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Left to right as one faces the group: *front row*, Lederer, Hilding, Miller, Hilger, Davison, Richards, LeJeune, Dixon, Moore, Broyles; *second row*, Murtagh, Equen, Brown, Salinger, Goodale, Silcox, Henry, Woodward, Thornell, Simpson, Boies, Weaver, McCall, Ogura, Ferguson; *third row*, Roberts, Schenck, Alexander, Van Alyea, Tremble, Welle, Cracovaner, Furstenberg, Tolan, Kline, Bordley, Lierle, Robb, Atkins, Putney; *fourth row*, Friedberg, Holmes, Baxter, Cardwell, Williams, Ireland, Walsh, Baker, Hill, Goldman, Harris, Kirchner, Lathrop; *fifth row*, Daly, McHenry, Holinger, Bryce, McGovern, Jones, Conley, Work, Arnold, Har-
rill, Munoz-MacCormick, Suehs, Alfaro, Lewy.

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TRANSACTIONS OF THE
EIGHTY-FOURTH ANNUAL MEETING
OF THE
American Laryngological Association

HELD AT
THE HOLLYWOOD BEACH HOTEL
HOLLYWOOD, FLORIDA
APRIL 21 AND 22, 1963

The Eighty-fourth Annual Meeting of the American Laryngological Association was called to order by the President, Dr. Francis E. LeJeune, at the Hollywood Beach Hotel, Hollywood, Florida on April 21, 1963 at 9:00 A.M.

REGISTRATION

The following Fellows were in attendance:

ALEXANDER, FRED W.	BREWER, DAVID W.
ALFARO, VICTOR R.	BROWN, LESTER A.
ARNOLD, GODFREY E. T.	BROYLES, EDWIN N.
ATKINS, JOSEPH P.	BRYCE, DOUGLAS P.
BAKER, DANIEL C.	CARDWELL, EDGAR P.
BAXTER, JAMES D.	CLERF, LOUIS H.
BOIES, LAWRENCE R.	CONLEY, JOHN J.
BORDLEY, JOHN E.	CRACOVANER, ARTHUR J.

DALY, JOHN F.	MILLER, ALDEN H.
DAVISON, FRANCIS W.	MOORE, PAUL M.
DIXON, FRED W.	MUNOZ-MACCORMICK, CARLOS E.
EQUEN, MURDOCK S.	MURTAGH, JOHN A.
FERGUSON, CHARLES F.	NORRIS, CHARLES M.
FRIEDBERG, STANTON A.	OGURA, JOSEPH H.
FURSTENBERG, ALBERT C.	PUTNEY, F. JOHNSON
GOLDMAN, JOSEPH L.	REED, GEORGE F.
GOODALE, ROBERT L.	RICHARDS, LYMAN G.
HARRILL, JAMES A.	RICHARDSON, JOHN R.
HARRIS, HERBERT	ROBB, JAMES M.
HENRY, GEORGE A.	ROBERTS, SAM E.
HILDING, ANDERSON C.	SALINGER, SAMUEL
HILGER, JEROME A.	SANDERS, SAM H.
HILL, FREDERICK T.	SCHENCK, HARRY P.
HOLINGER, PAUL H.	SILCOX, LOUIS E.
HOLMES, EDGAR M.	SIMPSON, W. LIKELY
HOOPLE, GORDON D.	SISSON, GEORGE A.
IRELAND, P. E.	SOOY, FRANCIS A.
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KIRCHNER, JOHN A.	TABB, HAROLD G.
KLINE, ORAM R.	THORNELL, WILLIAM C.
LATHROP, FRANK D.	TOLAN, JOHN F.
LEDERER, FRANCIS L.	TREMBLE, G. EDWARD
LEJEUNE, FRANCIS E.	VAN ALYEA, OLIVER E.
LEWY, ROBERT B.	WALSH, THEODORE E.
LIERLE, DEAN M.	WEAVER, DELMAR F.
MCCALL, JULIUS W.	WEILLE, FRANCIS L.
MCGOVERN, FRANCIS H.	WILLIAMS, HENRY L.
MCHEMRY, LAWRENCE C.	WOODMAN, DEGRAAF
MELTZER, PHILIP E.	WORK, WALTER P.

OPENING SESSION

SUNDAY MORNING, APRIL 21, 1963

The first Scientific Session of the Eighty-fourth Annual Meeting of the American Laryngological Association, Inc., held at the Hollywood Beach Hotel, Hollywood, Florida, convened in the Pageant Room on Sunday morning, April 21, 1963, at nine thirty-nine o'clock, immediately following the Executive Session, Francis E. LeJeune, M.D. of New Orleans, Louisiana, President of the Association, presiding.

PRESIDENT LEJEUNE: With the aid of this historic gavel I wish to call the Eighty-fourth Annual Meeting of the American Laryngological Association to order.

PRESIDENTIAL ADDRESS

FRANCIS E. LEJEUNE, M.D.

Fellows of the American Laryngological Association and Guests: It is my privilege to welcome you today to the Eighty-fourth Annual Meeting of the American Laryngological Association and to extend to you a cordial invitation to participate in the discussion of the scientific papers prepared for your consideration. Because of the length of the program we shall have to adhere strictly to the time limit for the presentation of papers and discussions.

The privilege of serving as President of this Association is the highest honor that can be bestowed upon a laryngologist, and for this, I am humbly and deeply grateful. The American Laryngological Association is a long-established, honorable society, with an enviable heritage, where a close bond of friendship develops among its members. My deep admiration for this Association was kindled and stimulated by my Chief, the late Dr. Robert Clyde Lynch, who was an

Presidential Address, American Laryngological Association, April 21-22, 1963, in Hollywood, Fla.

From the Department of Otorhinolaryngology, Ochsner Clinic, New Orleans.

ardent and loyal member. Upon returning from these annual meetings, he would review the programs and relate some of the interesting discussions with individual members whose names I held in awe and reverence. I soon began to realize that membership in such an organization was indeed a great honor—one which would be entirely beyond my reach. My election to Fellowship in 1936 was, therefore, one of the highlights of my professional career and kindled within me a determination to exert my greatest effort to prove myself worthy of this honor.

As time went on I learned to enjoy and appreciate the tradition, ideals, aims, and character of the American Laryngological Association. I am reminded of a passage which Hesseltine¹ paraphrased from *Co-operation* (Sunshine Magazine, Litchfield, Ill., vol. 35, No. 7, July, 1958) which I think is justly applicable: "Two most precious things anyone or anything—man or business—can have are integrity and noble character. They are as fragile as an orchid, and as beautiful. As precious as a gold nugget, and as hard to find. As powerful as a great turbine, and as hard to build. As wonderful as youth, and as difficult to keep." This in spirit exemplifies my appreciation of the American Laryngological Association and the outstanding men who comprise its membership.

Ours is a great heritage entrusted to us by our predecessors. Heritage is not earned; it comes to us as a gift from those who have preceded us. It is given in trust that we shall nurture it, adding, we hope, to its brilliance and enhancing its value before passing it on to younger shoulders. Membership in such an organization carries with it responsibilities. Acceptance of this responsibility imposes upon each of us the exercise of care and scrutiny in the selection of new members. For this reason, selection of candidates merits our most serious consideration. We should hold sacred the privilege of exercising the power of casting a vote for new members. In our democratic method of selecting such members a brief historical sketch and a bibliography of each proposed candidate are sent to the membership, together with a ballot containing adequate space for comments. Our secretary has repeatedly stressed the importance of making comments on each ballot, as this is of inestimable value to the Council in the further discussion of candidates. This year only 80 per cent of the ballots sent out by the secretary were returned. This means that 20 per cent of the membership failed to cast their vote on the proposed candidates. Only 21 per cent of those who voted returned ballots

containing comments. I do not believe that this omission was deliberate or due to lack of interest, for we all fully realize our obligations as members of this Association. I particularly wish to call this to the attention of the newer members, as most of them failed to return their ballots. True it is that occasionally a candidate may not be known personally, but sufficient biographic and bibliographic information is given so that some opinion can be formulated. Our goal is a 100 per cent return of ballots, and this can be accomplished with your cooperation.

A few years ago, we were deeply concerned because of the scarcity of physicians seeking otolaryngological residencies. It is gratifying to know that today there are few good residency programs in our specialty that are not filled. This was well brought out last year by Dr. Dean Lierle in his Presidential Address, and I am happy to report that this year more men in the senior medical class in my locality have expressed a desire to specialize in otorhinolaryngology than in any previous years. Dr. Lierle informs me that there is such a large increase in applicants for Board certification in Otolaryngology that an additional examination must be held this year. Must we attribute this to an exploding population, to good salesmanship on the part of the otolaryngologist, or to increased interest in our specialty? With the increasing population and the extended years to man's life, the demand for well trained otolaryngologists will be greater. The responsibility of teaching others our art is an ancient and honored tradition among physicians. The American Medical Association, American College of Surgeons, American Board of Otolaryngology, and Residency Review Committee are largely responsible for the excellence of our residency programs in our various training centers. We need only to listen to the younger men appearing on our national programs discussing intricate medical and scientific problems with poise and full knowledge to be assured that our specialty is being handed down into safe hands.

It would be comforting to our Founding Fathers to know that the Association they founded is in such capable hands. If we could for a few moments project ourselves into a world of make-believe and invite our Founding Fathers to spend a day with us reviewing briefly the progress of medicine during the past twenty-five years, they would be amazed at the revolutionary changes that have occurred in this short space of time. The discovery of insulin, the antibiotics, penicillin, the poliomyelitis vaccines, and the steroids has practically

eliminated many of the dreaded diseases and effectively controlled many others.

Our Founding Fathers would be further amazed to know that the fear of laryngeal diphtheria is no longer with us, and that acute mastoiditis, lateral sinus and cavernous sinus thrombosis, and osteomyelitis are indeed rare diseases. The phenomenal progress in otologic practice in the restoration of hearing by fenestration, and later, stapedectomies, tympanoplasties, and other otological procedures, has given added comfort and pleasure to thousands who lived in a silent world. The discarding of hearing aids after these surgical procedures is remindful of the miraculous cure of the crippled who cast away their crutches at the Shrine of Our Lady of Lourdes.

Treatment of malignant tumors of the head and neck with roentgen-ray, cobalt, and chemotherapeutic agents gives greater promise of success than in the past. Surgical procedures, in properly selected cases, directed to these areas are highly successful. Pneumonectomies, arterial perfusion, radioisotopes, and super-voltage radiation are but a few in a mounting armamentarium that will eventually control cancer, the most dreaded of diseases. The brilliant and daring techniques of vascular and cardiac surgery are monumental accomplishments. The amazing development of electronic aids, such as the medical computer now being used in several medical centers, gives promise of much aid to the physician by receiving, recording and storing medical symptoms. The Laser, Maser and Magna Scanner would mystify our forefathers, as it mystifies me. Not only would they be astonished at these developments, but they would also be intrigued by the wonderful work of medical scientists on the mysterious code of inheritance which determines the basic nature of every living organism.

We are indeed proud of the accomplishments in the field of otolaryngology in the space of a few years. However, ours is not the only group of medical men who have felt a sense of pride in the progress of our profession. Osler expressed the thoughts of his time admirably when he said, "Never has the outlook for the profession been brighter. Everywhere the physician is better trained and better equipped than he was twenty-five years ago. Disease is understood more thoroughly, studied more carefully and treated more skillfully. Diseases familiar to father and grandfather have disappeared and the Public Health measures have lessened the sorrows and brightened the

lives of millions." This statement is as applicable today as it was in the time of Osler.

The present progress of medicine, made possible by the continuous and unabated amount of medical research, not only would appall our Founding Fathers, but gives assurance that the future promises fantastic weapons in the fight against disease that well nigh stagger the imagination.

Our forefathers experienced much pride and satisfaction in the skilled writing of prescriptions for various diseases. Times have indeed changed and our Founding Fathers would be astounded that only 10 per cent of prescriptions written by physicians today are compounded by the pharmacist. The other 90 per cent are for medications, prepared by reputable pharmaceutical establishments, in the form of tablets, capsules, spansules, cough mixtures, and the like. Of drugs in common use a number of years ago, the principal survivors are morphine, the barbiturates, and aspirin. The antibiotics and steroid compounds now account for the majority of prescriptions.

Indeed, it has been a day replete with dramatic surprises for our Founding Fathers. And now, before concluding, I am sure they would want me to warn you of the growing tendency of some physicians to seek publicity in newspapers and magazines. The medical profession has always justly frowned upon such undue publicity, deeming it unworthy of the followers of Aesculapius. The Judiciary Committee of our State Medical Societies has, as a rule, been able to contain the few who attempted to break this barrier. However, of late, there has been increasing evidence of physicians seeking the limelight. I am concerned particularly with the increasing publicity in trade publications with physician's pictures and in some instances, even their telephone numbers. A physician's devotion to his profession, loyalty to his medical organizations, participation in their activities, good patient-doctor relationship, and practice of ethical and conscientious medicine will rapidly establish his status on a firm and honorable foundation and earn for him the deserved respect and high esteem of his colleagues as well as of the public.

In closing I wish to express my thanks particularly to our genial secretary, Dr. Lyman Richards, whose guidance is largely responsible for this entire program, and to the members of the Council for their generous help and loyal support throughout the year. To the entire membership I wish to express, again, my thanks and deepest gratitude

for the honor of serving as your president for this year, an honor I assure you, that I shall cherish forever.

REFERENCE

1. Hesseltine, H. C.: Does Change Imply Betterness? J.A.M.A. 177:899-902 (Sept. 30) 1961.
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PRESIDENT LEJEUNE: We are fortunate today in having as our guest of honor a man who needs no introduction to any medical audience. His fame as an outstanding speaker, educator and lecturer is known worldwide.

He has been honored with the presidency of nearly all the medical associations of which he was a member, including the American College of Surgeons, the American Cancer Society, the Cordell Hull Foundation, the Pan-Pacific Medical Association, and twelve others.

He has been honored by foreign governments and was made an Honorary Member of the Royal College of Surgeons.

He is Professor Emeritus of Surgery at Tulane University and the co-founder of the Ochsner Clinic in New Orleans.

He has been a generous contributor to the medical literature.

He is a man of tireless energy and just to relate his activities in the last two or three days:

On Thursday he was the guest of honor at the Extension Club in New Orleans, where he was made an Honorary Member. Friday afternoon and Friday night he lectured in San Francisco before one of the graduate Medical Assembly classes, he left California yesterday morning and on his flight the plane stopped briefly in New Orleans for its routine stop, where he was joined by his charming wife, and then they completed their flight to Miami and eventually to Hollywood Beach.

So we welcome him here this morning, but this is just an ordinary thing for this man Ochsner, who is a tireless individual.

I wish to present to you my good friend, Alton Ochsner, of New Orleans, Louisiana, who will now talk to us.

THE MEDICAL PROFESSION AS AMBASSADORS OF GOOD WILL

ALTON OCHSNER, M.D.

(Guest of Honor)

NEW ORLEANS, LA.

I am greatly honored to have this opportunity to talk to you this morning. Although my title was assigned to me, I could not have chosen a more timely one because we are now living in the most hazardous period of the history of our country, and I would like to discuss what we as physicians can do to combat this hazard.

For thirty years I have been making visits to Latin America because of my interest in the people there. On my first visit, I became aware of the fact that the Latin Americans looked up to us as a nation and were friendly toward us, although they were envious of us. As my Latin American friends have said, "Why do you want us to like you? We are never going to like you. We are always going to be jealous of you because you are big and powerful and we are small and relatively poor. What you ought to do is have us respect you."

I think that is one of the mistakes that our country has made. We want other nations to like us. We have had the respect of the Latin American nations throughout the years but now we have lost that. About a month ago I was in Panama attending an Inter-American Air Force Conference. A former president of Panama and a good friend of mine said, "Why don't you people learn? Why don't you lead the Americas in this fight against communism? We, as Latin Americans, have lost all respect for the United States." I think that our image in Latin America now is at its lowest ebb. There are probably two reasons for this. One is that some of our former policies and attitudes toward other countries, particularly Latin America, have been objectionable to them. We are inclined to be condescending toward other peoples, because we erroneously consider ourselves better than anyone else in the world. A number of years ago, when

I was in India, a friend of mine, who is a professor of surgery at Bombay, said to me, "Why do you people refer to us as 'underdeveloped nations'? We realize that we don't have some of the material things that you have in the United States, but our culture is older than yours," which is certainly true—"and although we do not have the material wealth that the United States has, we are trying to improve ourselves. Why don't you refer to us as 'nations under development' rather than 'underdeveloped nations'?" This, of course, is the same thing but has a nicer connotation. Why don't we give these countries credit for what they are doing? Why do we have to insist upon everybody acting and living the way we do? There is no good reason for it.

Last summer, as president of the International House in New Orleans, I headed a cultural mission to Spain. This group of thirty-two people saw Spain as probably few tourists see it. We traveled by bus to sixteen cities, stopping in small hamlets and talking to the people. The Spanish people were happy, well fed, and well clothed, but by our standards they were poor. They didn't have modern plumbing and electric refrigeration, but they were about as happy as anyone could be. Yet we criticize them because they are poor. One evening, after a dinner which had been given for us by the city officials, we went to the home of a vice mayor, who lived in modest circumstances. He did not have electric refrigeration, but he had two magnificent paintings. By our standards he was poor but by his own he was richer than we.

Who are we to say by what standards other peoples should live? Why do we want everyone to be like us? Our government and our industries abroad have made mistakes but most of them were made a long time ago. The image that Latin American had of us deteriorated to the poor image of today partly but not primarily because of these mistakes. The primary reason for this deterioration is communist infiltration. We are fighting the worst war in the history of our nation. It is a cold war and one which we have consistently lost up to the present time, including last October, when the President took such a definite stand in the Cuban crisis, which everyone acclaimed. However, although he originally demanded on-site inspection, this was never carried out, and at the same time he assured Castro we would never invade Cuba. Because of this our standing among the Latin American nations has suffered.

Every time we make a move, every time we negotiate with the communists, we lose. But why? It is because communist infiltration techniques are insidious and clever. There is a small group of hard core communists in Latin America, who are people such as you and I. They are not known as communists, but they infiltrate and gradually influence the thinking of the masses so that everything that we do, whether it is good or not, is immediately interpreted as evil.

For instance, not long ago in one of the Central American republics an orphanage was built with American capital. It seems inconceivable that people could be criticized for building an orphanage. Yet, by subtle innuendoes, the communists were able to make the natives believe that the Americans built the orphanage not to help orphans but to brain-wash them into being anti-national.

This is not a fight between the peoples of the free world and the Russians and Chinese. It is a much more basic fight than that. It is a fight between atheism and theism. The goal of our opponents is a global domination, and Krushchev himself said that he would have complete control of the United States in another ten or fifteen years—and he will, unless we do something about it now.

Everyone said that Cuba would never fall, and I was one of those who believed this. I thought that a country whose people were predominantly Catholic, such as Cuba, would never succumb to an atheistic ideology. It has been said that the Latin American countries are susceptible to communism because of the tremendous difference in economic status of the people. This certainly is undesirable, but such economic conditions cannot be corrected overnight. Nor can they be corrected by edict but this can be accomplished by a gradual process of education.

If it were a question of inequality, the last Latin American country that should have embraced communism is Cuba. The per capita income in Cuba was higher than any other Latin American country, and Cuba was prosperous. It is true that it was ruled by a dictator, and none of us would condone dictatorship. Moreover, Castro denied being a communist, claiming to be a good Catholic. That he was a communist was obvious because he had participated actively in the communistic conquest of Guatemala. As soon as he came into power, however, he admitted that he was communistic—which was known before—and immediately began to rule in the typical communistic manner, which is characteristic of the techniques of communism.

In all previous wars we have fought honorable people—people with integrity. I, however, believe there is another attribute even more important—loyalty. I think loyalty is a more desirable characteristic than integrity (if one had to choose one of the two) because it is impossible to be loyal and not honest whereas one can be honest, in the ordinary sense of the word, and not loyal. The communists have no concept of integrity. They believe that they are justified in using any means that is necessary to achieve their goal, and they do.

Only day before yesterday I was talking with a San Francisco physician who knows former President Dwight Eisenhower well. He told me that Eisenhower had said that we are now facing the most critical period in the history of our nation. For this reason, it behooves all of us, as citizens, to do our part to preserve our way of life.

Too many of us have become complacent. We can't be bothered. We think this is a problem for the State Department. But we can't leave this to the government for two reasons. In the first place, our government is hindered by the poor image other nations have of us. Latin Americans immediately object to anything that comes out of the official United States because they have been indoctrinated to consider us as an imperialistic nation.

The other reason is that as individuals we can do a great deal more than the government. This particularly applies to physicians, who can play an important role in international affairs. We physicians are a privileged group, particularly in our role in international relations, because medicine is the one facet of activity which has no boundaries. All physicians speak the same language although they may use different tongues. Any physician would be cordially received in any nation except possibly behind the Iron Curtain.

I never appreciated the importance of this until two years ago when I made a trip around the world, serving as a consultant to the Surgeon General of the Air Force. In Vietnam our only military personnel was MAAG (Military Advisory Assistance Group). Upon arrival the Commanding General told me, "This is the most frustrating job I have ever had. I have been here five years. I am trying to make these people realize that we are here to help them and expect nothing in return. They can't understand that we have no ulterior motive and they can't realize that we are doing this only in the hope of preserving world peace."

I spent four days at the hospital discussing vital problems with the physicians of the Vietnam army. I gave seven talks with the help of an interpreter. We talked frankly and became close friends. The night before I left they gave a reception for us at the leading hotel. After I returned to the States, I received a letter from the Commanding General in which he wrote, "You have made the difference. These people are now coming to us. They realize the Americans are sincere and we can now get through to them." It wasn't I as a person but I as a physician that made it possible for me to penetrate the barrier. The Commanding General could not penetrate that barrier because he was representing the government of the United States but as a physician I could. Physicians throughout the world understand each other because the same cancer affects a Vietnamese as affects us.

As a result of this experience, it occurred to me that we, as physicians, have a golden opportunity, and therefore an obligation to wage this cold war much more effectively than anyone else can. The communists have been using their physicians in this way for some time. Two years ago, when the All-Pakistan Medical Conference was announced, both the Russians and the Chinese asked permission to send a delegation of ten physicians each to the Conference, which met in Karachi, Pakistan. The Pakistanis were—and I think probably still are—anti-communistic. They told the communists that they could send two delegates from each country. They then appealed to our Ambassador for representation from the United States. The Ambassador went to the State Department, and the State Department in turn asked the American Medical Association for representatives. The A.M.A. frankly told them they had no funds for this purpose. The State Department then learned that I would be in that area representing the Air Force at that time and they asked me to represent the United States at the Conference, which I did. Actually, I think the five days I spent at that conference were probably the most fruitful of my entire tour of duty as Consultant to the Surgeon General of the Air Force. I gave two talks and was asked to discuss every paper presented. Although the Russians and Chinese had representatives there to spread their propaganda, I felt that I, as an American, was able to offset this.

As a result of this trip, I began to realize more and more that in physicians we have a weapon which we should use and which the communists have used most effectively. If 10 or 12 of our most eminent physicians could officially represent the United States at each

of the national medical meetings throughout the world (and I am sure they would gladly contribute their time), we could exert considerable international influence. One of our Congressmen arranged for me to speak with a State Department official about this. I had misgivings about what could be accomplished unless I could talk with the top official because I realized that some of the personnel in the Diplomatic Service are convinced that no one except one trained in diplomacy can influence political thinking.

Unfortunately, I doubt that the person I saw even knows that I was in his office, because I had an appointment at two-thirty, I saw him at two forty-five, and at two forty-eight he excused himself because he had another appointment. Nothing was apparently accomplished. One of the probable reasons for this is that the State Department cannot imagine that physicians could influence the thinking of the people of other nations, and the other reason is that actually the cost of such a project would be minimal, probably less than \$100,000 a year, and it is difficult for our government to comprehend that anything can be effective that costs so little!

As illustrative of what can be done by ordinary citizens to halt the spread of this evil ideology, two years ago a group of us in New Orleans started the Information Council of Americas (INCA), which is preparing magnetic tapes. These "Truth Tapes" are Spanish narrations by Cuban refugees, many of whom were originally supporters of Castro, because they thought he was a liberator from their dictator, Battista. On these tapes they relate their experiences in the "communist paradise." These Truth Tapes are now being broadcast over 106 stations in sixteen different countries in Latin America.

We hope to educate the masses through this medium because such a large part of the Latin American population is illiterate. Practically everyone has a small transistor radio, which was given to them by the communists, who wanted to reach these countries through Cuba. We are sending our messages to these people through the same radios. We hope to stem the tide of communism south of the border by alerting the Latin American masses to the evils of communism by showing them its effect on one of their cousin nations, Cuba.

In conclusion, I should like to re-emphasize that physicians have a wonderful opportunity to do something toward promoting international peace and I hope that we can soon make a concerted effort in this direction.

41 QUESTIONS,—AND SOME OTHERS

ARTHUR W. PROETZ, M.D.

ST. LOUIS

Twelve years ago in response to recurring appeals from budding investigators for something to investigate I put in print 40 questions which I felt still needed answers.

When our President graciously suggested that I review them, as of today, my curiosity was aroused and I am responding. The result of this survey is best expressed in a short comment by Bergen Evans on his own book, *The Natural History of Nonsense*, when he revised it, also after 12 years: "That so few changes are required," he writes, "is at once pleasing and depressing; it is gratifying to have seen so clearly, but dismaying to have been ignored so completely."

A detailed report of progress on each question in the list would be tedious even if I had the broad acquaintance with the literature to make it; however I do see enough of the journals to attempt a fair estimate.

The first thing to strike one is that the dozen problems among the 40 which have received any attention worth mentioning have been practically smothered with it, while the rest have been only lightly touched upon or passed up altogether.

While the mass of research has greatly increased in the last decade, due to funds and facilities now available, it too often lacks imagination and diversity and tends to clump around a few popular topics.

When a subject comes into the limelight, especially a surgical one, there is a rush to get on the shiny bandwagon and be heard, not with one publication but half a dozen in as many journals.

Without being a moralist at the moment, one cannot help observing that there are few soloists and fewer composers on a bandwagon, and that the tootlers are merely there for the ride—unless, of course, they are subsidized, which is something else.

Among my 40 questions a round dozen have received brilliant answers.

—Much work and thinking have been given to malignancies of the larynx and to the influence of tobacco smoke on the incidence of lung cancer.

—The action of the epiglottis has received some illuminating demonstrations with the aid of x-ray cinematography, which have stirred up new questions and discussions.

—Only a month or two ago there appeared a masterly monograph from Oxford on the cytology and behavior of cilia. It is not a clinical work but comes very close to first principles.

—Hilding has contributed persistently to our knowledge of the physiology of both upper and lower respiratory tracts.

—Countless words of many people have been printed on the physiology of hearing and the nature of tinnitus and tone losses in various ranges under many conditions.

—There has been worldwide emphasis on the answer to my burning question, number 40 (how can we keep that window open?), and the one on the nature of otosclerosis.

—One regarding the nature of otitis externa and the pathology of the external auditory canal in general has had the unremitting attention of Senturia and some others with signal success.

—The surgical restoration to usefulness of air and food passages following injury or malignancy has made notable progress.

—The question dealing with the reputedly intimate connection between the nose and the genitalia (thrown in, if I remember correctly, to help bring the quota up to 40) had its fruition this year around St. Valentine's day in a sleek 160 page monograph on masculine frigidity and nasal stress—from Paris—including a good deal on what to do about it.

—There are still 17 questions on the list which, so far as I know, have gone unnoticed: on musical interpretative centers, auditory and visual; on the nature of absolute pitch; atrophic rhinitis; cerebrospinal rhinorrhea and its immunity to infection; vacuum in sinuses; the sinuses and mental disease; parageusia in the ageing; on the purpose

and nature of yawning, and ten more, some clinical, some physiological, all practical.

—Thirteen have been investigated with various degrees of thoroughness. True progress has been retarded in some instances; clinicians encountering a case of a rare disease have been content simply to report it, adding lists and tables of other cases found in the literature, but nothing more.

Among the problems open to more thorough research are things like the behavior and competence of the sinus ostia, in certain diseases and under various treatments, the endocrines and the respiratory system, the granulomata, diaplacosis, allergy and air conditioning.

—Space medicine which had no place among my questions because it scarcely existed at the time has far outstripped me by propounding its own questions and promptly answering most of them.

—Two important fields not included in my list have been steadily and brilliantly pursued, both of them as it happens by the group at Ann Arbor: the salivary glands and the nerves of the nose. They are being carried forward on this program.

This fairly covers the forty questions, for now. Number 41 I shall put aside for a moment and go on to those designated in the title as "some others." These have been put in a separate category because they have no place in either clinic or laboratory, but they do concern investigators intimately in bringing their labors to light, and they keep poking their way into the consciousness of anyone paging through the journals. Here they are:

—What is the most acceptable, therefore the most effective, way of presenting an important scientific investigation in a clinical journal? For the sake of acceptability by other scientists and also because he has been living with his data day and night for some time it is understandable that an author wants to set down all the little details, every mathematical equation, every flicker of the needles on all the -ometers.

It is equally understandable that the clinician, who comprises the bulk of the readers, wants primarily the results in a form that he can remember weeks later when confronted by a patient, secondarily a few data to round out the picture and cover any doubts, and finally not any of the mathematics, even if he can comprehend them.

If the piece runs to 40 pages he skips it and moves on to something more digestible. If this happens often, he lets his subscription lapse.

What to do?

—Boil it down and file away the tables and equations in the lab. to be available to the few others working on the same immediate problem, on request?

—Print an adequate resumé of a dozen pages, stating problems, methods and conclusions and file the main opus with the American Documentation Institute in Washington?

—Print it in extenso in one of the basic science journals seen by clinicians chiefly in libraries?

The answer is any of these.

A journal devoted to printing complete 40-page articles on highly specialized subjects, might appear desirable, but it could not survive, unless subsidized, for each article would interest too few of its subscribers.

Next question:

—What accounts for the present tendency of authors to write papers in groups? Formerly a rugged individual was conversant enough with all shades of his problem to take it on alone, and still come up with something lasting (sometimes even while practicing medicine). Now it seems to require a committee with all of the members in the byline.

—Are problems today too difficult for one brain, or is it possible that large grants now easily available are converting research into a pleasant way of life for too many easy-going medics, so badly needed for treating patients?

—How many have the talent, curiosity and training in research method necessary to produce results commensurate with the expenditure? Some man-by-man reports on productivity would be interesting in the press releases of agencies reporting how many millions spent last year. A brain can turn out an idea, but not the U.S. mint. Nowadays mountains in labor often do, actually, bring forth only little white mice.

—What do you suggest doing about some so-called ethical doctors who issue statements, together with their portraits, to appear

in the lay press, in throw-away journals, and other similar advertisements?

—What can be done by you and me to further the recent move to designate each therapeutic chemical by one official name and to require the use of that term exclusively for all purposes?

—When will we abandon the strange notion that it takes ten years for a team of technologists to teach a man medicine, but that skill in the art of medicine will descend on him in no time like a mantle from heaven, intuitively? Also, how many of the technologists would have the know-how to teach him that art, had they a mind to?

—Where do you find these technologists heading for help when they themselves get sick?

These are only questions, but there is no harm in asking. Perhaps I had better stop while I still have a few friends and get back to Question 41, which keeps raising its ugly head with every perusal of the literature.

It is simply this:

—What on earth has become of the nose?

12 WESTMORELAND PLACE

A SURVEY OF SKIN GRAFTS AND PEDICLE FLAPS FOR REPAIR OF NASAL DEFECTS

JOHN B. ERICH, M.D.

ROCHESTER, MINN.

Nasal defects which involve a loss of skin are becoming increasingly common as a result of trauma, surgery and irradiation. Unless such wounds are extremely small, it is not possible to undermine and approximate their skin margins without distorting the nose. Only by the use of skin grafts can such disfigurement be avoided.

Every loss of nasal tissue produces some disharmony in facial contour and, not infrequently, a deformity that is unsightly. Such defects seldom are viewed with indifference; instead, they are a source of self-consciousness and discontent to most patients. Anything that can be done to eliminate or improve these acquired irregularities of the nose is associated with definite psychologic benefits and with genuine appreciation.

In reviewing the various skin-grafting procedures which have been advocated for the repair of nasal defects, one finds that these techniques have undergone, through trial and error, a series of gradual steps in perfection. Yet, in recent years an insignificant amount of new material has been added to the mass of information already presented in the literature on this subject. Nevertheless, the wide variety of useful skin transplants is such that the selection of the most appropriate method of treatment in any individual case is somewhat confusing and often a matter of much concern. Any practical consideration of this problem requires the preparation of a detailed but simple classification of these grafts together with a rough draft of their commendable and adverse features.

Although surgeons do not agree on all phases of this subject, I have found that, by grouping the grafts for nasal repair and the factors which influence their selection in the following manner, an outline can be developed which is especially useful to me for purposes of discussion and treatment:

1. *Four* different types of skin grafts (one of which is the group of pedicle flaps) are available for nasal repair.
2. *Four* groups of pedicle flaps of skin can be used for nasal repair.
3. *Four* different pedicle flaps can be included in each of these groups, thus making a total of 16 flaps in all.
4. *Four* factors must be considered in the selection of a graft or a flap in any individual case.

The first three groups in this outline provide the basis for a simplified classification which can be presented as follows:

SKIN GRAFTS AND FLAPS USED FOR NASAL REPAIR

- A. *Full-thickness skin grafts* (dissected grafts)
- B. *Split-thickness skin grafts* (shaved with razor or dermatome)
- C. *Composite grafts* of skin, subcutaneous fat and sometimes cartilage from the ear
- D. *Pedicle flaps of skin*
 - I. *Contiguous flaps* (elevated in tissues adjacent to defect)
 1. *Rotation flaps*
 2. *Transposition flaps*
 3. *Trap-door flaps*
 4. *Pleated flaps*
 - II. *Neighboring flaps* (elevated in areas close to defect)
 1. *Vertical forehead flaps*
 2. *Sickle-shaped temporal flaps*
 3. *Loop-shaped supra-orbital flaps*
 4. *Nasolabial cheek flaps*
 - III. *Distant tubed flaps* (prepared on chest, abdomen, back)
 1. *Clavicular tube flaps*
 2. *Pectoral (chest) tube flaps*
 3. *Thoraco-epigastric tube flaps*
 4. *Posterior thoracic or scapular tube flaps*
 - IV. *Distant arm flaps*
 1. *Simple open arm flaps*
 2. *Tubed arm flaps*
 3. *Simple open pectoral-wrist or abdominal-wrist flaps*
 4. *Tubed pectoral-wrist or abdominal-wrist flaps*

In planning the repair of any nasal defect due to an external loss of tissue, the foregoing list of grafts and flaps should be considered. The fact that so many types of skin grafts and pedicle flaps are available for repair should make it obvious that no one of them is capable of correcting every type of defect satisfactorily. Moreover, each has specific advantages and disadvantages. The selection of a graft or flap for any individual patient depends on the size, shape and location of the defect, the age and general physical condition of the patient, the length of time required to complete a reconstruction with various flaps, the patient's financial status and the condition of the tissues surrounding the defect and at the various donor sites.

While it is apparent that many circumstances affect the selection of a flap for repair of a defect of the nose, the preferred method theoretically should be the one which offers the most desirable aesthetic result in the shortest possible time. I have found it convenient to divide the factors which influence the choice of pedicle flaps of skin for nasal repair into the following four groups.

Aesthetic Consideration. Although the skill of the surgeon has much to do with the effectiveness of the final result, the aesthetic outcome is greatly influenced also by the color, texture and thickness of the skin in the flap. Skin on the forehead or cheek closely resembles the skin of the nose. Moreover, the lower part of the neck and upper limits of the chest provide skin that is cosmetically acceptable for nasal repair. However, on the back, abdomen and lower anterior portion of the chest wall, the integument is not ideal, because of its whiteness and excessive thickness.

Time Element. The time involved in elevating and transferring the various pedicle grafts which have been enumerated has a decided influence on the selection of a flap in each case. If the patient is old, his general condition poor or his financial resources especially limited, one is not going to choose a flap which requires innumerable stages in separation even though it might effect a more pleasing reconstruction. On the other hand, in young people or in healthy individuals without financial worries, one would consider the flap which promises the most desirable aesthetic result regardless of the amount of time required to complete the surgery.

Condition of the Tissues at the Site of the Defect and at the Proposed Donor Site. Any surface of the body involved by scarring,

senile changes or alterations due to irradiation is a poor site for the elevation of a pedicle flap or for reception of a free skin graft.

Size and Location of the Defect. If the area to be covered with skin is small, one could consider the use of a contiguous flap, but in larger defects a neighboring or distant pedicle defect becomes obligatory. Furthermore, the location of the defect on the nose greatly influences the choice of a flap. This factor is particularly well illustrated in the vertical forehead flap, which, because of limited length, often will not reach a defect involving the nasal tip.

SELECTION OF SKIN GRAFTS FOR NASAL REPAIR

Split-thickness, full-thickness and composite grafts are free skin transplants; that is, they are completely detached from the donor site and maintain their viability only through the vascular supply in the bed of tissue in which they are placed. They cannot be expected to heal in tissues whose blood supply is inadequate because of previous irradiation or excessive scarring, nor can they be employed over a hole or perforation which has no blood supply at all. Consequently, in nasal surgery these three types of skin grafts have limited usefulness because they can be employed only on superficial surface defects in which the underlying or adjacent tissues are fairly normal. Although some of these grafts (full-thickness and split-thickness) always are lacking in adequate thickness, free grafts provide a quick method of repair and do not produce scarring or distortion of the surrounding tissues as do some pedicle flaps, especially those of the contiguous variety.

Full-Thickness Skin Grafts. If the nasal defect consists of a superficial loss of skin and the area is not too large, a full-thickness skin graft dissected from the posterior auricular region or from the upper eyelid is more desirable than are other free grafts, because the skin obtained in these situations closely resembles in color and texture the skin of the face. However, when the area to be covered is large or involves the whole nose, enough skin to cover the denuded surface cannot be dissected from behind the ear or upper eyelid. Consequently, one is forced to use a full-thickness skin graft from the supraclavicular region or a split-thickness skin graft from the abdomen, thigh or arm. In my experience, a full-thickness dermal graft taken from the supraclavicular region has a more satisfactory color for nasal transplantation than do split-thickness grafts, but the former is often thick, and healing may be unsatisfactory. Conse-

quently, when the nasal area denuded of skin is large, I prefer to use a split-thickness graft obtained from the abdomen or thigh.

Split-Thickness Skin Grafts. Although (shaved) split-thickness skin grafts are thin and usually heal nicely, they often present a poor color match for the rest of the nose. Then, too, split-thickness as well as full-thickness skin grafts have another objectionable feature: namely that of developing pigmentation in their deeper layers. Although this complication does by no means always occur, it is one that cannot be prevented and one that ruins an otherwise pleasing result. Fortunately, however, such pigmentation can be appreciably lessened by derm-abrasion.

Composite Auricular Grafts. When the defect involves a very small loss of tissue in the region of the columella or the free edge of a nostril, repair can be effected quickly and effectively by the use of a composite graft (skin, subcutaneous tissue and occasionally cartilage) taken from the ear. Usually the defective area on the nose must be changed by sharp dissection either into a sort of wedge-shaped opening or into a rectangular space which can be filled or covered with a very thin composite graft. If the defect is large, a pedicle flap of skin becomes necessary for repair because composite grafts which are too thick quickly undergo necrosis and are lost.

USE OF PEDICLE FLAPS FOR NASAL REPAIR

As previously mentioned, free skin grafts cannot be employed over perforations, in tissues whose blood supply is inadequate due to scarring or to atrophic radiation changes or for reconstruction of the nose. In all such instances, pedicle flaps are a necessity for proper repair.

Both simple open and closed tube flaps of skin may be utilized for correction of defects about the nose. By simple pedicle graft, I am referring to any flat, ribbonlike flap of dermal and subcutaneous tissue which is elevated in locations that permit direct transference to the defect.

A tubed flap is elevated as a ribbonlike mass of skin and subcutaneous fat left attached at each end for a blood supply; the free margins are turned under and approximated to make a tube-like structure in which all raw surfaces are eliminated even when the graft is transferred, thereby preventing infections and the development of excess scar tissue in the flap. Furthermore, tubing a flap actually

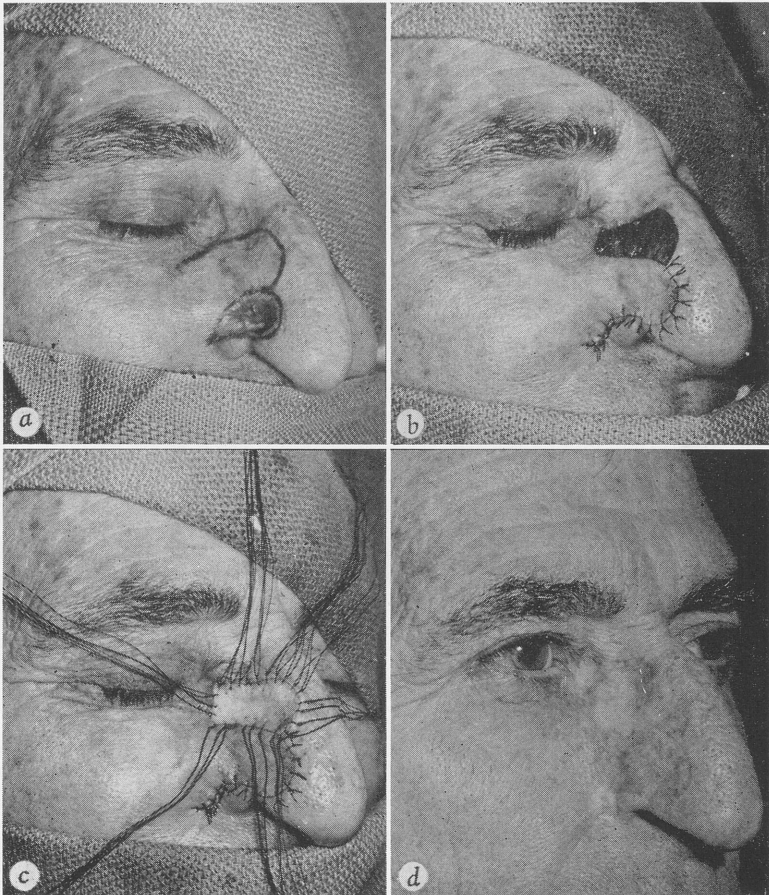


Fig. 1.—*a*. Postoperative defect of right side of nose. Rotation contiguous flap is outlined above defect. *b*. Contiguous flap elevated and rotated down to cover defect. *c*. Remaining denuded surface on side of nose covered with a postauricular full-thickness graft. *d*. Final cosmetic result.

increases its blood supply. For the repair of defects about the nose, tubed flaps may be elevated on the neck, trunk or arm.

Necrosis of many pedicle skin grafts can be expected if they are elevated and transferred in on stage, because the blood supply is not adequate to nourish the entire flap. This difficulty, however, can be eliminated by means of a delayed procedure in which the flap

is elevated in a series of successive stages that are designed to transfer the circulation to the pedicle. By these operations, the vessels in the pedicle are stimulated to become larger until they are able to nourish the entire flap, which then can be transplanted without subsequent danger of sloughing. It is true, of course, that short flaps with wide pedicles often can be elevated and transplanted in one operation without the risk of necrosis.

CONTIGUOUS PEDICLE FLAPS

Contiguous pedicle grafts are simple open flaps which are elevated in tissues immediately adjacent to the nasal defect and are advanced, rotated or folded to cover the defective area.

For a small loss of tissue on the nose, contiguous flaps offer the quickest method of repair inasmuch as they can be elevated and transferred in one surgical procedure. Moreover, their color and texture are almost identical to those of the remaining skin on the nose. Still, contiguous flaps do not provide a completely satisfactory restoration, because they produce noticeable scarring in the tissues bordering the defect. Moreover, these grafts have a limited degree of usefulness since they can be employed only for small tissue losses and can be elevated only in tissues which are free of radiation changes and scarring.

Contiguous Rotation Flap. A rotation flap is designed to be elevated and rotated on its axis so as to cover a nasal defect (Fig. 1). This type of contiguous flap can be utilized over any small area which is deficient in skin and which does not involve the free edge of the nostril. When spread over a perforation, the flap should be made sufficiently large so that its margins will extend well beyond the edges of the hole. Even though it covers the perforation, a flap that is too small will become puckered by scar tissue and will produce a repair that is cosmetically unsatisfactory.

Contiguous Transposition Flap. A contiguous transposition flap is prepared in such a way that, after elevation, it can be transposed from the donor site to cover the defect. From a technical standpoint, the flap is not rotated but raised and transferred to a new location over the defective nasal area. The transposition flap is especially well adapted for small areas of tissue loss on the nasal bridge near the glabella (Fig. 2) or bordering the cheek. In these situations, a small flap in the glabella region or on the cheek can be prepared and rearranged in one surgical procedure to make a covering for the defect.

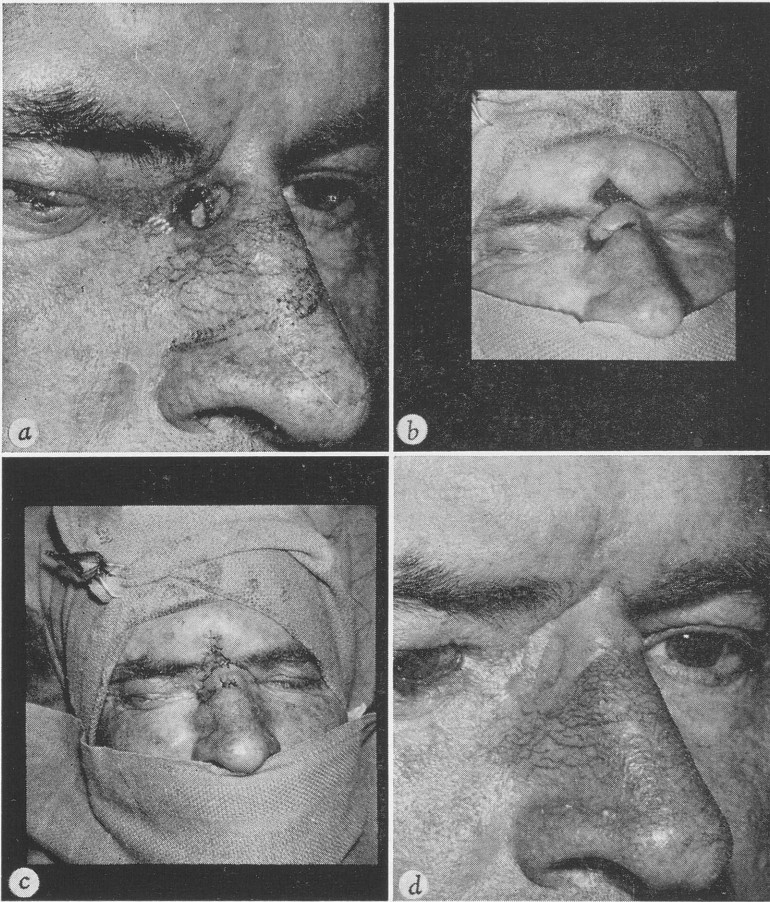


Fig. 2.—*a*. Postoperative defect on right side of bridge of nose corrected with a transposition flap from glabella. *b*. Small contiguous flap in glabella transposed to cover defect on right side of nose. *c*. Flap sutured in place. Donor site is also sutured. *d*. Final cosmetic result.

Because the surrounding tissues are soft and pliable, the donor area can be obliterated by approximating and suturing its margins.

Contiguous Trap-Door Flap. The contiguous trap-door flap is elevated in tissues adjacent to a small hole through the nose, the pedicle being located along the free edge of the healed perforation. After elevation, the flap is turned over like a trap door and inlayed into the

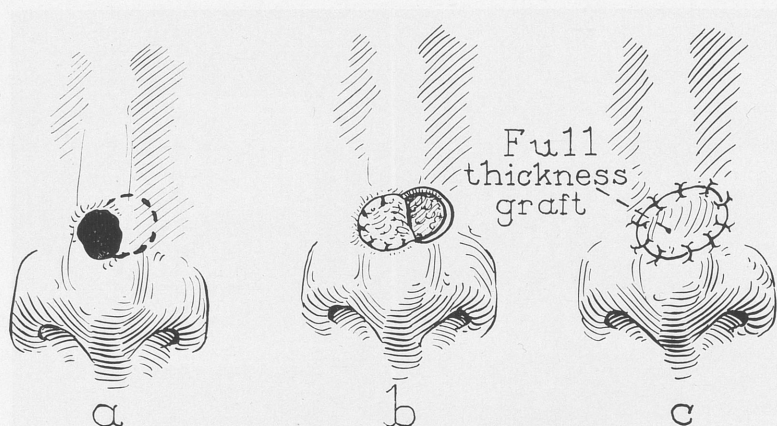


Fig. 3.—Postoperative perforation of nose corrected by trap-door contiguous flap and full-thickness skin graft. *a.* Contiguous flap outlined. *b.* Flap is turned over and sutured into defect. *c.* Postauricular full-thickness graft inlaid into defect and over flap. (Reproduced from Erich, J. B.: Repair of Nasal Defects Involving Tissue Loss. *Journal-Lancet* 77:259-272 [Aug.] 1957.)

defect (Fig. 3). The flap thus makes a lining for the perforation, which must be covered with another pedicle flap or, more commonly, with a free skin graft.

Contiguous Pleated Flap. A contiguous flap, which I designate as "pleated," is designed to correct the deformity caused by a small loss of tissue along the free edge of a nostril. The flap is elevated above the healed defect, turned down and doubled back on itself (like the hem on a garment) to form a fold which makes a new free edge for the nostril (Fig. 4). After transference of the flap in this manner, an irregular raw surface is left on the side of the nose, which can be covered with a full-thickness postauricular skin graft.

NEIGHBORING PEDICLE FLAPS

Neighboring flaps are simple open pedicle grafts which are elevated in the same general anatomic region as the defect and are long enough to permit direct transference to the defect. For repair of losses of tissue about the nose, neighboring flaps are elevated on the forehead and along the nasolabial fold.

In defects which are too extensive to be corrected by contiguous flaps, the use of pedicle grafts on the forehead or cheek should be considered unless the skin in these areas is badly scarred, shows alterations due to irradiation or is involved by senile changes such as keratoses or epitheliomas. Flaps on the forehead or cheeks give excellent cosmetic results because they provide skin similar in color and texture to the skin of the nose. Although some of them require more time in preparation than do contiguous flaps, all are less time-consuming than are the distant tubes. Unfortunately, forehead pedicle grafts

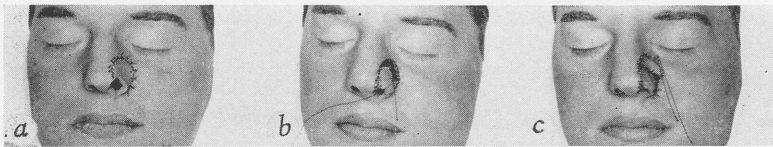


Fig. 4.—Postoperative defect on free edge of nostril corrected with contiguous pleated flap (exhibit). *a*. Flap based along free border of defect is elevated and sutured back in place to obtain a better blood supply. *b*. Two weeks later the flap is elevated, turned down and folded on itself to form free edge of nostril. *c*. Denuded surface above flap is covered with a postauricular full-thickness skin graft.

have one extremely undesirable feature, namely, that of producing conspicuous scar tissue at the donor site in some cases. Although this is not so important in women, who can style their hair to cover the area of cicatricial deformity of the forehead, in men the scar is often most objectionable. I do not wish to leave the impression here that the forehead is always deformed after elevation of a flap in this region; on the contrary, the residual scarring occasionally is inconsequential.

Vertical Forehead Flaps. The vertical flap, which is elevated in the center of the forehead or over one brow, is based below, extends upward to the hairline and can be turned down to cover an area denuded of skin (Fig. 5). Except in bald-headed persons, the vertical forehead flap, even when curved laterally, is limited in length by the hairline and thus ordinarily is adaptable only for defects involving the upper half of the nose. However, most vertical forehead flaps can be prepared in one or two stages to provide considerable tissue for quick nasal repair. Their most objectionable feature is that they

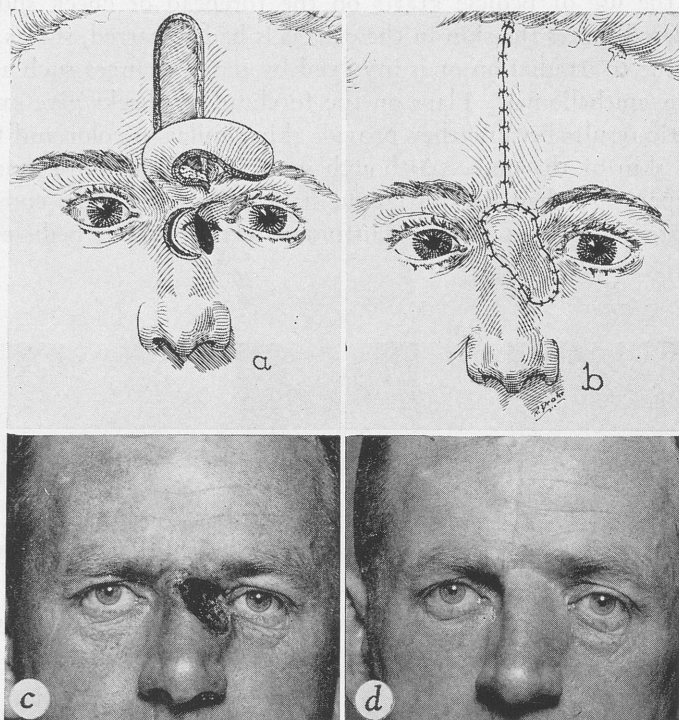


Fig. 5.—Large postoperative defect on left side of nose corrected with a vertical forehead flap. *a*. Drawing of vertical forehead flap which is elevated and turned down to cover defect on left side of bridge of nose. Small flap adjacent to defect is turned over for a lining. *b*. Completed plastic procedure with wound at donor site sutured. *c*. Defect of left side of bridge of nose before healing has occurred. *d*. Final postoperative result.

unavoidably may leave conspicuous scarring over the frontal area; consequently, this form of neighboring graft should not be utilized without first making certain that the patient is thoroughly aware of this possible complication. A vertical forehead flap, the width of which measures at least one-third of the length, usually can be elevated and transplanted in one operation. If, however, the flap is much narrower than the above-suggested dimensions, it is less likely to slough if moved in two stages about two weeks apart. If the flap is intended to cover a perforation, it usually must be lined with a free split-thickness skin graft which is inserted under the flap during its primary elevation. Three weeks after transference, the flap has

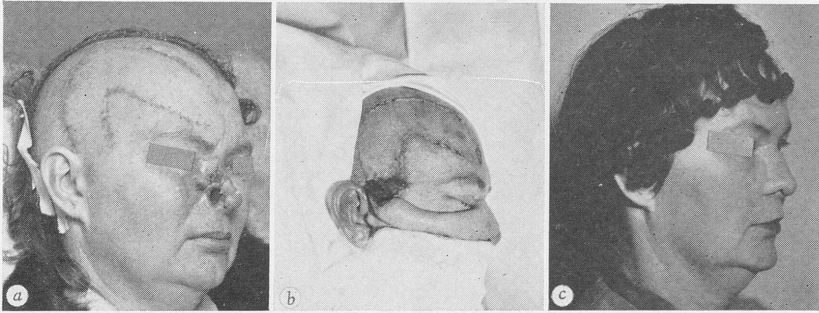


Fig. 6.—Large postoperative defect on right side of nose corrected with a sickle-shaped temporal flap. *a.* Defect on side of the nose and outline of sickle flap. Frontal pedicle had not yet been severed. *b.* Sickle flap transferred to defect. Resultant denuded surface in temporal region has been covered with a split-thickness skin graft from abdomen. *c.* Final cosmetic result. Unused part of flap has been returned to scalp. Residual scarring and split-thickness skin graft on right side of forehead is evident. Patient tries to cover it with her hair.

secured a good enough blood supply from the edges of the recipient wound to permit severance of the pedicle and return of the unused part to the forehead.

Sickle-Shaped Temporal Flap. The sickle flap, so called because it roughly assumes the shape of a sickle, is constructed in the temporal region and is based in the pre-auricular area to include the temporal artery. After elevation, the flap can readily be extended to reach the nose (Fig. 6). Of all the forehead flaps, the sickle-shaped temporal graft produces the least amount of visible scarring on the forehead and, consequently, has a wider range of acceptance. However, sickle flaps supply a limited amount of tissue and consequently can be used only for partial nasal defects. Usually, they do not offer enough hair-free skin for total reconstruction of the nose or even the nasal tip.

Sickle flaps leave little visible scarring, because most of the flap is located back of the hairline; consequently, the unused part of the flap when returned to the scalp becomes covered with hair to hide the scars.

In my experience, every one of the flaps discussed in this paper that cannot be moved to the recipient area in one or two operations

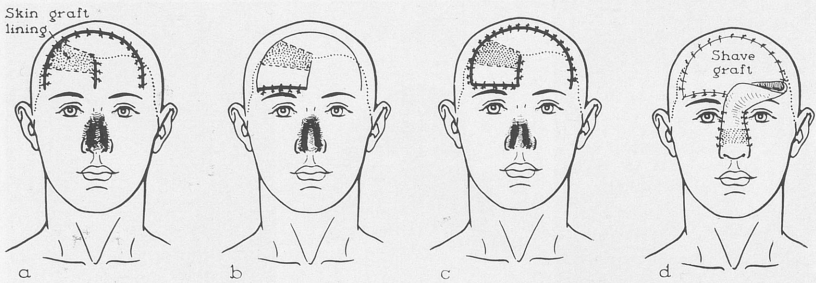


Fig. 7.—Stages in elevating and transferring loop-shaped supra-orbital flap to nasal region. *a*. Flap is completely elevated and undermined as indicated by solid lines. Dotted area represents a thick-shaved skin graft which is inserted with its raw surface up to act as a lining when flap is transferred to reconstruct nose. After lining is in place, flap is sutured back in position as indicated. *b*. Ten days later one pedicle is cut across above brow to stimulate vessels in remaining pedicle to become larger. Wound is sutured. *c*. Ten days later entire pedicle flap is elevated and sutured back in position to stimulate blood supply. *d*. Ten days after that, flap is brought down and folded to form columella and nostrils. Resultant denuded surface on forehead is completely covered with a split-thickness skin graft as indicated. (Reproduced from New, G. B., and Erich, J. B.: *The Use of Pedicle Flaps of Skin in Plastic Surgery of the Head and Neck*. Springfield, Illinois, Charles C. Thomas, Publishers, 104 pp., 1950.)

requires four surgical procedures in elevation and transference. This observation applies not only to simple open flaps but to the tubed grafts as well. During the first operation, the entire sickle flap, which we are discussing here, is elevated and replaced without disturbing its two (pre-auricular and frontal) extremities. In ten days, the distal pedicle in the frontal area is severed; if a skin graft is needed for a lining, it is inserted at this stage. Ten days to two weeks later, the flap is again raised and replaced, and this represents another stage in delay to improve the blood supply. At the fourth operative procedure, the flap is re-elevated and transferred to cover the nasal defect. Should one find that the flap extends across an eye, it is most important to wrap the pedicle in vaseline or paraffin gauze to protect the eye from hairs growing out of the flap. If the lateral wall of the nose is to be reconstructed, the distal edge of the flap is turned under to produce the free edge of the nostril.

Three weeks after transference to the nasal region, the unused part of the flap can be detached and returned to the scalp.

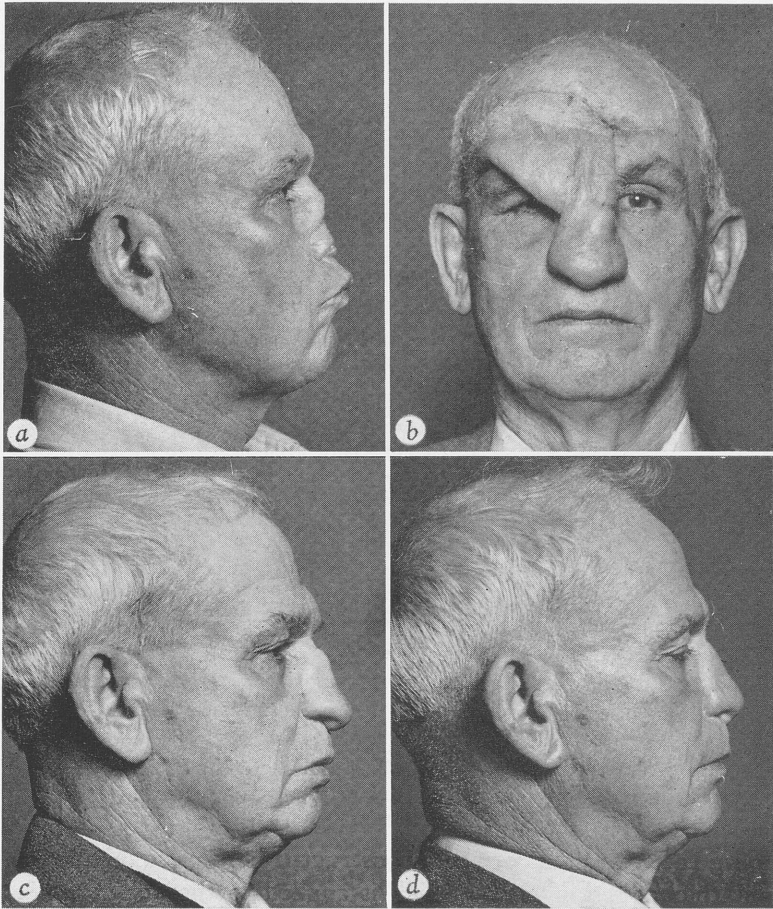


Fig. 8.—Postoperative loss of nose corrected with a loop-shaped supra-orbital flap. *a.* Defect. *b.* Supra-orbital flap has been folded to form columella and nostrils and brought down to nasal region. Denuded surface on forehead is covered with a split-thickness skin graft. *c.* Unused part of flap has been returned to forehead. *d.* Reconstructed nose reduced to normal contour by operations shown in Figure 9.

Loop-Shaped Supra-orbital Flap. The loop-shaped supra-orbital flap is constructed with a pedicle over one brow that includes the supra-orbital artery for a blood supply. The stages in elevating and moving this flap are illustrated in Figure 7. Again, four operations are required for transference to the nasal region. The color, texture and contour of the graft are especially well adapted for building an

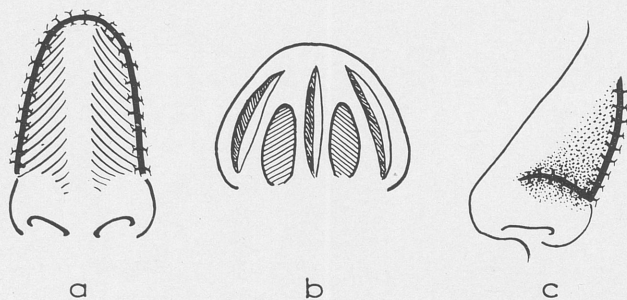


Fig. 9.—*a*. Incision to remove excess fat and subcutaneous tissue along lateral aspects of nose. *b*. Elliptical pieces of skin and subcutaneous tissue removed from columella and either ala in order to narrow these structures. *c*. Lateral incision made above ala through which subcutaneous tissue is removed. This operation makes a sulcus-like depression above ala and makes ala itself more prominent. (Reproduced from New, G. B.: Total Rhinoplasty Using a Forehead Flap. *Tr. Am. Soc. Plastic & Reconstructive Surg.* 13:33-42, 1944.)

entire nose (Fig. 8). Were it not for the fact that this flap leaves much scarring on the forehead, I would prefer it to all others for total nasal reconstruction; but in view of this one undesirable feature, I favor the use of a large clavicular or chest tube flap.

When the supra-orbital flap is transferred to the nasal region, the free end is folded to form the columella and nostrils (Fig. 8*b*). If the defect extends up to the glabella, then that portion of the flap forming the upper half of the reconstructed nose must be lined with a split-thickness skin graft, which is applied to the undersurface of the pedicle during the initial stage of its elevation.

Following transference of the flap in total nasal repair, four operations are required to complete the reconstruction. First, the unused part of the flap is severed and returned to the forehead. After a two-month period of waiting to allow for shrinkage, the three operations shown in Figure 9 are indicated for establishment of normal nasal contour. In the first of these procedures, the reconstructed nose is incised along its upper and lateral borders (Fig. 9*a*). The skin flap within this line of incision is undermined and turned down so that excess fat on the sides and dorsum of the nose can be removed; the flap is then replaced and sutured. Two weeks later, elliptical pieces of skin and subcutaneous fat are removed from the lower border of



Fig. 10.—Epithelioma on right side of nose removed and area covered with a nasolabial cheek flap. *a*. Epithelioma before removal. *b*. Nasolabial flap elevated and transferred into postoperative defect. End of flap has been folded over to form free edge of nostril. Donor site has been sutured.

the columella and the lateral wall of each nostril, thereby giving the nares a more natural contour (Fig. 9*b*). With the passing of another two weeks, the final operation to accentuate the alae is undertaken; this involves lateral nasal incisions through which subcutaneous tissue is excised above the alae to make these structures seem more prominent (Fig. 9*c*).

In some instances, the bridge or tip of a reconstructed nose lacks adequate support and must be upheld with an implant of costal cartilage or bone. For this purpose, I prefer a piece of bone taken from the crest of the ilium. Removal of the cortex from a block of iliac bone leaves a cancellous graft which is easily shaped to the required dimensions with files and rongeurs. Moreover, it heals in place quickly and does not warp as costal cartilage so often does.

Nasolabial Cheek Flap. The nasolabial flap is a simple open graft based above and prepared in one or two stages along the nasolabial fold. After elevation, it can be swung directly over to the nose for repair of small defects on the lateral wall of a nostril (Fig. 10). However, nasolabial flaps provide only a small amount of tissue and are used most advantageously in older people who have loose, wrinkled and redundant skin. In such individuals, the flabbiness of the adjacent tissues at the donor site permits ready approximation of the skin

edges, leaving a linear scar along the nasolabial fold that ultimately becomes almost invisible. In younger patients, however, such a wound may be closed under considerable tension, thus producing a wide, conspicuous scar associated perhaps with distortion of the angle of the mouth.

