the ten supraglottic cancers in this series was the thyroid cartilage invaded, in contrast to the epiglottic group where it was seen in about half the specimens.

In four of the ten cases, the arytenoid mucosa was involved with cancer to a degree that supraglottic laryngectomy would have required removal of one arytenoid in three cases, both arytenoids in the fourth case.

In serial sections, supraglottic cancer showed the same tendency to remain confined to the tissues above the larynx that the surgeon and pathologist observed in the gross specimen (Fig. 6, Table VI).

TABLE VI

SUPRAGLOTTIC CANCER (10)

Treated by total laryngectomy	9
In four of these nine, cancer invaded soft tissues over	
one arytenoid	3
both arytenoids	1

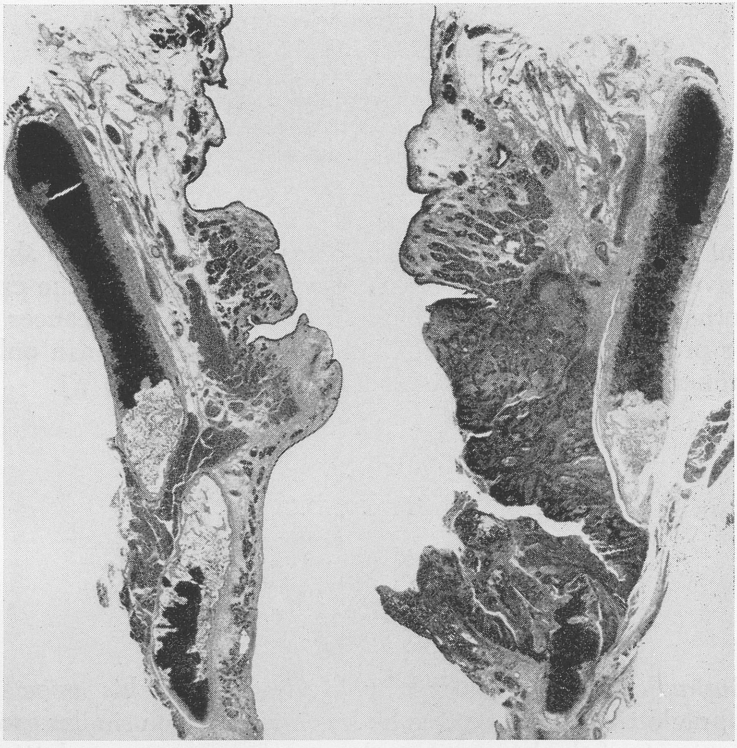

Fig. 5.—Infraglottic cancer with cord fixation and invasion of the cricoid. By comparing the normal part of the cricoid ring on the left side of the photograph one can see that only the ossified portion of the cricoid has been destroyed by the cancer.

Transglottic Cancer. This is a term that is almost inescapable for the clinician and the radiologist. It describes the behavior of a large, deep growth occupying the tissues above and below the ventricle better than any other term in current usage. Cancer which crosses the ventricle may very likely have originated there, according to serial section studies reported by LeRoux Robert in 1936. In any event, the transglottic cancer is characterized by invasion of the "paraglottic space," an area described by Tucker,[19] and delineated in a convincing fashion by the dye injections performed by Pressman and his associates.[15]

Cancer which invades this space exhibits an aggressive growth pattern, infiltrating the laryngeal framework in most cases, and emerging from the confines of the larynx by direct extension be-

Fig. 6.—Supraglottic cancer, showing no tendency to cross the ventricle in either this or other sections.

tween the thyroid and cricoid cartilages at the cricothyroid membrane (Fig. 7).

Of 19 transglottic growths, 15 invaded cartilage (Figs. 8 and 9). Sixteen of the 19 reached or penetrated the lateral parts of the cricothyroid membrane (Table VII).

TABLE VII

TRANSGLOTTIC CANCER (19)

Cartilage invaded		15
Thyroid	8	
Cricoid	5	
Arytenoid	5	
Against thyroid cartilage but not invading		4
Cancer at or into cricothyroid membrane		16

Fig. 7.—Transglottic cancer growing toward the cricothyroid space. This pattern was observed in 16 of 19 transglottic growths. Cutting the cricothyroid muscle in this location, or the cricopharyngeus more posteriorly, frequently carries the risk of cutting into tumor in growths of this type.

Pyriform Sinus. Pyriform sinus growths are usually far advanced when first seen. Nearly half of them in this study invaded the thyroid cartilage (Fig. 10). Invasion of the nearby posterior cricoarytenoid or interarytenoid muscles occurs, with resulting limitation of vocal cord motion (Fig. 11). As the pyriform sinus cancer enlarges it may surround the lateral edge of the thyroid cartilage, spill over its upper edge or grow forward within the loose spaces of the ossified portion of the thyroid ala (Fig. 12). It may grow forward into the paraglottic space and resemble a transglottic growth. It may then escape the confines of the larynx by way of the cricothyroid membrane just as any other cancer which has invaded the paraglottic space (Table VIII).

Epiglottic Cancer. Of ten epiglottic cancers, nine invaded the epiglottic cartilage, usually by growing through the fenestrae, as

Fig. 8.—Transglottic cancer destroying the cricoid ring and the lower edge of the thyroid ala.

TABLE VIII

PYRIFORM SINUS CANCER (24)

Invading cartilage		11
Thyroid	11	
Cricoid	1	
Invading Posterior Cricoarytenoid muscle		3
Invading Interarytenoid muscle		1
Invading Ventricular band		1
Invading Cricoarytenoid joint		1

described by Ogura[14] and by Bocca[20] (Fig. 13). Five specimens showed invasion of the thyroid cartilage anteriorly or along its upper lateral edge, verifying the clinical impression that these growths were not resectable by horizontal supraglottic laryngectomy. Such

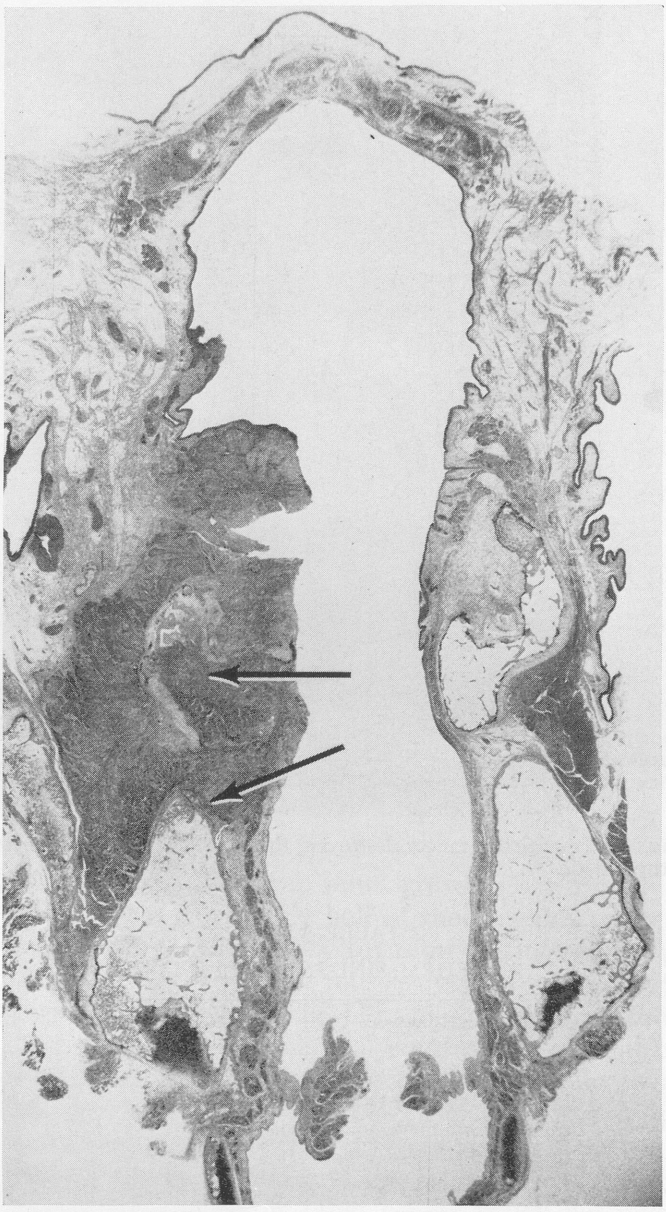

Fig. 9.—Transglottic cancer destroying arytenoid cartilage (upper arrow) and beginning to invade the upper edge of the cricoid (lower arrow).

Fig. 10.—Pyriform sinus cancer (C) invading thyroid cartilage and interarytenoid muscle.

invasion should be suspected if the lower edge of the epiglottic growth reaches or closely approaches the epiglottic petiole near the anterior commissure. (This was true in three of four such cases in this series.) It is particularly true of poorly differentiated cancer (Fig. 14) or of tumors which are grossly ulcerative.

Pre-epiglottic space invasion occurred in over half this group, usually by direct growth forward through the epiglottis, or more inferiorly, through the thyroepiglottic ligament (Table IX).

Cord Fixation. Limitation of motion or vocal cord fixation usually indicates deep invasion of the thyroarytenoid muscle by cancer.

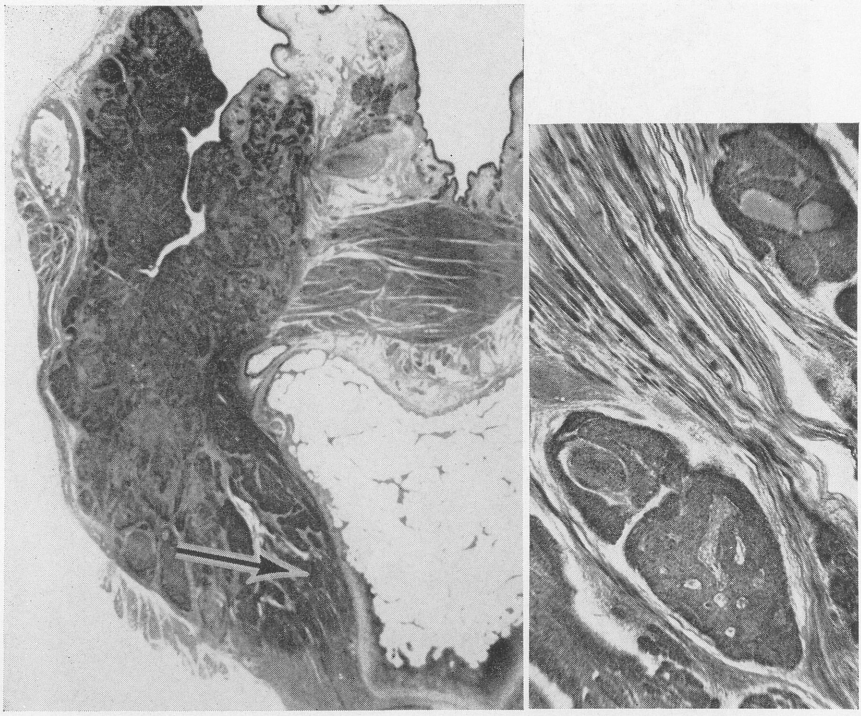

Fig. 11.—A: Pyriform sinus cancer invading posterior cricoarytenoid muscle at arrow. B: High power view of same area.

TABLE IX

EPIGLOTTIC CANCER (10)

Invasion of Epiglottic cartilage	8
Invasion of Thyroid cartilage	5
Invasion of Pre-epiglottic space	6

While this is most commonly seen in glottic cancer, such invasion may occur as part of the spread of cancer from areas other than the true cord. Much of this spread may be confined to the deep tissues, the tumor usually growing forward deep to the conus elasticus (Fig. 15).

Nearly a third of the cases in this series exhibiting cordal fixation were transglottic lesions, some of them showing relatively little mucosal involvement along the true cord.

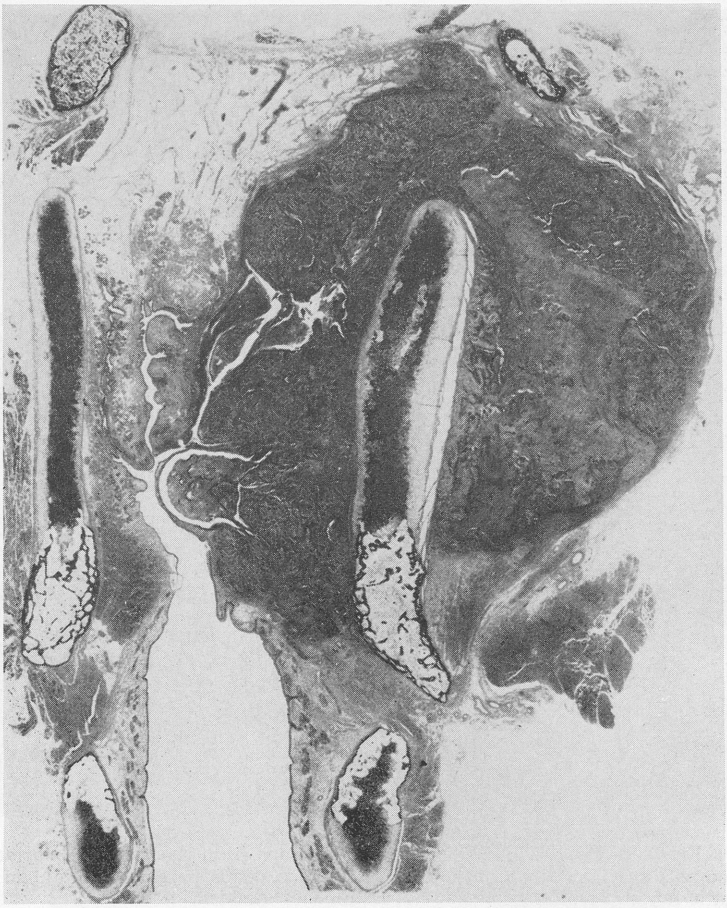

Fig. 12.—Pyriform sinus cancer growing around the upper edge of the thyroid cartilage and presenting itself as a mass palpable over the thyroid ala.

Other causes of limited mobility of the vocal cord, or of actual fixation, are less common and include invasion of the arytenoid cartilage or of the neighboring interarytenoid or posterior cricoarytenoid muscle; advanced fibrosis after radiotherapy; edema developing during radiotherapy. While chronic inflammatory infiltration has been observed in the deep tissues near the advancing edge of the growth, it is unlikely that this could produce a significant degree of fixation (Table X).

Radiation Failures. A precise analysis of the 21 radiotherapy failures in this series is not possible at present since it will require

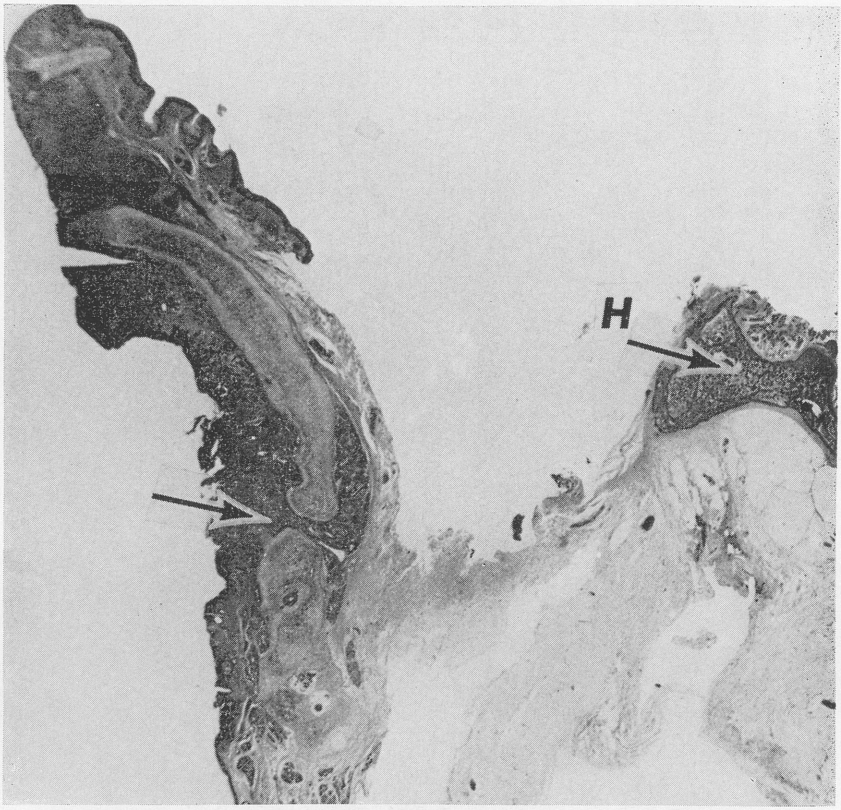

Fig. 13.—Epiglottic cancer growing forward from the laryngeal surface to occupy a submucosal position in the vallecula. It is passing through an epiglottic fenestra which ordinarily houses mucous glands.

