

















TRANSACTIONS

**Transactions**

**American Laryngological Association**

---

SEVENTY-FIFTH VOLUME



"DOCENDO DISCIMUS"



TRANSACTIONS  
OF THE  
SEVENTY-FIFTH ANNUAL MEETING  
OF THE  
American  
Laryngological Association

1954

HELD AT THE HOTEL STATLER

BOSTON, MASS.

MAY 27TH AND 28TH, 1954

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PUBLISHED BY THE ASSOCIATION

ST. LOUIS, MO.

1954

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FELLOWS OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION - HOTEL STATLER, BOSTON, MASS. - MAY 27, 1934.

THOSE IN ATTENDANCE AT THE SEVENTY-FIFTH ANNUAL MEETING

- Top Row:* Moore, Sooy, McCall, Broyles, Hansel, Weille, Woodman, Tremble, Walsh, Woodward, Equen, Heatly, Eagle, Kline, Lynch, Wilkerson, Lederer, ....., Batson.
- Second Row:* Holinger, Putney, Van Alyea, LeJeune, Whalen, Galloway, Havens, Beatty, Ballenger, Boyden, Engler, Simpson, Beck, Priest, Murtagh, Hickey, Morrison, L. F., Theobald, Maxwell, Jackson, C. L., Davison, Meltzer, Pressman.
- Front Row:* Salinger, Roberts, Tucker, Fenton, Coates, Proetz, Schenck, Cuning, Harkness, Cody, Dixon, Taylor, Clerf, Hill, Brown, McMahon, Hoople, Boies.



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TRANSACTIONS OF THE SEVENTY-FIFTH ANNUAL  
MEETING

OF THE

**American Laryngological Association**

HELD AT

THE HOTEL STATLER, BOSTON, MASS.

MAY 27 AND 28, 1954

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The Seventy-Fifth Annual Meeting of the American Laryngological Association was called to order by the President, Dr. Gordon F. Harkness, at the Hotel Statler, Boston, Mass.

REGISTRATION

The following Fellows were in attendance:

ASHLEY, REA E.	DAVISON, FRANCIS W.
BALLENGER, HOWARD C.	CUNNING, DANIEL S.
BECK, AUGUST LEO	DIXON, FRED W.
BOIES, LAWRENCE R.	EAGLE, WATT W.
BOYDEN, GUY LEE	ENGLER, CLARENCE W.
BROWN, J. MACKENZIE	FENTON, RALPH A.
BROYLES, EDWIN N.	FURSTENBERG, ALBERT C.
CLERF, LOUIS H.	GALLOWAY, THOMAS C.
COATES, GEORGE M.	GOODALE, ROBERT L.
CODY, CLAUDE C.	HANSEL, FRENCH K.

HARKNESS, GORDON F.	MURTAGH, JOHN A.
HAVENS, FRED Z.	NASH, C. STEWART
HEATLY, CLYDE A.	ORTON, HENRY B.
HICKEY, HAROLD L.	PRIEST, ROBERT E.
HILL, FREDERICK T.	PROETZ, ARTHUR W.
HOLINGER, PAUL H.	PUTNEY, F. JOHNSON
HOOPLE, GORDON D.	RICHARDS, LYMAN G.
IRELAND, P. E.	ROBERTS, SAM E.
JACKSON, CHEVALIER L.	SALINGER, SAMUEL
KELLY, JOSEPH D.	SCHALL, LEROY A.
KLINE, ORAM R.	SCHENCK, HARRY P.
LEDERER, FRANCIS L.	SIMPSON, W. LIKELY
LEJEUNE, FRANCIS E.	TAYLOR, H. MARSHALL
LIERLE, DEAN M.	THEOBALD, WALTER H.
LYNCH, MERCER G.	TREMBLE, G. EDWARD
MARTIN, ROBERT C.	TUCKER, GABRIEL
MAXWELL, JAMES H.	VAN ALYEA, OLIVER E.
MCCALL, JULIUS W.	WEILLE, FRANCIS L.
MCCART, HOWARD W. D.	WHALEN, EDWARD J.
MCMAHON, BERNARD J.	WILKERSON, WILLIAM W., JR.
MCNALLY, WILLIAM J.	WILLIAMS, HENRY L.
MORRISON, LEWIS F.	WOODMAN, DEGRAAF

## GUESTS

Walter Aagesen, Anderson, Ind.  
 Lucian W. Ahnfordn, New Orleans, La.  
 A. Sinha, New York, N. Y.  
 Fred S. Arragg, Lawrence, Mass.  
 John J. Ballenger, Evanston, Ill.  
 L. J. Barnes, Los Angeles, Calif.

Benjamin Bofenkamp, Minneapolis, Minn.  
J. J. Baker, Gallipolis, Ohio  
David W. Boisseau, Seattle, Wash.  
D. S. Bolstad, Dearborn, Mich.  
Jules Brahy, Montreal, Canada  
David W. Brewer, Syracuse, N. Y.  
Lester A. Brown, Atlanta, Ga.  
Edgar P. Cardwell, Newark, N. J.  
Joseph A. Costrino, St. Louis, Mo.  
Terrence Cawthorne, London, England  
Marcos Chaimovich, Santiago, Chile  
Claude C. Cody, III, Houston, Texas  
T. L. Curran, Hartford, Conn.  
S. E. Dalton, Atlantic City, N. J.  
K. M. Day, Pittsburgh, Pa.  
Lincoln C. Dickey, Cleveland, Ohio  
H. D. Emswiler, Columbus, Ohio  
Maurice G. Evans, Chestnut Hill, Mass.  
W. H. Evans, Youngstown, Ohio  
Charles F. Ferguson, Boston, Mass.  
George Ferguson, Durham, N. C.  
Jesse S. Fifer, Frederick, Md.  
G. S. Fitz-Hugh, Charlottesville, Va.  
N. F. Gordon, Milwaukee, Wis.  
J. A. Fisher, Asbury Park, N. J.  
Stanton A. Friedberg, Chicago, Ill.  
Joseph L. Goodman, New York, N. Y.  
Glenn Greenwood, Los Angeles, Calif.  
Arthur J. Gorney, Boston, Mass.  
I. S. Gupta, New Delhi, India  
Esme Hadfield, Oxford, England  
A. E. Hammond, Detroit, Mich.  
Fred E. Hamlin, Roanoke, Va.  
H. James Hara, Los Angeles, Calif.  
J. A. Harrill, Winston-Salem, N. C.  
E. W. Harris, Columbus, Ohio  
V. K. Hart, Charlotte, N. C.  
Robert Henner, Chicago, Ill.  
Raymond L. Hilsinger, Cincinnati, Ohio  
Oscar Hirsch, Boston, Mass.  
W. B. Hoover, Boston, Mass.



Leland R. House, Los Angeles, Calif.  
John T. Jenkin, Hilo, Hawaii  
George Kelemen, Boston, Mass.  
Walter D. Klestadt, Fall River, Mass.  
William A. Lell, Philadelphia, Pa.  
Julius Lempert, New York, N. Y.  
Pen Jeng Lin, Taipei, Taiwan  
Carl W. Lupo, Brooklyn, N. Y.  
Carlos E. Munoz-MacCormick, Santurce, Puerto Rico  
J. C. Macdonald, Oklahoma City, Okla.  
A. S. Macmillan, Boston, Mass.  
F. A. Macneil, Winnipeg, Canada  
Marlow W. Manion, Indianapolis, Ind.  
Roland F. Marks, Walnut Creek, Calif.  
W. Raymond McKenzie, Baltimore, Md.  
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Paul M. Moore, Cleveland, Ohio  
L. E. Morrissett, Greenwich, Conn.  
Werner Mueller, Jamaica Plain, Mass.  
Rex Murphy, Denver, Colo.  
Charles M. Norris, Philadelphia, Pa.  
E. W. O'Brien, Rochester, N. Y.  
Joel J. Pressman, Los Angeles, Calif.  
P. E. Provost, Augusta, Me.  
George F. Reed, Boston, Mass.  
John E. Regan, Taunton, Mass.  
Lorenz W. Ruddy, Sacramento, Calif.  
C. R. Salkeld, Bournemouth, England  
James B. Shannon, Montclair, N. J.  
R. Gourishankar, Travancore, India  
Louis E. Silcox, Philadelphia, Pa.  
T. T. Smith, Omaha, Neb.  
Francis A. Sooy, San Francisco, Calif.  
Hilton H. Stothers, New York, N. Y.  
E. A. Stuart, Montreal, Canada  
J. M. Sutherland, Detroit, Mich.  
Harold G. Tabb, New Orleans, La.  
Marvin Tamari, Chicago, Ill.

Ronald Taylor, Vancouver, B. C.  
Richard B. Hadley, Rye, N. Y.  
Theodore W. Walsh, St. Louis, Mo.  
J. S. Wendel, Detroit, Mich.  
George L. Whelan, Philadelphia, Pa.  
William L. Wood, Red Bank, N. J.  
Nelson A. Youngs, Grand Forks, N. D.

## INTRODUCTION TO PRESIDENT'S ADDRESS

The Opening Session of the Seventy-Fifth Annual Meeting of American Laryngological Association, held at the Hotel Statler, Boston, Massachusetts, May 27, 1954, convened at 2:10 P. M., Dr. Gordon F. Harkness, President, presiding.

PRESIDENT HARKNESS: The Diamond Jubilee Meeting of the American Laryngological Association is called to order by this historical gavel presented to this Society in 1911 by Samuel Johnston, J. H. McHarkin, and D. Bryson Delavan. It is one of our treasured possessions in our archives of the Association.

## PRESIDENT'S ADDRESS

### A PERSPECTIVE FROM THE TWILIGHT ZONE

G. F. HARKNESS, M.D.

DAVENPORT, IOWA

Oh! What a wonderful generation

Oh! What a marvelous day

I've got a beautiful feelin'

Everything has gone my way.

There's a bright golden haze

in medicine's meadow

Advancements as high as an elephant's eye

wherever I gaze

Seems like they're climbing clear up to the sky

Oh! What a wonderful contemplation

Oh! What a marvelous day

But what of the coming generation

Greater things will pass their way.

With apologies to "Oklahoma" and "Oh! What A Beautiful Morning."



To have lived in a generation that has seen the birth of the incandescent bulb as a means of illumination; to have heard almost from its beginning the recording of the human voice on the phonograph; to have witnessed the development of electric power in industry and in the home, and the gasoline engine as a motive power in the automobile; all these and more. During this time the old horse streetcar was replaced by the electric street car and it in turn largely replaced by bus transportation, the interurban railroad systems came into being and have practically gone. From the old dirt roads the entire country has a system of paved highways. Even the telephone, in my early youth, was a novelty while now it serves everyone not only locally but nationally and internationally as a means of communication. I vividly remember the thrill of learning what the Wright brothers had done heralding the birth of our modern air transportation from what seemed an almost fantastic beginning. And now with the motion pictures and television we have not only entertainment but a great advance in audio-visual education. Truly a most remarkable generation.

In the vanguard stands the progress of the science of medicine and surgery, preventive and therapeutic. Time does not permit my cataloguing these accomplishments. They are or should be familiar to you except to say that they excell all other accomplishments for the benefit of society.

To have seen the dreaded diptheria relegated to a rarity among the infectious diseases, to have witnessed the introduction and use of the products of the internal glands not alone in their entirety but of some of their components and then synthetic substitutions of the same in modern therapy. In addition, the great aid to human and animal nutrition with the role played by the vitamins. Truly startling contributions. The identification and specificity of micro-organisms in causing disease had its birth and development in a preceding era but it was during my professional life that we learned of antibodies and antigens and the *modus operandi* by which nature overcame these enemies from without. And we are now in that wonderful period thru the use of sulfonamides and the antibiotics of conquering these extraneous foes not only with a shotgun blast but specific means of identification as to sensitivity of individual organisms to these therapeutic aids.

And what does the future offer? A revolutionary era just in its beginning. What with the isotopes and their radio active trac-

ings and with the fission of the atom it behooves us to pause and weigh the possibilities of the destruction of modern civilization as against the peaceful opportunities for a better world for all in which I feel sure, the science of medicine will have its full share. Truly a heritage which you and I have received and one which we should guard zealously and wisely.

Fifty years past our specialty was a lusty member in the medical field. It has a proud inheritance from able predecessors. We can look with pride on men in otolaryngology whose names have joined that galaxy of physicians deserving of being remembered by posterity. But looking back fifty years, what was otolaryngology and why did it attract men out in the highways and byways of medical practice? Today as then the medical profession exists to serve all the people of this land and the greater part of that service has to be rendered out in the highways and byways rather than in just the large metropolitan and teaching centers. Otolaryngology developed and attracted men predominantly as a surgical specialty. We acknowledge that during this period there was a lack of proper appreciation of upper respiratory physiology, the influence of allergy and nutrition and other recognized factors, as we know them today. It was during this era that the theory of focal infections as the cause of many distantly related conditions reached its peak. Lucky indeed were the tonsils and devitalized teeth that, with the slightest suspicion, escaped removal. During this period approved surgical indications presented themselves in such a frequent incidence that men with good basic training could and did develop and maintain a creditable technique out in the highways and byways for many surgical procedures. Also during this period there were otolaryngologists who gave special attention to maxillo-facial surgery; to plastic procedures and oral surgery. But, it is to be remembered that there were other men outside the field of otolaryngology who were devoting time and effort in these now so called borderline fields. The greater incidence of surgical patients in the otolaryngological field made it attractive as a surgical specialty, together with its economic rewards. The question of special franchises for certain geographical areas did not seem to be so important since there seemed to be enough for all.

No group can stand in the way of progress. Inevitable changes come to all phases of life and social orders. Science is probably the most unyielding and dictatorial master crushing opposition that insists on retaining outmoded principles as laid down by our fore-

fathers. Industry recognizes this, retains what is good and charts a new course or falls by the wayside. This applies equally to the phases of medical progress.

The keystone of our medical arch is what is best for the patient in prevention and therapeutic care. It is our solemn duty to examine and approach these problems not from any selfish motives but to avoid disturbing this keystone and if necessary chart a new course.

Let us consider the inevitable changes that have taken place in the otolaryngological field. The science of medicine with the introduction of the antibiotics simply eliminated much of the acute surgery of this field. Recognition of the allergic factors shifted many patients from the surgical side to the medical side of otolaryngology. Elimination of acute conditions from the surgical field has as a part of preventive medicine markedly reduced the chronicities that formerly required surgery. The medical aspect of otolaryngology has assumed a more important position.

If one is going to be a master craftsman within a special field of medicine, assuming good basic training, he must be able to develop within his private practice a sufficient number of certain diagnoses to prevent his deterioration in technique and skill. Even our teaching institutions subdivide the field and do not expect every staff member to be the master in all its subdivisions. The fenestration operation which does not attack pathology but rather by-passes it to improve function demands the most careful diagnostic discrimination as well as social and economic considerations. The surgical technique, most meticulous, under magnification certainly should not be approached lightly by the occasional operator but left to those who have spent many long and arduous hours in preparing themselves and who knows that out of a clear sky perhaps through biochemical research something will be found to influence the pathology of otosclerosis and out the window will go the fenestration operation.

Again major surgery of the larynx for malignancies requires an incidence of patients and properly trained hospital personnel to be able to give the patient all that he is entitled to receive.

From the medical side the American Society of Audiologists, a non medical group, are trained to diagnose and prescribe hearing aids. They may be compared to the otologist as are the optometrists to the ophthalmologists and it is the former who prescribe far more re-



fractive prescriptions. This is no attempt to decry or justify this practice but simply stated as an instance that affects the otologist in private practice.

The current literature of the last few years registers the names of a number of prominent otolaryngologists who have written relative to this so called problem. A partial list includes the names of J. M. Brown, L. G. Richards, F. L. Lederer, H. B. Orton, D. M. Lierle, C. McCaskey, J. M. Robb, R. L. Boies, G. D. Hoople, F. D. Woodward and others together with one American Medical Journal editorial.

An analysis of views expressed reveals a general acknowledgment that important changes have taken place, most of them for the benefit of the patient. Their conclusions however do not parallel their acknowledgements. Some simply scoff at the idea of otolaryngology being a dying specialty, deride the fact that anyone should even suggest such an idea. The author likewise objects to terming otolaryngology a dying specialty. But what is lacking is a constructive program as to how to meet these changes, which are changes in fact, particularly as it affects the private practice of otolaryngology. Time does not permit nor should I take your time repeating the statistical studies of Richards, Lierle, Boies and others. Their statistics verify the fact that fundamental changes have taken place.

While perhaps not germane to my theme the current writings among the teachers shows that otolaryngology has its problems in our teaching institutions. Their conclusions seem to be that these problems are not to be solved by dictums or demands but by mutual agreements in each institution between various departments, with a division of clinical material and that the service delivered will bring its own reward. Some say improve the undergraduate otolaryngological teaching, that this will automatically increase a greater interest in otolaryngology and will automatically fill the authorized residencies in otolaryngology which in 1951 had 33% unfilled out of 408 offered. It has been suggested that undergraduate teaching to the man going into general practice be so improved that the general practitioner will be able to diagnose and take better care of minor otolaryngological conditions and be able to recognize serious pathologies when present. If this suggestion means better care for the patient we should agree but it certainly does not improve the condition of the otolaryngologist in private practice.

Richards made a significant distinction between the science of otolaryngology and the practice of otolaryngology. He wisely stated it was at times difficult to integrate one with the other and confessed an inability to solve the problem. The general consensus of opinion seems to be that in research and scientific otolaryngology there is a most enticing field. This however does not materially affect our problem, if it is a problem.

What of the invasion of medical practice from without the medical profession. As your delegate it took three meetings of the house of delegates before I was able to have established, by the American Medical Association, a definite status and limitations of those with only dental degrees. The American Dental Board of Oral Surgery attempted to establish the rights of their diplomates to have independent hospital status in admitting, treating and discharging hospital patients and for conditions that had no contiguous connection with diseases of the teeth and jaws. As a guide to hospital staff organizations the house of delegates in 1953 unanimously reported that dental oral surgeons should be subordinate to a department of surgery and that dental oral surgery is to be limited to diseases of the teeth and jaws and lesions of contiguous soft tissues directly related to the teeth and jaws but excluding malignancies. Association and cooperation with the dental profession is desirable but not by invasion.

As a resume, one might epitomize the factual conclusions of authors who have written recently relative to otolaryngology. Teachers acknowledge there is a lack of interest among younger men in otolaryngology. They acknowledge that there are problems even in teaching institutions, generally concluding that the solving of departmental differences is to be made at the local level in each institution by mutual agreement. To create greater interest some advocate more time be given to otolaryngology in the under graduate curriculum, others, better training in otolaryngology for the man who is going into general practice so that he can make a proper examination, treat in a better way minor conditions encountered and recognize serious pathology when present. Also, it is suggested that better undergraduate instruction will automatically stimulate interest in postgraduate otolaryngological training. These suggestions are but fostering the individual's hopes and desires. They do not meet the problem as it faces the man in private practice nor does it give the answer to the young man asking for aid in deciding and charting his own course.

Let us not delude ourselves into thinking that our assuming jurisdiction over certain geographical areas is in a way akin to a sovereign state exercising the right of eminent domain for the public good. It is also well to remember our public relations and that the title otolaryngology in the public's and some professional minds is only directly related to conditions of the ear, nose and throat. This has been ingrained in their thinking for a long time and you can't change it by mere assertions.

The time has come, I believe, to acknowledge and point out the factual changes that have taken place. This means cooperation from teaching institutions: this means recognizing the economic limitations of the average man in training: and finally accepting the fact that with present day legitimate aspirations the title of otolaryngology by itself is outmoded in the public and many professional minds.

I believe that in looking into the future one must first acknowledge that changes have taken place and that if we of a special field of the practice of medicine have a program to offer it must be a positive program based on certain fundamentals, divorced from selfish motives from any group and appointed without rancor or anger. General fundamentals should subscribe to the following.

1. The keystone of the practice of medicine is what is best for the patient.
2. The only justification for any special field of medicine is better care for the patient.
3. The program must recognize and be organized to include the care of the general population which is far greater than the clientele of teaching institutions, most of them in the large metropolitan centers.

#### Individual fundamentals:

1. The program must recognize the economic limitations to the man in training.
2. The graduate in medicine, with a proper basic training, in any special field of medicine always remains a doctor of medicine and surgery. Once this concept is lost the individual's horizon becomes so narrowed that he will fail to deliver the services to which the patient is entitled.



3. The prerogatives of a doctor of medicine are wide but with them goes a solemn responsibility not to abuse them. Some of the subdivisions of a special field of medicine may not be for every man basically well trained in the fundamentals of his specialty.

4. The so called border line fields, medically and surgically, are ethically open to the properly trained otolaryngologist but not by any exclusive franchise. Recognition and not antagonism should be shown towards those who by their training honestly feel qualified to work in these so called border line fields without having had a basic training in otolaryngology. One might cite as examples the general surgeon in neck surgery and the maxillo-facial surgeon in some of its variations. Medically one must recognize that the general allergist has grounds for feeling that the upper respiratory manifestations are but part of a general allergic sensitivity.

Fundamentals relative to reaching institutions:

1. A positive program requires the cooperation of autonomous departments, a recognition of the economic limitations of their trainees who are going out into private practice, all combined for the benefit of future patients.

Such a positive program does not mean in any way an attempt to dictate teaching programs but it does seem reasonable that a department of otolaryngology in its residency training should be able to ascertain the inclinations of residents towards these borderline fields and if the department in itself does not offer such training then there should be the opportunity for the trainee to receive basic training in that particular subdivision towards which he is inclined even if received from another autonomous department.

Fundamentals relative to public relations: In private practice one becomes known to a large extent by the title that he individually assumes and one cannot wisely go counter to the conception of that title by the public and his professional colleagues. If one basically trained under the title otolaryngologist and has received sufficient training in the borderline fields of his choice then the title otolaryngologist is not sufficient but there is required the additional title of head and neck surgeon, maxillo-facial surgeon, plastic surgeon of the head or allergist of the upper respiratory tract. It is up to otolaryngological teaching departments to see that he gets sufficient training to assume this title be it within its own department or from some other department.

I offer these simply as observation from one looking in retrospect from the twilight zone of private practice.

A final observation relative to medical meetings and post-graduate courses: formal medical meetings such as this meeting with their scientific programs are most valuable adjuncts to the practice of medicine. Secondly they give the opportunity for acquaintanceship, fellowship and friendships which as one's years in practice lengthen become one of the most cherished possessions of one's professional life. One has only to look over the listings as published and be almost appalled at the multiplicity of medical meetings. To these of national regional type there is added those of local nature, from state to county and hospital staffs until one wonders what time is left for legitimate recreation and quiet reading in one's own study. Many papers of a didactic or research type are better digested when read after publication, in the privacy of one's own library. I would, however, like to comment on what to my mind is one of the finest developments that in the last few years has gained greater impetus. These meetings have not the disadvantage of travel distance and are thereby limited in drawing area. The groups are smaller but are also limited to those with serious purpose of mind and in no way holiday bound. They are not didactic in character but primarily clinical, dry or actual, and offer something that one does not get from the literature. They offer much for the young man who is building, one whose economics do not permit too long an absence from his practice and yet feels it is the time that clinical associations are most valuable. I am referring to the continuation of clinical studies given for a day periodically for a stated number of times for a fee. They differ from staff seminars. Numerous teaching institutions are developing them, even state associations such as Massachusetts has developed programs in four areas of the state to overcome the disadvantage of travel distance. The feature of a fee charge makes the enrollment more selective and increases the responsibility of the teaching staff through clinical discussions to provide something that those in the audience feel that they can take home and make practical application of the knowledge gained.

In closing I want to thank you for patiently having listened to a few of my thoughts and to thank you sincerely for the honor of being able to preside at this seventy-fifth meeting of the most honored and senior society of otolaryngology in this great country of ours.

## BLOOD DYSCRASIAS AND THEIR RELATION TO LARYNGOLOGY

WILLIAM DAMESHEK, M.D. (By Invitation)

BOSTON, MASS.

This is the 75th Anniversary of this august Society. It so happens that this year marks the 100th Anniversary of the birth of Paul Ehrlich, the great German immunologist, chemist, hematologist and chemotherapist. Paul Ehrlich was born 100 years ago and at about the time this Society was founded began his studies in the staining reactions influencing blood cells. From that he went on to specific chemotherapeutic methods and to the development of the field of immunology and its relationship to hematology. That is a great anniversary in hematology and I thought I might mention it at this time.

Hematology has, I must say, relatively little relationship to the field of otolaryngology and it is therefore with considerable trepidation that I speak to this Society in this field.

Hematology is largely concerned with the blood cells, namely, the red blood cells, the white blood cells, the blood platelets. It is concerned with the blood forming tissues which are the bone marrow, the lymphoid tissue and the reticulo-endothelial tissues as well as those of the plasma cells, and it is also concerned with the hemostatic mechanisms.

With respect to the blood forming tissues there can be no doubt that in infancy and early childhood the lymphoid tissue is dominant in comparison with the bone marrow; thus in infancy and childhood, lymphoid tissues throughout the body are what might be termed hyperplastic in terms of the adult tissues. Therefore in infants and children there are larger lymph nodes throughout the body. They have larger tonsils and adenoids than the adult population and I often wonder whether large tonsils and adenoids so commonly sacrificed with tonsillectomy may perchance be removed unnecessarily, because after all lymphoid tissue is an important functional element of the entire body having to do not only with removing material which is deleterious but also in the production of antibodies. As time has gone



on the site of antibody production has gradually shifted from the reticulo-endothelial system, in which it was supposed to be most active, towards the collection of lymphocytes which are present in the various lymphoid bearing areas throughout the entire body and the tonsils and adenoids are largely collections of lymphoid tissue. It is distinctly possible that the large tonsils and adenoids of infancy and childhood attest to the temporary dominance of lymphoid tissue in that age period.