DISTANT TUBE FLAPS

The distant tube flaps which I employ for nasal restoration are all elevated on the trunk. Some surgeons make use of flaps tubed along the course of the sternomastoid muscles or across the lower part of the neck. However, in my opinion, such grafts seldom are satisfactory; they produce too much visible scarring at the donor site and too often do not provide sufficient tissue for satisfactory repair.

In recent years, I have been agreeably impressed by the excellent nasal reconstructions which can be effected with a clavicular tube or with the upper end of a chest tube flap (Fig. 11). The skin near the clavicle resembles closely in color and texture that of the face; even more important is the invisibility of objectionable scar tissue at the donor site when the patient is clothed. Compared with forehead flaps, the only real disadvantage associated with clavicular and chest tubes is the increased time required to complete the nasal reconstruction. It is true, of course, that one encounters a little more difficulty in folding a clavicular or chest tube graft to form the columella and nostrils than occurs with a forehead flap, but the ultimate result is just as good. Consequently, only in patients with much scarring on the anterior chest wall, in men with a heavy growth of hair on the chest and shoulders, in individuals who are unconcerned about post-operative scarring on the forehead or in those who, for various reasons, must have the reconstruction completed in the shortest period of time do I consider the use of a forehead flap. Except in these circumstances, I prefer a clavicular or chest tube graft.

Clavicular Tube Flaps. A clavicular tube flap which is elevated parallel with or obliquely across the clavicle is ideal for smaller defects of the nose. It supplies skin with a good color for facial repair, but occasionally there is not enough tissue for total nasal reconstruction. As in sickle-shaped temporal flaps and loop-shaped supra-orbital flaps, four surgical procedures are necessary for transference of one end of a clavicular tube to the nasal region. During the first operation, the tube is constructed; in determining its size, much depends on the dimensions of the defect and the distance to the nose. However, a

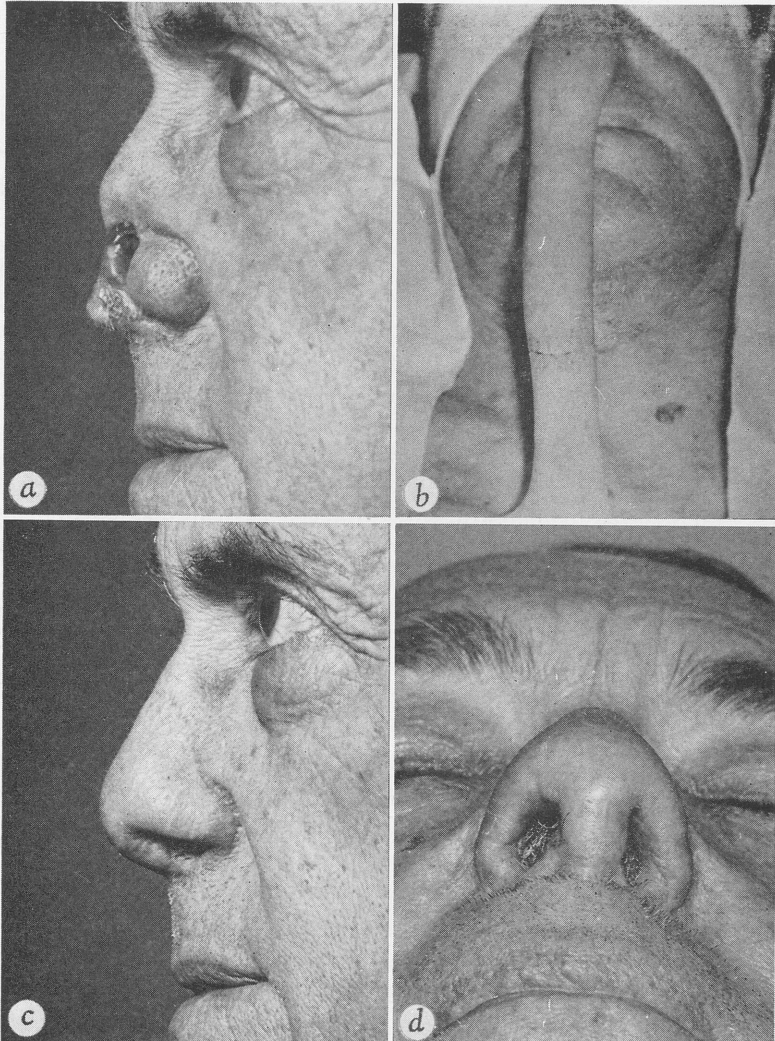


Fig. 11.—*a*. Postoperative loss of lower half of nose. *b*. Chest tube flap transferred to reconstruct nose. *c*. and *d*. Final result.

slough in the tube is unlikely if one holds rigidly to the rule of never permitting the length to be greater than three times the width. Furthermore, if the lower mesial end of the flap is to be used for nasal reconstruction, the tube must be made long enough to reach the nose without undue tension; otherwise, interference with circulation in the tube can be expected. If one finds that the flap cannot be stretched to the nasal region without excessive traction or torsion on the pedicle, the detached extremity of the tube must be joined to the side of the neck as an intermediate stage; later, the opposite end can be used for nasal reconstruction.

Before the second operation is undertaken, about a month should pass to allow for the development of an adequate blood supply in the tube. In the second and third stages of delay, the end of the flap to be used for nasal repair is gradually undermined. In the fourth operation, the flap is transferred to the nasal region.

If the loss of nasal tissue is due primarily or secondarily to irradiation changes in the skin, it has been my observation that the tube may undergo some degree of necrosis after it has been moved to the nasal region in spite of several stages in delay and regardless of the fact that the skin in the clavicular area never was directly irradiated. This tendency to sloughing can be avoided by deferring the transference of the tube for 6 to 8 weeks after the third operation noted above. This period of delay seems to stimulate the vascularization of the tube so that subsequent sloughing is most unlikely.

Pectoral (Chest) Tube Flaps. A pectoral (chest) tube flap is elevated in a more or less vertical direction on the anterior chest wall, and the stages in its preparation are similar to those of a clavicular tube (Fig. 11). If time is relatively unimportant or if there is much hair on the lower part of the chest, it is advisable to use the upper rather than the lower end of the flap for nasal reconstruction, because the color of the skin higher up on the chest wall tends to match the facial tissues more satisfactorily and usually is relatively free of hair. However, one cannot stretch the upper extremity of a chest tube flap to the nose without first attaching the lower end to the side of the neck as an intermediate stage.

Thoraco-epigastric Tube Flaps. Thoraco-epigastric tubes are elevated along the course of the thoraco-epigastric vein. They are used rather infrequently for nasal repair because of several undesirable features, the most significant of which are poor color and excessive

thickness of the skin. Moreover, the usual thoraco-epigastric tube is extremely long and requires not only an extended period of time in preparation but also a special method of construction; that is, during elevation it must be bridged in the center to ensure an adequate blood supply. In this procedure two tubes are made in tandem; the bridge of undisturbed skin between them acts as a common pedicle. When the tubes are completely healed and a good blood supply has been established, the bridge can be gradually incised and tubed to convert the two tubes into one long structure. Although a thoraco-epigastric tube seldom is employed in nasal surgery, there are nevertheless instances in which such pedicle grafts are especially useful, as in the patient with extensive facial deformity due to residual scarring from a burn. In such situations, dermal tissue is needed not only for reconstruction of the nose but also for repair of large cicatricial areas on the face.

A thoraco-epigastric flap need not extend from the axilla to the inguinal region; when prepared in the lower half of this area, it amounts to nothing more than an abdominal tube. If constructed only in the upper half, it might better be called a "subaxillary" tube.

Posterior Thoracic or Scapular Tubes. Posterior thoracic tubes are elevated in a vertical direction on the back, usually in the scapular area, and are transferred either directly or by migration to the nasal region. Pedicle tube flaps on the back never are completely satisfactory, because the skin is thick, and healing may consequently be delayed. Moreover, the patient finds such a tube undesirable because he cannot lie comfortably on his back. As a matter of fact, the only cases in which I would consider this type of graft are those in which a large amount of tissue is needed to repair a defect involving the nose and adjacent part of the cheek or in the exceptional patient for whom there is no other satisfactory skin available for transplantation. Since a flap on the back is not as long as a thoraco-epigastric tube, the former can be prepared in a shorter period of time. In addition, the lower end of a tube elevated on the back usually will reach directly to the nose without the intermediary attachments that are generally necessary when a thoraco-epigastric tube is utilized.

DISTANT ARM FLAPS

These simple and tubed distant flaps are constructed on the arm itself or are elevated on the trunk and carried to the nasal area on the upper extremity. In general, these flaps have a color that is a poor

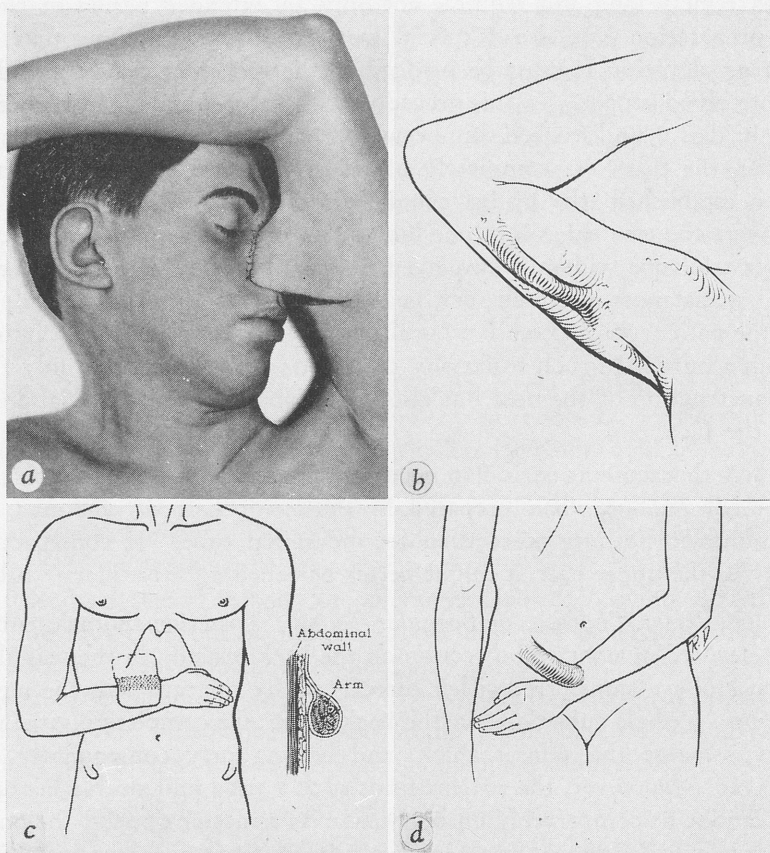


Fig. 12.—*a*. Reconstruction of nose with an open arm flap (exhibit). See text regarding advantages and disadvantages of this flap. *b*. Double pedicle flap of skin elevated and tubed along posterior aspect of upper arm. See text for advantages and disadvantages of this tube flap for nasal repair. *c*. Open pectoral-wrist flap which can be used for nasal repair. A rectangular-shaped flap of skin is elevated on anterior wall of chest. A flap of skin of similar size is elevated on arm near wrist and turned backward with its raw surface exposed. Flap on chest then is attached over raw surface of arm and turned-back wrist flap. Dotted area illustrates flap of skin from wrist, which has been turned backward. Three or four weeks later an upward apron-like extension of flap is excised from anterior wall of chest and is carried directly to nasal defect on wrist. *d*. Abdominal-wrist flap which can be used for nasal repair. Tube flap is elevated on anterior abdominal wall; one end is attached to wrist as illustrated. Later, opposite extremity of tube is detached, and wrist carries pedicle flap to nasal region.

match for that of the skin of the face, and some have a precarious blood supply. Although useful for surface repair, the skin on the arm is too thin for effective nasal reconstruction. On the other hand, the skin on the abdomen is often too thick. As a matter of fact, flaps elevated or migrated on the arm have only one attribute of real merit; they offer a means of repairing deformities of the nose in less time than would be required by some of the pedicles previously described. However, I believe that arm flaps should not be used except in young adults; when employed for reconstruction in older individuals, the enforced period of immobilization of the upper extremity not infrequently can result in permanent disability as far as free mobility of the arm is concerned. I have had patients who after a prolonged period of fixation never did regain full motion of the arm. Consequently, pedicle flaps on the arm have a limited degree of usefulness.

Simple Open Arm Flaps. Any simple open flap elevated on the upper arm must be varied in contour to fit the dimensions of the nasal defect (Fig. 12a). Even when constructed with a wide pedicle, the vascularity often is deficient and areas of necrosis are likely to develop. Accordingly, I would not recommend the use of such a flap except for surface repair in young people who have no other areas on the body in which an acceptable flap could be prepared.

Tubed Arm Flaps. A pedicle flap, elevated and tubed along the long axis of the arm, develops a satisfactory blood supply in time and occasionally can be used to advantage in smaller defects of the nose (such as loss of the columella) in young adults (Fig. 12b). However, the average individual does not have a big enough arm with loose enough skin to permit construction of a tube flap sufficiently large to be of practical use. Consequently, there are few patients for whom this type of graft can be utilized.

Simple Open Pectoral-Wrist or Abdominal-Wrist Flaps. A simple pectoral-wrist or abdominal-wrist flap is an open pedicle graft elevated on the chest or abdomen and attached to the wrist, from which the flap is transported to the nose (Fig. 12c). This flap, which is usually of rectangular shape, is prepared on the anterior chest or abdominal wall. A flap of skin of similar size is elevated on the arm near the wrist and turned backward with its raw surface exposed. The pedicle graft on the chest or abdomen is then attached over the raw surface of the arm and the turned-back wrist flap. When the raw surfaces of the flaps are healed together, a matter of 3 or 4 weeks, an upward apronlike extension of the flap on the trunk is excised

and is carried to the nasal region on the wrist. This technique provides a method whereby considerable skin from the chest or abdomen can be transplanted in a relatively short period of time. Unless the flap is elevated high on the chest wall, the color of such flaps is poor for nasal repair; moreover, the skin often is too thick and the blood supply sometimes inadequate. Therefore, such flaps should not be used in cases in which the final cosmetic result is especially important, and they never should be employed in older patients.

Tubed Pectoral-Wrist or Abdominal-Wrist Flaps. A tubed pectoral-wrist or abdominal-wrist flap (Fig. 12*d*) involves the construction of a tube on the anterior thorax or abdomen, one end of which is attached to the wrist for transportation to the nose. If the tube is short, it can be elevated, tubed and sutured to the wrist in one operation; otherwise, it must be prepared and left alone for four weeks before being connected to the wrist, after which another three or four weeks must elapse before the tube can be transferred to the face. Although these tube flaps supply a large amount of well-vascularized tissue for transplantation, they too have a poor color, are often too thick and should be utilized only in young adults.

SUMMARY

In summary, nasal defects which require skin grafts for repair have been discussed. For losses of nasal skin and subcutaneous tissues, four definite types of skin grafts including pedicle flaps have been considered. The flaps have been divided into four groups, each group being comprised of four different flaps. In addition to a discussion of the advantages and disadvantages of these 16 flaps, four factors which influence their selection have been presented. Four stages in the preparation and transference of some of these flaps have been presented. Furthermore, in total nasal reconstruction, four consecutive operations for restoring normal nasal contour have been outlined.

MAYO CLINIC

HYPERPLASTIC SINUSITIS - A FIVE YEAR STUDY

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This report is based on a study of the case histories of fifty patients who had chronic hyperplastic sinusitis, who were treated and observed by me over a period of at least five years.

I have attempted to unravel the etiologic factors, record the methods of treatment and make a fair assessment of the results of treatment. All patients included in this study had pus and polyps in the nose and density in one or more sinuses shown by roentgen examination. Pus and polyps in one or more sinuses were demonstrated by operation and the nature of the exudate was shown by smears and cultures. Only cases of extensive disease are included in this series.

Seventeen patients had food or inhalant sensitivity in addition to sinus infection. When present, food or inhalant allergy was treated according to currently accepted methods.

Hyperplastic sinusitis may be defined as a type of chronic sinus disease manifest by polypoid thickening of the nasal and sinus mucosa. In my opinion this type of disease is due to local sensitization of the submucosal tissues to bacterial proteins. The infecting organism is usually a staphylococcus. I think this type of polypoid sinusitis is a manifestation of bacterial allergy.

Schenck¹² said, "When infection of allergic ethmoidal tissue occurs, a vicious train of events follows. This tissue cannot combat infection with the same efficiency as can non-allergic tissue. The impaired response to acute infection facilitates progress to a chronic status, and chronic infection becomes established. Bacterial sensitiza-

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tion is then possible because of the chronic focus of infection in the ethmoid cells."

I have seen hundreds of patients who had severe seasonal pollen rhinitis, but none of them developed polyps *unless* they, in addition, had infection in the sinuses.

Rowe and Rowe¹¹ imply that food allergy is the usual cause of nasal polyps. This has not been my experience, but not all of my patients were investigated for nonreaginic food allergy.

ANALYSIS OF DATA

All fifty patients were followed for at least five years after their first operation. Nineteen patients were followed for ten or more years, and two have been observed for twenty years.

TABLE I
AGE OF PATIENTS AT THE TIME
OF FIRST OPERATION

1 to 10 years	0
11 to 20 years	1
21 to 30 years	2
31 to 40 years	13
41 to 50 years	14
51 to 60 years	12
61 to 70 years	7
71 or over	1

There were 25 females and 25 males in this series. Twenty-one of the fifty patients had asthma and one of these had Loeffler's syndrome.

Seventeen of the fifty patients had co-existing food or inhalant sensitivity requiring specific treatment.

As is shown in Table I all but three of the patients were over 30 years of age. It seems to me that if a patient were destined to develop food or inhalant sensitivity he would do so before the age of 30. This strengthens my conviction that bacterial allergy due to

sinus infection is a cause of hyperplastic sinusitis. A more descriptive term is chronic suppurative and hyperplastic sinusitis because pus and polyps within the sinuses were demonstrated at the time of operation.

The types of surgical treatment utilized are listed in Table II.

TABLE II
SURGICAL TREATMENT—50 CASES

1. Removal of polyps, bilateral intranasal ethmoidectomy and bilateral Caldwell-Luc	7 cases
2. Removal of polyps, bilateral ethmoidectomy, 3 times and later bilateral Caldwell-Luc	2 cases
3. Removal of polyps, bilateral intranasal ethmoidectomy and bilateral intranasal antrostomy	14 cases
4. Removal of polyps, bilateral ethmoidectomy, 2 times and bilateral intranasal antrostomy	4 cases
5. Removal of polyps, bilateral intranasal ethmoidectomy, 3 times, and bilateral intranasal antrostomy	6 cases
6. Bilateral intranasal ethmoidectomy	13 cases
7. Bilateral intranasal ethmoidectomy, 2 times	1 case
8. Bilateral intranasal antrostomy	2 cases
9. Unilateral intranasal ethmoidectomy and intranasal antrostomy	1 case

A review of the case histories of these patients suggests a few generalizations. 1) A high submucous resection of the septum is usually necessary to permit good visualization of the ethmoid area. 2) Removal of the anterior end of the middle turbinate or removal of the lateral half of the middle turbinate gives much better visibility. It has been said that intranasal ethmoidectomy is a "blind procedure." This is not the case if the above measures are carried out. 3) If the patient needs an ethmoidectomy he also needs an antrostomy on the same side. 4) Those patients having long-standing disease need a Caldwell-Luc procedure. 5) Hypertrophied obstructing inferior turbinates should be treated by linear cauterization. To perform linear cauterization I use a needle electrode attached to the cutting current. This sacrifices very little mucosa but creates a scar reaching

from the mucosa to bone. Soft hypertrophies on the upper third of the septum can also be reduced in size with this same electrode.

In 1922, J. L. Goodale⁷ presented before this Association a paper in which he described 16 cases very similar to the 50 I have presented. He wrote: "Operative treatment of sinusitis consisted in adequate removal of diseased conditions in the ethmoid bone with the establishment of large openings into the antra to permit the spontaneous evacuation of the antral fluid." He was well acquainted with food and inhalant sensitivity and excluded them as causes of this type of sinus disease. He also said that in his experience vaccines were of relatively little value so long as the disease state of the sinus persisted.

Goodale said: "In all the above cases skin tests for foods and pollens were negative, or if positive, the influence of these factors upon the asthma were definitely eliminated. Unless otherwise stated the operation consisted in the exenteration intranasally of the ethmoids and establishment of an opening into the antrum through the lower meatus."

After reviewing my cases I find myself with conclusions very similar to those written by Dr. Goodale 40 years ago.

Lederer¹⁰ has a section on hyperplastic rhinitis with the synonyms nasal asthma; nasal polyps; nasal polyposis. He lists the etiology as long-continued attacks of chronic rhinitis or sinus disease. Under the section headed "Chronic Ethmoiditis," he states: "In this disorder the inflammatory process is initiated by a similar pathologic process in the neighboring sinuses or the nose. The disease may be either suppurative or hyperplastic." In my experience it is usually both. In describing his technique of intranasal ethmoidectomy, Lederer says removal of the middle turbinate is helpful. Lederer also says "within the strict limitations of its indications, intranasal surgery of the ethmoid labyrinth has proved satisfactory." . . . "The surgeon must possess a greater combination of anatomic knowledge, caution and manipulative skill than is demanded in any other form of nasal surgery."

Grove⁸ says and I agree: "Postoperative treatment with antibiotics and steroid hormones, particularly meticorten, is most helpful in promoting good healing and avoiding a recurrence of the polyps. . . . The group which had had complete surgery showed over twice as much improvement as that with incomplete surgery."

RESULTS OF TREATMENT

Prompt relief of nasal obstruction which had caused loss of sleep was the most gratifying result of surgical treatment in all cases.

Thirty-seven patients required no additional polypectomies or sinus operations following their first operation. The nine patients who had Caldwell-Luc procedures and ethmoidectomy required no subsequent sinus surgery.

Following surgery, 42 patients had return of sense of taste and smell even though it had been absent from one to ten years.

Asthma was totally relieved or greatly benefited in 16 of the 21 asthmatic patients.

There were no deaths. One patient had severe bleeding at the time of operation and one patient had enough bleeding to require packing on the eighth postoperative day.

COMMENT

In my 1953 paper⁴ I stated that my purpose was to utilize the *smallest* amount of surgery that would produce permanent relief from symptoms. I am now convinced that extensive disease requires extensive surgery. One patient had polyps removed 100 times before I did bilateral Caldwell-Luc and ethmoid operations.

The treatment methods I listed in 1953 were as follows:

1. Antibiotics in bactericidal dose.
2. Vaccines to increase immunity.
3. Surgery to remove tissues which have suffered irreversible damage and also to provide ventilation and drainage.
4. Management of co-existing food or inhalant sensitivity.

Ten more years of experience have altered my ideas somewhat. Because 21 of the 50 patients had asthma, I have completely given up the use of penicillin. Anaphylactic reactions to penicillin are more frequent in asthmatic patients. I do use either erythromycin or tetracycline, two or three grams per day, during the week following operation. Some type of staphylococcus is the infecting organism in most of these patients.

I tried both stock and autogenous vaccines for a number of years but the results were so unconvincing that I have completely given up the use of vaccines.

Very thorough careful surgery seems to be the one method which has stood the test of time, but as is obvious from the previous data, repeated operations are necessary in some cases. Thirteen of the 50 patients had multiple operations. The conclusion seems obvious—that the first operation was not sufficiently complete. Two patients went elsewhere after my intranasal surgery had failed to give satisfactory relief. Both patients had bilateral Caldwell-Luc procedures following which their nasal symptoms cleared up and their asthma which had previously been severe gives no trouble except at the time of acute respiratory infections once or twice a year.

Schenck and Kern¹³ found that 83% of their asthmatic patients were improved by the Caldwell-Luc procedure and they state: "These data suggest that the Caldwell-Luc operation offers perhaps greater hope for relief than do other forms of antrum surgery and we believe that in the case of extensive antrum disease, notably with polypoid change, it is the method of choice."

In this particular series there was only one patient who had strictly unilateral suppurative and hyperplastic disease. This patient had had known food and inhalant sensitivity causing asthma for many years before she developed the sinus infection. She had recurrent asthma due to known food and inhalant sensitivity since childhood but did not develop the sinus infection until age 62.

In the paper presented before this Association in 1953,⁴ twelve of the fifty patients described had unilateral disease. This, I think, is the strongest evidence that hyperplastic sinusitis is due solely to bacterial allergy. Pus and polyps were present in only one side of the nose and roentgenological examination of these patients showed clouding of the antrum and ethmoid only on the side containing polyps. Some of these individuals did, in addition, have a history of food or inhalant sensitivity and had positive skin tests but the opposite side of the nose appeared normal and roentgenogram showed clear sinuses on the normal side.

Dixon⁵ in 1945 presented a study of the records of 125 of his patients who had required an intranasal ethmoidectomy and who had follow-up observation of at least 3½ years, some of them as long as

20 years. Almost all of his patients had satisfactory relief of symptoms. There were no deaths and no complications. It was Dixon's report that first aroused my enthusiasm for intranasal ethmoidectomy. I agree with his statement that failure to achieve satisfactory results may be listed under four headings: "1) Ill-advised attempts by those not qualified to perform this type of surgery; 2) the incomplete removal of pathologic tissue by those qualified; 3) uncontrolled allergy; 4) inadequate postoperative care."

Dixon stated: "For this study I have selected only those patients who are most difficult to manage, those having the entire nasal cavity filled unilaterally or bilaterally with polyps." Thirty-three, or 26%, of his patients had unilateral disease, a figure quite close to the 24% which I reported to this Association in 1953.

Thirty-four of Dixon's 125 patients had recurrences in five years and these patients averaged two operations each during that period of time.

We see many patients who have had chronic suppurative sinusitis for years who do not develop polyps. Those who develop polyps must have some defect in physiology which permits the tissues to become sensitized. Clinical observation makes me think that a relative deficiency of glucocorticoids is a factor permitting the development of the hyperplastic response to infection. Surgery alone would of course not correct this factor.

The following case report will serve to indicate why I think an endocrine insufficiency, probably adrenocortical, may be the basic defect in physiology which permits the development of this type of sinus disease. It also serves to show the limitation of some of the methods of treatment which were utilized with but temporary amelioration of symptoms.

REPORT OF CASES

The patient, J.S., was first examined by me in 1942, when she was 14 years of age. Her chief complaint was complete nasal obstruction on the left side of four months' duration. Her first operation in August 1942 was removal of polyps and left intranasal ethmoidectomy. A second operation in September 1949 was a bilateral intranasal ethmoidectomy, removal of polyps and submucous resection of the septum. At this time the patient also had granular hypertrophies on the lateral wall of the nose on each side in front of each middle turbinate. These areas were treated by electrocoagulation. All skin tests were negative. The third operation in May 1950 was a bilateral intranasal ethmoidectomy and removal of polyps. Three ethmoidectomies to remove regrowth of polypoid tissue should be enough to

make anyone realize that he was not getting at the real cause of the disease. The patient's fourth admission in August 1950 was for treatment of severe asthma. Previously she had had severe asthma every time she had a head cold. This also suggested bacterial allergy. Her brother had polyps and her mother had asthma which was relieved during pregnancy. This suggested poor endocrine inheritance.

She was admitted for the fifth time in November 1950 when her chief complaints were nasal obstruction, yellow nasal discharge, cough and asthma. Chest roentgenograms were "typical of tuberculosis," but repeated sputum examinations were negative for the presence of acid fast bacilli and smears showed the presence of many eosinophils. Her blood count showed 22% eosinophils. Treatment with penicillin, plus 75 mgm of cortisone daily produced rapid clearing of all nasal symptoms and complete disappearance of the pulmonary densities, thus indicating Loeffler's syndrome. The patient had six more hospital admissions during the next two years for treatment of suppurative rhinitis, bronchitis and asthma. Each time all nasal and bronchial symptoms cleared up within a week in response to cortisone, *with* or *without* penicillin. Each time cortisone, 75 mgm daily caused disappearance of polypoid tissue in the ethmoid areas and disappearance of the red, granular hypertrophies on the upper half of the septum with return of the sense of smell.

Her fourth and last operation in February 1953 was a bilateral Caldwell-Luc procedure. Smears made from the pus in her antra showed the presence of many eosinophils, many neutrophils and culture showed a non-hemolytic staphylococcus. She had a blood eosinophilia of 24%.

Cortisone was administered during the postoperative period. Ten days after discontinuing the administration of cortisone she had a recurrence of asthma and recurrence of the red granular tissue on the upper half of the septum on each side. Each time she was given two 25 mgm cortisone tablets per day her asthma cleared up promptly and the granulomatous tissue on the septum flattened out. During 1953 the patient followed a cereal free diet without benefit. Both stock and autogenous vaccines were tried without benefit and the autogenous vaccine made her symptoms worse, even when given in a very small dose. It soon became evident that she would be free of symptoms only when taking 1½ cortisone tablets per day. She had four courses of cortisone therapy during 1953. With each course her cough and asthma stopped within a week and the granulomatous tissue on the septum flattened out within a week.

She was married in July 1951 but never became pregnant until she started taking cortisone in February 1953. She had a healthy baby boy in December 1953. She was maintained almost continuously on cortisone and later on prednisone. She had a healthy baby girl born in July 1956. I lost track of her until May 1960. She had been treated for alleged "tuberculous" from November 1959 until April of 1960, when her attending physician wrote to me and found out about her previous diagnosis of Loeffler's syndrome. Since that time she has been in good health, her maintenance dose of steroid being 7½ mgm of prednisone daily. I saw her in June 1960; the nasal mucosa looked normal, there was no discharge, no crusting, the olfactory fissures were open and there was a wide passage up to each ethmoid. Both sphenoid ostia were visible and the linings looked normal. A follow-up letter in December 1962 said that she is in good health and had had no asthma since March 1962 when she had a cold.

This case report extending over 20 years is but one of many similar stories which have convinced me that poor adrenocortical function is one of the factors predisposing to the development of hyperplastic sinusitis and that a *high eosinophil count in the blood means poor adrenocortical function* and not food or inhalant sensitivity. Some of this patient's eosinophil counts were as follows: September

1949—12%; May 1950—12%; August 1950—5% after an adrenalin injection; November 1950—6% after i.m. cortisone; December 1950—5% after i.m. cortisone; January 1951—13%; June 1952—0% at the time of an acute infection; November 1952—19%; February 1953—24%.

The following case report shows the many errors that can be made by attributing the presence of polyps entirely to food or inhalant sensitivity.

CASE 2. The patient, H.S., a female, aged 61, was first examined by me in September 1947. She said she had had many head colds ever since she went to California in 1928 and she had had chronic nose trouble ever since. A submucous resection was performed in the fall of 1928 and she had had polyps removed four times, the last time in March 1946. She had been greatly troubled with nasal obstruction since early 1947 and she had had no sense of taste or smell since 1930. She had excess nasal discharge which was usually yellow, but she had no marked postnasal discharge and no cough. She had had many skin tests and was told she was sensitive to weeds, grasses, feathers, food and bacteria. She had had many allergy injections without apparent benefit. She is clinically sensitive to feathers and to overstuffed furniture. For three months she tried a diet of nothing but cooked fruit and chicken, but her symptoms were no better. She says the injections of staph and strep vaccine made her symptoms worse. She had also tried a penicillin nasal spray. Examination showed polyps in both ethmoid areas. All the anterior sinuses were dark on transillumination. Washings from the left antrum contained mucopus; no return flow could be obtained from the right antrum. Culture made from the sinus washings showed a pure growth of staph aureus. X-ray examination showed marked clouding of all sinuses. The treatment was bilateral intranasal antrostomy and bilateral intranasal ethmoidectomy. Following operation she had a return of sense of smell for the first time in 17 years. A Christmas card two years later said that her health was good and her nose was comfortable.

In my opinion poor adrenocortical function is a factor predisposing to the development of hyperplastic sinusitis, as well as to infectious asthma. This idea is not new. Garretson,⁶ a neurologist, wrote in 1935 as follows: "The symptom of allergy is always a vagotonic symptom and the cure of this symptom is obtained only by overcoming the hypoadrenia; therefore find out the cause of the hypoadrenia, eliminate this cause, activate the suprarenals, and you will cure the patient."

In the present study, fifteen of the patients who had asthma found that they frequently had to take small maintenance doses of prednisone, usually one or one and a half 5 mgm tablets daily in order to remain free of asthma. So long as they continued this schedule they continued to be free of nasal symptoms as well as free of asthma. Two of the fifteen found that they had more satisfactory relief when taking small maintenance doses of Acthar gel, 10 units, every day or every second day. Six of the fifteen found that acute respiratory infections could be quickly controlled only if they increased the dose of prednisone to three 5 mgm tablets daily.

It is experience with this group of patients that has convinced me that there is a defect in the pituitary-adrenal axis that permits the development of both hyperplastic sinusitis and infectious asthma.

One of the fifteen had a complication due to corticosteroid therapy. He was an extremely psychoneurotic individual who increased his corticosteroid dose while not under my care and developed a bleeding peptic ulcer.

Five of the patients who had received maintenance therapy with either Acthar gel or prednisone for several months were able to discontinue this type of medication and be free of symptoms for several years. So it is not true that initiation of corticosteroid therapy means lifelong dependence. Good judgment on the part of the physician and cooperation on the part of the patient are essential.

Brandon's book² entitled "Corticosteroids in Medical Practice" is an extremely useful reference. Brandon says: "Clinical experience in allergy and antibody studies indicates that the concomitant use of ACTH or cortisone in doses used in man does not interfere with the hyposensitizing process to specific pollens or other antigens.

"During cortisone or ACTH administration the delayed inflammatory tuberculin type reaction is prevented or abolished within a few days in animals and men.

"There is a normal diurnal variation of corticosteroid levels in man. The maximum output of 17-hydroxycorticosteroids in the urine is between 8 and 10 A.M., being about three times higher at that time than between midnight and 2 A.M." This I think is the explanation of why most asthmatic patients have acute attacks during the hours following midnight.

The normal daily adrenocorticosteroid output in man has been estimated to be the equivalent of 12.5 to 40 mgm hydrocortisone per day. Plasma levels of adrenocorticosteroids in pregnant women may reach 400% of the levels in non-pregnant females. This fact probably explains the case histories of women who are subject to asthma except during the last six months of pregnancy.

Brandon says: "Adrenal insufficiency may occur in times of stress and require small supplements of corticosteroids. Reduced tolerance to stress is more common in the older age groups and in debilitated patients who have had repeated operative procedures."

Cooke³ wrote: "A type of eosinophilic infiltration is also well known in the so-called hyperplastic or hypertrophic forms of rhinitis and sinusitis, with or without polyps.

"We have been increasingly impressed by the presence of blood eosinophilia in asthma of purely infective origin.

"When the infection is localized in the sinuses an allergic reaction to bacteria may result, with subsequent formation of eosinophil cells in the infected tissues and thence in their secretions, but this only occurs in those individuals capable of sensitization.

"In infective asthma secondary to sinus disease an excess of eosinophil cells in the sputum and antral discharge is practically a constant finding, whereas an increase in the blood is found in but 61% of our series. These facts lead us to believe that in this condition the eosinophil production is a local histogenous one and represents a local bacterial allergic reaction."

The fact that there are large numbers of eosinophils in the exudate as well as in the tissues of patients having bacterial allergy has been amply demonstrated by Grove and Cooke⁹ and Silcox,¹⁴ as well as in our own patients.

Baldwin¹ says: "The physical examination of the asthmatic patient should start with the nose. Here, the presence of nasal polyps or pus in the nasal passages usually indicates infection.

"Rarely is the use of antibiotics by inhalation preferable to adequate oral administration.

"Nasal surgery has an important place in the management of infectious factors in the asthmatic. In children, infected adenoids and tonsils should be removed.

"When antrums are infected, repeated irrigations may clear up the infection. When improvement does not result, the cutting of a window or permanent opening into the antrum is rarely a rewarding procedure. Here the infection still remains in the antral membranes. The operation of choice is the Caldwell-Luc with exenteration of infected tissues.

"Many of these patients need the thorough exenteration of the Caldwell-Luc operation.

"Many of the bad results attributed to nasal surgery, especially in this category, are due to incompetent and incomplete surgical technique. Vaccine therapy in the management of infectious factors in asthma is not a substitute for antibiotics or surgery when indicated."

The need for multiple operations, meaning the need for an operation one, two or three years after the first one can, of course, be attributed to inadequate or incomplete surgery the first time. Another fairly obvious conclusion is that a new infection can be the stimulus which reactivates the hyperplastic tissue response in a susceptible individual. Prompt control of acute infections by antibiotics plus administration of corticosteroids in dose no larger than 10 mgm per day of prednisone will in most cases promptly control the acute infection and prevent recurrence of the hyperplastic tissue.

CONCLUSIONS

1. Hyperplastic sinusitis is a manifestation of bacterial allergy.
2. Complete surgical removal of diseased tissue is necessary in advanced cases.
3. A temporary or permanent subclinical adrenocortical insufficiency is a conditioning factor leading to the development of this type of disease.
4. Patients having this disease have an inherited or acquired endocrine defect predisposing them to recurrence.
5. Small maintenance doses of corticosteroids can prevent recurrence.

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DISCUSSION

DR. SCHENCK: Dr. Davison is to be commended for his courage in challenging some old conceptions in regard to this controversial entity and for his originality in offering fresh suggestions as to its etiology. He has offered convincing evidence, based upon his study of 50 patients, that chronic hyperplastic rhinosinusitis is the result of the synergistic action of allergy and infection, and that the ultimate polyposis is due to the antigenic effect of micro-organisms, chiefly staphylococci present in the soft tissues. The age differential in his reported cases is offered as evidence that inhalant rather than food allergy fosters bacterial sensitization; this age incidence may also be attributed to the fact that perennial nasal allergy may be present for 20 to 30 years before nasal occlusion due to polyps or the onset of bronchial asthma impel the patient to seek medical aid.

If the concept of bacterial sensitization is accepted then the surgical principles outlined by Dr. Davison must be approved because wherever micro-organisms operate as an antigen, failure to remove them perpetuates local tissue changes whereas their removal eventuates in regression or arrest of these changes. He is, therefore, on sound ground when he states that "aeration and drainage" is inadequate and that bacterial antigen must be eliminated by the total removal of the tissues containing the responsible micro-organisms.

There is increasing evidence to support the theory of bacterial sensitization. Electrophoretic analyses of polyp fluid, carried out with concomitant histologic studies of polyp tissue, indicate that specific antibodies (reagins) are present in polyp fluid and that not only eosinophiles but also lymphocytes, plasma cells and granulocytes infiltrate the polyp. The fetish of the eosinophile has obscured the role of other cells. There are higher gamma globulin values in polyp fluid than in serum and the content of antibodies in polyp fluid may occasionally be 100 times

that of the serum. From a biochemical point of view, these antibodies possess the characteristics of gamma globulin. Ample plasma cells are present in most nasal polyps and there is an absolute and relative increase of gamma globulins with the number of plasma cells directly proportional to the increase in gamma globulins; few plasma cells are found in polyps with low gamma globulin values. No correlation has been found between the number of lymphocytes and eosinophiles present in the polyp and the gamma globulin content. Since the higher the gamma globulin concentration, the greater the plasma cell infiltration in the polyp, it is logical to conclude that there is local production of gamma globulin in the polyp and this is supported by the histologic demonstration of sufficient numbers of plasma cells and lymphocytes to provide for the local manufacture of antibodies.

The recently discovered role of the basophile in producing the lesion of cold urticaria also bears mention here. Although it makes up no more than 2 per cent of the leukocyte population it is loaded with chemicals involved in a variety of critical physiological processes—heparin, histamine and perhaps plasmin as well; it may prove to be the cell responsible for the histamine shock of anaphylaxis. Mast cells appear more prominently in chronic inflammation and, therefore, may be of significance in chronic hyperplastic rhinosinusitis.

I congratulate Dr. Davison on his presentation which contains so many intriguing angles that for the first time I regret the limitation of time for further discussion.

DR. HENRY L. WILLIAMS: Mr. President, Members of the Society and Guests: I will take the hint that has just been given and try not to give a paper. Fortunately I didn't prepare anything in advance even though Dr. Davison was kind enough to send me an advance copy of his paper.

There is much in this paper that I agree with; especially the general principles that he has enunciated. Of course one can't make a good discussion—at least I can't—by agreeing with anything, so I will pick out the few points that I found after diligent search that I would like to disagree with.

Amongst them first and foremost is his suggestion that the middle turbinate be removed in intra-nasal surgery. This is something that I am strongly opposed to. I believe that the removal of the middle turbinate will eventually result in atrophic changes not only in the nose but in the posterior pharyngeal wall and these patients eventually become very uncomfortable. I think that the functioning tissue of the turbinate should be preserved as carefully as possible. One should make every effort to preserve it. This is not always possible, of course. We are not all perfect surgeons—at least I am not—and sometimes the thing is gone after I get through, but it isn't because I didn't attempt to preserve it.

And another thing that I am not very much in favor of is the cauterization that he suggests. I very much prefer, if it is absolutely necessary to get more space because of the hypertrophy, or hyperplasia, whichever it is, of the inferior turbinate or middle turbinate, submucous resection of the anterior end of the turbinate. I think one particular proposer of this technique was Howard House, who has retreated from rhinology and become a mere otologist. This I strongly advise because it does not destroy the cavernous tissues.

The third point of disagreement: nothing is mentioned here about the naso-antral window. It is all Caldwell-Luc. Of course I know that in the East it would seem that no one knows how to do a proper naso-antral window, so it should not amaze me that he advocates the Caldwell-Lucs. If a proper naso-antral window is done, however, one proceeds with the operation with greater speed. There is less

extensive tissue injury and consequently faster healing. One has almost as complete a visualization of the interior of the antrum as one does through the Caldwell approach—I object to adding Luc's name because actually his procedure is nothing like the operation suggested by Caldwell. Luc advocated taking off the middle and inferior turbinates and resecting the entire naso-antral wall so as to turn the antrum into part of the nasal cavity. This is not my idea of restoring normal function to the maxillary antrum. I prefer therefore to refer to this operation by the name of the distinguished Canadian physician who suggested this technique of draining the maxillary sinus via the nasal floor.

As a fourth point for discussion I would like to suggest that one of the difficulties we have in considering hyperplastic sinusitis as an expression of allergy is that actually we don't know what an allergy is. The immunologists have not completely solved the problem. We talk glibly of the effects of allergy, but do we know what we are talking about? I have "committed" papers on this subject which I sometimes regret because I can easily see now that I was in a state of considerable ignorance at the time I wrote them. Now I know so much that I know I know nothing. I hesitate to ascribe hyperplastic sinusitis to bacterial allergy. I am not sure what bacterial allergy is. Possibly even Dr. Schenck doesn't know exactly what it is. Therefore I think that while the therapeutic, surgical and medical principles that have been adduced can hardly be improved on, I doubt that the hypothetical explanations are completely adequate to the subject. I think the immunologists are going to have to do a lot more work before we can say that we have conquered this particular problem.

I heard no mention of the local use of cortisone or the corticosteroids, which I believe has been mentioned by Dr. Baker and others. I was thinking particularly of the work of Drs. Dill and Bolstad. I believe that this should be seriously considered in the after-care of these patients. If a tendency toward recurrence appears, local steroid therapy will counteract it better than parenteral use without the side effects which can occur.

DR. FRED W. DIXON: Mr. President, Fellows, and Guests: I wish to thank Dr. Davison for a preview of his paper. I think his success can be due to two things: control of the allergy and infection, and second, adequate operation, giving ventilation and drainage.

While I try to control the allergy, I do not unduly delay the operation for, postoperatively, the patient is frequently symptom free.

I have no patience with the rhinologist who removes polyps piecemeal at each office visit.

Dr. Davison performs more intra-nasal antrotomies than I do during his ethmoidectomies. I find that by unblocking the ethmoid sinuses, the infection in the other sinuses will often resolve.

Antibiotics, plus irrigation of the maxillary sinus through the natural ostium, is frequently sufficient.

One point that I wish to stress is to leave as much of the middle turbinate as possible. When this is done, a curtain remains, thereby deflecting the inspired air. This prevents the air from striking directly the posterior pharyngeal wall which produces a dry mucosa and crusting (the hot spot that causes so much discomfort to the patient in the winter).

I would like to ask Dr. Davison whether he has had success with Depomedrol.

I believe Dr. Davison has shown the fallacy of "once a sinus, always a sinus" and in its stead has shown that, while a guaranteed cure cannot always be promised, the patient can be assured of living in comfort.

DR. DAVISON (closing): Mr. President and Fellows: I would like to thank my discussers for being so charitable. Some of my ideas are not orthodox.

I think that Dr. Schenck's term chronic hypertrophic rhinosinusitis is a good one.

Most of these patients are in the age group subjected to severe stress. I really think that these people have an exhaustion of some part of the endocrine system. I think the high eosinophil counts are an index of poor adrenocortical function rather than of "allergy."

I was glad that Dr. Williams brought up the question of how much one should remove of the middle turbinate. I remove only the anterior end or the lateral half. It is perfectly possible to take a knife, split the turbinate and take off the polypoid tissue laterally, thereby preserving the olfactory fissure. That, of course, is what lets these people have a return of their sense of smell. Five to ten years' observation has shown that they do not develop atrophy or crusting. Dr. Williams evidently slept through the projection of my Table No. II, which showed that 27 patients had intranasal antrostomy and that only 9 had a Caldwell-Luc type operation.

Despite his disparaging remarks I think Dr. Williams' papers dealing with the subject of allergy are still important contributions. Dr. Williams and I both admit our ignorance of the fundamental defect in physiology permitting the development of bacterial allergy. He dumps the problem in the lap of the immunologists. I think progress can be made by a good rhinologist working with a good endocrinologist.

I appreciate Dr. Dixon's remarks. His paper, as I said, stimulated me to do more intra-nasal ethmoidectomies.

I haven't had enough experience with Depomedrol to give an opinion.

I think Dr. Baker's paper describing the local use of steroids last year was an important one. I didn't include his ideas in this paper because I was describing people who had very extensive disease.

THE VALUE OF VACCINE THERAPY IN RESPIRATORY INFECTIONS

EDLEY H. JONES, M.D.
AND
PATRICK G. McLAIN, M.D.
(by invitation)

VICKSBURG, MISS.

The purpose of this presentation is to call your attention to a weapon in our therapeutic armamentarium for treatment of respiratory infections which, apparently, is currently neglected; but which, when properly employed can be quite effective. I refer to bacterial vaccines.

Respiratory vaccine therapy, understandably, is a controversial subject. For example, the immunizing properties of typhoid vaccines are generally accepted, but it is also recognized that routine inoculation will not always provide immunity to massive exposure.³ Vaccines made from bacteria found in the respiratory tract do not possess as great immunogenic properties as typhoid vaccine, and the same degree of response cannot be expected. Numerous studies have been reported showing clinical response. At least one study⁷ has been made of patients without infection compared with patients suffering with staphylococcal infection, reporting the titer of agglutinins and units of antitoxin in the blood before and after a series of ten injections of combined staphylococcal vaccine and toxoid. Definite increase in levels were noted after treatment; there was, generally, less increase in the infected group, as would be expected. The tuberculin type of bacterial allergy has been proven but the sensitizing and immunizing type cannot be similarly proven and is not so generally accepted; while the principal proof is clinical observation, many clinicians agree that bacterial allergy of both types is a clinical entity. This boils down to the fact that those who obtain good results with

vaccine therapy heartily endorse it; but are quick to point out that it is of value only when used properly, and in properly selected cases.

In earlier years there was a great deal of interest in this subject, Baird² reporting his review of nearly 400 articles published up to 1955, and Blatt's⁴ review of microbial allergy listing 829 references "nearly all published in 1956 or 1957." Since then there has been comparatively little interest, a fairly careful review of the literature from 1958 to 1962 failing to reveal a single published article on bacterial vaccines, though many were published on viral vaccines.

Apparently it has been presumed, and more or less accepted as a fact, that antibiotics will eliminate respiratory infections. This is far from true. Unquestionably, antibiotics have saved many lives, but they have not eradicated respiratory infections, and never will. Further, we believe that the frequent and promiscuous use of antibiotics for mild respiratory infections interferes with and lessens antibody response, the patient becoming more and more susceptible with more and more recurrences. We are seeing an increasing number of such patients. Careful studies fail to reveal any pathology other than the current infection. It is worthy of comment that the majority are not allergic patients. The commonly accepted conclusion is that they have lowered resistance and are very susceptible to respiratory infections. We think this a passive, unsatisfactory explanation and believe the basic etiologic factor to be inadequate antigen-antibody response on the part of the patient. We think it important to recognize this basic factor and, along with indicated treatment of the current infection, prescribe therapy to stimulate greater antigen-antibody response. We employ vaccines for this purpose and our results show that at least 90% of selected cases are definitely benefitted by proper vaccine therapy.

There are also the difficult groups of patients reported by Goodale⁸ that have required and undergone sinus surgery, yet continue to suffer with chronic infection that are benefitted by vaccine therapy.

Vaccine therapy is not a "cure-all" for respiratory infections and should not be used promiscuously. The fundamental principles should always be followed with every patient—a careful history, thorough examination, adequate laboratory and x-ray studies, and proper therapy, including allergy regime when indicated, and necessary surgery. These procedures are successful in the great majority of cases. When not successful, and the patient suffers from chronic infection, or

recurrent episodes of acute infection, proper and adequate vaccine therapy will be effective in a large percentage of patients.

For the past 35 years one of us has employed vaccines as an adjuvant in the treatment of allergic rhinitis. In recent years we have been fortunate in having many problem cases, such as those described above referred to us. During these years we have worked out a method that is usually effective. However, experience has shown that no one method is successful with all patients and different techniques must be employed in difficult cases. For this reason we will review techniques reported by others before presenting our experience.

A few years ago oral vaccines aroused some interest, possibly because of the ease of administration. They are still available and are still being recommended by some physicians. The rationale of oral vaccine therapy has never seemed logical to us and we do not recommend it.

The first vaccines prepared for injection were of necessity autogenous vaccines, and autogenous vaccines are still widely used. One variation is to grow the organisms on blood agar using the patient's blood, it being presumed only organisms pathogenic to the patient will grow; we have tried this method and our experience has not impressed us that it has any value. Autogenous vaccines can be made by any good clinical laboratory, usually contain only bacteria considered pathogenic and can be prepared in the desired concentration. Goodale and Mangiaracine⁸ reported 88% improved to excellent results in a series of refractory patients with autogenous vaccine of "young cultures" (three or four hours) of coagulase positive hemolytic staphylococcus aureus, the vaccine including the culture medium. Anderson and Hauser¹ recommend autogenous vaccines made from the original cultures, not subcultured and not washed, inactivated by a chemical rather than by heat (which they believe destroys much or all of the antigenic properties). Autogenous Bacterial Antigen Complex[®] (mentioned below) can also be prepared. Crump⁵ obtained cultures from asthmatic children by bronchoscopy stating that "... in our hands the use of bronchoscopic vaccine in asthma in children is the *sine qua non* of therapy." Baird² does not recommend autogenous vaccines, stating that in the growth of a culture "... the bacteria have changed their characteristics, and have descended about 72 generations more or less from the original bacteria."

There are now available many and various preparations of stock vaccines, including various combinations of catarrhal or respiratory vaccines, as well as staphylococcic, streptococcic and staphylostreptococcic vaccines. Variations such as Sero-Bacterins,* toxoids and toxoids combined with vaccine are available. Serobacterins are prepared by treating the bacteria with immune rabbit serum and these preparations must not be given to patients allergic to rabbit serum. Bacterial Antigen Complex† is available in two types and, as mentioned above, autogenous preparations can be made. Staphage Lysates‡ are available for aerosol and injection. We have had no experience with the latter two preparations.

The usual method of administration has been to give an initial dose of 0.10 cc to 0.25 cc subcutaneously, increasing subsequent injections by the same amount but not usually giving larger doses than 1.0 cc. The smaller doses may be given at three to five day intervals, the larger doses at weekly intervals. Maintenance doses may be repeated every two to four weeks. Some allergists have combined vaccines with other antigens in desensitization therapy, but in recent years many allergists have employed the intradermal method only.

It is generally agreed that an intradermal injection stimulates at least ten times the antibody response of the same dose given subcutaneously and is much safer, never causing systemic reactions. Hansel⁹ described a method of "optimum dosage" giving minute doses of staphylococcus toxoid intradermally. At the other extreme, Baird² recommends huge doses given subcutaneously and uses Serobacterinaccine Mixed with a high bacterial content (7 to 8 billion organisms per cc in the preparations available). He recommends doses of 0.2 cc, 0.4 cc, 0.8 cc, 1.2 cc, 1.8 cc and 2.5 cc, repeating the latter dose several times when adequate. If favorable results are not observed he increases the dose 0.5 cc each injection until reaching 5.0 cc, divides doses over 1.0 cc giving one-half in each of two sites, divides doses over 3.5 cc in four injections, in some cases repeats smaller doses every 2 to 4 weeks as maintenance therapy. In discussing reactions to these large doses he states, "Based on experience over a quarter of a century I can say categorically that the large doses are never dangerous. Occasionally, and only occasionally, they cause a fairly severe *immunity* type reaction (chill, temperature, etc.) but I have never known them to cause a *sensitivity* (dangerous) type of reaction."

* Merck, Sharp & Dohme

† Hoffman Laboratories

‡ Delmont Laboratories

The procedure we follow, as mentioned above, is to first obtain a detailed history and make a complete examination, with adequate clinical laboratory studies, x-rays and diagnostic skin tests when indicated, surgery if necessary. We agree with Davison^{6a} and others that the diagnosis of bacterial allergy can be made from the history. Certainly, an adequate history of chronic or recurrent respiratory infections can be obtained. One important finding when examining patients between acute episodes is a thicker-than-normal mucus discharge, perhaps a little more profuse than normal, heavily laden with bacteria. We collect such specimens in a sterile Davison specimen collector for culture. Another important factor is the hemoglobin level. It is true that many patients, particularly children, get along satisfactorily with a hemoglobin of 12 grams but we insist that all these "problem cases" have, or are built up to a hemoglobin of at least 13 grams, preferably 14 grams.

As a rule, we make intradermal tests with vaccines before prescribing vaccine therapy. Over the years we have varied the vaccines and will try any new one that offers any additional value, including it thereafter if its value is established. At present we are using: 1) Cutter Laboratories' M.V.R.I. 057-16, 2) Hollister-Stier Laboratories' Bacterial Vaccine, 3) Lederle Laboratories' Catarrhalis Combined Bacterial Vaccine 1974-37, 4) Merck Sharp & Dohme's H. Influenzae Serobacterin Vaccine, Mixed No. 4750, 5) National Drug Company's Staphylococcus Toxoid-Vaccine Vatox, and 6) Parke-Davis and Company's Catarrhalis Vaccine (Combined) Bio. 615. Since we make these six tests at one time we dilute the vaccines with equal parts of sterile diluting fluid and the patient is given 0.02 cc of each intradermally. The 30 minute flare reaction is recorded, as well as the 24 hour erythema reaction. The flare reaction is of more importance in estimating the value of Serobacterins and toxoids, the 24 hour reaction of more importance in the others.

If the patient has minimal reactions to these tests vaccine therapy is not recommended. A rare exception to this rule is when the patient's history reveals repeated bacterial infections, particularly staphylococcic infections. If the patient has definite reactions to some of the tests but not all, only those vaccines causing reactions are employed, combining them so that patient gets only one injection. We prefer intradermal injections and the size of the test reactions indicates the dose. If the reactions are severe the composite vaccine (individually made up for each patient) may be diluted 1:1, 1:2, or

1:3, depending on the severity of the reactions, later and gradually increasing to full strength. The starting dose is always 0.02 cc and the patient is requested to note the 24 hour erythema reaction and report it when returning for the next injection. We get more accurate reports by asking them to place a coin over it; was the area of erythema larger than a 25c piece (24 mm), a 50c piece (30 mm)? We desire a 24 hour erythema reaction of about 30 mm diameter, subsiding in another 24 to 48 hours. As long as the desired reaction is obtained that same size dose is repeated but when the reaction is smaller than 25 mm the next dose is increased 0.01 cc and such increases are continued as necessary to obtain the desired reaction; it being remembered that doses larger than 0.10 cc cannot be satisfactorily given intradermally. When the reaction is larger than a silver dollar (38 mm) the next injection is decreased; and *it is a cardinal rule that succeeding injections must not be given until all erythema from preceding injections have subsided*. (A small area of reddish induration, 3 to 5 mm in diameter may persist for weeks; this is not to be confused with erythematous reaction).

Short courses of injections are rarely effective and we seldom prescribe a series of less than ten injections, usually continuing this therapy for six months. If the treatment is started during the summer months, we like to continue it through the winter months, and until our bad weather is over.

Our experience shows this type of therapy effective in about 80% of our patients. When this has been followed for as long as 2 or 3 months without noting perceptible improvement we do not admit defeat but re-evaluate the patient and our treatment. The history is reviewed to be sure we have not overlooked anything pertinent. We re-examine the patient, again running smears and cultures. If pathogenic bacteria are present, we repeat the antibiotic sensitivity culture. If no real change is noted and the problem is lack of response to therapy, we run a gamma globulin estimation. Davison^{6b} has emphasized the value of this test, and of administering gamma globulin along with indicated antibiotic therapy, and continuing vaccine injections. If the gamma globulin level is normal and the culture shows pathogenic bacteria that we think will respond to autogenous vaccine therapy, we have an autogenous vaccine made. We always check the potency of autogenous vaccine with intradermal tests. We have used autogenous vaccines, usually intradermally, but have employed both intradermal and subcutaneous methods concur-

rently in some patients. Our results with autogenous vaccines have not been sufficiently impressive to justify our using them routinely. We have obtained good results in some cases by combining intradermal injections of toxoid (usually staphylococcus toxoid, but will use streptococcus toxoid or the combination if this is indicated by previous studies) and subcutaneous injections of Serobacterin-Vaccine Mixed[®] following the method of Baird,² though we have never given the very large doses. We are not sure the latter is not a form of protein shock therapy rather than immunizing or desensitizing therapy, but we do know we have obtained clinical results in some patients by this method.

The patient's response is individual, and this individual response is our guide not only for increase of dosage but also for duration of therapy and repeating the course. We find some patients can take a course of only 10 or 12 injections with prompt response, and may not require any further therapy for years; others will get good results but voluntarily return in a few months for another series of injections, some taking a short course each fall; the real "problem cases" may require therapy for two or three years, suffering some degree of relapse whenever treatment is discontinued. We routinely continue concomitant intradermal vaccine therapy in all patients with allergic rhinitis as long as they require desensitization therapy with other antigens.

The following reports illustrate typical cases:

REPORT OF CASES

CASE 1. Mrs. R.L.M., WFM 42, housewife and social worker, seen August 24, 1959 with history of frequent respiratory infections the previous winter, again suffering recurrences and requesting treatment for prevention of these episodes. History was negative for allergy and otolaryngologic examination was negative. Intradermal tests with half-strength vaccines revealed a 24 hour moderate reaction to one, moderately severe to two and severe to two. These were combined and a series of 15 weekly intradermal injections was prescribed. She returned the next year (Sept. 26, 1960) with a history of complete relief from respiratory infections and was so pleased she requested the course of treatment be repeated, and it was again prescribed. She was last seen on January 26, 1963 with a report of having been free of respiratory infection until December, 1962, though she had not had any vaccine since January 10, 1961. At her request, vaccine therapy was again prescribed.

This report illustrates the type of case that requires a series of injections every year or two.

CASE 2. Mr. B.H., WMM 40, plumber, was seen October 18, 1958 with an infected right radical mastoid cavity and an allergic rhinitis. The ear condition was finally diagnosed therapeutically as atrophic otitis, both conditions due to allergy to

dust, molds, and bacteria. Desensitization therapy and the usual allergic regime were prescribed. This was continued until December 2, 1960 when he no longer reacted to dusts and molds and that therapy was discontinued; vaccine therapy was continued until March 25, 1961 when it, too, was discontinued. He was re-tested for dusts and vaccines on August 25, 1961 and vaccine therapy prescribed during the winter, discontinuing it in February, 1962. He has been tested twice since then, last on August 3, 1962 with negligible reactions, and no treatment other than cleansing the radical mastoid cavity every 6 months; and has been free of symptoms.

This report illustrates the type of case that requires treatment for 2 to 5 years before building up enough resistance to discontinue therapy. How long this particular patient will be free of symptoms is problematical but some of these patients remain free of symptoms for years; others have to return in a few years for additional therapy.

CASE 3. Mrs. L.O.C., WFM 28, housewife and social worker, was first seen on April 30, 1959. She was referred by an internist at a local Clinic with a history of severe cough of 3 years' duration starting each Fall and lasting through Spring, present during day but worse at night, with severe paroxysms; stated she may have had some wheezing. She had consulted several physicians who had prescribed various antibiotics, antihistamines and cough remedies, with little benefit. She had a very thorough examination at the Clinic which included routine blood studies, BMR, chest and sinus x-rays and bronchoscopy; all of which were reported normal except slight cloudiness of the right maxillary sinus; and the culture obtained by bronchoscopy grew slowly, reported pneumococci and non-hemolytic streptococci. This history was quite suggestive of respiratory allergy and diagnostic skin tests were included in her examination. Surprisingly, her reactions to pollens, dusts, molds and miscellaneous antigens were all negative; she had negative reactions to three of the five vaccine tests, but very definite reactions to the other two. Nasal examination revealed some thick discharge draining from the right antrum; the nasal smear was reported + neutrophils, + mononuclears, no eosinophils and +++ mucus with +++ bacteria. An antibiotic sensitivity culture was requested. We made a tentative diagnosis of tracheobronchitis, secondary to residual infection of the right maxillary sinus; told the patient we would treat her with antibiotics and vaccines but if she did not respond promptly, we would advise sinus surgery. The indicated antibiotics were prescribed along with a series of eight weekly injections of vaccines. She reported some relief from coughing after the first two injections, then had a mild URI, took a second course of the same antibiotic, continued the injections and was completely relieved after 3 months; however, weekly injections were continued until December, 1959. She returned in July, 1960, pleased with her progress and requested prophylactic vaccine therapy. She was re-tested with vaccines and had reactions to four; these were combined and prescribed. She was last seen May 12, 1962, reporting she had had very little trouble since her visit two years earlier, but had noted some postnasal discharge the previous weeks. Again the nasal smear failed to reveal any eosinophils and was reported + neutrophils, +++ mucus and + bacteria. Vaccine tests were again made and this time she had very definite reactions. Vaccine injections were again prescribed.

This report illustrates a case whose history suggested respiratory allergy but reacted only to tests with bacterial vaccines, yet obtained relief from vaccine therapy. It is possible she will require a course of vaccine therapy every year or two, but she has obtained relief without sinus surgery.

CASE 4. Mrs. R.R.M., Jr., WFM 29, housewife, was referred to us on April 3, 1961 with a history of recurrent pyelitis with one attack of pyelonephritis, refrac-

tory to all treatment which had included cystoscopies, repeated cultures and many courses of antibiotics. The referring physician noted she had recurrent attacks of tonsillitis, most of which were accompanied by pyelitis and expressed the opinion that tonsillar infection was a contributing, if not the etiologic factor. When seen she had a moderately acute upper respiratory infection and we requested sinus x-rays, which were negative. An ABS culture was run, the indicated antibiotics prescribed and as soon as the infection was under control tonsillectomy was performed under local anesthesia. Although antibiotics were continued during hospitalization she had a flare-up of pyelitis following surgery, requiring a longer hospital stay. The tonsils were sent to the laboratory in a sterile container; culture revealed Beta hemolytic *Staphylococcus aureus*, *staphylococcus albus*, Beta hemolytic streptococcus, *hemophilus influenzae* and *neisseria catarrhalis*. An autogenous vaccine was made from the staphylococci and streptococci. This vaccine was administered by the referring physician and the record is not available, other than that it was given subcutaneously, starting with a small dose and increasing to a maximum of 1 cc, with some local reaction, and at least 10 injections were given. She was next seen on July 5, 1961 with a moderately acute rhinosinusitis. It was then learned her course had not been satisfactory; while the episodes were milder she had continued to have recurrences of both respiratory and kidney infection. Incidentally, the nasal culture then obtained did not grow either staphylococci or streptococci. After controlling the acute episode we advised further vaccine therapy under our direction, and gave her weekly intradermal injections of her autogenous vaccine with subcutaneous injections of *Hemophilus Influenzae* Sero bacterin. These were continued until her autogenous vaccine was exhausted in February, 1962. Tests with stock vaccines were then made, and treatment continued with these until May 1962, when vaccines were discontinued. During this period she had shown steady improvement, with only one episode of urethrotigonitis about 10 days after starting injections, a mild URI in September; and a moderately severe RI in January, 1962, and was pleased with her progress.

She was not again seen until November 13, 1962 when she reported she had been free of both kidney and respiratory infection until the previous week, and was suffering with mild acute pharyngitis at that time. As soon as that episode was controlled, vaccine therapy was again instituted. The following week she was back with another mild respiratory infection but since then has been free of both respiratory and GU infections.

This report illustrates a case of recurrent respiratory infections with much more severe recurrent genito-urinary infections which had not responded to repeated courses of antibiotics and other therapy; failure to recover fully after removal of diseased tonsils followed by a short course of autogenous vaccine; and slow but good recovery with continued vaccine therapy. Her future course will determine how long vaccine therapy will be required.

There is one other situation in which we have found vaccine helpful. We have all been consulted by patients complaining they are catching cold, the only symptoms being some malaise and possibly some sniffing, examination negative except for mild congestion of the nasal and pharyngeal mucosa. These are usually viral infections and the prescriptions of choice have been rest and aspirin. In recent years an antihistamine has been combined with aspirin in such preparations as Coriforte,[®] or with an oral decongestant as in Triaminic,[®] or all three as in Triaminicin[®] and Ornade.[®] Sulfonamides or broad-

spectrum antibiotics are often prescribed to prevent secondary bacterial infection. In this type of case we have found that a small intradermal injection of vaccine, 0.02 cc to 0.05 cc, depending on the patient's age, weight and previous experience with vaccines, seems to add to the effectiveness of the above measures.

SUMMARY

The value of bacterial vaccine therapy has been presented and emphasized. Such therapy is not a "cure-all" but can often be effective in patients with chronic respiratory infections, or who suffer repeated episodes of acute respiratory infection and have not responded to other therapy.