TABLE X

VOCAL CORD FIXATION (30)

Location of primary cancer:	
Glottic	11
Glottic with infraglottic extension	3
Transglottic	9
Pyriform Sinus	4
Infraglottic	1
Anterior Larynx	1
Epiglottis	1

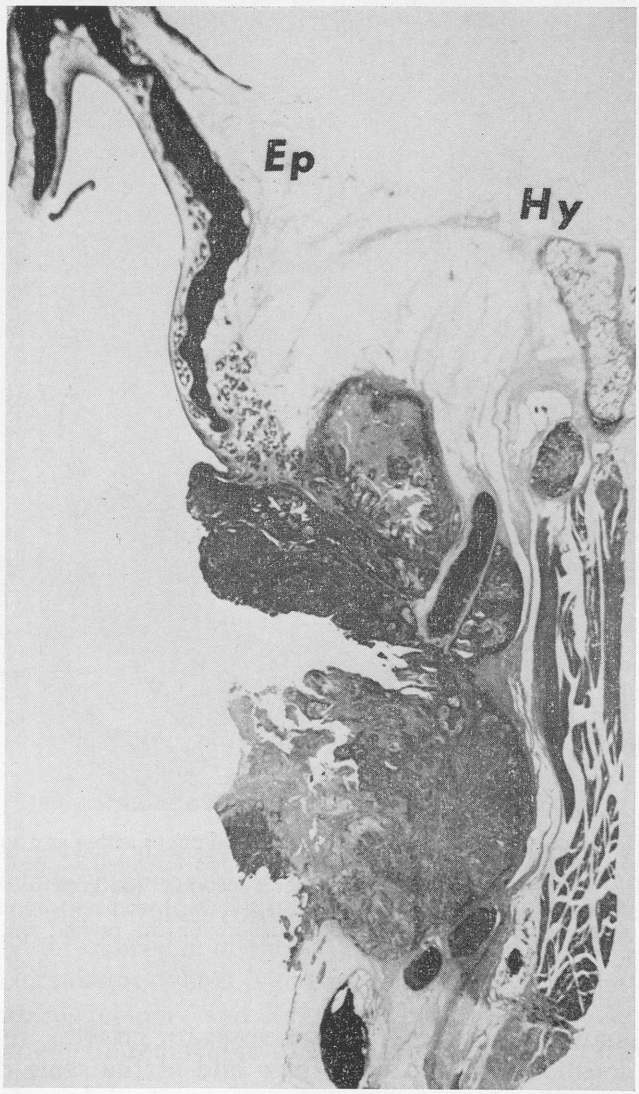

Fig. 14.—Cancer at base of epiglottis invading thyroid cartilage and extending into pre-epiglottic space (sagittal section). Ep: Epiglottis, Hy: hyoid bone.

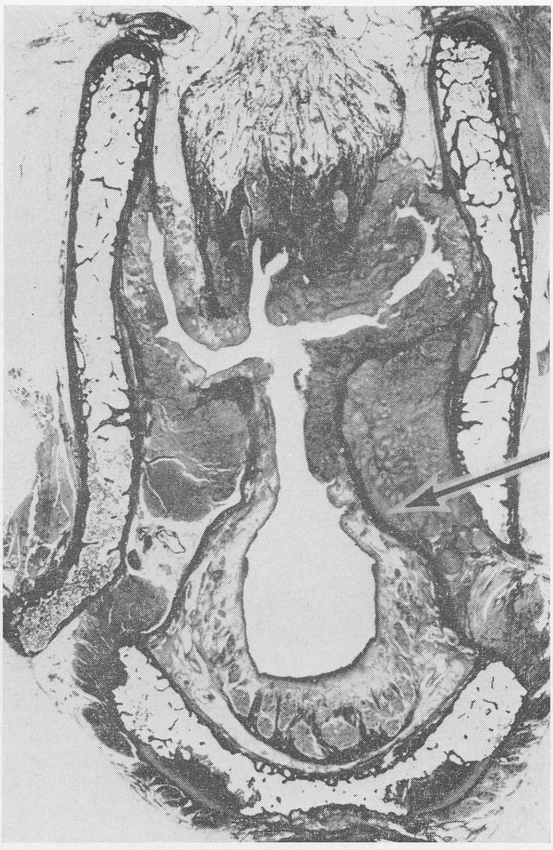

Fig. 15.—Elastic tissue stain showing conus elasticus acting as a soft-tissue barrier to the medial growth of cancer which has extended forward from a transglottic position as seen on more posterior sections. The growth is entirely deep to the conus at the arrow, and exhibits the usual tendency to escape the confines of the larynx by growing outward between the thyroid and cricoid cartilages.

a similar analysis of radiotherapy successes for growths in the same anatomic locations, of the same stage and of the same degree of differentiation, fixation, size, etc.

Invasion of Laryngeal Framework. In most instances where cancer invades the thyroid, cricoid or arytenoid cartilage, the invasion is nearly always confined to that portion of the cartilage which has ossified. While aggressively-growing cancer occasionally invades cartilage directly, this has been the rare exception in this series (Fig. 16).

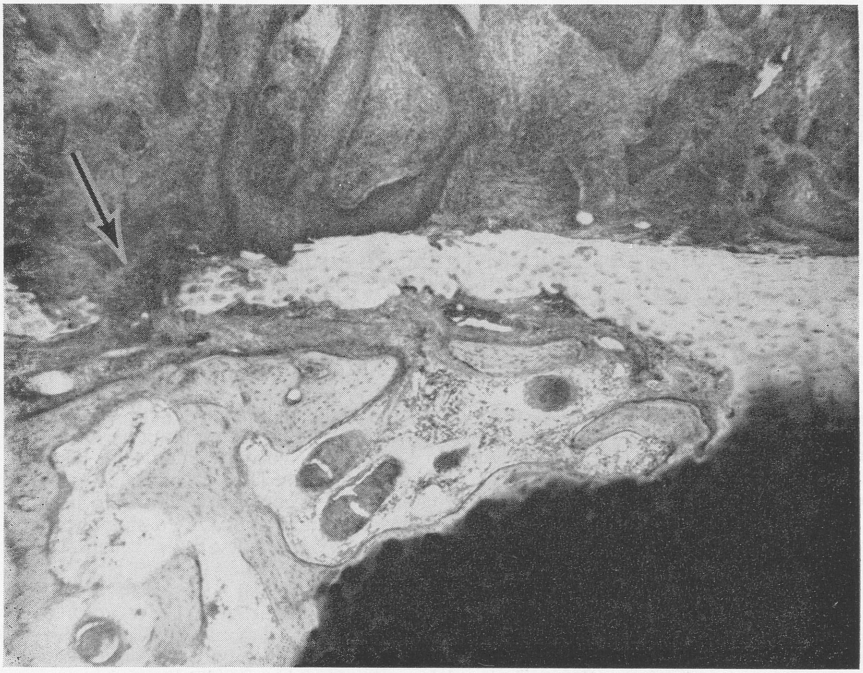

Fig. 16.—High power view showing tendency of cancer to invade the ossified portion of the laryngeal framework, the cricoid plate in this instance. The cancer grows on the surface of the cartilage in the lower part of the photograph and invades the open spaces of the ossified part of the cricoid at the arrow.

DISCUSSION

Vocal cord fixation indicates deep invasion of the thyroarytenoid muscle by cancer which often reaches, and sometimes invades the thyroid ala. In fixed cord cases with invasion of cartilage it is usually the inferior edge of the thyroid ala or the framework at the anterior commissure which is destroyed. This observation indicates that hemilaryngectomy and less extensive operations carry a high risk of inadequate removal of cancer in the case with a fixed cord.

Vocal cord fixation can result from invasion by cancer from other primary sites within the larynx. Of 30 cases with a fixed cord, 11 were primarily glottic cancer, nine transglottic and four pyriform sinus. Although other reasons for vocal cord fixation were occasionally observed, the most frequent finding in these fixed cord cases was invasion of the paraglottic space by cancer, regardless of the site of origin.

Mobile Cord. Thirteen of 14 cases showed no cancer deep to the conus elasticus. Three of these had been removed by hemilaryngectomy, the rest by total laryngectomy because of radiation failure and other causes shown in Table II.

Invasion of the cartilaginous framework was found in 14 of 19 transglottic cancers, 11 of 24 pyriform sinus, and in two of the three infraglottic cancers. These findings illustrate the necessity for wide surgical resection and probably indicate an important reason for the high incidence of radiotherapy failure with cancer in these locations.

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Acknowledgment—I am greatly indebted to Doctors Peter Hukill and Robert Hutter of the Department of Pathology for their many helpful consultations; to Doctor Yasushi Murakami, my research associate in Laryngeal Neurophysiology, for his skillful assistance with the preparation of photographic materials; to my two technical assistants, Winifred Craig and Sylvia Freeman, for their continuing efforts to improve the quality of our sections; and to my colleagues who have contributed surgical specimens.

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DISCUSSION

GABRIEL F. TUCKER, JR., M.D.: I think a thing which should be apparent to all of us is the fact that there is a great variety of ways in which tumor may go in the third dimension, perpendicular to the mucosa. We are beginning to get some idea as to what these may be, but I think the practical question which overrides all of this is "What good will this do us in the treatment of patients?" We hope this will lead the clinician who sees these sections, and I am sure John agrees with me on this, to think of and be impressed by the third dimension of a tumor, to try and estimate this clinically and apply it to treatment.

Clinical radiographic studies must be correlated in this light. It also means that we need to revise our system of classification so that we include the third dimension of a tumor in our clinical and pathologic thinking.

JOSEPH L. GOLDMAN, M.D.: I would like to comment on the effect of irradiation on cancerous invasion of the cartilage. Dr. Kirchner has shown so many instances of invasion of the cartilage by cancer. In our serial section studies of larynges which have been treated by pre-operative irradiation, we have found many instances of cartilage replaced entirely by fibrous tissue with no evidence of cancer, which would indicate (much to our surprise) that cancerous invasion of cartilage can be cured by irradiation. I believe that it is important to appreciate and understand this in the whole concept of pre-operative irradiation of cancer of the larynx.

DR. KIRCHNER (closing): I would like to comment to my friend Joe Goldman that I really wonder whether we can say that the larynx is free of cancer if we don't find the cells in serial sections. I feel that in spite of the value of this technique, you can't always tell whether a cell is viable, particularly in these thick celloidin sections of 20 microns. To know whether this particular cell has a potentiality of malignant growth I think is really asking a lot of the technique, even with Dr. Goldman's beautiful studies. I wonder whether time is not the one measuring device that we have here and that we really can't say, looking at the sections, that five years from now the patient may not meanwhile have developed cancer. This is why I say that it is too early, at least, for me, to make any conclusions about the study.

THREE YEARS EXPERIENCE WITH CRYOSURGERY IN HEAD AND NECK TUMORS

DANIEL MILLER, M.D.
BOSTON, MASS.

Cryosurgery as a technique is not new, having been used mainly by dermatologists intermittently over the past 70 years. However, the incidence of failure in the treatment of cancers of the head and neck is of such proportions depending on the area, the nature of the tumor and its proclivity for resistance to conventional methods of therapy, such as with radical surgery, Cobalt 60 x-radiation and/or radium, that it has led some of us to look for other adjunctive measures for help in such problems.

X-ray therapy equipment, no matter how sophisticated, is only as useful as the men behind the machines. The technical and more radical approaches for extirpation of disease with the surgical knife is only as successful as the dexterity and judgment of the physician wielding the knife. Cryosurgery as a tool is less well understood than x-radiation in its effect on tissues; and certainly the cutting blade is a much more precise method of approach. However, cryobiologists have supplied us with considerable information regarding the effect of freezing on various tissues *in vitro* as well as *in vivo*. The precision with which freezing temperatures can be applied to tissue that must be destroyed has improved as we have learned more about the techniques of utilizing the equipment available for such purposes. Just as the x-radiation equipment has been improved upon over the past 50 years, so has the cryosurgical equipment become more sophisticated. In 1961, Cooper[1,2] reintroduced the concept of destruction of tissue with freezing techniques and since then there has been considerable interest in this approach.

It is now three years since we have had the opportunity to work with cryosurgery and feel the time has come to present to otolaryngologists the status of this technique as we have experienecd it to date. Prior to embarking upon a review of the type of pathology treated in 275 patients observed over these few years it should be stated unequivocally that no statistical long-term cure rate should be reported by this technique until a considerably greater length of time has elapsed, and until more cancer centers have had an opportunity to assess their results during a long-term trial.

Initial trial with the cryosurgical equipment was directed, for the most part, at palliation of symptoms and signs of advanced cancer. Its major purpose was to eliminate bulky obstructive tumor, reduce or eliminate pain, help reduce dysphagia, eliminate odor and, on occasion, reduce airway obstruction; and finally, of great importance, to give some hope to patients that something more could be done for them when all other methods had failed.

The equipment utilized includes the Linde Cooper closed probe utilizing the 2.7 mm tip and the 9.5 mm tip. The results with this equipment were variable; therefore, this led to the utilization of the Brymill open-ended probes with direct application of liquid nitrogen at −196°C using spray tip apertures of .5 mm to 1 cm. A comparative study of the two types of equipment was reported at the Academy meeting in 1968 and is presently being published in the Transactions.[3] The study to determine the tissue temperature depth obtained using thermocouples at varying depths revealed that the Brymill self-pressurizing apparatus with direct application of liquid nitrogen has proved to be the more effective cryosurgical delivery unit for destruction of tissue, although the closed Linde probes are still very helpful in areas where direct nitrogen cannot be delivered because of the immediate anatomy.[4] Both cryosurgical instruments are utilized by us, depending, however, upon the situation at hand.

In order for cryosurgery to have a destructive effect it has been widely accepted that a critical temperature level of at least −20 to −25°C must be reached in the treated tissue.[5] Only the 1 cm aperture at the open end Brymill probe with liquid nitrogen pouring through it was able to reach a 2 cm depth in a convenient length of time, namely, four minutes. It was possible in a ten minute period application with the closed 9.5 mm Linde probe to reach a depth of only 1 cm. We have determined that cartilage is very sensitive to cryogenic injury and even the smallest 2.7 mm Linde closed probe was able to produce destruction of the cartilage of the auricle in a few

minutes. The direct application of liquid nitrogen caused destruction in less than one minute using even the .5 mm aperture.

We have applied the liquid nitrogen through the .5 cm Brymill probe directly to the carotid artery of a dog producing a solid iceball and elimination of pulsation, only to have it return within a period of 30 seconds after discontinuing the application of the freezing liquid nitrogen. Thirteen days later a section of the artery revealed an intact wall with a loss of nuclei in the muscle cells and very little other change. The resistance of large walled blood vessels to cryosurgery destruction is evident.[6] This does not preclude the blow-out of a carotid artery should there be a tumor which is of a different substance that has already invaded the wall of the artery. Sensory nerves are extremely sensitive to cryosurgery and pain fibers are eliminated almost immediately upon application of the freezing technique; thereby pain is eliminated in most patients with this technique.

We have had an opportunity to treat over 275 patients with early neoplastic disease as well as T-4 lesions. The greater majority of these patients had advanced carcinoma, inoperable and incurable. Pain has definitely been relieved in most instances for long periods of time, extending from several months to one and one-half years. Tumors and defects at the root of the nose which have caused such pain that necessitated morphine around the clock resulted in the elimination of morphine and other narcotic analgesics with cryosurgery. In some instances, it has necessitated two or three courses of cryo-therapy separated at long intervals of many months, to control the symptomatology and bulk of tumor for a reasonable duration of time.