With respect to blood cells and their reactions in connection with laryngology, the first cell that I would like to discuss is the red blood cell. When red cells are reduced anemia is stated to be present. The other day I mentioned with respect to the very interesting moving picture by the Japanese Takahara, that with chronic iron deficiency over a lengthy period of time, iron is depleted in *all* the body cells, not only in the red blood cells. An individual with chronic iron deficiency develops not only a low color index or hypochromic anemia but actually a lack of iron in all his body tissues, with the result that various changes develop in most of the body cells. Thus the tongue becomes atrophied, cheilosis develops at the corners of the mouth, changes in the esophagus occur. These changes have been known to laryngologists for many years under the designation of the Plummer-Vinson syndrome. These authors described the relationship of low color index anemia with a peculiar disturbance of the esophagus characterized by dysphagia, due to thinning out and web-like formations of the esophagus, also by the presence of koilonychia that is, spoon finger nails. More recently Waldenström has termed this disturbance *sideropenic dysphagia* which means dysphagia due to iron deficiency.

Recently I saw another type of deficiency in association with an esophageal abnormality, a type that had never been previously described. Dr. Orvar Swensen at our hospital performed an esophago-enterostomy for congenital atresia of the esophagus, quite a formidable operation as you know. After the lapse of some years the child developed an anemia of the pernicious anemia variety with high color index, large blood cells and the like. It seems that with the stomach having been bypassed over a period of years, there finally developed a lack of intrinsic factor with the result that the individual developed a B<sub>12</sub> vitamin deficiency and finally "pernicious" anemia. That sort of situation although rare might conceivably develop in other similar detour operations of the esophagus.

Now with respect to the white blood cells, a deficiency in white blood cells of an extreme degree in which the white cell count may go down to levels of 100-500 has been well known to laryngologists under the name of agranulocytic angina or agranulocytosis for a good many years. This disorder was first described by Schultz of Germany in 1925 as agranulocytic angina. More recently we have been talking about it as agranulocytosis. In the great bulk of the cases the disorder is secondary to the use of one or another chemotherapeutic agent. As we have gone along in this era of antibiotics we see more and more reactions to these remarkable drugs of which agranulocytosis is one. Aminopyrine was, of course, the drug chiefly implicated in agranulocytosis and finally was legislated out of existence in Denmark and in various other countries and largely went out of use in this particular country. However, its place has been taken in this country and elsewhere by a new material called butazolidin. Butazolidin being almost identical with pyramidon, cases of agranulocytosis are now being reported in various parts of the country in response. The reaction is due to the development of a drug hypersensitivity, which brings me to the types of reactions which drugs may produce or induce in various individuals.

Some individuals develop what appears to be a direct attack by the drug on the bone marrow with the result that anemia or leukopenia, or thrombocytopenia, or pancytopenia, namely a total bone marrow involvement, takes place. On the other hand, some individuals develop a reaction on the blood forming tissues by an indirect mechanism, and it is in this indirect mechanism that we have been most interested in recent years. This has to do with the development of autosensitization or auto-immunization. Paul Ehrlich, to allude to him again, felt that the individual could not develop an *auto*-antibody, that is, an antibody that could attack his own cells but it has been demonstrated not only in our work but in the work of others that *auto*-immunization affecting the red cells, white cells and platelets may result as the end product of drug or other hypersensitivity. This indirect reaction has assumed increasing importance in the development of hemotoxic reactions. Aminopyrine and butazolidin I referred to as the chief white cell reactors but I should put in a word of caution about the use of antihistaminics which, if used in considerable concentration over a period of years, and intermittently administered over a period of time, may also result in agranulocytosis. So even the antihistaminics or antiallergic drugs may induce agranulocytosis.

The therapy of agranulocytosis, as you know, has been revolutionized by the use of antibiotic agents. It was with some trepidation, a number of years ago, that we used for the first time an antibacterial agent in the treatment of this disorder. At first we administered sulphathiazole, but with the advent of penicillin, the results were even more gratifying and of no possible harm to the bone marrow. The outlook in agranulocytosis, which was death in the great majority of cases, is now relatively favorable providing antibiotics are in high concentration.

Another white cell reaction which is of considerable interest from the laryngological standpoint is infectious mononucleosis. If the tonsils are not present the tonsillar tabs become considerably hypertrophied and the curious thing is that the individuals having this disease develop a very severe pain as the result of the infectious mononucleosis, an angina. You look at the throat in such cases and you find relatively little, but the individual complains of considerable pain. Some individuals who do have their tonsils with infectious mononucleosis develop a very severe tonsillitis and the various signs of Vincent's angina affecting the throat. In that disease the infectious agent, whatever it might be, presumably a virus, causes a very striking lymphoid hyperplasia throughout the entire body, involving not only the lymph nodes and the tonsils and adenoids but the mesenteric nodes within the abdomen, the spleen and various other

parts of the body. Thus the individual with mononucleosis develops a generalized white cell reaction, at times resembling leukemia. Acute leukemia as you know is a generalized white cell proliferation of neoplastic type involving either lymphoid structures throughout the body or the bone marrow or the reticuloendothelial cells. The acute leukemias with total involvement of the entire bone marrow result not only in white cell disturbances and proliferation of nodes but in hemorrhagic manifestations to which I will allude in a moment.

Lymphosarcoma is one area where the laryngologist and the hematologist may come together at times. Lymphosarcoma involving the tonsil is a very distinct entity. Lymphosarcoma represents a localized white cell tumor involving lymphoid tissue anywhere in the body. Thus lymphosarcoma may arise in a lymph node. It may arise in the spleen. It may arise in the mesentery of the mediastinum and it may arise in the tonsil as well. Under such circumstances considerable unilateral tonsillar enlargement may develop. Our ther-



apy of lymphosarcoma and of leukemia has advanced considerably with the advances made in chemotherapeutic methods in the last several years. Whereas previously we had only x-ray therapy we now have such potent chemotherapeutic agents for leukemia and lymphosarcoma as aminopyrine, the nitrogen mustards, the antifolics, triethylene melamine or T.E.M., and various other chemicals that are of distinct importance in this field.

The next type of disorder which is probably of most importance to you is that of the hemorrhagic disturbances. The hemorrhagic disturbances may be of importance in any special field of medicine. They may be subdivided into those with (1) platelet deficiency or thrombo-cytopenia, (2) those with a coagulation disturbance of which hemophilia is an example, and (3) those having to do with disturbance of the blood vessels. There is another type of hemorrhagic disturbance which the pulmonary surgeons and the urologists particularly may come in contact with. I allude to the fibrinolytic mechanism where suddenly the blood clot is lysed very quickly and very marked hemorrhage ensues.

Thrombocytopenia may arise as an idiopathic disease as you know. Thrombocytopenic purpura may arise secondarily to leukemia but in any event the absence or distinct lowering of the platelet level is a very important thing. The idiopathic disorder may develop acutely in an otherwise well or previously well individual. Thrombocytopenia may develop as the result of drug sensitivity. Individuals taking quinidine intermittently for various cardiac disturbances may suddenly develop thrombocytopenia, bleeding from the mouth, blood blisters, and bleeding from the throat. Ordinarily thrombocytopenia with lesions of the buccal mucosa and pharynx is associated chiefly with acute leukemia and if one does see thrombocytopenia in the buccal mucosa or elsewhere, particularly in association with gingival manifestations, the possibility of acute leukemia should be kept in mind.

There have been a good many developments in the field of thrombocytopenia and its therapy and in the preoperative management of the individual having thrombocytopenia. We have been using recently plastic bag blood for the preservation of platelets. We use fresh blood which is put into plastic bags, that is, the donation of blood is made not in a glass bottle but in a plastic bag (Fenwal Co. of Framingham, Mass.) and we find that the platelets are better

preserved. This medium is water repellent, and these plastic bags are utilized for fresh blood transfusions, particularly in hemorrhagic disease. They may also be used for fresh blood transfusions for other disorders.

The coagulation disorders namely the hemophilias, have always been of importance in laryngology. I can well remember my intern days when I was first on the so-called Blood service at the Boston City Hospital when we went to see a boy who had been operated upon for peritonsillar abscess and it turned out that he had hemophilia with a considerable hemorrhage into the peritonsillar space. Of course his management was exceedingly difficult as can be imagined.

The blood vessel disorders have some relationship to the laryngologist. For example, streptococcus sore throat, which may be quite severe, in some children is followed by a lag period of something like a week or two in some cases and then by a generalized vascular purpura, known as the Henoch-Schönlein type purpura. Henoch-Schönlein purpura is probably due to an auto-immunization affecting the blood vessel wall with the development of a vascular purpura. Periarteritis nodosa is a disorder which is being seen in increasing incidence, I think largely because of the increasing use of the antibiotics. But the blood vessel disturbance that we have to deal with chiefly in relationship to laryngology is that of hereditary telangiectasia or the so-called Rendu-Osler-Weber disease. In that condition there is no hematologic disturbance whatever. The red cells, white cells and platelets are perfectly normal but the blood vessel walls throughout the body and particularly at exposed areas are unusually fragile, dilated and tortuous. Under such conditions severe epistaxis may develop from the nose, and the individual may have nose-bleeds very frequently. Our experience with that disease has not been a very good one. Patients with hereditary telangiectasia have been cauterized on repeated occasions without much of any effect and various other local measures have been used in that disease without too much effect too. Finally after a period of years patients with this disease develop a very severe hypochromic iron deficiency anemia, which is very difficult to control.

That finally brings us to the question as to whether or not routine bleeding studies should be performed in the field of laryngology with particular relationship to the tonsillectomy operation. Should an individual have a bleeding time, a clotting time and platelet count,

for example, as screening measures in the prophylaxis of tonsillectomy? In my own experience I find that this is hardly of any value whatever and I think myself that the bleeding time as it is ordinarily performed in the clinical laboratory, the coagulation time as it is ordinarily performed using capillary tubes can well be dispensed with. I do believe though that routine careful questioning of the patient and the patient's family for any possibility of bleeding disturbance in the past is of distinct value. From the standpoint of whether or not to do routine bleeding studies prior to tonsillectomy, my choice would be rather to take a very careful history, not only of bleeding in the patient but of bleeding in the patient's family.

I have given you some stray remarks about some possible relationships between laryngology and hematology. I hope they have been to some extent informative.

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#### DISCUSSION

DR. ROBERT L. GOODALE: I would like to ask Dr. Dameshek, if he has any comment to make on the so-called pseudohemophilia, about which Dr. Jacobson has spoken so frequently here.

DR. SAMUEL SALINGER: I want to corroborate what the essayist had to say about routine coagulation and bleeding time as a means of discovering whether a patient will bleed, particularly in his T. & A. operation. We have performed this routinely in a large series of cases and were very much surprised at times to find a patient with very low bleeding and coagulation time to have a very severe hemorrhage. Contrary wise I do agree with him that the personal history is very important. We always make it a point to inquire into it. The information thus obtained may be very valuable.

DR. WILLIAM DAMESHEK: It was good to have my old classmate, Bob Goodale, ask a question with reference to the hemorrhagic disturbance that we have called pseudohemophilia. In the last few years there has been a tremendous advance in hemophilia. Now hemophilia has been subdivided into various types, including PTA and PTA deficiency.

With reference to pseudo-hemophilia, that is a disturbance in which the prime characteristic is an increased bleeding time and a tendency on the part of the individual to bleed excessively in response to small traumas, such as minor operative procedures. That this is a distinct entity I am quite certain. It may be of familial origin. It may be transmitted by either the mother or father. It does not have the same hereditary patterns as hemophilia. It has no particular importance unless the individual requires an operation, minor or otherwise, and unfortunately it is not helped too well by transfusions as is hemophilia; so that local therapy becomes all important. There one has to use gel foam with thrombin



and various other measures of that type. Pseudo-hemophilia is not a very well defined entity by some, but on the other hand, I think it is something to be cognizant of and it can only be recognized by a bleeding time disturbance. Normal bleeding time is one to three minutes. In that condition it may go up to 8, 10, 15, 20 minutes.

I am glad to hear that my remarks about the routine use of coagulation time and bleeding time are corroborated by an experienced otolaryngologist. I think that there has been altogether too much attention paid to these routine studies which frequently do more harm than good, because they are done in routine fashion and are often of little, if any value.

Mild cases of hemophilia that bleed excessively with operation may have a perfectly normal coagulation time in a glass tube and particularly in a capillary tube. Capillary tube estimations of coagulation time are absolutely worthless and many estimations of coagulation time in small glass tubes are also worthless. The best way to get around this is to do coagulation times in silconized tubes where the matter of glass surface is disposed of by water-repellant medium corking the glass.

The bleeding time is also a very difficult test to be certain of and unless done accurately and by very careful technique, utilizing the forearm, it is apt to be inaccurate. Therefore again I would recommend that these routine tests be done away with and the patient and the family asked very carefully about bleeding manifestations.

Thank you very much for your attention.

## THE USE OF STAPHYLOCOCCUS TOXOID IN OTOLARYNGOLOGY

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The following clinical observations on the use of Staphylococcus toxoid in otolaryngology are presented as a preliminary report, as further investigation must be conducted with the correlation of bacteriologic, immunologic, cytologic and hematologic studies.

For many years we<sup>1</sup> have emphasized the value of cytologic studies of the secretion of the respiratory tract in the diagnosis of allergy and of infection. More recently, Bryan<sup>2</sup> has shown that changes in epithelial cells of the respiratory secretions may be considered as characteristic of virus infection. With the introduction of the antibiotics, there has been a change in the patterns of infection as well as some alteration of virulence of certain organisms. The use of routine bacteriologic studies in the practice of otolaryngology is imperative today, and the development of effective vaccine therapy is now just as important if not more so than previously.

During the past 50 years various antigenic and allergenic agents such as bacterial, fungus and virus vaccines, non-specific proteins, chemical agents, pollens, dusts, etc.—have been introduced into the field of therapy, chiefly as injectable substances. The therapeutic application of these various agents has been based upon the principle of developing the tolerance of the patient by beginning with a small dosage and increasing it gradually until satisfactory results were obtained. If an exacerbation of symptoms or a focal reaction occurred, either the dosage was reduced or the treatment was discontinued. In other instances general reactions were noted to appear usually only after the larger doses had been given. With certain agents employed in the treatment of specific conditions, good results have been obtained. On the other hand, many failures have been encountered, and yet in general there has been no tendency to modify the original principle of this "build-up" type of dosage.

The principle of the use of the small optimum effective dose, especially in the injection of vaccines was introduced as long as forty years ago but the method never received general recognition in spite of the fact that it offered many advantages such as the absence or avoidance of all types of reactions, as well as a high percentage of good therapeutic results.

In certain infectious diseases, such as measles, mumps, scarlet fever, small pox, typhoid fever, poliomyelitis, etc.—long lasting or permanent immunity either follow the attack or may be induced artificially in some instances by the injection of vaccines. By the use of vaccines, some of these diseases may be reproduced but the ideal method is one by which it is induced in a very attenuated rather than in the severe natural form. There are many infectious conditions such as those which affect the respiratory, intestinal, genito-urinary tract, the skin and special organs, caused by a variety of gram-negative and gram-positive organisms for which vaccine therapy has proved to be either non-effective or if so, of very short duration. For these conditions the antibiotics have been most effective.

Although the very great therapeutic value of the antibiotics is appreciated there is evidence that their use is being abused in that they are administered in instances in which they are not indicated or are given in too large doses, sometimes over a long period of time. Allergic and other side reactions especially those resulting from the disturbance of the normal flora of organisms in the respiratory and gastrointestinal tracts and the development of antibiotic resistant strains, are not infrequently encountered.<sup>3</sup> Secondary monilial infections involving, particularly, the mouth and gastrointestinal tract may also complicate the picture.

With the wide-spread use of the antibiotics, there has appeared clinical evidence of a decrease in immunity or resistance to infection, especially in the respiratory tract. We have encountered a significant number of instances of repeated upper respiratory infections and pansinusitis in young children, especially in those to whom antibiotics, and particularly nose drops, have been administered repeatedly. Some of these children have not passed through the normal course of a cold or sore throat without antibiotics. Since the acute infection has been attenuated in this manner, there is no definite stage of resolution reaction, at which time the acute process quickly improves and subsides. The natural immunity to the organisms concerned is



not stimulated or increased so susceptibility to reinfection occurs. In many cases in which reinfection occurred, especially during the fall and winter, we noted a high incidence of residual sinusitis. Large quantities of muco-pus could be evacuated by suction or by displacement and suction. It is evident that these children never recovered completely from the head colds, so the residual infection in the sinuses also played a part in reinfection.

In the adult we have encountered a significant number of cases of monilial infections of the mouth, tongue, tonsils, soft palate and pharynx, secondary to the use of antibiotics. In several instances a recurrence of this infection reappeared if antibiotics were taken as long as six to twelve months later. In two cases, the ingestion of beer after many months, reproduced a marked glossitis and stomatitis. From these observations it is apparent that the restoration of the normal flora of bacteria may be a slowly delayed process.

Although there has always existed a great need for effective methods of immunization to the common bacterial infections, such as colds, sore throats, pneumonia and various types of skin infections especially those caused by the staphylococcus, with the introduction of the antibiotics that need has become much greater. In a large percentage of the cases of infection of these types, because of their mildness, the use of antibiotics is not indicated. On the other hand an effective vaccine would be not only indicated but also preferred. Its use may be imperative in those instances in which the antibiotics are not tolerated. A type of therapy in which smaller doses of antibiotics may be administered to attenuate the infection combined with the use of effective vaccine dosage offers the possibility of a more safe and sane method of treatment, which is not likely to be followed by the above complications.

In discussing the matter of chemotherapy, Mellon<sup>4</sup> emphasizes the importance of not neglecting the stimulation of the natural defensive powers. His presentation on the relation of so-called "host factors" in modern chemotherapy are worthy of consideration. In 1935, Mellon<sup>5</sup> reported his observations on the use of antistreptococcal serum for the purpose of depriving hemolytic streptococci of their virulence. Of a series of 125 patients with severe subacute infections from this organism which usually caused a fatal outcome in many instances, 90 per cent recovered with the use of this serum.

It was believed that the effects obtained were not the result of specific antibodies contained in the serum, because the doses which produced maximum effects were very small, averaging from 0.01 cc to 0.10 or 0.20 cc. The majority of the patients in this series exhibited varying degrees of sensitivity to horse serum. It was noted incidentally, that a mild degree of serum sickness often heralded the early and complete recovery of the patient. This was especially true when this complication disguised itself as an arthritis. The action of specific antibody also could not explain Mellon's observation of the healing of varicose ulcers of long-standing which had become infected with the erysipeloid type of hemolytic *Streptococcus*. Neither could the clinical responses manifested by a leucocyte rise as well as an increase in blood calcium, blood phosphate and sometimes the chlorides, be attributed to an antiserum. There was also a disappearance of general and local edema associated with severe infections. Mellon believes that the mechanism of action is a "desensitizing" or an "anti-shock" effect. He refers to the good results previously reported from the production of hyperpyrexia and the use of non-specific protein, especially when these agents were not employed in too large doses. The production of shock reactions from overdosage were responsible for obscuring the undoubted clinical value associated with the small, desensitizing doses, which presumably are anti-shock in effect. The size of the dose therefore, must be considerably under the toxic or shock threshold. Mellon also found that the nonspecific effect could be produced by antidiaphtheritic serum as well as anti-streptococcic serum. Apparently, antibodies, tissue enzymes or other antibacterial substances are liberated when the small optimum dose is employed. The reaction may be nonspecific and the response of the "anamnesic" type.

Ehrich<sup>6</sup> has demonstrated experimentally that large doses of antigen depress the immune mechanism and localize the antigen with damaging tissue effects. Antibody response is depressed, as manifested by the prevalence of large monocytes and histocytes. Low doses result in antibody production as shown by the predominance of lymphocytes and plasma cells.

Although various bacterial and virus vaccine preparations have long been in use, there has been in general a lack of a standardization of materials as well as dosage. Since there is always some variation in individual responses or a range of optimum effective dosages, more difficulty is added to the problem where there is a lack of reliable

standardization. Some antigenic preparations are much more potent than others. Marked local or general reactions may be produced with comparatively small doses of some vaccines or only with large doses or more concentrated material with others. Mixed stock or autogenous respiratory vaccines are usually prepared from organisms such as streptococci, staphylococci, pneumococci, H. Influenza, M. Catarrhalis, etc., The organisms are killed or attenuated by heat or formaldehyde treatment. Then standardization is based upon the number of killed organisms per cc; this is usually 2,000 million per cc. This is not a very reliable standardization of potency. Some observers use the intradermal skin test as a measure of potency or of individual susceptibility or sensitivity. Serial dilution titration testing, by which the end point or weakest dilution which will produce a minimum positive reaction, may be of some value in determining potency or therapeutic dosage. A stock vaccine may be standardized clinically with some degree of accuracy by using it over an extended period of time on a large number of patients.

A trend toward the use of small doses of virus vaccines in which no reactions are produced is exemplified in the recent works of Salk and his associates.<sup>7</sup> They employed an emulsification of a large number of strains of influenzal viruses in light mineral oil. These vaccines were much more effective than similar aqueous preparations. It was found that there was no advantage in employing more than the quantity of virus necessary to achieve the maximum antibody level. High antibody levels lasted as long as four months.

Using the same technic of preparing emulsified vaccines, Salk<sup>8</sup> more recently has perfected a poliomyelitis virus vaccine which has been biologically tested on monkeys and which is now available in limited quantities for clinical trials.

Various bacterial toxins converted into the attenuated state as toxoids have been more effective on the whole than the standard killed bacterial preparations.

In general there is therefore a trend in the direction of the employment of attenuated live, yet still potent materials which are subjected to some form of biological standardization. Furthermore there is a trend towards the use of smaller doses not accompanied by reactions of any kind and doses which are maintained at an optimum effective level.

## STAPHYLOCOCCUS TOXOID

Among the infections which are encountered in the field of otolaryngology, those caused by streptococci and staphylococci are the most important to be considered. Vaccine preparations of these two types of organisms have long been in use. A standardized toxoid preparation, however, has been available only for the staphylococcus. In some commercial preparations, staphylococcus toxoid is combined with staphylococcus vaccine. Streptococcus vaccine may also be included in this combination. If a streptococcus vaccine is employed with staphylococcus toxoid the optimum range of effective dosage must be determined for each separately before being used in a combination.

At the present time we are concerned in the report of our observations on the use of staphylococcus toxoid only, rather than on a combination of vaccines and toxoids. On the basis of the use of staphylococcus toxoid over an extended period of time on a variety of clinical cases, we have been able to establish certain basic therapeutic principles of dosage which are also applicable to the employment of other vaccines and toxoids as well as to a variety of allergenic and pharmacologic agents (Fig. 4).

Staphylococcus toxoid has been used for the prophylactic treatment of recurrent boils, carbuncles, styes, and pustular acne since 1933. It has been suggested that the reactions noted in some cases indicate a hypersensitivity to staphylococcus proteins. The best results were noted in patients with a high sensitivity.

*Standardization:* Staphylococcus toxoid is prepared from a highly hemolytic strain of *Staphylococcus aureus* and is tested for its ability to stimulate antitoxin production. A unit of toxoid is the amount obtained from a dermonecrotizing unit of toxin (the least amount of toxin which by intradermal injection in a susceptible rabbit will produce an erythema, with a central necrosis at least 5 by 5 mm in diameter). Detoxification of the toxoid is effected with formaldehyde. It is carried to the point where the intradermal injection of 0.1 cc into the skin of the susceptible rabbit will no longer cause necrosis.

*Tolerance Test:* The manufacturers recommend a preliminary toxoid sensitivity test before the institution of treatment because in rare instances local and general reactions to 10 units (0.1 cc no. 1 dilution, 100 units per cubic centimeter) have been noted.



The sensitivity test is made by the intracutaneous injection of one unit (0.1 cc of a 1:10 dilution of Dilution 1). At the end of 24 hours the test is read and recorded as 1+, 2+, 3+, or 4+ according to the following results:

- 1+: Erythema less than 10 mm
- 2+: Erythema 10 to 15 mm
- 3+: Erythema over 15 mm without necrosis
- 4+: Over 15 mm with necrosis (rare)

*Dosage:* The initial dosage based on the above tests is as follows:

- 1+ reaction—0.10 cc Dil. 1 (100 $\mu$  per cc) or 0.01 cc of Dil. 2 (1,000  $\mu$  per cc), (10 units)
- 2+ or 3+ reaction—0.10 cc of a 1:10 dilution of Dil. 1 (1 unit)
- 4+ reaction—0.10 cc of a 1:100 dilution of Dil. 1 (0.1 unit)

By this plan of treatment, injections are recommended by the subcutaneous or the intramuscular route and are given twice weekly. In general, the dosage is increased by 0.2 cc with each injection until the top dose of 1.0 cc of Dilution 2 (1,000 units) is reached. This usually requires about 10 injections. After the maximum dosage is established, the injections are given at weekly, sometimes at three to four week intervals.

In the above discussion, it was stated that the best results were obtained in patients with the highest degree of sensitivity. In these instances, very small doses were used. The initial dose was as small as 0.1 unit. In the treatment of furunculosis of the ear canal and styes, we noted very dramatic effects with doses of from 1/100 to 2/10 unit. These results suggest that the small optimum dosage is more satisfactory and that the build-up to the maximum tolerance of 1,000 units is unnecessary.