While the literature is too voluminous for detailed review, the various methods reported effective have been discussed.

We have described our methods which have been effective in at least 90% of selected cases.

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DISCUSSION

DR. ROBERT L. GOODALE: The subject of vaccine therapy for respiratory infections, as we all know, has gone through various periods of popularity and disbelief, but I think that of late we have had a greater awareness of the value of vaccine therapy both for improving the antibody level in the patient who is repeatedly afflicted by specific bacterial infections and also for desensitizing those cases who have an allergic history complicated by infection of the respiratory tract. In some instances, as we know, these cases are primarily allergic to some other factor, such as pollen, dust, food, drugs, and so forth; but also, they are subject to bacterial infections. In my experience these are chiefly Staphylococcic diseases and it is fully as necessary to desensitize to the specific bacterial allergen as it is to the pollen or other substance whatever it may be. In certain instances it has seemed to me that where the allergist has cleared the patient by negative skin tests for allergens and the patient still has allergic symptoms and obvious signs of chronic bacterial disease, it may well be that these are essentially cases of bacterial allergy. This is not a new concept. My father, Doctor J. L. Goodale, in the *Boston Medical and Surgical Journal* for February 17, 1921, in an article entitled, "Preliminary Notes on Skin Reactions Excited by Various Bacterial Proteids in Certain Vasomotor Disturbances of the Upper Air Passages," cited fifty cases in which Staphylococcus albus and aureus chiefly, and a few others such as Staphylococcus citreus and Micrococcus tetragenus, had been isolated from cases which had very definite allergic histories and on whom positive skin tests had been elicited by the intradermal method. These tests were controlled by injection of a sterile solution which did not produce the same reaction which we characteristically call a positive allergic test.

Be that as it may, as time went on, bacterial allergy was practically forgotten and I think we have now come full circle around so that it is a tremendous help to desensitize for Staphylococcus aureus where the patient has chronic sinusitis with recurrent polyposis and hyperplastic rhinitis and in whom even the most thorough surgery seems to be only partially successful. In fact, many of my cases have only responded satisfactorily when they were put on long courses of autogenous Staphylococcus vaccine, given according to allergic methods of gradually increasing doses and carried around the year on a perennial schedule.

Doctor Jones has used other bacteria than Staphylococcus aureus and seems to be getting extremely good results. My own feeling is that we should use organisms other than Staphylococcus aureus where it is a question of allergic sinusitis. In these cases, the antibody factor is not as important as the desensitization to the bacterial protein.

At the Massachusetts Eye & Ear Infirmary, due to the enthusiastic work of Miss Anita Mangiaracine, we have used an autogenous Staphylococcus aureus vaccine which has been prepared in a somewhat different way from the commercial strains. I believe it is rather unique. I will not quote the method of preparation in full. This will appear in the published discussion.

PREPARATION OF STAPHYLOCOCCUS VACCINE

A culture is taken from the region affected and plated routinely. If a coagulase positive hemolytic Staphylococcus is isolated, it is transplanted into the following medium.

1. Infuse 500 grams of lean beef hearts—overnight in a liter of water.
2. Boil for 20 minutes the next morning. Strain.
3. Add 20 grams of Bacto neo-peptone, 10 grams of salt; adjust the Ph to 9.2. This alkalinity precipitates out all the phosphates.

4. Boil hard for 20 minutes; cool for 3 hours or more.
5. Strain through cloth. With HCl bring ph down to 7.0.
6. Add 0.05 grams of dextrose.
7. Tube and flask, and autoclave at 15 lbs. for 20 minutes.
8. Transplant from plates the strain of Staph. aureus isolated. Incubate overnight.

From an 18-24 hour culture in the special medium above, the organism is transplanted as follows:

- a. For every 5 cc of broth, 0.1 cc of overnight culture (cf. above) is used. Usually make up to 40 cc of the vaccine. Therefore, 0.8 cc of the overnight culture to 40 cc of broth is used.
- b. Grow for three or four hours.
- c. Kill the organisms by boiling in a water bath 7 minutes. Transfer to sterile vaccine bottle.
- d. Tri-cresol (3 cc of a 2% solution/40 cc) is added to the vaccine bottle as a preservative.
- e. Check for sterility by growing a culture of 1.0 cc of the vaccine for 48 hours. If sterile - vaccine is ready to use.

The rationale of this is that in the three or four hour growth, the organism produces a capsule which is transitory and later disappears. This coincides with the production of a toxoid, which is liberated into the culture medium itself. It may be noted that the entire preparation becomes the vaccine to be used. The organisms are not strained out and washed or in any other way deprived of this toxoid material.

One of the objections to the autogenous vaccine that has been raised is that after a certain number of generations of the organism, it undergoes some biological changes, i.e. mutations from the original strain; however, in the three or four hour growth hardly enough time has elapsed for this to be an important factor. 1:100 and 1:10 dilutions are made. Beginning with a 0.2 cc IM dose of the 1:100 dilution, we gradually increase the strength and amount of the subsequent doses according to a schedule which takes about twelve weeks to reach the maximum dose of 0.5 cc. of the undiluted vaccine.

I give the doses once a week. It has been suggested that they be given on three consecutive days with a four-day interval between each series. Actually I do not think it makes much difference provided the material is given regularly and over a sufficiently long period of time. To reach the maximum dosage, I give twelve injections at weekly intervals and then repeat the final dose of 0.5 cc every two weeks for three or four doses and then once a month. On this schedule, I carry the patient around the calendar.

So far it has worked out exceedingly well in the stubborn cases of bacterial allergy which complicates chronic respiratory infection. I have very much enjoyed hearing Dr. Jones' paper.

DR. SAM H. SANDERS: Although the title of Drs. Jones' and McLain's paper is listed on the program as "The Value of Vaccine Therapy in Respiratory Infection," their discussion is limited to bacterial vaccine as stated in the second sentence. To inject a discussion of virus vaccines would be introducing a different subject entirely and would distract one's thought from the subject so ably presented.

The essayists do not suggest the use of bacterial vaccine on any and every patient but advise against their promiscuous use. The use of the vaccine is advised

in a selected group. This group is selected from those patients who continue to have chronic infections or recurrent episodes of acute infections after receiving the treatment indicated from the information obtained from quote "a careful history, thorough examination, adequate laboratory and x-ray studies, and proper therapy including allergic regime when indicated and necessary surgery." They state that these procedures are successful in the great majority of cases but when not successful, proper and adequate vaccine therapy will be effective in a large percentage of patients.

Unfortunately there is very little agreement among the otolaryngologists on what is a thorough examination: what are adequate laboratory and x-ray studies. The majority of otolaryngologists either ignore allergy or consider house dust the only allergen of any importance in perennial allergy. Dr. Jones knows that allergy is very important in the majority of these patients. He also knows what adequate surgery is but anyone not knowing the type of work that Dr. Jones and Dr. McLain do could assume bacterial vaccine therapy is being recommended for all patients who have any acute or chronic upper respiratory infection. I am sure it is not the intent of the essayists to leave this impression.

The essayists' methods of determining the proper and adequate vaccine therapy is to determine the dose and select the patients to be treated from their intradermal skin reaction to a one-half strength dilution of any upper respiratory bacterial vaccine that has proven to be effective. They are now using the following vaccines:

1. Cutter Laboratories M.V.R.I. 057-16
2. Hollister-Stier Laboratories Bacterial Vaccine
3. Lederle Laboratories Catarrhalis Combined Bacterial Vaccine 1974-37
4. Merck, Sharp and Dohme's H Influenzae Serobacterin Vaccine Mixed No. 4750
5. National Drug Co. Staphylococcus Toxoid Vaccine Vatox
6. Parke-Davis Catarrhalis Vaccine (Combined) BIO 615

The thirty-minute flare as well as the twenty-four hour erythema reaction to the .02 intradermal test is taken into consideration in determining the dose and the vaccine to be administered. The size of the future dose and interval between doses depends on the size and duration of the reaction.

Many allergists are of the opinion that very little information can be gained through bacterial or vaccine skin tests. Similar substances are contained in the six products mentioned and being tested by Drs. Jones and McLain. Simon¹ used almost the same method recommended by the essayists. The lack of patients' response to the vaccine which gave the largest skin reaction persuaded him to go one step further. Dr. Simon thought more definite information could be obtained from the use of bacterial antigen complexes (Hoffman Laboratory). The results of the tests were essentially the same.

Bacterial antigen complexes, in contrast to vaccines, are acellular and crystal clear. Ninety to ninety-five per cent of the non-essential sensitizing bulk of the vaccine has been eliminated, and up to 94 per cent of the sensitizing nitrogenous substances such as meat extracts, peptones, et cetera, present in the filtrate, have been removed.

The process used retains the essential protective substances of the bacterial cells such as polysaccharides in pneumococci, the "M" substance in hemolytic strep-

¹Simon, S. William, M.D., and Rinard, Lela A., B.S.: Bacterial Antigen Complexes (Hoffman) An Evaluation of Skin Test Specificity Versus Patient Reaction. *Annals of Allergy* 19:877-83 (Aug.) 1961.

tococci, and the bovin antigens in gram-negative bacteria. These substances are combined by the process of co-precipitation with the antigenic substances present in the filtrate. Such antigenic substances are scarlet fever toxin, hemolysins, leucocidins, fibrinolysins, dermonecrotic toxin, coagulase and hyaluronidase.

Bacterial antigen complex preparations apparently combine all antigens necessary for antibacterial and antitoxic immunity. Thus, they offer in one preparation, as, for instance, staphylococcal bacterial antigen complex, the antigenic equivalent of cellular vaccines prepared from staphylococci and the antigens in staphylococcus toxoids. The method used prevents denaturation, deterioration and subsequent loss of valuable antigenic properties which may occur in the production of vaccines by repeated washings and the use of heat or chemicals. These complexes are produced in several different bacterial combinations and standardized.

The tests were first done on individual strains of organism and later in pooled stock. A dose of .02 was given intradermally and the readings taken in twenty minutes and twenty-four hours. The patients were seen daily and carefully interrogated. Dr. Simon's conclusions were that in testing with bacterial antigen complexes of various combinations of organisms there apparently is no correlation between immediate or delayed skin tests and patients' reactions.

Practically all allergists use various vaccine preparation on the majority of their patients, probably because they hope to prevent upper respiratory infections or they hope the vaccine will help the secondary infection in the upper and lower respiratory tract. Some of these cases are considered bacterial allergy but if there is such a condition as bacterial allergy, it would be very difficult to differentiate it from a low grade infection if skin tests are eliminated.

Contrary to what would be expected from this discussion we use a great deal of the various bacterial products to stimulate antibody formation. They are used in combination with allergenic extracts. The patient has to receive an injection anyway, so we include a little "free" vaccine; admittedly a very unscientific procedure.

Vaccines used in the treatment of chronic infections, when the focus remains undisturbed, have been disappointing. Vaccines must help some of the people but a method of determining who will and will not obtain results before trial, has escaped me except in one type of case. Occasionally one sees a patient who has recurrent bacterial infection. Their resistance is low and general condition poor. If .03 of the usual stock vaccine produces a local reaction and they will not tolerate a build-up of over .07 without local or systemic reaction one can expect excellent results if the vaccine is given in small doses every three or four days for a long period of time.

Dr. Jones has had thirty-five years of experience in the administration of bacterial vaccines. He has developed a method of testing and administering vaccines that is satisfactory to him and his patients. Anyone observing the system in operation could probably see its merits. If Dr. Jones says it is good it is worth a try.

Many of us are critical of the use of bacterial vaccines because we know their use has been abused. We "hold out" for definite indications and proof of results. This is too much to expect. Vaccines have been used over a long period of time and accepted. They are of value and many of our patients recognize this even though some physicians are too stubborn to admit it.

I agree with the essayist when he limits the indication for vaccine to those patients in whom the irreversible diseased tissue has been removed, after failing to respond to local and systemic medication, and symptoms of persistent or recurrent infections persist.

DR. JAMES M. ROBB: Mr. President, Members and Guests: It is not only a great privilege but an abiding honor for me to be here at this meeting. I had this same problem that Dr. Davison was discussing, a hyperplastic sinusitis. I was operated on. I played a little football and hockey and the partition in my nose was as crooked as a dog's hind leg. If you ever have trouble explaining to a patient how crooked the septum is, just use that "as crooked as a dog's hind leg."

I had Dr. Lee Simpson in Detroit straighten the partition and I said, "Lee, you have got to do more than this."

He said, "Robbie, I can't do that to you."

Two months afterwards I went down to Memphis, Tennessee, and Dr. Likely Simpson, who is sitting right there beside me, did what I call an all-American. He did a Caldwell-Luc, an ethmoidectomy and a partial middle turbinectomy. Not every vestige of everything was taken out, I am sure of that.

As a sequel of that all of my troubles disappeared, so that an all-American is a necessary job, in which you do a submucous resection, a Caldwell-Luc and an ethmoidectomy, not removing all of the middle turbinate. You can see that I feel pretty good. I am willing to take on anybody about ten years younger at any time. I just wanted to mention that on the side.

SECRETARY RICHARDS: Excluding Dr. Lathrop, Dr. Robb.

DR. ROBB: Excluding Dr. Lathrop, yes, he was a boxer, so I just can't keep up with him. It is important in this work to do a really complete job and I was delighted to hear Davison talking.

Now, second, in response to Dr. Jones, I personally have never received vaccine therapy, but I have had a great many patients with exactly the problem that Dr. Jones has discussed. What he was presenting is a very important part of the problem. I was very glad to hear Dr. Davison present his paper because here I am as a sample.

DR. JOSEPH L. GOLDMAN: It has been a gratifying experience that you placed these two papers on our program and I wish to congratulate Dr. Davison and Dr. Jones for delivering these papers and for bringing these subjects to our attention.

We have not been directing our thoughts along these lines in recent years as much as we should. Actually immunology is becoming a very important subject in medicine. In the last several years Dr. Medower of England and Dr. Lederberg of this country were awarded Nobel Prizes for their work on the immunology, influences and genetics of bacteria.

The reason that I wish to make these remarks is to help avoid the development again of the concept of focal infection that we used to talk about years ago. I think that we should be very clear what we mean by bacterial allergy. Years ago this term was used rather loosely and the term became discredited. For instance, asthma was regarded as being a bacterial allergic disease and ultimately it was proved that this was not so.

There are certain diseases which have been shown to be the result of bacterial allergy, namely periarthritis nodosa, erythema nodosum and some cases of nephrotis. These are instances in which the focus of infection has been of distant origin and the process is the result of the products of bacteria. These, in the real sense, are examples of bacterial allergy.

In talking of hyperplastic sinusitis I think that we have to be very careful not to imply that this type of nasal disease is entirely the result of bacterial allergy. It may be that originally the tissues were sensitized by bacteria and then a train

of events developed which are unrelated to bacteria. Actually, the situation is one of hypersensitivity of the tissues and once the tissues become hypersensitized, they can react to various stimuli, be it bacterial, physical conditions, emotional conditions, constitutional conditions, or the endocrine system.

Some years ago we made a study of membranes removed from the sinuses and nose in cases of asthma. These tissues were sectioned for the purpose of looking for bacteria and we found isolated, few bacteria in the tissues on rare occasions. In other words, if these tissues do develop alterations, the stimuli must come from other sources than bacteria, once these tissues have become sensitized.

Just a few remarks about vaccine. A few years ago we tested animals with various bacteria for the purpose of creating protection. As Dr. Goodale mentioned, there are certain selective bacteria which can act as excellent antigens to improve resistance to these particular bacteria. For instance, staphylococcus aureus, as Dr. Goodale mentioned, is an excellent antigen. The pneumococcus also is an excellent antigen, but other bacteria do not act similarly, so that a vaccine should be used for a specific micro-organism if possible. Usually cultures will reveal a particular micro-organism in the chronic sinus, such as the staphylococcus or pneumococcus. That particular micro-organism should be used for a vaccine to improve the patient's resistance. I think that we have to be careful not to give the impression, once again, that we can use bacterial vaccine to prevent colds and infections of all kinds.

DR. DAVISON: Dr. Jones is quite right in saying that many people do have beneficial results from vaccines. Other people do not. There is a parallel in the immune reactions of horses used in the preparation of diphtheria antitoxin. At the antitoxin farms it is soon apparent that certain horses make lots and lots of antitoxin and other horses do not. They simply don't respond. They are healthy animals, but they do not make antitoxin; hence they are retired either to the plough or to the glue factory.

DR. JONES (closing): I thank all the discussors. I am glad Dr. Goodale cleared up that point of no protein shock reaction to the culture media of his vaccine. I think I will try it.

Dr. Sam Sanders was also scheduled to discuss my paper. This presentation has been made one day earlier because of the illness of other speakers. Dr. Sanders has been delayed by attending the funeral of a friend. He was kind enough to send me a copy of his discussion, which I think has definite value. It will appear in the *Transactions*.

Also, I thank Dr. Robb. I am glad he approved. Since he also discussed Dr. Davison's paper I will also thank him on behalf of Dr. Davison.

Now, Dr. Goldman, I am not sure we see eye to eye on this subject. You are interested in staphylococci and pneumococci as antigens. Certainly streptococci belong in that group. I think hemophilus influenzae belong there. I don't buy the idea that only gram-positive organisms have antigen-antibody value.

I am not too sure yet about bacterial allergy. I am quite sure that Dr. Schenck and Dr. Williams could discuss this at some length and I think I would enjoy hearing it. I do point out that we use the skin reaction of the individual patient as our guide and as long as we do so, we are making our treatment individual rather than "Here, this is the procedure we will give to everybody that walks in with a cold." I think we get good results. In other words, we individualize, not generalize, the treatment.

Dr. Davison's articles have really been an inspiration in preparing this paper and I appreciate his discussion.

ALLEVIATION OF APHONIA OR DYSPHONIA

THROUGH INTRACHORDAL INJECTION OF TEFLON PASTE

GODFREY E. ARNOLD, M.D.

JACKSON, Miss.

PURPOSE OF VOCAL CORD INJECTION

The chief reason for vocal disabilities in cases of laryngeal paralysis is deficient glottal closure resulting from abducted immobilization of the paralyzed cord.⁵ The same aerodynamic principles govern the deficient function of the vocal generator in all other cases of organic loss of glottal closure: following chordectomy; subsequent to vocal cord trauma; as well as in cases of congenital deficiency or acquired myopathy of the vocalis muscle (internus paresis). Many cases in each category have been successfully treated with intrachordal injection.

INJECTABLE SUBSTANCES

As discussed in a previous paper,⁷ a suitable substance should have the following properties: 1) it must be well tolerated by the tissues; 2) it must not be resorbed in time; and 3) it must be finely dispersed in a harmless vehicle in order to be injectable through the long needle of the Brünings syringe. Experience with various substances have been reported which included the following: autogenous and homogenous cartilage particles;^{1,4} heterogeneous bovine bone dust; tantalum powder suspended in glycerine;⁷ and tantalum oxide suspended in glycerine, or applied to tissues in other ways.⁷ With further experience, it developed that Teflon powder[®] mixed with glycerin appeared most suitable for our purpose because it complied with the three chief requirements mentioned above.¹⁰

INDICATION OF INTRACHORDAL INJECTION

The main purpose of intrachordal injection is to improve glottal closure for phonation. It is indicated in the following conditions.

Unilateral laryngeal paralysis with abducted position of the paralyzed cord causes marked hoarseness with air waste. Injection of the affected cord with a filling substance will compensate for its paralytic atrophy, and shift it to the midline where it can be met by the normally movable opposite cord for glottal closure during phonation. It follows that intrachordal injection is indicated mainly in the condition of complete and combined paralysis of both laryngeal nerves of one side with intermediate position of the atrophic and bowing paralyzed cord.³

Congenital dysplastic dysphonia is due to hypoplasia of the vocalis muscles in both cords with hypotonic bowing during phonation. This condition is sometimes combined with the clinical appearance of a congenital vocal cord sulcus on one or both vocal cords. Owing to the insufficient glottal closure, the voice is hoarse, weak, and characterized by great air waste with shortened phonation time. After the two cords have been filled in two separate sessions to a more normal fullness, glottal closure appears improved.

Following chordectomy for circumscribed malignancy of one cord, the excised cord is replaced by a cord-like scar band. Usually, the regrown scar band is not sufficiently broad to form good glottal closure with the other cord. After it has been filled with an injectable paste, the scar band becomes fuller, stronger, and broader so that it offers a better point of apposition to the vibrating other cord. In this manner, the aphonic condition following the life-saving chordectomy procedure may be corrected by developing a fairly loud, clear, and serviceable voice.

Vocal cord defects following excessively radical excision of vocal cord nodules or polyps may be filled up to compensate for the deficiency in glottal closure. This type of micro-phono-surgery requires considerable experience and practice. It has been successful in certain selected cases of co-operative and understanding patients.

CONTRAINDICATIONS

Intrachordal injection should not be considered before all possible attempts at vocal rehabilitation by voice therapy have been made. As

is well known, many patients are capable of overcoming their vocal disability through systematic development of intralaryngeal compensation and better exploitation of the vocal-auditory feed-back mechanism. These matters were discussed in previous papers.^{8,9}

For the same reason, injection should not be considered before six months have elapsed since the onset of laryngeal paralysis. Spontaneous recovery is possible within this time. Moreover, the state of paralytic atrophy cannot be judged before the period of potential spontaneous recovery has gone by.

Every effort should be made to ascertain that the presenting vocal disability is actually due to deficient vocal closure. If psychogenic factors are responsible for dysphonia, such as may be the case following thyroidectomy, the most successful injection could not ameliorate the patient's emotional adjustment. Such examinations of functional capacity proceed as explained in previous papers.^{2,5,6} As a general rule, marked dysphonia in case of median position of the paralyzed cord cannot be of organic or mechanical origin. Instead, these cases are usually due to emotional factors.

It should be obvious that bilateral laryngeal paralysis is not suitable for injection because narrowing of the glottis would increase the respiratory difficulty.⁶

In view of the war-time experience with paraffin injection, caution should be exercised when foreign bodies are present in the larynx. To these belong fragments of shells or bullets. Subsequent injection may break open the encapsulated foreign body and cause re-infection with perichondritis.¹

An expectant attitude is advisable in cases of congenital amusia, that is, in patients with deficient musical ability.⁹ It has been recognized that many cases of so-called "functional" dysphonia are due to the deficient feed-back mechanism of incomplete auditory monitoring. This is the chief reason why non-musical patients find it much more difficult to compensate for any type of vocal disability. Musically gifted persons are able to overcome the functional deficits in laryngeal pathology with relative facility. In contrast, those without a musical ear encounter great difficulty in coping with any disturbance of laryngeal function. For this reason, musical patients will derive considerably greater benefit from intrachordal injection than those without musical ability.

SELECTION OF SUITABLE PATIENTS

When this new procedure was proposed to the first patients, the question was often asked whether the suggested operation could possibly do any damage to the residual voice. For this reason, a test injection has been recommended. It consists of the injection of about a half cc of gelatin into the paralyzed or otherwise deficient cord. A suitable preparation of injectable gelatin is manufactured by the E. Bischoff Co. of Ivoryton, Conn., or by the Ames Co., Elkhart, Ill. under the name of My-B-Den. This test injection has the following advantages.

The patient can judge for himself what vocal improvement he may expect from the injection procedure. Moreover, he gets used to the procedure of intrachordal injection with local anesthesia and direct laryngoscopy, thus losing his apprehension of the permanent procedure and becoming more co-operative.

The surgeon may estimate how much substance he should inject at a particular point of the cord. He can also practice the details of the injection procedure, finding out what technical difficulties he may encounter with the final operation.

The postoperative course can be followed. It was interesting to observe that all patients without exception demanded that the permanent implantation be performed without delay. Having experienced a marked vocal improvement during the three days that the gelatin injection was effective, these patients were very unhappy when the predicted relapse to the original dysphonia occurred after the gelatin was absorbed.

Skeptical patients may be shown by this completely reversible and temporary test operation what they may expect from the permanent injection procedure. No further explanations need be given. Conversely, the surgeon is in a position to judge whether the patient's auditory monitoring mechanism will be adequate to derive full benefit from improved glottal closure. In other words, unsuitable candidates with emotional instability, or functional vocal derangements, may be rejected before the reversible final injection has been done.

TECHNIQUE OF OPERATION

Direct laryngoscopy with local anesthesia is the procedure of choice.¹⁰ Even though any type of laryngoscope may be suitable, we

use the Holinger anterior commissure laryngoscope with side opening (Pilling No. Br. 204) which facilitates introduction of the injection syringe. In order to have both hands free for this delicate procedure, the scope is stabilized with the Lewy laryngoscope holder. General anesthesia is not advisable because the patient should phonate intermittently during the procedure so that the improvement of glottal closure may be judged.

At present, we use the original Brünings syringe which is loaded with 2 cc of Teflon-glycerin paste. With one of the models furnished by the Storz Company of St. Louis, Mo., the Ethicon Teflon tube fits snugly into the loading tube. Another model has a smaller inner bore of only 5 mm which requires a special adapter to be screwed on the Teflon tube. The straight canula of the Brünings syringe has been modified by the Storz Company in the following manner. The total length of the detachable canula is 19 to 20 cm. Its terminal needle of gauge 18 or 19 has a length of 13 to 14 mm with a short beveled tip. A wider needle gauge punches too big a hole into the cord through which the implant may escape again.

An equal mixture of 10% cocain and 2% pantocaine solutions has been found very efficient for local anesthesia in this procedure. The Holinger laryngoscope is introduced in the usual manner and fixed with the Lewy holder against the patient's chest. Pushing the same side of the thyroid cartilage inward with the left hand which rests outside of the larynx, the cord to be injected is well fixed in optimal exposure.

The loaded Brünings syringe is introduced until the needle tip passes the glottis. It is moved gently across the cord to be injected until the needle tip pushes the homolateral ventricular fold as far laterally as is possible. Pointing the needle in an oblique lateral direction, it is advanced through the floor of Morgagni's ventricle to a depth of a few millimeters. The exact depth of penetration depends on the dimensions of the cord. The chief points of correct procedure should be practiced with cadaver larynges.

The first injection is made into the midportion of the ligamentous cord, that is, between the anterior commissure and the vocal process. A deposit placed in this area will correct the concave bowing of the cord, pushing its medial border toward the midline. Depending on the degree of bowing to be corrected, about 0.2 to 0.3 cc of Teflon

paste is deposited laterally to the mid-portion of the deficient cord until its medial margin appears straight.

In cases of laryngeal paralysis with abducted position of the paralyzed cord, a second implant should be placed laterally to the vocal process. Injecting another 1/3 cc of Teflon paste, the arytenoid is shifted medially so that the deficiency of posterior glottal closure is corrected. If necessary, a further deposit of 0.2 cc may be added between these two implants. No injection should be made into the anterior third of the vibrating cord. At this point it would act in a similarly disturbing manner as a vocal cord node or polyp. In order to avoid more severe postoperative reactions, no more than 0.5 to 0.75 cc of paste should be injected at one time.

When a chordectomy scar has to be built up, individualized judgment is necessary. As a rule, the tight scar tissue will not permit the injection of too great an amount of paste. Otherwise, it might rupture with subsequent escape of most of the implant. It may be necessary to expand and build up scarred chordal tissue in several sessions by adding a few tenths of a cc at a time.

REPORT OF CASES

In addition to the cases described in a previous paper,¹¹ the following two examples may serve to illustrate the amount of vocal improvement that can be achieved. At the time of their treatment tantalum powder was used for injection. It was thus possible to study the dispersion of the injected foreign body within the vocal cord structure by means of radiography.

CASE 1. A Russian sailor, aged 68, noticed the gradual onset of hoarseness in 1961. A few months later, he could not talk above a faint whisper. Studied carefully at the New York Eye and Ear Infirmary, all investigations proved negative. A viral neuropathy of the vagus nerve was therefore assumed.

In July 1961, all ENT findings were negative, except for complete immobilization of the left cord in intermediate position. The voice was aphonic.

On July 7, 1961, a suspension of 0.2 cc tantalum powder in glycerin was injected into the left cord. There was no pain or other discomfort during the subsequent days. A week later, the voice sounded much louder and fairly clear. In August 1961, the voice had



Fig. 1.—Tantalum powder implant (0.2 cc) for correction of paralytic dysphonia, five weeks after injection. Note fairly regular filling of cord with slight extension into ventricular fold and subglottically.

lost some of the previously improved quality. The injected cord was not near enough to the midline and appeared bowing. This explained the incomplete result. Lateral and frontal radiograms showed the position and extension of the injected tantalum powder (Figs. 1, 2).

The patient hoped to regain the vocal improvement which he had experienced for a short while. On August 14, 1961, 0.2 cc of tantalum oxide powder mixed with glycerin was injected into the left cord. There was fever, moderate pain, and slight laryngeal edema during the subsequent days. The same observation was made in a few other cases treated with tantalum oxide. One week later, the reaction had subsided. The injected cord had been shifted to the midline and closed well with the other cord. Accordingly, the voice had greatly improved: it was loud, almost clear, and of pleasant pitch. Subsequently, the patient returned to Russia and was not seen until August 7, 1962. At that time, his larynx gave the appearance of cardal paralysis in the midline, with good glottal closure. The injected cord



Fig. 2.—Frontal tomograph showing dispersion of tantalum powder within vocal cord with slight extension into ventricular fold and subglottically.

was white, smooth, straight, and without signs of irritation. The voice sounded normal for a man of age 69. The vocal qualities were as follows: Range G - c¹; speaking pitch around A; phonation time (PhT) 15 sec. When last seen on January 25, 1963, about 18 months after surgery, the larynx continued to look favorable, and the voice was loud, clear, pleasant, and without fatigue. A vocal recording confirmed this subjective impression.

CASE 2. A man, aged 55, who studied voice physiology as a hobby, concluded from his readings at a public library that he suffered from dysplastic dysphonia. Presenting his case in a modest and pleasant manner, he wondered whether his chronic hoarseness could be relieved by tantalum injection.

Examination in January 1961 showed normal ENT findings, except for typical dysplastic dysphonia due to bilateral vocal cord sulcus with a bowing glottis. Characteristically, the high tones sounded best, and the voice was better whenever the patient had a



Fig. 3.—Fairly regular shape of tantalum powder implant (0.2 cc) for correction of dysplastic dysphonia, six months after injection.

cold. Vocal range was narrow ($c^0 - c^1$), Ph T was short (5 sec), there was great hoarseness, and poor volume.

In March 1961, 0.2 cc of tantalum suspension was injected into the mid portion of the left cord. Two days later, the voice sounded greatly improved. During the subsequent months, vocal improvement continued until the following acoustic data were noted: normal range from D to e^1 ; speaking pitch around A; Ph T 20 sec. Figures 3 and 4 demonstrate the radiologic appearance six months later.

Returning frequently for further observation and technical discussion of his case, the patient appeared very pleased, but was willing to have more injections to correct his residual huskiness. In October 1961, the right cord was injected (tantalum) to make it more even and straight. As a result, the voice became clearer, louder, and Ph T increased to 25 sec. Some residual bowing of the left cord was corrected with Teflon paste in April 1962. Subsequently, the voice sounded practically normal, retaining its widened range, good volume, but having increased again in Ph T: 30 to 40 sec. When

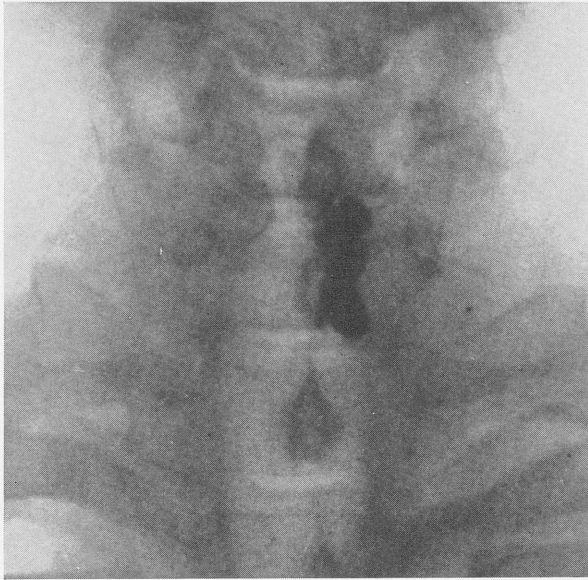


Fig. 4.—Frontal radiograph showing circumscribed extension of implant lateral and below injected cord.

last seen in January 1963, the cords moved normally, closed well, appeared straight and smooth. The voice was satisfactory in every respect.

In this ideal case of a highly motivated, unusually co-operative, and well adjusted patient, an excellent result could be achieved, thanks to the combination of many favorable circumstances. Even in purely organic conditions, the patient's psychologic attitude plays an important role.

CONCLUSIONS

It has been understood for a long time that the chief cause of hoarseness in cases of laryngeal paralysis is deficient closure of the glottis. Other types of organic glottal deficiency follow the same aerodynamic principles. In such cases, improvement of vocal function may be achieved through surgical narrowing of the excessively wide glottis by increasing the bulk of the paralyzed or otherwise deficient cord. An abducted cord may be simultaneously shifted to the midline.

In addition to the patient's subjective satisfaction with his improvement, the favorable changes of vocal ability may be analyzed according to acoustic, phonetic, and physiologic standards. Determining the various parameters of vocal quality, the surgeon or his assisting voice therapist can objectively compare vocal function before and after the operation. Tape recordings are particularly impressive because the original state of the voice is quickly forgotten.

Among the various qualities of the voice, the following improve most conspicuously after chordoplasty in the majority of cases: general speaking ability, speaking pitch, vocal intensity, clarity of the voice, phonation time, and the range of available tones. The phonetic details of vocal improvement have been discussed in a previous paper.¹¹

As is true with all types of vocal rehabilitation, therapeutic results do not depend on the mechanical and surgical problems of the operation alone. Being a psycho-social function which is deeply rooted in emotional life, the voice not only reflects many feelings and moods, but is also influenced by the patient's personality structure. For these reasons, prognosis may be less good when emotional imbalance, nervous over-irritability, or other psychologic factors retard the patient's adjustment to his new voice. It has been known for a long time that congenital amusia, i.e., lack of musical talent, is one of the chief disadvantages which may prevent the patient from taking full advantage of improved glottal function.

SUMMARY

Certain types of hoarseness or aphonia may be due to organic factors. Among these, the organic loss of glottal closure is one of the chief causes of phonic break-down. Glottal closure may be improved through the injection of well-tolerated substances into a deficient vocal cord. Teflon paste has proved to be a suitable substance for such purposes.

Indications and contraindications of intrachordal Teflon paste injection have been discussed. More than with other surgical procedures, the selection of suitable patients determines the outcome of this procedure. Likewise, the technique of the operation requires a minimum standard of experience and skill. Pertinent details have been indicated.

In addition to previously reported results, two case histories illustrate the vocal improvement that may be achieved with this

technique. Certain specific conclusions may be drawn regarding the vocal benefit obtained by patients treated in this manner. It should not be forgotten, however, that the mechanical and surgical problems of improved glottal closure are complicated by the individual patterns of the patient's psychologic personality structure.

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DISCUSSION

DR. ARTHUR J. CRACOVANER: Dr. Arnold is to be complimented on this work.

I have observed with great interest the development of the idea and the difficulties involved in finding a suitable substance for this injection. The concept is an important addition to our operative treatment of laryngeal defects. On occasion we have referred to Dr. Arnold for this type of treatment patients who have undergone the removal of a vocal cord. Although a scar frequently forms that acts as a vocal cord, there are times when the approximation of the cords is not sufficient to give an adequate voice.

This injection has improved the voice, changing it from a hoarse whisper to an audible sound. I do not recall a personal experience with other types of defects

that have been treated by this method. I can understand, however, the possibility of improving the voice in some types of paralysis, in congenital defects or traumatic defects.

The recording is new to me, but I am sure you will agree that it is very impressive.

I can remember attempting to improve the voice in postoperative laryngofissure cases by placing cartilage in the larynx in order to narrow the glottic chink so that the good cord could approximate the operated side. This was not very successful. The injection method of treatment has proven easier and has given far better results.

It is easy to understand the use of gelatin as a trial procedure when the method was in the experimental stage. Dr. Arnold did not give his complete paper, but in his paper he mentions the difficulties he had with the various injectable materials. He evolved the steps through which he went. One of them was the injection of gelatin as a trial procedure before he injected tantalum or teflon. I wonder if this is necessary now. It means an additional hospital admission and it is doubtful if one obtains enough useful information to warrant the time and the expense.

An interesting observation is the assertion by Dr. Arnold that it is unwise to inject the anterior one-third of the vocal cord, since it would act as a growth on the cord. Apparently one must be careful and selective in choosing the site of the injection.

The cases described in Dr. Arnold's paper were patients who had received injections of tantalum. I question if he has used teflon long enough to determine whether or not it is going to give permanently good results. Teflon paste has only recently been used and therefore its worth is still to be evaluated.

Again let me say that this appears to be a very useful procedure.

DR. ROBERT B. LEWY: The method that Dr. Arnold describes works effectively and efficiently. He deserves great credit as a pioneer and innovator of the procedure. The degree of voice rehabilitation and improved glottic control is truly remarkable.

One word of warning must be sounded: the frictional components involved in using a paste in a long needle are considerable. For safe and accurate work instruments must be used that surmount this problem. The instruments which he demonstrated do surmount this problem.

I wish to show you a series of slides and also to present a tape recording.

The slides:

1. Picture of paralyzed vocal cord;
2. Picture of paralyzed vocal cord after teflon injection;
3. Instruments;
4. Spectrograph of voice before and after injection;
5. Tape record of voice before and after injection.

DR. DEGRAAF WOODMAN: Mr. Chairman and Fellows: I have been very much interested in Dr. Arnold's work—especially his technique to control the Aphonia, also the work of Dr. Willard Goff of Seattle, who uses a paste of ground bone.

Certainly the presentation here is beautiful. He has shown a technique as to where and how to instill this space filling material, and he has shown examples of excellent functional results.

There is one thing I am worried about. That is the reaction obtained from the long-term use of this type of foreign body. I am just old enough to be able to

recall the reactions from the use of paraffin, in which resulting paraffinomas did not develop for periods of even ten years after the original instillation.

May I suggest that he try the patient's own centrifuged blood, taking the precipitate and using just enough serum to act as a vehicle for injection. Now one might immediately say: "Well, this is blood and it will quickly be absorbed." I do not believe this is necessarily so as I recall two patients who were operated upon for Arytenoidectomies when we were first doing them at the Medical Center, and the surgeon had accomplished his work in such a dry field that he did not feel the need of leaving in a drain. In fact his first two cases were closed without drains and both of them developed hematomas so that the lateral wall of the glottis was pushed medially to such an extent that neither of these cases could be decannulated after many weeks of observation. Hence I feel that a hematoma may last a long while.

At least in using blood he would be using something compatible with the patients' own tissues.

One might say that Tantalum has been used in various parts of the body without ill results, but the use of a plate or prosthesis is a little different from diffusing a suspension of metal into soft tissue spaces. I feel we should be cautious and think about the use of such a foreign body in this area.

I hope that Dr. Arnold will, if he has a sufficient series of cases, at least give a trial to this suggestion.

DR. LYMAN GILDER RICHARDS: While he is coming up, could I just ask whether he feels the prospects of success in this are better after a paralysis of nonoperative cause or in a case such as, for instance, laryngofissure, where the cord inevitably is replaced with some degree of scar tissue?

Are your results liable to be better in the nonoperative case?

DR. ARNOLD (closing): I wish to thank the benevolent discussers for their kind remarks.

Dr. Cracovaner has mentioned that in the beginning of these studies I did a test operation by injecting resolvable gelatin. I do that nowadays but rarely because I do not have to practice a procedure any more. However, some patients are unduly apprehensive about the procedure. In such cases one may do the test operation to show them what and how much they may expect. A test operation may be advisable when psychological factors are suspected, or when a patient is highly non-musical in contrast to Dr. Lewy's patient who could sing so well. We have found that people who are not able, or have never been able to sing are less capable of compensating for a vocal disability than are those with an interest in singing.

Dr. Cracovaner also wondered how long teflon has been in use and the answer is for more than two years. The cases treated with tantalum injection, as I have shown, were observed before that time.

Dr. Lewy's presentation was most impressive. I was amazed at how well his patient could talk because you must remember that this man had a pneumonectomy, retaining only one lung. Consequently his breath was remarkably good.

Dr. Woodman has given me a valuable suggestion to which I am going to devote earnest thought. At first I thought, of course, that blood would be quickly resolved, but it may be that in this area it may have the desired effect.

Dr. Richards is perfectly correct in assuming that surgical cases, such as following chordectomy, are not as easily corrected as are cases of laryngeal paralysis. A paralyzed cord retains its external form, whereas scar tissue following a trauma or chordectomy is necessarily inferior to an otherwise healthy cord.

I thank you very much for the opportunity to present this paper.

LARYNGIC DESCRIPTIVE DISCREPANCY

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CHICAGO, ILL.

The American Laryngological Association was founded in 1879, inaugurating the study of the larynx as a specialty. During the 84 years that have elapsed almost every meeting has contributed something to the study of the vast problem of cancer of the larynx. Over this span we have the same awareness possessed by our laryngic forebears, viz., that successful management of laryngeal carcinoma is subject to correct diagnosis, which in turn is dependent upon a more exact assessment relative to site, extent, degree of invasiveness, and other morphological factors. The current laryngic diagnostic inventory makes available indirect and direct laryngoscopy, laminography and cineradiofluorography, as well as histopathologic studies. Each modality has within its range the establishment of a tentative or a positive diagnosis but more often than not, several approaches will be necessary in arriving at a complete evaluation. Moreover, the operative experience, together with a thorough macro- and microanalysis of the surgical specimen eventually gives us the clues which will permit a more exact assessment of existing therapeutic modalities.

With the increasing emphasis on classification and staging of carcinoma of the larynx in relationship to treatment, it is obvious that the factors and the circumstances influencing the morbidity and mortality are worthy of periodic inquiry. It may be an oft told story but it bears repeating. A review of the problems involved may advantageously be conducted in the light of the historical utterances of our predecessors, the current literature as well as selected examples from personal experience. This is especially true if we employ the latter to illustrate the adage, "Experience is the name everyone gives to his mistakes" (Wilde). It is rightly said that "it demands almost

superhuman courage to select material from one's own humiliating experiences, and with the best intentions it is only human nature to develop amblyopia and deafness in regard to these deficiencies. There is a more comfortable method of enumerating the mistakes of others since it carries with it an unworthy sense of triumphant condemnation of the other fellow as well as the implication of personal infallibility" (Adolphe Abrahams).

INDIRECT LARYNGOSCOPY

Historical Perspective. Over a century and a half ago, in 1807, Philip von Bozzini of Frankfurt-am-Main described a double cannula with a mirror placed at an angle at the end. It was supposed to transmit light through one compartment, reflect it from the mirror onto the parts examined, and the image, received on the mirror, was then reflected back to the eye through the other compartment. It was thought that the light passing in would interfere with the perception of the reflected image if one tube was used. A wax candle with a reflector behind it supplied the illumination. This instrument was reported to have been used successfully to view the larynx.

In 1829, Benjamin Guy Babington presented before the Hunterian Society "an oblong piece of glass, set in silver wire, with a long shank. The reflecting portion was to be held against the palate, whilst the tongue was held down by a spatula." He called it a glottoscope. The names and efforts of Cagniard de la Tour (1825), Senn of Geneva (1827), Bernati (1832), Robert Liston of London (1837), Beaumès of Lyons (1838), Liston of London (1840), Selligie (a mechanic), Adam Warden, Ephraim Cutter, and Avery (1844), all antedated the singing teacher, Signor Manuel Garcia of London (1855) in their attempts to view the larynx. Ottokar Chiari, in 1902, in his book, "Krankheiten der Oberen Luftwege," as well as J. Gottstein in his book, "Die Krankheiten des Kehlkopfes," in 1890, credited Ludwig Türck, of Vienna, who in 1860 wrote "Praktische Amleitung zur Laryngoskopie," as the real discoverer of laryngoscopy, "even if there were more or less attempts by others to undertake examinations in isolated instances." Then there was Johann Nepomuk Czermak of Budapest (1858), whom Chiari credited with being the first to utilize the Türck mirrors illuminated by reflected light. Ernest Krackowitzer in 1858 and later in 1861, Louis Elsberg (the first President of the American Laryngological Association), Strangenwald, Church, John H. Douglas and Horace Green were credited with introducing

indirect laryngoscopy to America. It was about this time that Turck and Semeleder began to teach laryngology in Vienna. "The Use of the Laryngoscope" was published by Morell Mackenzie in 1868 with a "Discussion of the Art of Laryngoscopy."

Over fifty years later, the state of laryngoscopy can best be summed up by a quote from MacKenty who in 1922 said, "Judging from the advanced condition of the great majority when they come for operation, the diagnosis of cancer of the larynx must either be extremely difficult or poorly understood by the profession." Of interest in this connection is the description of a patient whom he observed in that year and continued to see, along with Chevalier Jackson, for the next five years. This patient began with a small cordal lesion and eventually had a large fungating mass that even the uninitiated might have diagnosed. MacKenty further commented, "After all these years of watching, circumventing every possible avenue of error, we now had, in spite of all our efforts, an advanced intrinsic cancer to deal with. If I had not the unerring skill and judgment of Dr. Jackson covering every move I might have blamed myself for overlooking the malignant condition in June." In fact the patient had an extensive carcinoma involving the entire left side of the larynx, including the aryepiglottic fold. The postsurgical period was catastrophic and in one year the patient succumbed to neck metastases.

Our predecessors in the specialty wrote tomes on the art of indirect laryngoscopy. They were much more dependent on this method, if not skilled, than were the generations of clinicians to follow. The method not only served a diagnostic purpose but also figured importantly in the treatment of infiltrative and ulcerative states in tuberculous laryngitis, as well as for the indirect removal of tissue for histologic study of neoplastic disease. For many years now, I have been impressed with the lack of descriptive uniformity of the laryngeal image as visualized by any given number of our present day observers at the same sitting. Aside from the diagnostic value of such observations, the lack of dependability relative to the description of site, size, color, degree of invasiveness, and the like, which are helpful in setting indications for treatment, is indeed appalling. Granted that there are instances where almost insurmountable difficulties are encountered in exposing the larynx to full view, sedation and topical analgesia, plus an infinite amount of patience and cajolery, may finally be rewarded. General anesthesia is frequently required. The

anatomy and the mobility are observed with the objective of assessing the lesion relative to its diffuse or localized character. The question arises whether or not the cord is of normal texture and exhibits the same looseness of the opposite or normal cord. If the process appears diffuse, several sites for biopsy may have to be chosen. The interpretation of mobility frequently rests with the particular examiners (and this, incidentally, may be on any given day). The question should be asked, "Does the cord appear to move en masse?" The same exacting technique of examination must characterize all clinicians if they are to speak a language common to every laryngologist.

It is frequently the case that one hears that the indirect laryngoscopic view differs greatly from that obtained on direct laryngoscopy. Aside from the problems previously alluded to, the latter method can, if correctly applied, add much to the information gained by the indirect method. The shortcomings of both methods must be appreciated and taken into account in each laryngoscopic evaluation.

DIRECT LARYNGOSCOPY

Historical Perspective. The birth of direct laryngoscopy is best depicted in an interesting preface of a book written in 1896 by Alfred Kirstein on "direct examination without a mirror" in describing "Autoscopy of the Larynx and Trachea":

"Ever since the time when progressive minds first endeavored to get a view of the interior of the human larynx they have been under the impression that the way from the mouth to the vocal cord, according to existing anatomical conditions, must of necessity form an angle. Under the influence of this conception, the truth of which no one ever seemed to doubt, no plan could have been conceived other than the one which finally, in the sixth decade of our century, resulted in the discovery of laryngoscopy by Garcia, Türck, and Czermak, namely, to overcome this difficulty by means of a reflecting apparatus (mirror) placed at the apex of the angle, or the introduction, into medicine, of the laryngoscope, the laryngological technique entered upon a path of development from which it has hitherto not departed in the slightest degree. The introduction of endolaryngeal surgery with the laryngoscope in 1862 by Victor von Bruns was the next brilliant development of Türck's and Czermak's great invention. Cocaine [Carl Koller did not employ it until 1884 in ophthalmic surgery] has freed laryngological surgery from its many difficul-

ties without at all changing its method. Thus, there has been but one creative thought which has hitherto directed all our laryngological methods of examination and treatment.

"It may be a rash undertaking for me to deviate from time-honored tradition, to open up an entirely new way of examination and treatment of the larynx and trachea, by teaching that we can view the interior of the air-passages directly, without the aid of optical appliances and operate with straight (uncurved) instruments in the same manner. Nevertheless, my undertaking is doubly justified. In the first place, because of the facts themselves, which may be demonstrated at any time, and which I have made known in a series of publications (in 1895) entitled 'Autoscopy of the Air Passages.' The fact that we have, up to this time, never viewed the interior of the larynx directly (without a mirror) is certainly not due to logical reasoning, but because we had no idea of such a possibility. Since, however, we have discovered that it can be done, and how we can do it, there can be no objection raised to our making full use of our new knowledge and ability. In the second place, this method is not intended to replace the laryngoscopic method, but to add to it. I must decidedly deny any prejudice on my part toward the laryngoscopic method; the very fact that I myself have shown that my direct method cannot render the indirect method superfluous throws into very strong light the value of the laryngeal mirror. Of course, many a laryngologist is convinced that the laryngological technique needs no additions; others may think differently. Only the future can decide this question."

In this book, Kirstein concluded that "Whoever believes in uninterrupted progress, in small things as well as large, may nurture the hope that in the future autoscopy will be developed to such an extent that it can ultimately be applied to every individual. This hope, however, I cannot share. The problems of autoscopy are confined within exceedingly narrow limits, their determining factors are well known, and I have expounded them sufficiently in this monograph. The limitations of autoscopy are of an anatomical nature, and the structure of our bodies will scarcely accommodate itself to our wishes. In other respects I now consider the problems of autoscopy definitely settled, until, at least, an entirely new creative thought arises to open up a new path."

The so-called "blind spots" of the larynx, especially those that originate or extend into the pre-epiglottic space, base of the epiglottis

or subglottic region, ventricles or thyroid cartilage, cannot always be completely assessed by indirect or direct laryngoscopy. As Chevalier Jackson stated, "Mirror examination is, of course, extremely important and always necessary, but, alone, the laryngeal mirror appearances are not conclusive. No mirror examination is complete unless the anterior commissure is clearly exposed to view. Death often lurks under an overhanging epiglottis. Moreover, it is not merely the anterior commissure but also the suprajacent, nearly vertical wall of the posterior surface of the epiglottis that must be seen. The laryngeal image, in health or disease, is made up of color, form and movement."

The literature of cancer of the larynx contains many descriptions of mirror appearances that were considered by their authors as important diagnostically if not absolutely characteristic. Each sign in turn has been discredited and discarded by subsequent authors.

Henry T. Butlin, who wrote on "Malignant Disease of the Larynx" in 1883, stated "There are three classes of cases: first, in which anyone and everyone can make the diagnosis; second, in which the better instructed or more experienced can make it; and the others do not; and the third class, in which the diagnosis is so obscure that no one can make the diagnosis unless the larynx is opened, and in some cases it is even then difficult to be sure of the nature of the disease." In 1908, Chevalier Jackson, in the proceedings of the Section of Laryngology of the American Medical Association, disputed the statement of Butlin, saying that to obviate any necessity for the exploratory laryngofissure he had been carrying out the following plan of procedure: "1. Consideration of the symptoms. 2. Indirect laryngoscopic study. 3. Direct laryngoscopic and bronchoscopic subglottic study. 4. Histologic study of an adequate specimen removed by direct laryngoscopic study. . . . The direct laryngeal speculum permits the use of forceps in any case with an accuracy impossible even in favorable cases by the indirect method." In view of the frequency of overlooked early cancer of the larynx Jackson advised that all cases of laryngeal disease in adults be regarded as malignant until proven otherwise. This was not given as a basis for surgery but as a diagnostic routine. In 1930, Sir St. Clair Thomson, referring to Butlin's statement, felt "This opinion remains true, although with added technical aids in diagnosis, there are few cases nowadays in the third group."

DESCRIPTION—A KEY TO MANAGEMENT

It is the rare laryngologist who can resist the temptation of classifying carcinoma of the larynx for the purpose of determining therapeutic indications. It is not entirely within the purview of this communication to make this objective a vital part of the presentation other than reiteration of the important role played by an exact laryngeal identification and evaluation as the *sine qua non* of success in treatment.

Presentation herewith of a practical grouping for the purpose of deciding treatment based upon site and degree of involvement is in no way intended to negate the efforts of numerous committees to reach an accord on the clinical classification and staging of cancer of the larynx and hypopharynx. It is fair to point out, however, that the results of their efforts have neither universally caught the fancy of clinicians nor been entirely acceptable to others as a basis for evaluating the various treatment modalities. Appreciation is due the efforts of the International Committee for the Study of Cancer of the Larynx (Rio de Janeiro, 1958); the International Union Against Cancer (Paris, 1958); a meeting of both of these groups (Paris, July 1959), and the American Joint Committee on Cancer Staging and End Results Reporting (sponsored by the American College of Surgeons, American College of Radiology, College of American Pathologists, American College of Physicians, American Cancer Society, and the National Cancer Institute), which was organized January 9, 1959. All of these groups, for the sake of a universal language, agreed on defining the extent of disease in terms of three components, the TNM system, i.e., 1) the primary tumor designated by the letter T, 2) the regional lymph nodes designated by the letter N, and 3) distant metastases designated by the letter M. Descriptive terms were also agreed upon relative to what constituted the anatomical limits, the regions, the lymph node metastases and clinical stages of cancer of the larynx. Our approach does not differ materially.

DEFINITIVE MANAGEMENT

<i>Site and Degree of Involvement</i>	<i>Treatment</i>
<i>Group I</i>	
Middle third	Irradiation or
Movable cord	Thyrotomy

Group II

Anterior third and commissure	Irradiation
Movable cord	Anterior commissurotomy
	Laryngectomy

Group III

Posterior third	Laryngectomy
Movable cord	(and neck)

Group IV

Groups I, II or III with	Laryngectomy
Fixation of cord or	and neck
Extension	

Group V

False cord	
Ventricle	Laryngectomy
Arytenoid	and neck
Aryepiglottic fold	
with or without nodes	

Group VI

Epiglottis	Laryngectomy and neck on side of
Vallecula	palpable nodes and later,
with or without nodes	opposite neck

Group VII

Hypopharynx	Laryngopharyngectomy and neck
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Group VIII

Fixed nodes	Palliative
Distant metastases	and supportive

There are two key questions which have been somewhat debatable: Does the lack of cord motility negate the indication for irradiation and is this modality contraindicated or does it hold out a possibility for encouraging carcinoma elsewhere when employed in the treatment of young adults? Many radiologists contend that neither premise is acceptable. It is well to point out, at this time, that carcinoma frequently is of multicentric origin and that a lesion on one cord may arise on the opposite cord by this means or by implantation. Diagnostic and therapeutic problems arise as a result.

NECK PALPATION

Speaking of meaningful descriptive discrepancies, one has but to refer to the variations of opinion occasioned by a group of clinicians who palpate a neck. Naturally, the patient who has obvious masses leaves no doubt for debate in this regard. It is amazing, however, to find clinicians so frequently at variance. It is estimated that palpation alone may account for an error of approximately 20%. This constitutes a clinical error in estimating the presence of metastases. Obviously, such a physical finding, when present, is of great importance. Together with the correct interpretation of the laryngeal image, if there is to be a proper selection of modalities, the suspected or known presence of positive nodes is important, inasmuch as we well realize that it is the primary adequacy of treatment that spells success or failure in cancer management. This frequently means that the primary lesion and node bearing area require removal in continuity. Observers have pointed out that the incidence of lymph node involvement runs alarmingly high (upwards of 50 per cent), irrespective of the size or duration of the primary lesion. This is especially true if the site of the lesion is not confined to the true cord. Even then, in our experience, the posterior one-third of the true cord has a marked metastasizing quality.

The delaying action of associated conditions or other diagnoses requires little elucidation. The presence of even a suggestive positive serological test or some indefinite bronchial or pulmonary roentgenographic finding may be blamed for the laryngeal picture. Many of the recently trained laryngologists have encountered so few instances of specific or chronic granulomatous disease of the larynx that they fail to recognize them when they do come to their attention. As rare as its occurrence has been in the past or will be in the future, the triad (syphilis, tuberculosis and carcinoma) is still capable of manifesting itself in the same patient. Even the most experienced laryngologist can ill afford to dismiss all else from his thoughts, other than cancer.

DIAGNOSTIC RADIOGRAPHY

Although the earliest roentgenologic diagnostic studies of the neck have been attributed to Scheier in 1896, it was not until 1922 that Coutard developed the technique of examination of the hypopharynx and larynx by means of lateral soft tissue roentgenograms of the neck. This method has remained the fundamental technique for

the evaluation of lesions arising in the larynx, and its value in the diagnosis and treatment of head and neck tumors has been ably described by Baclesse, Coutard, Pendergrass, Young, Fletcher and others.

Before 1940 almost all the information available concerning roentgen examination of the soft tissues of the neck was based on observations made with the patient in the lateral position. Early in the evaluation of laryngeal neoplasms, small occlusal films were placed in the hypopharynx (Rethi and Waldapfel). This technique and other methods of frontal roentgenography make use of low kilovoltage with short target skin distances and are restricted in scope and lack detail. However, improvement in film contrast and the advent of the fine focus shock-proof roentgen tube made it possible to obtain more adequate ventral projections of the larynx. Tsuiki who followed a technique initiated by Schoen (1953) and Reinicke (1954), utilized high voltage roentgenography. Fluoroscopic and spot-film examination of the larynx is considered by Schreiber (1962) to be a simple, efficient, and diagnostically useful method. Four exposures are made on two 20 x 25 cm films, postero-anterior and lateral, during quiet respiration and while phonating a high-pitched "E". This method has the advantage of providing views of the larynx in two projections and in two stages of physiologic activity on two films obtainable in a few minutes time.

It is possible to localize laryngeal neoplasms, which develop along frontal planes, such as tumors of the false cord and lateral subglottic region. Nevertheless, there is the continuing problem of the superimposed spine in obtaining routine frontal roentgenograms of the neck. Furthermore, laryngeal tumors usually appear as homogeneous masses and there is a need to enable differentiation of the tumor from its surrounding structures, which planigraphic studies of the same region allow one to do. The planigraph or tomograph has been used in clinical medicine since 1931, but it was Felix LeBorgne, who in 1936 published the first anteroposterior planigraphic study of the larynx. This method was particularly valuable in disclosing subglottic neoplasms and delineating an airway narrowed by tumor.

Radiopaque visualization of the tracheobronchial tree was first introduced by Jackson, who in 1918 reported the use of bismuth subcarbonate through a bronchoscope for the localization of foreign bodies. Samuel Iglauer of Cincinnati later injected iodized oil to

examine the larynx, trachea, and bronchial tree, but this method was not further developed until 1936, when Jackson utilized lipiodal for the contrast examination of the larynx and pharynx. With the synthesis of more satisfactory radiopaque media for uniform mucosal coating, a number of methods of contrast laryngography were reported by various authors. In 1957, Powers, McGee and Seeman described a simple and reliable contrast technique for the roentgen examination of the larynx and pharynx utilizing oily Dionosil. Through a curved cannula, 5 to 15 cc of this solution is instilled into the laryngopharynx after adequate topical anesthesia. Spot roentgenograms are obtained in the frontal and lateral planes employing high KV, rapid exposure techniques. Film pairs, exposed during quiet nasal respiration, phonation, Valsalva, and modified Valsalva, comprise the study.

During a three year period Powers and his associates employed contrast laryngography in the diagnosis of 99 malignant neoplasms, precisely determining the site of origin of the tumor, the area of involvement, the size of the tumor, the status of surrounding structures, and mobility of the cords in 92% of the cases. In 1960 Ogura and his group (which included Powers) reported on the evaluation of a series of laryngograms which provided very accurate information on the exact site and extent of laryngeal tumors. In a series of 96 patients the accuracy of pre-operative evaluation by x-ray studies and laryngograms was 92 per cent, whereas by clinical study alone, they were only accurate in 78 per cent of instances.

Fabrikant, Richards, Tucker, and Dickson have recently (1962) added television image intensification fluoroscopy and cineradiofluorography to contrast laryngography in the evaluation of laryngeal neoplasms, which they feel enhance the value of the method, despite some loss of detail in these examinations.

TISSUE BIOPSY

There are laryngologists who stress the fact that biopsies should only be obtained by those capable of following through with definitive care. Distortion of the original indirect or direct image of the larynx presents sufficient variations so as to modify a clinician's thinking and decisions relative to correct management. He is confronted with a difficult decision; to treat a patient who has no residual disease

or to observe one who has been treated inadequately. Other influencing factors of importance are:

1. Inadequate tissue, both in size and depth
2. Improper site (edema or ulceration)
3. Faulty tissue technique
 - a. Instrumentation
 - b. Exposure
 - c. Labeling
 - d. Imbedding
4. Morphological description
 - a. Biologic error
 - b. Technical error

Aside from the technical difficulties that may be encountered in the proper exposure of the larynx, especially in the so-called unco-operative patient, small lesions are frequently "nibbled" off rather cautiously in order to preserve the integrity of the cordal edge. A deeper bite, or one that would include normal tissue with that which is altered, would compromise the voice and, if the lesion is benign, could have manifold repercussions. It is clear, however, that "pinch" biopsies often lead to noncommittal biopsy reports. Such biopsies may not always reveal tumor cells, and, therefore, only positive findings can be considered dependable. Negative findings do not necessarily "rule out" the possibility of cancer. Cytologic studies have shown some correlation, but usually in situations where diagnosis could be established by other means. Unfortunately, there are no absolute criteria for making a positive diagnosis of malignancy from a single cell observed in a smear. Other factors concerning technique require little or no elucidation. However, in line with the diagnostic discrepancies which occur, the handling of tissues from the moment specimens are removed from the biting forceps, to the time the slide is read by the pathologist, becomes a matter of prime importance.

Diagnostic delays are occasioned by a number of the previously mentioned factors including:

1. Insufficient tissue
2. Further removal of tissue for biopsy because of inconclusive findings
3. Lack of recognition of extremely suggestive histologic signs of malignant alteration

4. Complacency due to a lack of co-ordination between the clinical picture and the interpretation of a suspicious biopsy.
5. Other diagnoses, including syphilis or tuberculosis
6. Poor response to local analgesia

The pathologist's task would be aided by a closer working relationship with the laryngologist, each sharing their specialized views and knowledge. The former's diagnosis must always be taken into account, but in view of occasional misinterpretation, there is room for mutual discussion. Such occasions arise when a tangential section of epithelium appears in the microscopic field and this is mistaken for malignancy by the inexperienced eye. Too often a suggestion of malignancy is not stressed sufficiently to the clinician. Paul Szanto of Chicago, a pathologist of considerable experience both generally and in the field of otolaryngology, points out that two forms of error may occur. The first is a biological one, wherein even the experienced pathologist may be challenged by "something that looks like carcinoma." He would insist on serial or subserial sections into the depth of the block of all tissue that has been removed for biopsy. Technical error characterizes the inexperienced pathologist, especially when it comes to interpretation of tissue removed from the larynx. Nonetheless, under either circumstance the pathologist must be aware of the clinician's dependence upon his histologic interpretation and the implication occasioned by further delay in arriving at the recognition of the presence of carcinoma.

It is of historical interest to note that the Jacksons laid down the "ground rules" that, if followed, would alleviate many of the problems that are encountered. Essentials which they believed should prevail and many which have previously been alluded to are:

1. There must be an ample amount of tissue.
2. It must be taken with precision.
3. If the growth is small all of it is removed and submitted.
4. If the growth is large ample specimens are taken from different parts including an edge of grossly normal tissue.
5. The entire specimen should be sectioned and the sections should be examined in the order in which they have been cut from the specimen.
6. If the first specimen is inconclusive the biopsy is repeated.
7. The histopathologist is a consultant in the case; he is given all

the important clinical and laboratory data; he is asked to look at the larynx with the mirror and through the direct laryngoscope; he sees more from the site from which the specimen is taken; if he wishes more tissue it is taken for him, amply and with precision. When a laryngofissure or laryngectomy is done the entire specimen is taken for study. He is always given every opportunity to follow the subsequent course of the disease in every patient. This kind of teamwork becomes of utmost value when unusually difficult diagnostic problems are presented. Moreover, the cumulative experience in the histopathology of laryngeal disease gives an approach to infallibility otherwise unobtainable.

It is obvious that the Jacksons went to considerable length to expound the *seventh* essential. The problems that apparently plagued them, their predecessors, and which still constitute one of our major descriptive discrepancies today, was to be solved by the cementing relationship of those espousing the same philosophy of good patient care.

Relative to the patient who brings along a positive biopsy from another laryngologist, it has been our practice to follow the recommendation Holinger made in discussing Maloney's paper: "We would be falling into a very serious pitfall if we simply took his slide, good as it may be and definite as the lesion may appear carcinomatous from the gross appearance, and went ahead with laryngectomy. Dr. Edwin Hirsch, pathologist with whom we have worked for many years, convinced me some time ago that it would be unwise to take a slide sent with a patient and look at it and say 'This is cancer!' with the idea that we go ahead with major surgery on that basis. His statement was 'How do you know that this is the slide of this patient? How do you know the sequence of events from the biopsy to the time it comes into your hands and mine is one that is not disturbed? How do you know that there is no possibility of error?'"

Maloney suggested what Butlin proposed in 1883 and what we have practiced for many years, when he advocated "Exploratory thyrotomy to establish (positive histological) diagnosis where all else has failed." We should recognize the definite indication of this procedure for diagnosis as well as management. Maurice F. Snitman suggested the "La-La" procedure, by which he implied "laryngeal laparotomy," to explore the extent of the tumor so as to more accurately determine the surgical procedure to be employed.