Cavernous hemangiomatas have been treated with successful eradication of the greatest bulk of the tumor no longer requiring further surgical excision.[7] Skin cancers,[8] including basal cell as well as squamous cell near the eye lids,[9-11] alar region, forehead, cheek and temple have been eliminated with a minimum of scarring and a minimum of loss of pigmentation in many patients with no recurrence of any of the lesions to date. The duration of treatment time and the hospital stay is minimal. Recurrent carcinomas of the floor of the mouth,[12,13] antrum cavities and metastatic masses of the neck have been kept under control for long periods of time. A persistent lymphosarcoma of the tonsil has been eradicated, there being no recurrence locally or anywhere else in the body for one and one-half years since cryosurgery was introduced, following the failure of

x-radiation therapy. Small carcinomas of the anterior tonsillar pillar have been treated with no recurrence to date, one year later. One carcinoma of the larynx, persistent or recurrent, following radiation therapy for a T-1 lesion has been free of recurrence since cryosurgery was applied as a secondary treatment, it now being ten months since the treatment.

It is necessary to emphasize that this is not a statistical report such as a five-year cure statistical survey, but rather a report as to events which have transpired with our experience during this limited period of time utilizing cryosurgery. Twelve patients with papillomata of the larynx have been treated with complete clearing of papillomata in four patients and recurrence in the others, although at a greatly reduced rate. The papillomata of children have not responded as well as the papillomata in the adult larynx. This may be a technical problem in view of the limited area for maneuverability with the small closed cryo probe in the smaller larynx of children. All children who have cryosurgery applications for recurrent papillomata should have tracheotomies performed prior to the maneuver. This is not necessary nor has it proved even suggestive in any of the adults.

Leukoplakia of the hard palate, soft palate and buccal mucosa have been treated with 45 to 60 seconds application of liquid nitrogen using a 1 mm aperture, with successful elimination of all leukoplakia. Its use in the treatment of juvenile angiofibroma has been of inestimable value when used correctly.[14] When first utilized it was thought that the tumor itself could be destroyed in view of the first attempt at this tumor by Work and his associates.[15] This proved to be insufficient to destroy the tumor through which coursed considerable warm blood, which prevented the destruction of sufficient tissue to be effective. However, we have since learned that the cryo probe can be inserted into the tumor at intervals during the course of its removal and reduce the amount of blood loss by 75 to 80 percent. The freezing technique is used not of itself as the method of destruction of tissue but as a method of sealing off the numerous blood vessels, enabling the surgeon to perform his dissection with greater ease.

Although it is more difficult to destroy osseous tissue with cryosurgery it is possible to destroy metastatic or direct extension neoplasm which has invaded the bone. If a sufficient area of bone is exposed with a loss of periosteum it is possible for granulation tissue

to grow out from the periphery, and skin graft may be successful if deemed necessary at a later date.

In concluding this brief report it is possible to say at this point in the development of this additional tool for the treatment of neoplasms of the head and neck, that cryosurgery has proven its value as an adjunctive means of palliation in the suffering of cancer of the head and neck as well as a curative tool in certain cancers of the head and neck. The technique for the proper application of the closed and open probes is readily learned and easily applied. This tool can be made more precise but only if used in the light of the present knowledge as to depth of penetration of the freeze, using thermocouples wherever possible. It is our feeling that many primary carcinomas of the head and neck now treated by x-radiation or by scalpel or electro surgery may very well be treated with cryosurgery. This treatment takes much less time. It can be done in many instances under local or no anesthesia. The patient's stay in the hospital is considerably lessened and the need for plastic reconstruction may be reduced considerably as there is much less deformity with its use. If necessary it can be used in repeated courses. It can also be used in conjunction with the other conventional methods of cancer therapy. As has been stated before, our optimism for the future use of cryosurgery is cautious optimism based on the information presented to you today.

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DISCUSSION

JOHN S. LEWIS, M.D.: There is one word of caution and that is in regard to the use of this modality in the treatment of juvenile laryngeal papillomatosis. I have treated three such cases with the cryoprobe on five or six different occasions during different sittings, and these patients subsequently developed laryngeal stenosis. I must point out that you are administering thermal injury to tissue and this is a predictable sequela of cryosurgery.

The other thing I would like to emphasize is the fact that cryosurgery is a palliation in the treatment of cancer, not a curative method except perhaps in basal cell carcinomas and the unusual case of lymphosarcoma that Dr. Miller mentioned.

GEORGE F. REED, M.D.: Dr. Miller said that he was not able to control the angiofibroma itself with the cryoprobe, presumably because of its great vascularity, but yet he told us of a couple of cases of cavernous hemangiomata that were cured by cryotherapy. This doesn't seem to fit either with the angiofibroma experience or with the dog experience with the carotid artery that he demonstrated and that has been shown before.

JOHN F. DALY, M.D.: Dr. Irving Cooper who devised the first probe gave the second one to the New York University group for use and we have used it in many conditions with rather limited results. We have found it useful in recurrences of nasopharyngeal carcinomas and in recurrences of tumors arising in the nose such as neuroesthesiomas. We have also used it in leukoplakia of the mouth and have had satisfactory results. We have been disappointed with the results in angiomas. An angioma of the tip of the nose in a young girl did not respond to multiple applications of the probe. These applications were made directly into the angioma through an incision in the nasal vestibule.

Our experience has not been the same as Dr. Miller's in dealing with the large, sloughing tumors in the oral cavity which bleed and become infected. In our hands the cryotherapy produced additional tissue necrosis which became infected again and still remained a stinking mess. A papilloma of the nasal vestibule did not respond to surgical excision and a skin grafting did respond with the applications of cryotherapy. We have used it also in the larynx without producing fibrosis or adhesions of the vocal cords but it did not eliminate recurrences.

Dr. Miller has carried the application of cryotherapy much beyond what we have done and this undoubtedly accounts for the successful results he has had.

J. RYAN CHANDLER, M.D.: After hearing Dr. Miller's initial report of his work at the meeting in Hollywood last year and after visiting him in Boston, we, too, began to use cryosurgery on selected cases of the head and neck and you might be interested in knowing our experience.

It has not been as good as Dr. Miller describes, particularly on lesions outside of the oral cavity, for the same reasons that Dr. Daly just mentioned. The necrosis of tissue, even in the oral cavity, produces quite an objectionable odor which can be helped some upon removal and debridement of the area.

One case which would be interesting to the group (I told Dr. Miller about it) is a 60-year-old man with recurrent carcinoma of the soft palate, tonsil fossa and nasopharynx. Following radiation therapy he was treated with three applications of extensive cryosurgery to result in the destruction, or apparent destruction, of the tumor, but there was bleeding from his internal carotid artery, I believe for the reasons that Dr. Miller mentioned. The man survived ligation of the common carotid artery, which was necessary for its control, and he is now some five months post last cryosurgical application without evidence of local disease, but he is hemiplegic.

JOSEPH H. OGURA, M.D.: About a year and a half ago Dr. Miller came to St. Louis and we obtained the same unit that he has.

I am really impressed by the palliation in some cases. If I might talk about two in particular, both of these had a maxillectomy for carcinoma with a recurrence and had been irradiated. On one, with the recurrence, we used the Brymil unit and one of them is a year now without any evidence of recurrence. The tumor shrank remarkably and although I am sure that there may be some tumor there, I am quite certain that this patient would not be alive today.

Now, the other one is a patient who had a jaw/neck dissection for a large cancer on the right side. Following this the patient developed a recurrence on the opposite side of the jaw, following which the patient received a heavy course of irradiation and had radiation persistence.

In this one patient we used a Brymil probe and I was simply amazed. The patient is now nine months. These are the two cases.

Now, the third one is a mucoepidermoid that recurred following maxillectomy. That one, too, is about six months, following two courses of freezing.

I would like also to reiterate what Dr. Miller said about papillomas. This particular one was so massive the patient had papilloma down in the bronchial airway as well as in the larynx and subglottis, with which we failed after repeated measures, including the use of vaccine. On this patient, we opened the larynx up and froze it completely with a Brymil unit, using the cryoprobe and repeated endoscopically with the new laryngeal cryoprobe by Brymil, this patient now has not had any papilloma recurrence since the last application, which is about eight months. Prior to this time we had repeatedly tried to take out the papilloma by endoscopic procedures as frequently as three months.

DR. MILLER (closing): From the standpoint of the treatment of papillomatosis with cryosurgery and web formation in youngsters, I have one patient who has a web which has formed and I am a little concerned about it. I am concerned about future applications. I think Doctor Lewis's suggestion as to problems relating to this therapy in a juvenile larynx is an excellent caution. It is probably due to technical difficulty inserting a small probe in a narrow field of visibility.

I should mention at this point Doctor Lewis's own extensive work with cryosurgery to glomus tumors of the middle ear, with which I have had no experience to date.

The second question, raised by Doctor Reed: The answer to the destruction of the cavernous hemangioma is different than the almost angiomatous type of lesion with a lot of fibrous tissue, as noted in this youngster. The thin walls of the small and medium size veins and the walls of the medium sized arterial vessels can be

destroyed with cryosurgery. The thicker the walls, as the thick walls in vessels such as the carotid artery and the thickness of the tissue about the arterial blood supply in the vessels in the angiofibroma, are probably the reasons why the cryosurgery doesn't get through to it. There is no question that there is a tremendous amount of warm blood that courses through an angiofibroma actively as a heat-sink which further prevents the cryosurgical destruction of it. The temperature does not drop below the —25°C in the depths of it, although a nice ice ball is seen.

From the standpoint of the treatment of metastatic tumors which have been described by Doctor Daly as having been unsuccessful, the answer to this lies in the fact that you were using the small 2.7 mm Linde probe. Tissue penetration studies presented last fall by us at the Academy Meeting reveals that the Linde 2.7 mm probe is not the proper applicator for this type of lesion. The 2.7 mm probe will not destroy beyond a couple of millimeters and, therefore, even though an ice ball is formed, there is insufficient destruction of the tissue underneath and this is why we also failed to get very far using that same little probe in our earlier studies. However, this small probe is most helpful to the neurosurgeon in the treatment of Parkinson's disease.

Now, the larger Linde probe, the 9.5 mm, if it could be used, would get to the depth of at least a centimeter in at least a reasonable period of time. There has been failure in the understanding of equipment. Unfortunately, the equipment was set to read —140°C on the Honeywell regulator, therefore, it was thought we must have —140°C deep within the tissue. This is not so. This is why we did those tissue penetration studies using thermocouple at varying depths. I think this explains some of our original failures using the small and large Lindo cryoprobe, especially the smaller one.

From the standpoint of the sloughing tumors, I have had the same experience you have had and I learned why. Again, the tissue penetration studies revealed that at two cm only the large one cm liquid nitrogen spray got to a depth of two cm. It explained why you obtained superficial slough and why I did in some of these tumors. Therefore, my approach to these large metastatic lesions is surgical, either with electrosurgery or with scalpel, excising the greatest bulk of the tumor. Get to the minimal level that you can safely; you can stop the bleeding with the cryo directly applied to tumor immediately thereafter. Then you know you have at least two cm of depth that you can still reach with the nitrogen. If you do not get to this level, you are going to get a flowering of this tumor within a period of a few weeks plus the slough plus the odor, and I think this explains why that has taken place in some of your cases.

I have had some experience with papilloma of the septum and lateral wall of the nose. They have responded very satisfactorily to cryosurgery.

Doctor Chandler's report of the carotid artery blowout, which I knew about from our own personal communication is explanable. As stated before in the presentation, if there is tumor invasion of the carotid artery, the tumor is of different substance than a muscular wall and, therefore, if it is destroyed with the freezing the wall will rupture at that point and a carotid blowout will then occur.

Doctor Ogura's experience is certainly most interesting and I am appreciative of the fact that he has reported those few cases demonstrating its value.

I repeat once again, for you Doctor Daly particularly, that our feeling about the value of cryosurgery is one of cautious optimism, but I think it has a definite place in our armamentarium now. This is not ready to replace surgery with the scalpel; it is not ready to replace x-radiation for the most part except in many skin lesions. Its proper place will be found by those of us who continue on with the work. This is the beginning of it.

Memorial

LYMAN GILDER RICHARDS, M.D.

(1893 - 1968)

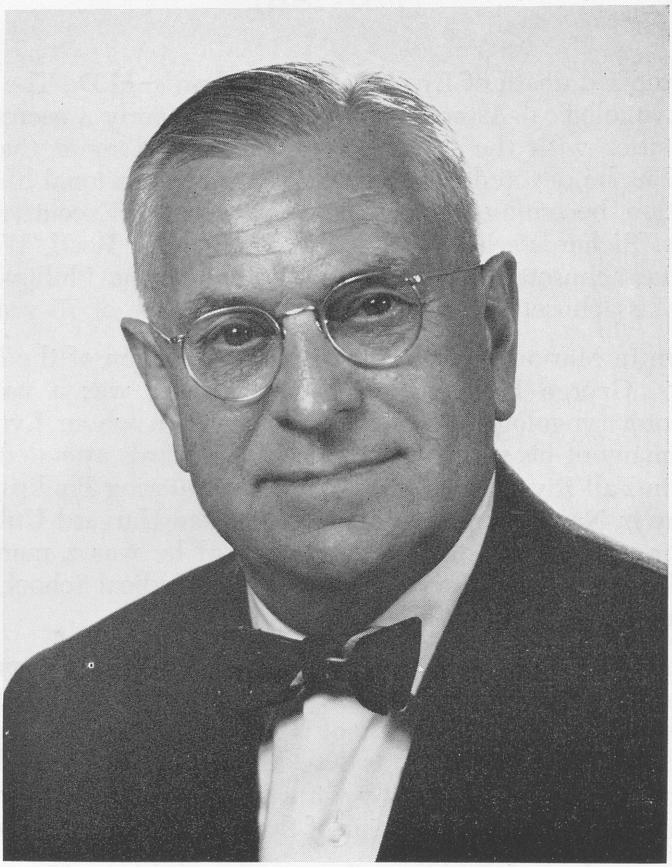
In the sad death of Lyman Gilder Richards, M.D., The American Laryngological Association has lost not merely a secretary, as his activities with the Association encompassed more than that. From 1959, he devoted practically his entire professional life to the Association, becoming what is better described as Executive Secretary. Dr. Richards who had lived at 12 Clovelly Road, Wellesley Hills, Massachusetts died on June 10, 1968 at the Phillips House of the Massachusetts General Hospital at the age of 75 years.

Born in Marion, Massachusetts, he was the son of the late Dr. and Mrs. George Lyman Richards. His father was a nationally known otolaryngologist of the old school, from whom Lyman inherited many of his sterling qualities. Dr. Richards attended public schools in Fall River, Massachusetts before entering Phillips Exeter Academy in New Hampshire. He then entered Harvard University, graduating cum laude in 1916. At Harvard he was a member of the track team. He then enrolled in Harvard Medical School, graduating cum laude three years later.

After a year with the Near East Relief in Turkey, he spent two years as a surgical resident at St. Luke's Hospital in New York, after which he took up the study of Otolaryngology with a two year residency at the Massachusetts Eye and Ear Infirmary. This was followed by postgraduate study with Dr. Chevalier Jackson at the Post-Graduate School of Medicine of the University of Pennsylvania.

He was chief of Otolaryngology at the Childrens Hospital and Peter Bent Brigham Hospital in Boston for ten years. He also served as Bronchoscopist at the Massachusetts Eye and Ear Infirmary and as Otolaryngologist for the Massachusetts Institute of Technology. During World War II he served at Pearl Harbor as a Commander in the Medical Corps, United States Navy.

He was a member of the New England Otological and Laryngological Society, serving some time as Secretary; the New England Pediatric Society; the American Academy of Ophthalmology and Otolaryngology; the American Broncho-Esophagological Society, serving as Secretary and President; the American Medical Associa-



LYMAN GILDER RICHARDS, M.D.

tion; the American Laryngological, Rhinological, and Otological Society, serving as Editor from 1933 to 1943 and as President in 1948. He was also a Fellow of the American Otological Society. In 1966, he received the Newcomb Award of the American Laryngological Association.

He is survived by his wife, Kathryn, a daughter, Mrs. James Manson of Concord, Massachusetts, and two sons, Robert Lyman Richards of Sherborn, Massachusetts and George Lyman Richards, of Duxbury, Massachusetts.