*Metallergic Hypersensitiveness—The Anamnestic Reaction:* In the use of staphylococcus toxoid injections we have noted that satisfactory responses may be obtained in certain allergic conditions, especially the allergic dermatoses as well as in a variety of bacterial infections with various organisms. In other words a non-specific effect may be obtained.

Urbach<sup>9</sup> refers to the term "metallergy" which was introduced to denote the fact that a specifically sensitized organism will respond to subsequent exposures of another type with specific allergic reactions presenting the same clinical picture as that elicited by the first allergen. The phenomena has also been called the "anamnesic reaction." For example, Mackenzie and Fruehbauer<sup>10</sup> have shown that rabbits sensitized to egg-white will after a while show no trace whatsoever of circulating antibodies to egg in their blood; these antibodies will reappear immediately, however, after the injection of typhoid vaccine. The same phenomenon has been noted in the use of bacteria instead of egg white. In an animal sensitized to a specific organism, antibodies could be made to reappear later following the injection of another different organism. This anamnesic effect is very strikingly reproduced by the therapeutic injection of staphylococcus toxoid. For example an ear canal infection caused by *pseudomonas aeruginosa* may respond very promptly to an optimum dose of staphylococcus toxoid.

It is generally known that in allergic individuals acute intercurrent infection (e.g. common cold, erysipelas) can for a while totally eliminate the allergic symptoms. The occurrence of a strictly specific shock of this type renders the organism temporarily incapable of reacting to other antigens. In other words an organism in combatting infection is not in a position at the time to create a sufficient number of antibodies to another antigen or allergen. With the appearance of the common head cold in an individual with nasal allergy the complete disappearance of the allergic process is exemplified by the total temporary replacement of eosinophiles by neutrophils in the nasal secretions.

In the treatment of such allergic dermatoses as urticaria, infantile eczema and contact dermatitis we have been able to obtain very satisfactory responses often following the first injection of staphylococcus toxoid. So far we have not been able to reproduce such startling results in respiratory allergy. Failure to do so has probably been the result of not establishing the proper optimum dosage. The non-specific effects obtained in the treatment of allergic conditions with staphylococcus toxoid have been produced also by a variety of non-specific agents such as peptone, tuberculin, pyrogen, milk, sulfur in oil, etc. Since staphylococcus toxoid is a standardized preparation it is a much more satisfactory material to employ because optimum effective doses can be more readily determined.

REPORTS ON USE OF STAPHYLOCOCCUS TOXOID  
IN OTOLARYNGOLGY

In 1941 Townsend<sup>11</sup> made a very comprehensive report of a group of 394 cases of acute recurrent rhinitis in which staphylococcus toxoid was employed in treatment. He used the skin test titration method to determine the size of the initial doses. From an analysis of the skin reactions, culture findings and results of treatment he noted that the patients appeared to be divided into two major groups:

Group I. (a) Frequent endemic head colds, (b) negative skin reactions, (c) staphylococcus albus (and aureus) predominant, (d) tolerant to increasing doses of staphylococcus toxoid, and (e) favorable results.

Group II. (a) Infrequent epidemic febrile throat and nose colds, (b) positive skin reactions, (c) occasionally staphylococcus aureus (streptococci predominant), (d) intolerant to increasing doses of staphylococcus toxoid, (e) unfavorable results.

On initial intracutaneous dose of three units (.03 of the No. 1 Dilution 100 units per cc) was used in the selection of cases for treatment. Townsend designated his method as that of the low dosage type, but the doses were low only in comparison to the massive ones usually employed. For example, he stated that the total dosage of 660 units in adults and 991 units in children was approximately one-fifth to one-tenth of the usual amounts of toxoid administered (3800 to 10,000 units). Of the entire group of 394 patients 193 were treated with toxoid. Sixty-five per cent had good results and 35 per cent had doubtful or unfavorable results. Of the cultures taken from the nose 66 per cent showed pure staphylococci. Of the 394 persons tested 63 per cent showed negative skin reactions.

As the result of his extensive studies Townsend stated that, "The cause of the different phenomena observed in different persons with the use of staphylococcus toxoid is unknown."

In the routine management of all cases of frequent and infrequent head colds it is of primary importance to determine whether the existing condition is infection or allergy. Sometimes the allergic patient has an acute infectious cold. The cytologic examination of the nasal secretions for the presence of neutrophils or eosinophils offers the only absolute method of making an accurate diagnosis.

In Townsend's series of cases no cytologic determinations were reported. It is evident therefore, that considerable difficulty was encountered in classifying the cases since no differentiation was made between infection and allergy.

Upon analysis of his two groups of cases it is reasonable to assume that Group I represented chiefly the allergy patients, because of the history of frequent colds, the predominance of staphylococcus albus which is usually a contaminant or a part of the natural flora, the tolerance to increasing doses of staphylococcus toxoid and the favorable results. In our experience allergic patients may have favorable results from the non-specific shock effect of the large doses.

The patients classified in Group II were quite evidently those with infections. As will be pointed out later patients with acute staphylococcus and streptococcus infections can tolerate only very small doses, for in this group the proper immunologic response to the infection must be obtained or the results will be unfavorable. If the dosages are even just slightly above the optimum level the infection is aggravated and the patient becomes worse instead of better.

In their report of clinical observations on the use of autogenous vaccine made from cultures of coagulase-positive hemolytic staphylococcus aureus Goodale and Mangiaracine,<sup>12</sup> a number of important points were emphasized in the treatment of bacterial infections of the eye and the respiratory tract. It was found that the production of coagulase was considered the best indicator of pathogenicity. Noncoagulase producers were considered as saprophytes or contaminants. Although staphylococci have been considered to be non-encapsulated in young (three-hour) rapidly-growing cultures capsules have been demonstrated. These organisms were thought to be preferable for the preparation of autogenous vaccines because of their greater antigenicity.

Although coagulase-positive hemolytic staphylococcus aureus is commonly prevalent in the nose and throat it is not pathogenic unless the balance between its virulence and the patient's immunity is upset in its favor. We are in agreement with Goodale and Mangiaracine that the antibiotics lower the resistance to bacterial infection. The occurrence of repeated respiratory infections in young children who had antibiotics for every acute episode is evidence to support this contention.



In their entire group of 113 cases representing various types of infection of the eye and respiratory tract Goodale and Mangiarcine reported that 88 or 78 per cent were definitely benefited by vaccine therapy.

The initial dose of vaccine employed in treatment was .20 cc of a 1 to 100 dilution given intramuscularly. This was repeated on three successive days, then a rest period was allowed for four days. Following this same plan of weekly schedule the dosage was gradually increased until at the end of the fifth week 1 cc of undiluted vaccine was administered.

In the discussion of the above paper Fenton<sup>13</sup> suggested the use of low dosage but did not state the amount or the dilution employed. He suggested that most satisfactory cultures from the nose are obtained from the region above the posterior end of the middle turbinate.

Clerf<sup>14</sup> expressed the opinion that in vaccine therapy very small doses should be given. He further stated that the whole problem of immunity is the production of antibodies and the individual must have infection in order to develop them.

In the preparation of antigenous vaccines from the streptococcus and pneumococcus, Mangiaracine pointed out that it is almost impossible to stimulate antibody formation when using beta streptococci or pneumococci unless living cultures are used. More and more evidence is appearing in the recent literature to indicate that attenuated live bacterial and virus cultural materials are being employed in the preparation of vaccines with greater antibody production and with better therapeutic results.

#### PRINCIPLES OF SMALL OPTIMUM DOSAGE

The principles of an optimum effective dosage are based upon certain specific physiologic or immunologic responses. The responses follow a very definite pattern and apply to a great variety of antigenic and allergenic agents. Vaccine therapy based upon these principles was introduced long ago but the plan has been followed by only a few observers. In 1926 Crowe<sup>15</sup> reported his observations of the "Treatment of Chronic Arthritis and Rheumatism" in a small volume. In 1928 he opened the Charterhouse Rheumatism Clinic in South London. In a revision of his book in 1932 he<sup>16</sup> reported a

comprehensive summary of 662 cases. It was stated that between 80 and 90 per cent of the patients received benefit from vaccine therapy.

The vaccines used consisted of 157 strains of streptococci and two types of staphylococci (*albus* and *aureus*). They were made into a stock vaccine which was administered to this group of patients. The stock preparation was found to be more satisfactory than antogenous preparations. At first the initial dose was established at 500,000 organisms but on account of reactions it was reduced to 50,000. The smallest dose employed was 100 organisms. In some instances reactions were noted from this dosage but no attempt was made to reduce it further. Later, however, Small<sup>17-18</sup> who began his observations on the Crowe principle in 1927, reduced the dosages even below one organism. Since there was a limit for measuring dosages in terms of organisms, he began the use of bacterial extracts with which dosages were calculated on a weight-volume basis. His extracts were prepared according to the following plan: 1 - 10 dilution =  $10^{-1}$ , 1 - 1000 =  $10^{-3}$  etc. In the treatment of rheumatoid arthritis doses as low as  $10^{-16}$ ,  $10^{-15}$ ,  $10^{-14}$  were employed.

#### REACTIONS AND RESPONSES

The reactions and responses of small doses of various agents, especially *Staphylococcus toxoid*, which we have noted, correspond closely to those reported by Crowe and by Small in the use of bacterial extracts in the treatment of arthritis. There is a definite zone of dosage in which sharp exacerbations of symptoms follow the injection. This zone is just above the optimum or the effective one. We have noted, for example, that if one unit of *Staphylococcus toxoid* causes an exacerbation of a dermatitis, an urticaria, or an auricular furunculosis, a dosage of about 1/10 to 2/10 of a unit is followed by a satisfactory response. Acute exacerbations last only one or two days and may be followed by improvement. Dosages immediately above the reactive range become less pronounced in effect and with still higher amounts, no effect at all is produced. When very large doses are eventually administered, a general reaction manifested by lassitude, drowsiness, general aching, malaise, headache, anorexia and possibly a slight rise of temperature is produced. It is noteworthy that the larger of the microdoses, which do not produce evident reactions, are still much smaller than those generally employed in large dosage therapy (Fig. 4).

With micro—or minute dosage, a certain amount of a given dilution may cause a sharp exacerbation of symptoms, yet 100 times this amount may cause no apparent effect. Even repeated doses within this range produce no reactions. On the other hand, the one-hundredth part of this dose, which was followed by an exacerbation of symptoms, may upon repetition cause repeated flare-ups or reactions.

A dosage established below that which causes an exacerbation of symptoms is the amount which produces the optimum therapeutic effect. The relief of symptoms usually lasts only three to four days but upon repetition of the same dosage, the effect is repeated. As a rule, the length of the period of relief increases as treatment is continued. If the period of relief last only one to two days, or if the intervals of relief between injections do not eventually lengthen, then an increase in the dosage is indicated. If a focal reaction occurs on the same dosage then a reaction is indicated.

#### TECHNIC OF MICRODOSAGE

As will be pointed out later we learned that the effective dosage should be approached from the very weak ineffective zone to the one which produces the most satisfactory results, thus avoiding the zone of reaction. In other words, the zone of reaction should not be established first and then the dosage reduced below this.

Trial doses of the weakest dilutions may be given at three- to five-day intervals, increasing each five to tenfold until the zone of effectiveness is reached. It is not necessary to proceed with larger doses to produce a focal reaction; as a matter of fact, it is not advisable. If intermediate rather than the weakest doses are employed in the beginning, a dose which is too large may cause a marked exacerbation of symptoms lasting for several weeks, making it difficult to establish the effective dose. As a rule, a rest period should be allowed until the exacerbation has completely subsided and immunologic balance has become restored.

*Analyses of Trial Injections:* In the treatment of arthritis Crowe points out that four conditions may arise following a trial injection:

1. No change—Dosage too small.

2. Improvement—Beginning within a few hours after the injection. Pain, stiffness and swelling become less. In furunculosis of the ear canal, relief of pain often occurs also within a few hours. In these instances, the dosage is correct or nearly so.
3. The joints become worse within 24 hours. This represents a *focal reaction* and the dosage is too large.
4. The patient becomes worse—such symptoms as lassitude, drowsiness, general aching, etc., indicate a *general reaction*, consequently, the dosage is very much too large.

The plan of subsequent dosage may be summarized as follows:

Trial Injection		Improve-	Focal	General
No. 1 results in:	No change	ment	Reaction	Reaction
With Trial	Increase	Maintain	Reduce	Reduce Dosage
Injection No. 2:	Dosage	Dosage	Dosage	Drastically

*Focal and General Reactions:* A very mild focal reaction lasting less than a few hours is not especially significant but would indicate a slight reduction of dosage, say about 50 per cent. If the reaction lasts longer than 12 hours, the dosage should be reduced by ten times.

General reactions are of greater significance as the symptoms may last for several weeks. After the reaction has entirely subsided, it is advisable to start a trial dosage 100 to 1,000 times weaker. It is really more advisable to begin trial doses with the very weakest dilutions thus avoiding focal or general reactions. A focal reaction is more noticeable to the patient than a mild general reaction. The latter should not be overlooked.

*Subsequent adjustment of Dosages:* As pointed out repeatedly elsewhere, initial effective doses may be administered in chronic cases at three- to five-day intervals, later five to seven, ten, fourteen, twenty-one and twenty-eight-day intervals. If the intervals of relief do not gradually and progressively lengthen, the dosage may be increased by 50 per cent or more, but too much increase should not be made in an effort to establish a longer interval too early in the course of treatment. An exacerbation of symptoms may result. After injection intervals have been lengthened to three or four



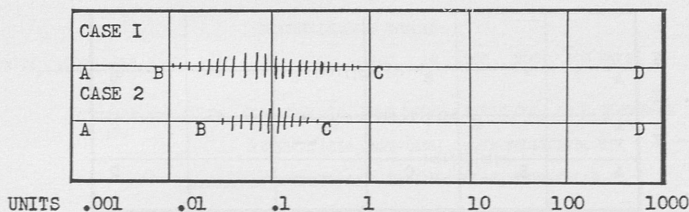


Fig. 1.—Ranges of reaction belt: Case 1 belt wide, Case 2 belt narrow.

weeks, further treatment may be discontinued but in some instances, may have to be continued for a year or longer.

In the use of this method of therapy it is important to learn the indications for manipulating the dosage. If the patient is showing satisfactory response the dosages must not be increased. It is usually advisable to try even a smaller dosage for in some instances we have noted that after several injections of the same amount there is a slight focal reaction. This indicates a reduction in dosage of about one-fifth.

*Reaction Zones:* The focal reaction zone below or above which no reactions occur varies in different individuals. The maximum is within 100, the minimum within ten times the increase above the effective range.

In Figure 1 the lines A B C D represent the entire range from ineffective dosage to general reaction. A B represents optimum effective dosage. B C represents the focal reaction range. The up and down movements of the broken line indicate the degree of reaction. Within this range note that the degree of reaction increases then decreases with increasing dosage, and that above C there is no reaction and incidentally no therapeutic effects. When very large doses are given such as near point D general reactions may be produced. Within this latter range there may be a nonspecific shock reaction with some therapeutic effect.

In case 1 the effective dosage lies between .001 units and about .01. The focal reaction range is from .01 units to 1 unit. In case 2 the effective range is below .05 units. The range of focal reaction is less than in case 1.

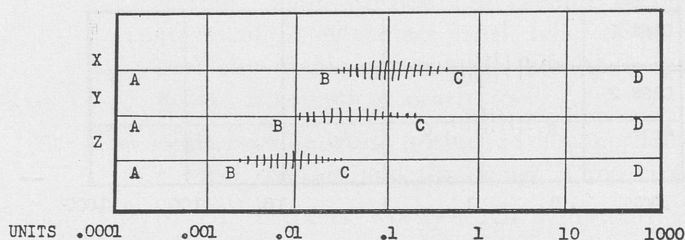


Fig. 2.—Change in range of dosage with prolonged therapy.

When treatment is extended over a prolonged period of time the range of effective dosage tends to decrease or shift to the left as shown in Fig. 2. X represents the situation at the beginning of treatment, Y after 2 to 6 weeks and Z after 6 weeks. If for example to top effective dosage is  $\frac{1}{2}$  unit at X, later it may drop to  $\frac{1}{10}$  unit and finally to  $\frac{1}{100}$  unit or less.

#### CLINICAL USE OF STAPHYLOCOCCUS TOXOID

When *Staphylococcus toxoid* was introduced in 1933 we used it in the treatment of furunculosis of the ear canal but without satisfactory results. Further use was discontinued until about ten years ago when we discovered that certain small doses ( $\frac{1}{10}$  to  $\frac{1}{2}$  unit) were effective in the treatment of urticaria. Later satisfactory results were noted in the treatment of various types of dermatitis such as infantile eczema, adult eczema and contact dermatitis. More recently we<sup>3</sup> found it effective in the treatment of the serum disease reactions following the administration of penicillin, such as urticaria, laryngeal edema, arthritis etc. The antiallergic effect of *staphylococcus toxoid* is non-specific as a variety of other bacterial and antigenic agents will produce the same effect. The evidence suggests that there is a stimulation of ACTH production.

Since small doses of *Staphylococcus toxoid* were effective in the treatment of allergic conditions, the application of this principle to the treatment of infections, such as furunculosis of the ear canal, hair follicle infections, in the vestibule of the nose etc. was found also to produce satisfactory results.<sup>19-20</sup> More recently it has been used in the treatment of acute respiratory infections with some instances of remarkable success (Fig. 3).

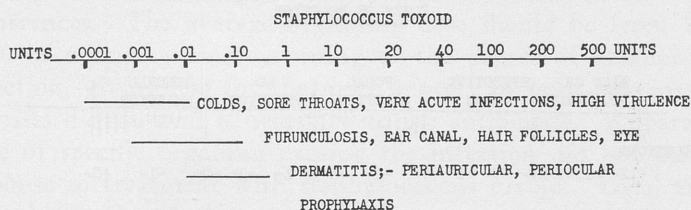


Fig. 3.

*Respiratory Infections:* Colds, sore throat, tonsillitis, acute sinusitis and bronchitis, serous otitis. For many years we have employed mixed respiratory vaccines in various weak dilutions for the treatment of these infections but the results were questionable and inconsistent, no doubt because of the lack of standardization of the materials and most important the failure to find the optimum range of effective dosage. More recently we began the use of staphylococcus toxoid employing the same dosages as established for furunculosis and for allergic conditions but no significant results were noted. Finally upon a more drastic reduction in doses by 10 to 100 times, definite immunologic responses were noted. More observations correlated with the bacteriologic findings and with invitro sensitivity tests must be carried out but the outlook in this field appears promising.

In some recent studies Tunevall<sup>21</sup> in Sweden investigated the behavior of certain respiratory infections in children at different ages. The behavior of the specific antibodies to such organisms as pneumococci, hemolytic streptococci, hemophilus influenzas, and pyogenic staphylococci were especially noted. Each of these organisms was found to have a predilection for a special age and it was noted that the antibody titers, as well as the ability to produce antibody in acute infections rose with increasing age. This development proceeded at different rates for different antibodies. Tunevall emphasizes the importance of these studies in the approach to the problems of the common infections in children at different ages. In 1949 ström<sup>22</sup> and his associates reported that in scarlatina, early antibiotic treatment greatly reduces the formation of antibodies. Tunevall does not advise the administration of antibodies for the milder respiratory infections but suggests that they be given in case of purulent complications.

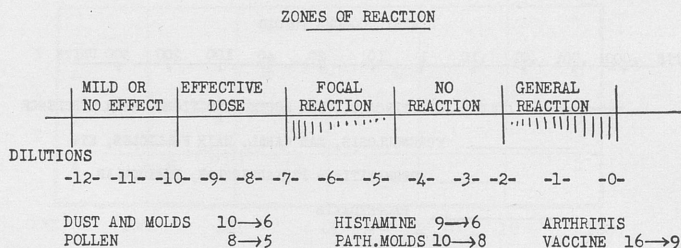


Fig. 4.

With the widespread use of antibodies today there is evidence that many of the respiratory tract infections are very much attenuated or less virulent than formerly encountered. There is apparently less antibody stimulation and less immunity produced. Following the use of antibiotics, antibody formation is further reduced. A knowledge of the proper use of bacterial vaccines to stimulate natural immunity and an abundance of antibodies is now a very timely problem.

In the administration of staphylococcus toxoid for respiratory and other infections we have noted that smaller doses (1/1000 to 1/100 units given every 3-4 days) are indicated with the more virulent infections. Further studies will probably show that vaccine dosage will have to be modified also according to whether it is employed before or after the use of antibiotics and according to the dosage of antibiotics administered. In some instances we have noted that the administration of Staphylococcus toxoid with a minimum of effective antibiotic dosage (250 to 500 mg of terramycin, acromycin, erythromycin or equivalent of other antibiotics) was followed by very satisfactory results.

*Skin and Hair Follicle Infections:* Since the Staphylococcus is a normal inhabitant of the skin, and since the skin is an important immunologic organ, maximum responses from the intradermal injection of optimum doses should be expected. In a group of 60 cases of furunculosis of the ear canal the response to vaccine and local treatment was noted within a few hours. In early cases the process was completely aborted. Pain was promptly relieved. In more advanced cases there was definite improvement and course of the disease



was shortened. By maintaining the injections at three to four day intervals until the infection had completely subsided there were no recurrences. The average beginning dose should be from 1/100 to 1/10 of a unit, varied according to the degree of acuteness of the infection, giving less for the more acute. In only two instances in 60 cases did we find it necessary to use antibiotics. Apparently the type of specific organism causing the infection did not modify the response to treatment with staphylococcus toxoid. Local treatment must be employed also as indicated.

In the treatment of hair follicle infections and infections of the skin of the vestibule of the nose, similar satisfactory results may be obtained. Seborrheic dermatitis or infection involving the canal and auricle as well as the eye brows which commonly spreads from the scalp promptly responds to toxoid therapy especially when combined with the use of Selsun (Abbott) shampoo on the scalp. In the eye with such conditions as styes, marginal blepharitis and conjunctivitis, responses to toxoid therapy also have been satisfactory.

*Allergic Dermatitis:* Dermatitis affecting the auricle and ear canal, the periauricular area, the periorbital region and adjoining parts of the face may be of the true atopic type, a contact dermatitis or a fungus allergy or infection (*Candida albicans*). The dermatitis may be complicated by secondary bacteria infection. The local application of Neocortef ointment is also advisable in most cases. Wet packs, lotions, ointments and powders are also used as indicated. Urticaria and angioedema may affect the various parts of the face, the lips, tongue, epiglottis, larynx and esophagus. The lesions may be localized or a part of a general reaction. In these cases the dosage of Staphylococcus toxoid varies from 1/100 to about 2/10 unit given intradermally at 3-4 day intervals (Fig. 3). In cases of fungus allergy or infection we have added the injection of an extract of a combination of *Monilia*, *Trichophyton* and *epidermophyton* in doses of .10 cc of 1 to 1 billion to 1-100,000,000. In general the doses should be gradually reduced if satisfactory results are to be maintained, thereby avoiding focal reactions because of a lowering of the threshold of tolerance.

*Post-Antibiotic Complications:* These complications consist chiefly of serum disease like reactions, fungus infections (*Monilia*), loss of normal flora of bacteria in the respiratory, gastro-intestinal and genito-urinary tracts, the development of infection of antibiotic

resistant organisms and the loss or reduction of normal bacterial immunity.

In the treatment of serum disease reactions, especially those caused by penicillin, manifested by urticaria, angioedema, generalized dermatitis, fever, joint pains etc. the response to *Staphylococcus* toxoid in doses of from 1/10 to 1/2 unit has been very striking. If toxoid administration alone is not satisfactory the administration of a short course (4-5 days) of ACTH and/or Cortisone therapy should produce prompt results.

The complications with monilial infection especially those observed on the tongue, cheeks, soft palate, tonsils, epiglottis, pharynx and esophagus respond to the injection of *Staphylococcus* toxoid combined with a monilial extract. The addition of injections of B-complex and the oral ingestion of acidophilis buttermilk or Lactinex Tablets and the avoidance of yeast containing foods such as bread, cheese, beer etc. should also be instituted.

The administration of *Staphylococcus* toxoid is also indicated in cases of infection with antibiotic resistant organisms especially with *Staphylococci*. Perhaps bacterial or virus vaccines should always be administered with all of the antibiotics. Certainly there is need for the restoration of natural bacterial immunity in these instances in which it is reduced from large doses or the repeated administration of antibiotics. It has been suggested that the antigenic stimulation of immunity by bacteria is absent when antibiotics with bacteriolytic properties are administered. And when bacteriostatic effects are produced the organisms are phagocytosed so very little if any antigenic material remains.

#### SUMMARY

1. *Staphylococcus* toxoid has proved to be of definite value in the treatment of certain infectious and allergic conditions encountered in otolaryngology such as: furunculosis of the ear canal and vestibule of the nose, seborrheic dermatitis of the auricles and face, acute respiratory infections, contact and atopic dermatitis of the auricle, periorbital area, nasal vestibule, urticaria and angioedema of the lips, tongue, pharynx and larynx; post-penicillin serum disease reactions and lowered immunity resulting from antibiotic administration.

2. The effectiveness of *Staphylococcus toxoid* is dependent upon the establishment and maintenance of an optimum small dosage.

3. Optimum dosage range is calculated upon the basis of certain immunologic responses. Effective doses were found to be below that which cause a focal reaction.