Relative to histological interpretation, no problem results when distinct metaplasia is present. Polemics, with subsequent delays in establishing a conclusive diagnosis, are occasioned by cellular interpretation that frequently does not coincide either with the indirect or direct laryngologic assessment of the lesion. Among these delaying descriptive terms which the laboratory report contains are:

1. Metaplasia
2. Pachydermia (this is more of a clinical than pathological terminology)
3. Leukoplakia
 - (a) with hyperkeratosis
 - (b) with basal cell proliferation
4. Carcinoma in situ (non-invasive carcinoma)
5. Papilloma

Under the above circumstances further and deeper removal of tissue becomes paramount. It is well, however, to make special reference to some of these terms as to what they mean to us. In an interesting discussion by McGavran, Bauer, and Ogura, epithelial hyperplasia was defined as a thickening with or without acanthosis; parakeratosis, as the surface accumulation of incompletely keratinized cells retaining their nuclei; keratosis and hyperkeratosis, as the formation of a granular layer with surface accumulation of completely keratinized cells, and atypical epithelial hyperplasia, as abnormal maturation of the epithelial cells with nuclear aberration, but of insufficient degree to be classified as carcinoma in situ. All of these, we believe to be varying stages of leukoplakia. Metaplasia implies the alteration of the normal respiratory type of epithelium into a squamous variety.

Keratosis is not a true neoplasm. Laryngoscopically, it appears as localized, chalky white elevations. Microscopically, there are varying degrees of keratin formation, a hyperplasia of prickly cells and a proliferation of basal cells to form projections into the underlying tunica propria. The basement membrane is usually distinct and the arrangement of cells quite orderly. At times, however, the cells tend to become irregular so that it may be difficult to be certain whether the lesion is benign or malignant, whereas the transformation to a stratified squamous type of carcinoma is unequivocal. It is unfortunate that some pathologists employ the terms keratosis, hyperkeratosis, pachydermia and leukoplakia interchangeably as though they implied the identical alteration. Frequently, on any given day,

any number of pathologists will disagree on interpretation and designation of these epithelial changes.

Leukoplakia presents two distinct varieties to which the laryngologist gives different clinical meanings. In the first form of leukoplakia there is a thickening of stratified squamous epithelium with keratin formation (leukoplakia with hyperkeratosis). To a clinician, all things being equal, this condition might respond to adjusting or changing the patients' habits, including an unfavorable working environment, voice abuse and smoking. The second form, however, is to be viewed with a degree of alarm, because it presents histologic changes seen in the first variety plus basal cell proliferation. Insofar as the larynx is concerned carcinoma in situ could readily be considered GRADE I carcinoma with evidence of carcinoma lurking not many millimeters away. We would warn the pathologist of using this designation as a "safety-valve" or an "escape" equivocal diagnosis.

Relative to papilloma, its presence in an adult is to be viewed with suspicion and calls for further inquiry and close observation. Papillomas reveal a core of well vascularized loose connective tissue covered with hyperplastic and branched squamous epithelium. The surface of the latter contains a variable amount of keratin, the prickle cell layer is thick and composed of well differentiated polygonal cells, and the basal cell layer, although regular and even, frequently reveals hyperchromatism of the nuclei, often sending peg-like projections into the underlying connective tissue. The basement membrane is intact.

In our experience, where atypical cells are reported by the pathologist, it is noteworthy that in almost all instances, further study has revealed the presence of malignancy. Inasmuch as it is unacceptable and even unthinkable for anyone to employ cancerocidal doses of irradiation or advocate surgery without positive histologic evidence of malignancy, the attitude of watchful expectancy has sounded the death knell of many patients.

A case in point was the classical MacKenty-Jackson patient previously mentioned, where five years of observation passed under their very well trained eyes, when the situation became so obvious, but beyond the control of all measures that were instituted. In the mind's eye of all laryngologists similar cases pass in kaleidoscopic review. It becomes the specialist to humbly acknowledge his shortcomings, especially in the light of the fact that courts hold him to a higher

degree of learning, skill and judgment than the generalist. This to my mind, in view of the numerous difficulties of establishing an exact diagnosis, is much too restrictive and punitive, but convince the plaintiff, his attorney, and a lay jury of this and you have an almost insurmountable hazard to overcome, which our laryngologic forebears had not need to concern themselves about.

Questions always arise as to whether or not the lesion was benign to begin with and later underwent malignant proliferation, or that mitotic cellular changes were present at the very outset but not in the particular material obtained on biopsy. Furthermore, the responsibility does not rest solely upon the laryngologist but on the pathologist who reads the microscopic slide. Nevertheless, there is never anything final about this reading, for continuous observation and eternal vigilance become the order of the day, so as to observe the slightest change in the cordal picture. Even then difficulties arise as are exemplified in the experience of all clinicians.

SUMMARY

In spite of technological advances over a span of 106 years since Ludwig Turck began to popularize indirect laryngoscopy and 68 years since Alfred Kirstein introduced the direct method, diagnosis of carcinoma presents a continuing challenge. We have not over the years greatly improved our visualization and verbal description of laryngeal lesions. Frequently, it seems extreme preciseness of visualization and description may not call for alterations in the ultimate management of the patient. There are instances, however, where it is all important. Nor is this appraisal of morphologic diagnosis limited to the laryngologist; similar problems of evaluation and even definition confront the pathologist in his description and diagnosis.

The frequent, if not constant, critical appraisal of diagnostic and therapeutic axioms passed down from former generations of laryngologists is essential to ensure a resolution of the problem of cancer of the larynx. The author has chosen to re-examine historically a number of aspects which relate to the deficiencies and pitfalls of diagnostic studies and techniques so as to direct attention to their existence and possible solution in the light of personal experience.

It is interesting to note that the past century has witnessed a number of outstanding advances in surgery and irradiation. The

development of new techniques and modalities has been coupled with considerable progress in informing and educating the profession and the public in general. This would tend to encourage patients to seek advice at an earlier stage of the disease. As a result of these advances and contributions to the literature extolling them, one would naturally expect a tremendous impact on survival rates. The reasons this is not the case is related to the diagnostic discrepancies that have been discussed and which necessitate more extensive forms of management. Also to be taken into account is the fact that for the last two decades patients have been given the benefit of surgical procedures not available to them before when their condition was judged as inoperable. It is understandable, therefore, that the morbidity and mortality rate, especially in this group, will affect the over-all figures.

It becomes apparent that all modalities and many disciplines acting in a co-ordinated therapeutic relationship must converge if we are to improve on the statistics relative to morbidity and mortality of laryngeal carcinoma. There is an increasing need for refining the techniques of indirect as well as direct laryngoscopy. We should increase the employment of laryngograms and exploit further the advantages of cineradiofluorography.

There is a need to interest more pathologists in the special problems posed by laryngeal tumors to the extent of having them as part of the team which is capable of viewing the neoplasm *in vivo*, just as does the radiotherapist. Frequent biopsies and long periods of observation have often been necessary to arrive at a diagnosis of cancer and by the time this occurs, the disease may have progressed to a stage which is difficult or impossible to eradicate. Should the biopsy study fail to reveal the conclusive type of pathology commensurate with the clinical picture, resort should be had to thyrotomy.

For many years, Maurice F. Snitman has studied surgical specimens by sectioning of the entire larynx in the frontal plane. This has proved to be an invaluable aid in the over-all assessment of the basic pathology and in the determination of the site of origin of the tumor (respiratory, transitional and stratified squamous epithelium). Studies of this type could be rewarding to follow responses to therapy; proclivities for growth, metastases, etc. The recent serial section studies of Gabriel Tucker, Jr. of the larynx can similarly throw light on the problem of management so as to improve statistics relative to morbidity and mortality.

Another not unimportant element which affects the laryngologist and his decisions is a human one, based upon his emotional content, his attitudes, his cumulative past experience and his physical state at the time of examination. At times, sentiment overshadows good reasoning and decisions relative to management are frequently not based upon logic. There are times when one modality or another (irradiation or surgery) may give the patient an equal chance for long term survival. Selection of the form of treatment to be advised is best determined by the skill and experience rather than a choice based upon timidity or sentimentality. The radiotherapist as an active participant of the team has much to offer. Not infrequently, paradoxical as it may seem, the therapist in reviewing the history and the findings, may be the very one who holds out for surgical intervention.

1853 WEST POLK ST.

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DISCUSSION

DR. ALDEN H. MILLER: In his exhaustive manuscript Dr. Lederer has interwoven the interesting history and development of laryngic diagnostic modalities with the deficiencies and problems inherent in their use in diagnosing cancer of the larynx and in indicating its successful treatment.

For the most part I can only agree heartily with his observations and second most of the points he makes.

I, too, am sure that an occasional exploratory diagnostic laryngofissure is necessary in demonstrating a neoplasm of the ventricle or false cord. The "blind spots" of the larynx that he enumerated cannot be over-stressed. I, too, have found that incomplete fixation of a cord does not always mean that the malignancy has extended off the cord. This possibility can be considered in evaluating the indications for either radiotherapy or surgery in those lesions, which on the basis of site and appearance only would usually have been treated conservatively.

The incidence of lymph node metastasis from posterior cordal lesions in my patients has not been nearly as high as Dr. Lederer has experienced.

Contrast laryngograms have been very valuable to me in delineating neoplasms of the under surfaces of the cords, the subglottic area and of the upper trachea. Lateral or frontal soft tissue roentgenograms of the neck, however, have given me little help.

I am most sure that close teamwork and interchange of experiences with one's pathologist is absolutely necessary. Correlation of clinical and surgical findings with the microscopic picture, and review of postoperative results with him have been most rewarding to me. For instance, recently, my pathologist is becoming convinced that a superficially spreading, verrucous carcinoma of the larynx can be attacked surgically in a less radical manner. This has resulted in my surgical treatment of these lesions being more conservative—and equally successful.

I, too, insist upon my pathologist "reading" all slides brought by the patient. I also insist upon our own repeat biopsy before doing a laryngectomy.

Recently the experiences of an increasing number of surgeons employing "conservation" techniques in laryngeal tumor surgery seems to point the way to the greatest change in our selection of therapy. The use of hemi-laryngectomy, transverse, vertical and anterior, would result in my disagreeing with Dr. Lederer's table of "definitive management" for his Groups of I to VIII based upon site and degree of involvement. Many lesions in Group III (posterior one-third with movable cord) might be cured without laryngectomy, especially if they were verrucous or superficial carcinomas. Three such patients of mine seem cured by laryngofissure.

Some of those lesions that appear to fall into his Group IV (cordal with fixation of cord) and his Group V (false cord or ventricle or arytenoid) are being successfully handled by hemi-laryngectomy.

Epiglottic (Group VI) lesions that do not come within one and one-half centimeters of the anterior commissure may be candidates for the transverse supraglottic laryngectomy. Recently, three such patients on our clinical service give every promise of eradication of their disease with conservation of a laryngeal air-way, phonation and satisfactory swallowing.

A word about "staging." The work of our committees in this area will certainly result in a better universal understanding of clinical descriptions but a universal understanding of microscopic descriptions and interpretations seems impos-

sible to me at this time. My own pathologist's conclusions regarding carcinoma in-situ and verrucous carcinoma could not be conveyed to anyone else through a table of staging. The newer conservation surgical techniques would seem to have to result in changes in the selection of treatment based on the T, N, and M staging. For the present I believe each surgeon must do his own staging based on each patient's individualized lesion.

I have enjoyed studying Dr. Lederer's entire paper; have gained from his historical research and his experiences and congratulate him upon its presentation.

DR. CHARLES MORGAN NORRIS: Dr. Richards and Members of the Association: I would like to say just one word about the matter of classification of laryngeal carcinoma. Much effort has been spent by the various committees on this project and the Joint Committee on Cancer Staging and End Results Reporting has recently published a proposed clinical stage classification based on the T N M System, in which letters and numbers are used to designate lesions of various types and prognostic implication.

The use of this classification and system of staging offers a number of problems, and I should like to draw just one analogy.

Some years ago, the system of nomenclature for the broncho-pulmonary segments proposed by Doctors Chevalier L. Jackson and John F. Huber was modified by one of the highly regarded thoracic surgeons who suggested that numbers instead of names be used for the various segments. Today, however, there are very few surgeons, radiologists, chest physicians or bronchologists who would designate a broncho-pulmonary segment by number.

I think that this may emphasize one of the large problems in classification and staging of laryngeal carcinoma (if the T N M System is to be used), namely that numbers and letters are not adequately descriptive. In other words, it is possible that such a system represents another example of "laryngic descriptive discrepancy."

DR. LEDERER (closing): I agree with my discussers and must acquaint the audience with the fact that I did not have an opportunity to give you my paper in extenso.

I am impressed, however, with the discrepancies which arise in the histologic diagnosis and these have been such time-taking affairs that this has constituted a delaying action.

Whether right or wrong, we follow a precept that Dr. Holinger has brought out on previous occasions. We do not accept a biopsy that is brought to us from elsewhere. At times this has paid off in behalf of the patient. However, at times this has been one of the delaying discrepancies which has been the undoing of the patient.

Now, there are two errors as far as the histology is concerned. One is a biologic one, wherein even the experienced pathologist may be challenged by "something that looks like cancer," where people will argue this out on a cellular basis. Drs. Ogura, McGavran and Bauer have given us considerable food for thought in this regard. They have divided them into the various types of cellular change. Whenever we deal with sections that are cut tangentially, we have seen a number of people who have come up with a diagnosis of carcinoma.

Now, relative to this interpretation, there is no problem that exists—please, I don't want to be misunderstood—there is no problem that exists when metaplasia is present. The polemics, with subsequent delays in establishing a conclusive diagnosis, are occasioned by cellular interpretation that does not coincide either with

direct or indirect laryngoscopy. Moreover, we have found universally that where atypical cells are reported by the pathologist, in our experience it is noteworthy that in almost all instances further study has revealed the presence of malignancy.

Now you know, gentlemen, that inasmuch as it is unacceptable, or unthinkable, even to give irradiation, much less do surgery, you can well imagine that until you get the conclusive biopsy there is a delay.

I am not talking about what is grossly visible and what is so apparent as tumor—whether this is happening to you or not, it does happen to us; that we do not always come up with the definitive diagnosis.

I appreciate the opportunity of airing this subject with you and I am sure that the succeeding two papers will dispel any anxieties you have about people not being able to make a correct diagnosis.

EXPERIENCE IN THE SURGICAL AND
RADIOLOGICAL TREATMENT IN 500 CASES OF
CARCINOMA OF THE LARYNX

DOUGLAS P. BRYCE, M.D.

(by invitation)

P. E. IRELAND, M.D. W. D. RIDER, M.B.

(by invitation)

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This review of our results of the treatment of laryngeal carcinoma was undertaken to compare our experience with radiotherapy and surgical excision, and to obtain guidance for our future planned therapy.

Within the scope of laryngeal carcinoma are lesions which respond extremely well to excision or radiotherapy, and those which are often unpredictably resistant to either. In addition adequate excision usually requires laryngectomy which remains as a severe handicap to the patient and a deterrent to the surgeon. The treatment of choice must be planned to produce maximum retention of laryngeal function without sacrificing survival.

The two present methods of primary treatment are in conflict on one point alone, and that is the conservation of the larynx. Radical surgery will produce by far the best survival rate, but at the expense of the loss of function. Irradiation will preserve function but suffers in comparison in that recurrences are very high particularly in advanced lesions. It has been the opinion of the clinical group at the University of Toronto and the Ontario Cancer Hospital that a combination of these methods of treatment is to be desired and would produce the best results. Thus, over the past ten years, there has been an increasing emphasis on primary radiotherapy particularly in

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the early stages of the disease, with surgical excision of the recurrences. This policy has resulted in a much greater emphasis being placed on the follow-up examination of patients, and in the co-operation of surgeon and radiotherapist. We have, furthermore, been required to a greater extent to support our opinions as to the proper treatment of any given lesion.

The diagnosis of recurrence of carcinoma at the earliest possible moment is essential to a program of pre-operative radiotherapy. It is very difficult to obtain a positive biopsy when tumor recurs deep to the surface epithelium. Increasing significance must be put upon persisting edema and inflammation, and changes in the clinical picture from one examination to the next. We feel that the importance of this follow-up examination cannot be overemphasized and that the examiner needs a great deal of experience in this particular aspect of examination.

Such a program also increases greatly the number of operative procedures carried out in irradiated tissue. The problems of post-operative necrosis and infection are more serious in these cases and must be planned for by improvements in surgical technique, and postoperative care. These technical difficulties alone should not be a contraindication if the combined method of treatment produces improvement in our results.

The present review is an attempt to assess our results and to form a basis for future planning. Six hundred and sixty-six cases were reviewed in order to form a basis of comparison for our recent experience. Techniques of treatment have changed so greatly that only the results of recent years have modern significance. Therefore, the patients seen during the years 1950 to 1960 were studied in more detail. Five year survival figures for this period of time were compiled but also a study of "successful" cases was adopted. This allowed us to include more recent cases which have not yet been followed for five years. A "successful" case is one in which the patient is free of disease either primary or secondary at his last examination. All cases have been followed for at least two years.

A further problem had to be solved in that classification of laryngeal carcinoma varies from center to center and makes comparison of figures difficult. It was decided to adopt the suggestion of the American Committee on International Classification of Laryngeal Carcinoma with certain modifications.

TABLE I
DISTRIBUTION OF LARYNGEAL CARCINOMA

REGION	SITE
GLOTTIC	VOCAL CORD ANTERIOR COMMISSURE
SUPRAGLOTTIC	POSTERIOR ASPECT OF EPIGLOTTIS VENTRICULAR BAND VENTRICLE
INLET	ANTERIOR THIRD MIDDLE THIRD POSTERIOR THIRD
SUBGLOTTIS	UNILATERAL SUBGLOTTIS ANTERIOR SUBCOMMISSURAL POSTERIOR SUBCOMMISSURAL
HYPOPHARYNX	PYRIFORM SINUS REGION RETRO-CRICOARYTENOID REGION POSTERO-LATERAL WALL

TABLE II
PRIMARY LESION ON ADMISSION

T CATEGORY	DESCRIPTION
I	ONE SITE NORMAL MOBILITY
II	ONE REGION NORMAL MOBILITY ONE SITE MOBILITY IMPAIRED ONE REGION MOBILITY IMPAIRED
III	TWO OR MORE REGIONS
IV	BEYOND LARYNX

TABLE III
REGIONAL NODES ON ADMISSION

N CATEGORY	DESCRIPTION
N ₀	NO NODES
N ₁	HOMOLATERAL MOVEABLE
N ₂	BILATERAL MOVEABLE
N ₃	FIXATION - UNILATERAL OR BILATERAL

TABLE IV

STAGE	CATEGORY		
	T	N	M
I	T ₁	N ₀	M ₀
II	T ₁ T ₂	N ₁ N ₀	M ₀ M ₀
III	T ₁ T ₂ T ₂ T ₃ T ₃ T ₃	N ₂ N ₁ N ₂ N ₁ N ₂ N ₀	M ₀ M ₀ M ₀ M ₀ M ₀ M ₀
IV	T ₄ or N ₃ or M ₁		

Table I provides for the division of carcinoma into five regions and each region into various sites. On the basis of the extent of the primary lesion a T rating is given as per Table II.

A further description of the lesion is given by the N rating which refers to secondary extension to the cervical nodes as in Table III.

TABLE V
5 YEAR SURVIVAL
DISTRIBUTION BY REGION

REGION	NUMBER	SURVIVAL	
		CR%	NET%
Glottic	329	53.0	61.0
Hypopharynx	150	13.6	14.7
Supraglottic	61	32.6	37.1
Inlet	49	22.5	28.1
Subglottic	14	*	*
Others (untreated inadequate description)	63	*	*
TOTAL	666	36.9	41.7

* Statistically not significant.

The letter M refers to distant metastases, and there are two categories, either M_0 when metastases are absent or M_1 when they are present. Staging of these lesions was accomplished in the suggested groupings seen in Table IV.

Such a classification and staging was relatively new and untried by our group, so that a further surgical staging was used, based on the type of surgical procedure which would classically be advocated for the lesion. This, it was felt, would provide a better means of comparison of surgical and radiotherapy treatment results. Thus an "early" stage refers to a lesion which would be treated by the classical laryngofissure operation. An "intermediate" stage lesion would require a laryngectomy or a laryngectomy and block dissection. A "late" stage in a glottic lesion includes all those cases with

TABLE VI

1934 - 1960

5-YEAR SURVIVAL BY TREATMENT

Primary Treatment (Combined)	Glottic		Hypopharynx		Supraglottic and Inlet	
	CR%	Net%	CR%	Net%	CR%	Net%
Surgery	62.5	72.9	17.2	17.9	53.0	63.0
Radiation	45.0	51.0	12.9	14.3	16.9	18.9

TABLE VII

1950--1960

5-YEAR SURVIVAL

DISTRIBUTION BY REGION

Region	Number	Survival	
		CR%	Net%
Glottic	229	55.8	65.3
Hypopharynx	109	10.4	11.6
Supraglottic and Inlet	72	31	38
Subglottic	9	57*	57*
Total	419	38.5	42.9

* Statistically not significant

secondary cervical extension or with the primary lesion on the border-line of operability. Such staging is probably rather loose at the present time due to the description of more recent surgical techniques, but does provide a basis for comparison of results for the surgeon.

Six hundred and sixty-six cases were reviewed, and of these, 66 were discarded either because they refused treatment or the description

TABLE VIII

1950 - 1960

5-YEAR SURVIVAL

DISTRIBUTION BY METHOD OF TREATMENT

Primary Treatment (Combined)	Survival					
	Glottic		Hypopharynx		Supraglottic and Inlet	
	CR%	Net%	CR%	Net%	CR%	Net%
Surgery	62.0	73.0	12.5	13.0	52.2	66.7
Radiation	55.8	65.3	10.4	11.6	5.3	6.3

of the lesion did not allow classification. The remainder were all adequately documented, treated, and followed. Table V shows the distribution of these cases and the five year survival rate by region.

The crude and net survival figures of all cases corresponds roughly to the results reported from most other unselected series.

Table VI illustrates the survival of all cases by primary method of treatment and region. The primary treatment refers to the original decision for treatment by the clinical group, and not the total treatment.

In the supraglottic and inlet regions which were combined to produce more significant figures, the superiority of surgical therapy on the basis of survival alone is apparent. The results in the hypopharyngeal lesions are very poor by either method.

In the years of 1950 to 1960 inclusive, four hundred and nineteen cases were studied and their distribution and five year survival figures are seen in Table VII.

The five year figures in this table do not differ greatly from the over-all group although there is a general slight improvement. The figures for subglottic carcinoma are not significant because of the small number of cases. In the past eleven years, the five year survival by treatment and region is shown in Table VIII.

TABLE IX

1950 - 1960

GLOTTIC STAGED

Stage	Primary Treatment	Cases	Successful				Nodes % of Total Treated
			Total		Laryngectomies		
			No.	%	No.	%	
I	Surgery	37	35	94	8	23	3.5
	Radiation	23	23	100	6	26	0
II	Surgery	51	37	72	31	84	24
	Radiation	49	33	79	9	27	18
III	Surgery	28	18	64	17	94	43
	Radiation	20	11	55	5	25	45
IV	Surgery	10	5	50	5	100	60
	Radiation	4	1	25	1	100	75
Early	Surgery	37	34	92	7	20	8
	Radiation	22	22	100	6	27	0
Intermed.	Surgery	71	52	73	45	87	30
	Radiation	63	43	68	9	21	23
Late	Surgery	18	9	50	9	100	61
	Radiation	11	3	27	1	33	54
All Stages	Surgery	126	95	74	61	64	28
	Radiation	96	68	71	20	29	23

These figures show a considerable improvement in the five year survival of glottic lesions treated by radiotherapy which is due to the improvement in techniques and the use of cobalt therapy. The reduced figures for the hypopharyngeal lesions are also probably due to the increased use of radiotherapy in this region. Supraglottic and inlet lesions continue to do very much better by surgical excision.

On the basis of the "successful case" and the salvage of the larynx a further study of the glottic lesion was carried out as seen in Table IX.

The successful cases as mentioned do not all have a five year follow-up period. The percentage of laryngectomies was calculated

TABLE X

1950 - 1960

GLOTTIC

SURVIVAL BY METHOD OF TREATMENT

Treatment (Primary)	2-Year		3-Year		5-Year	
	CR%	Net%	CR%	Net%	CR%	Net%
Partial Laryngectomy	86	94	77	92	73	87
Laryngectomy	63	75	56	68	46	58
Laryngectomy and Block	84	91	79.1	90.5	77	77
Cobalt	81	85	73	77	61	67
Other Radiation	22	36	22	30	24	33

on the successful cases only. In the early stages of glottic lesions, therefore, primary radiotherapy will produce a survival figure comparable to surgery and in the successful cases three-quarters of the patients will retain their larynxes. Only in the late cases does primary surgical excision produce results which warrant universal laryngectomy.

It might be argued that the delay of excision resulting from radiotherapy would cause an increase in dissemination of the tumor to cervical nodes. This is not supported by our results.

A further breakdown in survival among the glottic lesions with reference to method of primary therapy is seen in Table X.

The interesting features of this table are the relatively good results with cobalt irradiation compared to other methods of irradiation and also in relation to laryngectomy alone. Laryngectomy with

TABLE XI

1950 - 1960

GLOTTIC

COURSE OF THERAPY

Cases	Primary Therapy	Subsequent Therapy		
		Further Surgery	Further Radiation	Combination
45	Partial Laryngectomy	8	1	2
52	Laryngectomy	2	9	
29	Laryngectomy and Block		2	
78	Cobalt	25		6
18	Other Radiation	2	3	1

TABLE XII

1950 - 1960

GLOTTIC - SURVIVAL

Stage	Primary Treatment	Cases	2-Year		3-Year		5-Year	
			CR%	Net%	CR%	Net%	CR%	Net%
Early	Surgery Radiation	37	95	95	83	94	81	92
		22	100	100	94	100	83	100
Inter-mediate	Surgery Radiation	71	72	85	64	79	57	73
		63	70	76	61	66	48	56
Late	Surgery Radiation	18	67	71	62.5	67	38	38
		11	9	11	0	0	0	0

TABLE XIII
1950 - 1960

HYPOPHARYNX - SURVIVAL

Stage	Primary Treatment	Cases	2-Year		3-Year		5-Year	
			CR%	Net%	CR%	Net%	CR%	Net%
All	Surgery Radiation	33	42	45	34	37	12	13
		73	27	28	21	22	10	11
I & II	Surgery Radiation	11	45	50	33	37	14	17
		15	46	50	45	50	10	17
III & IV	Surgery Radiation	22	41	43	35	37	12	12
		58	22	23	16	17	10	10

block dissection has produced the best over-all figure except for partial laryngectomy in which much earlier stages are treated.

In Table XI a breakdown of the subsequent therapy in glottic cases which have been treated primarily by specific surgical procedures or radiation is presented. It is apparent that radiation by itself is not sufficient but must be complemented by careful follow-up and surgical excision for recurrences.

Survival figures by method of treatment and the surgical staging of early, intermediate and late are shown in Table XII.

It is again demonstrated in the early stages, that relatively comparable results to surgery can be obtained by radiotherapy as a primary method of treatment.

Our experience with hypopharyngeal lesions has up to the present time been discouraging. The results are outlined in Table XIII.

The results of surgical excision appear to be slightly superior to radiotherapy but still far from satisfactory. In Table XIV the high percentage of spread to cervical nodes is seen and again it is demonstrated that some larynxes can be saved, if radiation is the primary method of treatment.

The lesions of the supraglottic region and inlet are undoubtedly best treated by surgical excision. However, the cases treated were chiefly in the late stages and the survival rates were not good.

TABLE XIV

1950 - 1960

HYPOPHARYNX - STAGED

Stage	Primary Treatment	Successful %	Nodes %	Larynx Saved %
All	Surgery	33	85	0
	Radiotherapy	21	68	20
I	Surgery	45	72	0
II	Radiotherapy	47	40	28
III	Surgery	27	90	0
IV	Radiotherapy	14	72	12

TABLE XV

1950 - 1960

SUPRAGLOTTIC AND INLET - SURVIVAL

Stage	Primary Treatment	Cases	2-Year		3-Year		5-Year	
			CR%	Net%	CR%	Net%	CR%	Net%
All	Surgery	40	75	83	66	75	52	67
	Radiation	32	31	35	28	31	5	6
I & II	Surgery	8	100	100	100	100	86	100
	Radiation	7	71	83	80	80	33	50
III & IV	Surgery	32	69	79	54	65	37	50
		25	20	20	18	19	0	0

Table XVI would suggest also that sufficient larynx can be saved in early cases by radiotherapy to encourage us to persist with this method of therapy.

Subglottic lesions in our series were very few. This does not refer to subglottic spread of a glottic lesion but to a tumor confined

TABLE XVI

1950 - 1960

SUPRAGLOTTIC AND INLET - STAGED

Stage	Primary Treatment	Successful %	Nodes %	Larynx Saved %
All	Surgery	68	62	0
	Radiotherapy	31	60	91
I	Surgery	88	37	0
II	Radiotherapy	71	14	80
III	Surgery	67	70	0
IV	Radiotherapy	20	72	100
Inter- mediate	Surgery	77	77	0
	Radiotherapy	77	10	87
Late	Surgery	59	73	0
	Radiotherapy	13	78	100

TABLE XVII

1950 - 1960 SUBGLOTTIC - ALL STAGES

5-YEAR SURVIVAL

Primary Treatment	Cases No.	Survival No.
Surgery	3	2
Radiation	4	2
Combined	2	2 (3 year)

to the subglottic region as the site of origin. Our meager experience is seen in Table XVII.

COMMENT

The cases reported have in the most part been seen by a clinical group with an increasing emphasis on joint decision for the method of therapy and with general consultation on follow-up and the choice of subsequent treatment.

We have considered that the early glottic lesion could be treated equally well either by radiotherapy, or surgery, but surgery would produce an inferior voice. There is another feature, however, in that a full course of radiotherapy is a serious and debilitating experience especially for the elderly. In old and poor risk patients limited surgery may be the treatment of choice.

In moderately advanced glottic lesions we believed that radiotherapy as a primary method of treatment would produce results comparable to surgery, and that the number of larynxes preserved would far outweigh any slight reduction in survival. The most important consideration in this group is the follow-up. In order to justify this method of management, recurrence must be recognized at the earliest possible moment and, if there is a reasonable suspicion, a laryngectomy and block dissection should be done despite a negative biopsy. Our results have supported our choice of the method for treatment of these lesions.

In the late stage of the glottic type we have favored surgical excision, sometimes with radiation before or after the surgery. Our results have been poor and the number of larynxes saved negligible.

The results in hypopharyngeal lesions have also been discouraging by any method of treatment. We have adopted recently a policy of elective pre-operative radiation with radical excision six weeks after the completion of radiotherapy and we hope that this will improve our figures. The results in this series would suggest that both radiotherapy and surgery fail miserably to cure disease in this region.

The inlet and supraglottic tumors have not been treated with any definite policy except that we have felt that the false cord lesions, with anterior excavation, must be operated upon. Our results would strongly support surgical treatment in these regions, except perhaps in the early stages.

CONCLUSIONS

Carcinoma of the larynx is a disease, which in favorable regions, and in the early stages, can be cured in a quite remarkable percentage of cases. Treatment should be by a combination of radiotherapy and surgical excision. Both methods of treatment are mutually supporting and not competitive. The most effective combination must be the decision of a combined clinical group. The follow-up is of permanent importance.

Radiotherapy as a primary method of therapy will, in combination with surgery, give the best results in early and intermediate glottic lesions and three-quarters of the survivors will retain their larynxes.

Improvement in the results of late glottic, inlet and supraglottic lesions as well as all hypopharyngeal lesions will be obtained by an elective program of therapy consisting of a full course of radiotherapy followed by subsequent radical surgical excision of the primary and secondary disease six weeks after the course of radiation. The decision for this operative procedure must not await evidence of recurrence of the disease.

We have not found previous irradiation to significantly affect subsequent surgical procedure, provided that the surgical technique takes this complication into consideration and that the procedure is properly timed.

UNIVERSITY OF TORONTO

DISCUSSION

DR. PAUL H. HOLINGER: Mr. Chairman, Dr. Bryce whom we now welcome as a Fellow member, and members of the American Laryngological Association:

This paper is another important contribution to the study of Cancer of the Larynx. It is a group effort and provides opportunity for comparative studies with other series, whether or not we wish to agree with the conclusions. It is of interest that the authors use the concept of a study of the *successful* cases versus an analysis of the failures, "all cases having been followed for at least two years."

The title, "experience with the treatment of 500 cases of Cancer of the Larynx," is somewhat misleading. There are 666 cases mentioned in the series, 10% of which are deleted for various reasons. Of the 603 cases remaining, 150 are cases of Hypopharyngeal carcinoma. These are important and well documented. Unfortunately, they will be lost in the literature unless the authors add to the title "Treatment of Cancer of the Larynx and Hypopharynx." Hypopharyngeal carcinomas are separate

entities; but, unfortunately, they are included as a separate "region" in the essayists' Table V of "Distribution of Laryngeal Carcinoma."

In adopting the classification of the American Committee on Classification of Laryngeal Carcinoma, they add still another region; "Inlet" lesions are added to the list of regions, subdividing these into anterior, middle and posterior sites. For an analysis of results, they are combined "to produce a more significant figure" with supraglottic lesions. Their supraglottic lesions include the ventricular bands, ventricles and posterior aspect of the epiglottis; and the glottic lesions consist of lesions of the vocal cords and anterior commissure. Thus, the "Inlet" lesions must bridge this somehow making it impossible for comparative statistical studies to differentiate the glottic and supraglottic groups. As a matter of fact, I am not quite clear as to the anatomical site or sites of "Inlet" lesions.

For the record and for comparison with other series, it should be pointed out that the authors vary slightly their staging from that developed by the American Committee on Classification. Obviously, they may if they wish, but it denies direct comparison.

In general, the statistics of cure rates are fair. They are better than some, but not quite as good as others. Possibly the classification modifications work against the series.

Particularly confusing to me are Tables V through VIII recording 5 year survivals of 1950 to 1960 series of patients; and Table XVI that indicates the percent of successfully treated cases. Table V lists the entire 666 patients; Table VI lists the 5 year survivals from 1934 to 1960 for comparison of therapy with the 1950 to 1960 group. Again, on a "five-year survival" basis, 1957 should be a shut-off point if the tables are accurate summaries of 5 year survivals. The conclusions drawn in regard to improvements in therapy could only too easily be negated in the next two or three years as a significant group of these patients actually reach the 5 year survival milestone. Possibly not, but this is quite a loophole.

Aside from the statistical analysis, certain conclusions may be further discussed. The authors are obviously shifting toward increasing the indications for radiation, first in the early glottic lesions to save the larynx and provide a better voice, and second, as primary treatment in "moderately advanced glottic lesions" to preserve the larynx of some at the expense of a slight reduction of survival. I agree in part with the first point with some exceptions as the authors indicated. But let us break the concept that the voice is invariably better after irradiation than after a limited cordectomy. It is time we challenge this statement because too often prolonged post-irradiation edema and chronic perichondritis produce huskiness that limits the conversation or speech of the preacher, lawyer, salesman or other professional voice user far beyond that of a similar individual with a fissure.

In regard to the second point, I feel the authors are advocating the concept of "try irradiation first; you can always operate." I admire the team work of the group and their diagnostic acumen that permits them to recognize residual or recurrent carcinoma in the irradiated larynx in sufficient time to resect the larynx and still have only a slight reduction in survival. They do indicate that this is done *on reasonable suspicion despite a negative biopsy*. This is probably a key that requires the keenest judgment to use. This philosophy, while possibly not new, is difficult to accept unless we introduce the concept of definite and planned surgery in the immediate post-irradiation period. The authors recognize the growing swing toward planned surgery after irradiation, but their statistics were not clear to me as to which patients had *planned surgery* after irradiation, and which had had surgery because recurrence was suspected or identified by a biopsy.

A wealth of material has been presented and I extend my congratulations to the authors for the amount of work we all know this has required. I hope that my remarks can be accepted as they are intended, namely, that the discussion will clarify some of the questions that arose in my mind as I had the opportunity to study the advanced draft of this paper.

DR. PAUL M. MOORE: I would like to mention a personal discouragement with the result of irradiation in a case that I acquired recently. As far as I could find out, this woman had an early lesion of one cord about two years ago. She refused surgery and had irradiation. I got her last November. We had to do a tracheotomy because the vocal cords were frozen and the larynx was full of carcinoma. She couldn't tolerate further irradiation. We did a laryngectomy. The tissues broke down. We made pedical flaps, but she still has a bit of a leak.

DR. IRELAND: I am sure I speak for Dr. Bryce too when I say that we wish to thank Dr. Holinger for his—I was going to say “destructive criticism” but it was not meant that way. I think a lot of the discussion was quite correct. I can not make the excuse of saying that explanations are going to be found in the printed paper because he had a copy of the paper for publication and we talked about this before the meeting today. I know he feels very strongly about staging and about having this standardizd as far as nomenclature goes. I think perhaps this is quite right.

On the other hand, at the beginning of this paper we stated that this was an attempt to make a survey on what we were doing about carcinoma and seek what usefulness we might get from the information that we obtained.

I don't think we care particularly whether these people are five-year cures or not. We are trying to find out, “Have we been doing the right thing, over the past ten years, in the way we are treating these patients?”

As Dr. Bryce stated, a large majority of these people were over the five years. The others were not presented as five-year cures but as the general picture. I would suggest, there may be some mix-up with regard to the tables and staging, especially as we know that Dr. Holinger is very much in favor of having a standardized type of staging.

I would also like to partly agree with Dr. Lederer and say that perhaps a few of us can be a little original at times and do something that we find is useful to us personally in classifications.

There are so many points that he made. I think Dr. Bryce did mention with regard to maintaining a good voice in carcinoma of the larynx. Also that he wasn't in the habit of radiating every one of these cases and he did mention laryngofissure. I think there is more about this in the written paper.

The strang thing is that when we went over these figures the best results that we had in any of the long-term series are the ones that had laryngofissure. This is exactly what I thought in 1935, or 1934, when I took my American Board, and we still discovered a large group of patients that I had operated on before the War who are still going strong. These had laryngofissure and this is all they did have done. I certainly think there is a place for limited surgery in the larynx and we do not send everybody for treatment by cobalt therapy.

I do not think we know the answer to treatment of this disease. I am sure this is true in our group and I hope somebody comes along who is clever enough to tell us what we are going to do about cancer.

Dr. Holinger did feel that it was radical to decide, on follow-up, to do a laryngectomy on patients without further positive biopsy. This group must be carefully assessed and frequently seen. We do know they have had cancer and doubt they are cured. We are aware of the radiation dosage and its adequacy. They persist with oedema, hoarseness and dysphagia. Duplicate biopsies have proved negative. These are especially true when there has been extension to the ventricle. After all, this person has a known cancer and most likely still has and I can see no objection to a laryngectomy. Perhaps we were just wrong and we should have taken out the larynx at the beginning.

As stated, there is a wealth of material and it is surprising what you can get from an analog computer. That is another thing which is going to make it difficult, in our staging. If we are going to change the staging so often then we are going to have punch cards of little value. Even now when you start a review you find you must go through the computer for everything only to find you must review also these histories. I am a little afraid that we may get to depend too much on punch cards and analog computers. I am scared to death of them myself and the answers they give you.

There is a lot more material that I hope we can get time to analyze, or get somebody to analyze for us so that we will be able to get more information from this series of cases.

To Dr. Paul Moore: I don't think that there is anyone who has not been discouraged about using cobalt on a carcinoma of the vocal cord. I certainly have been. You said two years for the recurrence, did you?

CHAIRMAN MOORE: Yes.

DR. IRELAND: Well, even four years—I have one very fine businessman who has now been four years and he comes back with carcinoma of the larynx and extensive secondaries. He just didn't come back to see anybody—and I don't suppose it would have made any difference. Perhaps the good Lord has a lot to do with it in certain instances.

I am very proud to have Dr. Bryce present this paper today and I was very proud to bring him up this morning and introduce him to this very distinguished group as a newly-elected member.

DR. BRYCE (closing): It seems to me, Mr. Chairman, that for someone whom everyone is so proud of, I am taking quite a beating.

It was not planned that I should enter the discussion at this stage because Dr. Ireland was going to conclude for me. However, I think I do need to protect myself a little.

When we prepared this paper we did so fully expecting that we would be criticized. This was our expectation and we were certainly not disappointed.

The staging and classification presented was used in order that we could gain experience with this method. We also classified our tumors surgically as you will see in the published paper.

We anticipated criticism of our method of reporting our cases which had not yet reached a five-year survival period. I stated quite clearly, however, that such a method was used deliberately to attempt to find out something about the cases treated by more modern methods since 1957. In our opinion, also, the five-year survival figure is a somewhat arbitrary yard-stick. It is most observers' opinion that the survival curve flattens very considerably after three years and I think that such

a method of reporting should not be unduly criticized. Furthermore many five-year survival cases have survived five years but still have their tumor, so that in this way, also, a five-year survival figure leaves something to be desired.

Dr. Holinger has criticized our statement that we would operate on a patient following radiotherapy if we were suspicious of a recurrence even with a negative biopsy. We feel that the patient's treatment should be decided on his first visit and if we have decided to operate on him following radiotherapy then this procedure should be carried out despite the clinical appearance of the lesion. It is our feeling that the surgical excision has been delayed too long when a recurrence is obvious. We recently had six cases of carcinoma of the pyriform sinus which we elected to excise following radiotherapy at a six-week post-radiation period. These were then serially sectioned and a search made for the tumor. In one case there was an obvious recurrence. In five cases there was no evidence of recurrence and of those five only in one could we find no carcinoma. In the other four islands of carcinoma were found throughout the section often a centimeter or more deep to the mucosa. A positive biopsy could not be obtained in these cases. We know from our experience that radiotherapy does not cure these cases, but it does cause the tumor to shrink and be less active and more favorable for surgical resection.

So, sir, thank you for the opportunity to protect myself and thank you, Dr Holinger, for your discussion.

TECHNIQUES IN LARYNGECTOMY
TO MINIMIZE POSTOPERATIVE COMPLICATIONS
AND PERMIT IMMEDIATE FEEDING

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(by invitation)
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This paper describes certain techniques for laryngectomy which minimize postoperative complications and permit immediate feeding. The techniques reduce the chances of pharyngeal fistula and hematoma, two of the major postoperative complications. Because they prevent healing by primary intention, they contribute to increased morbidity and mortality.

Fistula formation occurs often enough that most surgeons are reluctant to begin early oral feedings. They believe it is best to delay and "let the suture line heal" before subjecting it to stress. Although fed through nasogastric tubes for a week or 10 days, patients nevertheless swallow saliva. Thus a fistula sometimes appears even before regular feedings commence. When fistula is discovered, it is customary to leave the nasogastric feeding tube in place until the fistula closes, sometimes weeks later.

Some patients develop hematoma. Then it is necessary to open the wound widely, evacuate the hematoma, and wait for healing by secondary intention. If the patient also develops a fistula, the difficulty is compounded. Necrosis of the carotid artery (especially in a patient who has received irradiation therapy) becomes a dreaded possibility.

To minimize these difficulties, the following technique of laryngectomy is presented. Most of the scheme differs from standard laryngectomy procedure and some aspects may be considered radical.

Yet in basic concept the technique does not differ from that employed by most experienced and careful surgeons, for it is based on the principles that underlie any method: tight closure of the pharyngeal defect, adequate drainage of the wound and pressure against skin flaps. This technique which was used first in 25 dogs is now used in all patients undergoing laryngectomy or laryngectomy and neck dissection on the otolaryngology service at University Hospital.

SYNTHETIC SUTURES

One part of the technique is using synthetic sutures, especially those made of nylon. Silk and catgut are the customary suture materials used to close the pharyngeal defect after laryngectomy. By now, however, many investigators have demonstrated that synthetic sutures cause less tissue reaction and are stronger than either silk or catgut. These new suture materials are made of nylon or dacron. Nylon sutures are monofilamentous, while dacron sutures are braided. Familiar nylon sutures are Ethilon,[®] Dermalon,[®] and Nylon.[®]

Synthetic sutures are available in several forms: on spools, in cut lengths, or attached to cutting and round needles. Their tying characteristics are somewhat different from those of other suture materials. A surgeon's knot followed by a complete square knot is the only secure way to tie synthetic sutures.

To close the pharyngeal defect, 4-0 nylon suture is strong enough except at the base of the tongue, where 3-0 suture may be required. I have used both nylon and dacron and *prefer nylon*, especially Ethilon. Another product, Tevdec,[®] formerly Tefdac, must be tied so carefully that it is impractical.

SUTURE TECHNIQUE

Many different suturing techniques are used to close the pharyngeal defect after laryngectomy. I prefer an interrupted stitch that inverts the mucosa. Any irregular mucosal edges should be trimmed so that no everted mucosa is left by accident. Mucosa cannot unite with mucosa any more than unbroken skin can unite with unbroken skin. There can be union only between two raw surfaces—in this instance, submucosa and submucosa.

After the larynx is removed, it is common for the pharyngeal defect to present a pouch of redundant mucosa at its lower end. This pouch should be cut off (Fig. 1).

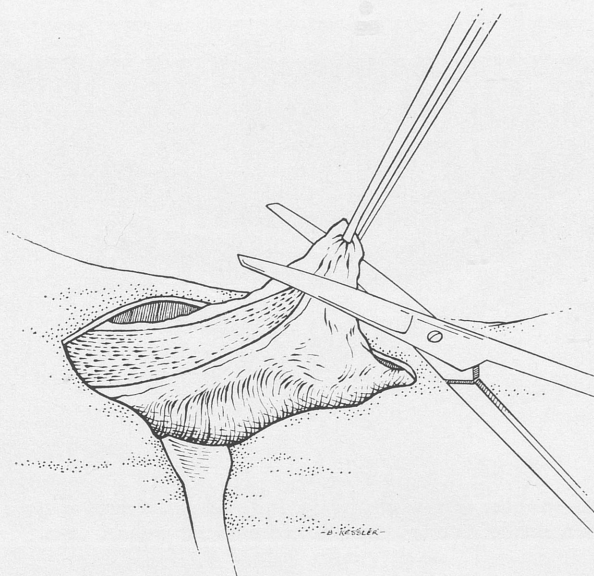


Fig. 1.—Trimming off a pouch of excess mucosa. Any irregular mucosal edges should also be trimmed to make sewing easier.

The first bite of the needle picks up strong submucosa about 5 to 10 mm from the cut edge; the second bite picks up the back of mucosa as near as possible to the edge of the pharyngeal defect, but it does not penetrate the mucosa; the third bite (on the opposite side of the defect) corresponds to the second; the fourth corresponds to the first. When a knot is tied, the two bites nearest the defect draw together and invert the mucosa; on top of that, pulled down tight, is a layer of substantial submucosa formed by the first and fourth bites. A series of these interrupted stitches with an average spacing of 4 to 5 mm closes the pharyngeal defect. Care should be taken not to place any of the first row of sutures in muscle because muscle provides little strength for the suture line. Muscular and facial layers can be pulled together later.

If the first layer is tight, there will be no fistula. If a gap is left or if the sutures untie, no matter how carefully any subsequent layer is closed, a fistula is likely.

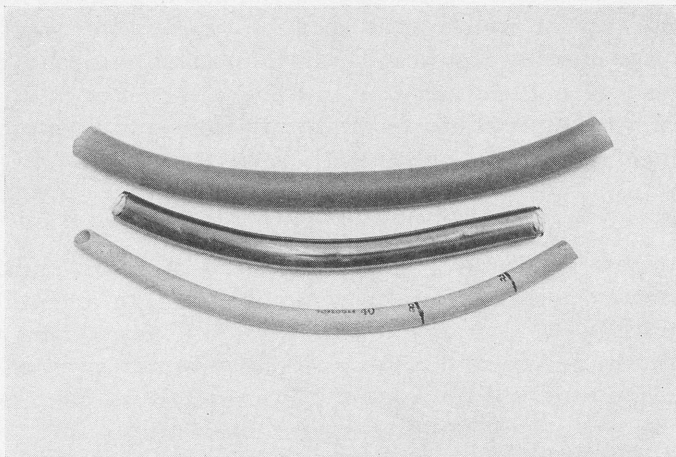


Fig. 2.—Rubber and plastic tubing for use as a 'darner' or form to sew across. Even smaller tubing may be needed in exceptional cases.

SEWING OVER A FORM

A flexible rubber or plastic tube serves admirably as a 'darner' or form over which to close the pharyngeal mucosa (Figs. 2 and 3). Preferably the tube should be white, and it should be no more than an inch in outside diameter (smaller sizes also should be available). It is used in the same way that a housewife uses a darner over which to mend socks. One end of the tube is passed into the esophagus and the other end directed into the oral cavity. After the pharynx is closed, the anesthetist pulls the form out the mouth.

The chief advantage of the 'darner' is that it enables the surgeon to detect gaps in the all-important first suture line. These gaps, which permit a fistula to form, are particularly easy to leave at the base of the tongue. In addition, the tube stabilizes the mucosal edges and makes suturing easier.

CLOSED SUCTION DRAINAGE

Another important part of the technique is closed suction drainage (Figs. 4 and 5). This type of drainage, which is much more efficient than the customary passive type drainage with hard rubber or Penrose tubes, eliminates the danger of hematoma and makes pres-

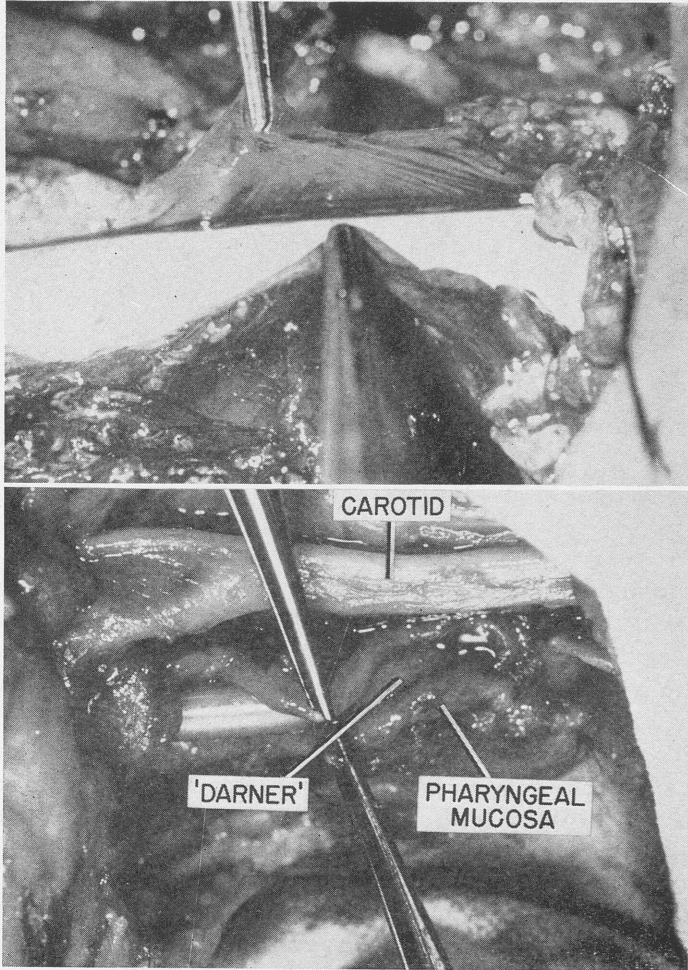


Fig. 3.—*Above*, a large white 'darker' in place. One end reaches the mouth and will be pulled out after the suture line is intact. Here there is ample mucosa. *Below*, a number 28 Robinson catheter used as a 'darker' in a patient with minimal mucosa for closure. Compare size of tubing with the common carotid artery.

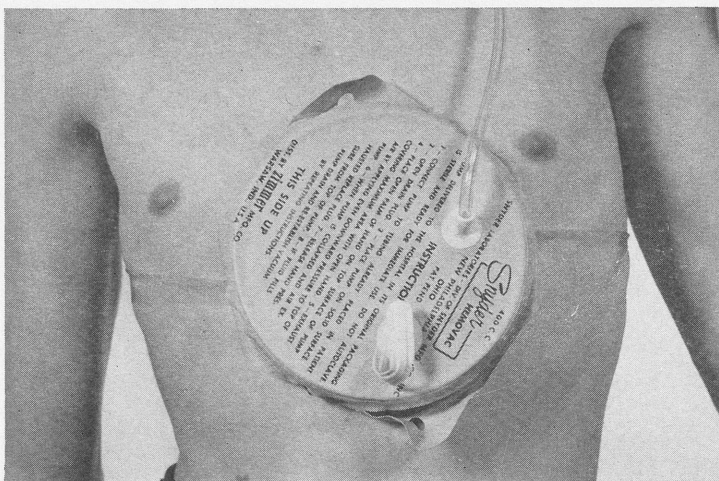


Fig. 4.—Closed suction drainage—the Hemovac unit. This is the sterile plastic container which is both the suction unit and the receptacle for blood.

sure dressings unnecessary. It enables the physician to follow the progress of the healing process. The closed suction system has drainage tubes that have an airtight connection with the suction source. The skin is sealed around the tubes by a purse string suture. If closed suction is to be effective, the suture line in the neck must also be airtight, but making it so is an easy matter. Other systems of drainage in which one tube sucks and another admits air to the neck are not the same. The chief advantages associated with a closed system of constant suction drainage are these:

1. Drainage is more efficient by closed suction than by rubber drains. In closed suction blood is evacuated as soon as bleeding occurs. In contrast, ordinary drains permit blood to collect in quantity; sometimes the clots that form cannot drain through even a large tube. By macerating the pharyngeal suture line, clots and liquid blood weaken it and contribute to formation of fistula.

The skin and suture line remain absolutely dry, since all drainage runs into a sterile plastic container. The container, which is both the suction unit and the receptacle for blood, can be emptied as often as necessary, usually twice a day. Suction is renewed by opening a

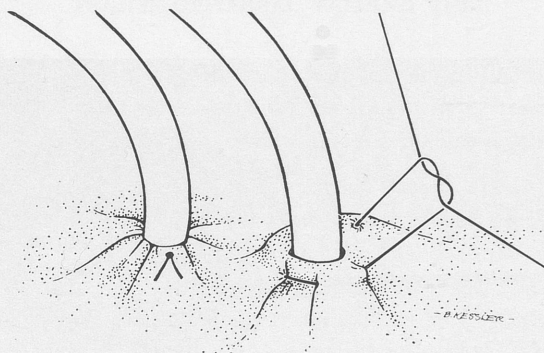


Fig. 5.—Drainage tubes passing through skin must be sealed tight. The needle from the Hemovac set seen in Figure 5-A, is used to draw the plastic tubes through the skin.

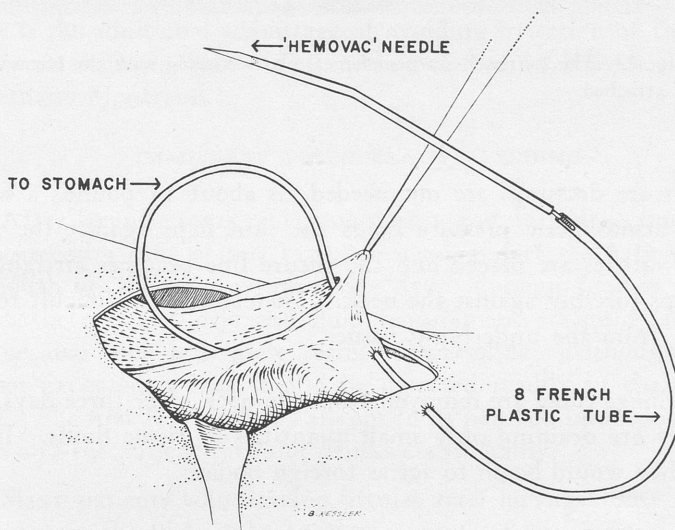


Fig. 6.—Transesophageal feeding tube passing through wall of esophagus and skin of lower neck.

seal on top, pressing down the lid to compress a spring in the container, and closing the seal.

2. No neck dressing is needed, either to collect blood or to achieve pressure. The only dressing is a plastic spray to protect the wound against bacterial contamination.

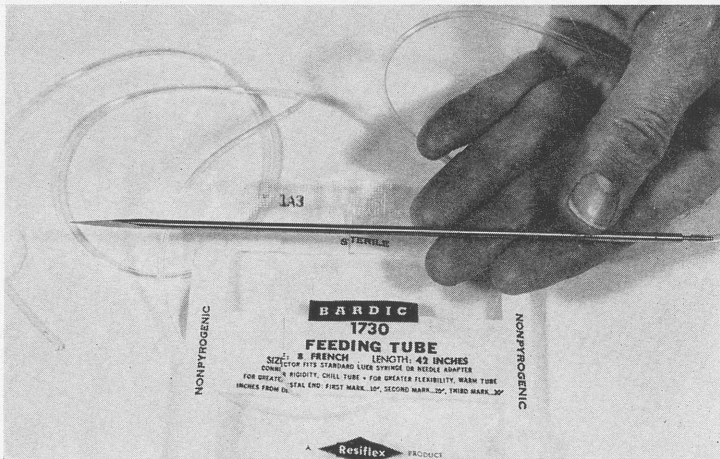


Fig. 7.—The 8-French transesophageal plastic tubing with the Hemovac needle attached.

Pressure dressings are not needed, as about 15 pounds a square inch of atmospheric pressure holds the skin flaps against the neck. As skin sutures are placed and the suture line becomes airtight, the skin snaps forcibly against the neck. Thereafter it is difficult to pull the skin from the underlying tissue.

Drainage tubes are removed from the neck after three days. By then they are draining only small quantities of tissue fluids. If left longer they would begin to act as foreign bodies.

There is great reassurance to being able to look at a wound and to know what is happening. When massive neck dressings cover a wound one really never knows what is going on until the dressing is removed. I often have hesitated to remove thick layers of gauze and mechanic's waste for fear of what I would find. Massive dressings intended to provide pressure sometimes come to do just the opposite. Blood wets the dressing and loosens it. Then by its own weight the dressing and attached skin pull away from the neck. One must try closed suction to appreciate its advantages.

TRANSESOPHAGEAL FEEDING TUBE

For those not wanting to feed their patients by mouth immediately, I recommend a transesophageal feeding tube instead of the usual nasogastric tube, which some writers say may predispose to fistula.

A small plastic feeding tube, size 8 French, is fitted with the needle from the Hemovac set and drawn through the esophagus a few centimeters below the pharyngeal defect (Figs. 6 and 7). The needle pulls the tube from inside the lumen through the esophageal wall and through skin of the lower neck. Finally, one end of the tube is inserted into the esophagus and the other taped carefully to the skin. The tube is large enough to permit feeding of formulas, but not so large as to cause a fistula when it is withdrawn a week later.

Transesophageal feeding tubes have not caused infections or fistulas in my patients, but they have increased their comfort greatly. There is the additional advantage of avoiding irritation of the suture line by a nasogastric tube. This method of feeding was described first by Fernand Montreuil.

IMMEDIATE POSTOPERATIVE FEEDING

After laryngectomy or laryngectomy and neck dissection, I now use no feeding tube of any type. Instead, I start the patient eating by mouth on the first postoperative day. Enough food is given to satisfy the entire caloric and fluid requirement. Liquids are given first because they leak more readily than solids. Although fistulas are not expected, a leak should be detected readily in the drainage tubes. The first few swallows are apt to be painful, but soon the pain leaves and the patient eats and drinks comfortably.

Many patients who develop fistulas after laryngectomy begin to do so before they leave the operating room. Saliva escapes through a leaking suture line and later other liquids simply follow the established tract. When we delay feedings until the seventh postoperative day, we feed patients when their suture lines are weakest. The wound is stronger both earlier and later.

TRACHEOSTOMY STOMA

The tracheostomy stoma is formed using 4-0 interrupted sutures. Nylon is particularly good here because this monofilamentous suture,

which causes no apparent reaction, can be left indefinitely without producing the crusting and stitch abscesses so common with silk and catgut. Infection is one of the chief causes of postoperative constriction of the stoma. If one forms the tracheostomy with nylon, and then does *not* insert a laryngectomy tube, healing proceeds under optimal conditions.

SUMMARY AND CONCLUSIONS

The two major postoperative complications of laryngectomy, fistula and hematoma, can be minimized by a technique that includes synthetic sutures, an interrupted stitch, a form over which to sew, drainage by closed suction, and a transesophageal feeding tube, or better, no feeding tube at all. Although the plan differs considerably from standard procedures, the basic concepts are those employed by most experienced and careful surgeons—tight closure of defect, adequate drainage and pressure against skin flaps.

Feeding by mouth on the day after surgery is recommended.

UNIVERSITY HOSPITAL

DISCUSSION

DR. GEORGE REED: Mr. Chairman, members and guests, it is indeed a pleasure to discuss Dr. Saunders' interesting presentation. I would like to commend him for bringing to our attention some of these finer points in the operative and postoperative care of laryngectomized patients. I would like to discuss briefly some of the points where Dr. Saunders and I would disagree and contribute some thoughts and observations from our experiences at the Mass. Eye and Ear Infirmary.

Dr. Saunders states that his techniques "permit immediate feeding." I would disagree in that this implies that other methods do not allow immediate postoperative feeding without dire consequences. Using other techniques, I have tried immediate feeding in many cases and although we have gotten away with it many times, we have felt that the incidence of fistula formation was higher in patients who were fed immediately than in those who were maintained on intravenous fluids, for a period of four or five days. I would agree heartily, however, that wherever possible the use of a nasogastric feeding tube should be avoided.

In references to using nonabsorbable sutures for the esophageal mucosa I would like to suggest the possibility of using wire instead of synthetic suture material. Although I do not have enough cases at the present time to draw any real conclusions, I have used 4-0 braided, stainless steel wire in approximately ten patients in recent months with very good results. Many of these patients have had pre-operative radiation and have had delayed surface wound healing but have not fistulized. Only one has fistulized—this being a patient who had 6,000 r of pre-operative radiation with considerable surface wound breakdown. Remarkably enough he did not fistulize until 6 weeks postoperatively, and this only a small five-millimeter opening which closed spontaneously. Wire sutures are somewhat more difficult to work with

than silk or synthetic material, but I find that by using two running layers the closure is very solid and quite easy to perform. The first layer is used to close the mucosa with inversion of the edges. The second layer encompasses the mucosa more distally and also any fragments of muscle which are available to strengthen the suture line.

I am sure we have all been impressed in recent years with the advantages of the probable closed suction drainage or "Hemovac" apparatus. I used to take great pains to be sure that the skin suture line was airtight in the operating room, but I find that this is unnecessary since if there are any leaks these are very quickly sealed off by serum. When using the Hemovac I agree that no dressing is needed, but I think the plastic spray is also unnecessary and constitutes a nuisance when the sutures are removed.

Early in the days of using closed suction, we were impressed with an increased incidence of wound infection, and finally realized that many of these infections were probably arising from the reservoir of the apparatus. It certainly stands to reason that in dealing with a wound that has communicated with the infected pharynx, that some organisms are almost certain to be drawn down through the tubing into the vacuum reservoir where they encounter a perfectly delightful culture medium in the form of aspirated blood and serum. It is quite easy to visualize infection from this culture medium going up the tube back into the wound. For this reason, we never leave the drainage system in place more than two or three days and insist on as much sterility as possible when emptying the reservoir. It is also a good technique to clamp the drainage tube when the vacuum is released while emptying the reservoir.

When we discussed this retrograde infection problem with Miss Mangiaracine, our bacteriologist, she suggested that it would be a very effective and inexpensive measure to merely insert a large dose of Penicillin and Streptomycin into the reservoir to prevent the active growth of organisms. Using all of these measures, we now feel that we can take full advantage of closed suction drainage without an undue amount of retrograde infection.

Finally, in regard to stomal techniques, I both agree and disagree with Dr. Saunders. I strongly disagree with his opinion that nylon sutures about the stoma produce less crusting, reaction and stitch abscesses than catgut. For years, we used heavy nylon sutures around the stoma, but only recently have I used catgut. I have been greatly impressed by the advantages of catgut. In my opinion, there is much less overall tissue reaction, both in the immediate postoperative period and two or three weeks later. Catgut also has the advantage of protracted self-removal, so that when there is tension about the stoma, there is no tendency to separation of the mucocutaneous edges with resultant granulation and tendency towards stenosis.

I would certainly agree wholeheartedly with Dr. Saunders, however, that whenever possible it is desirable to use no tracheal tube. I feel quite certain that irritation from the presence of a tube tends to promote stomal stenosis and should be avoided if possible. We now use a tracheal cannula for only two or three days and most patients are discharged from the hospital without a tube. We also find that the presence of the cannula and the need for its care and management are psychologically distasteful to most patients, and they are much happier when they can be discharged from the hospital without this additional psychological hindrance.

I would again like to congratulate Dr. Saunders on this timely compendium of laryngectomy techniques, and for bringing them to us in this forthright fashion.

DR. HERBERT H. HARRIS: Doctor LeJeune, I hesitate to come up to the podium and discuss this paper. First of all, I thought someone else would because of the

importance of this subject. I do, however, wish to take this opportunity to say a few words regarding this presentation.

I agree in principle with practically everything that Dr. Saunders has presented. I was never impressed with the value of closed drainage until recently when I started using it. The fact that I have not used it many times makes me somewhat doubtful as to its total value as compared with conventional drainage and pressure dressings. Perhaps I should make no definite statement regarding its usage, though I am sure it has some value.

I do wish to impress that it is absolutely necessary to close the raw edges of the mucosa and not suture the opposing epithelial surface of the mucosa. I believe Dr. Saunders stressed this thoroughly, however. A horizontal mattress lock stitch is used to close the mucous membrane and fascia in separate layers, and the muscle layer with interrupted sutures. If infection takes place, the catgut will be absorbed more easily than will a nonabsorbable suture. I doubt that we have had more than 10% fistulas. Perhaps this may be high—actually it is only a guess—but it does include a fairly large number of radiated cases.

Now, this may not be an exact picture, but I do not allow patients to eat within ten days following surgery. Having watched wounds heal, I have discovered that they are not very tight at six or seven days, but show very good union by the tenth day. If you have observed the skin at seven days you have noticed that the wound is easily opened; however, at ten days it is very difficult to separate. This has been my criteria for judging when to allow patients to eat.

I have never been impressed that a feeding tube did any harm. I am a little disturbed to find that Dr. Saunders is concerned about the tube producing ulceration, or in some way causing the wound to break open. I certainly believe that when the cricoid is in place that a long standing nasogastric tube will produce a post-cricoidal ulcer, but I doubt that it will in this instance.

Regarding the tracheotomy tube, there is some advantage in having one, although, perhaps, no great disadvantage in not having one. I do feel that when sutures are around the opening, perhaps some secretions may become dried and entangled in these sutures presenting a problem. Perhaps this is too conventional, but I still insist that a cone dressing about the tracheotomy tube holds the skin in good apposition to the tracheal mucosa and obliterates a dead space. The most important single item, I believe, in preventing strictures of the tracheostomy is absolute closure. Approximating the mucous membrane and the skin meticulously and not allowing small pieces of cartilage to remain between the edges are most important factors in obtaining a satisfactory stoma.