He was a member of Phi Beta Kappa, Alpha Omega Alpha, Pi Eta Club, Harvard Club of Boston, and the Massachusetts Institute of Technology Faculty Club.

Honors, of themselves, meant little to Lyman. He repeatedly refused to be even considered as President of the American Laryngological Association, an honor which he supremely deserved. From the time he first assumed office until his death, all of his professional activities were centered in his service to the Association. Many of us will recall the picture taken at the Annual Meeting in Hollywood Beach in April of this year. As sick as he was, he directed every activity of the Association, even to the sitting of the members for this annual picture. In everything Lyman did he was a perfectionist.

During his career as Secretary he served under the following Presidents: McNally, Broyles, LeJeune, Hilding, Furstenberg, Holinger, Pressman and, finally, Boies. All of these can testify to his dedication to the Association and the value of his services.

He had very rigid standards in all medical disciplines and never would deviate from them for any matters of expediency. This was something he had undoubtedly inherited from his father.

On the other hand he was a many-sided individual, interested in sports, especially skiing and golf, and music and the theater. He was an excellent violinist playing with professional skill. Many of the older members will recall occasions when, at some social meetings of the Societies, Lyman would take the violin from the leader of the orchestra and really put life into it. One of his hobbies was the theater and he had the ability to put on some of the finest sketches that our societies have ever seen. These were always carefully rehearsed and professionally directed by Dr. Richards. For an example, during his Presidency of the Triological Society, we were all delightfully entertained by a program based upon the

then popular shows, including the Travels of Fred Allen and the duet between Mr. Gallagher and Mr. Shean. Probably the last production, and one which showed his talents to the 'nth degree, was his "This is your Life" dedicated to Dr. LeRoy A. Schall on his retirement. This he directed to the most minute detail and was a fitting tribute to Dr. Schall.

Perhaps his closest friend was his former classmate at Harvard Medical School, Dr. C. Harold Jameson, now living in Camden, Maine. I am indebted to Dr. Jameson for the privilege of seeing a letter from Dr. Richards written just before his untimely death. In this he apologizes for his poor penmanship but states he is confined to his bed, trving to get his anginal attacks quieted down, before having a cardiac catheterization and indicated heart surgery. He states he is obviously getting worse and has no other choice. He makes reference in this letter to his interest in becoming a member of a Boston organization known as "The Speckled Band." He speaks of his plan to write his thesis qualifying for membership during his convalescence from the proposed heart surgery. This organization referred to is one concerned with Sherlock Holmes tales by Sir Arthur Conan Doyle.

It is obvious that he realized the situation and he faced it with supreme courage as evidenced by the minute details he went into in arranging for the funeral services that should be carried out.

So lived and died Lyman Gilder Richards, M.D., eminent otolaryngologist, whose professional career was characterized by dynamic devotion and dedication. The American Laryngological Association will cherish his memory for many, many years to come.

F.T.H.

Executive Sessions

EXECUTIVE SESSION

SUNDAY MORNING, MARCH 23, 1969

The opening session of the Ninetieth Annual Meeting of the American Laryngological Association, Inc., held at the Roosevelt Hotel, New Orleans, Louisiana, convened in the Grand Ballroom on Sunday morning, March 23, 1969, at eight twenty o'clock, Francis W. Davison, M.D., Danville, Pennsylvania, President of the Association, presiding.

The following candidates were elected to Fellowship:

Active Fellowship

Dr. A. H. Andrews, Jr., Chicago, Ill.

Dr. J. R. Ausband, Winston-Salem, N.C.

Dr. J. T. Dickinson, Pittsburgh, Pa.

Emeritus Fellowship

Dr. W. W. Eagle

Dr. A. C. Furstenberg

Dr. A. G. Rawlins

Dr. A. T. Smith

Dr. E. P. Cardwell

The Nominating Committee was then elected from the floor as follows:

Francis E. LeJeune, M.D., New Orleans, La., Chairman

Daniel C. Baker, M.D., New York, N.Y.

Emanuel Skolnik, M.D., Chicago, Ill.

Victor Alfaro, M.D., Washington, D.C.

G. Slaughter Fitz-Hugh, M.D., Charlottesville, Va.

ANNUAL BUSINESS MEETING

Monday Morning, March 24, 1969

The meeting reconvened for the Annual Business Meeting of the Association on Monday morning, March 24, 1969, in the Grand Ballroom at eight-twenty o'clock, President Davison presiding.

Secretary Lathrop then presented the report of the secretary.

REPORT OF THE SECRETARY

The Eighty-Ninth Annual Meeting of the American Laryngological Association was held on April 25 and 26, 1968, at the Hollywood Beach Hotel, Hollywood Beach, Florida. The President, Dr. Lawrence R. Boies, presided. Fourteen scientific papers were presented. Seventy-eight Fellows and 192 guests were in attendance.

The Librarian, Historian and Editor, Dr. F. Johnson Putney, reported the deaths of the following:

Emeritus Fellows: Dr. Thomas Gittens
Dr. Joseph D. Kelly

Active Fellow: Dr. Joel J. Pressman

The Newcomb Award was presented to Dr. Walter P. Work.

The following were elected to Fellowship:

Active

Dr. J. R. Chandler, Miami, Florida

Dr. David D. DeWeese, Portland, Oregon

Dr. Richard T. Farrior, Tampa, Florida

Emeritus

Dr. Clyde A. Heatly, Rochester, New York

Dr. G. Edward Tremble, Montreal, Canada

Corresponding

Sir Terence Cawthorne, London, England

Dr. Fumio Nakamura, Kyoto, Japan

Honorary

Dr. Justo M. Alonso, Montevideo, Uruguay

Dr. Thomas C. Galloway, Evanston, Illinois

Dr. Eelco Huizinga, Groningen, Netherlands

Dr. Georges Portmann, Bordeaux, France

Dr. Luzius Ruedi, Zurich, Switzerland

The following officers were elected for the coming year:

President: Dr. Francis W. Davison

First Vice-President: Dr. DeGraaf Woodman

Second Vice-President: Dr. John A. Murtagh

Secretary: Dr. Frank D. Lathrop

Treasurer: Dr. Alden H. Miller

Librarian, Historian and Editor: Dr. F. Johnson Putney

First Councilor: Dr. Albert C. Furstenberg

Second Councilor: Dr. Paul H. Holinger

Third Councilor: Dr. Francis A. Sooy

Fourth Councilor: Dr. Lawrence R. Boies

The next Annual Meeting will be held at the Hollywood Beach Hotel, Hollywood Beach, Florida, on the mornings of April 24 and 25, 1970. The American Otological Society will hold their meetings on the mornings of April 19 and 20 and those of the Triological Society on the mornings of April 21, 22 and 23. The American Broncho-Esophagological Association will hold their meetings on the afternoons of April 20 and 21, the American Society for Head and Neck Surgery on the afternoons of April 22 and 23 and the American Academy of Facial and Plastic Reconstructive Surgery on the afternoons of April 24 and 25.

Since the last Annual Meeting the Association has lost through death the following:

Dr. Lyman G. Richards, an Active Fellow since 1934, who died on June 10, 1968.

The present Fellowship of the Association is:

Active 96 Honorary 10 Corresponding 12 Emeritus 23 Total 141

Respectfully submitted,

Frank D. Lathrop, M.D., Secretary

(On a motion by Dr. Lederer seconded by Dr. Putney, the secretary's report was accepted.)

REPORT OF THE TREASURER

Exhibit A

AMERICAN LARYNGOLOGICAL ASSOCIATION
GENERAL FUND
OPERATING STATEMENT AND FUND BALANCE
FISCAL YEAR ENDED MARCH 31, 1969

Income			
Dues (91 members at $75.00, 3 new members at $115.00)		$7,170.00	
Registration fees—1968 Annual Meeting, Hollywood Beach, Florida		70.00	
Sales of "Transactions"		1,030.00	
Interest on certificates of deposit and savings accounts		544.92	
Total income			$ 8,814.92
Expenses			
1968 Annual Meeting:			
Banquet and cocktail party	$1,299.13		
Guest speaker	476.00		
Projectionist	263.72		
Reporting service	458.31		
Total annual meeting expense		2,497.16	
Council meeting expense		992.31	
Cost of "Transactions"		3,413.89	
General expense:			
Stationery, printing and postage	1,643.11		
Clerical hire	450.00		
Auditing	175.00		
Treasurer's bond	63.00		
Donations—Centurion Club, American Council of Otolaryngology	600.00		
Gifts	514.50		
Bank charges and foreign exchange	90.50		
Total general expense		3,536.11	
Total expenses			10,439.47
Net income (loss) for the Year			(1,624.55)
Fund Balance, March 31, 1968			11,191.65
Fund Balance, March 31, 1969 (Exhibit B)			$ 9,567.10

Exhibit B

AMERICAN LARYNGOLOGICAL ASSOCIATION
TRUST FUND
OPERATING STATEMENT AND FUND BALANCE
FISCAL YEAR ENDED MARCH 31, 1969

Income

Interest on U.S. Government bonds		$ 458.20	
Dividends on common stock		264.06	
Dividends from mutual funds:			
Reinvested in funds	$ 3,278.43		
Less: loss on exchange of Boston Fund for Massachusetts Investors Trust stock	1,190.77	2,087.66	
Donation to Memorial Fund		500.00	
Net income for the Year			$ 3,309.92
Less: Award from Casselberry Fund retained earnings			200.00
Net Income for the Year			3,109.92
Fund Balance, March 31, 1968			56,310.08
Fund Balance, March 31, 1969 (Exhibit C)			$59,420.00

TRUST FUND ASSETS

Cash		$ 9,913.65	
Accounts Receivable—E. F. Hutton & Company		14.80	
Investments (Exhibit D):			
U.S. Savings Bonds, Series H (at cost, par and maturity values)	$ 6,500.00		
Common stocks:			
Purchased (at cost)	$5,672.38		
Donated (market value at date of donation)	2,362.00	8,034.38	
Mutual fund stock (at cost)	34,957.17	49,491.55	
Total Trust Fund Assets			$59,420.00

CASH SUMMARY

Cash in Commercial Account, Bank of America, Western-Santa Monica		$ 2,363.78
Cash in Savings Account, Bank of America, Western-Santa Monica Branch		7,116.97
Cash in Time Certificates of Deposit, Bank of America		10,000.00
Total Cash		$19,480.75
Allocation:		
General Fund (Exhibit A)	$ 9,567.10	
Trust Fund (above)	9,913.65	
	$19,480.75	

Exhibit C

AMERICAN LARYNGOLOGICAL ASSOCIATION
INDIVIDUAL TRUST FUNDS
MARCH 31, 1969

	Principal and Retained Earnings 3/31/68	*April 1, 1968 to March 31, 1969*			*Principal and Retained Earnings 3/31/69*
		*Earnings**	*Donations*	*Awards*	
Casselberry	$ 8,506.74	$ 424.49		$ (200.00)	$ 8,731.23
Johnston	7,873.89	392.94			8,266.83
De Roaldes	5,419.21	270.43			5,689.64
Newcomb Trust	3,091.32	154.26			3,245.58
Newcomb Prize	721.54	36.00			757.54
Research	24,597.77	1,227.43			25,825.20
Memorial	6,099.61	304.37	$500.00		6,903.98
	$56,310.08	$2,809.92	$500.00	$ (200.00)	$59,420.00

* Net income for the year is allocated to each fund in the ratio it bears to the total balance of the trust funds at the beginning of the year.

Exhibit D

AMERICAN LARYNGOLOGICAL ASSOCIATION
TRUST FUND INVESTMENTS
MARCH 31, 1969

U. S. GOVERNMENT BONDS

Six $1,000.00 Series H. bonds (at cost)	$6,000.00	
One $500.00 Series H bond (at cost)	500.00	
Total U. S. Government bonds		$ 6,500.00

COMMON STOCKS:

	Number of Shares	*Cost*	*Market Value at Date of Donation*	
American Telephone & Telegraph Co.	70	$4,845.02		
International Business Machine Corp.	1		$ 517.00	
International Business Machine Corp.[1]	7	803.98		
McDonnell Douglas Corp.[2]	112		1,170.00	
McDonnell Douglas Corp.[3]	14	23.28		
McIntyre-Porcupine Mines, Ltd.	15		675.00	
		$5,672.38	$2,362.00	8,034.38

MUTUAL FUND—Massachusetts

Investors Trust (at cost)	1,938.974	34,957.17
Total Trust Fund investments		$49,491.55

[1] Includes 5¼ shares received in stock dividends.
[2] Includes 86 shares received in stock dividends.
[3] Includes 13.44 shares received in stock dividends, and exercised rights to purchase .56 share.

ACCOUNTANT'S REPORT

To the Members of the American Laryngological Association.

Gentlemen:

I have examined the statements of cash receipts and disbursements of the AMERICAN LARYNGOLOGICAL ASSOCIATION, including Trust Funds, for the fiscal year ending March 31, 1969. My examination was made in accordance with generally accepted auditing standards, and accordingly included such tests of the accounting records and such other auditing procedures as I considered necessary in the circumstances.

Cash receipts and disbursements were verified as recorded to date, and all securities were examined or accounted for as of this date.

In my opinion, the accompanying statements of cash receipts and disbursements present fairly the financial position of the American Laryngological Association and its Trust Funds at March 31, 1969, and the results of its operations for the year then ended, in conformity with generally accepted accounting principles applied on a basis consistent with that of the preceding year.

Respectfully submitted,

JOHN MACOMBER MCGEE, JR.
Certified Public Accountant

(On a motion by Dr. LeJeune duly seconded from the floor, the report was accepted.)

REPORT OF THE NOMINATING COMMITTEE

FRANCIS E. LEJEUNE, M.D.: (Chairman of the Nominating Committee) The Nominating Committee wishes to submit the following as Members of the Council:

Dr. Alden H. Miller
Dr. Joseph Atkins
Dr. Stanton Friedberg
Dr. Frank Lathrop
Dr. Charles Norris
Dr. F. Johnson Putney
Dr. Paul H. Holinger
Dr. Francis A. Sooy
Dr. Lawrence R. Boies
Dr. Francis W. Davison

There were no further nominations from the floor. It was moved, seconded and duly passed that the report be accepted.

REPORT OF THE LIBRARIAN, HISTORIAN AND EDITOR

The Eighty-ninth Meeting of the American Laryngological Association was held on April 25 and 26, 1968, at the Hollywood Beach Hotel, Hollywood, Florida. There were 78 Fellows and 192 guests in attendance. The meeting was ably reported by the Martin C. Johnson Reporting Service whose representative, Mrs. Annabelle Square, gave efficient and dependable service as usual. The completed papers were sent to the Annals Publishing Company within three weeks and were published in the June issue of the Annals. All of the material for the 1968 Transactions together with the corrected discussions were in the hands of the printer by July 1, 1968, and the Transactions were mailed to the membership on September 20, 1968.

The prompt publication was due largely to the efforts of Dr. Ben Senturia and his staff aided by the procurement of a second printing firm. With this cooperation we can anticipate equally prompt distribution in the future, but it is imperative that all papers be turned in at the annual meeting in their final form ready for publication. Forwarding of the completed manuscripts with illustrations prior to the annual meeting to the Annals Publishing Company has been an additional help in securing early publication. Last minute changes and corrections by the authors invariably delay the final form of the manuscript so that it is not available at the time of the annual meeting.

According to Council Directive, 250 copies of the 1968 Transactions were ordered and 108 books were sent from the publisher to the active and honorary members.

There were 48 standing orders for the Transactions and 18 additional single orders. Orders for 56 back issues were filled.

Complimentary volumes were sent to the honorary members, the Library of Congress, the National Library of Medicine for the Index Medicus and the families of the deceased members.

The cost of the 1968 Transactions was $3,413.89 which represented an increase of $236.07 over the previous year. The sale of the Transactions from January 1 to December 21, 1968, amounted to $1,420.00. Accounts outstanding amount to $120.00.

The increased receipts represented the sum collected from the 1967 Transactions mailed February 15, 1968, the 1968 Transactions mailed September 20, 1968, and a large order for back issues. It

does not represent an increase in sales for the calendar year 1968 and can be expected to diminish in succeeding years as the publication date becomes more standardized.

Respectfully submitted,
F. Johnson Putney, M.D., Editor

It was moved, seconded and duly passed that the report be accepted.)