4. An increase or decrease in dosage is made only with the idea of maintaining the effective range.

5. No progressive build-up in dosage is recommended.

6. No general or constitutional reactions are encountered.

7. These principles of optimum dosage are applicable in the use of other bacterial or virus vaccines as well as a variety of other antigenic and allergenic agents.

8. The establishment or stimulation of natural immunity by use of vaccines should be employed in combatting infection in every instance in which it appears advisable, instead of giving antibiotics.

9. The antibiotics decrease the production of antibodies and lower natural immunity.

10. Combined vaccine therapy with minimum doses of antibiotics may be advisable.

11. Bacteriologic, immunologic and cytologic studies should be correlated with these therapeutic procedures.

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## DISCUSSION

DR. ROBERT L. GOODALE: Dr. Hansel kindly sent me a copy of this paper to read. Inasmuch as at the Massachusetts Eye and Ear Infirmary the Ophthalmological Department has had some experience with the staphylococcus toxoid, I was induced to undertake a few studies on the use of autogenous vaccine, with the same purpose that Dr. Hansel mentions of supplementing the antibiotic approach to these infections with a more positive approach, that is, one of the active immunization. Dr. Scott and Dr. Proctor of the Eye Service carried out some experiments with staphylococcus toxoid. I think they had about 50 cases of staphylococcus conjunctivitis. They came to the conclusion that it was not as effective as the autogenous vaccine which Miss Mangiaracine of our Bacteriology Department made of young, seven hour cultures of coagulose positive hemolytic staphylococcus aureus. With that as the present approach in our department we find we are getting some very good results. With regard to small doses to avoid producing general reactions, I think we should be very careful not to produce



marked general reactions. The question of dose is I think an individual one. I have not had the experience with the very minute doses that Dr. Hansel has used.

At any rate we are getting some very good results with the vaccine. I think it would be nice to know more about the toxoid. I have no personal experience but I know from the experience of the members of the Eye staff on the other side of our house that they have not had as good results. I would like to know what brand he uses. It might make a difference.

DR. FRANCIS W. DAVISON: I am not ready to state that Dr. Hansel has the final answer with regard to correct doses, but I agree with his principles. We can benefit many patients with low grade infections by immunization with vaccines, vaccine filtrates or toxoids. I also agree with the principle just stated by Dr. Goodale that we should keep the doses small enough to avoid systemic or focal reactions.

In defense of clinical investigation, I should like to call your attention to the man who first used foxglove. He did not know what he had, but he knew that he was helping his patients.

DR. FRENCH K. HANSEL: Staphylococcus toxoid is available from several commercial sources. We usually obtain the No. 1 Dilution which contains 100 units per cc, and make serial dilutions from it.

In the use of vaccines the effective dosage is more easy to determine with standardized materials. Since the patient is always a variable factor some of the difficulty of establishing dosage is therefore minimized.

The value of the use of small dosages has been emphasized by many observers. Mellon reported in 1935 that small amounts (.02 to .05 cc) of antistreptococcus serum would produce remarkable immunity and improvement in cases of serious streptococcus infections such as cellulitis. The effect was not necessarily specific for similar results could be obtained with diphtheria or tetanus antitoxin.

## THE VENOUS NETWORKS OF THE NASAL MUCOSA

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Recording the variations in the size and arrangements of blood vessels has occurred since the beginning of anatomy. Curiously, in these lists, be they in English, French, German or Japanese, there is no mention of the variations of the vessels nor the architectural patterns within the nose. This is true for both the arteries and the veins. The reasons for this lack are easy to find. Formerly, the nose was treated as an organ like the spleen or kidney. Variations are listed for the anterior facial vein or the internal maxillary artery, but only for their extra-nasal course. The last seventy-five years, the life span of the American Laryngological Association, has seen the birth and development of rhinology as a specialty. As late as 1880, there were arguments as to whether the head mirror was a useful instrument. Opponents contended that the mirror should be fixed to the light rather than to the head, and the observer and patient oriented accordingly. The detailed study of the nose began in 1882 with Zuckerkandl's<sup>1</sup> well-illustrated *Anatomy of the Nasal Cavity*. The small size of the nasal vessels makes them difficult to dissect. Further, the general dissecting room has little time to spend on parts concerned with a medical specialty. Zuckerkandl's descriptions of the nasal blood vessels were augmented by accounts of serious bleeding in clinical practice. Among the early reports was the one of Little,<sup>2</sup> who described bleeding from the lower anterior part of the septum. A few years later, Kiesselbach<sup>3</sup> went over the same ground. The terms "Little's area" and "Kiesselbach's area" denote a region of the septum prone to bleed. We have assumed the bleeding to be due to a peculiar arrangement of blood vessels located here. Some of us have used one eponym for venous bleeding and the other eponym for an arterial flow. Close survey of papers of Little and Kiesselbach does not justify this differentiation. Kiesselbach

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was indefinite while three of Little's four cases were definitely arterial bleeding. Neither author described the anatomy of the vessels. Perhaps we should use the eponyms to describe the area without any presumptions as to the vascular pattern. Recently Professor Swindle,<sup>4-6</sup> making use of his extensive familiarity with comparative forms, gained through association with zoological collections, has given detailed accounts of the nasal arteries and veins and their finer connections. He reported the arrangement in the reindeer and the kangaroo since he considered these forms to represent types. He described interrelations of vessels rather than their gross distribution. Harper<sup>7</sup> in 1947 gave a preliminary report of his studies of the venous network in the human nose. In his summary he indicated that the arterial supply had a course in the periosteal layer or in the bone itself and that the finer ramifications of the arteries broke up into the rich venous network of the substantia propria of the nasal tissues.

Detailed study of the finer ramifications of the vascular system cannot be made by dissection. Injection methods are necessary to see the small vessels. After the injection has been made, there are several ways of handling the material. One of the methods is that of corrosion. This consists in using an injection mass which becomes solid and then, by one of the several means, destroying all of the surrounding tissues and retaining only a cast of the interior of the vessels. The method began with Schwammerdan in 1672. A somewhat parallel method, probably just as old although its age is difficult to determine, uses a dried specimen. Here the vessel is filled with a mass, and the soft parts allowed to lose substance in cold running water for two or three weeks, after which the parts are dried. The filled vessels now stand up sharply against the shriveled remains of the soft tissues. Most of our older book illustrations of vessels were made from specimens prepared by this method. A modern method is generally attributed to Spalteholz,<sup>8</sup> who in 1911 placed on a firm scientific basis the making of so-called transparent preparations. The Spalteholz technique was used by Swindle. Recently I have had the good fortune to have available for corrosion specimens a plastic mass which obviates most, if not all, of the drawbacks of the earlier masses. I owe this to the cooperation of Dr. Ernest Rose and the High Polymer Research Laboratory of H. D. Justi and Son, Inc., dental manufacturers in Philadelphia. This mass is a methyl-methacrylate which can be made to polymerize at room temperature. The shrinkage is not more than 15 per cent. Intimate

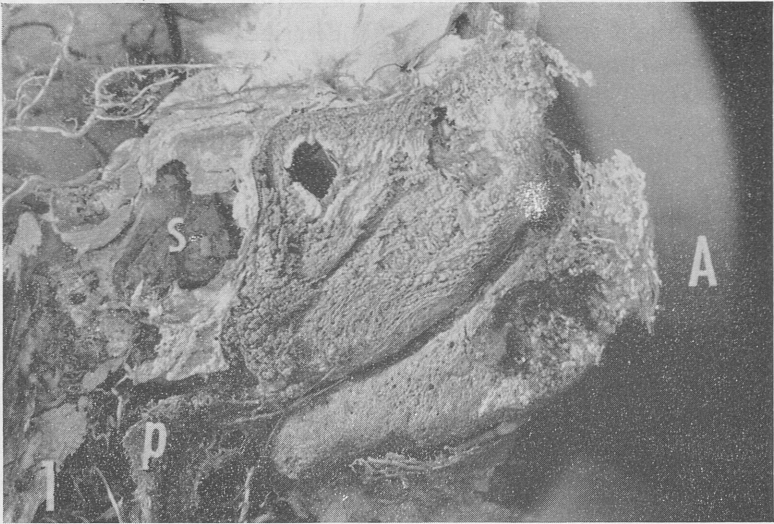


Fig. 1.—Corrosion specimen. Human, adult.. Left lateral nasal wall. A, anterior; P, rich plexus of veins in the eustachian tube area; S, sphenoid sinus with lining of veins. Note, that while only veins are shown, the lateral wall anatomy appears normal, except for the artifacts produced in sawing the specimen.

relations of the blood vessels to one another, over large areas, are more readily seen than in transparent preparations. The transparent preparations which I have made earlier are better understood by a study of the corruptions.

The following description of the veins of the human nose is based upon a series of corrosion preparations made with the new methacrylate mass mentioned above. These corruptions give an over-all view of the veins of the nasal mucosa, the paranasal sinuses, the nasal vestibule, and the nasal skin. They show the location of the principal draining veins and suggest several clinically significant relationships.

The specimens here reported are adult and new-born human material. The method chosen for injecting the veins may seem unorthodox. When it is recalled that with minor exceptions there are no valves in the veins in the head and neck, the logic of the method





Fig. 2.—Corrosion specimen. Human, adult. Same specimen as Figure 1, but seen from without. A, anterior; L, probe in the cylindrical venous plexus surrounding the nasolacrimal apparatus; O, ostium of the maxillary sinus from within the sinus. The continuity of the nasal veins and the sinus veins is obvious.

will be apparent. In one specimen, a deep brachial vein was isolated and the injecting cannula was directed toward the trunk. By this method, the fluid methacrylate solution entered the trunk veins to flow into the deeper neck veins. From these neck veins, the solution passed upward to the head and eventually to the veins of the nasal mucosa. In other specimens, the external jugular veins was the site of injection, the cannula being directed toward the head. In still others, one or both internal jugular veins were used. In every instance, the mass was fluid enough to find numerous escape outlets so that undue pressure was not developed. In using new-born material, it is necessary to ligate enough arteries so that a reflux cannot occur back to the head on the arterial side by way of the open foramen ovale and the patent ductus arteriosus. The vascular system was not profused before injection since in my experience it is not necessary. Although I am completely familiar with the pulsed pressure method of injection and use it on occasion, I did not feel that

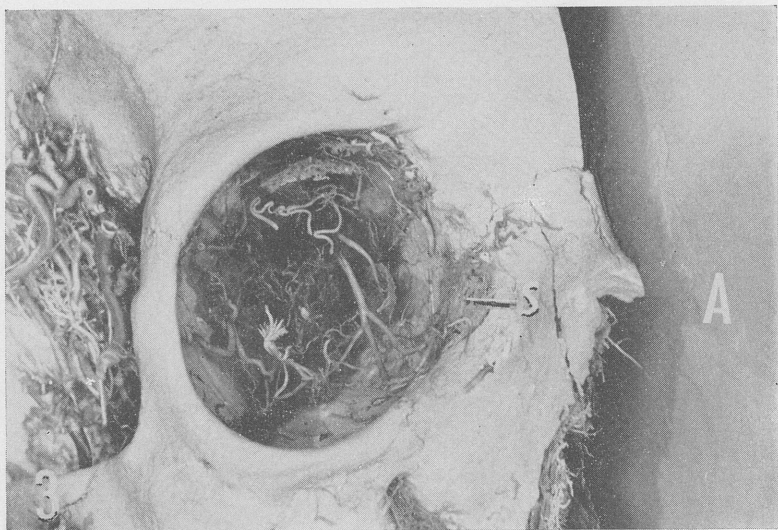


Fig. 3.—Corrosion specimen. Human, adult. Dorsum of the nose and right orbit. A, anterior; S, plexus of the saccus lacrymalis.

it was necessary in these specimens. All of our material was at least one week post-mortem, and all had been preserved by refrigeration.

The low viscosity and the easy flowing characteristics of this mass made the direct injection of the turbinates, as practiced by Zuckerkandl, unnecessary.

A corrosion specimen of the veins of the lateral nasal wall is startling in that it appears that the soft tissues are fully represented by the network of the cast (Fig. 1). It is as if the surface covering had been removed and everything else remained. Looking at such a specimen, it seems difficult to find place for the arteries and glands known to be present in the turbinates. This is due in part to the fact that with continuous pressure, most of the venous channels have been opened, and the corrosion preparation actually gives us the appearance of the veins when engorged, as in an inflamed state. By actual measurement, the mat of veins is from 1 mm to 4 or 5 mm in thickness, depending upon the area. The thickness is greatest in the rhinopharynx and on the turbinates. The veins themselves range



Fig. 4.—Corrosion specimen. Human, adult. The nasal septum seen from the left side. A, anterior; V, the coarser plexus over the vomer. Other points noted in the text are readily made out.

from a diameter of 0.1 mm to 1.0 mm or slightly larger. The veins are rarely more than a millimeter from each other, usually much closer. There are many interconnections. In the olfactory area the principal venous stems have a vertical course reflecting the course of the olfactory nerve filaments (Fig. 1). Over the rest of the lateral wall, they have the same general sweep as the nasal conchae themselves; that is, they slope obliquely downward and backward. At the ostia of the sphenoidal and maxillary sinuses, the network continues through into the cavities of the sinuses themselves. Here the veins are of the smaller ranges of diameter averaging less than 0.5 mm. It is to be remembered that these dimensions are for the internal diameters since these are casts. The mat of veins is about 0.5 mm in thickness. Although the normal sinus mucosa is pale and almost transparent, these specimens show that there is a rich venous plexus. Obviously it is capable of extemporaneous distention. As stated above, this injection represents something of the order of a maximum filling. Whether the sudden clinical thickening of the

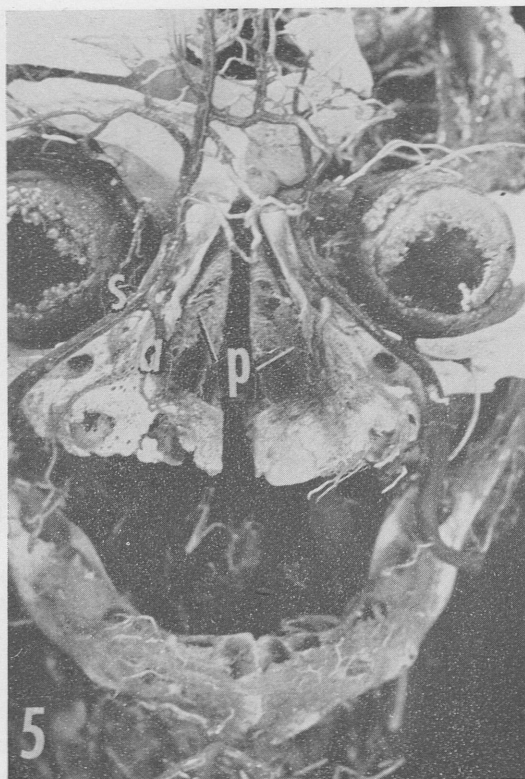


Fig. 5.—Corrosion specimen, human, neo-natus. P, indicates the venous plexus within the nasal cavity, and on the left side of the specimen the plexus of the skin of the nasal tip has been preserved. A, covers an alar vein and S, covers a saccus vein.

sinus membranes is due to engorgement with blood or to edema, a rich vascular network is necessary for either change to occur.

In specimens where the lateral bony nasal wall has disintegrated during maceration, the course of the naso-lacrimal duct can be followed upward by following the peri-lacrimal network of veins (Figs. 2 and 3). This network forms a definite cylindrical mesh which follows upward and encompasses the saccus lacrimonasus. In these specimens, it looks as if the saccus were in place. Actually, all we see is the cast of the interior of the veins of the peri-saccus plexus. From



this peri-saccus plexus, one or more veins run directly to the anterior facial vein.

This extensive communication of the peri-saccus veins with the veins of the nasal mucosa seems to offer an explanation for the swollen saccus area seen in young children during an acute coryza. The clinical picture looks like an acute dacryocystitis except that an acute inflammation here, as a primary entity, is practically unknown. Further, with adequate shrinkage of the nasal mucosa, the swelling of the saccus area subsides in a few hours. It would be difficult to say whether the swelling was due to the venous engorgement of the peri-saccus veins or whether the engorgement of these veins produced an obstruction to the duct itself. The latter explanation may be the proper one since such swelling is not encountered in adults, where the size of the duct is much larger. This plexus of the lacrimal

apparatus has been known for a long time, but it is rarely described and seldom illustrated.

The valveless continuity of the sinus venous network with that of the nasal cavity could account for the presence of fluid in the sinuses during an acute coryza without an actual infectious invasion of the sinus cavity. The early venous engorgement responsible for the increased nasal secretion unquestionably produces a sinus secretion.

As the venous mat of the nasal cavity nears the vestibule, the surface vessels seem as rich, but there are fewer layers. Here the network of the mucous membrane continues with the subcutaneous vestibular network of veins, and these in turn with the subcutaneous nasal veins over the alar cartilages. From this subcutaneous plexus, veins which could be called inferior alar veins, connect with the anterior facial vein.

The continuity of the submucosal and the subcutaneous network of veins over the alar cartilages furnishes the anatomic basis for the red and swollen nasal tip characteristic of the early stages of an acute infection (Fig. 3). The skin is in the same injected state as the mucous membranes in the nose. Parenthetically, the acute tenderness of this skin is due to its close attachment to the perichondrium. This is similar to the outer surface of the ear. Over the upper cartilage and the bone, the skin has a loose subcutaneous tissue, and the venous plexus is not so rich.

It is scarcely necessary to point out the rich venous plexus of the rhinopharynx which folds around the orifice of the eustachian tube (Fig. 1). The swelling of this orifice is so well-known that emphasis is unnecessary.

The nasal septum bears a network of veins similar to the lateral wall. Here the network is only 0.5 mm to 0.1 mm in thickness. The veins run parallel to the course of the olfactory filaments in the upper part of the nose and run in an irregular, horizontal course in the lower portion. They seem to parallel the course of the ethmo-vomer suture. In our specimens, the venous network covering the vomer is composed of larger and more widely separated veins than the network covering the perpendicular plate of the ethmoid. This is true in our small series and may be a universal finding. I feel that this should be studied further before expressing a final opinion.

It will be seen (Fig. 4) that in addition to the usually named veins of drainage, there is a whole series of veins from the nose which join the anterior facial vein beginning at the level of the sacculus lacrimalis and proceeding downward. Some of these veins now grouped as external nasal veins might properly be given special names because of their clinical significance, e.g., the vein of the lacrimal sac and the alar veins.

It might be asked whether the venous pattern shown to exist in the nose is characteristic of the veins of the other mucous membranes when demonstrated by this method. To take the immediately adjacent tissues of the mouth as a good example, it can be categorically stated that the venous network is neither as rich nor as thick as it is in the nose.

#### SUMMARY

The mucous membrane of the nasal cavity is pervaded throughout by an interlacing venous network consisting of vessels of from 0.1 mm to 0.5 mm and even larger in internal diameter. These veins are a valveless network. They communicate everywhere with each other and with the veins of the underlying bone. The thickness of the venous network parallels the thickness of the mucous membrane itself. It must be recognized that even within the sinuses with their thin mucosa, there is a rich venous network in a more or less collapsed state.

Photographs by Mr. Alex Vaskelis.

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## CONGENITAL ANOMALIES OF THE LARYNX

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Congenital anomalies of the larynx are of interest to the embryologist and clinician. This subject is reviewed and the various types of anomalies seen in the past ten years analyzed according to the symptoms they present, the laryngoscopic picture and the therapy which was instituted.

The congenital anomalies of the larynx will be discussed under the following headings: 1) congenital laryngeal stridor; 2) congenital webs; 3) congenital atresia; 4) congenital subglottic stenosis; and 5) congenital cysts and laryngoceles.

An understanding of the embryology of the larynx is necessary for an appreciation of the development of its anomalies. The larynx undergoes differentiation between the fourth and tenth weeks, and noxious agents affecting the developing embryo at this time may cause malformations of this structure, just as they may adversely affect other organs developing during the same period. The upper portion of the larynx arises from the pharyngeal floor in the region of the primitive glottis. That part from the vocal folds caudally forms around the stem of the trachea.<sup>1</sup> The epiglottis develops as a rounded prominence that elevates midventrally from the bases of the third and fourth arches. This soon alters its shape and consolidates into a transverse flap. It becomes concave on its laryngeal surface and in the middle of fetal life differentiates cartilage internally. The slit that opens from the floor of the pharynx into the trachea is

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the primitive glottis. It is bounded on each side by a rounded eminence, the arytenoid swelling of fourth and fifth arch origin. These two swellings begin to grow in a tongueward direction and on meeting the primordium of the epiglottis they arch upward and forward against its caudal surface. In the seventh week this results in the original, sagittal slit adding a transverse groove to its upper end, so that the laryngeal orifice becomes T-shaped. However, the entrance to the larynx ends blindly for some time because fusion of the epithelium in the upper larynx has obliterated the lumen. When the epithelial union is dissolved (ten weeks) the entrance becomes more oval in contour and a pair of lateral recesses is evident in the restored cavity. These recesses are the laryngeal ventricles. Each is bounded cranially and caudally by projecting lateral shelves, the caudal pair are the vocal folds which later differentiate elastic tissue.

The epithelial larynx is supported by dense mesenchyme derived from the fourth and fifth branchial arches. Early in the seventh week this mass shows localized condensations that foretell the laryngeal cartilages. The laryngeal muscles also originate from the same branchial arches and consequently continue to be innervated by the vagus nerves which supply those arches.

#### CONGENITAL LARYNGEAL STRIDOR

So-called congenital laryngeal stridor is a transient form of stridor in infants which is associated with a softness or abnormal flaccidity of the laryngeal tissues. For this reason, it is occasionally referred to as "laryngomalacia" or "congenital flaccid larynx." However, as Kelemen<sup>2</sup> has indicated, stridor can be produced almost anywhere in the larynx by bulky parts or protruding folds; thus, stridor is a symptom which is generally associated with respiratory obstruction rather than with a specific disease entity, and consequently the term is somewhat ambiguous. Therefore, in every infant with stridorous, noisy respirations, a differentiation must be made as to whether the symptoms are produced by the soft, flaccid structures of the larynx, the clinical entity of congenital laryngeal stridor, or by some organic obstruction due to other congenital anomalies, or a lesion acquired in infancy.

During the past seven years, 305 infants with congenital laryngeal stridor have been seen. Of these 199 were males and 106 females. Only one of these required tracheotomy. Typically, the stridor is present from birth and is usually audible only on inspiration. It may

be constant or intermittent and may vary with the position of the infant, being louder when the infant is on its back and quieter or absent when the infant is on its abdomen. The fluttering nature of the stridor may lead one to suspect that the infant's hypopharynx is filled with mucus, and that aspiration will relieve the condition. Synchronous with the stridor may be fluttering intercostal, supra-sternal or epigastric retractions.

A number of infants with congenital laryngeal stridor were noted to have funnel breast (*pectus excavatum*). The association with micrognathia, as suggested by Schwartz<sup>3</sup> was by no means apparent in this group of patients. Many of the stridorous infants had been irradiated for an ostensibly enlarged thymus gland. Littleton et al<sup>4</sup> discuss the fallacy of this too common diagnosis. Of course, if the gradual relief of congenital laryngeal stridor coincidentally follows thymic irradiation, it is then incorrectly concluded that the thymus was to blame.

Laryngoscopy is necessary to establish the diagnosis definitely and to rule out other causes of stridor. In the majority of cases, the epiglottis was the structure from which the fluttering sound originated, although the arytenoids were frequently involved with the epiglottis. Typically, the epiglottis is curled and may be tube-shaped in extreme instances.<sup>2</sup> The reason for this curling is not entirely understood, but it may be explained by a review of the development of the epiglottis which originates from the bases of the third and fourth arches. It is possible that the anterior half, or third arch contribution, has a propensity for more rapid growth than has the posterior half, or fourth arch contribution. This difference in the rates of growth of the two halves of the epiglottis would explain the curling of the structure in the infant, and the extreme curling in infants with congenital stridor. As the curled epiglottis is drawn into the laryngeal aperture with each inspiration, the typical stridor is produced (Fig. 1 A and B). Arytenoids which are long and flaccid flutter with each inspiration (Fig. 1 D), and they, too, are drawn into the laryngeal lumen along with a flaccid, curled epiglottis.

The one case of congenital laryngeal stridor which required tracheotomy was an extreme example of this condition. The infant had stridor from birth, and laryngoscopic examination when he was six weeks old showed the typical laryngeal flaccidity which characterizes congenital laryngeal stridor. The epiglottis was soft and

curled and the loose aryepiglottic folds and flabby arytenoid prominences were drawn into the laryngeal aperture on inspiration, alternately one above the other in a fluttering type of motion. When the tip of the laryngoscope was passed into the vallecula and the laryngeal structures brought under tension through lifting the base of the tongue, the stridor was appreciably reduced. Advancing the laryngoscope under the epiglottis and to its tuberculum resulted in a complete disappearance of the stridor as the tension was increased and the aryepiglottic folds were retracted laterally by the lip of the laryngoscope. Bronchoscopic examination of the trachea showed no evidence of subglottic or tracheal obstruction but all symptoms returned as the laryngoscope was withdrawn. No therapy seemed indicated or necessary in view of the patent airway and the absence of other organic obstruction of the airway. At the age of five months, the stridor and respiratory difficulty had increased; and in addition, because of the respiratory distress, the infant could not feed properly and had hardly gained more than a pound over his birth weight. There had been three attacks of cyanosis accompanied by convulsions. On the evening of readmission to the hospital for study, his laryngeal difficulty became so pronounced that a tracheotomy was indicated and was performed with a bronchoscope inserted to provide the infant with an adequate airway. Repeat studies of the larynx, esophagus and tracheobronchial tree showed nothing more than the flaccid larynx to account for the stridor. Since his tracheotomy, the child is eating and developing normally. There have been no further episodes of cyanosis or convulsions.