May I thank Dr. Saunders for this constructive presentation. Although I do not think I disagree with him on many points, I believe I shall continue to use the nasogastric feeding tube.

DR. SAUNDERS (closing): I just wish to thank Dr. Reed and Dr. Harris very much for their several helpful suggestions.

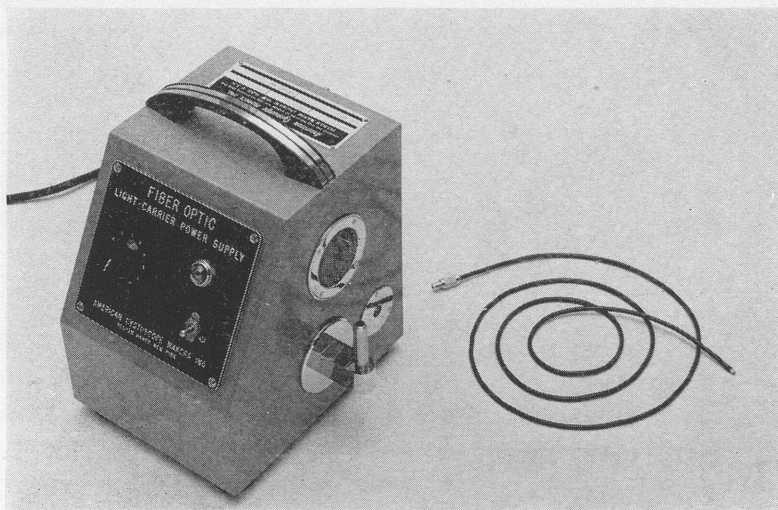
PRESENTATION OF INSTRUMENTS

Mr. President and Gentlemen: I have been interested in vision and illumination for some time. This development of the transmission of light, through small flexible glass fibers, seems to answer many questions. This principle of light transmission has been known for many years, and has been studied by Baird, Hansell, Van Heel and Hopkins. This advanced development has been done by the American Cystoscope Makers, Inc., of New York City. Light is transmitted down a bundle of flexible glass fibers from the light source to the tip of the instrument used—laryngoscope, nasopharyngoscope, telescope, head light, etc. The application of this principle is unending. The light is cold and can be directed in any angle. The illumination obtained is roughly 10 times the light of a grain of wheat electric bulb.

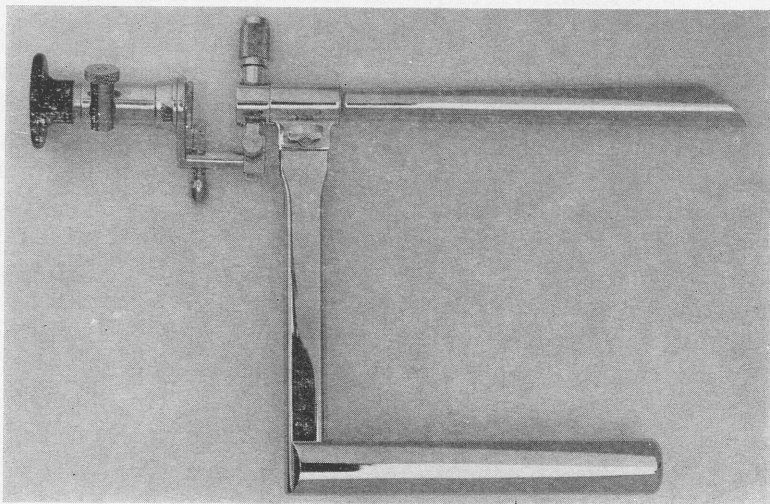
Here, Mr. President, is a laryngoscope, nasopharyngoscope, telescope, and a small bundle, that can be fitted to bronchoscopes and laryngoscopes.

EDWIN N. BROYLES, M.D.

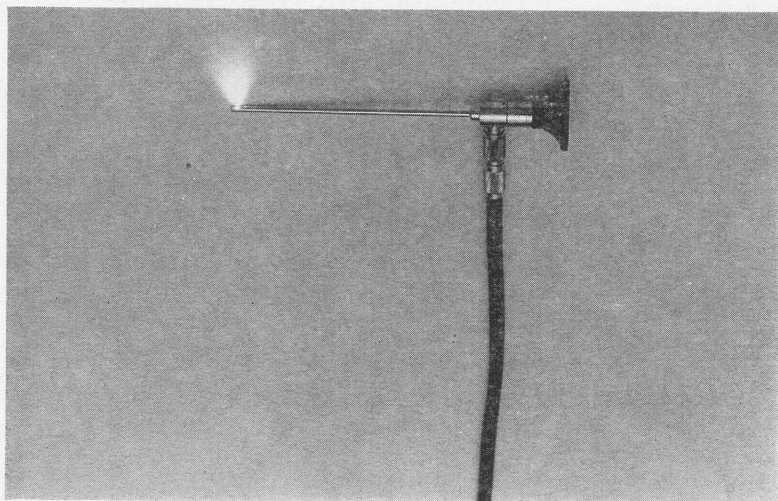
From the Department of Otolaryngology, Johns Hopkins Hospital.



LIGHT SOURCE



LARYNGOSCOPE WITH MAGNIFIER AND FIBER OPTIC LIGHTING



NASOPHARYNGOSCOPE WITH FIBER OPTIC ILLUMINATION

CANCER OF THE MAXILLARY SINUS

CLINICAL CLASSIFICATION AND MANAGEMENT

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(by invitation)

(by invitation)

SYRACUSE, N. Y.

The purpose of this paper is to analyze critically our experience in reference to present day methods of managing carcinoma of the sinuses.

Why Is Cancer of the Sinuses So Repeatedly Mismanaged? A paramount reason is the difficulty of diagnosis. In reviewing 59 cases of maxillary carcinoma we find the presenting symptom frequently misleading. Too often the patient has been seen by the dentist, the internist, the neurologist and even the psychiatrist for a bizarre chain of symptoms usually related to pain somewhere about the face. In the complete work-up negative x-ray findings of the sinuses give false security. Equivocal x-rays are hard to discern and are valueless unless followed by exploratory antrotomy. Early erosions of the posterior wall of the antrum are difficult to interpret even if laminagraphs have been taken.

How Can We Improve Our Methods for Earlier Diagnosis? If a patient is consistent in relating bizarre complaints to one or the other side, the otolaryngologist should be alerted to the possibility of malignancy in or around the paranasal sinuses. Diagnoses of late malignancy in or around the paranasal sinuses. Diagnoses of late malignancy can be made clinically by anterior and posterior rhinoscopy followed by confirmation biopsy. Usually, however, more difficult and subtler techniques must be employed for early diagnosis. In order of importance we suggest antral washings with Papanicolaou stains; sinus x-rays with Caldwell, Waters, basal and lateral views; and, occasionally, radiopaque study with water soluble contrast media. If symptoms persist and, particularly, if there is a past history of sinus trouble we advocate taking a look. We cannot agree with those who

believe an exploration of an antrum is a radical or extreme procedure. It can be reassuring if negative and life-saving if an early diagnosis is made.

The thoracic surgeon, the neurosurgeon, and the abdominal surgeon explore their particular areas when signs and symptoms justify a look. Perhaps the conservatism of the last decade with its hesitance to perform surgeries on the respiratory mucous membranes still prevails somewhat and serves to deter exploration even when indicated.

Malignant disease of the sinuses can be the result of a peculiar metaplasia associated with repair of epithelium destroyed by episodes of chronic sinusitis.^{1,2}

How Do We Classify Our Cases? Which Are Considered Early and Which Late? Recently we have attempted the classification of cancer of the antrum similar to the system of classification used by the Joint Committee on Cancer Staging and End Results Reporting for Cancer of the Larynx.³ Task forces have been adapting this system for many anatomic areas but, to date, have not been assigned the region of the maxillary sinuses. In this classification the important factor is that the T concerns the location of involvement in the antrum as well as the size of the lesion. In the TNM classification the pathology of the lesion is not taken into account.

In reviewing the literature, classification is missing in almost everyone of the discussions of cancer of the antrum. Öhngren, however, in addition to dividing the tumors according to various locations, also includes in his classification, references to three histological degrees of malignancy. The final criterion in his evaluation is the presence or absence of local or distant metastasis. He lists his topography groups in rising degrees of malignancy:

- 1) Tumors anterior, inferior and medial.
- 2) Tumors anterior, inferior and lateral.
- 3) Tumors superior, posterior and lateral.
- 4) Tumors superior, posterior and medial.

The question of pathology and grades of malignancy in the lesions arises. Certainly the histopathology is of great importance in estimating the lethal potential of a tumor. Perhaps, for a thorough evaluation of a series of cases, one may apply the TNM classification to the three histological groups suggested by Öhngren as follows:

- 1) Basal cell, adenomatous carcinomata (cystadenoma, endo-thelioma, etc.), myxomatous sarcoma, papillomatous carcinoma.
- 2) Squamous cell carcinoma, well differentiated, moderately differentiated; sarcoma other than above, except melanosarcoma.
- 3) Anaplastic squamous cell carcinoma; melanosarcoma.

Another Öhngren division, "a malignancy plane" runs from the inner canthus of the eye to the angle of the mandible, separating even further the benign from the malignant areas.

Using the above classification, cases may be placed on the following table:

TABLE I

Taken from Acta - Otolaryngology
Supp. 19, 1933

ÖHNGREN

	Topography	Histol. Mal.	Metastasis
Group I	a. Ant., inf. med.	M ₁ & M ₂	Mg.
	b. Ant., inf. med. Sup., post., lat.	M ₁	Mg.
Group II	a. Ant., inf., med.	M ₃	Mg.
	b. Ant., inf., lat. Sup., Post., lat.	M ₁ , M ₂ or M ₃	Mg.
Group IIIa.	Sup., Post., Med.	M ₁ , M ₂ or M ₃	Mg. or Pos.
	b. Ant., Inf., Med. Ant., Inf., Lat. Sup., Post., Lat.	M ₁ , M ₂ , or M ₃	Pos.

An application of the TNM system would show a more anatomic and specific detail than Öhngren's classification. It would also take into consideration the separation of distant and local cervical metastasis in grading the severity of the disease, and includes the potential severity of fixed cervical nodes in the clinical staging of cancer of the antrum.

We propose the following clinical staging system for cancer of the maxillary sinus as adapted from the TNM System developed by the Joint Committee on Cancer Staging.

T = Tumor of primary lesion and its extent.

N = Lymph nodes of the region and their condition.

M = Distant metastasis.

Within each letter element, increasing involvement is categorized by the combination of the capital letter with a numerical suffix.

As involvement of the host extends, the following designations appear:

T0 = No tumor clinically.

T1, T2, T3, T4 = Tumor location - ascending degrees of lethal potential.

Regional lymph node involvement is designated:

N0 = No involvement shown clinically or by diagnostic methods.

N1, N2, etc. = Ascending degrees of nodal involvement.

Distant metastasis is designated in a like manner:

M0 = None observable clinically or by diagnostic methods.

M1, etc. = Ascending degrees of metastatic potential of host.

The primary antral neoplasm is frequently gross and so it often is difficult to determine with certainty the primary site of growth. An attempt should be made to do this, however, since with this particular malignancy, the location of the disease is the most important factor in determining the treatment and the future prognosis.

With these facts in mind the following has been selected as the breakdown for T in our classification for cancer of the antrum.

T1, N0, M0

1. Invasion of the anterior wall or
2. Inferior naso-antral wall.
3. Anterior medial palate.

T2, N0, M0

1. Invasion of the lateral wall without muscle involvement.
2. Invasion of the superior wall without orbital involvement.

T3, N0, M0

1. Pterygoid muscle invasion.
2. Orbital invasion.
3. Invasion of anterior ethmoid cells without involvement of the cribriform plate.
4. Invasion of anterior wall of antrum with skin involvement.

T4, N0, M0

1. Invasion of cribriform plate.
2. Invasion of pterygomaxillary fossa.

3. Extension to nasal fossa or other antrum.
4. Invasion of pterygoid plate.
5. Invasion of posterior ethmoid cells.
6. Extension to ethmo-sphenoid recess or sphenoid sinus.

The lymphatic metastases are represented as N0, N1, and N2. There is no breakdown as to bilateral metastasis of cervical nodes but essentially whether or not nodes were palpable, present but not fixed, or present and fixed.

T4,N1,0 and/or T4,N2,0

N0	= No clinically palpable cervical lymph node(s) (metastasis not suspected)
N1	= Clinically palpable lymph node(s) that are not fixed (metastasis suspected)
N2	= Clinically palpable lymph node(s) that are fixed
M0	= No distant metastasis
M1	= Clinical and/or radiographic evidence of metastasis except to the cervical lymph node regions.

From these categories of T N and M a similar administration of staging for cancer of the antrum is suggested, again following the method of the Joint Committee of Clinical Classification and Staging of the Larynx. This system appears logical and reasonable in evaluating the proper treatment and prognosis for this disease. It also should be of tremendous value in that an accepted basis for reporting cure rates of cancer of the antrum can be established. In this regard the evaluation of reported cases and techniques would then be more meaningful in the overall control and cure of the disease.

STAGE I

T1,N0,M0

Tumor invasion of the anterior wall without skin involvement (T1); or
 Tumor of inferior naso-antral wall without skin involvement (T1); or
 Tumor of anterior medial palate (T1).
 No clinically palpable cervical lymph node(s), metastasis to nodes not suspected (N0).
 No distant metastasis (M0).

STAGE II

T2,N0,M0

Tumor invasion of the lateral wall without muscle muscle involvement (T2); or
 Tumor invasion of the superior wall without orbital involvement (T2).

No clinically palpable cervical lymph node(s), metastasis to nodes not suspected (N0).

No distant metastasis (M0).

T3,N0,M0

Tumor invasion of anterior ethmoid cells without involvement of the cribriform plate (T3).

Tumor invasion of pterygoid muscle/with or without combination of T2 (T3); or

Tumor invasion of orbit in combination with T2 (T3); or

Tumor invasion of anterior wall of antrum with skin involvement (T3).

No clinically palpable cervical lymph node(s); metastasis to nodes not suspected (N0).

No distant metastasis (M0).

T3,N1,M0

Primary tumor as in T3 (Stage II).

Clinically palpable cervical lymph node(s) that are not fixed; metastasis to node(s) suspected (N1).

T4,N0,M0

Tumor invasion of posterior ethmoid cells/with or without T3 (T4); or

Tumor invasion of cribriform plate (T4); or

Tumor invasion of pterygoid maxillary recess (T4); or

Tumor extension to other antrum (T4); or

Tumor extension to ethmo-sphenoid recess or sphenoid sinus (T4); or

Tumor invasion of pterygoid plate (T4).

No clinically palpable cervical lymph node(s), metastasis to nodes not suspected (N0).

No distant metastasis (M0).

T4,N1,M0; and T1,T2,T3; or T4,N2,M0

Primary tumors T1, T2, T3 or T4 as described in Stages I, II and III.

Clinically palpable cervical lymph node(s) that are fixed. Metastasis to node(s) suspected (N2).

No clinical or radiographic evidence of distant metastasis (M0).

T1,T2,T3 or T4,M1

Primary tumor T1, T2, T3 or T4 as described in any preceding stage, associated with

Clinical or radiographic evidence of distant metastasis (M1).

How Do We Determine Which Modality to Use as the First or Prime Method of Treatment? There can be no standard formula applied when choosing a modality until therapeutic methods of treat-

ment for cancer of the antrum improve and become more predictable. At present we use a scientific equation but this must be balanced against a human equation. With increased experience and improved methods one does, however, alter his policy from time to time.⁵ The following is our present concept of therapy:

For *Stage I* cases we insist on surgery as the prime treatment. We had two cases; both were treated by surgery and both are alive. In *Stage II* cases a radical maxillectomy alone or combined with exenteration of the orbit has been our therapy of choice, although recently we have advocated radio-Co-60 pre-operatively for Stage II, followed by definitive surgery two to three months post-radiation. We have reason to believe fewer orbital exenterations may be mandatory by following this order. In the past, surgical failures in Stage II received radiation by one of two methods (usually by concentrated supervoltage to recurrent areas and occasionally by intracavitary radium). Our survival rate for Stage II cases has been 75% (3 out of 4 cases are alive).

Stage III cases begin to present grave problems because exenteration of the orbit is mandatory in all cases (except in T1,N1,M0 and T2,N1,M0) if surgery is the prime treatment. In our experience all these cases have required postoperative radiation, so if there is any doubt as to the patient's psychological response to surgery it may still be wiser to precede the surgery with a full course of supervoltage radiation. We agree with Hilger⁶ that irradiation first in these cases might lessen the metastatic potential and certainly will not complicate total surgery at a later date. Also, in certain cases, irradiation will reduce the amount of surgery necessary by circumscribing and reducing the tumor mass.

We agree with Schall⁷ that intracavitary radium does have its place in the treatment of antral cancer. The problem of treating by radium alone results from the rapid fall in dose with the distance from the radium source; the dose falling as the square of the distance. This modality can, of course, be used only for tumors of limited extent. Since it is difficult to be certain that even a small tumor is so limited, intracavitary radium is never used as a prime method of treatment. Our survival rate for Stage III cases is 44% (4 out of 9 cases are alive).

Our overall survival rate for Stage IV cases is 9% (4 out of 44 cases are alive). For this reason we obtain our biopsy confirmation

with as little surgical trauma as possible. We treat the patient with supervoltage x-ray (cobalt 60) and, depending upon the clinical response, follow the x-ray with maximum surgery or subtotal surgery combined with more cobalt or intracavitary radium, or both.

What Surgical Procedures Do We Offer These Patients? Our operative procedures can be classified as either maximum resections or subtotal resections. Maximum resections mean the exenteration of the orbit in continuity with a radical maxillectomy. We use the standard operative procedure en bloc maxillectomy combined with exenteration of the orbit as advocated first by Watson,⁸ and Martin,⁹ and later by Frazell,¹⁰ Conte¹¹ and Tabb.¹² We do not compromise on our bony incisions but do remove the entire zygomatic arch and ascending ramus of the mandible if the disease is deep in the pterygo-maxillary fossa or is deeply invading the pterygoid and temporalis muscles. Anything less than this we consider subtotal surgery which includes the radical maxillectomy and the partial removal of the maxillary bones.

COMMENT

Since 1939 the management of maxillary carcinoma at the Upstate Medical Center at Syracuse, New York, has undergone wide changes. From 1939 to 1951 all cases were treated only with radiotherapy. There were no survivals in this group. Since 1951 most cases have been treated with combined modalities. The youngest patient was 29 years old, and the oldest was 87. Fifty-two of the 59 cases showed evidence of x-ray changes. Of these, 37 showed bone destruction. Two cases were classified as Stage I; 4 as Stage II; 9 as Stage III, and 44 as Stage IV. *Twenty-nine* cases were treated with combined modalities, eight were treated with radical surgery alone and twenty-one were treated entirely with irradiation. One patient received no treatment after biopsy. Thirty patients died under two years after treatment was initiated. Of those deceased, 7 died from other causes and were without recurrent local disease, and four died from remote metastases which dispels the theory that antral lesions do not metastasize distantly. For 25 patients life was extended over one year and they became well adjusted. Nine patients had life prolonged over one year but were unable to adjust to their disabilities. Perhaps this last group should not have had treatment. Eleven patients had life extended less than one year but they had made a satisfactory adjustment.

A gross 34% of our cases are in the five-year cure group. This figure includes the seven patients who died of other causes without showing local recurrence at autopsy. It does not include as cured those living but lost to follow-up. Since we have used combined modalities of treatment a net 22% of our patients are alive over five years without recurrence. In a selected group of 32 cases, where the prescribed combined treatment was considered controlling and adequate, we had a gross 47% and a net 28% cure rate for over five years. (Nine cases over five years, two of these over seven years.)

Although this series is a relatively small one, we believe it is representative and significant in evaluating the improvement in modern methods. Our observations are comparable with other series reported in the literature and, no doubt, these percentages will be maintained until there are further improvements in the modalities for treatment.

SUMMARY

An analysis of present-day methods for treating carcinoma of the paranasal sinuses has been presented. A plea is made for a high index of suspicion of neoplasm of the sinuses in light of common signs and symptoms about the head. A stronger plea is made for exploratory surgery in suspicious cases.

The individuality of the clinical aspects of each case must be conscientiously weighed. We believe the patient will be afforded the greatest opportunity for cure with the combined efforts of the radiotherapist and the surgeon.

The surgeon must understand as completely as possible the psychologic architecture of the patient who must consider radical surgery. We must consider pain as a factor and select methods that will relieve pain. We must often use the modality in Stage IV cases which offers the most reasonable outlook in palliation for the individual. The patient who has achieved added years of life but is in constant pain, is unable to speak intelligibly or is unable to swallow, is a failure. If these situations can be anticipated, it is better to use conservative methods of treatment. The patient will live fewer weeks but his last days will be happier. Review of the literature and our own cases convinces us that but a few patients with Stage IV maxillary sinus carcinoma will be cured, but it is in our power to prolong life in most of these cases. The decision as to which case to prolong is difficult to make and is a tremendous responsibility. The decision as

to which modality of treatment to use to prolong these cases is even more difficult to reach and should be made only after careful consideration of the many factors discussed above.

An attempt has been made to classify cancer of the maxillary sinus according to the degrees of severity. We have proposed an adaptation of the TNM system as developed by the Joint Committee on Cancer Staging. It is our hope that this or a similar system for classifying lesions of the maxillary sinuses will be universally accepted among otolaryngologists so that the proper evaluation of reported cases and their treatment will be more meaningful.

UPSTATE MEDICAL CENTER

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DISCUSSION

DR. HAROLD G. TABB: Doctor Sisson has today given us a frank evaluation of his experience in the management of carcinoma of the maxillary sinus with a proposal for classifying these lesions so that otolaryngologists will have a common ground in discussing their results.

There is little that I can disagree with in this presentation, so would like to emphasize several points.

As brought out by the author, patients with carcinoma of the maxillary sinus are seldom seen early. We have found a delay of three to six months in our cases between initial symptoms and the beginning of definitive treatment. With this delay, we are forced, in many instances, to manage a more advanced carcinoma with its poorer prognosis.

It is evident from the literature and substantiated by the author that earlier diagnosis appears to be our only key in increasing the salvage rates for cancer of the maxillary sinus, but the disease is so insidious that it often defies early diagnosis. Chronic sinusitis, polyps, and nasal obstructions all interfere with local ventilation and drainage of the nose and create what might be termed a climate for malignancy. Because of the frequency with which antral densities prove malignant, and because of the importance of early diagnosis, it is wise to explore all suspicious densities in the sinus. I agree with the author wholeheartedly in his philosophy that looking into an antrum whenever an unexplained, persistent density is present is a wise plan.

The oldest surgically cured cancer of the maxillary sinus in our series is one in which the diagnosis was made quite accidentally. After removal of a severe nasal septal deformity, a carcinoma was found on the lateral wall of the nose extending into the medial wall of the antrum. A maxillectomy was performed and the man is alive and free of disease now, eleven years after surgery.

I compliment Dr. Sisson for proposing a logical method of classification for malignant diseases of the maxillary sinus along the lines similar to that as developed by the Joint Committee of Cancer Staging for laryngeal carcinoma. This appears to be a realistic method of nomenclature and I hope that it will be favorably received.

As brought out here today, there are still only two methods of therapy at hand which are capable of controlling cancer of the maxillary sinus: namely, surgery and x-ray. Chemotherapy, although quite promising, does little more at this time than enhance the effects of irradiation.

Surgery today still offers the best chance of permanent cure in early cases of antral cancer. In looking back over our series of almost ninety cases, I find that radical surgery has given us the largest percentage of successful results and was most successful in neoplasms confined to the floor, anterior or medial antral walls. These are properly designated as early or stage I lesions by Dr. Sisson.

Over the past three years, I also have come to realize that super-voltage irradiation has a definite complimentary place in the treatment of antral carcinoma (we also prefer it), prior to surgery. Large Lesions which, in the past, would have required resection of the maxilla in combination with the orbit can now be removed in many cases without jeopardy after pre-operative irradiation and with preservation of the orbit.

My reasons for using pre-operative irradiation are the same as those advanced by Goldman in the treatment of laryngopharyngeal cancer, namely, that pre-operative irradiation destroys the early actively dividing cells and inhibits mitosis in the mature cells which may not be killed by x-ray. Since the young fragile cancer cells are usually at the periphery of a tumor, prior irradiation provides a more cancer-sterile field in which to surgically extirpate the cancer focus.

I wish to compliment Dr. Sisson for bringing us his latest methods of managing a most distressing disease.

DR. IRA A. POLISAR: Doctors Sisson and Johnson are to be congratulated on this paper and Dr. Tabb on his discussion.

I have just a few thoughts and I thought perhaps Dr. Sisson might help me with one question.

I didn't hear any histologic classifications in this group. I think, from my own small experience, that you are probably likely to run into a wider distribution of histologic types here than you are in carcinoma of the larynx. I mention this because in certain groups of tumors which may arise here sarcoma perhaps (except for easily recognizable chondro- or osteo-sarcomas which can be considered solitary) and more particularly the plasma cell tumors might, and perhaps should, be considered as possibly of multicentric origin. I wondered how they felt about doing the big operation in this type of case even if there is no clinical evidence of distant disease.

I would like to relate this thought, for example, to the seven per cent which I think Dr. Sisson mentioned as showing metastasis and wonder how many of that seven per cent, other than epidermoid carcinoma and also excluding those which had broken out into the soft tissues, were of histologic types such as plasma cell or sarcoma.

DR. FRANCIS L. WEILLE: Mr. Chairman, Fellows and Guests: I wish to tell you of two cases that I have had experience with that impressed me aside from the hopelessness of this illness.

The first was a carcinoma of the naso-antral wall that I excised about eleven years ago with a margin of safety around the tumor. The patient had no irradiation and is perfectly normal with no deformity. The operation was done through a Moure procedure. All she has now is an atrophic rhinitis on the operative side.

The second case is just the opposite. A man came in with his antrum and ethmoid full of myxosarcoma with involvement of his skin and the hard palate. There was extension to but not through the septum. I thought he was hopelessly ill and I told his insurance company this was the case.

I did a palliative Moure for him, merely for what Dr. Schall called "drainage" at the time, and gave him up as incurable. I did use electro-coagulation of the recurrent tumor on the floor of the nose on that side, but I thought he was just hopelessly ill and the less one did to him the better. His surgeon and family doctor called me one day and asked if I would take out the upper jaw after the technique of Dr. Tabb, which I reluctantly did. Now, this man is six years postoperative and without tumor. Certainly myxosarcoma is not supposed to be radio-sensitive, according to Dr. Schultz, of the M.G.H. Tumor Clinic X-ray Department, but the man did have irradiation in addition to his two operations and now is well.

DR. JOHN S. LEWIS: First of all I would like to thank Dr. Sisson and Dr. Johnson for this very excellent presentation.

Recently I have reviewed 416 cases of cancer of the paranasal sinuses, primarily adenocarcinoma and epidermoid carcinoma, which comprise about 90 to 95 per cent of all the tumors found in the area. It was interesting in going over the material that one could not accurately describe (from this classification Dr. Sisson presented) the exact anatomical location or extent of the tumor unless one did a surgical extirpation.

It was our experience that 40 per cent of tumors of the antrum showed no evidence of bone destruction at the time of diagnosis. The other cases did show clouding on x-ray, with some suggestion of a polyp or some other pathology present.

And I must emphasize that you cannot adequately classify the location of a tumor in the nasal cavity or paranasal sinuses without surgical extirpation.

As far as pre-operative radiation is concerned, we had about 60 cases in that group and it is interesting that our over-all cure rate was much higher by just primary surgery alone than with those that were irradiated with pre- or post-operatively. The cure rate by radiation alone as a primary method of treatment was 14 per cent and that by surgery exceeded 35 per cent and I think this is of considerable importance.

Incidentally the cervical metastasis in this large group was about 25 per cent, which is much higher than one is led to believe in looking over the various case reports in the literature.

DR. JOHNSON (closing): Mr. Chairman, Members and Guests of the American Laryngological Association: To recapitulate a few of our points:

Neoplasms of the maxillary sinus are more lethal, we believe, in their location rather than by their size alone. When the primary location is bad the prognosis is also bad.

The majority of our cases—44 out of 59 cases—were in Stage IV and the results were approximately 10 per cent, one case out of ten being salvaged. This is the disease we are most generally dealing with and when we speak of cure rates for carcinoma of the maxillary sinus, most of our cases are going to fall into this category, with a very unfavorable prognosis.

In the future, if we are to critically evaluate reported series of this disease, it would be well to know how many cases fall into Stage IV, or any other similar classifications, and if the results are improved, what techniques were employed to attain this, for it is the Stage IV cases where we must improve.

From our review of these cases, we feel we are in a better position to evaluate therapy for patients in the various stages and we certainly feel that, since our results for Stage IV, where most of our cases are classified, are so poor, that more heroic procedures, perhaps in combination with neurosurgery, should be attempted.

Conversely, if the prognosis is only 9 or 10 per cent in an unstable personality, then we can consider conservative palliative measures.

The consideration of pathology that Dr. Polisar brought up is very important and generally this has not been critically evaluated in the past literature. For an example, one essayist included a case of angiofibroma in his series of cases. Öhngren did include pathology in his classification and in this way more critically evaluated his work. He divided his histological classification into the three following divisions:

One, the basal cell and the adenomatous carcinomas (such as cystadenoma, endothelioma, and so forth), the myxomatous sarcomas, as Dr. Weille mentioned, and papillomatous carcinomas; so therefore Dr. Weille's case of myxomatous sarcoma would fall in one of the most favorable pathological categories, according to Öhngren's classification.

The second division was mainly moderately well differentiated squamous carcinomas and sarcomas other than the ones in the first division.

The third division was anaplastic squamous cell carcinomas and melanomas.

It would seem obvious that we should critically evaluate the pathology. For an example, certainly a cylindroma and an anaplastic squamous cell carcinoma of the antrum cannot be considered to be one and the same disease, even though they are in the same location.

We have heard repeatedly, and only as recently as yesterday from Dr. Lederer, of the importance of the pathologist understanding the nature of neoplasms of the larynx. This is equally important in treatment of carcinoma of the antrum. We therefore hope that if we can work out an accepted classification of the sinuses, we can also include the pathology so that we can better understand where we are going and what we are doing.

We wish to thank Dr. Tabb and Dr. Lewis for their comments, and also Dr. Polisar and Dr. Weille for their discussions.

FUNCTION IN THE CRIPPLED LARYNGOPHARYNX

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(by invitation)

NEW YORK, N. Y.

The combined and common usage of the oral and pharyngeal cavities for the purposes of handling food and the passage of air give this functional region of the head and neck special significance. This double functional responsibility is balanced in such a way that the demands of one system alternate with those of the other system, permitting each to use this conduit with rare communal efficiency. It is a perversity in development that the separation of this common system should place the more dominant air passage in the ventral position with the entrance to the gullet directly over it. When the anatomy and capacity of one system is seriously altered, crippling of the other will result. The significance of this phenomenon is accentuated by the adaptations required of the larynx following extensive extirpations of the gullet.

One of the purposes of the larynx is to protect the lower air passages by sphincteric closure of the supraglottic structures. This basic protection is enhanced by the forward movement of the larynx, increase in the antero-posterior diameter of the hypopharynx as the bolus passes through a relaxed cricopharyngeus muscle. The glottis is then opened with an ascending column of air after the bolus has passed.

From the Departments of Otolaryngology and Radiology of the College of Physicians and Surgeons, Columbia University, and the Otolaryngological and Radiological Services of the Presbyterian Hospital in the City of New York.

Crippling of the oral and pharyngeal cavities alters the combined functions of swallowing and breathing in a variety of ways.

These deficiencies are usually compensated by the adaptation of the residual function to meet the physiological demands. Rarely did the elimination of one specific organ or function in the gullet irrevocably alter the basic capacity of these two systems to continue to function as a unit. A practical working principle is that 50 per cent of any part of the functional system may be extirpated without the fear of serious impediment. When the resection includes more than 50 per cent of one system or includes 50 per cent of several regions or systems, a definite physiological handicap is created. This deficiency interferes with the formation and transportation of the bolus in the presence of the functional glottis. These were clinically manifest by serious alteration in eating habits. A group of thirty patients with severely altered swallowing mechanisms were studied over the past three years, using cineradiographic methods with the hope of acquiring better understanding of these adaptive phenomena.

The methodology of the test permitted the patient to swallow a bolus according to his habit and capacity. It was felt that this allowed a more valid estimation of his functional status than a rigid standardization of the size, consistency, rhythm and quality of the bolus. It was therefore possible to study the various techniques and mechanisms that each individual employed in order to overcome his handicap. Certain patients could form and handle the bolus according to instruction, and when this was possible, this was added to the study. Other patients had great difficulty in handling the bolus under any circumstance and in these, the study was limited or not done.

ETIOLOGY

Alterations in the swallowing and breathing mechanism could be divided into five different categories:

1. Partial or total glossectomy, associated with partial mandibulectomy and neck dissection.
2. Partial or total pharyngectomy with neck dissection.
3. Certain nerve paralyses were found to be important. Combinations of paralyses of the vagus nerve superior to the pharyngeal nerves and hypoglossal nerve greatly increased the functional disability of this region.

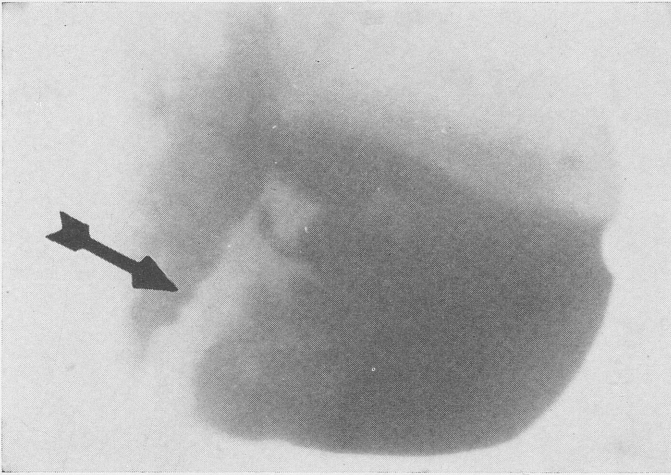


Fig. 1.—Frame from a cine-radiographic swallowing study of F.R., a 75 year old male, after resection of the horizontal ramus of mandible and right half of the tongue and floor of mouth. Reconstruction of the oral cavity including suturing of residual tongue onto floor of mouth. The vagus was not cut. Because of relative immobility of the tongue during swallowing there was failure of complete occlusion of oro-pharynx by coaptation of tongue and posterior pharyngeal wall despite compensatory increased activity of pharyngeal musculature. The latter is manifest in the illustration by the serrated contour of the posterior pharyngeal wall (arrow). Note failure of epiglottis to rotate inferiorly and cover the laryngeal vestibule. Small amount of barium did escape into the larynx and trachea despite apparent complete closure of laryngeal sphincter.

4. Scar tissue contraction, when it caused distortion, interfered with the opening of the sphincter or closure of the glottis.

5. The psychological factors associated with failure of function in this region were felt to be significant in many instances. The basic fear of the disease which necessitated such a massive and crippling operation was compounded by the anxiety, confusion, embarrassment and dependency associated with the rehabilitative phase. Coughing and choking were constant threats. The inevitable struggle of the instinct of hunger against the frustrations of not being able to swallow properly and the disassociation of the breathing and swallowing rhythms added to the management problem. Although the hunger phenomenon could be controlled by feedings through an oral-gastric tube or a gastrostomy, this did not prove to be a satisfactory



Fig. 2.—Partial paralysis of the right pharynx following section of the right hypoglossal, and superior laryngeal nerve and trauma to the right vagus. The muscle tone of the normal left pharynx seems to throw the barium bolus to the flaccid right pharynx so that most of the bolus runs down the right laryngo-pharyngeal gutter.

substitute for the swallowing mechanism. The absolute necessity of airway dependency upon tracheostomy did not enhance the swallowing technique even though it facilitated pulmonary toilet.

THE PROBLEM

The preservation of a functional glottis in the presence of a crippled gullet depended upon the residual capacity of five basic functions relative to the gullet and one essential function relative to the larynx: 1) elevation and posterior displacement of the tongue against the palate and pharynx; 2) elevation and anterior movement of the larynx; 3) upward movement of the hyoid bone; its "dumping rotation" on its axis; 4) backward and downward motion of the epiglottis; 5) contraction and stripping action of the pharynx. All contributed to the facility of the swallowing act. None of these functions alone was absolutely essential for swallowing and most

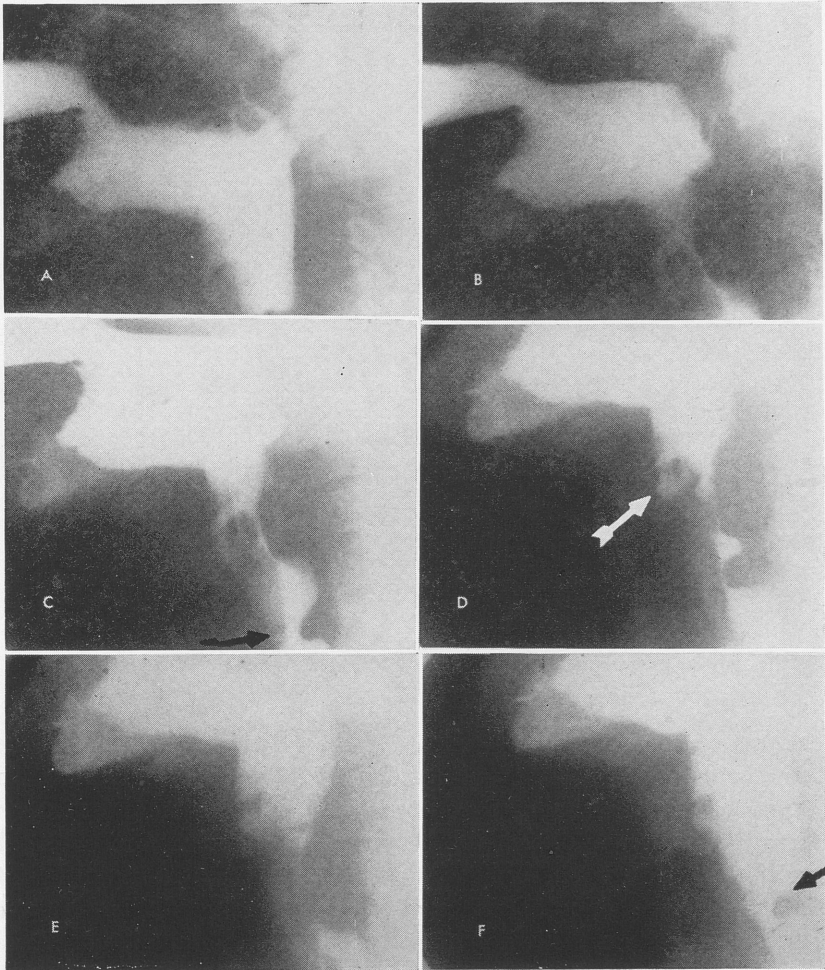


Fig. 3.—Cine-radiographic swallowing studies 6 years after a total glossectomy and left radical neck dissection.

A. Absences of tongue permits barium to descend to level of inferior boarder of mandible. Postpharyngeal musculature is relaxed.

B. Note marked forward movement of posterior pharyngeal musculature probably in compensation for the absence of the tongue.

C. Wave of pharyngeal constriction progresses inferiorly and approaches an incompletely relaxed cricopharyngeus muscle.

D. Pharyngeal contraction wave moves closer to the cricopharyngeus. Note barium in the laryngeal vestibule (white arrow) but none in the glottis or trachea.

E. Pharyngeal constriction wave merges with cricopharyngeus.

F. Relaxation of pharynx but incomplete relaxation of cricopharyngeus (black arrow).

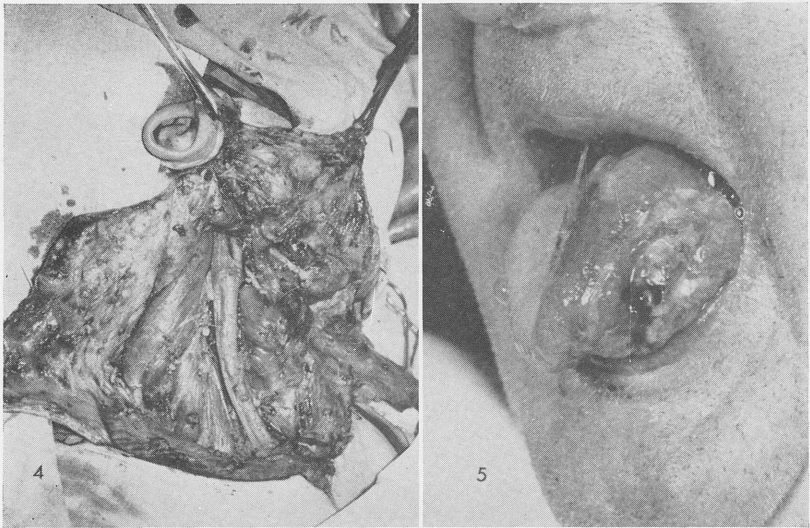


Fig. 4.—No significant crippling from thyroidectomy and neck dissection.

Fig. 5.—Cancer of the tongue requiring composite resection of half of the tongue, mandible and neck resulting in moderate crippling of oral cavity.

patients could adjust successfully to the loss of half of them. If the loss of any of these functions was added to a mutilated or paralyzed larynx prohibiting adequate closure in protection of the glottis, then a grave situation resulted. Obviously, resecting a portion or all of the tongue and the floor of the mouth interferes with movement of the tongue. Resection of a portion of the mandible and its muscular attachments interferes with the elevation of the hyoid bone and thyroid cartilage. Resection of the suprahyoid muscles creates the same deficiency. When the hyoid bone is added to the excised specimen, the deficiency is further exaggerated.

None of the above alterations interfere with the action of the constrictors of the pharynx. Resection of a portion of these constrictor muscles does not establish as serious an impediment as a paralysis of the pharyngeal constrictors. Repair of the pharyngeal musculature after the resection of the localized cancer partially re-establishes the contractile activity of this region. In contrast, this action is seriously altered when the area is atonic as a result of a local hemiparalysis. With only half of the pharynx contracting against the flaccid muscu-

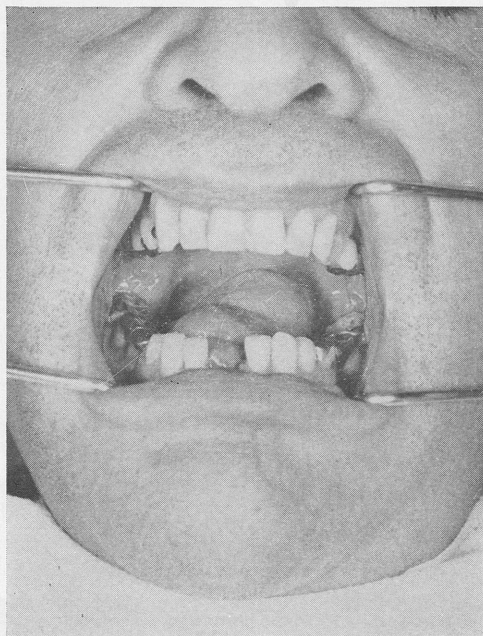


Fig. 6.—Crippled oral cavity following partial glossectomy and neck dissection. The rudiment of tongue is restricted in the mouth because of fixation to mandible.

lature on the paralyzed side, there is a shift of movement to the inert side permitting partial loss of control of the bolus. If anesthesia and partial paralysis of the larynx are added to this situation, the swallowing handicap is so significant that nasogastric and tracheostomy tubes frequently have to be maintained in position for several months until a functional adaptation is accomplished by the residual regions.

The problem resolves itself around the interference in creating, shaping, handling, transport, and control actions of the muscles of the pharynx. Gaining control of the bolus in areas that are deficient, distorted, limited in action, or nonfunctional has a great chance of success by depending upon the instinctual mechanism on the part of the patient rather than intellectual analysis of the swallowing act. If stimulation of the basic swallowing reflex in the gullet can be attained, the other minor deficiencies associated with puddling and spillage can be overcome by practice and special exhaling techniques.

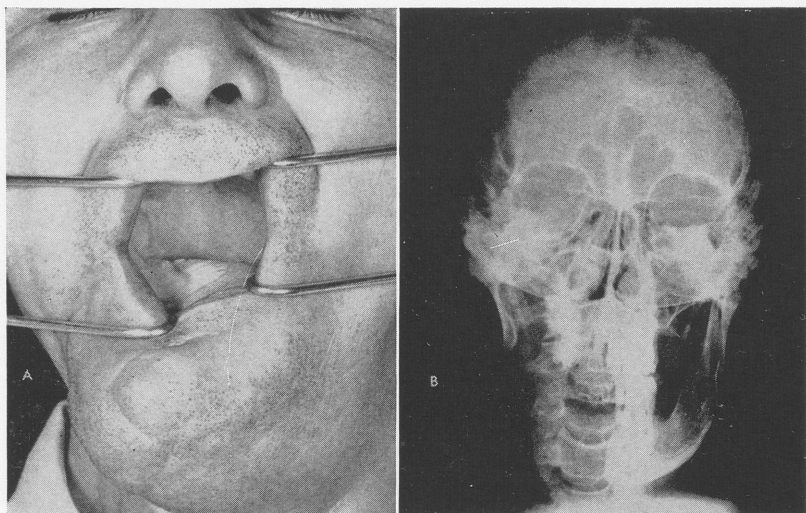


Fig. 7.—A. Crippled oral cavity with residuum of tongue fixed to cheek and palate giving some flexibility but limiting function. B. Shows mandibular deficiency following composite resection. Attachment of a mobile tongue to the cheek affords better function than fixing it to the mandible.

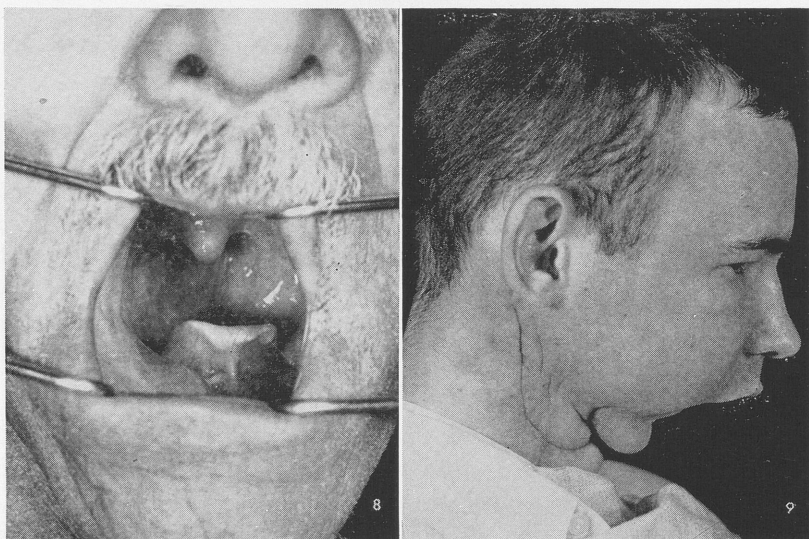


Fig. 8.—Crippled oral cavity following total glossectomy and neck dissection. Patient takes liquid diet by mouth and has a fair voice. Larynx is intact.

Fig. 9.—Severely crippled oral cavity from loss of mandible, tongue and bilateral neck dissection. No oral function save as a receptacle. Persistent aspiration was a problem.

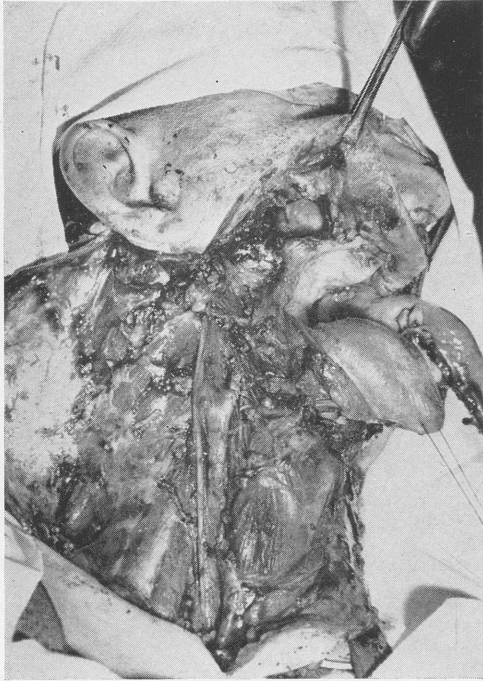


Fig. 10.—Resection of palate, maxilla, tonsil, mandible and neck resulting in only moderate crippling of oral cavity because tongue is active.

The exhaling techniques consist of a small cough at the end of the swallowing act in order to clear the supraglottic area of any residuum. Further clearing is obtained by repetitive swallowing and stripping maneuvers. Simulation of the basic swallowing act is accomplished by regional function modification in the residual tissues of the wound. This includes hyperactivity of any of the muscles that can contribute to the fundamental maneuvers of the tongue, glottis, and larynx that are required in the normal swallowing act. The residual muscles in the oral cavity, palate, and those remaining attached to the hyoid bone or in the suprahyoid area accept increased responsibility. The mylohyoid muscle complex is one of the most important single groups as demonstrated by the prolonged aspiration associated with the resection of the antero-bilateral mandible, floor of the mouth, suprahyoid structures, and neck dissection. This complex, however, is not exclusively essential. Hyperactivity occurred particularly in the pharyngeal

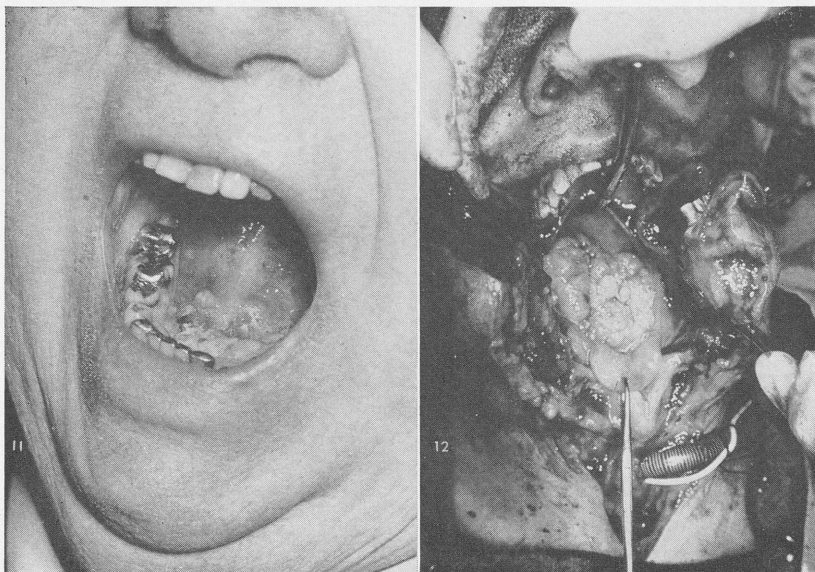


Fig. 11.—Cancer of floor of mouth and mandible with metastases to submental area. Resection of these structures with mylohyoid complex causes severe crippling.

Fig. 12.—Cancer of pharynx compromising the larynx, repaired by free skin graft tubing results in inert gullet.

musculature. This phenomenon was unusually exhibited in an individual who had undergone a total glossectomy and neck dissection and who had excellent control of the bolus without spillage or puddling. The constrictors of the pharynx moved forward to the site which would ordinarily be the base of the tongue completely closing the pharynx and then moved back to accept the bolus.

Perhaps the best technique in rehabilitating the swallowing mechanism is the use of "gulping." "Gulping" satisfied all of the instinctual requirements for swallowing, setting up a rhythmical movement under control of the medulla. It is devoid of the refinements of specialized control and contains a self-perpetuating mechanism simulating normal swallowing with less chance of error than any preconceived technique. Many patients were reluctant to even attempt to gulp down a glass of water because of the fear of failure, aspiration, coughing, and choking. One should be prepared to aspir-

ate the trachea under these circumstances, but if the patient accomplishes this maneuver on the first try, he has a considerable advantage in his break-through toward rehabilitating the swallowing mechanism. Additional aids which may provide some benefit to the patient are moving the bolus to the most functional side of the throat in preparation for swallowing, leaning to one side or the other and employing the effects of gravity to its maximum degree. In the end, however, the patient must master this problem according to his own determinations. This requires practice in swallowing his own saliva and then the daily passing of an oral-gastric feeding tube until the tracheal secretions have disappeared. It is beneficial for the patient to practice in front of a mirror, observing the fact that the thyroid cartilage moves upward upon each swallowing act; otherwise the protection of the glottis is curtailed.

CONCLUSIONS

1. The constrictural rhythmical act of swallowing embraces an architecture of groups of muscles, nerves, cartilages, and bone, each contributing a refinement in the physiologic significance of swallowing.
2. The relation of this phenomenon to the passage of air is a unique combination of systems in this region.
3. There is no single structure or organ that is absolutely essential for the swallowing act.
4. The swallowing act can survive extirpation of as much as 50 per cent of its constituents.
5. Serious crippling of swallowing is caused by extirpation of a group of functional components with a resultant combined deficiency in structure and in capability often associated with distortion and paralyzes. It is not necessarily the amount or quantity of tissue that is extirpated as much as the combination of actual impediments which are inflicted upon the functional capability of the region.
6. Rehabilitation consists of simulation of swallowing on the instinctual level consisting of hyperactivity, hypertrophy, and functional modification of the residual muscles and tissues in the wound.

DISCUSSION

DR. JOSEPH H. OGURA: Mr. Chairman, Members and Guests: I would like to again compliment Dr. Conley for this very timely subject because I think too much emphasis has been given to radical surgery of the neck and not enough to the functions and to their rehabilitation problems.

This presentation is of particular importance because of the methods used. An analysis of function of cineradiographic means can be done by slow motion and stop-down methods. Other methods can be employed; namely, electromyographic techniques, which in the future I believe will be the method used to complement the study of cinefluoroscopic examination.

We have used the cineradiographic methods in over fifty cases and we have learned a lot about the functional aspects of the various surgical procedures employed.

I would agree that the anterior and posterior displacement of the tongue to close off the hypopharynx in initiating the bolus is important. I am certain the pharyngeal constriction is of prime importance in carrying out the second phase because when you have paralysis, grave difficulties arise in the swallowing mechanism.

Preservation of glottic function is important, yet we can modify the glottis extremely, to the point of removal of 50 per cent or even more of the larynx with good swallowing function.

We have made electromyographic measurements of the mylohyoid muscle, pharynx, cords and cricopharyngeal muscles. There seems to be an orderly sequential firing of the muscle groups with a delay of 5 to 10 milliseconds in an orderly manner.

This work has been patterned after Bosma. At the present time we can infer that with certain segmental areas of pharyngeal paralysis, alteration of pharyngeal deglutition is noted.

I think it is important to stress some of the things Dr. Conley has stated. With resection of the jaw, if you preserve one-half of the tongue and one anastomosis the remaining tongue to the cheek, reasonable swallowing is maintained. However, when you resect the anterior jaw, swallowing is crippled. Drooling and the difficulty in handling secretions become an extremely serious problem.

What are some of the other factors that apparently are important and support what Dr. Conley has stated? As we descend into the pharynx and laryngopharynx, I can state from my experience with 97 consecutive conservation operations—in only two cases have we had difficulty; one which had a persistent postcricoid ulcer, and the second instance which followed conservation surgery after heavy irradiation. In the latter case I think fibrosis was the important factor here in preventing good swallowing.

One can state that sacrifice of the epiglottis, the false cords, the aryepiglottic fold and one arytenoid is consistent with good deglutition.

One important thing which was not mentioned is good pulmonary function. I think it is essential when conservation procedures are employed, or indeed even when other radical surgical means are used, that we determine the status of pulmonary function by several pulmonary function tests, such as M.B.C., vital capacity, nitrogen washouts, etc. I never try to attempt conservation procedures when the

nitrogen washout tests are greater than 3 per cent, which indicates chronic debilitating disease.

With our 97 patients we have found that the following factors seem to be important:

One, a good functioning tongue is essential.

Two, the elevation of the larynx to close off the hypopharynx against a posterior displaced tongue. Even when you resect the supraglottic areas, the hypopharynx is closed off and assists in dumping the barium into the open esophagus.

Three, pharyngeal constriction is very important in assisting this. Segmental resection is important in rendering the final act of swallowing competent. For example, a cricopharyngeal myotomy can overcome a hyperplastic bar that Dr. Conley talked about. We see this consistently in our cases in which deglutition was difficult. In the last 50 cases of our conservation surgery we have performed an elective myotomy at the time of surgery and this has materially assisted in the quick rehabilitation of the patients.

Lastly, the gulping action, the patient's desire to want to master this technique, and the closed airway system. One must remove the tracheotomy tube quickly, and the exhalation cough, which he stressed, is important in rapid rehabilitation.

DR. EDGAR P. CARDWELL: There is one point that may have some significance. In these patients who have difficulty in swallowing I think perhaps that sometimes the gulping process may not succeed at first because of aspiration. I have had some success by having the patient stand on his head to do his first swallowing (that is, with hips on the bed and elbows on the floor or on a stool). In this position any food or fluid runs out of the trachea. The patient gains confidence and from practice, muscle control of the swallowing mechanism. I thought maybe that you would have a try at that. I feel we have gotten the feeding tube out earlier by this method. The patient then gradually learns to swallow in the upright position. Sometimes it takes a month or more.

I enjoyed this paper and it is certainly going to be helpful in our problems with the elderly patient who has swallowed with difficulty.

DR. CONLEY (closing): I want to thank the discussers for their interesting and, might I say, curious comments. I had a vision of Grandma Moses being asked to stand on her head and then swallow this bowl of cereal.

I am appreciative of Dr. Ogura's experience and comments on this because, as you can see, he has had an extremely vital relationship with perhaps the most critical area of the swallowing mechanism, namely, the area of the combined inlet, where the airway and the gullet separate from the common conduit. In his conservation operations at this site I feel that he has made splendid progress in the resolution of this problem. I gather that he still has a few minor problems with his patients in this area because he is still improving the technique.

CINE-RADIOGRAPHY OF THE LARYNX AND PHARYNX

F. JOHNSON PUTNEY, M.D.

GERALD D. DODD, M.D. ROY R. GREENING, M.D.

(Motion Picture Presentation)

DISCUSSION

DR. JOHN A. KIRCHNER: Actually there is very little to disagree with in this paper.

I think Dr. Dodd has given us an excellent resume of the various conditions in which cinefluorography of the pharynx and larynx is helpful in diagnosis. These conditions are the ones in which motion or the sequence of events during the act of swallowing must be studied. These include:

1. Cancer of the larynx and hypopharynx, in order to help determine the degree of fixation.
2. Dysphagia or nasal regurgitation in a postoperative patient after the larynx has been removed.
3. Aspiration, cough or choking of a patient when there is some suspicion of paresis or paralysis in the pharynx.
4. Hypopharyngeal diverticulum. Small diverticula are particularly hard not only to operate, but to demonstrate and I think that cine gives us a very reliable tool.

Not knowing what Dr. Dodd was going to show, I brought a couple of slides.

(Slide) This, for example, demonstrates the use of motion. This patient had a laryngectomy at another institution, but he still has his hyoid bone in place and had developed a good bit of dysphagia after the operation. The conventional film showed this huge mass behind the barium column. This was interpreted by the radiologist as a space-occupying lesion, probably a residual tumor or a recurrence. Under fluoroscopy and cinefluorography this mass was seen to change its shape and size. It flattened out and was obviously a mass of muscle which had been detached from the larynx anteriorly at the time of the laryngectomy and was contracting in an ineffectual and purposeless manner. This could be seen on laryngoscopy as a soft mass on the posterior wall of the hypopharynx. The cine was a great help in arriving at a correct diagnosis.

(Slide) This patient had paralysis of the pharynx. Here is a woman who was unable to swallow, was aspirating and choking. This conventional film showed what was interpreted to be a filling defect in the hypopharynx and a diagnosis of hypo-

pharyngeal carcinoma was made. Laryngoscopy was performed and no tumor was found. We went back and watched this under the fluoroscope—we did not have cines at the time, but the technique of course was the same. We found that the entire pharynx filled and then this side (indicating) emptied itself, but this side (indicating) continued to puddle the bolus of barium. What she had was a paralysis of the side on your right. The side that had not filled was the normal side. The cine would have been a great help to us in this situation too.

I heard a man near me during the showing of this film say that the barium swallow that Dr. Dodd showed was so rapid that it was very difficult to identify any of the structures. We also have had this problem. We found that if you run the film at 48 frames a second instead of 16 or 24 as is usual you get not only a better visual relation of the parts, but you increase the chances of getting at least one frame that shows the structure you are looking for.

The cine technique certainly does have its disadvantages and its shortcomings, as Dr. Dodd has pointed out. The detail does not compare with laryngography or laminography if you take single frames and compare one with the other.

(Slide) When radiological help is necessary I think you have to use all three of these. You have to do laryngograms, you have to do laminograms and cines if you have any problem in localizing the lesion.

This is a little out of focus and I hope you can see it, but the cine was of no help to us on this patient, who had a tumor of his epiglottis. We were considering doing a partial laryngectomy—a supraglottic type of resection. We were able to show with a laryngogram that the lower end of the lesion was well above the vocal cords.

(Slide) The laminogram showed that the mass was on the free edge of the epiglottis.

This slide shows the lesion resected. This is the hyoid bone and this (indicating) shows the tumor. We have quite a good margin below it. Without the laminogram and the laryngogram this would have been rather difficult to demonstrate. We certainly could not tell with laryngoscopy where the lower edge of the tumor was.

(Slide) This slide shows a huge supraglottic tumor which had extended into the pre-epiglottic space. This extension is a little difficult to demonstrate by cine studies.

The laminograms and even the conventional laryngograms are much better for pre-epiglottic extension in a tumor of this type.

I like Dr. Dodd's system of pushing the larynx to one side so as to be able to clear the cervical spine. I think this is a very good technique. If it allows him to get pictures like he showed us without the use of contrast material it will save not only time but it will save the patient's good nature and the surgeon's patience as well.

We have found similarly that if you rotate the patient under the fluoroscope when you are going to do laminograms that you may be able to line up the long axis of the vocal cord or the ventricle in such a way as to get a good shot at the larynx with your laryngograms before you actually start them. This is sometimes a problem, particularly with hypopharyngeal tumors where the larynx is slightly rotated and a little hard to line up.

I have enjoyed listening to Dr. Dodd and watching his film very much.

DR. DODD (closing): Someone asked just how the light was obtained to do these cine studies. This is electronic magnification of the fluoroscopic image, the intensity of the fluoroscopic image on the order of about 3,000 times, which enables us to photograph with conventional cine film the details of the fluoroscopic screen. Normally with a regular fluoroscope this would need 20, 30 M.A., which would be prohibitive for the patient, but with units of this type for fluoroscopy you can work at one-half mill for the making of the film. You can work on patients like this from 6 to 8 mills at 95 kv. The dose is limited and certainly is kept to a small area.

Secondly, insofar as the rapidity of movement on these films, I would like to remark that we tried to show a fair amount in a limited period of time. Normally in doing these, without editing the film, we would do a minimum of 30 frames per second, which gives a much longer run. Secondly, the projection speed that we use is commonly on the order of 15 frames per second instead of the normal 30 or possibly 60. If necessary, we can stop and advance or reverse at one frame per second, so that a very graphic representation and individualization of the frames can be obtained and this is not a problem.

I would also like to point out that also inherent in the cine, due to grain size on the film and so forth, is some of the blurring that you may have noticed on the screen. Normally in projecting cines they should be kept to approximately life size or possibly twice life size. This reduces artifacts and grain considerably and on a smooth screen a great deal more detail is apparent.

I want to thank you very much for the opportunity of appearing here and I thank Dr. Kirchner for his discussion.

INVESTIGATIONS IN LARYNGEAL PHYSIOLOGY: THE CANINE LARYNX

PART II

DAVID W. BREWER, M.D.

SIDNEY T. DANA, M.D.
(by invitation)

SYRACUSE, N. Y.

Fortunately, canine subjects are readily available and easily anesthetized. They present many still unexplored opportunities for the study of laryngeal physiology. Last year at the annual meeting of our association we reported to you on our initial studies of the canine larynx.¹ The motion picture briefly reviewed the anatomy and pointed out certain differences between the canine and the human larynx. It also showed the specific effect on vocal cord motion produced by stimulation of each of the intrinsic muscles. The still active freshly excised larynx, as well as the *in situ* larynx with intact blood and nerve supply, were used. With the exception of the interarytenoid muscles identical effects were evident in the freshly excised human larynx. In addition, electrical stimulation of the recurrent laryngeal nerves agreed with the findings of Nakamura² that the use of low frequency stimulating currents (around 20 to 30 cycles per second) caused opening of the cords while higher frequency currents closed the cords.

Our present investigation deals with variations in electrical activity of the intrinsic laryngeal muscles during respiration. Three basic considerations will be discussed: 1) methods of electrode placements; 2) methods for recording onset of inspiration; and 3) the effect of Nembutal® anesthesia on these muscles.

This study was supported in part by a grant from the Francis Hendricks Endowment Fund for Medical Research, administered by the Trustees of Syracuse University.

REVIEW OF THE LITERATURE

The work of previous investigators studying the electrical activity of the intrinsic laryngeal muscles demonstrates a diversity of approaches and results as summarized in the table (Fig. 1). For example: cats and goats were studied by Murtagh³ and later by Murtagh and Campbell⁴ using a Grass Model III inkwriting system connected to a pneumatic apparatus for recording external and intraluminal pressure changes related to respiration, while the cat anesthetized with Chloralose® and Urethane® was the experimental subject of Green and Neil⁵ who recorded action potentials by photographing the image on the cathode ray oscilloscope. A similar recording device was used by Nakamura⁶ but his subject was the dog anesthetized with Pentobarbital®. Fink and his colleagues⁷ studied awake human subjects by correlating Grass Model III electromyograms with cinematographic and roentgenographic data. Faaborg-Andersen⁸ reported on respiration and extensively on phonation in the awake human using a Disa three-channel oscilloscope with simultaneous continuous photographic recording.