REPORT OF THE AMERICAN BOARD OF OTOLARYNGOLOGY
REPORT TO THE AMERICAN LARYNGOLOGICAL ASSOCIATION
BY THE REPRESENTATIVES FROM THE
AMERICAN BOARD OF OTOLARYNGOLOGY

The President of the American Board of Otolaryngology for 1969 is Walter Work, M.D. and the Secretary is Dean Lierle, M.D. According to its records, approximately 6,127 men have been certified by the Board since its inception in 1924. Less than 1 percent are foreign graduates. It is estimated that 3,759 certified otolaryngologists are living today, although the American Medical Association gives a total of 5,430 who list themselves as specialists of whom 3,280 are Board certified and 2,150 are not Board certified. The discrepancy may be in the relatively few remaining who came in as "Eye, Ear, Nose and Throat" specialists. The increasing number of candidates has necessitated two examinations per year, consisting of oral and clinical (with patients) discussions with the members of the Board and associate examiners.

Two examinations were held in 1968, one in New Orleans, March 10 to 15, 1968 where 91 candidates were examined of whom 73 were granted certification and 18 were failed or conditioned. The second was held in Chicago, October 19 to 25, 1968, where 102 candidates were examined of whom 84 passed and 18 were conditioned or failed.

The "Guide Lines" concerning the examination have been revised during this past year (2 copies attached) with some significant changes:

1. Applicants trained by the preceptorship method will not be eligible for examination after July 1, 1969.

2. No time interval will be required between the completion of the residency program and making application for examination.
3. Each application must include a letter from the director of the residency program attesting to the satisfactory completion of the applicant's residency program or a part thereof.
4. An accepted application designates the candidate as "Board Eligible." The applicant remains "Board Eligible" for three years from the date of the mailing of the notification of acceptance by the Secretary-Treasurer of the Board. If, at the termination of this period of time, a candidate has failed to appear for examination, the application fee and "Board Eligible" status are forfeited.

The Board nominates four representatives to the 12-man Tripartite Residency Review Committee with equal representation from the American College of Surgeons and the American Medical Association. The four representatives from the Board are: David DeWeese ('69); G. O. Proud ('72); Burton Soboroff ('70); Harold G. Tabb ('71). Dr. Dean Lierle is the Secretary representing the American Board.

The following additional items of interest to the Fellows of the American Laryngological Association were discussed at the Board meeting on October 1968:

1. The members reaffirmed their policy in regard to Doctors of Osteopathy taking residency training in Otolaryngology: namely, "graduates of schools of osteopathy who do not hold M.D. degrees are not eligible for appointment in residencies in Otolaryngology approved by the Council on Medical Education and Hospitals of the American Medical Association."
2. The Board reaffirmed its position concerning nonmedical personnel performing major surgical procedures, including the statement of the Trustees of the A.M.A. defining the work of the oral surgeon as being limited to diseases of the teeth and jaws and lesions of contiguous soft tissues related to diseases of the teeth and jaws exclusive of malignancies. In this regard it is of interest to note that the American College of Surgeons formulated the following statement: "Pursuant to its ideals for optimum care of surgical patients, the Regents of the American College of Surgeons affirm

that major surgery involves total medical and surgical care of the patient. In order to provide such optimum surgical care, those who perform major surgical procedures should be doctors of medicine with adequate graduate education in surgery."

3. It was recommended that Dr. George Reed, as delegate to the House of Delegates of the A.M.A. for Otorhinolaryngology, continue to examine the pros and cons of the Quinn Report to determine what is best not only for Otolaryngology but also for the A.M.A. and other specialty groups.

Respectfully submitted,
Frank D. Lathrop, M.D.
Paul H. Holinger, M.D.

(On a motion duly made and seconded from the floor the report was accepted.)

REPORT FROM THE BOARD OF GOVERNORS, AMERICAN COLLEGE OF SURGEONS

The fifty-fourth annual meeting of the Board of Governors of the American College of Surgeons was held at the Shelburne Hotel in Atlantic City, New Jersey, on Sunday October 13, 1968. Dr. Charles W. McLaughlin, Jr., Chairman of the Board of Governors presided. Dr. Jonathan E. Rhoads, Chairman of the Board of Regents, reported that the number of Fellows in the College will exceed 30,000 as of this year and that the number of initiates in the surgical specialties surpassed the number of general surgeons. He also reported that a new Financial Consultant for the management of the pooled funds of the College has been engaged and early indications are that the performance of this fund has been improved. Although the dues were raised from 40 to 60 dollars two years ago, there probably will be a further increase in dues in 1970 due to constantly increasing costs.

Reports of the various study committees were presented. Dr. Curtis P. Artz, Chairman of the Study Committee on Supplemental Teaching Aids for Undergraduate Teaching, recommended a project involving the use of written and audio-visual aids to be offered to various universities for inclusion in their undergraduate teaching

programs. W. B. Saunders Company has agreed to undertake a pilot project of about three chapters in this area. It was moved, seconded and carried that the Board of Governors recommend to the Board of Regents that the College undertake this project of producing course material as outlined for teaching medical students.

Dr. William F. Hollister, Chairman of the Study Committee on the Problem of the Continuing Education of the Practicing Surgeon, stated that his Committee did not favor compulsory continuing education or re-examination of the practicing surgeon at this time. The Committee studies will be continued for another year.

Dr. Stanley O. Hoerr reported for the Study Committee on Means of Improving the Relations between the Board of Governors and Specialty Societies. The results of a questionnaire to the Governors of these Specialty Societies were reviewed. A majority of Governors believe that a closer relationship between their societies and the College is desirable. The Committee suggested that the Specialty Societies should send their representatives on the Board of Governors for more than one term to provide continuing contact; and that the Governor should always render an annual report to his Society.

During the afternoon the Governors met in eight separate groups to consider a variety of problems that interest the College as follows.

1. Continuing Education of the Practicing Surgeon.
2. What Should Be the Direction and Future of the American College of Surgeons.
3. How Can the College Help Guide the Public in the Selection of A Competent Surgeon.
4. Can Minimal Qualification Be Established for First Assistants in Major Surgery.
5. How Can the College Best Support the Association of Operating Room Nurses and Surgical Technicians.
6. How Can the College Best Support the Program of Its Fellows Outside the Continental United States and Canada.
7. Medical School Education. Should There Be A Second Look at the Present Day Methods of Selecting Medical Students?
8. How Can Surgeons More Effectively Contribute to An Improved Design of Operating Room Suites.

At 3:00 P.M. the Governors reconvened to hear reports from each of these discussion groups and voted to continue the discussion groups next year.

Your representative on the Board of Governors is a member of the Committee to Study The Problem of Continuing Education of the Practicing Surgeon and has also been appointed to the Committee to Study the Future Financial Requirements of the College.

Respectfully submitted,

W. Franklin Keim, M.D.

(It was moved, seconded and duly passed that the report be accepted.)

REPORT OF THE AMERICAN LARYNGOLOGICAL RESEARCH FUND COMMITTEE

A report of the activities of the American Laryngological Research Fund Committee was presented to the Council at the mid-winter meeting in New York, January 4, 1969. Only one communication has been received this year, and that was from Dr. J. Wind, University of Ziekenhuis of Amsterdam. The request stated he had completed his research study on the "Comparative Anatomy of the Larynx," and now he wished to receive a grant to "publish this monograph as a university thesis, and at the same time as a commercial edition." The amount requested was $2,000.00. The members of the committee were unanimous in questioning whether Laryngological Research Funds should be used for this purpose. A review of the purposes of this Fund indicates this request does not conform to the rules and regulations governing the Fund. The majority of the committee, therefore, voted not to support this request.

Respectfully submitted,

Francis E. LeJeune, M.D., Chairman

OLD BUSINESS

SECRETARY LATHROP: At the Mid-winter Council Meeting the Council proposed an amendment to the Constitution and By-Laws.

As noted in my Post-Council Letter to the Fellowship dated January 21, 1969, the Council voted to submit to the Fellowship at

the Annual Meeting a proposed change in the Constitution and By-Laws whereby Article VI, Section 10-b relating to presenting at the Annual Meeting of papers previously read at another meeting or published in nonotolaryngological journals might be presented "at the discretion of the Council."

Article VI, Section 10-b now reads:

> No paper shall be read before the Association which shall have been printed or been read before another body.

It is proposed that Article VI, Section 10-b be changed to read:

> No paper shall be read before the Association which shall have been printed or been read before another body excepting at the discretion of the Council.

(On a motion by Dr. Cracovaner seconded by Dr. Miller the change in Article VI, Section 10-b was unanimously approved.)

NEW BUSINESS

PRESIDENT DAVISON: At the Council Meeting two days ago the question was brought up of having a History Committee to prepare a Centennial volume. This is our Ninetieth Meeting, so it is only nine more years until we will be celebrating our Centennial. Since Dr. Boies suggested the desirability of having a Centennial volume, with which we all heartily agreed, he was named Chairman. He very graciously accepted.

As you know, Dr. Boies did a great deal of work in preparing the Centennial volume for the Otological Society, which is beautifully done.

We have named to the Committee thus far Dr. Francis Lederer, Dr. George Reed and Dr. Lathrop ex-officio. I think this requires action as to whether or not we do wish to have a History Committee, which implies that we are planning to have a Centennial volume.

(Dr. Holinger moved that the History Committee be appointed to proceed with plans for a Centennial volume. The motion was seconded by Dr. Fearon and unanimously passed.)

Our Constitution and By-Laws have not been revised for ten years, and there have been a number of changes which should be

included in the published volume. Our current edition is exhausted. Dr. Alfaro has accepted the position as Chairman and Dr. Goodale has agreed to serve on this Committee with Dr. Lathrop ex-officio.

DR. KEIM: I just wondered whether this was going to be a full-dress revision or is this just incorporating the amendments that have been passed into the newly printed edition.

PRESIDENT DAVISON: As things stand, this will simply bring up to date the things that you, as the membership, have approved, and put it in orderly fashion.

This Committee also is entitled to make recommendations and in the normal course of events, those recommendations have to be approved by the Council and then presented to the membership before they become official. As a result, there will be no independent action by this Committee except to make recommendations.

(On a motion by Dr. LeJeune seconded by Dr. Daly the matter of revision of the Constitution and By-Laws was approved.)

PRESIDENT DAVISON: The matter was brought up of having a certificate of membership. As you may know, the other societies, including the American Otological Society, have a certificate of membership, but the American Laryngological does not. I don't know that it has even been brought up before.

The Council rather approved the idea of having a certificate of membership, so we are bringing it up for your consideration.

LOUIS H. CLERF, M.D.: I should like to indicate that we have no "members" in this Association. They are all "Fellows." I have seen times when the lecturer speaks of the "members." We have been told for a long time there are no "members," there are "Fellows," so let us have a "Certificate of Fellowship."

PRESIDENT DAVISON: We certainly thank Dr. Clerf for his very proper recommendation, "Certificate of Fellowship." (Dr. Cracovaner moved and Dr. Fearon seconded that a Certificate of Fellowship be prepared. The motion was duly carried.)

SECRETARY LATHROP: Just one point on this Certificate of Fellowship: this will be issued to all new Fellows, but, according to my understanding, if the old Fellows wish a Certificate, it will have to be directed to my attention so it can be prepared.

Now, would you like to have that or shall we make this retroactive?

PRESIDENT DAVISON: It is the general consensus that this "Certificate of Fellowship" be made retroactive.

DR. SALINGER: Has any thought been given to issuing a new Index, bringing the material since the last Index was issued up to date? I don't remember what date that was but it was some years ago.

PRESIDENT DAVISON: I remember clearly because I spent many hours making up the last one in 1963, and Dr. Proetz prepared the volume in 1943. Dr. Broyles did a ten-year Index in 1953. At that time the Council said, "Why don't we just have an Index in the back of each volume?"

DR. PUTNEY: That has been done since Dr. Davison brought it up to date in 1963. If you will notice, each one of the subsequent Transactions has an index each year. We had thought that perhaps in another ten-year period (1973) a ten-year summary might be made, but each volume contains an index, so it is current from year to year.

JOSEPH P. ATKINS, M.D.: Just a question of clarification about the Committee on Laryngological Research Fund. Does this reading of the By-Laws indicate that Canada is the fifty-first state, so far as eligibility for research funds is concerned?

PRESIDENT DAVISON: So long as we have active Canadian Fellows, I think that this includes North America.

HENRY L. WILLIAMS, JR., M.D.: And Puerto Rico and Mexico.

PRESIDENT DAVISON: Dr. LeJeune, do you wish to have any further clarification or statement of policy?

DR. LEJEUNE: This reads: First, to make a survey of persons in this country with definite problems etc. The question that comes up is the interpretation of this particular sentence.

PRESIDENT DAVISON: I think properly this should be referred to the Committee on the Revision of the Constitution, since there is a slight divergence of opinion. With your permission, I will refer this to the Committee to be approved by Council action and brought to the Fellowship at the next Annual Meeting.

EXECUTIVE SESSION

MONDAY MORNING, MARCH 24, 1969

The meeting reconvened in Executive Session on Monday morning, March 24, 1969, in the Grand Ballroom at twelve-two o'clock, immediately following the adjournment of the Scientific Session, President Davison presiding.

(On a motion by Dr. LeJeune duly seconded from the floor the new Council was unanimously elected.)

PRESIDENT DAVISON: Will the new Council convene here at the platform? (Meeting of new Council.)

SECRETARY LATHROP: The Council has organized and selected:

President: Dr. Alden Miller

First Vice President: Dr. Joseph Atkins

Second Vice President: Dr. Stanton Friedberg

Secretary: Dr. Frank Lathrop

Treasurer: Dr. Charles Norris

Librarian: Dr. F. Johnson Putney

First Councilor: Dr. Paul Holinger

Second Councilor: Dr. Francis Sooy

Third Councilor: Dr. Lawrence Boies

Fourth Councilor: Dr. Francis Davison

PRESIDENT DAVISON: Alden, it gives me great pleasure to turn over to you the historic gavel and the symbol of office. This was presented to the Association in 1911. We hope you will take care of it carefully—I personally kept it in the First National Bank!

We know you have the know-how, the ability and the energy to ably conduct the affairs of the Association for the coming year. We're happy to have you take over.

PRESIDENT MILLER: Thank you, gentlemen, very much. It is awfully hard to try to find words to say how you feel about something like this. Maybe what helped me is what one of the Fellows said, "Well, Al, this caps your career." Well, that to me is so true.

I can't think of anything in the world that is greater than this Fellowship and this job and I just hope that I can fill it like the other Fellows have before.

I can't help but tell you a wee bit about what this Society means to me, and I know means to the rest of you.

There aren't very many of us out on the West Coast. I can remember Dr. J. Mackenzie Brown saying there just was nothing else in this world except the American Laryngological Association, and I can always remember when Colby Hall and Lou Morrison were elected to the Fellowship what a great thing it was to them and that it has been to me, and again I think you very much.

At this time, I want to try to tell Dr. Davison what I think you have all felt for the wonderful job that he has done in having given us this fabulous program, both scientific and social, for the last two days and last night.

On behalf of the Fellowship, Dave, here is the Past President's Key to try to remind you how wonderful we think your job has been.

(The meeting was adjourned at twelve-ten o'clock.)

§

THE NEWCOMB AWARD

The Newcomb Award is presented each year by the American Laryngological Association to a Fellow who has made outstanding contributions to the literature of Laryngology and/or as a mark of esteem and appreciation for services to the Association.

In research, teaching and patient service as well as in meaningful contribution to the laryngological literature, Dr. John Kirchner, New Haven, Connecticut, is eminently qualified for the Award.

The Newcomb Award is presented to Dr. Kirchner with the sincere congratulations of the Fellows in the Association on this well deserved honor.

List of Fellows

LIST OF FELLOWS

Date Indicates Year Admitted to Active Fellowship

Attendance at meetings is shown by figures following each name.
Asterisk (*) indicates year paper was read.

Active Fellows

1962. Alexander, Fred W., 10515 Carnegie Ave., Cleveland 6, Ohio, 1962, '63*, '64, '66, '68, '69.

1958. Alfaro, Victor R., 917 - 20th St. N. W., Washington 6, D.C., 1958, '59, '60, '61, '62, '63, '64, '65, '66, '67, '69.