The recognition that the typical laryngoscopic picture of congenital laryngeal stridor is present does not rule out other causes of noisy respiration. This is illustrated by the following case:

L. L., male infant three months of age, was first examined on June 7, 1946 because of crowing respirations since birth. Laryngoscopy revealed a curled epiglottis and flaccid arytenoids typical of congenital laryngeal stridor and this diagnosis was considered confirmed by the laryngoscopic picture. However, his stridor persisted and he was re-examined on October 23, 1950 at the age of four years at which time a definite expiratory wheeze was present. Barium studies of the esophagus revealed an oblique, faint indentation crossing the esophagus at the level of the aortic arch in the antero-posterior view, and in the lateral view there was a marked posterior indentation. The trachea was seen to be considerably narrowed and pushed anteriorly at the level of the indentation. Laryngoscopy and bronchoscopy at this time revealed a normal larynx, but the posterior wall of the trachea was compressed above the bifurcation. Undoubtedly the fluoroscopic studies of the esophagus at the time of the first examination, which are now done routinely in infants with stridor, would have revealed the aortic arch deformity; but the

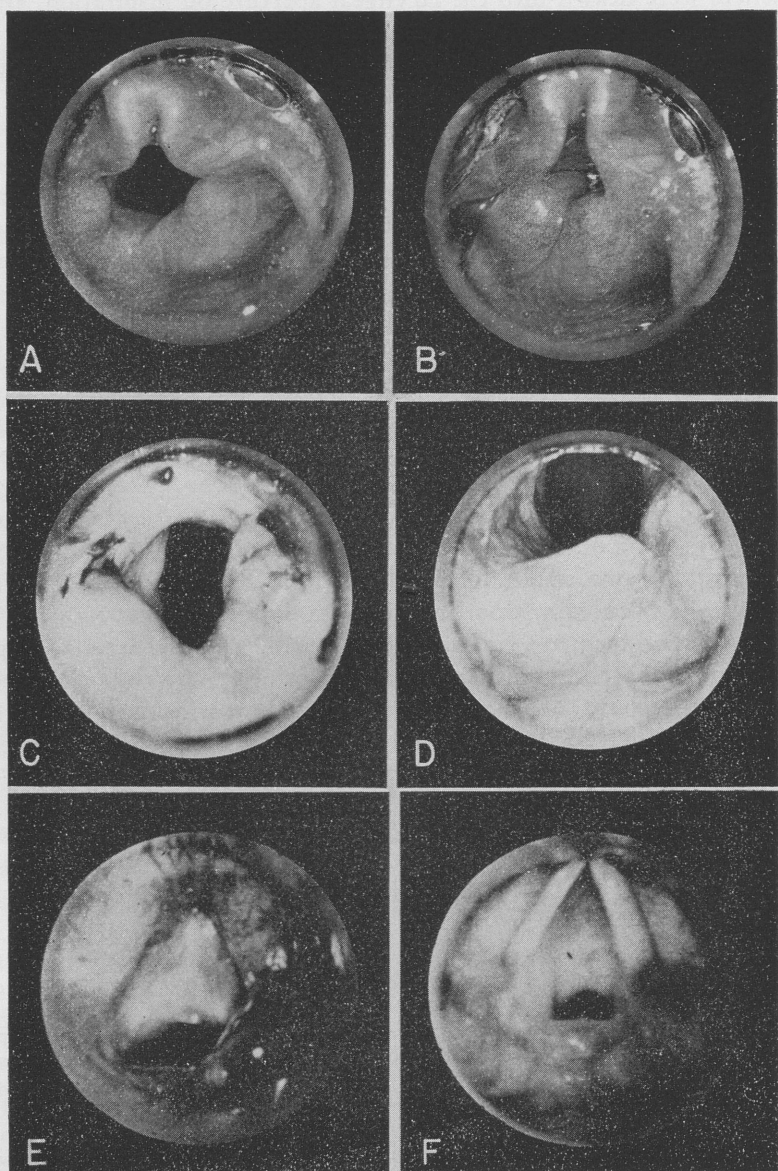




Fig. 1.—Direct laryngoscopic photographs of the larynx showing congenital laryngeal anomalies.

A and B.—Expiration and inspiration, respectively of an infant three months of age with congenital stridor.

C.—Congenital supraglottic web between the aryepiglottic folds at the level of the false cords.

D.—Flaccid, elongated soft parts of the left arytenoid in an adult being drawn into the larynx during inspiration. The right arytenoid followed over the left as inspiration progressed.

E.—Congenital fusion of the anterior half of the cords simulating a laryngeal web.

F.—Symmetrical subglottic stenosis at the level of the cricoid cartilage.

(Figures 1 E and F and Figures 4 and 6 are enlargements of frames of 16 mm motion picture films.)

laryngoscopic evidence of the typical picture of congenital laryngeal stridor made it appear that further studies were unnecessary in view of the positive findings. The diagnosis of a vascular ring was substantiated at surgery by Dr. Willis J. Potts. A similar case was reported by Wissler.<sup>5</sup>

Finlay<sup>6</sup> reported the clinical course and post mortem findings of three children in one family, all showing congenital stridor. Two of the children died shortly after laryngoscopy, which was performed under general anesthesia. The last case, which was not laryngoscoped, suffered episodes of choking during feedings, and was hoarse. Slight inspiratory retractions were present. Barium swallows showed notching of the posterior wall of the esophagus. Recurrent attacks of pneumonia caused the child's death and post mortem examination revealed an anomalous right subclavian artery and a small larynx. A fourth sibling was normal in all respects.

A complete study of infants with stridor should include a history of the nature of the birth and the time of onset of the stridor, a careful physical examination, especially noticing the character of the cry and its timing and observing whether the fluttering inspiratory sounds are synchronous with fluttering thoracic movements. X-ray studies should include frontal and lateral x-rays of the neck and chest and fluoroscopic studies of the esophagus. Finally, the endoscopic examination is made, confirming or denying the presence of the typical picture of congenital stridor as described above. All types of lesions which may obstruct the airway must be considered. The absence of x-ray evidence of an organic obstructing lesion does not establish the diagnosis of congenital laryngeal stridor by elimination. Only the direct laryngoscopic view can confirm this diagnosis, and rule out such other lesions as laryngeal paralysis or webs which would give no characteristic x-ray findings in infants.

As the infant with congenital laryngeal stridor grows, the larynx becomes larger and more firm, and the abnormal folds gradually uncurl. The stridor frequently disappears between the twelfth and eighteenth months. It is extremely rare that any active therapy is indicated and removal of the bulky parts seems not only unnecessary but dangerous.

#### CONGENITAL WEBS

Nineteen patients with congenital laryngeal webs have been observed (Table 1). There were nine males and ten females. In thirteen cases the web consisted of a membrane across the anterior por-

TABLE 1.  
SUMMARY OF FINDINGS IN PATIENTS WITH CONGENITAL LARYNGEAL WEBS

NO.	PT.	AGE	SEX	SYMPTOMS	POSITION OF WEB	CHARACTER OF WEB	TREATMENT
1	T. T.	5 yr.	M	Hoarseness	Glottis	Membrane	Dilatation multiple
2	L. J.	1½ yr.	M	Hoarse cry	Glottis	Membrane	Dilatation multiple
3	P. R.	1 mo.	M	Dyspnea; absent cry	Glottis	Transparent membrane	Dilatation single
4	L. B.	6 wk.	F	Severe dyspnea; weak, absent cry	Glottis	Fusion of cords	Tracheotomy dilatations
5	E. G.	11 da.	F	Hoarse cry; dyspnea	Glottis	Membrane	2 dilatations
6	M. S.	3 mo.	F	Weak cry; mild dyspnea	Glottis	Fusion of cords	Repeated dilatation
7	W. L.	2 wk.	M	Dyspnea, cyanosis, absent cry	Glottis	Dense membrane, small posterior opening	Tracheotomy incision and dilatation of web
8	B. F.	1 da.	F	Dyspnea, cyanosis, absent cry	Supraglottic	Supraglottic membrane	Incision and dilatation, tracheotomy—died
9	M. F.	31 yr.	F	Hoarseness	Glottis	Small anterior commissure web	Dilatation
10	R. P.	25 da.	F	Hoarseness	Supraglottic	Fusion of false cords	Dilatation

11	B. S.	6 yr.	M	Dyspnea and hoarseness; assoc. with patent ductus	Glottis	Membrane	None
12	J. F.	12 yr.	F	Hoarseness	Glottis	Membrane	Dilatation
13	A. H.	7 yr.	F	Hoarseness	Glottis	Membrane	Incision, dilata.
14	N. B.	17 yr.	M	Hoarseness	Glottis	Membrane	Incision, dilata.
15	R. T.	15 mo.	M	Hoarseness, dyspnea	Supraglottic	Supraglottic, fibrous membrane level of false cords, almost cartilaginous in character; membranous web across anterior half of true cord.	Dilatation
16	F. B.	2 yr.	F	Hoarseness, dyspnea	Glottis	Membrane	Dilatation; this child later developed papilloma
17	G. M.	8 mo.	M	Weak cry, hoarseness and dyspnea	Glottis	Membrane	None (mental deficiency)
18	W. L.	10 da.	M	Cry without sound, dyspnea and cyanosis	Glottis	Membrane	Forceps and bougie dilatation
19	L. B.	3 yr.	M	Squeaky, high pitched cry, hoarseness	Glottis	Membrane across anterior two-thirds of larynx	Dilatation with laryngeal bougies



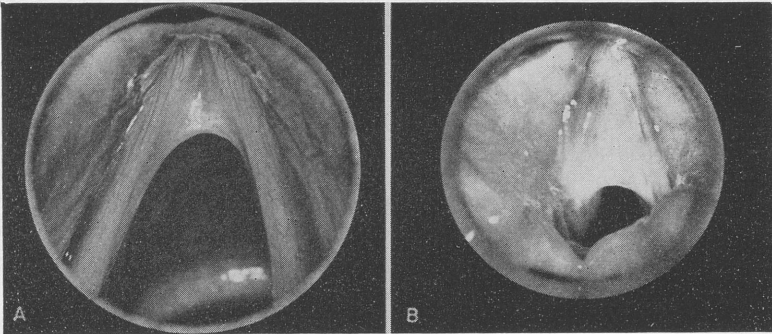


Fig. 2.—Typical congenital membranous webs of the larynx. A. Web across the anterior commissure of a 31- year old female who had been hoarse since birth. B. Web involving the anterior two-thirds of the larynx of a seven year old girl referred from her school because of speech difficulty. A history of hoarseness and dyspnea since birth was obtained.

tion of the glottis at the level of the superior surfaces of the cords, through which the outlines of the phonating edges of the cords could be distinguished. The degree to which the glottis was covered varied from small anterior commissure webbing (Fig. 2 A) to almost complete closure of the glottis (Fig. 2 B). In the majority of cases the anterior two-thirds of the glottis was involved, the tough membrane originating along the entire cord with the exception of the vocal process of the arytenoids; it arched anteriorly to give a posterior chink for respiration.

In one infant an extremely thin, transparent web extended across the anterior one-half of the larynx at the level of the superior surface of the true cords. This infant, a new-born, made no sound on crying and a direct laryngoscopy was performed for diagnostic purposes. The membrane was visualized, split with laryngeal alligator forceps and its edges immediately retracted into the laryngeal mucosa. The infant cried audibly at once and on subsequent examination the larynx appeared entirely normal.

In two infants, the congenital glottic web assumed a configuration which differed from that described above; it appeared to be a fusion of the anterior half of the cords rather than a membranous sheet (Fig. 1 E). In these two infants the anterior half of the

phonating edges of the cords appeared to be in contact with each other, the cords separating posteriorly as an inverted "V." The passage of dilators through the larynx demonstrated a fibrous character to this anterior commissure fusion which extended subglottically and was not similar to the webs described above.

The congenital webs in three patients were supraglottic, an apparent fusion of the anterior half of the false cords; the true cords below were normal in configuration and motility. The appearance of this condition in one patient is shown in Fig. 1 C. The description of the second supraglottic web is incorporated in the following case report:

B. F. was delivered normally at term on September 12, 1951. She was admitted to the hospital at the age of six hours because of marked inspiratory stridor, thoracic retractions and a high-pitched, weak cry. X-rays of the chest and neck showed no abnormality. Direct examination of the larynx was made with an infant laryngoscope and a most unusual picture was immediately observed. The base of the tongue was normal and the epiglottis was extremely small and was drawn posteriorly. The mucosa covering the posterior surface of the cricoid cartilage was visible as the base of the tongue was raised, this mucosa extending anteriorly as a more or less rounded, membranous surface. A tiny, pin-point opening through which respirations were taking place was seen in the midline in the center of this rounded mass. A 3 mm x 20 cm infant bronchoscope was inserted through this opening, dilating it and improving the airway; the true cords and glottic aperture could be visualized below it and appeared to be essentially normal. As the bronchoscope passed through the opening and beyond the cords into the trachea, the respirations became quiet and without effort. The bronchoscope was withdrawn and the larynx reinvestigated by lifting the tip of the epiglottis with the laryngoscope. A membrane appeared to cover the larynx, extending across the laryngeal aperture at the level of the aryepiglottic folds and the false cords. The anterior portion of this membrane was exceedingly thin and transparent, but the posterior aspect was fibrous and thickened and drew the superior surfaces of the arytenoids and the posterior portion of the aryepiglottic folds together over the glottis. A laryngeal knife was inserted into the posterior opening through which the bronchoscope had passed and the web was incised, releasing the tension which held the arytenoids and aryepiglottic folds together. The lumen of the larynx was then visible and the true cords were found to be essentially normal in their appearance and their motility. Following the procedure the patient's respirations were quiet and unobstructed. Consequently, the tracheotomy which had been contemplated was postponed, at least temporarily, to determine whether there had been sufficient improvement in the airway to make the tracheotomy unnecessary. Five days following the original laryngoscopy, the procedure was repeated and the membrane was redilated with laryngeal forceps. After another five day period, the larynx was dilated with a No. 12 laryngeal bougie, but fifteen days later laryngoscopy revealed considerable stenosis of the anterior half of the larynx and it became obvious that dilatation was not entirely satisfactory in keeping the airway open. Consequently, a tracheotomy was performed over a 3 mm bronchoscope and dilations were then carried out weekly.

On December 2, 1951, the baby took fluids poorly, became irritable, and began expelling excessive blood-tinged mucus from the tracheal cannula. Coarse

rhonchi were present over both lungs and x-rays showed diffuse pneumonitis bilaterally. Acute respiratory distress increased in spite of aspiration and antibiotic management and the next day the child developed convulsions and became cyanotic. She expired on the following day.

Post mortem examination revealed pneumonia involving the right middle, right lower and left upper and left lower lobes. Little evidence of the supraglottic web remained. The epiglottis was abnormally mobile close to its base and was bent posteriorly to lie over the glottis. In its anteroposterior diameter, the glottic chink measured six millimeters. The mucosa over the cords, arytenoids, and epiglottis was edematous, and the mucosa over the cords suggested slight denudation. Sections of the epiglottis and cords revealed the mucosa to be thinned in focal areas. A rich infiltrate of small round cells was present in the submucosa and the blood vessels were dilated and engorged with red cells.

The trachea and bronchi contained a bloody, frothy, viscous material. Except for the tracheostomy, the rings appeared normal and the trachea was not remarkable. Microscopic sections revealed no changes, except for edema of the mucosa and a few inflammatory cells in the submucosa.

Thus, of the nineteen congenital laryngeal webs, twelve consisted of moderately thick membranes across the anterior one-fourth to two-thirds of the membranous portions of the cords; one was a thin, transparent sheet across the superior surfaces of the cords; two were a fusion of the anterior half of the cords without an apparent membrane of any kind between the cords; and three were supraglottic webs which consisted of a membrane connecting the false cords and aryepiglottic folds but not involving the true cords below. The nineteenth patient was an adult who had been hoarse since birth who had a small membrane (Fig. 2 A) across the anterior one-fourth of the larynx. Serology and chest x-rays were negative and she had had no laryngeal inquiry or surgery of any kind. The history and appearance suggests a congenital laryngeal web, but, obviously, absolute proof other than the clinical picture is lacking.

Symptoms of laryngeal webs are present at birth or shortly thereafter and usually consist of a hoarse or weak cry, or a complete absence of sound with crying efforts. Respiratory distress is the second symptom, the degree depending upon the percentage of the glottis which is covered by the web. Three of the thirteen patients with membranous glottic webs were children of school age, referred because of speech difficulties.

The correct diagnosis can only be made by a direct laryngoscopic view of the larynx, but it is imperative that preparations for a possible tracheotomy be completed before the examination is undertaken. This measure is necessary whenever an endoscopic pro-

cedure is being performed in the face of respiratory obstruction; not that the endoscopy will precipitate the tracheotomy, but because of the nature of the pathology which may be found.

Therapy in these patients has been conservative. Two of the infants with membranous glottic webs and one with a supraglottic web have required tracheotomy. In the remaining patients direct laryngoscopic dilations have been employed. Dilatation of the larynx, occasionally associated with use of the Knight<sup>7</sup> combination scalpel and dilator have proven effective and the more extensive extra-laryngeal surgical procedures have not been necessary. One death from an extensive bronchopneumonia occurred in a tracheotomized infant who had a supraglottic web.

Congenital stenosis, webs, and atresia of the larynx are best explained embryologically by the fusion theory of laryngeal development. Hudson<sup>8</sup> reports that nine cases of congenital webs of the larynx were seen at Babies' Hospital between 1932 and 1949. The hospital admits approximately 4,000 patients yearly. He presents one case of a web extending between the ventricular bands leaving a very small posterior airway. In his discussion of this subject, Clerf<sup>9</sup> presents three cases of laryngeal webs. Two of the webs reported were between the ventricular folds. Clerf explains congenital laryngeal stenosis by the embryological fusion theory. As mentioned in the review of the embryology of the larynx, the developing vocal cords are adherent to each other during the seventh week. After about two weeks, this epithelial fusion dissolves and the opening into the larynx is reestablished. Failure of adequate dissolution may result in development of webs, bands, or complete atresia. McHugh and Loch<sup>10</sup> found reports of 133 congenital webs in the literature and presented an additional case of their own. The majority of the webs (75 per cent) were at the level of the true cords. Supraglottic webs accounted for 1.5 per cent of their series, and 3.7 per cent extended posteriorly. An extensive bibliography of 169 items accompanies this excellent study.

#### CONGENITAL ATRESIA

Congenital atresia of the larynx is obviously incompatible with life unless the condition is recognized at once at birth and immediate steps are taken to establish an airway. One such case is reported by Jackson and Jackson.<sup>11</sup> Direct laryngoscopic examination of a "blue baby" that had ceased to make respiratory movements immediately



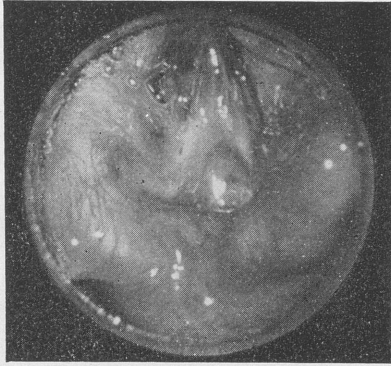


Fig. 3.—Congenital atresia of the larynx of an infant ten months of age. A tracheotomy had been performed at birth when it was realized that while the infant was making strong respiratory efforts, there was no air exchange.

after delivery revealed a complete membranous occlusion of the glottis. A 3 mm bronchoscope was pushed through the web into the trachea and oxygen and artificial respiration administered. The infant responded and after an hour was breathing on his own and the bronchoscope was withdrawn. At six years of age, examination showed thickened cords.

A similar, dramatic instance is herein reported:

D. C. was normally delivered after a full term gestation. The obstetrician noted strong respiratory efforts, with no evidence of air exchange. Thinking quickly, he rapidly performed a tracheotomy with the instruments at hand, establishing an adequate airway before damage from anoxia could take place. This infant was examined at Children's Memorial Hospital on September 13, 1950 when it was ten months old. On lifting the epiglottis, no aperture could be visualized. The epiglottis, hypopharynx, false cords, and arytenoids were seen to be normal. The true cords, however, were joined by a sheet of white fibrous tissue through which their outlines could be distinguished (Fig. 3). No actual lumen was visible, although a pinpoint dimple in the posterior commissure suggested an opening; however, there was nothing to suggest that air passed through on inspiration or cough. The atretic area felt extremely firm, possibly cartilaginous in character, and even with considerable effort it would not permit the passage of the tip of a No. 12 bougie. Efforts to establish a lumen by puncture from above and by retrograde procedures have not been successful. After further growth has taken place other attempts will be made to open through the obstructing membrane. McNaught's<sup>12</sup> method of placing a thin plate of tantalum in the midsagittal plane of the larynx through a slot in the thyroid cartilage, after dividing the membrane, may be effective here. It should be noted that this infant has other congenital anomalies such as hyperelastica cutis (Ehlers-Danlos Syndrome).

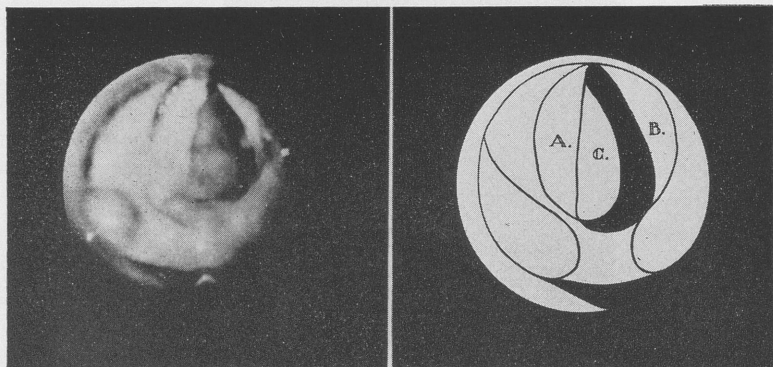


Fig. 4.—Direct laryngoscopic view of subglottic stenosis. The photograph was taken from a 16 mm motion picture frame and the structures are identified in the line drawing on the right. A. Left cord. B. Right cord. C. The left subglottic swelling as viewed through the glottis from the right. A similar swelling under the right cord caused the subglottic stenosis.

#### CONGENITAL SUBGLOTTIC STENOSIS

This clinical picture is a difficult one to evaluate (Figs. 1 F and 4). It consists of a thickening of subglottic structures and sometimes of the true cords themselves which causes respiratory obstruction of a severe degree in new born infants. The least laryngeal inflammation precipitates a tracheotomy since the limiting cricoid cartilage does not permit swelling of tissues in any direction other than inwardly, at the expense of the airway. The point of greatest obstruction is generally 2-3 mm below the level of the superior surface of the true cords. Biopsy of one such area showed no inflammatory reaction. In two cases, a severe obstructing deformity of the cricoid cartilage was found at necropsy, suggesting that moderate cartilaginous deformities may play an important role in other infants in this category.

Thirty-four cases of congenital subglottic stenosis of the larynx have been seen during the past seven year period. There were twenty males and fourteen females. The earliest symptom of this condition beginning at birth was stridor with or without episodes of cyanosis depending upon the severity of the stenosis. In mild cases, recur-

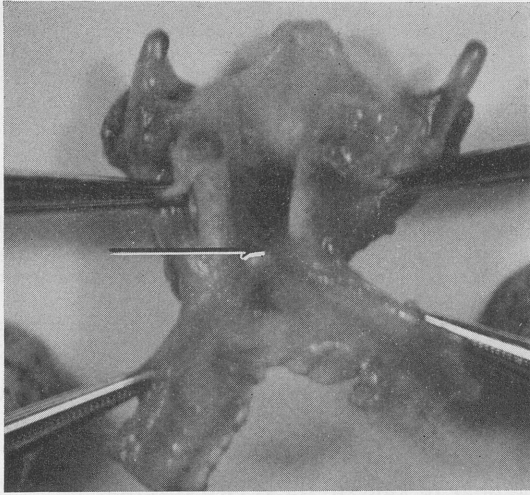


Fig. 5.—Cartilaginous deformity of the arch of the cricoid. The larynx is opened from posteriorly and the level of the stenosis is indicated by the arrow.

rent episodes of croup were the only suggestion of this condition, with repeated superimposed respiratory infections simulating laryngotracheobronchitis, although characterized by severe obstruction with minimal evidence of infection. In severe cases with progressive respiratory distress, tracheotomy becomes an urgent procedure. Fourteen of the thirty-four infants with congenital subglottic stenosis required a tracheotomy.

Mild stenosis usually disappears with growth of the larynx. In more severe cases, periodic dilatations of the larynx are performed to stretch the lumen to a more adequate caliber. This can be done without fear of resulting edema leading to complete obstruction of the larynx in patients who have been tracheotomized, but such therapy carries some risk if done without a tracheotomy. With an inflammatory component, adequate antibiotics, hydration and a humidified atmosphere are of utmost importance.