In considering the activity in a specific muscle such as the thyroarytenoid, Nakamura⁶ was unable to record any activity in the dog under Nembutal anesthesia, but later indicated that activity could be observed. Faaborg-Andersen⁸ in the awake human was unable to show absence of activity in any muscle when the patient held his breath. Further, he found that greater activity was seen in the posterior cricoarytenoid muscle during expiration when the subject was at rest but changed to inspiration when the subject underwent forced respiration. Fink et al⁷ reported human cricothyroid activity both during forced inspiration as well as forced expiration.

From Figure 1 there appear to be significant differences in choice of subject, anesthetic agent, method of recording respiration, type and sensitivity of recording equipment and results obtained. Each of these investigations has added much to our knowledge and at the same time has provided us with many further questions to be answered.

METHOD

A total of thirty mongrel dogs weighing 10 to 25 kilograms were anesthetized with an initial dose of 30 mgms/kilo of Nembutal intravenously. Supplemental doses of 6 to 30 mgms were administered as

PHASE OF RESPIRATION WITH GREATEST MUSCLE ELECTRIC ACTIVITY

	Faaborg-Anderson	Fink et. al.	Green & Neil	Nakamura et. al.
SUBJECT	HUMAN	HUMAN	CAT	DOG
CRICOTHYROID	Inspiration	Inspiration* Expiration	—	Expiration
THYROARYTENOID	Inspiration	Expiration**	Expiration	No Activity
POST. CRICO-ARYT.	Expiration*** Inspiration	—	Inspiration	Inspiration
LAT. CRICO-ARYT.	Inspiration	—	Expiration	Expiration
INTERARYTENOID	Inspiration	—	—	Expiration
ANESTHESIA	Topical Laryng. Awake	Local Sub-Q Awake	Urethane Sleeping	Pentobarb. Sleeping
RESP. SIGNAL	Voluntary & Thermo-electric	Voluntary	Pneumatic	Pneumatic

* Inspiration during forced inspiratory effort and Expiration during forced expiratory effort.

** Expiration during deep respiration.

*** Expiration during quiet respiration and Inspiration during deep respiration.

Fig. 1.

needed. Duration of examination of each dog varied from three to seven hours. The larynx was exposed by suspension laryngoscopy in order to insert electrodes, observe cord motion and to permit simultaneous cinematography when desired. An external approach was always used for the cricothyroid muscle and occasionally for the other muscles. Blood and nerve supply to the larynx was never interrupted. One or two Disa three-channel electromyographs* provided simultaneous monitoring and recording of action potentials (Fig. 2). Electrodes used were Disa concentric needle electrodes types 13 K 53 and 13 K 13 (Fig. 2). Respirations were recorded by techniques to be described under the next heading. During the recording notations were made on observations of both animal and laryngeal activity as well as technical data pertinent to measurements of electrical activity. After sacrificing the animal the larynx was examined for evidence of hemorrhage into the muscles and for additional anatomical studies.

* A second Disa Electromyograph machine was made available to us through the courtesy of Mr. Bernard Cohen of EMG Associates, Inc., New York City.

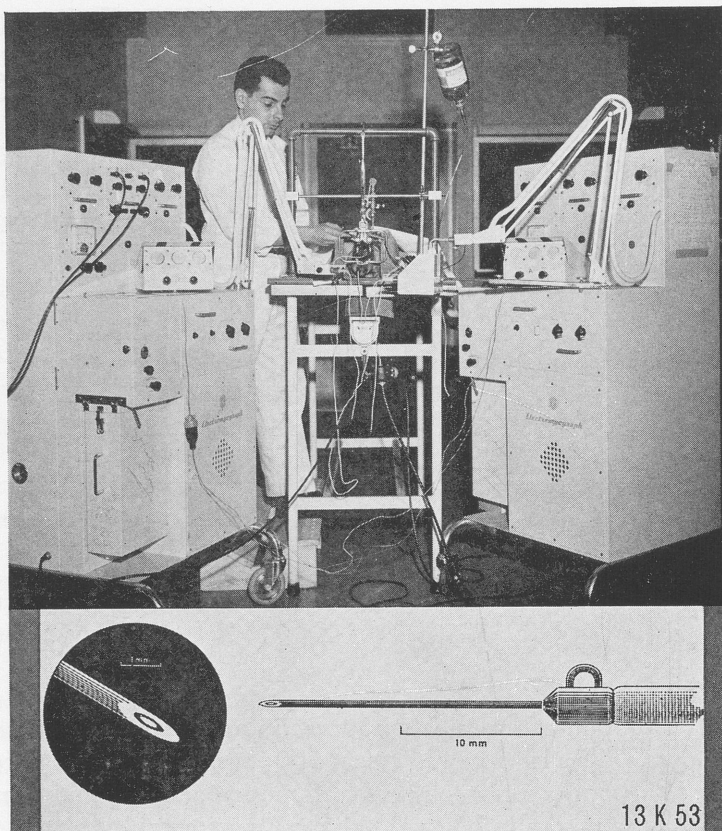


Fig. 2.—Canine subject with self-retaining laryngoscope in place. Two Disa Electromyographs permit 6 channel recording of action potentials by means of concentric electrodes such as (13 K 53).

RESULTS

A. *Electrode Insertion.* An effort was made to fulfill three self-imposed criteria. 1) The recording portions of the electrode must lie in the designated muscles irrespective of the thickness of overlying mucosa or overlapping adjacent muscles. 2) No splinting or removal of muscle pull on structures of the larynx or its attachments should exist, otherwise laryngeal physiology might be disturbed. 3) On insertion of the needle electrodes as little trauma as possible should

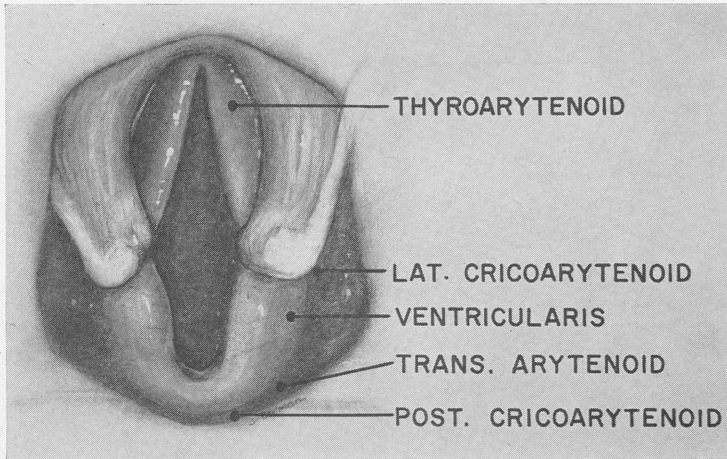


Fig. 3.—Laryngoscopic view with sites of electrode placement indicated.

occur since hematoma formation alters the electrical activity in the portion of the muscle so involved.

Three separate approaches were used plus combinations of all three in an effort to test and attempt to meet the above criteria. 1) An open surgical approach via supra-hyoid pharyngotomy; 2) percutaneous insertion, and 3) a per-oral insertion.

Duration of individual experiments and numbers of electrodes used precluded meeting all criteria mentioned above. For the majority of experiments a combination of approaches were used. This combination followed the technique developed by Faaborg-Andersen⁸ for human electromyography except for the following: 1) a self-retaining laryngoscope was used instead of the laryngeal mirror; 2) direct surgical exposure of the cricothyroid muscles frequently was used; and 3) the lateral cricoarytenoid was approached from the lateral aspect of the arytenoid cartilage rather than through homolateral thyroarytenoid muscle. The points of peroral insertion, based on previous anatomical and stimulation studies,¹ are indicated in Figure 3.

A few words should be mentioned regarding the ventricularis muscle. Though not found as such in the human larynx, it is analogous to the oblique portion of the interarytenoid muscle. It lies cephalad, medial and anterior to the transverse arytenoid muscle in

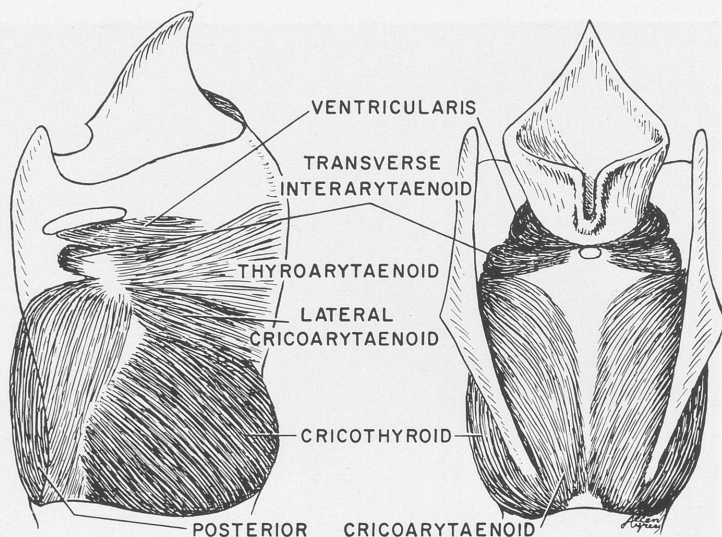


Fig. 4.—Canine laryngeal muscles—the ventricularis and the transverse arytenoid differ from the human interarytenoid in size and position.

the dog (Fig. 4). Both right and left ventricularis muscles are inserted into a special midline cartilage called the interarytenoid cartilage. Each also has a direct muscular connection with its fellow of the opposite side. Both the ventricularis and the transverse arytenoid muscles help close the posterior commissure as seen on stimulation studies.¹

B. *Methods for Recording Onset of Inspiration.* Criteria set up for choosing a means of signalling onset of inspiration include the following: 1) simultaneous recording on the electromyographic record; 2) an easily obtainable signal; 3) a signal which was regularly reproducible; 4) a sharply demarcated trace; and 5) a trace measurable both as to time and amplitude.

Respiratory Thermistor. A respiratory transducer, also known as a thermistor or special resistor which changes its electrical values in response to slight changes in temperature, was inserted into the airway approximately 2.5 cm above the glottic chink. When connected through an accessory Wheatstone bridge it recorded on one channel of the electromyograph. One might expect a "cold" deflec-

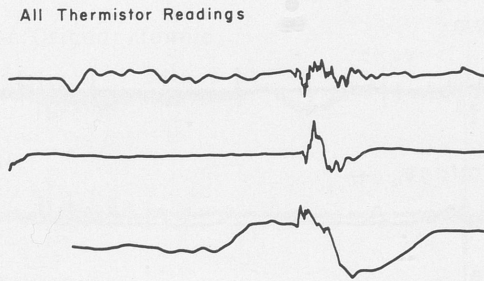


Fig. 5.—Examples of thermistor recordings. Note variations in base line and onset of increased activity.

tion on inspiration and a “warm” deflection on expiration. This was not routinely observed and the thermistor traces required both experience and additional information for any attempts at valid interpretation. In addition, the onset actually followed laryngeal muscle activity. In no instance did it occur simultaneously with or precede the activity of the posterior cricoarytenoid muscle.

Figure 5 presents a composite trace showing examples of three such recordings.

Ala Nasi. One day the flaring of the alae of the nose suggested that the ala nasi muscle be used as a monitor of onset of inspiration. On inserting a needle electrode into this muscle, activity was found present only on inspiration, the onset was sharply demarcated from the line indicating zero electrical activity, the onset preceded activity in the diaphragm and intercostals, the activity closely approximated the onset of activity in the posterior cricoarytenoid muscle and occurred at measurable intervals after expiratory activity in the remaining laryngeal muscles had ceased. Figures 6, 7 and 8 illustrate ala nasi activity recorded simultaneously with the intrinsic laryngeal muscles.

It appeared that the ala nasi muscle was the most accurate extra-laryngeal indicator of inspiratory onset and that in the larynx the posterior cricoarytenoid was the only indicator of onset of inspiratory activity.

C. *Effects of Nembutal Anesthesia.* After an initial anesthetic dose of Nembutal it is difficult to record activity in some dogs while

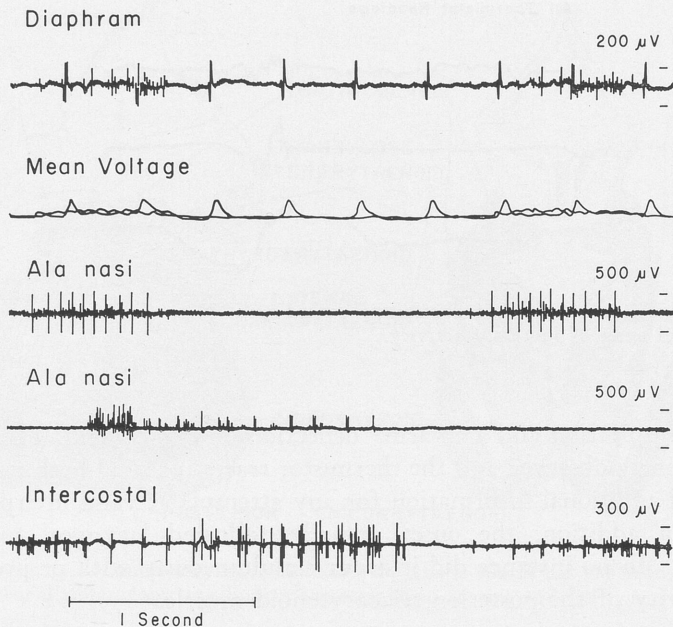


Fig. 6.—Diaphragm recording has cardiograph superimposed. Activity in the diaphragm and ala nasi occurs only during inspiration. (Subject No. 26; B-6) The bottom two traces show ala nasi active on inspiration and intercostal presenting primary expiratory activity.

it is easy in others. By waiting for the dog to lighten up to a stage of anesthesia comparable to upper Stage III in humans, it was possible to record muscle activity at all times in all of the intrinsic laryngeal muscles. Then by giving an additional dose of Nembutal the dog could be taken down into deeper planes of anesthesia for a period of time varying from a half hour to several hours depending on the accumulated dosage. During these progressive stages from lighter to deeper anesthesia serial recordings of muscle activity were obtained. Figures 9 and 10 present changes resulting from 0.4 cc of Nembutal given to a dog that was light, yet not whining or straining on the table. Within two minutes there was a slowing of the respiratory rate and a decrease in amplitude of electrical activity. The thyroarytenoid did not record at three minutes and by 15 minutes the cricothyroid no longer demonstrated regular rhythmic activity. At 25 minutes cricothyroid activity had returned and by 30 minutes both

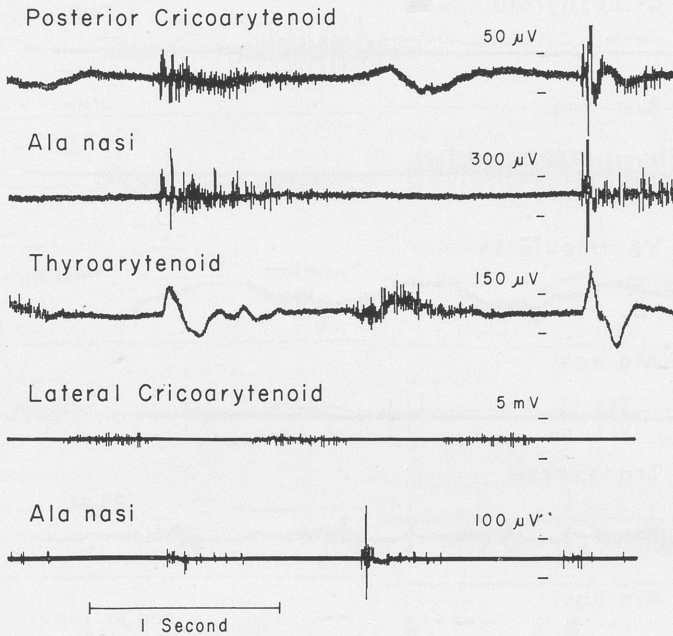


Fig. 7.—Posterior crico-arytenoid and ala nasi active only during inspiration. Thyro-arytenoid is active during expiration. Artefact is due to thyro-arytenoid needle motion on inspiration. (Subject No. 19; A-5) Lateral crico-arytenoid active only during expiration. (Subject No. 26; C-2)

muscles were nearly as active as they were before the run commenced. Addition doses of Nembutal were observed to depress all of the other muscles studied in several subjects with recurrence of activity appearing in reverse order.

During lighter planes of anesthesia while attempting to phonate, or while hyperpneic in acidosis, a marked change of activity in the posterior cricoarytenoid muscle activity repeatedly was observed. Figure 11 indicates activity occurring simultaneously with that evident in both the ventricularis and the transverse arytenoid muscles. These latter two muscles were active only during the expiratory phase in all instances. Surprisingly little activity is evident in the posterior cricoarytenoid during the short inspiratory phase.

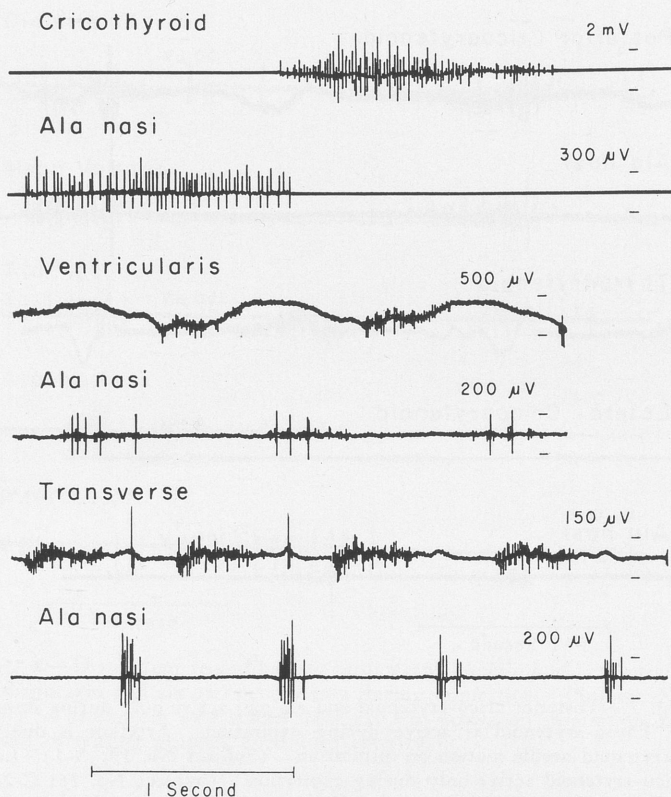


Fig. 8.—Upper two traces: Cricothyroid active only during expiration while ala nasi shows inspiratory activity. (Subject No. 25; A-1) Middle two traces: Ventricularis active only during expiration. (Subject No. 26; A-9) Lower two traces transverse: Transverse active primarily during expiration. (Subject No. 26; C-6)

COMMENT

Electrode Insertion. Several approaches for electrode insertion were tried. First, following Nakamura's⁶ technique, the pharynx was incised above the hyoid bone and the larynx elevated with its blood and nerve supply intact. This gave the operator excellent visual control. However, with the possibility of later human application in mind, and aware that additional pull on the stabilizing inferior constrictor and absence of stabilization from the suprahyoid muscles

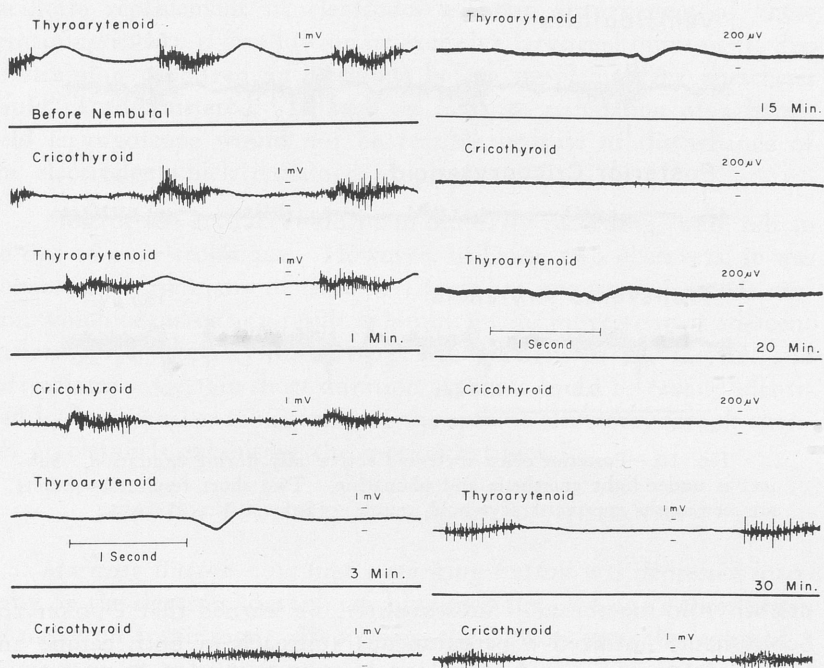


Fig. 9.—Serial recordings indicating effect of an additional dose of 0.4 cc of Nembutal (30 milligrams per cc) on thyro-arytenoid and crico-arytenoid muscles. Note progressive decrease in activity of thyro-arytenoid then cricothyroid, particularly at the 20 minute interval. At 30 minutes both muscles are again active. (Subject No. 5; A-9)

presented two uncontrollable factors, it was abandoned. In addition, it was noted in earlier stimulation experiments¹ that when the cricoid cartilage is fixed (as when the freshly excised larynx is mounted on a plastic tube) the thyroid cartilage exhibits marked lateral torsion, and no motion of the cricoid occurs. On the other hand, a self-retaining laryngoscope splints the thyroid cartilage much as do the strap muscles and suprahyoid muscles.

Percutaneous insertion through the skin and into the cricothyroid muscle was easily accomplished following the technique of Faaborg-Andersen.⁸ Also, the thyroarytenoid could be reached via the cricothyroid membrane using both external and laryngoscopic landmarks. This was done, however, after midline incision and exposure of the

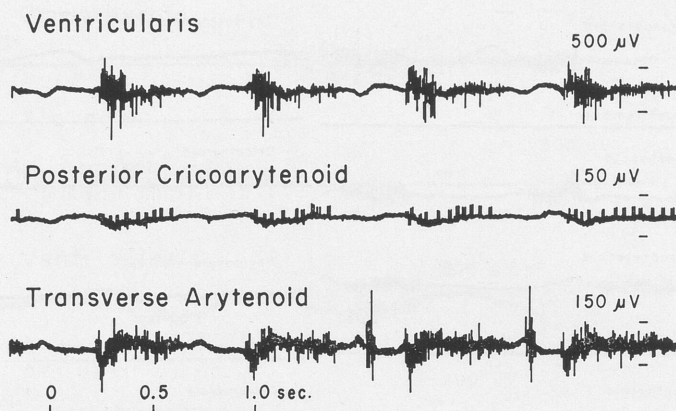


Fig. 10.—Posterior crico-arytenoid active only during expiration. Subject is under light anesthesia and phonating. Two short respiratory bursts are seen in the transverse arytenoid muscle. (Subject No. 25; A-5)

cricothyroid membrane. Subsequently, we learned that Dunker and Schlosshauer⁹ utilized a percutaneous approach in both canine and human subjects to reach the thyroarytenoid muscle. In our hands this was not an innocuous procedure for on several occasions edema in the floor of the ventricle and hematoma from repeated placements were noted. The lateral cricoarytenoid, ventricularis and transverse arytenoid could be reached with visual control by direct laryngoscopy while the posterior cricoarytenoid required blind cricoid cartilage fenestration for insertion.

Peroral insertion was relatively easy after becoming more familiar with the structures of the canine larynx.¹ It is of interest to note that in the dog rapid occurrence of edema in the floor of the ventricle occurs when the electrode is placed lateral to the lateral margin of the vocal cord just as trauma in the human stimulates the same visible reaction. In order to facilitate peroral insertion and to permit continued observation of the vocal cords in the anesthetized dog, a suspension laryngoscope was used. This also permitted simultaneous motion pictures to relate cord motion and interpretation of so-called artifacts in the action potential patterns recorded electromyographically. However, the presence of the laryngoscope splinted vocal cord motion to a variable degree depending upon the amount of tension applied to

the structures by the several suspension adjustments. It did, however, facilitate replacement of electrodes without disturbances of those remaining in situ if swallowing or excessive laryngeal movements due to straining dislodged an electrode before supplementary anesthesia could be administered. In such an instance reinsertion of a hand-held laryngoscope would not be feasible because of disturbance of the electrodes which remained in place.

Most of the artifacts evident in our early recordings were felt to be due to poor technique. However, utilizing two observers, it was possible to relate many of these "artifacts" to imparted electrode motion which occurred as a result of simultaneous movement of adjacent structures (hypopharynx, cartilaginous portions of the larynx, and soft palate). Certain short duration artifacts could be readily identified by loud speaker monitoring of the specific electromyograph channel or channels exhibiting the motion artifact.

METHODS FOR RECORDING ONSET OF INSPIRATION

Marking Button. At first a *marking button* was depressed manually on the electromyograph machine each time the dog's chest began to move on inspiration. Depressing the button optically exposed a linear strip at the upper edge of the paper film. This method gave only approximate information and exhibited an increasing error as the respiratory rate became more rapid. It was therefore discarded.

Percutaneous Insertion. Simultaneous recording of action potentials from intercostal muscles gave variable results. When *intercostal muscles* in the caudad interspaces were used initial insertion might exhibit early inspiratory activity. If the needle had to be replaced only expiratory activity might then be recorded. More cephalad interspaces yielded a superimposed electrocardiograph trace. First and second interspace muscles often did not record when the dog was under deeper anesthesia during the first hour or two after his initial dose of Nembutal.

Percutaneous electrode insertion gave good recordings but again electrocardiographic tracings frequently were superimposed. In addition, diaphragmatic activity came much later than activity in certain intrinsic laryngeal muscles. It was therefore felt not to be an accurate indicator of onset of inspiration for this investigation.

Thermistor. The rapidity of action of the thermistor made it difficult to correlate the thermistor oscilloscopic trace and the laryn-

geal motions observed through the laryngoscope. The trace was of such a character that it did not allow loud speaker monitoring.

Ala Nasi. The ala nasi provided our most reliable extralaryngeal indicator of inspiratory onset. With superficial electrodes taped to the ala in the awake human, recordings were obtained in inspiration only unless superimposed voluntary activity occurred.

Effects of Nembutal Anesthesia. In lighter anesthesia many variations were encountered. Of particular significance was the finding, encountered regularly, that the posterior cricoarytenoid muscle shifted its major activity from inspiration to the expiratory phase when the dog was light and attempting to phonate or when hyperpneic in acidosis. (The inspiratory activity was of shortened duration and lesser amplitude.) This phenomenon occasionally occurred with other muscles but not with the predictable regularity of the posterior cricoarytenoid. This is a subject now under further investigation.

One dog used on three previous occasions remained active and running around the room long after the routine initial dose of Nembutal. A second identical dose was then given and bizarre traces were obtained, unlike any previously or subsequently observed. It appeared that this animal had developed a sensitivity to the anesthetic agent and exhibited excitement phenomena reminiscent of humans sensitive to the same agent.

CONCLUSIONS

1. Access to the intrinsic muscles of the canine larynx for electrode insertions was best afforded by a combined approach utilizing either an open surgical or percutaneous approach for the cricothyroid muscles; and a peroral approach for the thyroarytenoid, lateral cricoarytenoid, posterior cricoarytenoid, ventricularis and transverse interarytenoid.
2. Search for a suitable signal of onset of inspiration indicated that the ala nasi muscle provided a sharply demarcated onset, was active only during inspiration, and preceded in onset the activity of the intercostals, diaphragm, and various thermistor placements.
3. Electrical activity can be demonstrated electromyographically in all of the intrinsic muscles of the larynx in the dog anesthetized with Nembutal. Each muscle is active during expiration except for the posterior cricoarytenoid which is active during inspiration. Varia-

tions are found in this pattern of activity especially in the posterior cricoarytenoid which frequently shifts its activity to expiration when the dog is lightly anesthetized.

4. The effect of additional doses of Nembutal upon the larynx is to depress neuromuscular activity. First, thyroarytenoid activity disappears, then cricothyroid, etc. Given an adequate dose of anesthetic, all activity can be abolished temporarily.

SUMMARY

Problems encountered and results obtained during investigation of intrinsic laryngeal muscle activity during respiration in approximately 30 dogs are reported. Techniques of electrode placement are reviewed and points of percutaneous and peroral insertions are described. Methods of recording onset of inspiration are reviewed and the usefulness of the ala nasi and particularly of the posterior cricoarytenoid as inspiratory monitors are noted. Observations on changes in action potential patterns observed electromyographically with the canine subject under varying depths of Nembutal anesthesia are reported. During quiet respiration as the depth of anesthesia increases a progressive diminution in both mechanical and electrical activity was observed. In addition, a sequential pattern of absence of recorded electrical activity was noted beginning with the thyroarytenoid muscle, then the cricothyroid, etc. Recurrence of activity as the anesthetic effect diminished occurred in reverse order. When hyperpneic or when under lighter anesthesia and attempting to phonate the posterior cricoarytenoid, previously active only during inspiration, was active primarily during "expiration" while by comparison inspiratory phase activity was diminished or absent. One subject exhibited an initial excitement reaction to Nembutal and after repeating the full initial dose bizarre phasic respiratory activity was noted unlike that previously or subsequently observed. Further studies presently are in progress.

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MICROPATHOLOGY OF RADIATION REACTION IN THE LARYNX

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CLEVELAND, O.

The purpose of this paper is to review the microscopic pathology that results from irradiation of the normal structures of the larynx.

The normal tissues of the larynx resist gamma radiation of less than 2500 r and will tolerate 5000 r in the usual divided therapeutic dosage. Dosage in excess of 5000 r is needed for the eradication of most squamous cell carcinomas of the larynx. There is variation of the amount of damage to normal tissues from individual to individual, as well as from tumor to tumor, which is yet unexplained in our present limited knowledge.¹⁻³

The sections presented were obtained from cases that have been irradiated with 250 kv units, cobalt 60, or super-voltage, receiving 7000 to 7500 r in an average of 40 doses covering a period of fifty days. All cases had been irradiated for the purpose of eradicating squamous cell carcinoma, but without success. It was impossible to tell from the sections, which type of therapy had been administered. The twelve specimens examined were obtained from six to twenty-four months following termination of the radiation, and representative sections will be presented.

EPITHELIAL CHANGES

Ciliated epithelium is highly susceptible to radiation while squamous epithelium is less susceptible, Figure 1. Ciliated epithelium ceases to function in a short time after therapy starts, with the accompanying loss of ability to move secretions out of the subglottic area of the larynx. With this loss of ciliary activity, there is a loss of function of the goblet cells and deep tubo-acinous mucous secreting glands especially in the ventricles and false cord. This produces the mucositis

and dry mucosa, with loss of the mucous blanket and resistant to infection afforded by an active mucous secreting ciliated epithelium (Fig. 2).

The oyster-like edema that is persistent in many irradiated larynges is hard to explain except on the basis of destruction of venous and lymphatic drainage. The submucosal area of such a case is edematous with general disruption of the tissues, hyalinization, small atrophic scattered cell nuclei, and wide unoccupied areas which in the living state may have been occupied by fluid.

The fact that keratotic squamous epithelium is resistant to radiation is well shown in Figure 3. The pseudo-sarcomatous changes reported by Lange and Baker were noted in a number of the specimens.⁴⁻⁶ This tends to confirm their observations that this occurred in irradiated squamous cell carcinomas, Figure 4.

Figure 5 shows the irradiation damage produced in the squamous cell carcinoma with fractionated dosage of 7000 r. The clumping of the chromatin of the nuclei with the arrest of mitosis and multiple nuclei formation is outstanding. Vacuole formation in the cytoplasm and shrinking with atrophy of the cells is marked.

MUSCLE CHANGES

Striate muscle is highly resistant to radiation, but in cancericidal doses undergoes atrophy. Fatty degeneration and Zenker's degeneration both occur. Zenker's degeneration is characterized by deposition of hyaline which bridges the distorted, wavy, pale, muscle fibers, and is most common in radiation necrosis of muscle. There is loss of striation and the substance is clear, translucent, homogenous, and takes the acid stain. The muscle nuclei are scattered or absent. If the degeneration is severe, fibrosis and complete hyalinization takes place, Figure 6. Muscle tissue, once destroyed, is replaced by scar tissue, and does not regenerate.

VASCULAR CHANGES

Of all the damage produced by irradiation in normal tissues, the most important is the damage produced in the blood vessels and lymphatics. Endothelium is susceptible to damage if the level of irradiation exceeds 1200 r and is usually irreversible. The lining endothelium of the lymphatics and capillaries undergoes early prolifer-

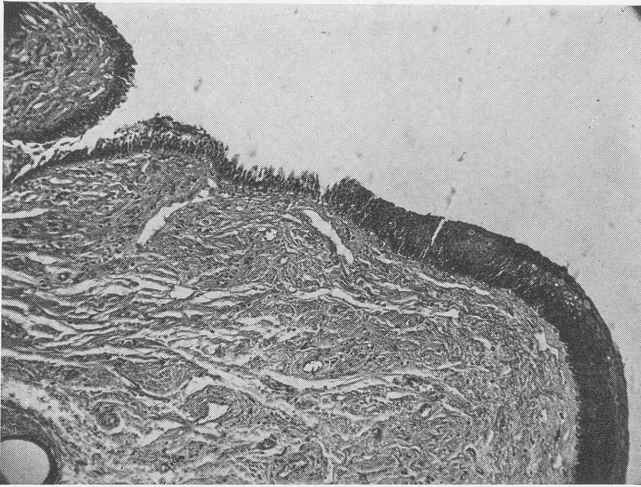


Fig. 1.—Shows the junction of squamous and ciliated pseudostratified columnar epithelium in the ventricle. The degeneration of ciliated epithelium of the ventricle is more marked than the relatively intact squamous epithelium of the true cord, which shows only slight vacuolization.

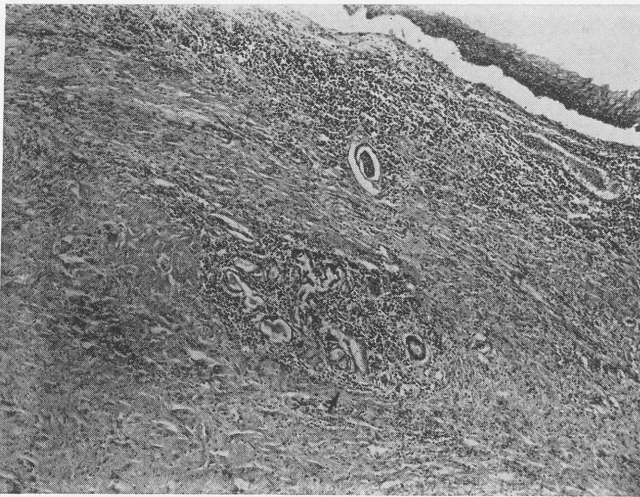


Fig. 2.—Shows the submucosal picture in the mucositis of the false cord with the sloughing of the squamous epithelium, infiltration of inflammatory cells, swelling of the submucosal tissues, fibrosis, necrosis, and hyaline formation. Complete degeneration of a deep tubo-acinous gland of the false cord (arrow).

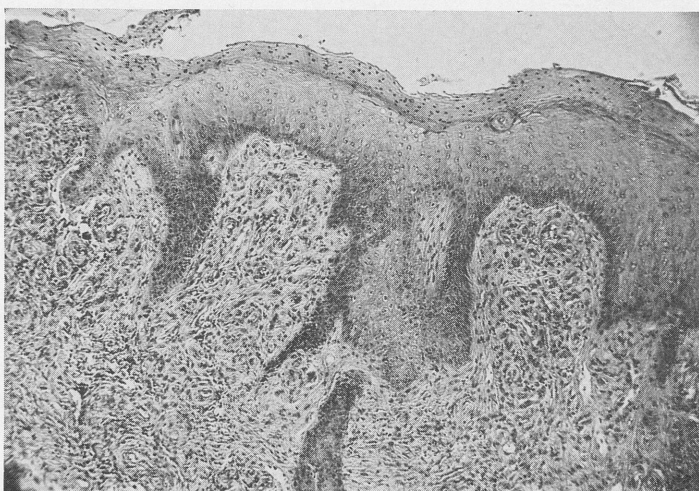


Fig. 3.—Shows heavily irradiated keratotic squamous epithelium which is little affected except for mild vacuolization of the nuclei. The submucosa is fibrotic with the pseudosarcomatous changes noted by Lange and Baker.

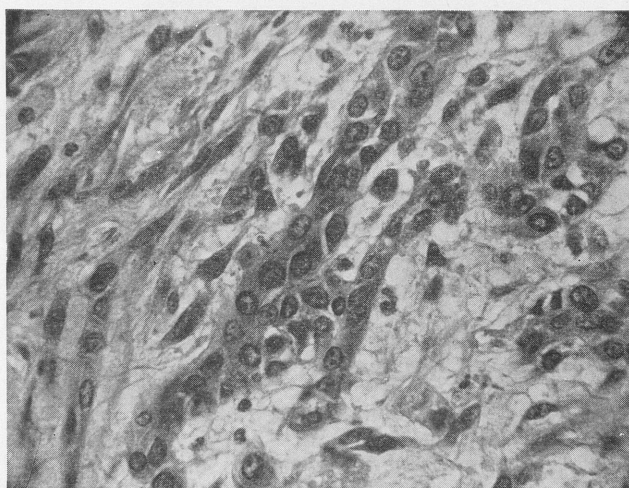


Fig. 4.—Pseudosarcomatous changes of an invasive squamous carcinoma seen in a number of these irradiated larynges.

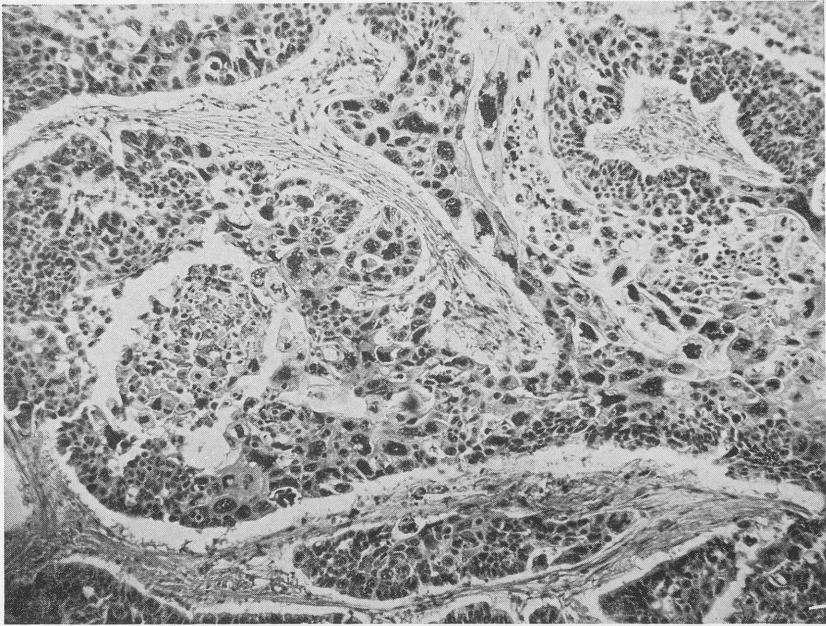


Fig. 5.—Squamous cell carcinoma showing irradiation effects. The nuclei of the tumor are partly pycnotic, and degenerated, with arrested mitosis. There are abnormal nuclei and multiple nuclei formation from chromosome injury with clumping of chromatin. Marked vacuolization of cytoplasm can be seen, with shrinking and atrophy of many of the cells.

eration followed by vacuolization of the cytoplasm and necrosis. This marked endothelial reaction results in early obliteration of the capillaries and lymphatics. The hyalinization of the collagen surrounding the capillaries and lymphatics aids in the obliteration of these structures, and as atrophy and fibrosis occurs, prevents their regeneration. Capillaries and lymphatic structures are notably absent in most of the section shown.

This endothelial damage is also marked in veins of all calibers with the additional damage due to subintimal fibrosis and hyalinization of the collagen with thickening of the walls at the expense of the lumen. The resulting phlebosclerosis may be more marked than in arteries of similar size.



Fig. 6.—Striated muscle with hyaline bridging, loss of striations, and the distorted, wavy, pale, necrotic muscle fibers, characteristic of Zenker's degeneration (arrow). Note the great reduction of muscle nuclei with early fatty degeneration and fibrosis (arytenoid muscle).

The arteries suffer the same damage to the endothelium and intima with marked hyaline thickening of the intima, Figure 7. In the tunica media, the elastic fibers degenerate and the smooth muscle shows hyalinization. The tunica adventitia atrophies, and is replaced by proliferation of fibroblasts, collagen formation, and fibrous bands of both hyaline and fibrous tissue (Fig. 8). With these changes, the smaller vessels are occluded and the larger vessels reduced in caliber with a resulting marked lowering of resistance and reparative powers of the irradiated structure. Unfortunately, these changes go on for months and years after irradiation and account for late fistulae, infection, and slough seen in the larynx long after the therapy is completed. Rupture of the larger vessels following infection and sloughing is well known to all. Large vessels become stiff, inelastic and

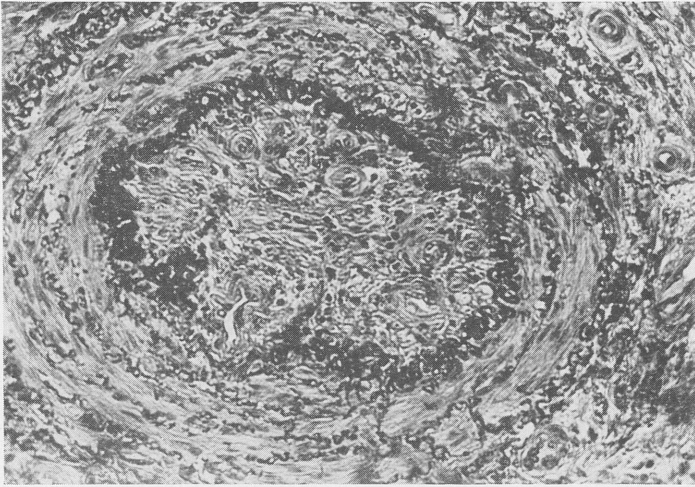


Fig. 7.—Illustrates marked endothelial proliferation in a small artery with obliteration of the lumen. The media is thickened, but elastic tissue is relatively intact.



Fig. 8.—Large artery with organized thrombus. The tunica media is fibrotic, the elastic fibers degenerate, the smooth muscles hyalinized. The tunica adventitia shows proliferation of fibroblasts, collagen formation, and fibrous bands of both hyaline and fibrous tissue.



Fig. 9.—High power section of thickened hyalinized perichondrium, with marked degeneration and distortion of the cells of the fibro-elastic matrix and elastica.

fibrous, and have been seen to split when not protected by supporting structures.⁷

CARTILAGENOUS CHANGES

Cartilage is slow to show changes to radiation in the absence of infection. Radiation chondritis and perichondritis develop slowly, months and years after exposure. The perichondrium showing degeneration of the nuclei and the superficial cartilage degenerating with engorgement of vessels and infiltration of polymorphonuclears, lymphocytes, and eosinophiles, without much change in the matrix of the cartilage. With time, aseptic necrosis develops. If the covering of the cartilage remains intact, it can stand large amounts of irradiation. However, with the impairment of lymphatic and vascular supply, it is easily infected and easily damaged by trauma. This often



Fig. 10.—High powered section of hyaline cartilage irradiated but not infected. The matrix is relatively unaffected, but the perichondrium is hyalinized and acellular. There is degeneration of many of the cells in the matrix, while others remain intact. The dense hyalinization of the acellular perichondrium is the main change.

results in extensive necrosis and sequestration with deformity of the larynx if healing does take place. Hyaline cartilage and fibro-elastic cartilage are similar in their reaction (Figs. 9, 10).

The effect of radiation on bone is mentioned because of the high percentage of bony areas in the thyroid cartilage. Radionecrosis of bone may take place beneath intact mucous membranes, and sequestration is very slow unless secondary infection occurs. Infection or trauma produce marked and gross disintegration of irradiated bone. There is little alteration in the bone to examination, except that it is much less susceptible to histolytic solvents. The bone seems more brittle, cell spaces and canaliculi are widened, irregular, reduced in numbers with abnormal spaces between the lamellae. The lamellae

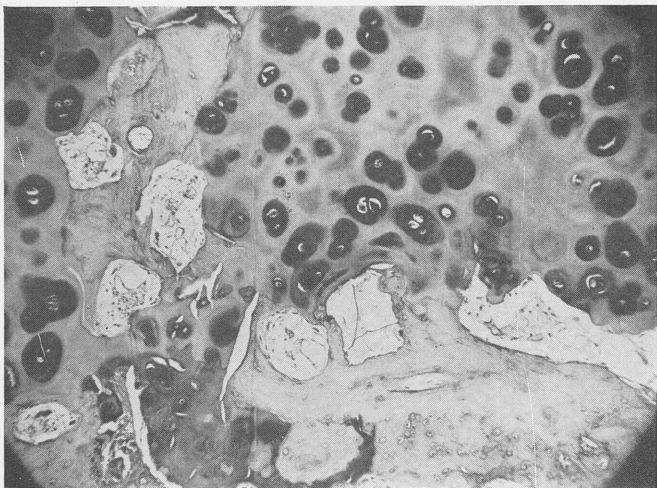


Fig. 11.—Calcified thyroid cartilage showing the degeneration and absorption of substance between the interface of the bone and cartilage with aseptic necrosis of the osteogenic layers. (Steven's H and E stain). There is little change in the substance of cartilage matrix.

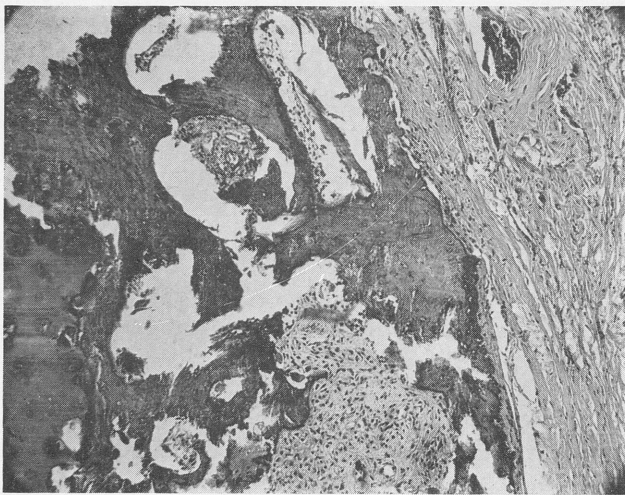


Fig. 12.—Typical fragmentation and disruption of the calcified thyroid cartilage when infected.

appear very hyaline as some unexplained change takes place in the lamellar substance. Atrophy and eburnation are late changes. Necrotic bone acts as an infected foreign body (Figs. 11 and 12).

COMMENTS

It is hoped that in the future, new techniques will evolve to lower the damaging dose of irradiation and increase its effectiveness against laryngeal tumors. Corgill's demonstration of peroxide infusion points the way.^{8,9} Other chemicals may help radiation to be more effective, and is a worthwhile subject for research. Crile and others demonstrated that heating made tumors more susceptible to irradiation.¹⁰ Possibly the combination of heat, super-oxygenation, and irradiation may be even more effective.

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DISCUSSION

DR. HERBERT H. HARRIS: It has been most illuminating to have read Dr. Alexander's paper and hear his presentation of the histopathological processes involved in the radiation of normal tissue. Frankly, my knowledge of this subject has been somewhat vague until I began to study some of our slides in more detail. I might inject here that this was done with a pathologist with whom I often consult. In general, I do not believe that I could add much to his paper. In nearly every instance his findings were similar to those which we found in reviewing the sections of several patients treated by radiation and subsequently operated.

In two instances I reviewed the tissues of the larynx from the anterior and posterior portions. Clinically, the larynx seemed to have received more radiation posteriorly than anteriorly. A calculation was made that when the wedges were used the larynx had received 5000 R to the anterior area and 7800 R to the posterior area. The radiation was given in sixteen treatments over twenty-eight elapsed days. The laryngectomy was done because of pain, edema and obstruction without evidence of resolution after six months. I could not differentiate microscopically more degenerative changes in the two areas and the pathologist with whom I consulted could not make a differentiation. True, the tissue of these two areas are different. The tissue responses, regarding blood vessel change, changes in the nuclei, the hyalinization, acellular elements and fibroblasts were not different on the basis of the amount of radiation; and, therefore, quantitative estimate on whether one tissue received more radiation than the other could not be made.

As did Dr. Alexander, we found on examining slides where 220 K.V. was used over a period of thirty-six treatments with a total dosage of 6500 R could I see any difference between the tissue response when 2 M.E.V. was used with a total dosage of 5000 R for sixteen treatments over twenty-eight elapsed days. Undoubtedly, if the same total dosage was given in a decidedly shorter period of time as against that given over thirty-six treatments, the tissue response should be different. Just where this difference can be seen microscopically, I do not know. One well-trained in examining postradiated tissue could tell much easier than could a pathologist who is not specially trained in examining radiated tissue.

The individual response to radiation microscopically is not at all easy to assay, but clinically there is a great difference, and probably examining a cross-section of the larynx would be of considerably more value.

It has been a privilege to have been asked to discuss this paper, and I wish to congratulate Dr. Alexander, a clinician, on his courage in presenting such a basic view as he has so ably done.

DR. JOHN A. KIRCHNER: I think this is a very important paper and I certainly enjoyed hearing it.

I would like to ask Dr. Alexander a question. He showed several sections of squamous cell tumors which had been radiated and in which the cells still appeared to be viable. I wonder whether he considers these cells necessarily cancer.

The reason I ask this is that the radiologist in New Haven has been interested in this question. He has taken some of these postradiated tumors and grown these cells in tissue cultures. He finds that they do survive and can be kept alive for an almost indefinite period of time. They seem to answer all the criteria we expect of living tissues, with one exception and this is that they do not divide. The cells do not reproduce themselves. I wonder whether such a cell, which morphologically is a cancer, is in fact a cancer if it does not divide.

DR. STANTON A. FRIEDBERG: I think this is a timely subject for Dr. Alexander to have brought before us, particularly so because of a tendency at present toward the use of irradiation preliminary to surgery. This is true in connection with bronchial and esophageal neoplasms, and occasionally with laryngeal tumors.

I was particularly happy to note that Dr. Alexander, by means of slides, gave a good explanation for some of the clinical phenomena we see after irradiation. The fact that these changes are progressive over a period of time accounts for something that we know clinically, namely that the irradiated larynx can be operated upon within a short time of completing therapy and the postoperative complications are not nearly as great as those which take place when the surgery becomes necessary some years later.

His slides illustrate why increased recognition and appreciation of the late development of carcinoma in irradiated fields is something to contend with.

He explained beautifully, I thought, the reason for the severity of the reaction of irradiated tissues to infection. We have all noted how severe the laryngitis of an ordinary infectious type can become and how persistent it is in the irradiated larynx. The complaint which many of these people have of extreme dryness in their throats was well illustrated by the vivid examples of atrophy of the glands of the submucosa.

In all these respects I think we are indebted to Dr. Alexander for a very interesting presentation.

DR. J. L. GOLDMAN: We indeed should be indebted to Dr. Alexander for a basic work of this kind because it helps us determine the direction in which we are going in the treatment of carcinoma of the larynx.

I would like to make these comments in order to emphasize that the local reaction occurs immediately after radiation and lasts for about three weeks. Dr. Alexander pointed out that some of the late changes are endarteritis, fibrosis and hyalinization, but these pathologic changes do not occur until a certain lapse of time after radiation. We think the period is after two months, so that surgery, after the third and before the sixth or eighth week, can be done with security.

DR. ALEXANDER (closing): In answer to Dr. Kirchner's question about whether these cells were viable or not, the only thing I can say is that these larynges were removed with actively growing cancer. I didn't show the sections of the growing cancers that were present that had not been eradicated by the radiation. We went up to 7500 R on some of them. However, it would be interesting to find out whether these cells would actively grow when they are encased in fibrotic tissue such as we saw in many of these larynges.

I want to thank Dr. Harris and Dr. Friedberg for their kind words.

I think Dr. Goldman's point about the ideal time to operate on these cases is well brought out in our studies of these sections. I think that between the six-week period and the two-month period is the ideal time to operate if you are going to us initial radiation and then operate. The skin reaction is beginning to subside. You do not get the fibrosis and sclerosis of the vessels developing in that time. It is only the lymphatics and the smaller arteries and arterioles that are affected that early. That is why you have somewhat less bleeding in these cases. I notice that Dr. Tabb mentioned the fact that his irradiated maxillary lesions bled less after they had received radiation when they were operated within two or three months.

MIXED TUMORS OF THE PAROTID GLAND
WITH EXTENSION TO THE
LATERAL PHARYNGEAL SPACE

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AND

DAN W. HABEL, M.D.

(by invitation)

ANN ARBOR, MICH.

Although much has been written about benign mixed tumors of the parotid gland in general, little has been written concerning deep mixed tumors which extend to the intra-oral and pharyngeal structures. Since the founding of the American Laryngological Association in 1879, a few cases with benign mixed tumors arising in minor salivary glands of the pharynx have been presented before the members of this Society. However, we have been unable to locate a paper in this Association's Transactions dealing exclusively with tumors arising in the parotid gland and presenting in the pharynx or the palate. Such patients often present interesting and complex problems in diagnosis and treatment. It is not entirely clear in all cases where these tumors originate in relation to the deep portion of the parotid gland. Further, the exact anatomical route of medial extension may be difficult to ascertain in some. During the past few years we have had the opportunity to treat six of these patients and wish to report our experiences. In addition, we wish to report six additional cases that were treated at the University of Michigan Hospital by other members of the Department of Otorhinolaryngology.

There is not entire agreement among surgeons as to the site of origin of these tumors. For example, Havens and Butler¹ reported forty-one cases and believed the tumors arose primarily in minor salivary glands or ectopic parotid tissue in the lateral pharyngeal and

From the Department of Otorhinolaryngology, University of Michigan.

retropharyngeal spaces. It is believed by some surgeons that many of the deep mixed parotid gland tumors arise from the stylomandibular prolongation of the parotid gland and thus grow into the lateral pharyngeal space. Gaughran² has pointed out that 74% of the anatomical parotid gland specimens examined showed a stylo-mandibular lobe. We believe that in addition to this origin, some may arise in the inferior extension of the retromandibular portion of the parotid gland which lies along the external carotid artery inferior to the stylomandibular ligament. We will elaborate on these points later.

Several methods for the surgical removal of these tumors have been proposed. An external surgical approach appears to be used frequently by most surgeons. An intra-oral approach is the method of choice by others in selected cases. In patients with extremely large tumors, a combination of both surgical approaches may be indicated.

It is not unusual for the palatal swelling to be found on routine physical examination. The patient, in this instance, is usually entirely without symptoms. There may or may not be palpable external swelling. Patients with tumors that arise in the stylomandibular projection of the gland usually have an external swelling located anterior and inferior to the tragus of the ear. Patients with tumors arising from the inferior extension of the retromandibular portion of the parotid gland usually present little or no external swelling.

Since the pharyngeal and palatal tumor mass is usually not found except on routine examination, one would not expect confusion with inflammatory lesions. However, it is not unusual to have a patient state that when the palatal mass was discovered, it was incised to establish drainage. One of the most useful methods in diagnosis is bimanual palpation. Even though there may or may not be an external mass, the examiner gets the sense of deep tumor formation and continuity by ballottement. This ballottement is felt over the external mass when present, otherwise it is felt along the angle and medial to the horizontal ramus of the mandible. This latter is particularly true in those patients where the tumors arise below the stylomandibular ligament and eventually extend medially. Patients with tumors arising in the stylomandibular projection of the gland and growing through the stylomandibular tunnel characteristically have tumefaction below and in front of the tragus of the ear as stated previously.

Since treatment and prognosis vary greatly between a malignant and benign tumor presenting intra-orally, it is important to make this differentiation pre-operatively if possible. Benign tumors are encapsulated while malignant tumors are infiltrative. This difference of growth can usually be described by the palpating finger. If malignancy is suspected, an incisional biopsy specimen should be obtained through an intra-oral route. If malignancy seems unlikely, an incisional biopsy should not be obtained. This is especially true if a mixed tumor is suspected. Patients with tumefaction in the lateral pharyngeal space or palatal areas cannot always be said to have a benign mixed tumor, for other tumors such as neurofibromas, neurilemmomas, leiomyomas, and lipomas may also grow in these anatomical sites. However, since these tumors listed are benign, the surgical treatment is essentially the same as with benign mixed tumors. Only by operation and subsequent microscopic tissue examination is it possible to make a correct diagnosis.

SURGICAL ANATOMY

It is not the purpose of this discussion to give a detailed summary of the anatomy of the parotid gland, but rather to point out some of the salient anatomical features of the gland itself and adjacent anatomical structures which are concerned with the deep growth of these tumors.

The parotid gland is enveloped in a dense fascia which, not only encases the gland, but divides it into many compartments. This compartmentalization occurs in the deep and superficial portions of the gland. The gland itself consists of a large flat superficial portion located in front of the ear and spreading over the masseter muscle. It dips deeply and fills the cleft between the mastoid tip and the ascending ramus of the mandible. There is an extension of the outer portion of the gland inferiorly as the tail of the parotid gland. The tail of the gland is bisected by the common facial vein. The medial extension of the gland into the cleft between the mandible and the mastoid tip is a constant anatomical finding. It is pierced by the VII cranial nerve. Gaughran² has stated that a deep extension of the gland evaginates the stylomandibular fascia which is called, by him, the stylomandibular extension of the gland (Fig. 1). Furstenberg³ called it the carotid extension and Faure⁴ called it the pharyngeal extension. In actuality, the fascia which the deep extension of the parotid evaginates, is a broad sheath extending from the base of the

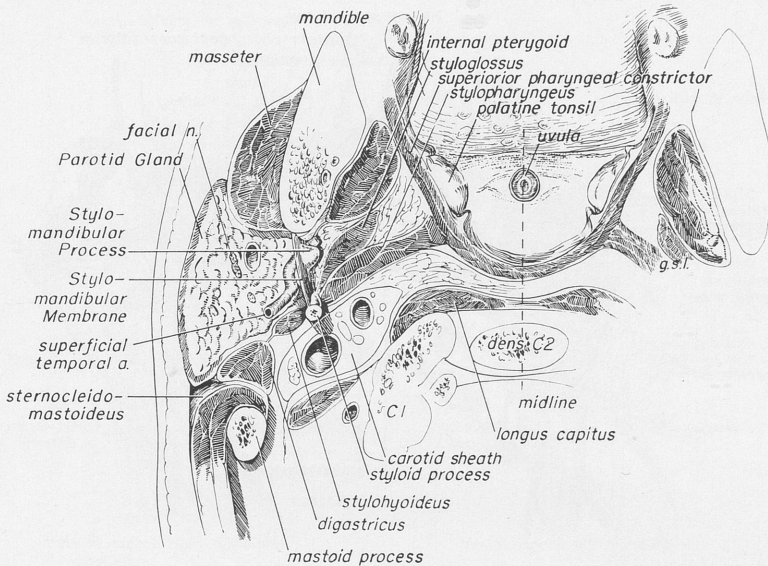


Fig. 1.—The anatomical relations of the parotid gland are shown in cross section. Note the stylomandibular fascia (membrane) with the stylo-mandibular extension of the parotid gland.

styloid process along its bony length to the posterior edge of the ascending ramus of the mandible. Scheldrup⁵ called this layer the stylomandibular membrane. Inferiorly, the condensation of these fascial fibers are designated as the stylomandibular ligament.

Patey and Thackray⁶ described the stylomandibular portion of the parotid gland and stated when tumors arise here they may pass deeply through the stylomandibular tunnel into the lateral pharyngeal space. When tumors pass through this tunnel they are constricted in their growth by fibers of the stylomandibular ligament and styloid process and hence are named dumbbell tumors by these authors (Fig. 2). They pointed out further the difficulties in removing these tumors in some patients and stated that since the stylomandibular ligament and styloid process both share in constricting these tumors, these structures must be dealt with by incision or fracture or both.

In addition to the stylomandibular projection of deep tumors, our surgical experiences and anatomical dissections suggest another deep prolongation of the parotid gland within which these tumors

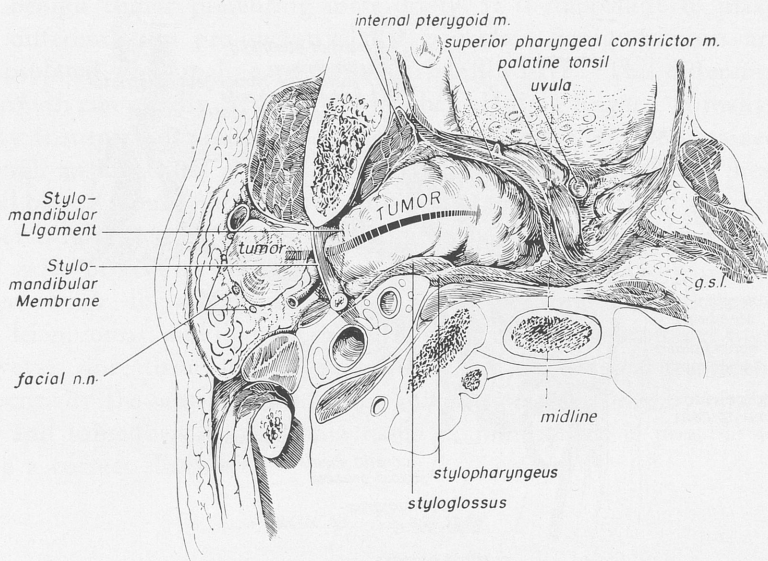


Fig. 2.—The dumbbell tumor described by Patey and Thackray is illustrated. The arrow depicts the extent of the tumor growth. Note constriction in the stylomandibular tunnel by the styloid process and stylomandibular ligament (condensation of fibers of the stylomandibular membrane). We report these as Group I cases.

might arise. In 1936, Scheldrup⁵ stated that abscesses in the lateral pharyngeal space could be approached anatomically through the stylomandibular membrane or through an area inferior to the stylomandibular ligament at the angle or horizontal ramus of the mandible. We have seen patients with abscesses in the lateral pharyngeal space where surgical incision and drainage was readily accomplished inferior to the horizontal ramus of the mandible directing the dissection inferior and medial to the stylomandibular ligament. Inferior to the stylomandibular ligament, the external maxillary artery pierces the fascia in its course from the carotid sheath. It comes in close relation to the substance of the gland and much superiorly divides into the superficial temporal and internal maxillary arteries. At a point where the external carotid artery pierces the parotid fascia below the stylomandibular ligament we have observed a medial extension of the retromandibular portion of the parotid gland. Since this is below the stylomandibular ligament we have designated this the

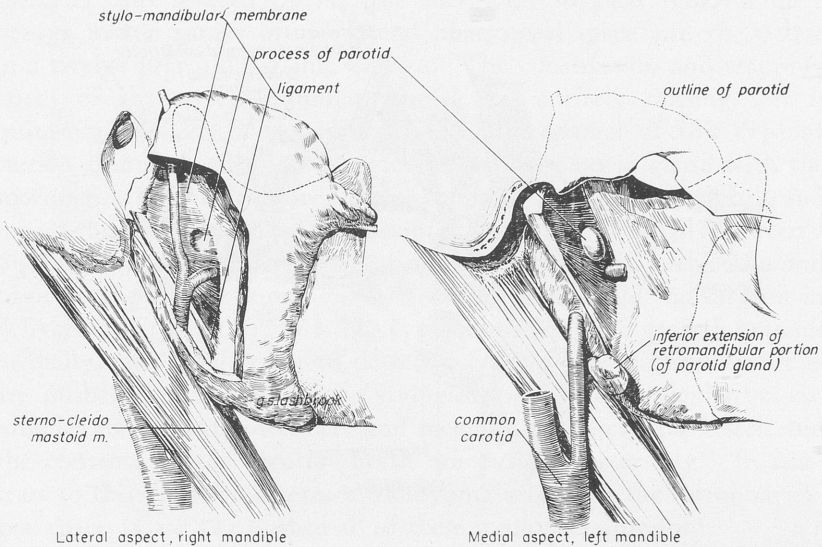


Fig. 3.—Note the inferior extension of the retromandibular portion of the parotid gland as it projects around and medial to the mandible inferior to the stylomandibular ligament. In this parotid gland extension we believe the round deep benign mixed tumors may originate. Such examples are presented in cases in Group II.

inferior extension of the retromandibular portion of the gland. Inferior in this instance refers to the stylomandibular ligament.