1969. Andrews, Albert H., 122 S. Michigan Ave., Chicago, Ill.

1962. Arnold, Godfrey E. T., 2500 N. State St., Jackson 6, Mississippi, 1962, '63*, '64*, '66, '69.

1958. Atkins, Joseph P., 3400 Spruce St., Philadelphia 4, Pa., 1958, '59*, '60, '61, '62, '63, '64, '65, '67, '68, '69.

1969. Ausband, John R., Bowman Gray School of Medicine, Winston-Salem, N. C.

1956. Baker, Daniel Clifton, Jr., 180 Fort Washington Ave., New York 21, N.Y., 1956, '57, '58, '59*, '60, '61, '62*, '63, '64, '65, '66, '69*.

1963. Baxter, James D., 1509 Sherbrooke St. West, Montreal, P.Q., Canada, 1963, '65*, '66, '67, '68, '69.

1944. Boies, Lawrence R., University Hospital, Minneapolis 14, Minnesota, 1944, '46, '47, '48*, '49, '50*, '51*, '53, '54, '55, '56, '57, '58, '59, '60, '61, '62, '63, '64, '65, '66, '67, '68*, '69.

1955. Bordley, John E., Johns Hopkins Hospital, Baltimore 5, Maryland, 1955, '56, '57, '59, '60, '62, '63, '64, '65, '66, '67, '69.

1959. Brewer, David W., 1100 E. Genesee St., Syracuse 10, N. Y., 1959, '60*, '61, '62*, '63*, '66.

1955. Brown, Lester Alexander, 490 Peachtree Street, N.E., Atlanta 8, Georgia, 1955, '56, '57, '58, '59, '61, '63, '65, '68, '69.

1940. Broyles, Edwin N., 11 East Chase St., Baltimore 2, Maryland, 1940, '41*, '42, '46, '48*, '49, '50, '51*, '52, '53, '54, '55, '56, '58, '59*, '60, '61*, '63, '64, '65, '67.

1963. Bryce, Douglas P., 170 St. George St., Room 320, Toronto, Canada, 1963*, '64, '66*, '67*, '68*, '69.

1968. Chandler, J. R., 1700 N.W. 10th Ave., Miami, Fla., 1968, '69*.

1967. Cocke, Edwin W., 899 Madison, Memphis, Tenn., 1967, '68, '69.

1964. Cody, Claude C., III, 1600 Memorial Professional Building, Houston, Texas, 1964, '65, '67, '69.

1957. Conley, John J., 139 East 36th Street, New York 16, N. Y., 1958*, '60*, '61, '62*, '63*, '64, '65.

1957. Converse, John Marquis, 722 Park Avenue, New York 21, N. Y., '60, '62*, '67.

1959. Cracovaner, Arthur J., 103 E. 78th St., New York 21, N. Y., 1959, '60, '61*, '62, '63, '65, '66, '67, '68, '69.

1960. Daly, John F., Faculty Practice Offices, 566 First Ave., New York 16, N. Y., 1960, '61*, '62, '63, '64, '65, '66, '67, '68, '69.

1951. Davison, Francis W., Geisinger Medical Center, Danville, Pa., 1951, '52, '53*, '54, '55, '56, '57, '58, '59, '60, '61, '62, '63*, '64, '65, '66, '67*, '68, '69*.

1966. Devine, Kenneth D., Mayo Clinic, Rochester, Minn., 1966, 1948*, '49, '51, '53, '54, '56, '57, '59, '62, '64, '65*, '66, '67, '68.

1968. DeWeese, David D., 3181 S.W. Sam Jackson Park Rd., Portland, Ore., 1968, '69*.

1969. Dickinson, John T., 1501 Locust St., Pittsburgh, Pa.

1952. Erich, John B., Mayo Clinic, 102 Second Avenue, Rochester, Minn. 1953, '56*, '57, '65.

1968. Farrior, Richard T., 509 Bay St., Tampa, Fla., 1968, '69.

1964. Fearon, Blair, Medical Arts Building, Toronto 5, Ontario, Canada, 1964, '66, '67, '68, '69.

1963. Ferguson, Charles F., 300 Longwood Ave., Boston 15, Mass., 1963, '64*, '66, '67*, '68, '69*.

1955. Fitz-Hugh, Glassell Slaughter, 2003 Spottswood Road, Charlottesville, Virginia, 1955, '57, '59, '60, '61, '62*, '64, '65, '66, '67*, '68, '69.

1956. Friedberg, Stanton A., 1725 West Harrison St., Chicago, Ill., 1956, '57*, '58, '59, '60, '61, '62, '63, '64, '65*, '67, '68, '69.

1959. Goldman, Joseph L., 1050 Park Ave., New York, N. Y., 1959, '60*, '61, '62, '63, '64, '65, '66, '67, '68, '69.

1940. Goodale, Robert L., 42 Fresh Pond Lane, Cambridge, Mass., 02138, 1940, '41, '42*, '44*, '46, '47, '49, '50*, '53, '54, 55*, '56, '57*, '60, '61*, '62, '63, '64, '65*, '68, '69.

1965. Hanckel, Richard W., 55 Doughty St., Charleston, S. C., 1965, '66, '67, '69.

1957. Harrill, James A., Bowman Gray School of Medicine, Winston-Salem, N. C., 1957, '58, '59, '61, '62, '63, '64, '66, '67, '68, '69.

1960. Harris, Herbert, 907 Hermann Professional Building, Houston 25, Texas, 1960, '61*, '62, '63, '64, '65*, '66, '67, '68, '69.

1959. Hart, Verling K., 1600 East Third St., Charlotte, N. C. 28204, 1959, '60, '62, '66, '68, '69.

1959. Henry, G. Arnold, 170 St. George St., Toronto, Canada, 1959, '61, '63, '66, '67, '69.

1943. Hilding, Anderson C., Research Laboratory, St. Luke's Hospital, Duluth 11, Minnesota, 1944*, '46, '47, '48*, '49, '50, '51, '53, '55, '56*, '57, '60, '61, '62*, '63, '64*, '65, '66, '67, '68, '69.

1955. Hilger, Jerome A., Suite 6A, 393 Dunlap St., St. Paul, Minnesota 55104, 1955, '56*, '57, '58, '59, '60, '62, '63, '64, '65, '66, '68, '69.

1945. Holinger, Paul H., 700 North Michigan Ave., Chicago 11, Illinois, 1946, '47, '49, '50*, '51*, '52, '53, '54*, '55, '56, '57, '58*, '59, '60, '61*, '62*, '63, '64, '65, '66*, '68, '69*.

1957. HOLMES, EDGAR M., 2000 Washington St., Newton, Mass. 02164, 1957, '59, '61, '63, '68.

1944. HOOPLE, GORDON D., 1100 East Genesee St., Syracuse 10, New York, 1946, '47, '48, '49, '50, '51, '52, '53, '54*, '55, '56, '58, '59, '60, '61, '62, '63, '64, '65, '66, '67, '68, '69.

1952. IRELAND, P. E., Room 82, Banting Institute, University of Toronto, Toronto, Ontario, 1952, '53, '54, '55, '56*, '57, '59, '61, '62, '63, '64, '66, '68.

1961. JONES, EDLEY H., 1301 Washington St., Vicksburg, Miss. 39108, 1961, '62, '63*, '65, '66, '67, '68, '69.

1964. KEIM, W. FRANKLIN, 75 Union St., Montclair, N. J., 1964, '66, '67, '68*, '69.

1965. KING, JAMES T., Baptist Professional Building, Atlanta, Ga., 1965, '66, '67, '69.

1963. KIRCHNER, JOHN A., 333 Cedar St., New Haven 4, Conn., 1963, '65*, '67*, '68, '69*.

1965. KNIGHT, JOHN S., 305 West 43rd St., Kansas City, Mo., 1965, '66*, '67, '69.

1963. LATHROP, FRANK D., 605 Commonwealth Ave., Boston 15, Mass., 1963, '64*, '67, '67*, '69.

1953. LEDERER, FRANCIS LOEFFLER, 1855 W. Taylor St., Chicago, Illinois 60612, 1953, '54, '55, '56, '57*, '58, '60, '61, '62, '63*, '64, '65, '66, '68*, '69

1936. LEJEUNE, FRANCIS ERNEST, Ochsner Clinic Building, Prytania and Aline Streets, New Orleans 15, Louisiana, 1937, '46*, '47, '50, '51, '52, '53, '54*, '55, '56, '57, '58, '59, '60, '61, '62, '63*, '64, '65, '66, '67, '68, '69.

1966. LEWIS, JOHN S., 3 East 71st Street, New York 21, N. Y., 1966, '67, '68, '69*.

1961. LEWY, ROBERT B., 25 East Washington St., Chicago 2, Ill., 1961, '62*, '63, '65*, '67, '68, '69.

1966. MCCABE, BRIAN F., Dept. of Otolaryngology, University Hospital, Iowa City, Iowa, 1966, '69.

1963. MCGOVERN, FRANCIS H., 139 South Main St., Danville, Virginia, 1963, '64, '66, '68.

1951. McHenry, Lawrence C., 301 N. W. 12th St., Oklahoma City 2, Okla., 1953*, '62, '63, '64, '65, '66, '68.

1931. McNally, William James, 1509 Sherbrooke Street West, Montreal, Canada, 1931, '33, '34*, '39, '44, '47, '49, '52, '53, '54*, '55, '56, '57, '58, '59, '60, '61, '65, '67*.

1952. Miller, Alden H., 1407 N. Vermont Ave., Los Angeles 27, Calif., 1952, '53*, '55, '57, '58*, '61, '62, '64, '66, '67*, '68, '69.

1965. Miller, Daniel, Deaconess Medical Building, 110 Francis St., Boston, Mass., 1965, '66, '67, '68, '69*.

1964. Montgomery, William W., 243 Charles St., Boston 14, Mass., 1964, '65*, '66*, '68*.

1958. Montreuil, Fernand, Hospital Notre Dame, 1560 E. Sherbrooke, Montreal 24, Canada, 1958, '60, '64, '67, '69.

1954. Moore, Paul M., 10515 Carnegie Ave., Cleveland 6, Ohio, 1954, '55*, '56, '57, '59, '60, '61, '62, '63, '64, '65, '66, '68.

1957. Munoz-MacCormick, Carlos Eduardo, 71 Park Terrace, Santurce, Puerto Rico, 1957, '58, '59, '60, '61, '62, '63, '64, '65, '66, '67, '68, '69.

1953. Murtagh, John A., 2 Maynard Street, Hanover, New Hampshire, 1953, '54, '55, '56*, '57, '58, '59, '61, '62, '63, '65, '66, '67, '69.

1937. Nash, C. Stewart, Medical Arts Building, Rochester 7, New York, 1937, '38*, '40, '42, '44, '46, '49, '50, '51, '52, '53, '54, '55, '56*, '58, '59, '60, '61, '62, '64.

1956. Norris, Charles Morgan, 3401 North Broad Street, Philadelphia 40, Pennsylvania, 1956, '57, '58*, '59, '60, '61*, '62, '63, '64, '66, '67, '68*, '69.

1961. Ogura, Joseph H., 640 So. Kingshighway, St. Louis 10, Mo., 1961*, '62*, '63, '64*, '65, '66*, '67, '68*, '69.

1958. O'Keefe, John J., 255 S. 17th St., Philadelphia 3, Pa., 1958, '59*, '60, '62, '64, '67.

1961. O'Neil, James J., 612 Medical Arts Building, Omaha, Nebraska, 1961, '62*, '64, '67, '69.

1961. Pang, Lup Q., 1374 Nuuanu Ave., Honolulu 17, Hawaii, 1961, '62*, '64, '66, '67.

1961. PASTORE, PETER N., 5503 Riverside Drive, Richmond 25, Va., 1961, '62*, '64, '66, '67, '69.

1967. PRATT, LORING W., 177 Main St., Waterville, Maine, 1967, '68, '69.

1951. PRIEST, ROBERT E., 79 S. Ninth St., Minneapolis 2, Minn., 1951, '53*, '54*, '56, '59, '60*, '61, '65, '67*, '68, '69.

1951. PUTNEY, F. JOHNSON, 80 Barre St., Charleston, S. C. 29401, 1951, '52, '53*, '54, '55, '56, '58*, '59, '60, '61, '62, '63*, '64*, '65, '66, '67, '68*, '69*.

1963. REED, GEORGE F., 766 Irving Ave., Syracuse, N. Y., 1963, '65*, '66, '68, '69*.

1956. RICHARDSON, JOHN ROBERT, P.O. 52, Searsport, Maine 04974, 1957, '58, '59, '60, '63, '64, '67, '69.

1959. SANDERS, SAM H., 1268 Peabody Ave., Memphis 3, Tenn., 1959, '60, '61, '62, '63, '65.

1964. SAUNDERS, WILLIAM H., N-820 University Hospital, Columbus, Ohio, 1964, '65, '66, '68*, '69.

1959. SILCOX, LOUIS E., 102 Lankenau Med. Bldg., Philadelphia 31, Pa., 1959, '60, '61, '62, '63, '64, '65, '66, '67, '68, '69.

1958. SIMONTON, KINSEY M., Mayo Clinic, Rochester, Minn., 1958, '59*, '62, '64, '66, '67, '69.

1962. SISSON, GEORGE A., Northwestern U. Medical School, 303 Chicago Ave., Chicago, Ill. 60611, 1962, '63*, '65, '66, '68, '69.

1964. SKOLNIK, EMANUEL M., 1853 West Polk Street, Chicago, Ill., 1964*, '65, '66*, '67, '68, '69.

1954. SOOY, FRANCIS A., University of California Hospital, 3rd and Parnassus Ave., San Francisco, Calif., 1954, '56*, '58, '60, '62, '63, '64, '65*, '67, '68*

1961. SUEHS, OLIVER W., 14 Medical Arts Square, Austin, Texas, 1961, '62*, '63, '64, '66, '68, '69.

1963. TABB, HAROLD G., 1430 Tulane Ave., New Orleans 12, La., 1963, '65, '66, '67, '69.

1954. THORNELL, WILLIAM C., 2508 Auburn Avenue, Cincinnati 19, Ohio, 1955*, '57, '59, '60, '63, '68.

1963. Tolan, John F., 1118 Ninth Ave., Seattle 1, Washington, 1963, '64, '65, '66, '67, '69.

1967. Trible, William MacL., 1234 19th St., N.W., Washington, D.C., 1967, '68, '69*.

1954. Walsh, Theodore E., 640 S. Kingshighway Blvd., St. Louis 10, Missouri, 1954, '55*, '56, '57, '58, '59, '60, '61, '62, '63, '64, '65, '66, '67, '68, '69.

1962. Weaver, Delmar F., 571 Fisher Building, Detroit 2, Michigan, 1962, '63, '64*, '65, '67*, '69.

1940. Weille, Francis L., 247 Commonwealth Avenue, Boston 16, Massachusetts, 1940, '41, '42, '44, '46*, '47, '48, '49, '50, '51, '52, '53, '54, '55, '56*, '57, '58, '59, '60, '61, '62, '63, '64, '65*, '66, '67, '68, '69.

1951. Woodman, DeGraaf, Talbottown Lane, P.O. Box 560, Easton, Md., 1951, '53, '54, '55*, '56, '58, '59, '60, '61, '62, '63, '64, '65, '66, '67, '68, '69.

1953. Work, Walter P., University Hospitals, Ann Arbor, Michigan, 1953, '54, '56, '58, '59*, '62, '63*, '65, '67, '68, '69.

Honorary Fellows

1946. Alonso, Regules Justo E., Mercedes 1233, Montivideo, Uruguay.

1957. (1925) Clerf, Louis H., 5575 Eighth Avenue, North, St. Petersburg 2, Florida, 1957, '58, '59, '60, '61, '63, '64, '65, '66, '68, '69.

1968. (1936) Galloway, Thomas C., 636 Church St., Evanston, Ill., '69.

1960. (1928) Hill, Frederick Thayer, Thayer Memorial Hospital, Waterville, Maine, 1960, '61, '63, '64, '66, '67, '68, '69.

1957. Huizinga, Eelco, Noorderhaven 7, Groningen, The Netherlands, 1957.

1967. (1933) Lierle, Dean M., University Hospitals, Iowa City, Iowa, 1967, '68

1957. (1930) Negus, Sir Victor E., Hill Farm Lodge, Haslemere, Surrey, England, 1957*.