The clinical history of the infant whose larynx appears in Figure 4 is as follows:

P. G. was born on August 2, 1948. She had difficult, stridorous breathing from birth, which became more marked in the third week of life. On admission to the hospital on September 4, 1948, her rectal temperature was 99, pulse 160, and her respirations were stridorous at the rate of 40 per minute. Intercostal and suprasternal retractions were present. She was considered to have laryngotracheobronchitis and was treated actively for this for four days, but respirations did not improve. A tracheotomy became necessary on the fourth day and was performed over a bronchoscope. On exposure of the larynx, mild edema of the arytenoids and swelling of the subglottic region were noted. Except for a few critical episodes, when the tracheal cannula became plugged with mucus, the postoperative course was uneventful. Further endoscopic examination at a later date revealed a definite subglottic stenosis. The thought of a double aortic arch with tracheal compression was entertained, but was not supported by esophagograms. Periodic dilatations of the larynx were carried out during the ensuing year and she responded well to treatment and the tracheal cannula was removed one year after tracheotomy was performed. The patient has had no further respiratory difficulty.

Anomalies of the cricoid cartilage are responsible for some cases of subglottic stenosis. Two infants mentioned above had severe cartilaginous deformities which necessitated tracheotomy. The following is a case report of one of these two infants:

M. M. was first seen on April 9, 1950 at four weeks of age in extreme respiratory distress. He had had respiratory difficulty from birth. A lateral x-ray of the neck showed marked narrowing of the airway at the level of the cricoid cartilage. Exposure of the larynx revealed a bony-hard stenosis below the level of the true cords, apparently at the level of the cricoid cartilage, and efforts were made to pass a bronchoscope beyond this point to establish an airway. This was unsuccessful because of the firm resistance. As the infant became more cyanotic an attempt was made to pass a small metal aspirator through the tiny opening in the subglottic space, but this also met with failure. An emergency tracheotomy was performed and artificial respiration was administered, but the infant failed to respond and expired. Necropsy showed the glottic and supra-glottic structures of the larynx to be normal. The cricoid cartilage was deformed and this deformity obviously was the cause of the obstruction. A shelf-like plate of cartilage, filling the convex inner surface of the arch of the cricoid cartilage extended posteriorly as a solid, rigid sheet, leaving only a small posterior opening (Fig. 5). In addition, hydrocephalus with complete closure of the foramina was disclosed.

#### CONGENITAL CYSTS AND LARYNGOCELES

Five cases of congenital cysts of the larynx have been seen in infants (Table 2). Two, reported previously,<sup>13</sup> accounted for respiratory obstruction and a muffled or absent cry in infants, each two months of age. Symptoms had been present since birth. The cysts originated in the right laryngeal ventricle and bulged into the glottic lumen, completely covering the right true cord (Fig. 6). They were filled with thick mucus; in one case a temporary trache-



TABLE 2.  
SUMMARY OF FINDINGS IN PATIENTS WITH CONGENITAL CYSTS OF THE LARYNX.

NO.	NAME	AGE	SEX	SYMPTOMS	LOCATION	TREATMENT
1	E. B.	2 mos.	F	Hoarseness and dyspnea	Right laryngeal ventricle	Endoscopic removal
2	F. H.	2 mos.	F	Aphonia, dyspnea and cyanosis	Right laryngeal ventricle	Tracheotomy and endoscopic removal
3	H. W.	2 mos.	F	Stridor and gagging	Left aryepiglottic fold	Repeated aspiration
4	C. J.	1 day	F	Crowing respirations, severe dyspnea	Right laryngeal ventricle	Endoscopic forceps removal
5	P. M.	15 yrs.	M	Stridor and muffled voice since birth	Left aryepiglottic fold and vallecula	Aspiration

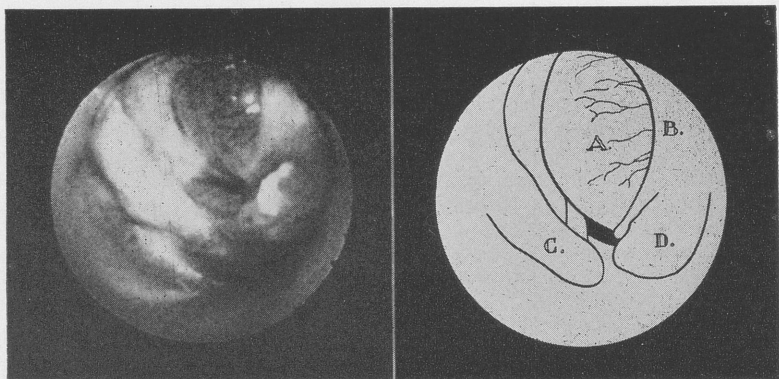


Fig. 6.—Congenital laryngeal cyst as seen through the direct laryngoscope. The line drawing on the right identifies the structures. A. cyst protruding from right laryngeal ventricle. B. Right false cord. C. Left arytenoid. D. Right arytenoid.

otomy was necessary after partial endoscopic removal, and in the second case endoscopic removal with cup forceps resulted in a normal cry and normal respirations without the necessity of a tracheotomy.

Three additional cases may be added to the previous report:

H. W., two months of age, was seen on January 6, 1947 because of stridor from birth and gagging during feedings. Moderate dyspnea and slight epigastric and suprasternal retractions were present and she had a muffled cry. Laryngoscopy revealed a large cyst in the left aryepiglottic fold, compressing the larynx and forcing the epiglottis to curl upon itself. The cyst was aspirated of two cc of thick, tenacious, mucoid secretion, resulting in collapse of the cyst and complete disappearance of symptoms. As it has refilled it has required periodic aspirations; each time the mother has noted the child becoming hoarse, she has returned for aspiration of the cyst as an out-patient. The procedure is accomplished rapidly without anesthesia and is accompanied by little trauma. Intervals between aspirations have gradually increased from one to two months to a period of eight months since she last required aspiration.

The fourth infant with a congenital laryngeal cyst, C. J., was admitted on December 5, 1952, at one day of age with crowing respirations since birth. The progressive respiratory distress had necessitated a tracheotomy at fourteen hours of age. Laryngoscopy revealed a grayish-yellow cyst similar in position to those in the first two infants. It appeared to arise from the right ventricle almost completely filling the laryngeal aperture. The cyst wall was removed with cup forceps and there has been no evidence of recurrence. Microscopic examination

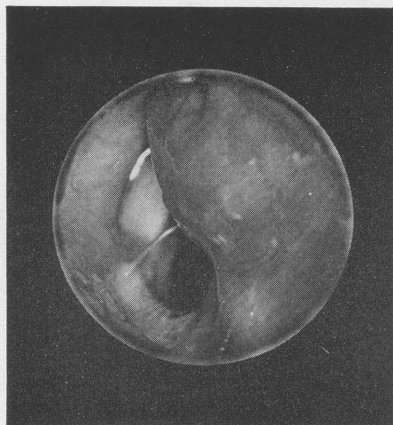


Fig. 7.—Direct laryngoscopic view of a large right-sided internal laryngocele. The sac involves the entire false cord and overhangs the glottis.

of the tissue which was removed showed the cyst to be lined with stratified columnar epithelium and to contain lymphocytes in its stroma. This histologic picture is consistent with congenital cysts of the larynx.

The fifth patient, P. M., a fifteen year old boy had had dyspnea, stridor and dysphagia since infancy. Symptoms had increased slowly and at the time of examination on April 12, 1952, he had considerable difficulty in breathing, talking and swallowing. Examination showed a large, round cyst, three to four cm in diameter on the left side of the pharynx, pushing the epiglottis to the right and apparently having its origin in the left aryepiglottic fold. Sixty cc of gelatinous fluid were aspirated with a heavy needle and the cyst collapsed to reveal a normal airway and normal vocal cords below. Recently symptoms have recurred and resection of the cyst seems indicated.

Most congenital cysts described in the literature are confined to the immediate neighborhood of the ventricular appendix.<sup>14-18</sup> It is probable that cysts are the result of simple disturbances in fetal development and not genuine embryonic malformations. Congenital cysts and laryngoceles have a similar development, but cysts do not communicate with the interior of the larynx. Laryngoceles admittedly become clinically perceptible only when swollen by air forced into them, or when filled with a collection of fluid; thus it is obvious that laryngoceles and cysts are often indistinguishable from each other. This is particularly true in adults, as Huckel<sup>19</sup> has

TABLE 3.  
SUMMARY OF FINDINGS IN PATIENTS WITH LARYNGOCELES.

NO.	NAME	AGE	SEX	SYMPTOMS	SITE	TREATMENT
1	F. P.	51	M	Aphonia, dyspnea, tracheotomy	Bilateral	Laryngofissure and bilateral resection
2	J. C.	43	M	Hoarseness 3 months	Left	Resection supraglottically
3	C. M.	63	M	Hoarseness 1½ years	Bilateral	Incidental finding on laryngectomy for carcinoma
4	H. S.	55	M	Hoarse 4 months	Right internal and external	Supraglottic excision
5	A. F.	55	F	Hoarseness 1½ years	Right internal	Endoscopic drainage with biopsy forceps
6	L. T.	30	M	Hoarseness	Right internal and external	None
7	S. M.	50	F	Hoarseness 2 years	Right internal	Endoscopic incision and drainage
8	B. K.	58	F	Hoarseness one year	Left	Excision supraglottically
9	M. T.	56	M	Hoarseness and dyspnea 2 months	Right	Endoscopic drainage, recurrence, supraglottic excision
10	F. S.	61	F	Hoarseness, occasional dyspnea 4 months	Right internal	Endoscopic drainage
11	D. P.	33	M	Hoarseness; 1 year swelling in neck after singing	Bilateral internal and external	None
12	E. M.	39	F	Hoarseness 1½ years	Right internal and external	Suprathyroid exposure and excision
13	R. B.	61	F	Hoarseness and dysphagia	Right internal	Endoscopic excision
14	A. C.	89	F	Hoarseness	Right internal	Endoscopic drainage
15	H. B.	63	M	Hoarseness 3 years	Bilateral internal and external	Endoscopic resection



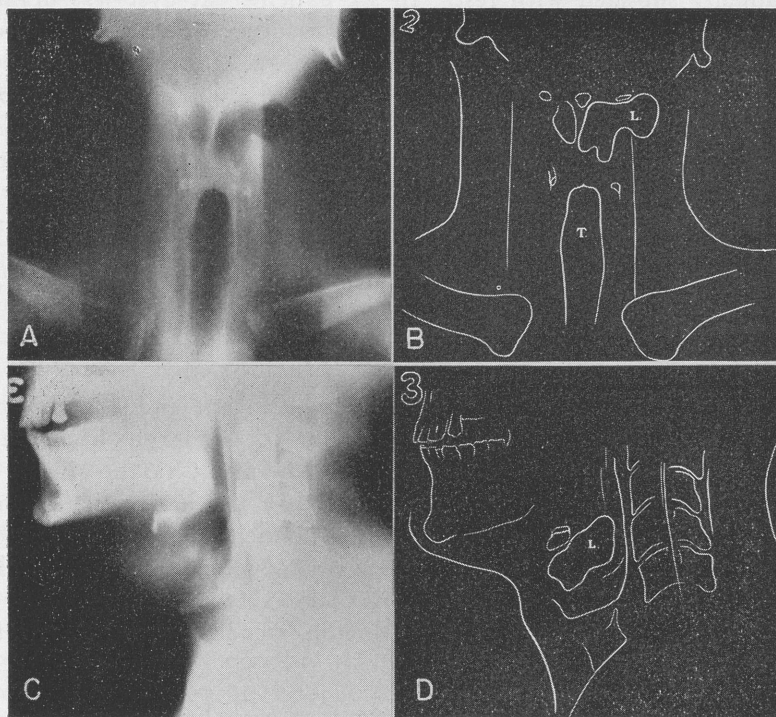


Fig. 8.—Antero-posterior and left lateral planograms of an internal and external unilateral (left) laryngocele. The line drawings clarify the significant structures. "L," the laryngocele, "T," the trachea.

already indicated. Congenital cysts are extremely rare in adults, probably because as hollow structures they fill and grow in intra-uterine life and are, therefore, observed directly after birth.

Fifteen cases of laryngoceles have been observed (Table 3). Eight were in adult males, seven in females. The sacs were unilateral in eleven cases (Fig. 7) and bilateral in the remaining four cases. In nine in whom the laryngocele was unilateral it was on the right, in two on the left (Fig. 8).

These lesions are considered in the category of congenital anomalies in view of the above discussion. Their presenting symptoms were hoarseness and, in occasional cases, some dyspnea. One patient,

a singer, complained of a swelling in the neck after singing which gradually subsided with rest. In six patients the cyst was drained by uncapping the superior surface of the false cord with biopsy forceps. In one additional patient this technique was used but the cyst recurred and consequently it was removed through an incision along the superior border of the thyroid cartilage. This approach was used in four additional cases and in a sixth patient, bilateral laryngoceles were removed through an external thyrotomy approach. In one case bilateral laryngoceles were observed as incidental findings in a laryngectomy specimen removed for carcinoma of the larynx. Two patients have remained untreated, having been seen for diagnosis only.

#### DISCUSSION

The primary differences between the infant and adult larynx are:<sup>20, 21</sup> 1) size; 2) the tissues of the infant larynx are softer; 3) the infant larynx is placed higher in the neck; 4) the axis of air entry is straighter in the infant larynx; 5) the laryngeal lumen is more funnel-shaped in the infant than in the adult. Negus<sup>22</sup> mentions that the infant cricoid cartilage is somewhat funnelled and that the antero-posterior diameter of the larynx is relatively greater in comparison to that of the trachea than in later life, at which time the cricoid is ring-shaped.

The symptomatology in the group of cases herein presented did not vary greatly. A weak or absent cry in an infant at birth suggests a laryngeal anomaly in spite of the fact that respirations may be unimpaired. However, in the infant who has dyspnea, stridor, or cyanosis, the character of the cry becomes of great diagnostic importance. The muffled, pharyngeal quality of cry of the infant with a thyroglossal duct cyst<sup>23</sup> or other pharyngeal obstruction is quite characteristic. An infant with a laryngeal (glottic) web generally has some dyspnea and indrawing in the suprasternal notch and epigastrium in addition to the weak cry, but this combination is by no means indicative of the diagnosis since it is often noted in infants with subglottic stenosis. Similarly, the loud, stuttering, crowing inspiration which characterizes congenital laryngeal stridor could by no means be considered diagnostic in itself since infants with bilateral recurrent laryngeal nerve paralysis and those with tracheomalacia, alone or associated with a vascular anomaly compressing the trachea had an identical sounding stridor.<sup>24</sup> For this reason, a diagnostic routine is established which, in addition to the history and physical

examination, consists of antero-posterior x-ray studies of the chest and neck, and particularly the lateral x-ray of the neck with the arms down and back and the head up and forward to obtain as much visualization of the trachea as possible. Fluoroscopic studies of the esophagus with barium or lipiodol as a contrast medium are often indicated as well to rule out possible compressing vascular anomalies or other anomalies of the tracheobronchial tree.<sup>25</sup>

Direct laryngoscopy is the only manner of inspecting the infant larynx and therefore is the most important single diagnostic procedure. It may be done with small laryngoscopes without administering an anesthetic to the infant. In acute laryngeal obstruction the instrumentation may precipitate complete respiratory obstruction; therefore, in the acute emergency direct laryngoscopy should be attempted only if the diagnosis cannot be established otherwise or if surgical relief of the obstruction through direct examination and aspiration, intubation, or tracheotomy is being considered. Instrumentarium should be ready for this eventuality when a direct examination of the obstructed larynx is contemplated. The difficulties of direct laryngoscopy in infants are due to the small size of the larynx and the curled, short, slippery, omega-shaped epiglottis. The larynx is easily displaced, and there is a decided tendency toward spasm of the larynx as the laryngoscope is introduced. This often makes visualization of the true cords and of the glottic airway difficult. The procedure must be done rapidly to avoid instrumental trauma. Treatment in this series has been conservative. Dilatation of laryngeal webs and subglottic stenosis and aspiration of laryngeal cysts rather than extensive external surgical procedures has, for the most part, given reasonably good results. Once the diagnosis of a congenital laryngeal anomaly in an infant was established, it was felt that the surgical procedures, other than endoscopic, could be postponed and approached more successfully when the infant had gained maturity. The exception to this was a tracheotomy if respiratory obstruction was severe or frequently recurring.

In a recent analysis of lesions necessitating tracheotomy in infants under one year of age,<sup>26</sup> twenty-seven of the total of sixty-two cases (44 per cent) required tracheotomy because of a congenital laryngeal anomaly, and nine (14 per cent) in addition were tracheotomized because of other congenital anomalies that necessitated extensive surgical repair. Only nineteen or 31 per cent of the sixty-two cases were tracheotomized for inflammatory disease; the remain-

ing seven (11 per cent) had neoplastic disease of the neck or larynx which obstructed the airway directly or by compression. This is in contradistinction to infants and children above one year of age in whom inflammatory disease was the etiology for which a tracheotomy was performed in approximately 90 per cent of cases. Of further interest was the fact that while the incidence of tracheotomy in infants for inflammatory disease such as acute laryngotracheobronchitis has steadily decreased over a period of fifteen years, the incidence of tracheotomy for other causes, such as congenital anomalies, laryngeal paralysis, polio, skull fracture, burns, tetanus, etc. has steadily increased.

Two groups of patients with laryngeal disease at birth are not included in this summary of congenital laryngeal anomalies. One is the group of laryngeal paralysis, either unilateral or bilateral, which were associated with cerebral agenesis, cervical meningomyeloceles, or extensive cardiovascular anomalies involving the left recurrent laryngeal nerve. The second group are the tumors of the larynx discovered at birth or shortly thereafter because of the respiratory obstruction they produced.<sup>27</sup>

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## DISCUSSION

DR. CHEVALIER L. JACKSON: Mr. Chairman, Members and Guests, I should like to ask Dr. Holinger, whether it is his impression that, in cases of congenital stridor benefit or improvement is caused by the introduction of the laryngoscope and consequent flattening of the epiglottis? I have had the impression that this was beneficial in some cases, and yet never accepted it as a proven fact. Recently, in talking with some of our colleagues in Philadelphia, I find they have a similar impression. I would like to know what Dr. Holinger thinks, from his large experience, about the therapeutic effect of the flattening of the epiglottis with the laryngoscope.

DR. GABRIEL TUCKER: I would like to compliment Dr. Holinger on the best moving picture I have ever seen. It is marvelous.

I had a patient just last week in whom there was an extremely long epiglottis, which Dr. Holinger has shown in several cases, and it was folded vertically so that the ary-epiglottic folds were brought together. The introduction of an anterior commissure laryngoscope spread the folds and the child is very much better.

I would like to ask Dr. Holinger, if he had that experience with the simple procedure where the child had intermittent stridor, the stridor was relieved when the child was placed on its face? When on its back the folds fell over the epiglottis. I would like to ask Dr. Holinger, repeating Dr. Jackson's question, if that procedure has proved of benefit in cases of this type with him?

DR. PAUL H. HOLINGER: Thank you, Dr. Tucker and Dr. Jackson! I have not found, or have not noticed that there is any therapeutic effect upon the larynx from the introduction of the laryngoscope. It would seem theoretically possible and certainly gives one the desire to try it and determine whether or not there is a change. I shall indeed look for it in the future.

## SOME OF THE INDICATIONS AND CONTRAINDICATIONS FOR HORMONAL THERAPY

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This discussion will be limited to ACTH, cortisone, estrone, testosterone, some of the indications and most of the contraindications.

The reasons for presenting this subject are as follows: 1) To review some of the indications for the use of hormones in the treatment of certain specific diseases; 2) to sound ample warnings about possible serious sequelae, if contraindications are not thoroughly understood and observed, and 3) to strongly emphasize what the writer believes to be a fact, namely, that hormonal deficiencies and imbalances are, as a rule, multiple and only one part of a general deficiency. Nutritional and electrolyte deficiencies and imbalances must be considered concurrently.

The latter will be stressed throughout this report as it seems to be the least understood, and certainly accounts for many therapeutic failures in the use of hormones. While adequate documentation is admittedly lacking, the writer has a large amount of clinical evidence pointing to its factuality. It is interesting to note that McCarrison<sup>1</sup> made some pertinent observations based on well controlled animal experiments as follows: "My studies have provided some interesting contrasts in the behavior of certain endocrine organs in the presence of food faults. Thus, the adrenal glands tend to undergo enlargement while the thyroid tends to atrophy in the presence of food deficiencies. When, on the other hand, the food is excessively rich in proteins and fats without any deficiency of vitamins, the thyroid tends to enlarge and the adrenals to diminish in size. In the former case the body appears to need more adrenal and less thyroid hormone; in the latter, more thyroid and less adrenal hormone. The combination of effects produced in the thyroid and

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adrenal glands in the presence of food deficiency is paralleled by the observation of Herring<sup>2</sup> that in rats fed on thyroid extract the adrenals almost double their weight, while their epinephrine-content is also increased; and by his more recent observation that the adrenals increase and the thyroid diminishes in size in consequence of pregnancy. The pituitary enlargement—occurring as it does in the organ resulting from thyroidectomy and from castration, the similarity being carried further when the thyroid and testicular atrophy resulting from avitaminosis are remembered.

“It seems, therefore, that both the functional perfection and the correlations of the endocrine organs are dependent upon a properly balanced and vitamin-containing food supply; dietetic deficiency means endocrine insufficiency.”

Until we have conclusive evidence to the contrary McCarrison's well documented and controlled animal studies of the behaviour of certain endocrine glands in the presence of various nutritional deficiencies should be accepted. There is a vast amount of clinical observations which confirms his experiments. He emphasizes throughout his book the crucial importance of balance in the endocrine secretions and balance of the vitamin-containing foods. He omitted discussion of the electrolytes, probably because so little was known at that time. Much knowledge is still lacking today.

It is of practical interest to note that in rats fed on thyroid extract the adrenals almost doubled in size with an increase in epinephrine content. It is not unusual to have patients report that they have been advised to take thyroid extract the remainder of their lives because of a low basal metabolic rate, without giving a thought to other means of promoting a more normal physiological activity of the thyroid. This and the misuse and abuse of the female hormones have not added to the prestige of our profession. The latter has at times been used almost to the point of brutality! It should be obvious to all that an imbalance is created which will produce a dysfunction throughout the entire delicate mechanism of the whole endocrine system.

The toxic effects of the imbalances have not been adequately stressed. There was a report recently published by Grant and Leopold<sup>3</sup> which seems to emphasize the dangers of large doses of cortisone (100 to 300 mg daily for three years) without the concurrent administration of potassium chloride and the sex steroids.



This young woman, 33 years of age with a severe rheumatoid arthritis, developed a Guillian-Barre syndrome with paralysis of both legs and the left facial nerve. Her face was moon-shaped.

The authors postulate that the Guillian-Barre syndrome was caused by an infection starting in the gastro-intestinal tract and involving the central nervous system. They further postulate that the cortisone may have had nothing to do with the syndrome, or perhaps it helped its development by breaking down the usual barriers of resistance. The writer respectfully suggests that neither postulation may be entirely correct. No one has demonstrated that the Guillian-Barre syndrome has an infectious genesis.

It seems quite likely that the cortisone given in such large doses over such a long period of time did have some direct effect by seriously disturbing the endocrine balance and the whole electrolyte system. It would have been interesting to know how this patient would have reacted had the following regime been carried out: 1) Adequate doses of potassium chloride and other electrolytes during the entire course of cortisone therapy with sodium restriction; 2) mixed sex steroids; 3) vitamin supplementation and, 4) adequate nutritional regime.

Kinsell and Partridge<sup>4</sup> summarize the present stage of hormonal therapy as follows: "The accumulation of knowledge and the development of concepts regarding the place of ACTH and cortisone in clinical medicine have followed an interesting pattern during the past two years. The first period might be termed the *phase of incredulity*. This persisted a relatively short time. The second phase would be dubbed properly the *era of extreme euphoria*. The third period is now with us. Its most outstanding characteristic is the *alarm reaction*."

Perhaps the word "alarm" should be changed to "caution." There is no doubt that hormones have great possibilities of producing dangerous sequela if the indications and contraindications are not thoroughly understood and obeyed. Until such time as we have a chemical or biological assay that will determine accurately a deficiency, or imbalance, we must proceed carefully along accepted, conservative routes and detour at great risks to our own reputation and to our patients' well being. One must be always alert to obvious changes in electrolyte metabolism, due to salt and water retention. This is easily detected by weight gain and tissue edema. Hormones

have such great potentialities for good that their cautious use should no longer be delayed. It is of vital importance to correct all other deficiencies and imbalances, thereby giving the endocrine glands a healthy and congenial chemical environment in which to function and to produce their own hormones by normal synthesis. The exception to this rule is when these glands have been destroyed by surgery, radiation or disease.

#### INDICATIONS FOR HORMONAL THERAPY IN OTOLARYNGOLOGY

*Asthma that is refractory to conventional treatment*, usually of the intrinsic variety. ACTH or cortisone is indicated. Results are excellent in selected cases. Keep the dosage low and reduce it some each day. Short courses of treatment as often as required are desirable and safe. The addition of sex steroids in older persons is nearly always indicated for hormonal balance.

*Hay Fever*. If the patient has not had or fails to respond to desensitization or antihistaminic therapy, ACTH or cortisone is indicated with helpful results. There are short, sometimes long, periods of relief.

*Perennial Allergy*. In older persons conventional therapy frequently fails. Cortisone combined with the sex steroids is usually quite rewarding. Again small dosages should be given over short periods of time to determine the continuing need.

*Drug Sensitivities*. In drug sensitivities, such as contact dermatitis, ACTH or cortisone and neo-cortef should be used locally. The results are good.

*Tension States*. Vascular headaches respond to sex steroids, Estrone and testosterone, cortisone 12½ mg twice daily. The results are very good in selected cases. One should discontinue medication frequently as a control to determine its need.