It evaginates the fascia inferior to the stylomandibular ligament and comes to lie anterior and medial to the lower posterior edge of the ascending ramus of the mandible (Fig. 3). In the normal parotid gland, this extension is small and, when present, lies in close proximity to the medial surface of the internal pterygoid muscle. Tumors arising in this inferior extension of the retromandibular portion would tend to expand medialward to the lateral pharyngeal space and palate. The parotid gland is separated from the submaxillary gland by a condensation of investing fascia called the angular tract. Morfit⁷ believes that tumors can grow medial to the ascending ramus of the mandible toward the pharynx and palate, and may also push downward arriving below the horizontal ramus of the mandible. Thompson⁸ also believes that round tumors may arise in the deep lobe stylomandibular portion of the parotid gland and present in the lateral

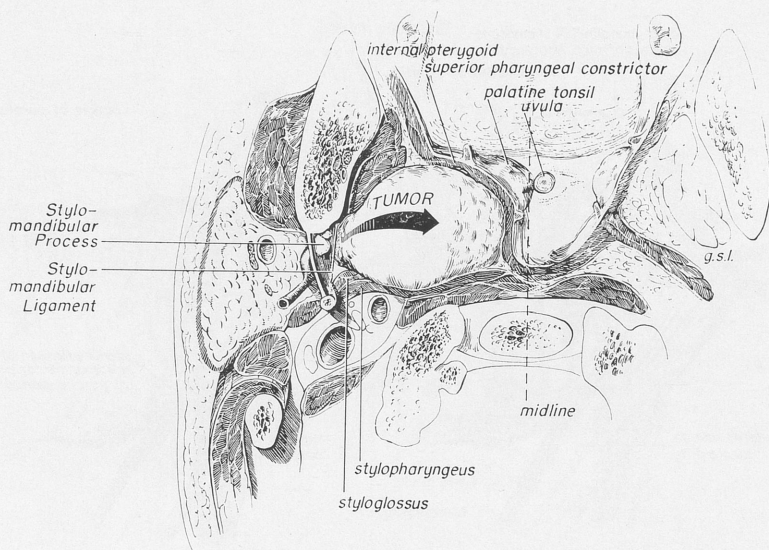


Fig. 4.—A round tumor is illustrated. Note that it originates below the stylomandibular ligament in the inferior portion of the parotid gland. The growth medially for both the round and the dumbbell tumors is the same after the stylomandibular ligament has been by-passed.

pharyngeal space as well as inferiorly near the angle and horizontal ramus of the mandible. Tumors which arise in the inferior extension of the retromandibular portion of the parotid gland are round in their growth as contrasted to the dumbbell shape of those tumors passing through the stylomandibular tunnel (Fig. 4). They present little or no external swelling as a rule. There may be only fullness below the angle and horizontal ramus of the mandible. There is no pre-tragal swelling as in the patients with dumbbell tumors. The clinical picture of the palatal and pharyngeal extensions may show no gross differences in either group. Upon bimanual palpation the examiner feels a sense of ballottement posterior to the angle and along the inferior ramus of the mandible. We believe, further, that in this latter group of patients, the facial nerve is not displaced as widely lateral as in patients with dumbbell tumors. It is possible to remove these tumors by exposing only the mandibular division of the VII nerve and not the whole trunk. The surgeon must occasionally remove the submaxillary gland in order for the excision to be successful. In one of the cases to be reported the inferior extension of the retromandibular

portion of the normal parotid gland was pulled through into the pharynx and severed during the intra-oral surgical removal of a benign mixed tumor of the lateral pharyngeal space and the palate in a twelve year old patient, Case. 6. The constriction and trapping effect of the stylomandibular tunnel was entirely missing. It is quite feasible also in the external surgical removal of this type of tumor to include adequate margins of normal parotid gland with the specimen. In this instance, both groups of patients (dumbbell and round tumors) the medial extension of the tumor is usually delivered by blunt finger dissection. The finger of an assistant within the oral cavity can push the tumor mass laterally and may aid the surgeon in delivering it. Further, in either type of tumor, once growth extends medially, the stylohyoid and posterior belly of the digastric muscles are pushed laterally while the styloglossus and the stylopharyngeus muscles are pushed posteriorly and medially. The carotid vessels and the contents of the carotid sheath are pushed posteriorly. In contrast to this growth pattern, a leiomyoma arising in the retropharyngeal space (Case 12) pushed all of these structures anterior.

Before going on with case presentations, it might be well to summarize our position regarding anatomy. The retromandibular portion of the parotid gland lies in a position posterior to the ascending ramus of the mandible. Gaughran² states that a stylomandibular lobe is present in 74% of cadavers examined. Patey and Thackray⁶ noted the origin of dumbbell tumors here. Marfit⁷ and Thomson⁸ believe also that round tumors arise in this deep lobe. We believe that round tumors may arise in the inferior extension of the retromandibular portion of the parotid gland and grow upward and medialward, to involve the lateral pharyngeal space and palate.

REPORT OF CASES

The twelve cases to be reported may be conveniently divided into the following groups.

Group I: Cases with tumors arising in the stylomandibular portion of the parotid gland showing medial growth to the pharynx and palate (dumbbell tumors).

Group II: Cases of deep mixed tumors of the parotid gland arising in the inferior extension of the retromandibular portion of the gland inferior to the stylomandibular ligament and growing medial and upward to the pharynx and palate.

Group III: Lateral pharyngeal space benign mixed tumors arising in a minor salivary gland (no connection to the parotid gland).

Group IV: Leiomyoma of the retropharyngeal space.

GROUP I

Cases with tumors arising in the stylomandibular portion of the parotid gland showing growth to the pharynx and palate (dumbbell tumors).

CASE 1. E.B., a white female, aged 40, noted a slow growing mass over the left parotid area anterior and inferior to the tragus of the ear for a period of twenty years. It was painless and non-tender. Upon examination of the patient at the University Hospital in 1949, a firm 4 x 3 centimeter mass was noted overlying the angle and ascending ramus of the left side of the mandible. There was no facial nerve involvement. A large mass protruded into the soft palate pushing the uvula and tonsil downward toward the patient's right. This mass was easily palpated by bimanual examination and was found to be in continuity. The patient was operated upon through an external incision. Since the tumor was large, it was divided; the outer half being removed and the inner extension was shelled out of its capsule. The patient's convalescence was uneventful. During the next six years, the patient developed a recurrence of the palatal portion of the tumor. In March of 1956, the mass and three small skin nodules were removed by external and intra-oral combined surgical approaches. Again in 1957 and 1959, the patient underwent surgical operation for local recurrence of the tumor. All surgical specimens were reported microscopically as benign mixed tumors. Recent follow-up examination showed the patient to be in good health without evidence of recurrent tumor.

CASE 2. L.C., a white male, aged 52, was examined at the University Hospital in the fall of 1959. He had noted a swelling in the right side of his face in the region of the parotid gland for seven years. It was painless. Facial nerve function was normal. Examination showed that there was a 1 x 1 centimeter mass anteriorly and slightly inferior to the tragus of the right ear. There was a large palate mass which pushed the uvula and tonsil to the patient's left. In December, 1959, the patient was operated upon through an external incision. In order to deliver the deep pterygomaxillary, lateral pharyngeal, and palate extension of the tumor, the lower fibers of the facial nerve were



Fig. 5.—Case 5. Note mass in area of right parotid gland anterior and inferior to the tragus of the ear. Scar of operation performed 7 years previously elsewhere is also shown. External masses in this area were present in all five patients of Group I.



Fig. 6.—Case 5. The palate mass was in direct continuity with the external mass as determined by bimanual palpation and later at surgical excision.

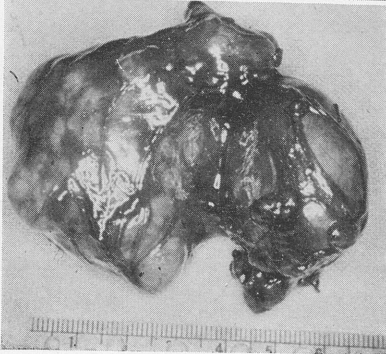


Fig. 7.—Case 5. Large dumbbell tumor showing constriction from the stylomandibular ligament and styloid process. The styloid process was fractured in order to deliver this tumor.

severed. The patient's convalescence was uneventful. Microscopic tissue examination showed the tumor to be a mixed benign tumor of the parotid gland. Follow-up examination showed that there was no evidence of recurrence of the tumor but the patient complained of a Frey syndrome.

CASE 3. R.G., a white female, aged 27, was examined at the University Hospital in the summer of 1956. Examination showed a 3 x 3 centimeter firm, non-tender mass at the angle and ascending ramus of the mandible on the right side. The palate and uvula were displaced to the left. Bimanual palpation showed the two areas to be involved by the same mass. The patient was operated upon in July, 1956 through external and intra-oral approaches. Due to the large size of the tumor, its contents had to be partially evacuated before the remaining mass and capsule could be delivered externally. The microscopic pathological report showed benign mixed tumor of the parotid gland. The patient's postoperative course was uneventful. She was lost to follow-up examination.

CASE 4. E.L., a white female, aged 56, was first examined in the University Hospital in 1956. In the right parotid gland area, just anterior and inferior to the tragus of the ear, there was a 1.5 x 2 centimeter firm, non-tender mass. The uvula and right tonsil were pushed to the left by the palate mass. Bimanual examination showed a single mass. The tumor was removed through an external approach. The capsule ruptured during removal. Microscopic tissue examination showed a benign mixed tumor arising in the parotid gland. Convalescence was uneventful. Recent check-up examination showed the patient to be in good health.

CASE 5. E.C., a white male, aged 69, was first examined at the University Hospital in June, 1962. Seven years previously an attempt had been made elsewhere to remove a mass from the right parotid gland. Within a month after this surgery, the patient noticed the mass still present. In June, 1962, the patient presented a parotid mass anterior to the tragus of the right ear (Fig. 5). In addition, a large right palatal mass was noted (Fig. 6). In July, 1962, the mass was removed through an external incision. In order to deliver the mass, the styloid process was fractured (Fig. 7). The superficial portion of the parotid gland was removed. Microscopic tissue examination showed a benign mixed tumor arising in the parotid gland. Convalescence was entirely uneventful.

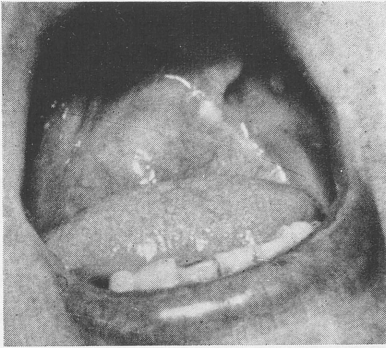


Fig. 8.—Case 7. This patient presented little or no external mass although there was a large mass in the pharynx and palate. It could be palpated beneath the horizontal ramus of the mandible on the left side bimanually.

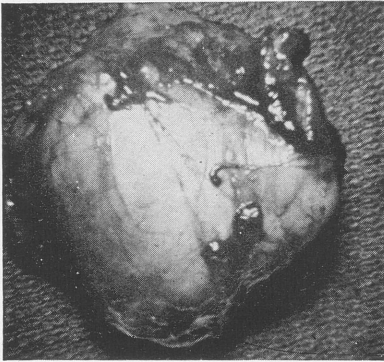


Fig. 9.—Case 7. Note the size of this round tumor. There are no areas of constriction as in the dumbbell tumors.

GROUP II

Cases of deep mixed tumor of the parotid gland arising in the inferior extension of the retromandibular portion of the gland inferior to the stylomandibular ligament and growing medial and upward to the pharynx and palate.

CASE 6. A white male, aged 12, was examined in June, 1956. On routine physical examination, a palatal mass occurring on the left side of the palate had been discovered one month previously. An attempted incision and drainage of this mass had been performed elsewhere. There was little or no external swelling. Facial nerve function was normal. A large tumor could be outlined by bimanual palpation. At St. Luke's Hospital in San Francisco in July, 1956, the tumor was removed through an intra-oral approach. It ruptured during its removal. It is significant to point out that in this case, the normal inferior extension of the retromandibular portion of the paro-

tid gland was pulled into the intra-oral wound and severed a distance away from the capsule of the parotid tumor. A tracheotomy was performed. Convalescence was uneventful. In the course of the next five years, the patient developed a recurrence and in March, 1961, again in St. Luke's Hospital in San Francisco, a 2 x 3 centimeter firm mass was removed from the lateral pharyngeal space through an external surgical approach. The specimens from both operations were reported as benign mixed tumors arising in the parotid gland. At the second operation, in addition to removing the recurrent tumor, the outer portion of the parotid gland was also excised. Convalescence was uneventful. Follow-up one year ago showed the patient to be in good health.

CASE 7. E.C., a white female, aged 72, was examined in March, 1961 at St. Luke's Hospital in San Francisco. For six years the patient had noticed an increased difficulty in swallowing and talking. There was a large palatal swelling extending into the pharynx (Fig. 8). There was little or no external swelling but this large firm mass could be palpated by bimanual examination. Ballottement was at the angle and along the horizontal ramus of the mandible. The patient's voice was typically "hot potato" in character. In March of 1961, the tumor was removed through an external surgical approach. A large round tumor was shelled out of its deep bed (Fig. 9). A generous portion of the inferior tail of the parotid gland was removed. Wide surgical exposure was obtained by removing the submandibular gland. Only the mandibular division of the VII nerve was identified since the tumor mass extended from below upward and medial to the stylo-mandibular ligament. The mass was not ruptured. The specimen was reported as benign mixed tumor arising in the parotid gland. A tracheotomy was performed. Postoperative convalescence was uneventful.

CASE 8. G.C., a white female, aged 62, had a benign mixed tumor of the lateral pharyngeal space removed 28 years previously through an intra-oral incision. A conduction hearing loss in the right ear was noted during all these years. In March, 1962, she noted a mass at the angle on the right side of the horizontal ramus of the mandible which was non-tender and non-painful. Upon examination at the University Hospital in April, 1962, bimanual examination showed a deep mass within the lateral pharyngeal space of approximately 3 x 3 centimeters in diameter. There was scarring of the side of the pharynx and torus of the right eustachian tube. The right



Fig. 10.—Case 8. Note the recurrent round tumor exposed in the lateral pharyngeal space. The incision must be extensive for adequate excision of normal parotid gland, exposure of the facial nerve and the carotid vessels and removal of the tumor.

tympanic membrane was thick, scarred, and retracted. Pure tone monaural audiometric studies showed a conduction hearing loss. The mass was excised through an external incision in May, 1962 (Fig. 10). Pathological report was again that of benign mixed tumor of the parotid gland. Postoperative convalescence was uneventful.

CASE 9. D.W., a white female, aged 30, was first seen at the University Hospital in November, 1959. For ten years she had noted a right parotid gland swelling at the angle of the mandible. It was firm, non-tender and approximately 5 centimeters in diameter. There was no visible palatal extension. However, a large deep extension of the tumor could be palpated bimanually medial to the angle and horizontal ramus of the mandible. There was a sense of ballottement in this area. In December, 1955, the tumor was excised from an external incision. In order to remove the tumor, the outer portion of the mass was incised and the portion of the tumor in the lateral pharyngeal space was then delivered as a separate specimen. Micro-

scopic tissue examination showed a benign mixed tumor arising from the parotid gland. Convalescence was uneventful. Recent follow-up examination showed no evidence of recurrence of the tumor and the patient appeared to be in good health.

CASE 10. B., a white male, aged 52, was first seen in November, 1948 at the University Hospital. Upon examination, there was no external mass over the parotid gland. The patient stated that a few months previously an attempt had been made to remove a tumor at the angle of the right side of the mandible elsewhere. In November, 1948, a large palatal tumor was excised through an external incision. During the course of removal the capsule ruptured. Ten years later, there was a recurrence of a mass deep within the lateral pharyngeal space as noted by bimanual palpation. This was re-excised in 1958 through an external incision. Microscopic tissue examination of all specimens showed a benign mixed tumor of the parotid gland. Recent follow-up examination showed no evidence of recurrence and the patient appeared to be in good health.

GROUP III

Lateral pharyngeal space, benign mixed tumor arising in a minor salivary gland (no connection with the parotid gland).

CASE 11. J.M., a white female, aged 37, was first examined in December, 1960. Upon routine examination two months prior, her local physician had found a large mass in the right lateral pharyngeal space which extended upward near the palate and into the nasopharynx. In December of 1960, at St. Luke's Hospital in San Francisco, a tumor mass was removed through an intra-oral approach. During the course of removal, the tumor was ruptured. In spite of this, the capsule was excised adequately. Pathological report of the specimen was a mixed salivary gland tumor of lesser salivary gland origin. It is pointed out that in this case the normal tail of the parotid gland was not attached to this tumor as was found in Case 6. Convalescence was uneventful and recent examination showed the patient to be entirely symptom free.

GROUP IV

Leiomyoma of the retropharyngeal space.

CASE 12. L.P., a white female, aged 15, was first seen at the University Hospital in July, 1962. Two months previously in rou-

tine examination, her physician had noted a swelling in the lateral pharyngeal space and palate on the left side. Examination confirmed the swelling in these areas. A mass could be palpated at the angle of the mandible and along the horizontal ramus. There was a sense of ballottement by bimanual palpation. The mass was firm and non-tender. There was no evidence of dysfunction of cranial nerves. In July of 1962, the mass was excised through an external surgical approach. The mass was found to be entirely free of the parotid gland. It pushed the stylohyoid and posterior bellies of the digastric muscles laterally. The carotid sheath and other styloid process muscles were pushed anteriorly. The mass lay on the transverse cervical processes and bodies of the vertebrae. It was impinged behind the styloid process. It extended to the nasopharynx, base of the skull and the retropharyngeal space. It was removed without incident. The facial nerve was exposed immediately at the beginning of the operation so that it could be protected at all times. Convalescence was uneventful. The patient has remained in good health. Paralysis of the left side of the tongue has been noted postoperatively. The microscopic pathological tissue examination showed leiomyoma.

COMMENT ON CASES

The first ten cases reported showed benign mixed tumors arising in the stylomandibular lobe and inferior extension of the retromandibular portion of the parotid gland. Case 11 was found to have a benign mixed tumor arising in the lesser salivary gland. Case 12 showed a leiomyoma.

It is interesting to point out that in Case 12, the diagnosis of benign mixed tumor was made pre-operatively. This case was included in this series to point out the difficulties in differential diagnosis and to note deep growth into the retropharyngeal space posterior to the carotid sheath and styloid process structures. The wide anterior displacement of the carotid sheath structures was striking in this case and even more so was adaptability. Benign mixed tumors arising in any portion of the parotid gland do not involve this fascial space. Like patients with deep parotid gland tumors, the facial nerve had to be exposed initially before the tumor could be removed. Partial excision of the parotid gland was accomplished only for adequate removal of the tumor and exposure of the facial nerve.

Case 11 was included to demonstrate that tumors of the lateral pharyngeal space and palate may occur entirely unassociated with the parotid gland.

Tumors arising in the stylomandibular lobe of the parotid gland must be considered for external operation in most instances (Group I cases). For successful removal, one must completely expose the main trunk and branches of the facial nerve as the first step in operation. The carotid vessels should be next identified and a hernia tape placed around them for control of possible later hemorrhage. In order to release the deep extension of these tumors, the constrictions at the stylomandibular tunnel must be released by fracturing the styloid process or by severing the stylomandibular ligament or both. Rupture of the capsule can best be avoided by these measures. It has not been necessary to resect or to split the mandible in any case. However, in some cases, smaller branches of the facial nerve have had to be sacrificed due to the size of the tumor. In other cases, the tumor had to be removed piecemeal or its contents evacuated before the capsule of the tumor could be removed. This should be avoided if at all possible. The intra-oral surgical route may be used occasionally in conjunction with external surgical approach in patients with extremely large tumors.

In the Group II cases, where tumors arise from the inferior extension of the retromandibular portion of the parotid gland, removal is accomplished most successfully by an external approach also. All patients may not require complete dissection of the facial nerve since these tumors are low and are placed medial to the main trunk of the nerve. Exposure of the mandibular branch only permits adequate removal of the tumor in most cases and further allows removal of a generous margin of normal parotid gland as well. The deep part of the tumor may be removed more readily in some cases after removal of the submaxillary gland. The carotid vessels should also be identified early for safety.

It is pointed out that in no instance did any of these twelve patients present permanent postoperative difficulty with facial nerve function except in those two patients where small branches of the facial nerve were severed intentionally. To date, the eleven cases of mixed tumors showed recurrences in five. If these five are analyzed, it is noted that three had inadequate excision initially elsewhere. Subsequent adequate excision, although repeated in some cases, did

apparently arrest the disease. In the other two cases where initial therapy was considered adequate, one case showed recurrence at five years and one case at the end of 28 years. It is important to point out that during follow-up examinations the physician should include bimanual palpation or otherwise early recurrent tumor masses in the lateral pharyngeal space will be overlooked.

The diagnosis pre-operatively was incorrect in Case 12 (leiomyoma). In none of the remaining eleven cases was malignancy suspected pre-operatively, hence no case underwent pre-operative biopsy. To date in several patients, in spite of recurrent tumors necessitating recurrent excisions, the pathological diagnoses in all eleven have been benign mixed tumors without evidence of malignancy.

A few comments might be made concerning all groups of patients. Nasal tracheal intubation should be carried out in all patients undergoing parotid gland surgery since this allows the mandible to be pulled forward thus giving the surgeon more space between the mastoid tip and the ascending ramus of the mandible in which to operate. Further, the patient's mouth should be made accessible as the patient is draped so that the surgeon's assistant can later place a finger in the mouth and push the palatal extension of the tumor into the external wound, if need be. A combined intra-oral and external surgical approach is probably not necessary in most patients. Resection or division of the mandible has been found to be unnecessary. A generous portion of the normal parotid gland should be removed in all cases approached externally. This latter statement implies that facial nerve dissection is dependent solely upon individualization of cases. The external and internal carotid arteries should be isolated and hernia tape placed about them, but not tied. This is to control excessive and unexpected hemorrhages during the course of excision of the tumors. The external carotid artery may have to be ligated in some cases. Every attempt should be made to deliver the tumors without rupturing the capsule. As pointed out, this may not be possible in some instances when large tumors are being excised. If the capsule does rupture and tumor cells are spilled into the wound, the wound should be irrigated with copious amounts of normal saline solution. Postoperative complications have been lessened by the use of antibiotic therapy, particularly in those patients where an intra-oral incision is used or when intra-oral and external incisions have been combined. In all external operations the deep tissues of the lateral pharyngeal space should be drained for at least 24 to 48 hours.

SUMMARY

Ten cases of benign mixed tumor of the parotid gland have been presented. In addition, one patient with a mixed tumor arising in a lesser salivary gland and one case of leiomyoma of the retropharyngeal space were reported. Five cases of mixed tumor arose in the stylo-mandibular lobe of the parotid gland and entered the lateral pharyngeal space through the stylomandibular tunnel. In five other cases, the tumor apparently arose in the inferior extension of the retro-mandibular portion of the parotid gland and entered the lateral pharyngeal space below the stylomandibular ligament and grew upward and medially. "Inferior" as used in this discussion refers to the stylomandibular ligament.

CONCLUSIONS

Surgical removal of these deep tumors should be planned along definite lines as outlined above. An external surgical approach is recommended in most cases.

OUT-PATIENTS DEPT.

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DISCUSSION

DR. JEROME A. HILGER: Mixed tumors in the parapharyngeal space may arise from gland structures other than the parotid. However, the presence of a large parapharyngeal mass with physical characteristics consonant with mixed tumor should be suspected of parotid origin and surgical philosophy be accordingly oriented. Doctor Work's well-chosen subject and excellent presentation makes this contention clear.

In the interests of time conservation and clarity, several discussion points can best be made through graphic illustration:

Fig. 1 When a tumor arises in the depth of the deep parotid lobe (position 1) it may avail itself of the anatomic ease of parapharyngeal space intrusion with presentation behind the tonsil and eventual oropharyngeal distortion. Its excision can most readily be effected through intrapharyngeal approach. Its deep terminus will be delineated by the medial and anterior aspect of the styloid process. Margination is not further favored by external removal of the parotid gland itself if an intact capsule is retained. Usually the capsular contact area with the deep aspect of the gland is much smaller than the circumferential contact with the enveloping "vital" structures of the parapharyngeal boundaries. We have removed "round" tumors of this origin based at the root of the styloid and obviously arising from the stylo-mandibular prolongation of the parotid as Doctor Work terms it. Therefore, not all "round" tumors arise retromandibularly and inferiorly. I would suggest that shape is not a valid differential guide to origin.

If violation of the tumor capsule occurs in this deep oropharyngeal dissection it is usually the consequence of "isthmus origination" (position 2) of the mixed tumor nidus. Growth medial and lateral from this point of origin results in the "dumbbell" tumor. Even if the lateral extension is not yet large, it is axiomatic the tumor will remanifest itself if the surgery is not completed by the external phase—total external parotidectomy—forthwith.

When the nidus of mixed tumor originates in position 3, it has been our feeling it rarely crowds medialward and "dumbbells" because lateral protrusion is the easier route.

Two of the points in surgical technique presented by Doctor Work warrant accent:

1) Intranasal intratracheal intubation through the opposite nasal space affords a clearer intraoral approach. In addition, a valuable gain in dissection space in the tight angle between vertical mandibular ramus and skull base is obtained in case supplementary external approach for deep lobectomy is found necessary. The distance gained through mouth closure and jaw protrusion eases the dissection posterior and deep to the vertical ramus. This anesthetic technique should be used in all excisional parotid surgery.

2) Division of the broad fascial band designated "stylomandibular ligament" frees the jaw angle for further protrusion. In addition the "dumbbell" tumor of isthmus origin (position 2) presenting as a continuous intrapharyngeal and external mass is "wasp-waisted" due to the indenting resistance of this ligament. When the ligamentous band is divided inferior to the mass it provides space for the deep lobule of the tumor which can be freed and delivered externally. Occasionally with unusual size of the deep component the mass will have to be delivered in two halves by fracture of the tumor "isthmus." This is undesirable but occasionally unavoidable.

I admired and learned from Doctor Work's presentation.

DR. JOHN F. DALY: We have had three experiences with this group of tumors. Two were the true dumbbell shaped tumors. The shape is rather important because it plays an important part in the surgical management, as well as the diagnosis. The large mass presenting on both sides of the stylomandibular ligament creates technical difficulties in excising it. It is almost impossible to deliver the tumor by either an external or an internal approach alone. By combining both approaches one can avoid dividing the tumor and the danger of seeding the wound.

In our first case we split the mandible in order to deliver the tumor. This can be avoided simply by dividing the stylomandibular ligament and retracting the mandible with a heavy hook.

The third case was interesting because it was rather small. It presented in the plug of tissue described by Thackeray and Petti. This is protrusion of the deep portion of the salivary gland into the tunnel. The patient was a doctor who had some kind of vague symptoms in this region and by palpation this covered the tumor when it was still small. A pharyngeal approach was made at another institution first and, because of its relatively small size, it was missed. We were able to remove it trans-orally. This tumor would fit into the second category of Dr. Work's.

These tumors are unusual tumors. Dr. Work's classification of these tumors is a very valuable contribution.

The chances of developing a tumor in this portion of the salivary gland are probably one out of a hundred, which adds to the significance of Dr. Work's report.

DR. WORK (closing): I wish to thank Dr. Hilger and Dr. Daly. The only thing I wish to state is that as otorhinolaryngologists it is hoped that our interests will continue in patients presenting such tumors.

NASAL REFLEX ARCS AND THEIR INFLUENCE IN NASAL OBSTRUCTION

A. C. FURSTENBERG, M.D.

ANN ARBOR, MICH.

I take it for granted that in this day of modern medicine we expect the vast majority of infants to be born with noses which function normally. Nevertheless we are frequently consulted by parents who bring their babies to us because of obvious nasal obstruction with its associated feeding problems and symptoms referable to the ears, nose and throat. In the majority of cases we have only to remove a mass of adenoids to restore free breathing and to assure the parents that the nose is in no way compromised by any of the congenital lesions and anomalies which have attracted their attention in current publications.

We have a deep compassion for the youngster who is born with some serious congenital defect or lesions of teratological origin, but these are not often encountered except in some of our large clinics where many patients are seen daily. The optimistic point of view I wish to emphasize is that these disorders are relatively rare when we take into consideration the rapid expansion of our population by the birth of normal children.

The challenging problems today pertaining to the respiratory function of the nose are comparatively benign, although numerous and frequently encountered throughout the growth and development of the child to adulthood and beyond for the span of life. What are these problems? How can they be solved? What can be done in a program of rehabilitation?

Everyone here is thoroughly familiar with the causes of nasal obstruction and the measures to be employed for their control or cure. Perhaps you will permit me, however, to emphasize one of the important etiological factors in nasal obstruction, namely, congestion

of the vascular bed of the nose. Under today's pressures it is not difficult to overlook the influences which are often responsible for this common disorder.

We occasionally interview a patient who is experiencing some social, economic, moral or religious conflict which has produced emotional states reflected to the nasal mucous membranes. An understanding of his difficulties requires an insight into the sociological and humanitarian aspects of medicine. The doctor must possess a knowledge of human conduct and behavior, a recognition of mental and physical reactions to emotional influences and an appreciation of the variable human capacities for surmounting these obstacles if he is going to recognize and treat properly patients who are the victims of this disorder. In short, this patient's nasal obstruction is on the basis of a neurosis, and psychiatric consultation and care are obviously needed.

Infections, both acute and chronic, are responsible in many patients for a congestion of the nasal mucosa. The physiologic effects of hot or cold air and exposure to cold water while swimming will frequently produce a sudden nasal obstruction. The nose is similarly influenced in some individuals by gravity. The patient tells us that when he retires at bedtime and lies on the right side of his face the right nasal passage becomes obstructed. If he turns to the left side the reverse is true. In this position, however, another factor probably comes into play, namely, the warmth of the pillow transmitted to his face and nose.

Endocrine disturbances must also be given some consideration in the etiology of nasal obstruction. We have all seen the vasomotor rhinitis of pregnancy which frequently occurs early and lasts for approximately two weeks after the infant has been born. Myxedema is occasionally a source of trouble. One of our distinguished faculty members recently consulted me on account of a marked nasal obstruction which had been a great source of annoyance for approximately two months. A brief examination showed an extensive congestion of the vascular bed of the nose together with the characteristic signs and symptoms of myxedema. When the iodides which he had been taking for asthma were withdrawn the nasal mucosa rapidly returned to normal.

I need only mention the adverse influences of allergy upon the nasal mucosa. We are all familiar with the diagnostic criteria of this

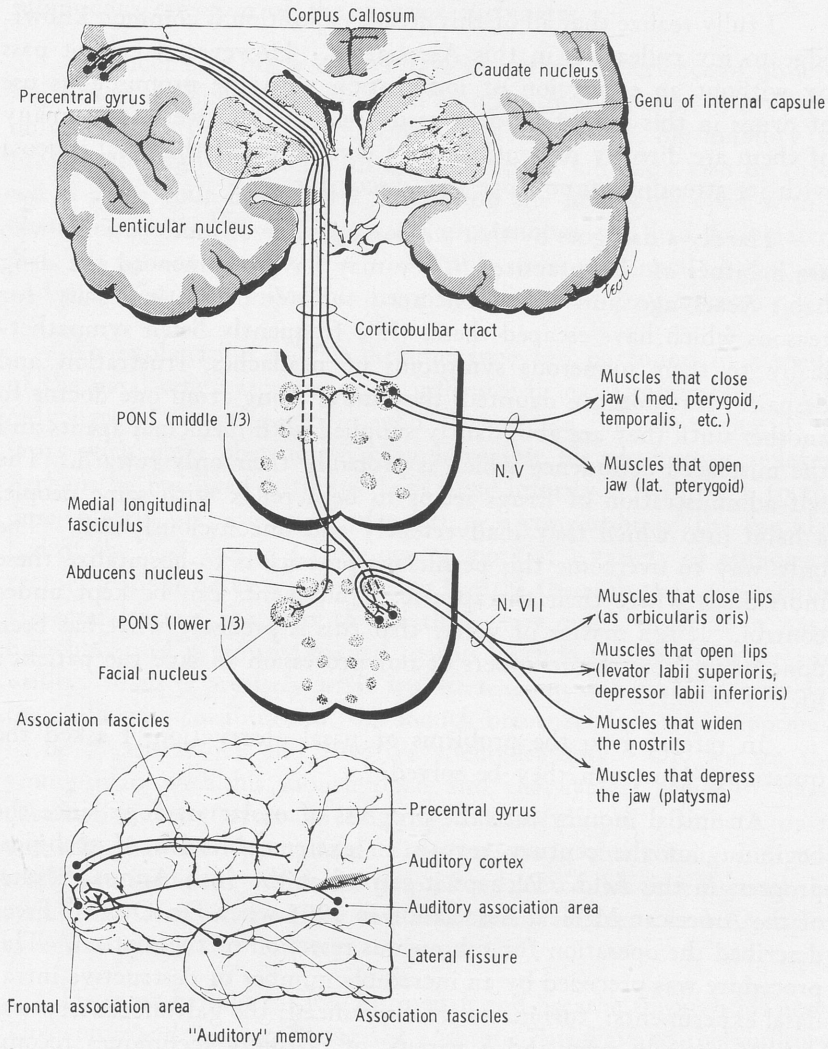


Fig. 1.

entity and the importance of its recognition and adequate treatment in the practice of otolaryngology.

I fully realize that all of this dreary exposition is common knowledge to my colleagues in this Association. However, I cannot pass by without an expression of indignation with the promiscuous use of drugs in this day of modern medicine. As you well know many of them are directly responsible for a congestion of the nasal mucosa with its attending annoyance and discomfort.

Hardly a day goes by that we don't see one or more patients who are habitués of this practice. They may have commenced the drug habit years ago and have continued self-administration daily for reasons which have escaped them. We frequently listen sympathetically to their numerous symptoms of headaches, frustration and despair. But nothing daunted, they move along from one doctor to another until they are abundantly supplied with medicinal agents and the misplaced confidence which is probably their only reward. The self-administration of drugs seems to be a reflex with some people, a habit into which they inadvertently and unconsciously fall. The only way to overcome this pernicious practice is to hospitalize these individuals where their therapeutic requirements can be kept under control. It is a matter of verity that this is precisely what has been done many times by members of our profession to save the patient's life.

In referring to the problems of nasal obstruction, I asked the question, "How can they be solved?"

An initial inquiry into the progress of otolaryngology since the beginning of the century reveals an extreme breadth of technical progress in this field. Perhaps it started at the 56th Annual Session of the American Medical Association in 1905 when Dr. Otto T. Freer described the operation for submucous resection of the septum. This procedure was preceded by an incredible number of destructive intranasal experiments: turbinates were sacrificed; the galvanocautery was indiscriminately used and a variety of crushing techniques recommended to straighten the septum. The pervasive popularity and use of this operation seems to have been established shortly after this date.

Since the turn of the century, many from the ranks of our specialty have established practicable surgical procedures for the relief of nasal obstruction. Despite some early grievous results, I think it can be said now that we have surgical methods that will successfully

relieve the patient of annoying and troublesome difficulty in breathing through the nose. The one most needed in my experience is the submucous resection of the inferior turbinates.

I wonder how many of you have shared my experience of obtaining a most satisfactory result from an operation for nasal obstruction only to have the patient continue to complain of his inability to breathe through the nose. In a check-up examination two or three weeks after your best efforts you found the nose wide open with a breathing space on both sides much greater than you had anticipated. Yet, the patient remained the implacable sufferer of nasal obstruction and may also have raised his voice in ridicule of your operation.

An explanation of this circumstance can be found in a study of the nasal reflex arcs and their influence in nasal respiration. It is only natural that the patient who has suffered for many months or years with nasal obstruction may have lost his respiratory reflexes. Perhaps he has lived without them since infancy. An injury to the nose in early life, a mass of adenoids, or a congestion of the vascular bed of the nose acquired from one or more of a variety of etiological factors made it necessary for him to breathe through the open mouth. He soon learned that mouth breathing was much easier than nasal breathing and promptly turned to this method for convenience and utility. This is precisely what we often see in our practice, namely, the child who continues to be a mouth breather after what appeared to be a satisfactory and effective adenoidectomy. Or, we see the young man with his mouth open and the typical facies of nasal obstruction long after an entirely successful and adequate nasal operation has been performed. The patient in this condition is not really handicapped. With the help of his doctor, his parents, and the acquisition of a little vanity, his respiratory reflexes can be restored to active usage.

He must be told to close his mouth and breathe through his nose. This command is relayed over the auditory pathways to the auditory cortex (bilaterally).

From the auditory (receptive) cortex, impulses are projected to the auditory association cortex where the command becomes meaningful, and is understood by the patient. This relay is to the auditory association cortex in the dominant or left hemisphere in right-handed people. (This is the hemisphere shown in the diagram.)

From the auditory association cortex, the impulses are relayed to other cortical areas—two of such relays are shown: (1) to the more rostral part of the temporal lobe where the impulses arouse the memory of past performances, namely, the memory of having been able at sometime to breathe satisfactorily through the nose, and (2) to the frontal association cortex. The auditory ("memory") cortex discharges also to the frontal cortex.

In the frontal association cortex, the type of response (the conscious response) is determined and association fascicles discharge, then, to the efferent path, namely, the corticobulbar tract.

The corticobulbar tract discharges (in part) directly (and in part through intercalated neurons which lie near the motor facial and trigeminal nuclei) to the appropriate facial and trigeminal motor neurons. The impulses carried over the corticobulbar fibers are partly excitatory and partly inhibitory. The excitatory impulses discharge to the motor neurons supplying the muscles which close the mouth and widen the nostrils. The fibers which are inhibitory discharge to the motor neurons which open the mouth and the jaw. Their inhibitory action, therefore, tends to prevent mouth breathing.

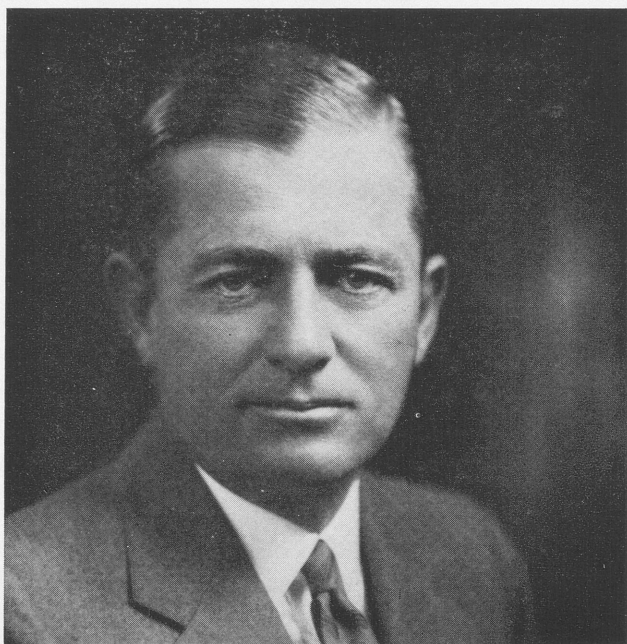
SUMMARY

1. Congestion of the vascular bed of the nose is a common etiological factor in the production of nasal obstruction. A submucous resection of the septum is not adequate for its relief. When combined, however, with a submucous resection of the inferior turbinates, the result is usually most satisfactory.

2. The respiratory reflex arcs are exceedingly important to nasal respiration. They can be activated for normal function by training and constant use.

ST. JOSEPH'S MERCY HOSPITAL

Memorials



WESLEY CREVLING BOWERS

WESLEY CREVLING BOWERS

1880 - 1963

Dr. Wesley Bowers, an Emeritus Fellow of the American Laryngological Association, was born in Belvedere, Warren County, New Jersey on November 27, 1880, and died on June 26, 1963. He received his early education in local schools graduating from the following institutions: Centenary Collegiate Institute in Hackettstown, New Jersey in 1900, Princeton University in 1904, the College of Physicians and Surgeons, Columbia University in 1908. He was awarded a surgical internship at St. Luke's Hospital in New York which included training in pathology and associated training at Sloan Hospital for Women.

On completion of his internship he spent some time under Dr. William R. Janeway and opened an office for private practice but soon decided on specialization in Oto-laryngology. He became associated with Edward Bradford Dench taking appointments in his clinics at the New York Eye and Ear Infirmary and at St. Luke's Hospital. At this period he also worked for a time with Dr. Edward C. Perkins.

He held clinical teaching appointments for a time at the College of Physicians and Surgeons and later at New York University Medical School but his real interest was in the resident training programs at St. Luke's and Bellevue Hospitals. He had much to offer in his teaching capacity in the wards and in the operating room.

He was a keen diagnostician and always alert for complications. He had a remarkable capacity for imparting to residents his judicious management of difficult problems. These qualities, together with his technical ability, made him the consummate otologist.

Dr. Bowers believed and insisted that dressings and treatments both pre- and post-operative could and should be carried out painlessly.

He was connected with St. Luke's Hospital for the fifty-six years of his professional life and he rose by steps from Ward Junior Interne to Consultant. He was admired and respected by the entire

hospital family. A largely attended memorial service was held in St. Luke's Hospital chapel and a fine portrait was hung prominently in the staff room, a tribute from the men in his department.

His publications were excellent in quality dealing principally with otological problems and having to do with temporal bone infection and its complications. With the advent of chemo-therapy he wrote wisely and soundly on their administration and warned of their masking effects.

He held memberships in a number of scientific societies: the American College of Surgeons, the American Otological, the American Laryngological, the Triological, the New York Otological, the New York Oto-Laryngological were but a few. At one period he held the Presidential office in the American Otological and the New York Otological Society.

His diversions were golf, duck and quail shooting, with an occasional trip to the Scottish moors for grouse. He was adept at both sports and in his latter years his golf score was in the seventies.

His first marriage was to Gladys Seward in 1916. She died in 1957. Surviving are their two children, a son, Wesley, Jr., and a daughter, Mrs. McLain B. Smith and four grandchildren. In 1962 he married Miss Clare Boyd who survives him.

V.McC.

JOHN H. FOSTER, M.D.

1876 - 1962

The American Laryngological Association lost a distinguished and highly regarded fellow member on October 1, 1962, with the death of Dr. John H. Foster. Dr. Foster, 85 years of age, had retired from active practice in 1945, to enjoy the fruits of his life's work, his deeds were many and his interest were varied.

Dr. Foster was born on November 20, 1876, in the farming community of Nelsenville, Texas, in Austin County, to Dr. and Mrs. G. W. (Martha Hoskins) Foster. Like his father, he devoted his life to helping his fellowman but upon retirement he returned to farming and ranching at Eagle Lake, Texas. Two years before his death he and Mrs. Foster had returned to Houston, living in the Plaza Hotel.

Dr. Foster graduated cum laude with a master of arts degree from Southwestern University at Georgetown, Texas, in 1896. He received his doctor of medicine degree cum laude from the University of Texas Medical Branch in 1900, having interrupted his studies to enlist during the Spanish-American War. He served with the First Texas Cavalry as hospital steward. Upon receiving his medical degree, he went to the St. Paul's Sanatorium in Dallas to serve his internship. After completion, he became an assistant physician at the Austin State Hospital but resigned in 1904 to do further study. His thirst for knowledge led him to New York City where he became house surgeon on the otolaryngologic service of the Manhattan Eye, Ear, and Throat Hospital in 1905. Having fortified himself with the best in education and a great deal of practical experience, Dr. Foster returned to Houston to open his own office in 1906. However, it was in October 15, 1907, when he formed his greatest alliance, with marriage to Miss Anne Vineyard.

Not being the type of man to stand still and let an opportunity pass, Dr. Foster again added to his education by taking postgraduate work in the clinics of London and Vienna in the summer of 1913. After this he served as a lieutenant in the Navy during World War I. Behind him was the honor of serving as president of the Harris County



JOHN H. FOSTER

Medical Society in 1910. Still to come were multiple honors, such as being president of the South Texas District Medical Society and the Texas Ophthalmological and Otolaryngological Society; vice-president of the American Laryngological Association; chairman of the eye, ear, nose, and throat sections of the Texas Medical Association and the Southern Medical Association. Dr. Foster was a member of the American Medical Association, the American Rhinological and Otolological Society, the American College of Surgeons, and the American Broncho-Esophagological Association. He was certified by the American Board of Otolaryngology. He received an honorary membership in the Texas Medical Association in 1947, having served as its president, vice-president and councilor of the Ninth District previously. He helped organize the Houston Eye, Ear, and Throat Hospital and Clinic. He held the degree of Doctor of Science, which was conferred upon him by his alma mater, Southwestern University, in 1933.

Dr. Foster's civic duties were well demonstrated and rewarded by such offices as member of the board of directors of the Houston First Federal Savings and Loan Association, serving as chairman in 1951 and was honorary chairman when he died. He was director of the First National Bank of Eagle Lake, Texas, from 1945 to 1960. At the time of his death, he was a director of the Houston Farms Development Company and secretary-treasurer and director of the Lakeside Elevator and Grain Company. He was formerly consulting physician for Hermann Hospital, the Southern Pacific Lines, and the Missouri Pacific Railroad.

Dr. Foster's popular personality was shown by his charter memberships to the Houston Country Club, the River Oaks Country Club, the Houston Yacht Club, the Doctor's Club, and the Eagle Lake Rod and Gun Club. He was a Shriner and an Episcopalian.

He is survived by his wife, a daughter, Mrs. Gus E. Cranz, of Forth Worth; two sisters, a brother, and two grandsons. Dr. Foster set a high example in citizenship, achievement, and usefulness. His mark upon the world will never be surpassed but will be fondly and awesomely remembered for many years to come.

H.H.H.



GORDON FOLETTE HARKNESS

GORDON FOLETTE HARKNESS

1880 - 1962

Gordon F. Harkness was elected to Active Fellowship in the American Laryngological Association in 1933 and remained an Active Fellow until the time of his death. He had an unbroken record of attendance at all meetings from 1946 through 1962. Dr. Harkness served as Vice-President of the Association 1940-1941, as Treasurer 1948-1953 and as President 1953-1954. His Presidential Address given at the Boston meeting May 27, 1954, presents beautifully the philosophy that guided his life and which one hopes will guide the lives of all physicians.

Dr. Harkness devoted his time to private practice except for the time he spent in service in World War I as Captain in the Medical Corps of the U.S. Army.

A brief resume of his academic career is as follows:

B.S. State University of Iowa, 1900.

M.S. State University of Iowa, 1902.

M.D. State University of Iowa, 1902; Member of Alpha
Omega Alpha.

1902 Intern, Mercy Hospital, Davenport, Iowa.

1903 Graduate work, Manhattan Eye and Ear Hospital,
New York City.

Dr. Harkness was certified by the American Board of Otolaryngology in 1925 and in his later years was a member of the Board of Directors of the American Board of Otolaryngology.

Dr. Harkness gave generously of his time to help solve the problems of organized medicine. He was President of the Iowa State Medical Society 1933-1934 and was a member of the Executive Board of the State Blue Shield Organization. For his services, he was given the Award of Merit of the State Society in 1953.

Dr. Harkness served as Chairman of the A.M.A. Section on Laryngology, Otology and Rhinology 1941-1942 and was the Delegate of this Section to the House of Delegates of the American Medical Association from 1948 until the time of his death in 1962. His tactful personality made him a most effective representative of Otolaryngology on the national front.

Gordon was a member of numerous medical societies, including the Iowa Academy of Ophthalmology and Otolaryngology, the American Laryngological, Rhinological and Otological Society, the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons.

In 1904 Dr. Harkness married Mabel Ann Gunsolus. Mrs. Harkness died in 1953. The devotion of his two daughters, Mrs. Louise Stoeltig of Baltimore and Mrs. Mary Rheinstrom of New York City, enabled Gordon to continue as an effective member of the medical team.

His published articles show wide range of interest and competence in many phases of otolaryngology as well as interest and competence in the field of ophthalmology. Gordon was admired and respected by all who knew him, especially by those who were close enough to have him as a personal friend. Were there more Gordon Harknesses the "public image of doctors" would be better than it is today.

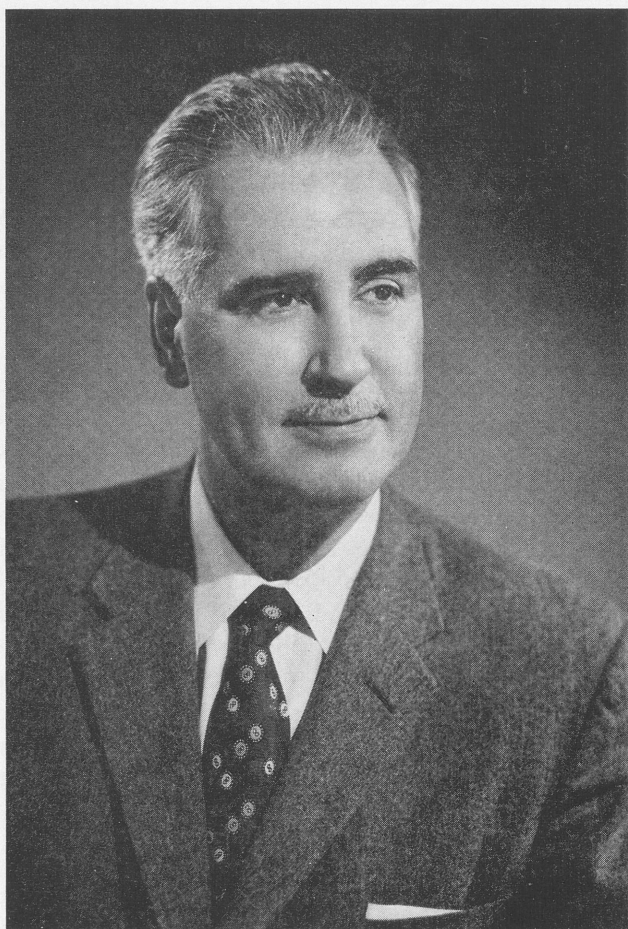
F.W.D.

KENNETH CLAYTON JOHNSTON, M.D.

1911 - 1963

Dr. Kenneth C. Johnston, Associate Professor of Bronchoesophagology in the Department of Otolaryngology of the University of Illinois, died on February 16, 1963. Dr. Johnston was born in Corinne, Saskatchewan, Canada; he received his undergraduate education there and his medical degree from the University of Manitoba. His early interest in diseases of the respiratory tract led to his appointment as Assistant Medical Director of St. Boniface Sanitorium adjacent to Winnipeg. To further his interest in diseases of the chest he moved to Chicago in 1947, specializing in bronchoesophagology and laryngeal surgery. He achieved national and international distinction for his work in these fields and held active or consulting positions on the staffs of the Research and Educational Hospitals of the University of Illinois, the Illinois Eye and Ear Infirmary, the Children's Memorial Hospital, Presbyterian-St. Luke's Hospital, St. Joseph's, Lake Forest, West Side Veterans Administrations and Chicago State Tuberculosis Hospitals. He contributed richly to the literature of his specialty and was a member of the American Laryngological Association, American Bronchoesophagological Association, the "Triological" and the Academy of Ophthalmology and Otolaryngology, and was a Fellow of the American College of Surgeons, the American College of Chest Physicians, and the Institute of Medicine of Chicago, to list but a few of his society affiliations.

Dr. Johnston felt strongly obligated to medicine and assumed the responsibilities of leadership in his quiet, competent, thorough manner. He was a member of the Chicago Laryngological and Otological Society, the Chicago Pediatric Society, and was on the Council of the Chicago Medical Society having been Secretary and President of its North Shore Branch. He was a Delegate to the Illinois House of Delegates, serving actively on many committees. His keen interest and participation in the clinical and educational programs in the medical school and the hospitals were felt and appreciated by every student, intern and resident on his service, to whom he gave so freely of his time.



KENNETH CLAYTON JOHNSTON

Dr. Johnston is survived by his widow, Ione Lattimore Johnston, and three sons, Kenneth, Robert and Ross. With them he participated in school and church activities. He was soft spoken, modest and had a fine sense of humor; he maintained a broad knowledge of his field and was accomplished in it. With his devotion to his family and patients, he exemplified all of those qualities associated with a respected physician and a kind, thoughtful person who achieved a position of distinction in his community.

P.H.H.



WILLIAM HOWARD JOHNSTON

WILLIAM HOWARD JOHNSTON, M.D.

1885 - 1963

William Howard Johnston was born on a farm near Drayton, Ontario, Canada, March 27, 1885. After only six months of retirement from active practice, he died on January 4, 1963.

As a boy of sixteen he came to the United States in response to encouragement by an older brother who was practicing medicine in Iowa, and studied pharmacy at Highland Park College in Des Moines. While practicing as a pharmacist in a small Iowa town, he decided to follow a cherished ambition to study medicine and, in 1906, entered the University of Iowa College of Medicine.

In 1910 he received his Doctor of Medicine degree, and after an internship in otolaryngology at Mercy Hospital in Iowa City, received his master's degree in medicine in 1911. During this phase of his education he found himself associated with one of the great pioneers and teachers of otolaryngology, Lee Wallace Dean, whose influence profoundly affected his subsequent career in that field.

From 1911 to 1929 he practiced his specialty in Muscatine, Iowa, whose county medical society he served as president for a term. His practice was interrupted in 1918 by a year of active duty with the Medical Reserve Corps of the United States Army as a First Lieutenant.

In 1929, his friend and preceptor, Dr. Lee Wallace Dean, then Professor of Otolaryngology at Washington University School of Medicine, called him to St. Louis for a year of teaching and research as Instructor in Otolaryngology, following which he moved with his family to Santa Barbara, California.

During nearly thirty-three years of active otolaryngology in Santa Barbara, he served as chief of the eye, ear, nose, and throat section at each of the three hospitals in that community, chief of staff at each hospital, and president of the Santa Barbara County Medical Society. He was active in the Methodist Church, the Rotary Club, and the Masonic Lodge, of which he was a thirty-second degree mem-

ber. In later years he was a member of the board of directors of the Memorial Cancer Foundation of Santa Barbara.

In 1925 he was certified by the American Board of Otolaryngology on whose Advisory Board he served during the Second World War. He subsequently became a Fellow of the American College of Surgeons, and a member of the American Academy of Ophthalmology and Otolaryngology (Vice-President in 1945-46), the American Laryngological, Rhinological and Otological Society, the American Laryngological Association, the American Otological Society, the Pacific Coast Tot-Ophthalmic Society, and the eye, ear, nose, and throat section of the Los Angeles County Medical Society. In 1943-44 he was Vice-Chairman of the Otolaryngology Section of the American Medical Association, and Chairman of that group in 1950. During his many years of organizational activity he read and published more than twenty papers relating to his specialty, and attended meetings and conventions in every major city in the United States and Canada.

In 1941 he was joined in partnership by his son, Dr. William R. Johnston, with whom he was associated for the remaining twenty-two years of his life. He is also survived by another son, Robert M. Johnston, a college teacher in San Luis Obispo, California, and a grandson, William P. Johnston of San Diego, California. His wife, Vera Jackson Johnston, whom he married in 1911, is living in Santa Barbara.

W.R.J.

FRANCIS WILLIAM WHITE, M.D.

1877 - 1962

Dr. Francis William White, an Emeritus Fellow of the American Laryngological Association, died on March 28, 1962, in New York City, at the age of 85 years.

Dr. White was born in the year 1877. He received the degree of Doctor of Medicine in 1903 from Jefferson Medical College of Philadelphia. Dr. White interned at Philadelphia General Hospital. He was certificated by the American Board of Otolaryngology in 1925.

Dr. White established a practice in medicine in New York City. He was consulting otolaryngologist at Willard Parker Hospital, the Manhattan Eye, Ear, Nose and Throat Hospital in New York City, Grasslands Hospital, Valhalla, New York, and North Hudson Hospital in Weehawken, New Jersey. He was Chairman of the Medical Advisory Board No. 16 of the Selective Service.

Dr. White was a fellow of the American College of Surgeons and held membership in the American Laryngological, Rhinological and Otolological Society, and the American Medical Association.

Executive Sessions

EXECUTIVE SESSION

APRIL 21, 1963

The Opening Session of the Eighty-fourth Annual Meeting of the American Laryngological Association, Inc., held at the Hollywood Beach Hotel, Hollywood, Florida, convened in the Pageant Room on Sunday morning, April 21, 1963, at nine-thirteen o'clock, Francis E. LeJeune, M.D., of New Orleans, Louisiana, President of the Association, presiding. The meeting opened in Executive Session.

The following were unanimously elected to Active Fellowship:

Dr. James D. Baxter, Montreal, Canada
Dr. Douglas P. Bryce, Toronto, Canada
Dr. Charles F. Ferguson, Boston, Mass.
Dr. Frank D. Lathrop, Boston, Mass.
Dr. John A. Kirchner, New Haven, Conn.
Dr. Francis H. McGovern, Danville, Virginia
Dr. George F. Reed, Boston, Mass.
Dr. Harold G. Tabb, New Orleans, La.
Dr. John F. Tolan, Seattle, Washington

The following men were elevated to Emeritus Fellowship:

Dr. Wesley C. Bowers, New York, N. Y.
Dr. Henry M. Goodyear, Cincinnati, Ohio
Dr. Robert C. Martin, San Francisco, California
Dr. Edward J. Whalen, Hartford, Conn.

PRESIDENT LEJEUNE: Gentlemen: I welcome you into the American Laryngological Association, one of the finest medical associations that I know anything about. You have just reason to be proud of

your election into this Association and I hope that you will show interest not only in attending our Annual Meetings, but also in participating and contributing to our scientific sessions. It is to you that we look for future leaders in years to come and I know that you will not be found wanting. God bless you.

The following men were elected members of the Nominating Committee:

Dr. Victor Alfaro

Dr. John Bordley

Dr. James Harrill

Dr. Walter Work

Dr. Julius McCall

PRESENTATION OF THE NEWCOMB AWARD

DR. HARRY P. SCHENCK: The Newcomb Award for 1963 has been conferred upon a tireless and devoted Fellow of this Association. He received the Casselberry Award twenty-seven years ago and, ever since, his attainments have pyramided until he has become an outstanding figure in Laryngology with world-wide recognition in the field of suspension laryngoscopy. For four long years he served as Treasurer of this Association and so efficiently that he was erroneously accused of Scottish ancestry. To his alma mater, Tulane University, he gave many years of loyal and faithful service, including fifteen years as Chairman and Professor of the Department of Otolaryngology. As co-founder of the Ochsner Clinic and co-founder of the Ochsner Hospital he has proven an able administrator. His active participation in organized medicine was rewarded by his elevation to the Presidency of the American Laryngological, Rhinological and Otological Society. I consider it a great privilege to present for the American Laryngological Association the Newcomb Award to our distinguished President, Francis Ernest LeJeune.

ANNUAL BUSINESS MEETING

MONDAY MORNING, APRIL 22, 1963

The Annual Business Meeting convened in the Pageant Room on Monday morning, April 22, 1963, at nine-five o'clock, President LeJeune presiding.

PRESIDENT LEJEUNE: The first order of business is the report of the Secretary, Dr. Richards.

REPORT OF THE SECRETARY

AMERICAN LARYNGOLOGICAL ASSOCIATION

The Eighty-third Annual Meeting of the American Laryngological Association was held on May 4th and 5th, 1962, at the Sheraton-Dallas Hotel, Dallas, Texas. The President, Dr. Dean M. Lierle, presided. Sixteen scientific papers were presented. Seventy-eight Fellows and one hundred and six guests were in attendance.

The Librarian, Historian and Editor, Dr. Francis W. Davison, reported the deaths of:

Emeritus Fellows:

DR. JOHN D. KERNAN

DR. ERNEST M. SEYDELL

DR. FRANCIS W. WHITE

Active Fellows:

DR. CHARLES D. BLASSINGAME

DR. RAE E. ASHLEY

The Newcomb Award was presented to Dr. Paul H. Holinger.

The Casselberry Award was presented to Dr. Hans von Leden.

The following were elected:

Active Fellowship:

Fred W. Alexander, Cleveland, Ohio

Godfrey E. T. Arnold, New York, N.Y.

George A. Sisson, Syracuse, N.Y.

Delmar F. Weaver, Detroit, Michigan

Emeritus Fellowship:

Colby Hall, Los Angeles, Calif.

LeRoy A. Schall, Barnstable, Mass.

The following officers were elected for the coming year, 1962-63:

President	Dr. Francis E. LeJeune
First Vice-President	Dr. Paul M. Moore
Second Vice-President	Dr. Jerome A. Hilger
Secretary	Dr. Lyman G. Richards
Treasurer	Dr. Alden H. Miller
Librarian, Historian, Editor	Dr. Francis W. Davison
First Councilor	Dr. Fred W. Dixon
Second Councilor	Dr. William J. McNally
Third Councilor	Dr. Edwin N. Broyles
Fourth Councilor	Dr. Dean M. Lierle

The next annual meeting of the Association will be held at the St. Francis Hotel, San Francisco, on June 18th, 19th and 20th, 1964.

The Association has lost by death during the past year:

Emeritus Fellow:

William H. Johnston

Active Fellows:

Gordon F. Harkness, Kenneth C. Johnston

The Council at its Annual Meeting in New York on January 12, 1963, recommended for:

Active Fellowship:

James D. Baxter, Montreal, Quebec

Douglas P. Bryce, Toronto, Ontario

Charles F. Ferguson, Boston, Mass.

Frank D. Lathrop, Boston, Mass.

John A. Kirchner, New Haven, Conn.

Francis H. McGovern, Danville, Va.

George F. Reed, Boston, Mass.

Harold G. Tabb, New Orleans, La.

John F. Tolan, Seattle, Wash.

Emeritus Fellowship:

Wesley C. Bowers, New York, N.Y.

Henry M. Goodyear, Cincinnati, Ohio

Robert C. Martin, San Francisco, Calif.

Edward J. Whalen, Hartford, Conn.

Dr. Samuel Salinger was reappointed Abstract Editor.

The present membership of the Association as of February 1, 1963 is:

Active	93
Emeritus	23
Honorary	5
Corresponding	14
	<hr/>
	135

Respectfully submitted,

LYMAN G. RICHARDS, M.D.

Secretary

Dr. Richards' Report was approved as read.

Dr. Alden H. Miller then presented the Report of the Treasurer. It was accepted as presented.

Exhibit A

AMERICAN LARYNGOLOGICAL ASSOCIATION
GENERAL FUND OPERATING STATEMENT

For the Period Beginning April 16, 1962 and Ending March 31, 1963

Income

Dues—90 members at \$75.00	\$6,750.00	
4 new members at \$115.00	460.00	\$7,210.00
	<hr/>	
Sale of <i>Transactions</i>		1,110.00
Interest received—Savings account		347.70
Registration fees—Annual meeting		360.00
		<hr/>
Total operating income		\$9,027.70

Expenses

Annual meeting—1962:

Banquet and cocktails	\$1,390.51	
Projectionist	122.47	
Photography	12.50	
Reporting service	295.46	
Programs	216.20	
Registration cards	9.50	
Entertainment	200.00	\$2,246.64
	<hr/>	

Council expenses:

Travel expenses	\$ 973.03	
Annual luncheon	136.97	1,110.00
	<hr/>	

General expenses:

Stationery, printing and postage	\$ 828.71	
<i>Transactions</i>	2,253.50	
Clerical hire	500.00	
Auditing	200.00	
Treasurer's Bond	62.50	
President's Key	13.38	
Loss on Canadian Exchange	5.81	3,863.90
	<hr/>	<hr/>

Total expenses		7,220.54
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Addition to General Fund		\$1,807.16
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General Fund balance, April 15, 1962		7,717.68
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General Fund balance, March 31, 1963		\$9,524.84
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Exhibit B

AMERICAN LARYNGOLOGICAL ASSOCIATION
TRUST FUND OPERATING STATEMENT

For the Period Beginning April 16, 1962 and Ending March 31, 1963

Income

Interest—Government bonds	\$ 1,487.59	
Dividends—Common stocks	84.87	
Donations to Memorial Fund:		
Dr. Harry Schenck (stock)	675.00	\$ 2,247.46

Expenses

Casselberry Prize—awarded to Dr. Hans Von Leden		100.00
Addition to Trust Fund		\$ 2,147.46
Trust Fund balance, April 15, 1962 (Exhibit C)		48,904.10
Trust Fund balance, March 31, 1963 (Exhibit C)		\$51,051.56
Trust Fund balance represented by:		
Government Bonds at par value	\$44,500.00	
Common stocks at market value, date of donation	2,362.00	
Total bonds and stocks (Exhibit D)	\$46,862.00	
Cash	4,189.56	\$51,051.56

Cash recapitulation

Cash in Trust Fund	\$ 4,189.56
Cash in General Fund	9,524.84
	<u>\$13,714.40</u>
Cash in Commercial account	\$ 1,787.09
Cash in Savings account	6,927.31
Cash in Savings deposit receipt	5,000.00
	<u>\$13,714.40</u>

Exhibit C

AMERICAN LARYNGOLOGICAL ASSOCIATION

REPORT OF TRUST FUNDS

March 31, 1963

Fund	Principal and Unused Income 4-15-62	Additions to Principal 4-16-62 to 3-31-63	** Income 4-16-62 to 3-31-63	Expenses 4-16-62 to 3-31-63	Unused Income 3-31-63	Principal and Unused Income 3-31-63
Casselberry	\$ 7,323.02	-----	\$ 235.47	\$ 100.00	\$ 135.47	\$ 7,458.49
Johnston	6,527.88	-----	209.90	-----	209.90	6,737.78
de Roaldes	4,492.90	-----	144.46	-----	144.46	4,637.36
Newcomb Trust	2,562.93	-----	82.40	-----	82.40	2,645.33
Newcomb Prize	598.20	-----	19.23	-----	19.23	617.43
Research	22,996.32	-----	739.43	-----	739.43	23,735.75
Memorial	4,402.85	\$ 675.00*	141.57	-----	141.57	5,219.42
	<u>\$48,904.10</u>	<u>\$675.00</u>	<u>\$1,572.46</u>	<u>\$100.00</u>	<u>\$1,472.46</u>	<u>\$51,051.56</u>

* Represents value of stock donated by Dr. Harry Schenck.

** Income has been allocated to each fund by the ratio that each fund bears to the total fund, as of 4-15-62.

Exhibit D

AMERICAN LARYNGOLOGICAL ASSOCIATION

TRUST FUND INVESTMENTS

March 31, 1963

	Serial No.	Par Value
GOVERNMENT BONDS		
Negotiable:		
U. S. Treasury Bonds	85-680	\$1,000.00
	24-955	1,000.00
	24-956	1,000.00
	24-957	1,000.00
	24-958	1,000.00
	24-959	1,000.00
	118-136 F	1,000.00
	118-137 H	1,000.00
	118-138 J	1,000.00
	277-504 D	1,000.00
	8357	5,000.00
	92-341	1,000.00

Non-negotiable:		
U. S. Treasury Bonds	1515	5,000.00
	2485	1,000.00
	2486	1,000.00
	12153 D	1,000.00
	12154 C	1,000.00
	12155 E	1,000.00
Eleven H Bonds		10,500.00
Four K Bonds		8,000.00
		<hr/>
		\$44,500.00
COMMON STOCK		
International Business Machine, 1 share	K-085881	\$ 517.00
McDonnell Aircraft Corp., 26 shares	S-012923	1,170.00
McIntyre Porcupine Mines, Ltd., 15 shares	NYA-4817	675.00
		<hr/>
		\$ 2,362.00
TOTAL BONDS AND STOCK		<hr/>
		\$46,862.00

ACCOUNTANTS' REPORT

To the Members of the American Laryngological Association
Gentlemen:

We have examined the accounts and financial records of the American Laryngological Association for the period beginning April 16, 1962 and ending March 31, 1963.

Cash receipts and disbursements were verified and all United States Government Bonds and common stock were physically counted and examined.

In our opinion, the accompanying statements present fairly the financial position of the American Laryngological Association as at March 31, 1963 and the results of its operations for the period beginning April 16, 1962 and ending March 31, 1963.

Very truly yours,
SERENE, KOSTER & BARBOUR
Certified Public Accountants

PRESIDENT LEJEUNE: Next is the report of the Nominating Committee. Dr. Alfaro.