1966. (1957) ONO, JO, 75 Zaimoku-cho, Azabu, Minato-ku, Tokyo, Japan.

1937. PORTMANN, GEORGES, 25 bis Cours de Verdun, Bordeaux, France, 1947.

1957. RUEDI, LUZIUS, Ramistrasse 100, Zurich, Switzerland, 1957.

Corresponding Fellows

1959. BARRETTO, PLINIO DE MATTOS, Ru Cons Torres, Homer 371, Sao Paulo, Brazil, 1960, '69.

1959. BATEMAN, GEOFFREY H., 55 Harley Street, London, W. 1, England.

1942. BATSON, OSCAR V., 203 South DeKalb St., Philadelphia 4, Pennsylvania, 1942*, '54*

1968. CAWTHORNE, SIR TERENCE, BT. 149 Harley St., London, W. 1, England, 1968.

1964. CLEVES, CARLOS, Carrera 14 No. 44-54 Apto. 202, Bogota, Colombia, S. A.

1966. DOHLMAN, GOSTA, Dept. of Otolaryngology, University of Toronto, Toronto, Canada, 1966.

1943. EGGSTON, ANDREW A., 653 Park Avenue, New York, N. Y., 1944, '49, '55, '60, '61*.

1968. NAKAMURA, FUMIO, Kyoto Prefectural U. Medicine, Kawara-Mach 1, Kamikyo-Ku, Kyoto, Japan

1964. PEREZ, ALFREDO C., Institito Celis Perez, Avenida Montes Deoca, Apartado 163, Velencia, Venezuela, S. A., 1965.

1924. SCHAEFER, J. PARSONS, 4634 Spruce Street, Philadelphia 39, Pennsylvania, 1934.

1952. TAPIA ACUNA, RICARDO, Av. Insurgentes Sur 300, Mexico 7 D.F., 1953, '57, '60*, '67.

1936. VIALLE, JACQUES, 27 Rue de France, Nice (Alpes Maritimes), France.

Emeritus Fellows

1951. (1944) BEATTY, HUGH G., 390 Oak St., Apt. No. 35, Columbus 15, Ohio, 1951, '52, '53.

1966. (1945) CAMPBELL, PAUL A., 321 Park Hill Drive, San Antonio, Texas.

1965. (1942) CANFIELD, NORTON, Harwood Hospital, Christiansted, St. Croix, Virgin Is.

1969. (1959) CARDWELL, EDGAR P., 2415 Oleander Drive, Wilmington, N. C.

1969. (1947) EAGLE, WATT W., 703 Professional Drive, New Bern, N. C.

1958. (1930) FIGI, FREDERICK A., Mayo Clinic, Rochester, Minnesota.

1969. (1932) FURSTENBERG, ALBERT C., St. Joseph's Mercy Hospital, Ann Arbor, Mich.

1963. (1928) GOODYEAR, HENRY M., 2012 Edgecliff Point, Cincinnati, Ohio 45206.

1960. (1940) HANSEL, FRENCH, 634 N. Grand Blvd., St. Louis 3, Missouri, 1962, '65.

1968. (1942) HEATLY, CLYDE A., 11 North Goodman St., Rochester 7, N. Y., 1942, '46*, '49, '50, '52, '53*, '54, '55, '57, '58, '60, '61, '65.

1961. (1950) KLINE, ORAM R., 896 Lake Ave., Woodbury Heights, N. J. 08097, 1961, '63.

1967. (1949) LINDSAY, JOHN R., 950 E. 59th St., Chicago 37, Ill., 1967.

1954. (1927) LUKENS, ROBERT M., 141 West Heather Road, Wildwood Crest, N. J.

1964. (1952) MCCALL, JULIUS W., 610 Rose Building, Cleveland 15, Ohio, 1964, '66, '68, '69.

1966. (1933) MCMAHON, BERNARD J., 150 North Meramec Ave., Clayton, Mo.

1959. (1927) MYERSON, MERVIN C., 436 Roxbury Drive, Beverly Hills, Calif.

1957. (1948) PHELPS, KENNETH A., 7200 34th St., South, St. Petersburg, Fla.

1969. (1951) RAWLINS, AUBREY G., 384 Post St., San Francisco, Calif.

1958. (1938) SALINGER, SAMUEL, 1260 East Alejo Road, Palm Springs, Calif., 1958, '59, '61, '62, '63, '64, '65, '66. '67, '68, '69.

1962. (1932) SCHALL, LEROY A., Scudder Lane, Barnstable, Mass., 1964, '66, '68, '69.

1964. (1934) SCHENCK, HARRY P., 326 South 19th Street, Philadelphia 3, Pa., 1964, '65*, '66, '68, '69.

1965. (1939) SIMPSON, W. LIKELY, 351 South Highland St., Memphis, Tenn.

1969. (1950) SMITH, AUSTIN T., 330 S. 9th St., Philadelphia, Pa.

1966. (1947) THEOBALD, WALTER H., 307 N. Michigan Ave., Chicago 11, Ill., 1966, '68, '69.

1968. (1950) TREMBLE, G. EDWARD, 1390 Sherbrooke St. W., Montreal, Quebec, Canada, 1950, '51, '52, '53, '54, '55*, '56, '57, '58, '60, '61, '62, '63, '65, '66, '67*, '68, '69.

1965. (1943) VAN ALYEA, OLIVER E., 135 South LaSalle St., Chicago 3, Ill., 1965, '66, '68, '69.

1961. (1941) VIOLE, PIERRE P., 241 South Windsor, Los Angeles, Calif., 1964.

1964. (1948) WILLIAMS, HENRY L., II, U. S. Veterans' Hospital, Minneapolis 17, Minn., 1964, '65, '66, '67, '68, '69*.

1951. (1935) WOODWARD, FLETCHER D., 1326 Rugby Road, Charlottesville, Va., 1959, '60, '61.

BENEFACTORS

Dr. John F. Barnhill
Dr. Gordon Berry
Dr. Edwin N. Broyles
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Dr. & Mrs. Lee Wallace Dean
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Dr. William E. Grove
Dr. Gordon F. Harkness
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Dr. & Mrs. George E. Hourn
Dr. Samuel Johnston
Dr. James E. Newcomb
Dr. Henry B. Orton
Dr. & Mrs. Lyman G. Richards
Dr. Samuel Salinger
Dr. Sam H. Sanders
Dr. Harry P. Schenck
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1951. Dr. Arthur W. Proetz

1954. Dr. Louis H. Clerf

1959. Dr. Albert C. Furstenberg

1960. Dr. Dean M. Lierle

1961. Dr. Frederick T. Hill

1966. Dr. Paul H. Holinger

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1929. Dr. Edward H. Campbell

1931. Dr. Arthur W. Proetz

1934. Dr. Anderson C. Hilding

1936. Dr. Francis E. LeJeune: Dr. Joel J. Pressman

1939. Dr. H. Marshall Taylor: Dr. Brien T. King

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1941. Dr. Noah D. Fabricant

1946. Dr. Paul H. Holinger

1949. Dr. Henry B. Orton

1962. Dr. Hans von Leden

1966. Dr. John A. Kirchner and Mr. Barry D. Wyke

1968. Dr. Joseph H. Ogura

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1952. Dr. William J. McNally
1953. Dr. Frederick T. Hill
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1955. Dr. Thomas C. Galloway
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1959. Dr. Harry P. Schenck
1960. Dr. Joel J. Pressman
1961. Dr. Chevalier L. Jackson
1962. Dr. Paul H. Holinger
1963. Dr. Francis E. LeJeune
1964. Dr. Fred W. Dixon
1965. Dr. Edwin N. Broyles
1966. Dr. Lyman G. Richards
1967. Dr. Joseph H. Ogura
1968. Dr. Walter P. Work
1969. Dr. John A. Kirchner

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(Year of Election)

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1885. Harrison Allen
1886. E. Fletcher Ingals
1887. R. P. Lincoln
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1889. J. N. Mackenzie
1890. W. C. Glasgow
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1917. Thomas H. Halstead
1918. Cornelius G. Coakley
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1920. Harris P. Mosher
1921. Harmon Smith
1922. Emil Mayer
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1924. Lee Wallace Dean
1925. Greenfield Sluder
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1930. Francis R. Packard
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1934. Dunbar Roy
1935. Burt R. Shurly
1936. William B. Chamberlain
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1939. James A. Babbitt
1940. Gordon Berry
1941. Thomas E. Carmody
1942-3. Charles J. Imperatori
1944-5. Harold I. Lillie
1946. Frank R. Spencer
1947. Arthur W. Proetz
1948. Frederick T. Hill
1949. Ralph A. Fenton
1950. Gordon B. New
1951. H. Marshall Taylor
1952. Louis H. Clerf
1953. Gordon F. Harkness
1954. Henry B. Orton
1955. Bernard J. McMahon
1956. LeRoy A. Schall
1957. Harry P. Schenck
1958. Fred W. Dixon
1959. William J. McNally
1960. Edwin N. Broyles
1961. Dean M. Lierle
1962. Francis E. LeJeune
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1964. Albert C. Furstenberg
1965. Paul H. Holinger
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1967. Lawrence R. Boies
1968. Francis W. Davison

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1885. H. A. Johnson
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J. O. Roe
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S. Johnston
W. H. Daly
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S. W. Langmaid
C. E. DeM. Sajous
C. E. Bean
J. H. Hartman
S. Johnston
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W. E. Casselberry
S. H. Chapman
A. W. deRoaldes
D. N. Rankin
H. S. Birkett
F. W. Hinkel

1899.	T. A. DeBlois	M. R. Brown
1900.	H. L. Wagner	A. A. Bliss
1901.	J. W. Gleitsmann	D. Braden Kyle
1902.	G. A. Leland	T. Melville Hardie
1903.	J. H. Lowman	W. Peyre Porcher
1904.	Thomas Hubbard	W. J. Freeman
1905.	J. L. Goodale	C. W. Richardson
1906.	G. H. Makuen	A. R. Thrasher
1907.	J. P. Clark	J. E. Rhodes
1908.	E. Mayer	F. R. Packard
1909.	C. G. Coakley	H. P. Mosher
1910.	Robert C. Myles	J. M. Ingersoll
1911.	F. C. Cobb	B. R. Shurly
1912.	A. W. Watson	W. Scott Renner
1913.	F. E. Hopkins	Geo. F. Shambaugh
1914.	Clement T. Theisen	Lewis A. Coffin
1915.	J. Gordon Wilson	Christian R. Holmes
1916.	Thomas H. Halsted	Greenfield Sluder
1917.	John Edwin Rhodes	D. Crosby Greene
1918.	G. E. Shambaugh	John R. Winslow
1919.	Francis R. Packard	Harmon Smith
1920.	Harmon Smith	W. B. Chamberlin
1921.	Dunbar Roy	Robert C. Lynch
1922.	George Fetterolf	Lorenzo B. Lockard
1923.	Hubert Arrowsmith	Joseph B. Greene
1924.	Ross H. Skillern	Gordon Berry
1925.	John E. Mackenty	Robert Levy
1926.	Lewis A. Coffin	William V. Mullin
1927.	Charles W. Richardson	Hill Hastings
1928.	Robert Clyde Lynch	Francis P. Emerson
1929.	William B. Chamberlin	Ralph Albert Fenton
1930.	Harris P. Mosher	James A. Babbitt
1931.	Joseph B. Greene	E. Ross Faulkner
1932.	Gordon Berry	Frank A. Spencer
1933.	E. Ross Faulkner	Thomas S. Carmody
1934.	Gordon B. New	Samuel McCullagh
1935.	Edward C. Sewall	H. Marshall Taylor

1936.	William P. Wherry	Harold I. Lillie
1937.	Frank R. Spencer	Bernard J. McMahon
1938.	Ralph A. Fenton	Frederick T. Hill
1939.	John H. Foster	Thomas R. Gittins
1940.	Charles H. Porter	Gordon F. Harkness
1941.	Arthur W. Proetz	Henry B. Orton
1942-3.	Harold I. Lillie	Dean M. Lierle
1944-5.	John J. Shea	Thomas C. Galloway
1946.	H. Marshall Taylor	C. Stewart Nash
1947.	John J. Shea	Frederick A. Figi
1948.	Henry B. Orton	Anderson C. Hilding
1949.	LeRoy A. Schall	Fletcher D. Woodward
1950.	W. Likely Simpson	Lyman G. Richards
1951.	Wm. J. McNally	Thomas C. Galloway
1952.	J. Mackenzie Brown	Edwin N. Broyles
1953.	Claude C. Cody	Daniel S. Cunning
1954.	James H. Maxwell	Clyde A. Heatly
1955.	Robert L. Goodale	Paul H. Holinger
1956.	Henry M. Goodyear	Robert E. Priest
1957.	Francis E. LeJeune	Pierre P. Viole
1958.	Charles Blassingame	Chevalier L. Jackson
1959.	James Maxwell	Oliver Van Alyea
1960.	Walter Theobald	Anderson Hilding
1961.	Julius W. McCall	P. E. Ireland
1962.	Paul M. Moore	Jerome A. Hilger
1963.	Paul M. Holinger	Lester A. Brown
1964.	G. Slaughter Fitz-Hugh	Daniel C. Baker
1965.	C. E. Munoz-MacCormick	Arthur J. Cracovaner
1966.	Lawrence R. Boies	G. Edward Tremble
1967.	John F. Daly	Stanton A. Friedberg
1968.	DeGraaf Woodman	John Murtagh

Secretaries and Treasurers

1879.	G. M. Lefferts	1895.	H. L. Swain
1882.	D. Bryson Delavan	1900.	P. E. Newcomb
1889.	C. H. Knight	1911.	Harmon Smith

Secretaries

1912. Harmon Smith
1918. D. Bryson Delavan
1919. J. M. Ingersoll
1920. George M. Coates
1933. William V. Mullin
1935. James A. Babbitt
1939. Charles J. Imperatori
1942. Arthur W. Proetz
1947. Louis H. Clerf
1952. Harry P. Schenck
1957. James H. Maxwell
1959. Lyman G. Richards
1968. Frank D. Lathrop

Treasurers

1912. J. Payson Clark
1923. George Fetterolf
1932. William V. Mullin
1933. James A. Babbitt
1935. Charles J. Imperatori
1939. Frederick T. Hill
1948. Gordon F. Harkness
1953. Fred W. Dixon
1958. Francis E. LeJeune
1962. Alden H. Miller

Librarians

1879. F. H. Bosworth
1883. T. R. French
1903. J. H. Bryan
1930. John F. Barnhill
1934. Burt R. Shurly
1935. George M. Coates

Librarian and Historian

1936. George M. Coates
1944. Louis H. Clerf

Librarian, Historian and Editor

1947. Harry P. Schenck
1952. Bernard J. McMahon
1955. Edwin N. Broyles
1960. Francis W. Davison
1964. F. Johnson Putney

PAST FELLOWS

Dates indicate original election to the Association

*Deceased

Honorary Fellows

*1908. Barnhill, John F., Miami Beach, Florida.
*1893. Birkett, Herbert S., Montreal, Canada.
*1878. Bosworth, Francke H., New York, N. Y.
*1917. Coates, George M., Philadelphia, Pa.
*1918. Dean, Lee Wallace, St. Louis, Mo.
*1881. Delavan, D. Bryson, New York, N. Y.
*1893. deRoaldes, Arthur W., New Orleans, La.
*1890. De La Sota Y. Lastra, Ramon, Seville, Spain.
*1923. Fenton, Ralph A., Portland, Oregon.
*1879. French, Thomas R., Brooklyn, N. Y.
*1880. Garcia, Senior Manuel, London, England.
*1903. Harris, Thos J., New York, N. Y.
*1907. Jackson, Chevalier, Schwenksville, Pa.
*1878. Johnston, Samuel, Baltimore, Md.
*1878. Lefferts, George Morewood, Katonah, N. Y.
*1914. Levy, Robert, Denver, Colo.
*1918. Lewis, Fielding O., Media, Pa.
*1883. Mackenzie, John N., Baltimore, Md.
*1881. Mackenzie, Sir Morel, London, England.
*1910. Masser, Ferdinand, Naples, Italy.
*1904. Mosher, Harris P., Marblehead, Mass.
*1910. Moure, J. J. E., Bordeaux, France.
*1937. Nager, F. R., Zurich, Switzerland.
*1918. Oliver, H. K., Boston, Mass.
*1906. Pierce, Norval Harvey, San Diego, Calif.
*1924. Proetz, Arthur C., St. Louis, Mo.
*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, N. Y.
*1890. Wright, Jonathan, Pleasantville, N. Y.