*Idiopathic Granuloma*. Here, ACTH or cortisone may be life-saving at times.

*Hemorrhages*. In nasal, orbital and bronchial hemorrhages or in those of unknown origin, probably vicarious menstruation due to a disturbance in the sex steroid levels, especially imbalances, sex steroids and cortisone are indicated.

*Degenerative Diseases*. In acoustic, ocular and other dysfunctions, occurring in the aging patient, sex steroids plus the conco-

mitant administration of ACTH or cortisone are indicated. Results are doubtful and still in the experimental stage but certainly worthy of further clinical trial before final evaluation.

#### CONTRAINDICATIONS FOR HORMONAL THERAPY

One should remember that the past is a learned prologue of the future. Until time tested, all new therapeutic measures may be quite beguiling, and at times illusory. Until complete information is available regarding the physiological effects, the mechanism of their action, the indications for, and the possible serious sequelae of the steroids, the following should be considered, for the time being at least, as contraindications: Malignant hypertension, congestive heart failure, severe diabetes, chronic glomerulonephritis, Cushing's syndrome, psychoses and psychopathic personalities, hirsutism, peptic ulcer, tuberculosis, active or recently healed. Certain experimental work has suggested that estrogens are carcinogenic when administered to animals which have inherited sensitivity to breast cancer. Great caution should be observed in women who give a familial history of carcinoma and the steroids are absolutely contraindicated if there is a personal history of breast or genital carcinoma.

This warning in no way applies to testosterone, the other sex steroid, which is so frequently indicated in women for tensions and hemorrhages. There is some accumulated evidence strongly suggesting that testosterone, the antimetabolite of estrone, may be a valuable therapeutic agent in the management of carcinoma of the female genital tract.

Much basic research must yet be done before the diagnosis of any hormonal deficiency can be put on a scientific status.

At the time of this writing the estimation of steroid blood levels is still a research problem. It will probably remain on a research basis for some time as the technique for accurate determination of these blood levels is a very delicate, and so far an unstandardized procedure.

The presumptive diagnosis of sex steroid deficiencies seems to be more within the range of our present clinical knowledge; at least it is more easily assumed than any other hormonal deficiency.

Women have told us from time immemorial that their tensions, migraine headaches, their nervousness, their allergies, even their asth-

matic bouts, have at times disappeared completely during pregnancy, only to return post partum. We have never given these voluntary statements the diligent attention that was warranted by the physiologic facts observed by these patients. In addition, it was observed that there were many more intractable allergic patients among women than men. Therefore, it seemed obvious that in women the body was supplied with something during pregnancy, perhaps a better balance, that did not exist in the non-pregnant woman or in the male.

Until better diagnostic methods are developed one must rely to a great extent on a detailed gynecological history, which is not without merit and the subcutaneous injection of a therapeutic dose of the steroid which is often quite helpful. It must be remembered that a positive diagnosis of a steroid deficiency cannot be made by laboratory or other objective methods.

The following questions should be answered by the patient if the physician is to be assisted in making a presumptive diagnosis of a sex steroid deficiency or imbalance: 1) Beginning with the menarche, did the patient exhibit any abnormalities such as menalgia, menorrhagia or amenorrhea? The last is especially important if it occurs in the obese young female or in the post partum period. 2) What was the condition of health during pregnancy? Did migrainous and other types of vascular headaches disappear or become less severe? 3) Did allergic conditions such as hay fever, asthma and perennial allergies improve or disappear entirely?

The injection of a therapeutic dose of either estrogen or androgen is a simple and practical test but it is in no way a completely reliable method of determining a steroid deficiency. It is suggestive and should be used. The patient must report the clinical response within 24 to 48 hours. If the patient observes a definite improvement in tension and other symptoms, the test must be considered as strongly suggestive of a deficiency or "slightly positive." It is frequently noted that the patient will report such a marked relaxation from tension that he will describe the reaction as one of complete exhaustion. This is more apt to happen when one of the androgens are being given, either to the male or female.

If there is no change at all, as frequently happens, the test should be recorded as negative. If the tension or other symptoms are made worse it is indicative of an over-supply already in the body of the



steroid being tested. Do not give the patient this steroid, but consider the opposite sex steroid. Estrogens and androgens are anti-metabolic. The gynecological history as outlined above has a much more conventional connotation than the therapeutic injection as it is not influenced so much by the emotions of the patient.

#### PRINCIPLES OF HORMONAL THERAPY

Before any specific therapy is suggested certain fundamental principles must be recognized. For example, the adult male produces a small amount of estrogenic substance which largely comes from the adrenal cortex, perhaps a small amount even from the testicle. The adult female produces significant amounts of androgen in the adrenal cortex. In the treatment of many conditions in both men and women several authors have suggested a ratio of 20 to 1 testosterone and estron. This ratio has not as yet been finally determined. There is great individual variation which must be carefully considered. It is therefore incorrect to speak of separate male and female hormones. No normal woman is without masculine secretion; no man is without female secretion. Someone has remarked "No Adam without an Eve."

Many investigators have postulated that the androgens probably have a direct effect on the pituitary gland by stimulation, which may in turn increase ovarian activity in the same manner. Anti-metabolic activity or neutralization between estrone and testosterone due to chemical structural resemblance has been espoused by Woolley<sup>5</sup> but this theory has not met with favor among endocrinologists.

There is already impressive clinical evidence to indicate that the concomitant administration of the sex steroids with cortisone may avoid some of the adrenal changes and negative nitrogen balance associated with the use of cortisone alone. This evidence strengthens the writer's postulation that if there is any endocrine deficiency it is nearly always multiple and is accompanied by nutritional and electrolytic deficiencies and imbalances. Vitamin C and the whole B complex given intravenously greatly enhance the therapeutic effect of all hormonal therapy, because they supplement other deficiencies.

Of course, an adequate nutritional regime is vital to the patient's welfare. Oral vitamin and mineral supplementation must be continued during the critical periods. The suggested therapy for a few

specific diseases to follow is, of course, crudely unfledged. Until we are better informed on the physiological effects of the hormones and the mechanism of their actions each physician must exercise profound and discriminating judgment in the selection of a relatively new and mostly untried therapeutic agent. Not all diseases listed as indications for possible hormonal therapy will be discussed.

We have accepted certain acoustic dysfunctions as a necessary part of aging. Nothing has developed so far to refute that time-honored conviction, perhaps nothing will. However, the widespread recognition of the interrelationships existing between steroids and metabolic processes emphasizes the importance of a possible endocrine approach to the problem of aging and it is hoped in the future to delay some of the degenerative changes which usually follow. Goldzieher and Goldzieher<sup>6</sup> point out that the loss of ovarian or testicular hormones in the aged withdraws one of the most important metabolic regulators of the organism. In support of this statement, attention is directed to the protein-building properties of both estrogen and androgen, their combined effects on calcium metabolism and the mineral content of the skeletal system. Even the central nervous system, these authors state, is under the influence of the gonadal hormones and strongly reacts to their absence.

This would again seem to support the writer's hypothesis that acoustic dysfunctions, as well as all the cranial and even the autonomic nervous system, probably react adversely when there is a deficiency in these important hormonal metabolic regulators.

Ample clinical evidence to support this hypothesis is not immediately available. Clinical investigations are going forward. Masters<sup>7</sup> points out that this has been deduced from the fact that up until the age of menopause the female has much less atherosclerosis than the male, and lower concentration of SF 10-20 molecules. However, the incidence of atherosclerosis increases as estrogen production diminishes, and when the female reaches the age of about 60, it is approximately the same as the male. Masters further states that, on this basis, it would appear desirable to administer estrogen in small doses to most postmenopausal women.

Extending the concept of metabolic interdependence, some workers, (Goldzieher and Goldzieher<sup>6</sup>) feel that steroid therapy cannot exert its optimum effect unless the nutritional requirements are adequately maintained. Vitamins are necessary for the elaboration

of basic enzyme systems, and the B vitamins as a group affect the intrinsic metabolism of all tissues. Furthermore, in line with the current trend, which increasingly emphasizes the concept of inter-relationship in human metabolism, Goldzieher and Goldzieher point out that vitamin supplementation in the aging patient is not only important from a nutritional standpoint but also necessary to increase the effectiveness of steroid therapy. It is believed by some that the steroid hormones may influence enzyme concentration, may be component parts of an enzyme system or may act as an accelerator or inhibitor of the enzyme systems. There is a multiplicity of enzyme systems, known and unknown, in the body. Also, there is a variability of structure and physiological activity. The enzymes are variously described as organic, frequently a protein or inorganic, a colloidal solution of a metal. Warburg's enzyme is an organic iron compound present in all tissues, and the primary factor in cell respiration. There are yellow enzymes with nine substances which take part in oxidations and reductions; all contain riboflavin. It will be seen from the above that proteins, metals and vitamins go to make up enzymes and that hormones may be component parts of an enzyme system. The importance of the pancreatic enzyme trypsin, as it may influence thrombotic lesions occurring any place in the body from the acoustic labyrinth to the extremities, has not been finally evaluated. The writer is now conducting such a study. It seems the pursuit of this investigation may be rewarding.

#### TREATMENT OF SPECIFIC DISEASES

*Asthma.* ACTH or cortisone in intrinsic asthma is definitely indicated and may be life-saving. Intravenous administration of 25 units of ACTH in 1000 ccs of 5 per cent glucose, a drop every three or four seconds. The infusions require six to eight hours to complete. Three or four such infusions, sometimes more, may be necessary. Fifteen units of ACTH in 500 ccs of 5 per cent glucose is frequently just as effective. The Thorn test for adrenal cortical function is important. In less severe asthmatic cases H. P. Acthar Gel® is the most satisfactory preparation for subcutaneous and intramuscular administration. Minimum dosage can be easily measured even in high potencies such as 80 Armour units per cc if an insulin syringe is used—H. P. Acthar Gel 40 to 60 units daily and continue two or three days as required, but reduce at least 10 more units each day.

If there is any question regarding cortical function or any other contraindication to ACTH, cortisone should be tried. Start the dosage at 75 mg or less per day and reduce 10 to 15 mg each day until 50 mg is reached. The 50 mg may be necessary for a few days, but the treatment should be discontinued as soon as possible. It appears to be safe to administer 25 mg of cortisone in divided doses over rather long periods of time. Watch for danger signs! Try small doses of ACTH 10 to 15 units subcutaneously daily for a short time. Rest periods with complete withdrawal are always indicated.

*Seasonal Hay Fever.* Hormonal therapy has not replaced the conventional preseasonal and seasonal desensitization although they may act synergistically with allergens. The hormones ACTH and cortisone should be used if the desensitization has failed completely or in part if the patient fails to respond to the antihistamines.

Symptomatic relief can be expected in nearly all cases. The duration of the relief varies greatly from a few days to a few weeks but long enough to assure ACTH and cortisone a definite place in our therapeutic program. Short courses of treatment, frequent rest periods, low dose schedules and regular observations make it a safe procedure.

Conservative optimism and continued study will, no doubt, clarify the picture so we may soon courageously administer these hormones with less fear of a serious sequela.

The concomitant administration of the sex steroids with ACTH and cortisone is nearly always indicated in older persons.

*Tension States.* There is an overlapping interest, in all branches of medicine, in the tension states. So frequently they are erroneously diagnosed as a "neurosis" or a "chronic nervous exhaustion." There is almost always a general deficiency background in which the sex steroids are most prominent. Benjamin<sup>8</sup> believes a favorable effect of the steroid sex hormones on circulation by relaxing arterioles and capillaries has been well established. He quotes Walker, "Since impaired circulation is at the bottom of the majority of all symptoms in the aging process, a circulatory improvement would be a self-evident advantage."

The above is of vital importance in the management of many acoustic dysfunctions of vascular origin; vascular headaches such as migraine, histaminic cephalgia, and many vasospastic conditions.



If the vascular condition any place in the body is thought to be of thrombotic origin, trypsin should be considered concurrently.

Goldzieher and Goldzieher<sup>6</sup> believes the duration of steroid therapy in the aged should not be limited in time but continued indefinitely since it is substitutional and symptomatic. They believe interruption of therapy ends most of the benefit.

#### CONCLUSIONS

- 1) Determine, if possible, a hormone pathogenesis of the disease to be treated.
- 2) Make certain that conventional therapy has failed.
- 3) Short courses of hormonal therapy with as small a dosage as possible is a safe procedure.
- 4) Observe the patient frequently and be alert for danger signs.
- 5) There is no chemical or biological assay at this time that will determine accurately a deficiency or imbalance of the hormones.
- 6) The presumptive subjective diagnosis of sex steroid deficiencies or imbalances is more within the range of our present knowledge. Case history and therapeutic test injection as described are helpful, but must be considered as only suggestive.
- 7) Hormones should be regarded only as one deficiency. All other deficiencies should be corrected concurrently. Supply the endocrine glands with congenial and adequate chemical media and they will usually synthesize their own hormones.
- 8) Discontinue all hormonal therapy as quickly as possible, except in the aged. Allow rest periods as a control to determine the continued need. Adequate dynamic therapy directed toward the correction of all suspected multiple deficiencies, even though there may be no grossly apparent pathologies, is often rewarded with gratifying clinical results.
- 9) Accentuation must again be placed on the importance of a balanced, combined, sex hormone therapeutic regime. This is especially true of the aging individual, male or female, showing signs of degenerative changes. Considering the aging female, it must be remembered that the estrogens are for replacement or supplementation of the diminishing natural hormones, while the androgens are given

for hormonal balance, and of equal or greater moment is their anabolic action in stimulating protein synthesis to increase muscular strength; androgens may also stimulate ovarian activity.

In the management of the aging male the androgens seem most important for their physiological activity in protein building, while the estrogens are chiefly for hormonal balance. The estrogens appear to have some beneficial effect on ectodermal tissues in the male. The writer seldom prescribes one sex hormone without the other. Both are given when ACTH or cortisone is being used.

10) The oral administration of estrogen, testosterone mixtures with nutritional supplement, is worthy of much more clinical trial. Estrogenic substances probably also have a stimulating effect on the output of ACTH and protein metabolism. Estrogens may stimulate osteoblastic activity and many cause an increased loss of hearing in women with otosclerosis during child-bearing periods. Testosterone, a known antimetabolic, might be indicated.

11) Hormonal therapy alone is never curative, usually helpful when indicated; always potentially dangerous and probably has great dormant possibilities as a new therapeutic tool when completely understood. At the present time hormones are of distinct value in the management of refractory allergic conditions in the tension states and probably are of value in certain degenerative changes occurring in the aging individual.

12) The androgenic hormones are chiefly of value as powerful protein anabolic agents, retaining nitrogen and building up protein. They are, therefore, the most universally accepted steroids for use in both sexes. When indicated, the estrogens and progesterones have a mildly anabolic action. The mechanism of this anabolic action is still unknown.

13) Clinical judgment is extremely important. One would not, of course, advise the androgens for women who already have obvious signs of masculinization such as dark hair with masculine distribution, oily skin and large bony frame. Neither would one suggest estrogen for women of the opposite type, unless specific indications were present.

14) Complete failure or, at least, disappointing results are almost sure to follow the correction of only one deficiency whether it be hormonal, nutritional or electrolyte.



Fig. 1.—Recurring intraorbital hemorrhage.

If the body metabolism has been such as to produce a deficiency in one hormone it most probably has depressed other endocrine secretions. Hormones have a synergistic action. The same is probably true of vitamins and minerals. I am willing to endorse the postulation that there is a harmonious interaction among hormones, vitamins and electrolytes. This is probably both catalytic and synergistic. They all form and function as enzymes. The enzyme trypsin is worthy of much more study.

#### REPORT OF A CASE

Only one case report is appended because well-founded knowledge of therapeutic results in hormonal therapy is still fragmentary. Conclusions may be premature, perhaps even fallacious. Criteria for evaluation is not well-defined. Other case reports will appear with another publication soon to follow.

Mrs. F. W. McR., aged 60 years, had seven intraorbital hemorrhages of unknown etiology from May 1 to September 15, 1953. The first attack occurred while she was sitting at a sewing machine. She appeared to have a "black eye," without much pain. The after effects of this hemorrhage lasted only a few days. The second episode, four weeks later, was much more severe and more painful. Blood oozed into the inner canthus through the lacrymal duct. Severe nasal hemorrhage was followed by nausea and vomiting of a large quantity of dark blood. She was admitted to a nearby hospital for a week. The third attack occurred in the early part of July and was even more severe. The vision in the left eye did not return when the swelling subsided as it had previously done. The other attacks

came on periodically until the middle of September when the seventh and last attack occurred; it was much less severe in every way. The patient's condition was as tragic as it was dramatic. She was seen by many consultants including an eminent neuro-surgeon.

Her menarche had begun at the age of 12 years. Almost from the beginning she had had severe menalgia which required bed rest and some sedation. Even as a child she passed large clots which usually relieved much of her menstrual discomfort. She has had a very painful hypertrophic arthritis for 26 years.

She had had five pregnancies: the first child was born when she was 17 years of age; the last child when she was 45. When she became pregnant the last time, 16 years ago, and for five years afterward, she had almost complete relief from her painful arthritis. However, it then returned with renewed severity.

During her menopause she had three severe uterine hemorrhages requiring long periods of bed rest and "shots" to control bleeding. These attacks occurred about once a year over a three-year period.

At the suggestion of another otolaryngologist I had the privilege of seeing this patient on August 4, 1953, after the fifth orbital hemorrhage had occurred. Her otolaryngological findings were negative. All laboratory findings including a left carotid angiograph were negative. Blood examinations revealed only a secondary anemia as would be expected.

She had seven transfusions which gave her some increase in strength but did not influence recurring hemorrhages. She had been given large quantities of antibiotics as one consultant proposed a diagnosis of erysipelas. Several hours were spent getting the gynecological history, and the presumptive diagnosis of an estrogenic deficiency or imbalance was made on the history alone. There were no other findings to substantiate this opinion. For academic reasons and to determine a single deficiency if possible, the patient was placed on oral estrogens (liquid premarin) on August 6, 1953. There was a marked improvement in her arthritic pain. She was able to walk around the ward and to take care of herself. She was euphoric and so were her physicians. This euphoric period lasted only a few days, after which her arthritic pain returned with increased severity.

On August 14, 1953, she had another severe intraorbital hemorrhage. She was put on cortisone, combined with the estrogenic substance, vitamins, minerals and an adequate nutritional regime plus



dilute hydrochloric acid. She is now continuing all therapy as outlined and her family physician is giving her ACTH for her arthritis. She has had no recurrence of the orbital hemorrhages.

*Comment.* It is, of course, impossible to conclude that her recurrent orbital hemorrhages were due to a steroid deficiency or imbalance. It is much more likely there was a general deficiency existing, including the steroids.

The gynecological history was strongly suggestive. The clinical results were quite convincing. An attempt to manage her therapy on a single deficiency basis proved futile. The balancing and correcting of all deficiencies seems to have been the proper clinical procedure. Her digestion and assimilation seem to have been greatly improved by the addition of dilute hydrochloric acid. The writer is in complete agreement with Hewlett and Scott,<sup>9</sup> who believe that "corticotropin and cortisone exert a beneficial effect on capillary fragility and bleeding phenomena in various types of thrombocytopenia. The improvement is not necessarily associated with an increase in circulating thrombocytes."

1110 PROFESSIONAL BLDG.

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## DISCUSSION

DR. FRANCIS L. WEILLE: Mr. President, Members and Guests, I was very much interested in what Dr. Roberts had to say and I think the paper is very timely.

I would like to mention two practical points: One, Dr. Francis Lowell of the Massachusetts Memorial Hospital and his group have found that 25 mg of cortisone four times daily for four days in hayfever has given an experimental group that they studied, around 75 patients I think, 60 per cent cures for the hayfever season. It is a useful point to have in mind and they claim that this dosage is not dangerous to anybody, as to the biochemical side effects or physiological side effects with this small dose and its duration.

The second point, a group of 23 status asthmaticus patients have been treated with the utmost care at the Massachusetts General Hospital over a long period of time, receiving dosages of about 75 mg of cortisone or more daily for a period of many months, about a year in other words. Their physiology and pathological physiology was watched with the greatest care and apparently everything was going along well when three of the patients developed fractures of the spine. Dr. Fuller Albright's group was called in to explain why this happened. Nothing seemed to have been wrong in the management of these cases. Well, it was very simple. Osteoporosis was mentioned here. You remember that cortisone depresses protein synthesis. In forming bone you have to have osteoid. To have osteoid you have to have normal protein and here osteoid was not formed. Bone is gradually removed in normal physiological processes always, not necessarily through osteoclastic activity and when a patient lost about 40 per cent of his body calcium through the urine he began to have fractures of the spine. It would be the spine or pelvis because those are the bones which bear most weight in the body. This is mentioned not to alarm you but to make you aware of the seriousness of prolonged ACTH or cortisone therapy. Once these fractures have occurred there does not seem to be much to do about it. The point is however that if you give the hormone for a great length of time you have to watch the calcium output in the urine in 24 hour specimens.

DR. SAM E. ROBERTS: Thank you, Dr. Weille, for your discussion.

While my results with hormonal therapy have been gratifying and the continued use of these valuable preparations is considered indispensable, I have not, however, observed many cures. Improvement and arrestability of conditions with a hormonal pathogenesis is easily and safely achieved.

I have stressed the dangers in hormonal therapy and I am glad Dr. Weille mentioned the spinal fractures.

Let me emphasize again that hormonal deficiencies and imbalances are nearly always multiple and a part of a general deficiency. To attempt to correct one without the others can only lead to therapeutic failure by creating an even greater imbalance, both electrolytic and hormonal.

A notable example of this imbalance is the continued misuse of estrone almost at times to the point of brutality.

Accumulating evidence is demonstrating the outstanding therapeutic value of the mixed hormones, estrone and testosterone in proper ratio in most tension states and many allergic conditions, in both men and women. This is especially true in the management of vascular headaches of all types and in circulatory disturbances any place in the body, including the labyrinth.

Where thrombotic areas are suspected, Parenzyme (trypsin) should be used concurrently with the mixed hormones.

In closing I wish to state that I feel very strongly that many patients are being incorrectly diagnosed as having a chronic nervous exhaustion or some form of a neurosis. These patients could be materially helped by the judicious use of mixed hormones and by the correction of other deficiencies and imbalances. This is especially true of the aging individual, but it does apply in a lesser degree to the younger persons.

# PRINCIPLES AND PROBLEMS IN SURGERY OF THE NECK

GRANTLEY W. TAYLOR, M.D.

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It is presumptuous to undertake a discussion of neck surgery with the idea that we are dealing with a specific organ or system, such as we assume when we refer to stomach surgery, or genitourinary surgery. The neck is a corridor, an essential passageway between the head and face above, and the thorax and the rest of the body beyond. Surgery of the corridor is commonly undertaken for the correction or relief of conditions originating beyond its borders, and presupposes understanding of the pathological and anatomical problems of the adjacent areas.

The neck is really the province of many fields of surgery, of importance to many of the surgical specialties. The neurosurgeon is concerned with intracranial sepsis, vascular anomalies, cervical disc protrusions, and peripheral pain problems. The orthopedist also deals with disc protrusions, as well as torticollis, anatomic anomalies, and radiculitis. The plastic surgeon copes with the problems of contractures and scars, and other reconstructive problems. The endocrinologist presides over the vast field of thyroid and parathyroid disorders. The thoracic surgeon has long considered the adjacent neck as his own field, from phrenic nerve interruptions to esophageal reconstructions. Finally, the oral surgeon and laryngologist have a major interest in this area, not only in coping with direct problems of the corridor passageways, but in dealing with metastases from malignant neoplasms invading the cervical lymphatics. In summary, the neck is a common meeting ground for many surgical specialties, and the interrelations of the fields calls for frequent cooperative attacks on disease in the area.

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The basic requirement for neck surgery is a complete familiarity with the anatomy of the region. While this is prerequisite for all surgery, nowhere are important structures so closely crowded and interrelated as in the neck. The identification and preservation of anatomical structures demand a painstaking and fastidious technique. Hemostasis must be accurate and complete, adequate exposure must be secured by proper placement of incisions rather than by forcible retraction, and careful sharp dissection is preferable to blunt prodding and rooting to avoid accidental damage. It must be added that placement of incisions must also conform to the requirements of a cosmetically acceptable scar.

The necessity for complete anatomical knowledge and fastidious technique implies that neck surgery should be performed only by experienced surgeons. The training of residents and apprentices in this exacting field must be under the direct supervision of a surgeon completely familiar with the contemplated operation, and each step of a procedure must be planned and understood. Even the simplest operations may present pitfalls and lead to complications in the hands of the careless and unwary. For example, one of my medical colleagues collected a series of patients with spinal accessory nerve damage following cervical lymph node biopsy. In another instance, out-patient local excision of a node resulted in injury to a trunk of the branchial plexus. Numerous other examples could be cited, but it seems unnecessary to elaborate the point.

Granted a skilled and experienced surgeon, expert anesthesia is the next essential for good neck surgery. While many procedures may be performed under local infiltration or field block anesthesia, they become wearing and fatiguing for both patient and surgeon. We prefer a general anesthetic agent, with provision for maintaining a free airway, and for suction to remove secretions. Adequate oxygenation must be assured at all times. Intratracheal intubation with an inflatable cuff is most satisfactory. Tracheostomy should be performed without hesitation if it is desirable.

A basic principle of neck surgery has been to preserve intact those structures which serve as passageway for essential functions of the body. Classic operations of the neck therefore required preservation of the cervical cord and spine, the esophagus and trachea, and the vessels concerned with the blood supply of the brain. These restrictions imposed limitations on the scope of dissections, notably in

coping with malignant neoplasms in which primary or secondary involvement of some of these inviolable structures anatomically precluded a successful total eradication of the disease.