DR. VICTOR R. ALFARO: Mr. Chairman, the Nominating Committee met yesterday, as ordered by the Society, and presents for your approval the following slate of members of the Council:

Anderson Hilding

Francis Davison

Paul Holinger

William J. McNally

Lester Brown

Edwin Broyles

Lyman Richards

Dean Lierle

Alden Miller

Francis E. LeJeune

Respectfully submitted by

John Bordley, James Harrill,

Walter Work, Julius McCall

and Victor Alfaro

The report of the Nominating Committee was unanimously accepted.

PRESIDENT LEJEUNE: The report of the Librarian, Historian and Editor:

REPORT OF THE LIBRARIAN, HISTORIAN AND EDITOR

Since our last meeting in May, 1962, the Association has suffered the loss of the following Fellows:

John H. Foster, M.D. (Emeritus Fellow) October 2, 1962

William H. Johnston, M.D. (Emeritus Fellow) January 4, 1963

Gordon F. Harkness, M.D. (Active Fellow) December 18, 1962

Kenneth C. Johnston, M.D. (Active Fellow) February 6, 1963

The Eighty-third Annual Meeting of the American Laryngological Association was held on May 4 and 5, 1962, at the Sheraton-Dallas Hotel, Dallas, Texas. The stenotypist, Mrs. Annabella Square, a representative of the Martin C. Johnson Reporting Service, was pleasant, efficient and dependable. I hope that her services can be obtained for future meetings. The Martin C. Johnson Reporting Service is a successor to the Master Reporting Company.

All essayists on the program submitted their manuscripts promptly and the transcript of the discussions was received three weeks following the meeting. These were promptly mailed to each discussant for correction.

According to Council directive, 250 copies of the 1962 *Transactions* were ordered.

The galley proofs were received from the publisher on July 6, 1962.

Copies of the 1962 <i>Transactions</i> mailed by the publisher to Active and Honorary Fellows on November 10, 1962, totalled	99
Standing orders and 1-year orders and copies to Emeritus and Corresponding Fellows mailed Nov. 14 - Mar. 2, 1963	85
<i>Transactions</i> remaining in hands of Editor	66
Cash received to date for <i>Transactions</i>	\$729.00
Bills outstanding	8.00
Editor's expenses from April 3, 1962 to April 14, 1963, for stationery, express charges and postage	97.55

In October, 1962, letters were sent to all Corresponding and Emeritus Fellows, inquiring whether or not they wished to receive the 1962 *Transactions* at the Council-established price of \$5.00. No *Transactions* were sent unless they were specifically ordered.

The cost of the 1962 *Transactions*, including the mailing and postage for copies sent to Active and Honorary Fellows was \$2,253.50. This was \$216.71 less than the cost for 1961 *Transactions*. The difference was chiefly due to the fact that the Editor was somewhat ruthless in editing the long-winded discussion in the Business Sessions, thereby reducing the number of pages from 300 for 1961 to 252 pages in 1962.

Dr. Salinger's abstracts were promptly prepared and were accepted for publication by the Editors of the *Laryngoscope* and the *A.M.A. Archives of Otolaryngology*.

Respectfully submitted,
F. W. DAVISON, M.D., *Editor*

Dr. Davison's Report was accepted as read.

PRESIDENT LEJEUNE: The Report of the American Board of Otolaryngology. Dr. Frederick Hill.

DR. FREDERICK THAYER HILL: Mr. President and Fellows: At the Fall examination of the American Board 114 candidates were examined. Of these 78 were passed and 36 either conditioned or failed, a failure percentage of about 33 per cent.

It is interesting to note that the waiting list of candidates for examination has increased to the extent that it is going to be necessary to carry on another examination in June.

PRESIDENT LEJEUNE: Thank you, Dr. Hill.

Dr. Fred W. Dixon then presented the report on the Annual meeting of the Board of Governors of the American College of Surgeons, held in Atlantic City, New Jersey, on October 14, 1962.

REPORT OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION
BY ITS REPRESENTATIVE TO THE BOARD OF GOVERNORS
OF THE AMERICAN COLLEGE OF SURGEONS

The Annual Meeting of the Board of Governors of the American College of Surgeons was held in Atlantic City October 14, 1962. It was attended by 138 Governors out of a possible 181.

The American Laryngological Association was represented by:

Dr. Dean Lierle who served on the Board of Regents.

Dr. Harry P. Schenck who served on the Executive Committee of the Board of Governors.

The following represent the Association on the Board of Governors:

Dr. Francis E. LeJeune, whose term expires in 1963.

Dr. Harry P. Schenck, whose term expires in 1964.

Dr. Paul H. Holinger, whose term expires in 1965.

There were two subjects discussed which I believe would be of interest to all of us.

1) "What Can Be Done About the Increase of Lawsuits Against Doctors?" It was reported that the number of lawsuits had increased by 3.2% each year; while the cost per claim had risen 33%. The most frequent claims for damages were unsatisfactory results, poor patient relationship, loose talk, excessive fees, inadequate records and early pressure of bills for service.

It was suggested that a committee of outstanding Doctors and Lawyers in the Community consider Medico-Legal cases as they arise and advise as to the merit of a claim.

The second subject was on a study made to determine "Whether the United States needs more Surgeons and, if so, in what Specialties should they be trained?" This was an interesting preliminary report of the data now being processed at a cost of \$10,000. In it will be given the number of Specialists in each area and the average charge for each operation in that locality. Part of this is now available.

With the expected increase in population within the next twelve years from 180 million to 235 million, the problem arises as to the number of Specialists needed and in what field. The average number of Otolaryngologists certified in the last four years was 73 per year.

The following survey showed the number of certified Otolaryngologists in proportion to the population:

Cities 25,000 to 50,000—(244 cities)—

One certified Otolaryngologist for each 35,000

50,000 to 100,000—(131 cities)—

One certified Otolaryngologist for each 21,300

100,000 and up—(130 cities)—

One certified Otolaryngologist for each 22,300

At the present time there are in the United States 3,829 full time N.E.T. Specialists. Of this number 3,450 are Board certified. (Health Manpower Source Book U. S. Department H.E.W.). Of this number only 411 belong to the American College of Surgeons. If we, as a group, are to influence the decisions of the College, every Fellow in the American Laryngological Association should belong.

In the questionnaire sent out by the American College of Surgeons the question was asked of the 411 American College of Surgeons Otolaryngologists as to their opinion of the supply in this field in their community. Twenty-three thought there were too many, while 183 replied just enough, and 183 not enough.

It would seem that the more educated the people become, the more they are demanding better qualifications and training of their Doctors. This, coupled with the increase in population, and the increase in the number of hospitals being built under the Hill-Burton Act, would lead you to believe that we are *not* training Otolaryngologists rapidly enough to meet the demand within the next ten years.

Respectfully submitted,

FRED W. DIXON, M.D.

Dr. Dixon's report was accepted as read.

PRESIDENT LEJEUNE: The proposed amendments to the Constitution and By-Laws will be taken up at a special meeting at the end of our Scientific Session this afternoon, because it would take much too much time to discuss that at the present time.

DR. CHARLES MORGAN NORRIS: Members of the Association, I would like to thank our President and our Secretary for allowing me to introduce Dr. Carlos Cleves, of Bogota.

Dr. Cleves is the Immediate Past President of the Colombian Society of Otolaryngology and he is the President of the Ninth Pan-American Congress of Otolaryngology, which is to be held in Bogota, Colombia, next February.

Dr. Cleves has come to tell us something of the plans which he and his colleagues are making for this Congress and I think we will all listen with interest.

DR. CLEVES: Mr. President and Members of the Society: I have to tell you that this is the first time that I have spoken in a foreign language and I think that it will be the last one because I feel so discouraged about it.

Maybe some of you know that in the last Pan-American Congress of Otorhinolaryngology and Bronchoesophagology that was held in Caracas last year Bogota was designated as the next city for the Ninth Pan-American Congress. The reason of my visit now to Florida is to announce this and to invite all of you to assist and to take part in this meeting.

The Scientific Program is already finished. It has been integrated by the most prominent personalities in our field. I brought with me a few copies of the preliminary program that later I am going to distribute among you. I hope that this will be the final one because all the participants have confirmed their assistance.

Now we are working on the social program—or may I say on the tourist program—because I know that one of the most important parts of these events is the relaxation of the daily routine.

I can assure you that all of you will be very welcome and you will spend a few nice days.

Now with the jets the trip is very short. It is only about two hours and forty minutes non-stop Miami to Bogota.

I hope to see all of you next year in Bogota. Also this invitation includes your wives, in spite of the well-known charm of the Latin-American girls that you will find there anyway.

EXECUTIVE SESSION

MONDAY AFTERNOON, APRIL 22, 1963

The meeting convened in Executive Session in the Pageant Room on Monday afternoon, April 23, 1963, immediately following the close of the Scientific Session, President LeJeune presiding.

PRESIDENT LEJEUNE: Gentlemen, we have to consider the proposed amendments to the Constitution and By-Laws of the American Laryngological Association. I shall ask our Secretary to read the proposed changes.

SECRETARY RICHARDS: In accordance with the Constitution as it now stands, which was revised in March of 1959, any changes and amendments have to be submitted to the Association at least a month prior to a regular meeting and submitted only to the Active Members. I think I am correct in saying that I did that several months ago, so you have all at least had a chance to look at these and, let us hope, also to think them over and consider whether you regard them as advisable or not.

These were all discussed and gone over by the Council with the feeling that the amendments, as we have them here, are advisable and constitute an improvement in the general set-up of the organization.

It takes a three-quarters vote by the Active Members present to make these changes.

I will read them and anyone who wants to discuss them or ask questions about them or object to them has every privilege in the world to do so before we put them into operation.

The first one involves Article IV, Paragraph 1—you may, none of you perhaps have a copy of the Constitution with you—page 6—it is not important. What I mean is that it is not important for you to have a copy of them with you. You can hear it. I will read it to you.

The requirements for admission to the Association are that the man be, amongst other things, a recognized specialist for a period of

ten years. We would like to amend—that is, the Council would like to amend it to “a recognized specialist of seven years or more,” the point being, we felt, that to require a man to have been a recognized specialist, naturally in laryngology and rhinology, for ten years delayed the entrance into the organization of a man who was thoroughly qualified to be a member, who would be a valuable contributor to it. To make him wait for ten years seemed a little too long and so we suggest that we cut this down and ask him only to have been a recognized specialist in laryngology and rhinology for seven years.

PRESIDENT LEJEUNE: Gentlemen, what are your wishes?

DR. SAMUEL SALINGER: Mr. Chairman, I would like to ask the question: what do they have to do in this seven years?

SECRETARY RICHARDS: That is a very good question. It says in the Constitution that he be a recognized specialist. It makes no stipulation as to the point at which he becomes a specialist. That, I think, is a debatable matter and perhaps should be more specifically stated as to just what that means in terms of years.

DR. HILL: Don't you think at least it should mean accreditation by the Board?

SECRETARY RICHARDS: All right.

DR. PAUL H. HOLINGER: He is a recognized specialist by his fellow specialists, if he has passed the Board examination.

SECRETARY RICHARDS: Would that please you? In other words, he cannot join this organization until he has had his Board for seven years. Is everybody happy with that?

(There was general assent.)

PRESIDENT LEJEUNE: Do we need a motion to pass that?

SECRETARY RICHARDS: Yes.

PRESIDENT LEJEUNE: Then I will entertain a motion.

(Such motion was duly made, seconded, and passed.)

The next one, Mr. Secretary.

SECRETARY RICHARDS: The next one is Article VI, Paragraph 10 (c), which now reads:

c. No paper, other than the President's Address, shall consume more than twenty-five (25) minutes in its reading without the unanimous consent of the members present.

It is suggested that this be amended to read:

c. No paper, other than the President's Address, shall consume more than twenty (20) minutes in its reading without the unanimous consent of the members present.

This amendment was unanimously approved.

SECRETARY RICHARDS: Now we come to a somewhat more momentous change. Article VI, Paragraph 8, reads that the Council shall organize by electing a First Vice-President, along with the list of other officers. It is suggested that that be changed to read that the Council shall organize by electing, among other people, a First Vice-President—and this is the change: “who will at the same time become the President-Elect.”

The matter of the President-Elect, of course, is current in the American Otological Society and in the Triological Society. It has never been current in the Laryngological Association. This suggestion has been made—I think Dr. McNally was one of the first to bring it up—on the theory that if a man is elected to the position of President without having been on the Council, as can readily happen, he has had no great contact with it. He is a little, perhaps, unfamiliar with the routine, and it would be helpful to him to have had a year to think the matter over, to plan his subsequent program and to that extent initiate him into his future duties a little more gradually and easily than if he were suddenly picked out of a hat, so to speak, and made the President of the organization without any preliminary experience.

Does anybody object to that?

DR. HILL: Mr. President, being one of the oldest members of the Association, I have a lot of respect for tradition and this has been traditional in this Association: that we have this framework of two vice-presidents. Now, if we change this it seems to me that the necessary thing would be to execute the Second Vice-President, because he would be simply a supernumerary.

SECRETARY RICHARDS: Which he is now, as a matter of fact.

DR. HILL: I don't care what the Triological and the Otological do. I am just old enough to like to keep in the tradition.

I think we have changed the Constitution and By-Laws two or three times since I have been a member. I believe I was chairman of the last committee that revised it. We tried at that time to keep our traditional pattern as well as we could—for example, the roll call—some other things that go back for years. I am not worried about anybody needing a year of training to come into the office of president. I can see a great advantage of the vice-presidencies coming down to other men, that it does mean that they will become presidents some time. So, from my angle, I would be very much opposed to making this change in our official circle.

PRESIDENT LEJEUNE: Thank you, Dr. Hill.

Any other comments? Dr. Dixon!

DR. DIXON: Mr. Chairman, I have always considered it a great honor to be the president of this Association. Not all can, however, have that honor. By having a First Vice-President and a Second Vice-President we can honor certain men that we feel should have some recognition. Like Dr. Hill, I would like to see these offices continued.

It occasionally happens that the Editor or the Treasurer or the Secretary no longer wishes to have that duty—after four or five years—and would like to be relieved. It usually happens that that Fellow then becomes the President. After four or five years he has had the necessary training. I think it is best to keep our old tradition.

It is up to the membership to vote on that. However, it seems to me that anyone who becomes the president of this Association should be well-informed. He could be well-informed if he inquires by letter or telephone call. I personally would like to vote against the amendment.

PRESIDENT LEJEUNE: Are there any other comments? Dr. Broyles!

DR. EDWIN N. BROYLES: Mr. President, it seems to me that as far as I can remember most of the gentlemen who have been elected to the presidency have been on the Council, if not of this organization then of other organizations. They certainly are well-acquainted with the running of medical societies; so I feel very much like Dr. Hill and Dr. Dixon.

PRESIDENT LEJEUNE: Dr. Schenck!

DR. SCHENCK: I see no advantage in making this change, Mr. President. I think it would be a step backward. I think all contingencies are taken care of by the present arrangement. It has worked over the years and experience should count for something. I don't remember any president who has come into office who has been ignorant of procedures and I see no advantage whatever in disturbing the set-up which has worked very well for half a century or more.

PRESIDENT LEJEUNE: Gentlemen, do I hear a motion?

DR. BROYLES: I move that we reject this amendment.

DR. HILL: Second the motion.

PRESIDENT LEJEUNE: Gentlemen, you have heard the motion. Is there any comment or discussion?

(There was none.)

All those in favor of the motion proposed by Dr. Broyles please say "aye." Motion—

SECRETARY RICHARDS: Wait a minute. It has to be a three-quarters vote. Is there anybody against it?

PRESIDENT LEJEUNE: Is anybody against it?

(There was no response.)

Motion passed.

SECRETARY RICHARDS: The next change is in reference to the Newcomb Award, which now reads as follows:

That the recipient of this award shall be selected by a committee of three Fellows, who shall not be members of the Council.

There is no argument about that. And:

That the Committee shall notify the Secretary of their selection one month before the Annual Meeting of the Council.

That is the way it stands now. The amendment is as follows:

The Award Committee, consisting of three Fellows who are not members of the Council, may select a candidate and submit his name and qualifications to the Council at its midwinter meeting for approval or rejection.

The decision on granting the award shall be the sole duty of the Council.

Those are the two important changes. These others simply are not in it but they are, I am sure, not creative of any argument:

The Secretary of the Association shall notify the candidate of his selection for the award as soon after the Council meeting as possible.

And:

The award shall be presented at the Annual Dinner of the Association.

Basically this comes down to, it seems to me, a very fundamental point which has come up not very often but once with some degree of acrimony and unpleasantness. We want to get it straightened out, that although the Newcomb Committee may select a name and submit it to the Council their decision does not compel the Council to approve it. The Council reserves its right—not very often, but possibly occasionally—to reject, for any reasons, the name suggested by the Newcomb Award Committee.

DR. HOLINGER: I recommend acceptance of this amendment.

(The motion was duly seconded.)

PRESIDENT LEJEUNE: All those in favor—

DR. HILL: May I speak in opposition? I hate to be the opposer here, but I feel—I see a little of the candidates' point of view, the Washington influence coming in here. It seems to me too bad to emasculate these committees, which, after all, are generally continuing committees—

SECRETARY RICHARDS: No, no. They are annual.

DR. HILL: Many of them have been continued—year after year, with one man changing.

SECRETARY RICHARDS: No, they are new every year.

DR. HILL: Well, many times—because I have been on committees—

SECRETARY RICHARDS: You were on the deRoaldes Committee. That is something else again, but the Newcomb Committee is appointed every year by the president.

DR. HILL: I still feel that perhaps it is just as well to give these people some authority.

SECRETARY RICHARDS: This is a very important point and I think it ought to be discussed because this can be dynamite if we don't get this settled.

PRESIDENT LEJEUNE: Any further comments?

DR. DIXON: I was instrumental in throwing in this amendment. The reason is that occasionally the Committee meets and they decide on a person. Sometimes they don't know all about this man as they should. It happened on one occasion that the man selected wrote a very bitter letter, resigning. He didn't want to belong to this Association. Nevertheless, according to the Committee, we had to give this man his award and I think it is wrong. I still think it is wrong. I think an award of this kind, an honor of this kind, should be given consideration. I don't believe it ever came up before and when it did come up we gave him the award but the Council was rather against it.

SECRETARY RICHARDS: May I interrupt this? Are you sure you are talking about the Newcomb Award and not the Casselberry Award?

DR. DIXON: No, I was talking about awards generally. I think that in any of these awards you should appoint a committee, the committee should report back to your Council and the Council decides whether or not they want to accept the recommendation of your committee. It doesn't happen very often but I think there should be a little discretionary power given to the Council to accept or reject this recommendation.

PRESIDENT LEJEUNE: Any other comments, gentlemen?

(Call for the question)

Dr. Broyles!

DR. BROYLES: Mr. President, I feel that when the president appoints a committee that he is doing it in good faith and I think when a committee nominates one of the Fellows, or a doctor, for the award, I believe we ought to go along with the committee. In other words, I think if the Council rejects the recommendation of a committee it is giving them a slap in the face. I think most of these committees really go into it as deeply as they can before they make their decision.

PRESIDENT LEJEUNE: Dr. Daly!

DR. JOHN F. DALY: I think the fundamental distinction is whether this committee is an advisory or recommending committee or whether it is one with executive power. I think if the committee actually makes the choice, then it has executive power. I think most committees are appointed as recommending committees, recommending back to the parent body, which would be the Council.

PRESIDENT LEJEUNE: Dr. Work!

DR. WALTER P. WORK: In my opinion you don't have to go to Washington, D.C. I have been educated in Ann Arbor and you just have to go right to the Medical School and their committees and their recommendations. If their recommendations aren't taken, there is all hell to pay.

PRESIDENT LEJEUNE: Any other comments?

SECRETARY RICHARDS: Just to refresh your memories, the statement as it now stands is this:

That the recipient . . . shall be selected by a committee . . .

DR. SCHENCK: The committee is not selected on the basis of an election? They are not an elected committee?

SECRETARY RICHARDS: They are appointed by the president.

DR. SCHENCK: Therefore they are advisory. They are not an elected committee and they only have an advisory function. What is more, in many instances the committee doesn't know who the candidate is—in the case of the Casselberry Award, for instance.

SECRETARY RICHARDS: There is quite a difference between the Casselberry and the Newcomb Awards.

DR. SCHENCK: But in this instance they get manuscripts under a *nom de plume*.

SECRETARY RICHARDS: That is the Casselberry Award. Let's let that go. That is coming up later. This is the Newcomb Award.

DR. SCHENCK: The committee is still not an elected committee.

SECRETARY RICHARDS: No, it is not. It is appointed by the president.

DR. SCHENCK: Therefore, as Dr. Daly has pointed out, they can only act in an advisory capacity.

PRESIDENT LEJEUNE: Is there a motion on the floor?

SECRETARY RICHARDS: Dr. Holinger made a motion but it was never acted on.

PRESIDENT LEJEUNE: Is there any further comment?

(There was no response.)

If not, are you ready for the question?

A FELLOW: What is the question?

SECRETARY RICHARDS: The question is that this should be changed from the statement: "That the recipient . . . shall be selected by a committee of three Fellows, who shall not be members of the Council"—change that to read: "The candidate shall be selected and his name and qualifications submitted to the Council for approval or rejection."

(Call for the question)

PRESIDENT LEJEUNE: All those in favor of this motion please signify by saying "aye"; all those opposed? The "ayes" have it. Motion passed.

The next one, please.

SECRETARY RICHARDS: Now we come to almost a similar situation with regard to the Casselberry Award. It now says—listen to this carefully, now—that:

The Award Committee shall determine the successful contestant or contestants, and shall inform the Secretary at least one month before the Annual Meeting of the Council.

On the basis of the consideration of a thesis submitted under a *nom de plume*, whose name is not even known to the Secretary—or should not be, certainly—that is to be changed to:

The Award Committee may select a candidate and submit his name and qualifications to the Council for approval or rejection.

That is the same situation as regards the Newcomb Committee. And it goes on:

The decision on granting the award shall be the sole duty of the Council.

DR. HOLINGER: I recommend acceptance of this amendment.

(The motion was duly seconded.)

PRESIDENT LEJEUNE: Any comments, gentlemen? Any discussion?

(There was none.)

Are you ready for the question?

(Call for the question)

All those in favor please signify by saying "aye"; those opposed? Motion carried.

SECRETARY RICHARDS: About the other two items there would certainly be no question:

The Secretary shall notify the successful candidate immediately after approval of the award by the Council.

And:

The award shall be presented at the Annual Dinner.

With regard to the deRoaldes Award, it now reads:

That the Committee shall notify the Secretary of its selection one (1) month before the Annual Meeting of the Council.

That is to be amended to read:

The Award Committee may select a candidate and submit his name and qualifications to the Council at the midwinter Council meeting for approval or rejection. The decision on granting the award shall be the sole duty of the Council.

DR. BROYLES: So moved. Recommend approval.

(The motion was duly seconded.)

PRESIDENT LEJEUNE: All those in favor of this motion please signify by saying "aye"; those opposed? Motion carried.

SECRETARY RICHARDS: There is an item regarding the Finance Committee—it is a little long. It now reads:

The Finance Committee, consisting of the Treasurer and two (2) other Fellows to be appointed annually by the Council. All Special Funds, unless otherwise ordered, and interim investments shall be controlled by this Committee.

We want to change this to read:

The Finance Committee, consisting of the Treasurer, the President and the Immediate Past President—that gives three people that the Finance Committee is to consist of rather than the Treasurer and two other Fellows not designating as to who they are—

may buy or sell subscription rights and exercise voting privileges. This Committee shall have power to sell, invest or reinvest all Special Funds of the Association as they deem advisable. No unusual expenditures, however, may be made by this Committee, except when so ordered by the Association at its Annual Meeting, or by resolution of the Council at any of its meetings. This Committee shall review the Common Stock holdings of the Association at least twice a year.

Dr. Miller, do you want to say anything about that?

DR. MILLER: No, it sounds good to me.

SECRETARY RICHARDS: The only difference that I can see basically—it details more exactly the financial transactions—the only difference is it specifies that the Finance Committee shall consist of the Treasurer, the President and the Immediate Past President.

DR. HILL: I would like to move the acceptance of that.

(The motion was duly seconded.)

PRESIDENT LEJEUNE: Any discussion of that motion?

(There was none.)

It has been moved and seconded that this amendment be accepted. All those in favor say "aye"; all those opposed? This motion is passed, Mr. Secretary.

SECRETARY RICHARDS: With reference to the Casselberry Award, it now says:

All manuscripts entered in a given year must be in the hands of the Secretary before December first.

The trouble with that, of course, is that it doesn't give the Casselberry Committee any time to go over these theses and send them from one person to another in time to bring in a report at the January Council Meeting. I would like to see that changed to:

All manuscripts entered in a given year must be in the hands of the Secretary before September first.

That gives the Casselberry Committee two months instead of one month to go over the theses.

PRESIDENT LEJEUNE: Do I hear a motion to that effect?

DR. BROYLES: So moved.

(The motion was duly seconded.)

PRESIDENT LEJEUNE: All those in favor please signify by saying "aye." Motion carried.

Now, Mr. Secretary.

SECRETARY RICHARDS: The next order of business, unless I am very much mistaken, is a meeting of the members of the Council to elect their officers.

PRESIDENT LEJEUNE: Will those members whose names are on the blackboard come up here for a very brief huddle.

(Secretary Richards read out the names and those Fellows who had been nominated to the Council came forward as requested.)

DR. LOUIS H. CLERF: Mr. President, I rise to a point of order. This group has not been elected. The nominations were closed but they were not elected.

PRESIDENT LEJEUNE: I apologize. Shall I entertain a motion, then? Will someone make a motion for the election and approval of these gentlemen, in accordance with the report of the Nominating Committee and that the Secretary be instructed to cast one ballot.

DR. HILL: So moved.

PRESIDENT LEJEUNE: Second?

(The motion was duly seconded.)

It has been moved and seconded that we elect, or approve, of the report of the Nominating Committee. All those in favor say "aye"; all those opposed? The Council is therefore elected.

(The Council then met briefly.)

SECRETARY RICHARDS: Mr. President, may I submit the following results of the meeting of the Council for 1963:

For President, Dr. Anderson Hilding
First Vice President, Dr. Paul Holinger
Second Vice President, Dr. Lester Brown
Treasurer, Dr. Alden Miller
Editor, Dr. Francis Davison
Secretary, Dr. Lyman Richards
First Councilor, Dr. William McNally
Second Councilor, Dr. Edwin Broyles
Third Councilor, Dr. Dean Lierle
Fourth Councilor, Dr. LeJeune

PRESIDENT LEJEUNE (to President-Elect Hilding): Andy, it is my great pleasure and privilege to hand you this historic gavel, symbolic of the great honor which has just been given you.

I know that under your guidance this Laryngological Association will have one of the best meetings that it has ever had. My congratulations! (Presenting gavel)

(The audience rose and applauded and remained standing.)

(President Hilding assumed the Chair.)

PRESIDENT HILDING: Thank you.

I suppose all men, when they receive a great honor like this, experience certain reactions and emotions. I think you must know that because you have heard many people try to express them.

Remembering an experience a couple of years ago, I am not going to try to express them. The occasion was a dinner at the Gustaphus Adolphus College in southern Minnesota, when certain alumni were to receive an honor called the Greater Gustavus Award. Each responded as he received the award. The first man responded something like this:

"When I was notified that I was to receive this award I was utterly surprised. I do not deserve it at all. I am completely unworthy"—and he went on in this vein for a while.

The second man got up and said, "When I was notified that I was to receive this award I was greatly surprised. I do not deserve it at

all"—and he went on and gave the same response, practically word for word.

The third one was a woman, a Ph.D., and, thinking that humble pie was a little bit hackneyed, I think she met the occasion very well. She said, "When I was notified that I was to receive the Greater Gustavus Award my friends were surprised, and as far as deserving it is concerned, because I was the tallest in the gymnasium class when attending this College I was placed first in the line when it came time to jump over that piece of equipment called a horse. As I picked myself up off the floor on the other side I decided that I deserved anything this institution could ever do for me.

So I shall simply content myself with saying, "Thank you. I shall do my best." And I am sure that when the news of this reaches Duluth my friends will be surprised.

My first duty is a very pleasant one. I am instructed that I am to present this to Dr. LeJeune for his presidency of the last year. (Presenting a key in a gift box to Dr. LeJeune)

DR. LEJEUNE: Thank you very much. I am deeply appreciative of this. I shall certainly preserve it as one of my most treasured possessions. Thank you very much.

DR. DIXON: Mr. President, there is something I should like to mention. Some years ago this idea of a pin or a key to be given to the retiring president was suggested by Dr. LeJeune. I think it is a very fine idea. Now, the cost of making the dies for the key and pin was defrayed by Dr. LeJeune. He refused to receive any recompense for this, saying that it was his wish to make the donation to the Society. I would like to have a rising vote of thanks to Dr. LeJeune for this donation to the Society.

(The audience rose and applauded.)

PRESIDENT HILDING: That, then, concludes our Eighty-fourth Session and I declare the meeting closed.

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

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*.
1958. ALFARO, VICTOR R., 917 - 20th St. N. W., Washington 6, D. C., 1958, '59, '60, '61, '62, '63.
1962. ARNOLD, GODFREY E. T., 2500 N. State St., Jackson 6, Mississippi, 1962, '63*.
1958. ATKINS, JOSEPH P., 3400 Spruce St., Philadelphia 4, Pa., 1958, '59*, '60, '61, '62, '63.
1956. BAKER, DANIEL CLIFTON, JR., 903 Park Avenue, New York 21, N. Y., 1956, '57, '58, '59*, '60, '61, '62*, '63.
1963. BAXTER, JAMES D., 1509 Sherbrooke St. West, Montreal, P.Q., Canada, 1963.
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.
1955. BORDLEY, JOHN E., Johns Hopkins Hospital, Baltimore 5, Maryland, 1955, '56, '57, '59, '60, '62, '63.
1959. BREWER, DAVID W., 1100 E. Genesee St., Syracuse 10, N. Y., 1959, '60*, '61, '62*, '63*.
1955. BROWN, LESTER ALEXANDER, 490 Peachtree Street, N.E., Atlanta 8, Georgia, 1955, '56, '57, '58, '59, '61, '63.
1940. BROYLES, EDWIN N., 1100 North Charles Street, Baltimore 1, Maryland, 1940, '41*, '42, '46, '48*, '49, '50, '51*, '52, '53, '54, '55, '56, '58, '59*, '60, '61*, '63.

1963. BRYCE, DOUGLAS P., 170 St. George St., Room 320, Toronto, Canada, 1963*.
1945. CAMPBELL, COLONEL PAUL A., 321 Park Hill Drive, San Antonio, Texas, 1946, '47, '49, '51, '57*, '61.
1942. CANFIELD, NORTON, Harwood Hospital, Christiansted, St. Croix, Virgin Is., 1942, '46, '47, '48, '49*, '50, '51, '56, '57.
1959. CARDWELL, EDGAR P., 109 So. Munn Ave., East Orange, N. J., 1959, '60, '61, '62, '63.
1957. CONLEY, JOHN J., 139 East 36th Street, New York 16, N.Y. 1958*, '60*, '61, '62*, '63*.
1957. CONVERSE, JOHN MARQUIS, 722 Park Avenue, New York 21, N.Y., '60, '62*.
1959. CRACOVANER, ARTHUR J., 103 E. 78th St., New York 21, N. Y., 1959, '60, '61*, '62, '63.
1960. DALY, JOHN F., Faculty Practice Offices, 566 First Ave., New York 16, N. Y., 1960, '61*, '62, '63.
1951. DAVISON, FRANCIS W., Geisinger Medical Center, Danville, Pa., 1951, '52, '53*, '54, '55, '56, '57, '58, '59, '60, '61, '62, '63*.
1941. DIXON, FRED W., 1027 Rose Building, Cleveland 15, Ohio, 1941, '42*, '44, '46, '47*, '49*, '50, '51, '52, '53, '54, '55, '56, '57, '58*, '59, '60, '61, '62, '63.
1947. EAGLE, WATT WEEMS, Duke Hospital, Durham, N. C., 1948*, '49, '51, '53, '54, '56, '57, '59, '62.
1954. EQUEN, MURDOCK S., Ponce de Leon Infirmarv. Atlanta 8, Ga., 1954, '55, '57, '58, '60, '63.
1952. ERICH, JOHN B., Mayo Clinic, 102 Second Avenue, Rochester, Minn., 1953, '56*, '57.
1963. FERGUSON, CHARLES F., 300 Longwood Ave., Boston 15, Mass., 1963.
1955. FITZ-HUGH, GLASSELL SLAUGHTER, 2003 Spottswood Road, Charlottesville, Virginia, 1955, '57, '59, '60, '61, '62*.

1956. FRIEDBERG, STANTON A., 122 So. Michigan Ave., Chicago 3, Illinois, 1956, '57*, '58, '59, '60, '61, '62, '63.
1932. FURSTENBERG, ALBERT CARL, Out-Patient Bldg., University Hospital, Ann Arbor, Michigan, 1933*, '36*, '38*, '46, '48*, '50, '51, '53, '54, '55*, '56, '57, '58*, '59, '60, '61, '62, '63*.
1959. GOLDMAN, JOSEPH L., 1050 Park Ave., New York, N. Y., 1959, '60*, '61, '62, '63.
1940. GOODALE, ROBERT L., 330 Dartmouth St., Boston 16, Massachusetts, 1940, '41, '42*, '44*, '46, '47, '49, '50*, '53, '54, '55*, '56, '57*, '60, '61*, '62, '63.
1957. HARRILL, JAMES A., Bowman Gray School of Medicine, Winston-Salem, N. C., 1957, '58, '59, '61, '62, '63.
1960. HARRIS, HERBERT, 907 Hermann Professional Building, Houston 25, Texas, 1960, '61*, '62, '63.
1959. HART, VERLING K., 106 W. 7th St., Charlotte, N. C., 1959, '60, '62.
1942. HEATLY, CLYDE A., 11 North Goodman Street, Rochester 7, N.Y., 1942, '46*, '49, '50, '52, '53*, '54, '55, '57, '58, '60, '61.
1959. HENRY, G. ARNOLD, 170 St. George St., Toronto, Canada, 1959, '61, '63.
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.
1955. HILGER, JEROME A., 350 St. Peter Street, St. Paul 2, Minnesota, 1955, '56*, '57, '58, '59, '60, '62, '63.
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.
1957. HOLMES, EDGAR M., 330 Dartmouth Street, Boston 16, Mass., 1957, '59, '61, '63.
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.

1952. IRELAND, P. E., Room 82, Banting Institute, University of Toronto, Toronto, Ontario, 1952, '53, '54, '55, '56*, '57, '59, '61, '62, '63.
1961. JONES, EDLEY H., First National Bank Building, Vicksburg, Mississippi, 1961, '62, '63*.
1963. KIRCHNER, JOHN A., 333 Cedar St., New Haven 4, Conn., 1963.
1963. LATHROP, FRANK D., 605 Commonwealth Ave., Boston 15, Mass., 1963.
1953. LEDERER, FRANCIS LOEFFLER, 1853 W. Polk Street, Chicago 12, Illinois, 1953, '54, '55, '56, '57*, '58, '60, '61, '62, '63*.
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*.
1961. LEWY, ROBERT B., 25 East Washington St., Chicago 2, Ill., 1961, '62*, '63.
1933. LIERLE, DEAN M., University Hospital, Iowa City, Iowa, 1933, '34, '35*, '37, '38, '39, '40*, '42*, '44*, '46, '47*, '48, '49, '50*, '51, '52, '53, '54, '55*, '56, '57, '58, '59, '60, '62*, '63.
1949. LINDSAY, JOHN R., 950 E. 59th Street, Chicago 37, Illinois, 1949, '50, '51*, '53, '55*, '56, '57, '58, '59, '60, '61, '62.
1952. MCCALL, JULIUS W., 610 Rose Building, Cleveland 15, Ohio, 1952, '53*, '54, '55, '56, '57, '58, '59, '60*, '61, '62*, '63.
1963. MCGOVERN, FRANCIS H., 139 South Main St., Danville, Virginia, 1963.
1951. MCHENRY, LAWRENCE C., 301 N. W. 12th St., Oklahoma City 2, Okla., 1953*, '62, '63.
1933. MCMAHON, BERNARD J., 150 No. Meramec Ave., Clayton 5, Missouri, 1934*, '35, '36, '38*, '41, '44, '46*, '47, '48, '49, '50*, '51, '52, '53, '54, '55*, '56*, '57, '58, '59, '60, '61, '62.

1931. McNALLY, WILLIAM JAMES, 1509 Sherbrooke Street West, Montreal, Canada, 1931, '33, '34*, '39, '44, '47, '49, '49, '52, '53, '54*, '55, '56, '57, '58, '59, '60, '61.
1954. MELTZER, PHILIP E., 285 Commonwealth Ave., Boston 15, Mass., 1954*, '56, '57, '58, '59, '60, '61, '62, '63.
1952. MILLER, ALDEN H., 1407 N. Vermont Ave., Los Angeles 27, Calif., 1952, '53*, '55, '57, '58*, '61, '62, '63.
1958. MONTREUIL, FERNAND, 1123 St. Joseph Blvd., E., Montreal, Canada, 1958, '60.
1954. MOORE, PAUL M., 10515 Carnegie Ave., Cleveland 6, Ohio, 1954, '55*, '56, '57, '59, '60, '61, '62, '63.
1957. MUNOZ-MACCORMICK, CARLOS EDUARDO, 71 Park Terrace, Santurce, Puerto Rico, 1957, '58, '59, '60, '61, '62, '63.
1953. MURTAGH, JOHN A., 2 Maynard Street, Hanover, New Hampshire, 1953, '54, '55, '56*, '57, '58, '59, '61, '62, '63.
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.
1956. NORRIS, CHARLES MORGAN, 3401 North Broad Street, Philadelphia 40, Pennsylvania, 1956, '57, '58*, '59, '60, '61*, '62, '63.
1961. OGURA, JOSEPH H., 640 So. Kingshighway, St. Louis 10, Mo., 1961*, '62*, '63.
1958. O'KEEFE, JOHN J., 255 S. 17th St., Philadelphia 3, Pa., 1958, '59*, '60, '62.
1961. O'NEIL, JAMES J., 612 Medical Arts Building, Omaha, Nebraska, 1961, '62*.
1961. PANG, LUP Q., 1374 Nuuanu Ave., Honolulu 17, Hawaii, 1961, '62*.
1961. PASTORE, PETER N., Box 25, 1200 East Broad St., Richmond, Virginia, 1961, '62*.
1954. PRESSMAN, JOEL J., Department of Surgery, UCLA Medical Center, Los Angeles 24, Calif., 1954, '56*, '59*, '60, '61, '62.

1951. PRIEST, ROBERT E., 79, S. Ninth St., Minneapolis 2, Minn., 1951, '53*, '54*, '56, '59, '60*, '61.
1951. PUTNEY, F. JOHNSON, 1712 Locust St., Philadelphai 3, Pa., 1951, '52, '53*, '54, '55, '56, '58*, '59, '60, '61, '62, '63*.
1951. RAWLINS, AUBREY G., 384 Post Street, San Francisco 8, Calif., 1951, '53*, '56, '58.
1963. REED, GEORGE F., Zero Emerson Place, Charles River Park, Boston 14, Mass., 1963.
1934. RICHARDS, LYMAN GILDER, 12 Clovelly Road, Wellesley Hills 81, Mass., 1934, '35, '36*, '37, '38, '39, '47, '49, '51*, '54, '56, '57*, '59, '60, '61, '62, '63.
1956. RICHARDSON, JOHN ROBERT, 403 Commonwealth Avenue, Boston 15, Massachusetts, 1957, '58, '59, '60, '63.
1945. ROBB, JAMES M., 633 David Whitney Building, Detroit 26, Michigan, 1946, '48, '49*, '51, '52, '55, '56, '57, '60, '61, '62, '63.
1953. ROBERTS, SAM E., 1100 Professional Bldg., Kansas City, Missouri, 1953, '54*, '55, '56, '57, '58, '59, '60, '63.
1959. SANDERS, SAM H., 1089 Madison Ave., Memphis 3, Tenn., 1959, '60, '61, '62, '63.
1934. SCHENCK, HARRY P., 326 South 19th Street, Philadelphia 3, Pennsylvania, 1934, '35, '36*, '37, '38, '39, '40, '41*, '44*, '46, '47, '49, '50, '51, '52, '53, '54, '55, '56, '57, '58*, '59*, '60, '61, '62, '63.
1959. SILCOX, LOUIS E., 102 Lankenau Med. Bldg., Philadelphia 31, Pa., 1959, '60, '61, '62, '63.
1958. SIMONTON, KINSEY M., Mayo Clinic, Rochester, Minn., 1958, '59*, '62.
1939. SIMPSON, W. LIKELY, 512 Physicians' and Surgeons' Building, Memphis 3, Tennessee, 1939, '41, '44*, '46, '47*, '48, '50, '51*, '52, '53, '54, '55, '57, '58, '59, '60, '61, '62, '63.
1962. SISSON, GEORGE A., 1200 E. Genesee St., Syracuse 10, N. Y., 1962, '63*.

1950. SMITH, AUSTIN T., Pennsylvania Hospital, 330 So. 9th St., Philadelphia 7, Pa., 1950, '51, '52, '53, '55, '56, '57, '58*, '59, '60, '61, '62*.
1954. SOOY, FRANCIS A., 490 Post Street, San Francisco 2, Calif., 1954, '56*, '58, '60, '62, '63.
1961. SUEHS, OLIVER W., 14 Medical Arts Square, Austin, Texas, 1961, '62*, '63.
1963. TABB, HAROLD G., 1430 Tulane Ave., New Orleans 12, La.
1947. THEOBALD, WALTER H., 307 N. Michigan Ave., Chicago 11, Illinois, 1947, '48*, '49, '50, '51, '52, '53, '54, '55, '56, '57, '58, '60, '61, '62.
1954. THORNELL, WILLIAM C., 2508 Auburn Avenue, Cincinnati 19, Ohio, 1955*, '57, '59, '60, '63.
1963. TOLAN, JOHN F., 1118 Ninth Ave., Seattle 1, Washington, 1963.
1950. TREMBLE, G. EDWARD, 1390 Sherbrooke St. W., Montreal, Quebec, Canada, 1950, '51, '52, '53, '54, '55*, '56, '57, '58, '60, '61, '62, '63.
1943. VAN ALYEA, OLIVER E., 135 South LaSalle Street, Chicago 3, Illinois, 1944, '46*, '47, '48, '50, '51, '52*, '53, '54, '55, '56*, '57, '58, '59, '60, '62, '63.
1954. WALSH, THEODORE E., 640 S. Kingshighway Blvd., St. Louis 10, Missouri, 1954, '55*, '56, '57, '58, '59, '60, '61, '62, '63.
1962. WEAVER, DELMAR F., 571 Fisher Building, Detroit 2, Michigan, 1962, '63.
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.
1948. WILLIAMS, HENRY L., 2nd, 54th St. & 48th Ave., Minneapolis 17, Minnesota, 1948, '49, '50*, '51, '52*, '53, '54, '55, '56, '57, '58*, '59, '60, '62, '63.
1951. WOODMAN, DEGRAAF, Talbottown Lane, P. O. Box 560, Easton, Md., 1951, '53, '54, '55*, '56, '58, '59, '60, '61, '62, '63.
1953. WORK, WALTER P., University Hospitals, Ann Arbor, Michigan, 1953, '54, '56, '58, '59*, '62, '63*.

Honorary Fellows

1957. (1925) CLERF, LOUIS H., 5575 Eighth Avenue, North, St. Petersburg 2, Florida, 1957, '58, '59, '60, '61, '63.
1960. (1928) HILL, FREDRICK THAYER, Professional Building, Waterville, Maine, 1960, '61, 63.
1943. (1918) LEWIS, FIELDING O., Providence Road, Media, Pennsylvania.
1957. (1930) NEGUS, SIR VICTOR E., 149 Harley Street, London, W 1, England, 1957*.
1957. (1924) PROETZ, ARTHUR W., 12 Westmoreland Place, St. Louis 8, Missouri, 1957, '58, '59, '60, '62.

Corresponding Fellows

1946. ALONSO, REGULES JUSTO E., Mercedes 1233, Montevideo, Uruguay.
1959. BARRETTO, PLINIO DE MATTOS, Ru Cons Torres, Homer 371, Sao Paulo, Brazil, 1960.
1959. BATEMAN, GEOFFREY H., 55 Harley Street, London W.1., England.
1942. BATSON, OSCAR V., 203 South DeKalb St., Philadelphia 4, Pennsylvania, 1942*, '54*.
1943. EGGSTON, ANDREW A., 653 Park Avenue, New York, N. Y., 1944, '49, '55, '60, '61*.
1961. FAABORG-ANDERSEN, K., Central Hospital, Nykøbing, Falster, Denmark, 1961.
1957. HUIZINGA, EELCO, Noorderhaven 7, Groningen, The Netherlands, 1957.
1950. LARSELL, OLOF, University of Oregon Medical School, Portland, Oregon, 1950*.
1957. ONO, JO, 75 Zaimoku-cho, Azabu, Minato-ku, Tokyo, Japan, 1957.
1937. PORTMANN, GEORGES, 25 bis Cours de Verdun, Bordeaux, France, 1947.

1957. RUEDI, LUZIUS, Ramistrasse 100, Zurich, Switzerland, 1957.
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*.
1936. VIALLE, JACQUES, 27 Rue de France, Nice (Alpes Maritimes), France.

Emeritus Fellows

1961. (1936) BALLENGER, HOWARD C., 723 Elm Street, Winnetka, Illinois.
1941. (1923) BARLOW, ROY A., Musquodoboit Harbor, Nova Scotia, Canada.
1951. (1944) BEATTY, HUGH G., 390 Oak St., Apt. No. 35, Columbus 15, Ohio, 1951, '52, '53.
1961. (1930) CAMPBELL, EDWARD HASTINGS, 133 So. 36th St., Philadelphia 4, Pa.
1958. (1930) FIGI, FREDERICK A., Mayo Clinic, Rochester, Minnesota.
1961. (1936) GALLOWAY, THOMAS CYRUS, 636 Church St., Evanston, Illinois, 1961, '62.
1951. (1928) GITTINS, THOMAS R., 408 Davidson Bldg., Sioux City, Iowa.
1963. (1928) GOODYEAR, HENRY M., 556 Doctors' Building, Cincinnati 2, Ohio.
1962. (1950) HALL, COLBY, 16100 Ventura Blvd., Encino, Calif., 1950.
1960. (1940) HANSEL, FRENCH, 634 N. Grand Blvd., St. Louis 3, Missouri, 1962.
1957. (1944) HAVENS, FRED Z., 102 Second Avenue S.W., Rochester, Minnesota.
1955. (1930) HOUSER, KARL M., 2035 Delancey Street, Philadelphia 3, Pennsylvania.
1951. (1928) JARVIS, DEFOREST C., Quarry Bank Bldg., Barre, Vt., 1952.

1960. (1942) KELLY, JOSEPH D., 140 East Fifty-fourth Street,
New York 22, N. Y.
1961. (1950) KLINE, ORAM R., 514 Cooper Street, Camden, N.
J., 1961, '63.
1954. (1927) LUKENS, ROBERT M., 141 West Heather Road,
Wildwood Crest, N. J.
1963. (1941) MARTIN, ROBERT C., 384 Post Street, San Francisco
8, California.
1959. (1927) MYERSON, MERVIN C., 436 North Roxbury Drive,
Beverly Hills, Calif.
1957. (1948) PHELPS, KENNETH A., 1850 Willow Road, Palo
Alto, Calif. 1958.
1958. (1938) SALINGER, SAMUEL, 1260 East Alejo Road, Palm
Springs, Calif., 1958, '59, '61, '62, '63.
1962. (1932) SCHALL, LEROY A., Scudder Lane, Barnstable, Mass.
1961. (1941) VIOLÉ, PIERRE P., 1930 Wilshire Boulevard, Los An-
geles 5, Calif.
1963. (1948) WHALEN, EDWARD J., 750 Main Street, Hartford,
Connecticut.
1951. (1935) WOODWARD, FLETCHER D., 1326 Rugby Road,
Charlottesville, Va., 1959, '60, '61.

BENEFACTORS

DR. JOHN F. BARNHILL	DR. FREDERICK T. HILL
DR. GORDON BERRY	DR. & MRS. GEORGE E. HOURN
DR. EDWIN N. BROYLES	DR. SAMUEL JOHNSTON
DR. WILLIAM E. CASSELBERRY	DR. JAMES E. NEWCOMB
DR. LOUIS H. CLERF	DR. HENRY B. ORTON
DR. CORNELIUS COAKLEY	DR. SAMUEL SALINGER
DR. & MRS. FRANCIS W. DAVISON	DR. SAM H. SANDERS
DR. & MRS. LEE WALLACE DEAN	DR. HARRY P. SCHENCK
DR. ARTHUR W. DEROALDES	DR. BURT R. SHURLY
DR. FRED W. DIXON	MR. LESTER T. SUNDERLAND
DR. WILLIAM E. GROVE	DR. H. MARSHALL TAYLOR
DR. WALTER H. THEOBALD	



Obverse of the deRoaldes medal

RECIPIENTS OF THE DEROALDES AWARD

- 1928. DR. CHEVALIER JACKSON
- 1931. DR. D. BRYSON DELAVAN
- 1934. DR. HARRIS P. MOSHER
- 1937. DR. LEE WALLACE DEAN
- 1943. DR. RALPH A. FENTON
- 1949. DR. GEORGE M. COATES
- 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

RECIPIENTS OF THE CASSELBERRY AWARD

1923. DR. GEORGE FETTEROLF AND DR. HERBERT FOX
1928. DR. RALPH A. FENTON AND DR. O. LARSELL
1929. DR. RICHARD A. KERN AND DR. HARRY P. SCHENCK
1929. DR. EDWARD H. CAMPBELL
1931. DR. ARTHUR W. PROETZ
1934. DR. ANDERSON C. HILDING
1936. DR. FRANCIS E. LEJEUNE: DR. JOEL J. PRESSMAN
1939. DR. H. MARSHALL TAYLOR: DR. BRIEN T. KING
1940. DR. FRENCH K. HANSEL
1941. DR. NOAH D. FABRICANT
1946. DR. PAUL H. HOLINGER
1949. DR. HENRY B. ORTON
1962. DR. HANS VON LEDEN

RECIPIENTS OF THE NEWCOMB AWARD

- 1941. DR. BURT R. SHURLY
- 1942. DR. FRANCIS R. PACKARD
- 1943. DR. GEORGE M. COATES
- 1944. DR. CHARLES J. IMPERATORI
- 1947. DR. HARRIS P. MOSHER
- 1948. DR. GORDON BERRY
- 1949. DR. GORDON B. NEW
- 1950. DR. H. MARSHALL TAYLOR
- 1951. DR. JOHN D. KERNAN
- 1952. DR. WILLIAM J. McNALLY
- 1953. DR. FREDERICK T. HILL
- 1954. DR. HENRY B. ORTON
- 1955. DR. THOMAS C. GALLOWAY
- 1956. DR. DEAN M. LIERLE
- 1957. DR. GORDON F. HARKNESS
- 1958. DR. ALBERT C. FURSTENBERG
- 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

PAST OFFICERS

Presidents

(Year of Election)

1879.	Louis Elsberg	1909.	J. E. Logan
1880.	J. Solis-Cohen	1910.	D. Braden Kyle
1881.	F. I. Knight	1911.	James E. Newcomb
1882.	G. M. Lefferts	1912.	George A. Leland
1883.	F. H. Bosworth	1913.	Thomas Hubbard
1884.	E. L. Shurly	1914.	Alexander W. MacCoy
1885.	Harrison Allen	1915.	G. Hudson Makuen
1886.	E. Fletcher Ingals	1916.	Joseph L. Goodale
1887.	R. P. Lincoln	1917.	Thomas H. Halstead
1888.	E. C. Morgan	1918.	Cornelius G. Coakley
1889.	J. N. Mackenzie	1919.	Norval H. Pierce
1890.	W. C. Glasgow	1920.	Harris P. Mosher
1891.	S. W. Langmaid	1921.	Harmon Smith
1892.	M. J. Asch	1922.	Emil Mayer
1893.	D. Bryson Delavan	1923.	J. Payson Clark
1894.	J. O. Roe	1924.	Lee Wallace Dean
1895.	W. H. Daly	1925.	Greenfield Sluder
1896.	C. H. Knight	1926.	Chevalier Jackson
1897.	T. R. French	1927.	D. Bryson Delavan
1898.	W. E. Casselberry	1928.	Charles W. Richardson
1899.	Samuel Johnston	1929.	Lewis A. Coffin
1900.	H. L. Swain	1930.	Francis R. Packard
1901.	J. W. Farlow	1931.	George E. Shambaugh
1902.	J. H. Bryan	1932.	George Fetterolf
1903.	J. H. Hartman	1933.	George M. Coates
1904.	C. C. Rice	1934.	Dunbar Roy
1905.	J. W. Gleitsman	1935.	Burt R. Shurly
1906.	A. W. deRoaldes	1936.	William B. Chamberlain
1907.	H. S. Birkett	1937.	John F. Barnhill
1908.	A. Coolidge, Jr.	1938.	George B. Wood

1939. James A. Babbitt	1952. Louis H. Clerf
1940. Gordon Berry	1953. Gordon F. Harkness
1941. Thomas E. Carmody	1954. Henry B. Orton
1942-3. Charles J. Imperatori	1955. Bernard J. McMahon
1944-5. Harold I. Lillie	1956. LeRoy A. Schall
1946. Frank R. Spencer	1957. Harry P. Schenck
1947. Arthur W. Proetz	1958. Fred W. Dixon
1948. Frederick T. Hill	1959. William J. McNally
1949. Ralph A. Fenton	1960. Edwin N. Broyles
1950. Gordon B. New	1961. Dean M. Lierle
1951. H. Marshall Taylor	1962. Francis E. LeJeune

Vice-Presidents

1879. F. H. Davis	J. O. Roe
1880. W. C. Glasgow	W. Porter
1881. E. L. Shurly	E. F. Ingals
1882. C. Seiler	S. Johnston
1883. S. W. Langmaid	W. H. Daly
1884. J. H. Hartman	G. W. Major
1885. H. A. Johnson	J. N. Mackenzie
1886. E. C. Morgan	S. W. Langmaid
1887. J. N. Mackenzie	C. E. DeM. Sajous
1888. W. C. Glasgow	C. E. Bean
1889. E. Holden	J. H. Hartman
1890. J. O. Roe	S. Johnston
1891. M. J. Asch	J. C. Mulhall
1892. S. Johnston	W. E. Casselberry
1893. J. C. Mulhall	S. H. Chapman
1894. C. C. Rice	A. W. deRoaldes
1895. J. Wright	D. N. Rankin
1896. T. M. Murray	H. S. Birkett
1897. A. W. MacCoy	F. W. Hinkel
1898. J. W. Farlow	M. R. Brown
1899. T. A. DeBlois	A. A. Bliss
1900. H. L. Wagner	D. Braden Kyle
1901. J. W. Gleitsmann	

1902. G. A. Leland	T. Melville Hardie
1903. J. H. Lowman	W. Peyre Porcher
1904. Thomas Hubbard	W. J. Freeman
1905. J. L. Goodale	C. W. Richardson
1906. G. H. Makuen	A. B. Thrasher
1907. J. P. Clark	J. E. Rhodes
1908. E. Mayer	F. R. Packard
1909. C. G. Coakley	H. P. Mosher
1910. Robert C. Myles	J. M. Ingersoll
1911. F. C. Cobb	B. R. Shurly
1912. A. W. Watson	W. Scott Renner
1913. F. E. Hopkins	Geo. F. Shambaugh
1914. Clement 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. Cuning
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

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	1933. William V. Mullin
1918. D. Bryson Delavan	1935. James A. Babbitt
1919. J. M. Ingersoll	1939. Charles J. Imperatori
1920. George M. Coates	1942. Arthur W. Proetz

1947. Louis H. Clerf
1952. Harry P. Schenck

1957. James H. Maxwell
1959. Lyman G. Richards

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

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 City.
- *1917. Coates, George M., Philadelphia, Pa.
- *1918. Dean, Lee Wallace, St. Louis, Mo.
- *1881. Delavan, D. Bryson, New York City.
- *1893. deRoaldes, Arthur W., New Orleans, La.
- *1890. De La Sota Y. Lastra, Ramon, Seville, Spain.
- *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 City.
- *1907. Jackson, Chevalier, Schwenksville, Pa.
- *1878. Johnston, Samuel, Baltimore, Md.
- *1878. Lefferts, George Morewood, Katonah, N. Y.
- *1914. Levy, Robert, Denver, Colo.
- *1883. Mackenzie, John N., Baltimore, Md.
- *1881. Mackenzie, Sir 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.
- *1909. Semon, Sir Felix, Great Missenden, England.
- *1878. Solis-Cohen, J., Philadelphia, Pa.
- *1889. Swain, Henry L., New Haven, Conn.
- *1914. Thomson, Sir St. Clair, London, Eng.
- *1903. Tilley, Herbert, London, England.
- *1914. Wagner, Clinton, New York City.
- *1890. Wright, Jonathan, Pleasantville, N. Y.

Corresponding Fellows

- *1892. Browne, Lennox, London, England.
- *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.
- *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.
- *1931. LasSagna, 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 City.
- *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

- * 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, Toronton, Canada.
- * 1891. Bryan, Joseph H., Washington, D. C.
- * 1913. Butler, Ralph, 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 City.
- * 1893. Coolidge, Algernon, Boston, Mass.
- * 1937. Crowe, Samuel J., Baltimore, Maryland.
- * 1941. Cuning, Daniel S., New York City.
- * 1913. Dabney, Virginius, Washington, D. C.
- * 1882. De Blois, Thomas Amory, Boston, Mass.
- * 1892. Farlow, John W., Boston, Mass.
- * 1922. Forbes, Henry H., New York City.
- * 1933. Foster, John H., Houston, Texas.
- * 1905. Freer, Otto T., Chicago, Ill.
- * 1940. Gatewood, E. Tribble, Richmond, Va.
- * 1880. Gleitsmann, Joseph W., New York City.
- * 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.
- * 1905. Halsted, Thomas H., Los Angeles, Calif.
- * 1896. Hardie, Thomas Melville, Chicago, Ill.
- * 1915. Hastings, Hill, Los Angeles, Calif.
- * 1888. Hinkel, Frank Whitehill, Buffalo, New York.
- * 1895. Hopkins, Frederick E., Springfield, Mass.
- * 1927. Hubbard, Thomas, Toledo, Ohio.
- * 1919. Hurd, Lee Maidment, Rowayton, Conn.
- * 1920. Imperatori, Charles J., Essex, N. Y.
- * 1904. Ingersoll, John Marvin, Miami, Fla.
- * 1939. Johnston, William H., Santa Barbara, Calif.
- * 1918. Kenyon, Elmer L., Chicago, Ill.
- * 1921. Kernan, John D., New York

- *1929. Kistner, Frank B., Portland, Oregon.
- *1885. Knight, Charles H., New York City.
- *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.
- *1896. Mayer, Emil, New York City.
- *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.
- *1939. Myers, John L., Kansas City, Mo.
- *1922. New, Gordon B., Rochester, Minn.
- *1878. Porter, William, Ocean Springs, Miss.
- *1901. Myles, Robert C., New York City.
- *1923. Newhart, Horace, Minneapolis, Minn.
- *1903. Packard, Francis R., Philadelphia, Pa.
- *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 City.
- *1905. Richards, George L., South Yarmouth, Mass.
- *1878. Robinson, Beverly, New York City.
- *1921. Sauer, William E., St. Louis, Mo.
- *1923. Sewall, Edward C., Palo Alto, Cal.
- *1930. Seydell, Ernest M., Wichita, Kansas
- *1907. Shambaugh, George E., Chicago, Ill.
- *1908. Smith, Harmon, New York City.
- *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.
- *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 City.
- *1942. Ashley, Rae E., San Francisco, Cal.
- *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 City.
- *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 City.
- *1915. Canfield, R. Bishop, Ann Arbor, Mich.
- *1934. Carmack, John Walter, Indianapolis, Ind.
- *1924. Carmody, Thomas E., Denver, Colo.
- *1889. Casselberry, William E., Chicago, Ill.
- *1883. Chamberlain, C. W., Hartford, Conn.
- *1917. Chamberlin, William B., Cleveland, Ohio.
- *1896. Chappell, W. F., New York City.
- *1882. Chapman, S. Hartwell, New Haven, Conn.
- *1902. Coakley, Cornelius G., New York City.
- *1913. Coffin, Rockwell C., Boston, Mass.
- 1918. Cox, Gerald H., New York City.
- *1880. Cushing, E. W., Boston, Mass.
- *1878. Cutter, Ephraim, West Falmouth, Mass.
- *1880. Daly, W. H., Pittsburgh, Pa.
- *1878. Davis, F. H., Chicago, Ill.
- *1941. Davis, Warren B., Philadelphia, Pa.
- 1926. Dennis, Frank Lownes, Colorado Springs, Col.
- *1901. Dickerman, E. T., Chicago, Ill.
- *1878. Donaldson, Frank, Baltimore, Md.
- *1947. Engler, Clarence W., Cleveland, Ohio.
- *1935. Eves, Curtis C., Philadelphia, Pa.
- *1919. Faulkner, E. Ross, New York City.
- *1914. Fetterolf, George, Philadelphia, Pa.

- *1901. Freeman, Walter J., Philadelphia, Pa.
- *1917. Friedberg, Stanton A., Chicago, Ill.
- 1940. Fuchs, Valentine H., New Orleans, La.
- *1897. Frothingham, Richard, New York City.
- 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 City.
- *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 City.
- *1938. Jackson, Chevalier L., Philadelphia, Pa.
- *1880. Jarvis, William C., New York City.
- *1878. Johnson, Hosmer A., Chicago, Ill.
- *1879. Johnson, Woolsey, New York City.
- *1960. Johnston, Kenneth C., Chicago, Ill.
- *1944. Jones, Marvin F., New York City.
- *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 City.
- *1878. Knight, Frederick Irving, Boston, Mass.
- *1898. Kyle, D. Braden, Philadelphia, Pa.
- *1880. Langmaid, Samuel W., Boston, Mass.
- *1878. Lincoln, Rufus P., New York City.
- *1911. Lockard, Lorenzo B., Denver, Colo.
- *1913. Loeb, Hanau W., St. Louis, Mo.
- *1897. Logan, James E., Kansas City, Mo.

- *1935. Looper, Edward A., Baltimore, Maryland.
- *1888. Lowman, John H., Cleveland, Ohio.
- *1919. Lynah, Henry L., New York City.
- *1952. Lynch, Mercer G., New Orleans, La.
- *1915. Lynch, Robert Clyde, New Orleans, La.
- *1879. McBurney, Charles, New York City.
- *1927. McGinnis, Edwin, Chicago, Ill.
- 1936. McGregor, Gregor, Toronto, Canada.
- *1913. McKimmie, O. A., Washington, D. C.
- *1945. McLaurin, John G., Dallas, Texas.
- *1885. McSherry, H. Clinton, Baltimore, Md.
- *1914. Mackenty, John E., New York City.
- *1898. Makuen, G. Hudson, Philadelphia, Pa.
- *1881. Major, G. W., Montreal, Canada.
- *1948. Maxwell, James H., Ann Arbor, Mich.
- *1881. Morgan, E. C., Washington, D. C.
- *1950. Morrison, Lewis F., San Francisco, Calif.
- *1940. Morrison, William W., New York City.
- *1886. Mulhall, J. C., St. Louis, Mo.
- *1925. Mullin, William V., Cleveland, Ohio.
- *1914. Munger, Carl E., Waterbury, Conn.
- *1892. Murray, T. Morris, Washington, D. C.
- *1881. Mynter, H., Buffalo, N. Y.
- *1893. Newcomb, James E., New York City.
- *1895. Nichols, J. E. H., New York City.
- *1927. Orton, Henry B., Newark, N. J.
- *1894. Park, William H., New York City.
- *1892. Porcher, W. Peyre, Charleston, S. C.
- *1927. Porter, Charles T., Boston, Mass.
- *1908. Randall, B. Alexander, Philadelphia, Pa.
- *1882. Rankin, D. N., Allegheny, Pa.
- *1902. Richardson, Charles W., Washington, D. C.
- *1930. Ridpath, Robert E., Philadelphia, Pa.
- *1879. Roe, John O., Rochester, N. Y.
- *1881. Robertson, J. M., Detroit, Mich.
- *1913. Roy, Dunbar, Atlanta, Ga.
- *1878. Rumbold, T. F., St. Louis, Mo.
- *1879. Seiler, Carl, Philadelphia, Pa.
- *1928. Shea, John Joseph, Memphis, Tennessee.
- *1893. Shields, Charles M., Richmond, Pa.
- *1909. Shurly, Burt R., Detroit 1, Mich.

- * 1878. Shurly, E. L., Detroit, Mich.
- * 1892. Simpson, William Kelly, New York City.
- * 1919. Skillern, Ross H., Philadelphia, Pa.
- * 1909. Sluder, Greenfield, St. Louis, Mo.
- * 1879. Smith, Andrew H., Geneva, N. Y.
- * 1932. Smyth, Duncan Campbell, Boston, Mass.
- * 1928. Sonnenschein, Robert, Chicago, Ill.
- 1924. Stein, Otto J., Chicago, Ill.
- 1934. Stevenson, Walter, Quincy, Ill.
- * 1911. Staut, George C., Philadelphia, Pa.
- * 1879. Tauber, Bernhard, Cincinnati, Ohio.
- * 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 City.
- * 1938. Vail, Harris H., Cincinnati, Ohio.
- * 1936. Voislowsky, Antonie P., New York City.
- * 1933. Wanamaker, Allison T., Seattle, Wash.
- 1896. Ward, Marshall R., Pittsburgh, Pa.
- * 1879. Ward, Whitfield, New York City.
- * 1886. Westbrook, Benjamin F., Brooklyn, N. Y.
- * 1924. Wherry, William P., Omaha, Neb.
- * 1924. White, Leon E., Boston, Mass.
- * 1939. Williams, Horace J., Philadelphia, Pa.
- * 1942. Wishart, D. E. Staunton, Toronto, Canada.
- * 1922. Wishart, David J. G., Toronto, Ontario.
- * 1940. Wood, V. Visscher, St. Louis, Mo.
- * 1896. Wollen, Green V., Indianapolis, Ind.
- * 1953. Wilkerson, William W., Nashville, Tenn.