Corresponding Fellows

*1892. Browne, Lennox, London, England.
*1938. Blair, Vilray P., St. Louis, Missouri.
*1940. Colledge, Lionel, London, England.
*1901. Collier, Mayo, Kearsney, Abbey, Kent, England.
*1893. Desvernine, Carlos M., Havana, Cuba.
*1930. Emerson, Francis P., Franklin, Mass.
*1961. Faaborg-Andersen, Knud, Nykobing, Denmark.
*1936. Fraser, John S., Edinburgh, Scotland.
*1901. Grant, Sir James Dundas, London, England.
*1887. Cougenheim, A., Paris, France.
*1894. Holden, Edgar, Newark, N. J.
*1919. Kelly, Adam Brown, Helensburgh, Scotland.
*1881. Labus, Carlo, Milan, Italy.
*1950. Larsell, Olof, Portland, Oregon.
*1931. LaSagna, Francesco, Parma, Italy.
*1926. Law, Frederick M., New York, N. Y.
*1921. LeMaitre, Ferdinand, Paris, France.
*1902. Lermoyez, Marcel, Paris, France.
*1897. Luc, H., Paris, France.
*1896. MacDonald, Greville, Haslemere, Surrey, England.
*1894. MacIntyre, John, Glasgow, Scotland.
*1903. McBride, P., York, England.
*1920. McKenzie, Dan, London, England.
*1919. McKernon, James F., New Canaan, Conn.
*1880. Meyer, Wilhelm, Copenhagen, Denmark.
*1910. Moure, J. J. E., Bordeaux, France.
*1896. Mygind, Holger, Copenhagen, Denmark.
*1950. Neil, James Hardie, Auckland, New Zealand.

*1919. Paterson, Donald Rose, Cardiff, Wales
*1941. Patterson, Norman, Herts, England.
*1919. Rogers, John, Jr., New York, N. Y.
*1894. Sajous, C. E. DeM., Philadelphia, Pa.
*1896. Schmiegelow, Ernst, Copenhagen, Denmark.
*1946. Segura, Eliseo, Buenos Aires, Argentina.
*1940. Soto, E. Fernandez, Havana, Cuba.
*1881. Thornton, Pugin, London, England.
*1913. Turner, A. Logan, Edinburgh, Scotland.
*1880. Whistler, W. McNeil, London, England.
*1901. Wingrave, Wyatt, Lyme Regis, Dorset, England.
*1894. Wolfenden, R. Norris, Kent, England.

Emeritus Fellows

*1936. Ballenger, Howard C., Winnetka, Ill.
*1923. Barlow, Roy A., Nova Scotia, Canada.
*1915. Barnes, Harry Aldrich, Kingston, Mass.
*1928. Beck, Joseph C., Chicago, Illinois.
*1921. Berry, Gordon, Worcester, Mass.
*1941. Bowers, Wesley C., New York, N. Y.
*1901. Brown, J. Price, Toronto, Canada
*1891. Bryan, Joseph H., Washington, D. C.
*1913. Butler, Ralph, Philadelphia, Pa.
*1930. Campbell, Edward H., Philadelphia, Pa.
*1897. Clark, J. Payson, Boston, Mass.
*1899. Cobb, Frederick C., Bradenton, Florida.
*1939. Cody, Claude C., Jr., Houston, Texas.
*1905. Coffin, Lewis A., New York, N. Y.
*1893. Coolidge, Algernon, Boston, Mass.
*1937. Crowe, Samuel J., Baltimore, Maryland.
*1941. Cunning, Daniel S., New York, N. Y.
*1913. Dabney, Virginius, Washington, D. C.
*1882. De Blois, Thomas Amory, Boston, Mass.
*1941. Dixon, Fred W., Shaker Heights, Ohio.
*1892. Farlow, John W., Boston, Mass.

*1922. Forbes, Henry H., New York, N. Y.
*1933. Foster, John H., Houston, Texas.
*1905. Freer, Otto T., Chicago, Ill.
*1940. Gatewood, E. Trible, Richmond, Va.
*1928. Gittins, Thomas R., Sioux City, Iowa.
*1880. Gleitsmann, Joseph W., New York, N. Y.
*1922. Goldsmith, Perry G., Toronto, Canada.
*1898. Goodale, Joseph L., Ipswich, Mass.
*1932. Graham, Harrington B., San Francisco, California.
*1906. Greene, D. Crosby, Jr., Boston, Mass.
*1917. Greene, Joseph B., Asheville, N. C.
*1950. Hall, Colby, Encino, Calif.
*1905. Halsted, Thomas H., Los Angeles, Calif.
*1896. Hardie, Thomas Melville, Chicago, Ill.
*1915. Hastings, Hill, Los Angeles, Calif.
*1944. Havens, Fred Z., Rochester, Minn.
*1888. Hinkel, Frank Whitehill, Buffalo, New York.
*1895. Hopkins, Frederick E., Springfield, Mass.
*1930. Houser, Karl M., Ardmore, Pa.
*1927. Hubbard, Thomas, Toledo, Ohio.
*1919. Hurd, Lee Maidment, Rowayton, Conn.
*1920. Imperatori, Charles J., Essex, N. Y.
*1904. Ingersoll, John Marvin, Miami, Fla.
*1928. Jarvis, DeForest C., Barre, Vt.
*1939. Johnston, William H., Santa Barbara, Calif.
*1942. Kelly, Joseph D., New York, N. Y.
*1918. Kenyon, Elmer L., Chicago, Ill.
*1921. Kernan, John D., New York, N. Y.
*1929. Kistner, Frank B., Portland, Oregon.
*1885. Knight, Charles H., New York, N. Y.
*1939. Large, Secord H., Cleveland, Ohio.
*1894. Leland, George A., Boston, Mass.
*1922. Lillie, Harold I., Rochester, Minn.
*1943. Lincoln, William R., Cleveland, Ohio.
*1928. Lyman, Harry Webster, St. Louis, Mo.

*1886. MacCoy, Alexander W., Philadelphia, Pa.
*1941. Martin, Robert C., San Francisco, Calif
*1896. Mayer, Emil, New York, N. Y.
*1951. McCart, Howard W. D., Toronto, Canada.
*1939. McCaskey, Carl H., Indianapolis, Indiana.
*1943. McCullagh, Samuel, New York, N. Y.
*1923. McKinney, Richmond, Memphis, Tenn.
*1928. MacPherson, Duncan, New York, N. Y.
*1939. Myers, John L., Kansas City, Mo.
*1901. Myles, Robert C., New York, N. Y.
*1922. New, Gordon B., Rochester, Minn.
*1923. Newhart, Horace, Minneapolis, Minn.
*1903. Packard, Francis R., Philadelphia, Pa.
*1878. Porter, William, Ocean Springs, Miss.
*1942. Potts, John B., Omaha, Nebr.
*1903. Renner, W. Scott, Buffalo, N. Y.
*1897. Rhodes, John Edwin, Chicago, Ill.
*1884. Rice, Clarence C., New York, N. Y.
*1905. Richards, George L., South Yarmouth, Mass.
*1878. Robinson, Beverly, New York, N. Y.
*1921. Sauer, William E., St. Louis, Mo.
*1923. Sewall, Edward C., Palo Alto, Calif.
*1930. Seydell, Ernest M., Wichita, Kansas.
*1907. Shambaugh, George E., Chicago, Ill.
*1908. Smith, Harmon, New York, N. Y.
*1923. Spencer, Frank R., Boulder, Colo.
*1927. Tobey, Harold G., Boston, Mass.
*1925. Tucker, Gabriel, Haverford, Pa.
*1892. Wagner, Henry L., San Francisco, Calif.
*1892. Watson, Arthur W., Philadelphia, Pa.
*1948. Whalen, Edward J., Hartford, Conn.
*1922. White, Francis W., New York, N. Y.
*1939. Wilson, J. Gordon, Old Bennington, Vt.
*1905. Wood, George B., Wynnewood, Pa.

Active Fellows

*1880. Allen, Harrison, Philadelphia, Pa.
*1917. Arrowsmith, Hubert, Brooklyn, N. Y.
*1879. Asch, Morris J., New York, N. Y.
*1942. Ashley, Rae E., San Francisco, Calif.
*1923. Babbitt, James A., Philadelphia, Pa.
*1906. Ballenger, William L., Chicago, Ill.
*1880. Bean, C. E., St. Paul, Minn.
*1949. Beck, August L., New Rochelle, N. Y.
*1904. Berens, T. Passmore, New York, N. Y.
*1924. Bigelow, Nolton, Providence, R. I.
*1938. Blassingame, Charles D., Memphis, Tenn.
*1893. Bliss, Arthur Ames, Philadelphia, Pa.
*1951. Boyden, Guy L., Portland, Oregon.
*1895. Boylan, J. E., Cincinnati, Ohio.
*1932. Brown, John Mackenzie, Los Angeles, Calif.
*1892. Brown, Moreau R., Chicago, Ill.
*1933. Buckley, Robert E., New York, N. Y.
*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, N. Y.
*1882. Chapman, S. Hartwell, New Haven, Conn.
*1902. Coakley, Cornelius G., New York, N. Y
*1913. Coffin, Rockwell C., Boston, Mass.
1918. Cox, Gerald H., New York, N. Y.
*1880. Cushing, E. W., Boston, Mass.
*1878. Cutter, Ephraim, West Falmouth, Mass.
*1880. Daly, W. H., Pittsburg, Pa.
*1878. Davis, F. H., Chicago, Ill.
*1941. Davis, Warren B., Philadelphia, Pa.
1926. Dennis, Frank Lownes, Colorado Springs, Colo.
*1901. Dickerman, E. T., Chicago, Ill.

*1878. Donaldson, Frank, Baltimore, Md.
*1947. Engler, Clarence W., Cleveland, Ohio.
*1954. Equen, Murdock S., Atlanta, Ga.
*1935. Eves, Curtis C., Philadelphia, Pa.
*1919. Faulkner, E. Ross, New York, N. Y.
*1914. Fetterolf, George, Philadelphia, Pa.
*1901. Freeman, Walter J., Philadelphia, Pa.
*1917. Friedberg, Stanton A., Chicago, Ill.
1940. Fuchs, Valentine H., New Orleans, La.
*1897. Frothingham, Richard, New York, N. Y.
1909. Getchell, Albert C., Worcester, Mass.
*1907. Gibb, Joseph S., Philadelphia, Pa.
*1940. Gill, William D., San Antonio, Texas.
*1878. Glasgow, William Carr, St. Louis, Mo.
*1913. Goldstein, Max A., St. Louis, Mo.
*1905. Grayson, Charles P., Philadelphia, Pa.
*1934. Grove, William E., Milwaukee, Wisconsin.
*1933. Harkness, Gordon F., Davenport, Iowa.
*1878. Hartman, J. H., Baltimore, Md.
*1945. Hickey, Harold L., Denver, Colo.
*1879. Holden, Edgar, Newark, N. J.
*1907. Holmes, Christian R., Cincinnati, Ohio.
*1882. Hooper, Franklin H., Boston, Mass.
*1893. Hope, George B., New York, N. Y.
*1938. Hourn, George E., St. Louis, Mo.,
*1939. Hunt, Westley Marshall, New York, N. Y.
*1901. Hyatt, Frank, Washington, D. C.
*1925. Iglauer, Samuel, Cincinnati, Ohio.
*1878. Ingals, E. Fletcher, Chicago, Ill.
*1882. Ives, Frank L., New York, N. Y.
*1938. Jackson, Chevalier L., Philadelphia, Pa.
*1880. Jarvis, William C., New York, N. Y
*1878. Johnson, Hosmer A., Chicago, Ill.
*1879. Johnson, Woolsey, New York, N. Y.
*1960. Johnston, Kenneth C., Chicago, Ill.
*1944. Jones, Marvin F., New York, N. Y.

*1879. Kealhofer, R. H., St. Louis, Mo.
*1942. King, Edward D., North Hollywood, Calif.
*1901. King, Gordon, New Orleans, La.
*1885. Knight, Charles H., New York, N. Y
*1878. Knight, Frederick Irving, Boston, Mass.
*1898. Kyle, D. Braden, Philadelphia, Pa.
*1880. Langmaid, Samuel W., Boston, Mass.
*1878. Lincoln, Rufus P., New York, N. Y.
*1911. Lockard, Lorenzo B., Denver, Colo.
*1913. Loeb, Hanau W., St. Louis, Mo.
*1897. Logan, James E., Kansas City, Mo.
*1935. Looper, Edward A., Baltimore, Md.
*1888. Lowman, John H., Cleveland, Ohio.
*1919. Lynah, Henry L., New York, N. Y
*1952. Lynch, Mercer G., New Orleans, La.
*1915. Lynch, Robert Clyde, New Orleans, La.
*1879. McBurney, Charles, New York, N. Y.
*1927. McGinnis, Edwin, Chicago, Ill.
1936. McGregor, Gregor, Toronto, Canada.
*1913. McKimmie, O. A., Washington, D. C.
*1945. McLaurin, John G., Dallas, Texas.
*1885. McSherry, H. Clinton, Baltimore, Md.
*1914. Mackenty, John E., New York, N. Y.
*1898. Makuen, G. Hudson, Philadelphia, Pa.
*1881. Major, G. W., Montreal, Canada.
*1948. Maxwell, James H., Ann Arbor, Mich.
*1954. Meltzer, Philip E., Boston, Mass.
*1881. Morgan, E. C., Washington, D. C.
*1950. Morrison, Lewis F., San Francisco, Calif.
*1940. Morrison, William W., New York, N. Y.
*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, N. Y.

*1895. Nichols, J. E. H., New York, N. Y.
*1927. Orton, Henry B., Newark, N. J.
*1894. Park, William H., New York, N. Y.
*1892. Porcher, W. Peyre, Charleston, S. C.
*1927. Porter, Charles T., Boston, Mass.
*1954. Pressman, Joel J., Los Angeles, Calif.
*1908. Randall, B. Alexander, Philadelphia, Pa.
*1882. Rankin, D. N., Allegheny, Pa.
*1934. Richards, Lyman G., Wellesley Hills, Mass.
*1902. Richardson, Charles W., Washington, D. C.
*1930. Ridpath, Robert E., Philadelphia, Pa.
*1879. Roe, John O., Rochester, N. Y.
*1945. Robb, James M., Detroit, Michigan.
*1953. Roberts, Sam E., Kansas City, Mo.
*1881. Robertson, J. M., Detroit, Mich.
*1913. Roy, Dunbar, Atlanta, Ga.
*1878. Rumbold, T. F., St. Louis, Mo.
*1879. Seiler, Carl, Philadelphia, Pa.
*1928. Shea, John Joseph, Memphis, Tennessee.
*1893. Shields, Charles M., Richmond, Pa.
*1909. Shurly, Burt R., Detroit, Mich.
*1878. Shurly, E. L., Detroit, Mich.
*1892. Simpson, William Kelly, New York, N. Y.
*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.
*1924. Taylor, Hermon Marshall, Jacksonville, Fla.
*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, N. Y.
*1938. Vail, Harris H., Cincinnati, Ohio.
*1936. Voislawsky, Antonie P., New York, N. Y.
*1933. Wanamaker, Allison T., Seattle, Wash.
1896. Ward, Marshall R., Pittsburgh, Pa.
*1879. Ward, Whitfield, New York, N. Y.
*1886. Westbrook, Benjamin F., Brooklyn, N. Y.
*1924. Wherry, William P., Omaha, Neb.
*1924. White, Leon E., Boston, Mass.
*1939. Williams, Horace J., Philadelphia, Pa.
*1942. Wishart, D. E. Staunton, Toronto, Canada.
*1922. Wishart, David J. G., Toronto, Canada.
*1940. Wood, V. Visscher, St. Louis, Mo.
*1896. Wollen, Green V., Indianapolis, Ind.
*1953. Wilkerson, William W., Nashville, Tenn.

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of the

AMERICAN
LARYNGOLOGICAL ASSOCIATION

1969

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