Modern advances in neck surgery have resulted in attempts to reappraise the inviolability of some of these structures, and to provide adequate alternative functioning pathways when the natural ones must be sacrificed.

I shall briefly consider some of these advances and their associated problems.

One of the first structures to be sacrificed with impunity was the internal jugular vein. This permitted control of the spread of infection, and opened the way to the development of the concept of radical neck dissection for malignant metastatic lymph nodes. There has been considerable experience with bilateral sacrifice of the internal jugulars, either simultaneously or in stages. This has been accomplished in numerous cases, but reports of disasters lead to the conclusion that the procedure is potentially dangerous or disastrous. A lumbar puncture needle connected with a manometer may permit some control of the sudden increase in intracranial pressure which follows occlusion of the vein.

Ligature of the common or internal carotid has long been recognized as dangerous and often fatal. When vascular anomalies are present, such as intracranial aneurysms or arteriovenous fistulas, collateral circulation may have developed which makes ligature safer. Procedures for gradual progressive occlusion over hours or days, as with a screw clamp, offer some promise of diminishing the dangers. Recently there have been explorations of free vessel grafts to replace sacrificed segments of the common and internal carotid vessels. Since the time of occlusion necessary to place a graft may in itself be long enough to cause brain damage, temporary by-pass plastic tubes may be helpful. Experimentally hypothermia during these operations appears to minimize cerebral damage.

Surgery of the pharynx has received great impetus since improvements in anesthesia and in the control of infection have controlled the prohibitive mortality and morbidity in this field. The discouraging radiation cure rate for malignant disease in this area has stimulated the operative approach. Reconstructions by flap grafts or free grafts around a mold or stent have been to some degree suc-

cesful. The swallowing function—passage of a bolus with protection of the airways from inhalation of food—is essential for the comfortable existence of the patient. In no field of surgery is it more important for the surgeon to evaluate his procedure not in terms of technical success but in terms of the increased span of comfortable or tolerable existence for the patient.

Radical surgery for carcinoma of the larynx is too firmly established to require discussion. While the voice is not an essential, vital function, there has always been a great reluctance to render a patient mute. The development of alternative pathways, either with the artificial larynx or preferably with esophageal voice, has permitted a freer and bolder attack on laryngeal cancer. Success with intrinsic carcinoma has encouraged operations for extrinsic carcinoma. The survival of large numbers of patients after laryngectomy has given time for the development of regional node metastases in many patients, and brings up the problem of regional lymph node dissections.

Neck dissection in the management of metastatic carcinoma of the cervical lymph nodes has been standardized for many years. Disagreements exist as to the indications for and the extent of dissection. In view of the high degree of error in clinical diagnosis of cervical lymph node metastases, we undertook some years ago a study to determine whether there are characteristics of the primary carcinoma correlating with the occurrence of lymph node involvement. In summary we found an increased occurrence of metastasis associated with longer duration, greater size, higher grades of malignancy, and invasive rather than proliferative mode of growth of the primary carcinoma. Recurrent cases, as opposed to those without previous treatment, presented a higher incidence of node involvement. These factors were of importance in varying degree in relation to all locations of primary carcinoma. There was marked difference between the sites of primary disease in the occurrence of metastases. In general, metastases were very common from primary carcinomas of the pharynx, extrinsic larynx, soft palate, tonsil and fauces; less common but frequent from the tongue and floor of the mouth; least common from the cheeks, lip, gingivae, intrinsic larynx and hard palate.

There has been some dispute as to the desirable extent of a neck dissection. Obviously, the objective is to remove all the nodes likely to be involved in metastases. While the anatomists have contributed greatly to an understanding of the normal lymphatic pathways of

drainage, their findings have been amplified and made concise by the clinical observations of numerous surgeons and pathologists. On the basis of our own experience we have considered a supraomohyoid dissection adequate to cope with metastases from most cases of carcinoma of the lips and cheek, reserving the radical dissection for the more malignant lesions of the tongue, floor of the mouth, soft palate and fauces, which drain directly into the deep cervical lymphatics.

The question of bilateral dissection arises for lesions encroaching upon the midline, and along with this, the decision as to whether both sides should be dissected at the same session. These questions must be solved for each individual case.

In recent years there has been a revival of employment of a one stage end bloc combined operation on mouth and neck, based upon the concept of the so-called "ideal" cancer operation which includes removal of the lymphatic vessels intervening between the primary focus and the regional node metastases. These operations are based on the assumption that the mode of lymphatic spread is by permeation, that is, a propagating thrombus of living cancer extending from from the primary disease to the involved node. While this mechanism may occur in some cases, especially in the late stages of the disease, the usual mechanism is embolic, and the intervening lymphatic vessels are free from involvement. Although the combined one stage operation is thus based on a false assumption, its employment has increased the scope of operation and the operability of many lesions, notably of the jaws, floor of mouth, base of the tongue, and the fauces. The increasingly radical nature of these operations has required development of reconstructive procedures by bone grafts and prostheses.

Another problem which arises in the neck is the appearance of a large node or nodes, demonstrated to be metastatic carcinoma by pathological examination, for which no primary carcinoma can be discovered. Frequently, repeated thorough examinations over a long period of time are necessary before an apparently insignificant primary carcinoma is discovered, usually at the base of the tongue or the pharynx. It is a tribute to the zeal and skill of our laryngological colleagues that there are fewer of these cases with undiscoverable sources now than there were twenty years ago. These cases used to be considered to be of branchiogenic origin, but many surgeons now doubt the existence of bronchiogenic carcinoma.



This brief summary has omitted many of the problems of neck surgery which are still under investigation even in the field of oral surgery and laryngology. Basically, all surgery of the neck requires recognition of the corridor function of the region, and must respect the anatomical and functional integrity of the passageways, or provide adequate substitutes for them. Viewed in this light, neck surgery is a broad field in which the possibilities for progress appear to be almost unlimited.

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#### DISCUSSION

DR. HENRY B. ORTON: Mr. Chairman and Members, I have enjoyed this paper very much. For many years, as a great many of you know I have been deeply interested in this field of surgery, and I agree with Dr. Taylor when he states that you must be absolutely familiar with the anatomy of the neck before you attempt to do very much in it.

On these problems of bilateral dissection of the neck, I heartily agree with him. Unless the second operation has had a certain interval of time before the second jugular is ligated in these cases, we may run into difficulty.

Dr. Taylor mentioned Dr. Wookie of Toronto. I would like to make a suggestion here so it is on the record, that Dr. Wookie's operation of lateral pharyngotomy and these plastic procedures were inaugurated by Sir Wilfred Trotter of London, and I had the pleasure in 1927 of spending the entire summer in London with Dr. Trotter doing these lateral pharyngotomies. It is Dr. Trotter's procedure and not Dr. Wookie's.

Again I wish to say how grateful I am to have been present to hear Dr. Taylor's paper.

DR. OSCAR V. BATSON: Mr. Chairman, Dr. Taylor, Fellows of the Association, I am grateful to Dr. Taylor for his mention of my work on the vertebral veins and for his references to the anatomic background of this problem. I first comment on the problem of bilateral ligation of the internal jugular vein. In 1944 in a conference on cerebral blood flow, I quoted the experiences of a Fellow of this Association, Dr. Phillip E. Meltzer, in bilateral ligation of the jugular veins with successful clinical results. The experiences of Dr. Meltzer were not unique in the days when mastoiditis was frequent. Today we are more concerned in finding out when bilateral ligation cannot be done, rather than in advocating ligation as a surgical procedure. Certainly the ligation of both internal jugular veins is a major step.

As I see the problem, it is a question of maintaining an adequate minute-volume flow of blood through the central nervous system. Omit the cases in which the brain cells are unable to use an adequate supply of blood and consider only the supply itself. The net volume flow can be reduced by blocking the arteries, or by blocking the veins. According to Gerard of Chicago, the central nervous system is thirty times more susceptible to anoxia than the peripheral tissues.

It is most necessary, therefore, to anticipate the problem of anoxia. The hazard varies with the different ages of the vascular system. In the young person with an elastic arterial tree, and a competent heart, the problem is simpler than in the older individual with the less elastic arteries and an aging heart. Unfortunately the radical neck dissection is done in this unfavorable age group.

When I described the vertebral vein system, I included with the vertebral veins, strict sense, all of the veins of the head and the neck, since they unite with the veins of the vertebral column. Venous blood may leave the head and neck, either by way of this peri-vertebral network or by way of the internal jugular veins.

I have studied four persons with occlusions of the superior vena cava. One of these had an occlusion of the superior vena cava and the vena azygos major. These occlusions were proved by exploratory operation. In this man, all of the blood from the head and neck and the blood from both upper extremities returned to the heart through anastomoses with the inferior vena cava. This man was a freight handler and as such did heavy lifting. He held his breath to stabilize his trunk in lifting. At such times the blood from the head and neck does not enter the thoracic cage but dams up in the vertebral veins and their associated tributaries. This enlarged network was able to substitute for the usual vessels. Nine months after the onset of this massive occlusion, this man had very few superficial veins visible. Two years after the onset of the occlusion, he had developed a caput medusae and was in general, much more comfortable.

Bilateral ligation of the internal jugular veins is obviously not as serious as a blocking of the superior vena cava and the vena azygos major. A professional man or office worker would have no occasion to enlarge the vertebral vein complex unless chronic constipation forced chronic breathholding upon him. Bilateral jugular ligation should be followed with a much stormier postoperative period in the office worker than in a labor or weight-lifter. With the general problem of anoxia in view, it may be possible to use medical supportive measures to aid the patient's post-operative condition.

On the arterial side, the ligation of the common carotid artery is quite different from the ligation of the internal carotid artery. The late Dr. Mont Reid of Cincinnati did a beautiful study on a series of cases with arterio-venous anastomoses. It was his idea that after ligation of the common carotid artery on one side, blood from the opposite external carotid artery, though connecting midline vessels filled the external carotid artery of the ligated side. From here blood entered the internal carotid artery. As Reid put it, the internal carotid artery of the ligated side becomes a branch of the external carotid artery of that side. This is so true that after ligation of a common carotid artery the temporal pulse may still be felt. Unfortunately this does not hold for all patients. Here again, it is a matter of the age of the arteries themselves since the carrying power of the rigid vessel, although dilated, is physiologically less than the carrying power of an elastic tube smaller in size.

A matter of collateral interest has been investigated by Dr. Samuel Hadden of Philadelphia. Hemiplegia may follow prostatectomy. The onset is generally in the early hours of the morning when the patient is inactive in sleep. Dr. Hadden concludes that the blood pressure has dropped to a point where the patient does not have a sufficient minute-volume flow through the brain. These cases of hemiplegia when properly treated may resolve in an hour or two. If it is not possible to give the heart support, the hemiplegia becomes permanent.

In radical neck dissections the limits of the dissection attempted have to be predicated in part upon the developed functional capacity of an alternate venous return flow and upon physiologic integrity of the heart and great vessels.

Thank you very much Dr. Taylor, for your clear exposition and for your interest in the vertebral vein system.

DR. FRANCIS L. LEDERER: Mr. Chairman, Members and Guests, at first sight and sound we have an impression of a marvelous surgeon and an administrator who would be a wonderful man to work with in the medical school. This plays a prominent role in all of our problems and since the paper deals with some of the problems of neck surgery, it is well that we mention them.

I was interested in a paper on "The Teaching of Medicine and Surgery as One Specialty" written by Oliver Cope, from Dr. Taylor's Department of Surgery at Harvard Medical School. It appeared in the *New England Journal of Medicine* in January of this year, and I quote, "Surgical specialties in their preoccupation with technique have tended to get too far out on the spoke. Otolaryngology and urology in particular give evidence of having lost contact with the hub. The other specialties too, such as gynecology and orthopedics, must be alerted to the shortcomings of the trend. It is time that they were drawn back and newer areas of special interest, such as thoracic surgery, should not be allowed to sever their contact with the hub. The practical step is the reintegration of otolaryngology, urology, gynecology and other special surgical interests into a broad department of surgery."

These polemics have been going on for some time and I am reminded of a remark of John W. Gerrie of Canada who stated: "Such a wide field necessitates full study and intensive training by those who are to undertake its practice. It is to be hoped that this training will be kept completely in line with the present requirements of Specialty training. However, one wonders if the emphasis on general surgical training, admittedly so important, has not gone far enough and if the incumbent trainee might not be given ample credit for time spent in familiarizing himself with the physiology of the mouth, with dentistry, with otolaryngology and ophthalmology, orthopedics, etc. These are part of a general understanding of the surgical problems of the body and more pertinent to the training at issue than say a knowledge of the relative merits of Bilioth I or Bilioth II in gastric surgery." We realize that the broad interest can be served and good training can therefore accrue. However, how much credit should be given to the man who is qualified in pathology and knowledge of the head and neck areas for which he has trained over a period of years, and would it not be well too that instead of dominance being sought on the basis of general surgical background the general surgeons work in this close harmony that Dr. Taylor intimated in allowing all residents to be trained in this area efficiently and effectively so that they too may carry on in this manner that Dr. Taylor speaks of?

Thank you!

DR. GRANTLEY W. TAYLOR: Gentlemen, I am honored to have provoked this discussion. I realize I probably opened topics which could preoccupy the Society for a considerable time.

I am grateful for Dr. Orton's referring to Dr. Trotter's work in this field. I didn't mean to attribute to Dr. Wookie any priority. Actually I remember doing a number of these operations with Dr. Edwards-Herman 20-30 years ago at the Huntington Memorial Hospital, lateral pharyngotomies with attempts at

reconstruction. The field fell into disuse because of the sepsis and disasters due perhaps to anesthesia. I think the whole field of this type of surgery has been reopened and reactivated by the improvements in anesthesia and in the control of infection. I don't know who started the pharyngotomies. I am sure Dr. Herman didn't. But neither did Dr. Wookie, neither did Dr. Trotter, but they all were working on principles which had been soundly laid down earlier. But I am glad to have attention drawn to Dr. Trotter's work.

I am honored to have Dr. Batson talk about this problem of circulation. I didn't realize that he would be present or I should have called upon him for discussion. I think that his contribution in relation to the spread of malignant disease, of his emphasis on the vertebral veins and their ancillary venous circulation is a major contribution. I think the problem in dissection in surgery is not the problem confronting the anatomist, because while his vertebral venous plexus is perfectly capable of carrying on adequate circulation, other things being equal, so often in our problems of practical surgery the other things are not equal. We cut a great deal of the accessory circulation which serves as an alternative pathway in a pure anatomical experiment. In the course of a dissection what so often in theory is adequate return, in practice proves not to be quite adequate. I have done bilateral jugular ligations, sometimes simultaneously, more often in sequence in two stages. I never do it without a good deal of trepidation, a good deal of anxiety for a considerable time and I have had misadventures and disasters in this.

The collateral circulation after common carotid ligation by the internal-external carotid hookup is of great importance. Suture of the external to the internal when the carotid bifurcation has to be resected is feasible, but again, in the course of dissections we sacrifice so many of the collaterals crossing from one side to another that often the common carotid ligature itself is a dangerous one.

The reference to Mont Reid's work with the pulsating aneurysm brings out what I said earlier, that a pre-existing vascular anomaly often has resulted in the development of adequate collaterals, which also Dr. Batson suggested when he said that sometimes the ligation of the common is not effective in controlling aneurysms. You must get closer to the scene because of the development of collaterals.

Dr. Lederer's quote from Dr. Cope on the overall training brings up Dr. Pressman's question of how should we train men who enter this field. In recent years we do not have special urological interns, residents at the Massachusetts General Hospital, but the urology is handled by passing through that department all of the general surgeons in their career and letting them all achieve a degree of familiarity with this field. At the Massachusetts General Hospital when I was training there the surgical residents all passed all too briefly through the otolaryngological clinic. At that time I had the privilege which I remember with gratitude of working briefly with Dr. Mosher. I don't suppose he ever would have taken any pride in my being there. It was a wholesome, helpful experience, and it gave me a certain degree of familiarity with the problems that lay in that field and interest in them. Eventually we may come to the point of having a basic board of surgery which will certify people that have had training in surgery and let them specialize from there out. Let us say the basic board of surgery would represent an examination passed as a sort of bachelor's degree, about three years after graduation from medical school, three years' experience in the fields of general surgery and then let them go and be otolaryngologists or orthopedic surgeons, or neurosurgeons. We certainly feel the lack of general training in the field of neurosurgery in which the boys go into it too early and have not enough appreciation of overall surgical problems. I agree that there is no sense in teaching a Billroth 1 to a man who



wants to do head and neck surgery, but he will have seen a good deal of surgical shock if he has taken care of Billroth 1's. It may be his knowledge and understanding of control and combat of surgical shock will stand him in stead when he gets off on a branch in the head and neck surgery, and similarly I think a broad grounding in general surgery is desirable and that the specialization should not take place at too early a stage. I have proposed to Dr. Schall from time to time that it might be desirable for him to integrate more closely with general surgery his training in his intern-resident group. If we could take his boys for a couple of years and ground them in general surgery, then they could more easily perhaps and more rapidly perhaps acquire the special techniques and field of knowledge requisite in laryngology. Let the people be trained first as overall general physicians and surgeons, and then enter their field of specialization rather than prematurely.

## A STUDY OF LYMPHOID TISSUE OF THE NASOPHARYNX

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In the course of the study being carried on at the Winthrop Foundation of the Massachusetts Eye and Ear Infirmary into the causes and treatment of hearing impairment, we found a significant number of children in whom intermittent tubal closure appeared to be related to recurrent adenoidal growth. In many of these young patients, moreover, neither surgery nor postoperative x-ray treatment of the lymphoid tissue put a stop to regrowth. We began to ask ourselves whether these cases required special surgical techniques. Was allergy perhaps at the bottom of the trouble? What could be done with irradiation? What kind of irradiation—x-ray or radium? We soon found, in searching for the answers to these questions, that we would have to begin at the very beginning and find out all there was to know about nasopharyngeal lymphoid tissue. What is lymphoid tissue? What purpose does it serve in the organism?

A survey of the literature made it clear that we were dealing with an extremely complex subject. Much of it was conjecture; a good deal of it, controversial. Even the most qualified students of lymphoid tissue confessed that many of the fundamentals were a mystery. Most involved of the myriad aspects of the problem, perhaps, was the endocrine question. Through that particular maze the otolaryngologist had best not wander without the guidance of the endocrinologist. Certain other aspects of the study could best be approached with the help of the pediatrician.

These areas still remain to be explored. At this juncture we have not got beyond a thorough survey of the literature, some histological studies which may be of interest, and an evaluation of the results that have been claimed for irradiation. Some of this work

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From the Winthrop Foundation, Massachusetts Eye and Ear Infirmary, Boston.  
Aided by a grant from Mr. Frederick Winthrop.

has already been done, notably so by Dr. Schenck<sup>14</sup> and others at the symposium held by the Academy of Ophthalmology and Laryngology in 1950. But it seems to me that the fundamental facts involved in a study so complex as this bears repeating.

The problem of nasopharyngeal lymphoid tissue resolves itself, as far as the otolaryngologist is concerned, into two distinct categories: the rhinological, and the otological.

A mass of adenoid tissue may obstruct the nasal airway and interfere with its function, often resulting in some rhinological disorder. When the mass is removed, the difficulty usually is cleared up. The tissue rarely recur in sufficient amount to again obstruct the airway; in any case, a second removal usually provides a permanent solution to the problem.

When the mass of adenoid tissue encroaches upon the tube simply by contiguity, any interference with tubal function can similarly be handled by removing the mass. This must be true of the great majority of patients with tubal disorders, to judge by the excellent results achieved in the great majority of adenoidectomies.

However, when tubal dysfunction recurs after operation, either following or in the absence of upper respiratory infection, then we know we are dealing not with a mere mass of lymphoid tissue mechanically obstructing the tube by contiguity, but with actual residual lymphoid tissue in the fossae of Rosenmuller, within the tubal orifice or on the torus or the salpingopharyngeal or salpingopalatine bands. This is the lymphoid tissue that poses the problem.

Now it has been conceded that lymphoid tissue everywhere in the organism is structurally and functionally the same. Therefore we can assume that what is true of lymphoid tissue, *per se*, is true of the lymphoid tissue of Waldeyer's ring.

#### HISTOLOGY

What is lymphoid tissue? Drinker and Yoffey<sup>6</sup> give the most concise definition of it as a mass of free cells, the vast majority of them lymphocytes of various sizes, together with a supporting framework of reticulum cells and of fibrous, elastic and sometimes muscular elements. Aschoff<sup>1</sup> differentiated between the tissue of the lymph nodes, which has afferent and efferent lymphatic vessels; the tissue in the mucous membranes, which has only efferent vessels; and the

lymphoid tissue of the spleen which has neither. We know, of course, that even within Waldeyer's ring itself we find tissue with and without glands opening into the crypts.

In the course of our investigation, we had to determine whether the masses we saw in the nasopharynx were actually hypertrophied lymphoid tissue, or merely edema resulting from infection or allergy. Therefore, at operation, the tissues were carefully removed from specific areas, such as the tori, fossae of Rosenmüller, the salpingopharyngeal and salpingopalatine bands, and studied histologically.

Sixty-six patients were chosen who were being operated on for ear symptoms, with bouts of deafness, associated with acute suppurative or acute secretory otitis. All of them when first examined had measurable hearing loss, but hearing improved following inflation of the middle ear in most of them, indicating reversibility of the loss. Primary tonsillectomy and adenoidectomy was carried out in 41; primary adenoidectomy alone in 2; secondary tonsillectomy and adenoidectomy in 1; and secondary adenoidectomy in 22.

There were removed 549 separate pieces of tissue, varying in size from 3-4 mm to half a centimeter. Well-defined secondary nodules were found in 437 of the pieces; 112 pieces proved not to be lymphoid tissue. From 55 of the patients, the tissue was removed from the torus; in 7, from the lateral band; and in the remaining, from the fossae of Rosenmüller. Secondary nodules were found in 79.6 per cent of the tissue; it is quite possible that if serial sections had been made, the percentage might well have been even higher. These cases represent true lymphoid tissue hypertrophy, and it was intensely active tissue, as judged by the great number of secondary nodules.

Losanow<sup>11</sup> states that lymphoid tissue is present in the embryo. According to Drinker and Yoffey, it can be found in the human throughout life. Scammon<sup>13</sup> found that no other body tissue, except the neural, develops so rapidly in the period from the first few months of life to the sixth year. The maximum peak is reached about the twelfth year, according to Scammon; then it begins to decrease, at first markedly, then more gradually until a more or less stable level is reached. Nevertheless, Kelemen<sup>10</sup> found well-developed, hyperplastic tonsillar tissue as late as the ninth decade of life. This "age involution" is to be distinguished from what Dougherty<sup>3</sup> calls "acute





Fig. 1.—Photomicrograph. One lobule, tightly packed with follicles.  
x 20 Heidenhain.

involution,” which occurs abruptly, regardless of age, in response to a number of physiological and pathological stimuli.

#### PHYSIOLOGY

What is the function of lymphoid tissue? It was largely on the basis of clinical observations that lymphoid tissue was accorded an important role in the body's defense reactions. Virchow<sup>17</sup> in 1860 was the first to formulate the concept that the lymph nodes act as barriers against infection, a concept that remained unchanged for the better part of a century. And once the concept was accepted for lymph nodes, it was extended to the other lymphoid tissues, the tonsils and other nasopharyngeal aggregates coming to be looked upon, in Drinker and Yoffey's words, as “somehow guarding the gateway to the alimentary canal.”

In more recent years, the defense theory has been elaborated to include: (1) the concept of lymphoid tissues as reaction centers where undesirable material is filtered out of the circulation, and (2) the postulate that antibody formation takes place in the lymphocyte.

The precise site of antibody formation has been debated vigorously. Recent studies favor the lymphocyte rather than the reticulo-endothelial cell, as Metschnikoff's followers believed.

Skoog's<sup>16</sup> experiments convinced him that under experimental conditions antibodies can be produced in the tonsil, but not to a degree comparable with that possible in other organs of the reticulo-endothelial systems. He concluded that the physiologic function of the tonsils was not to immunize the organism in the ordinary sense but to stimulate the reticulo-endothelial system to produce antibodies.

More recently, Ehrich and Harris,<sup>7</sup> extending the work done earlier by proponents of Bunting's theory that the lymphocyte was the site of antibody formation, satisfied themselves that cellular response within the lymph nodes during antibody formation is chiefly lymphocytic. These writers say that, if their reasoning is correct, the lymphocyte goes into action only after bacteria and other formed antigens have been properly prepared by the action of micro- and macrophages. It may be that antibodies are formed by the cooperation of lymphocytes with polymorphonuclear leucocytes and macrophages. This concept, they reason, would make clear why reticulo-endothelial blockage may or may not interfere with antibody formation.

Doughterty, Chase and White<sup>4</sup> concluded from certain of their experiments that antibodies are concentrated in the lymphocyte. And in 1952, Tore Wesslén,<sup>18</sup> in experiments undertaken to determine if the lymphocyte was a carrier or a producer of antibodies, concluded that lymphocytes in the form in which they enter the blood stream do not contain any antibodies but are capable of producing them.

Now, what of the function of nasopharyngeal lymphoid tissue? The literature on this score is limited. According to Forster,<sup>8</sup> the faucial tonsil is, in conjunction with other lymphoid tissue of the pharynx, considered to be a protective barrier against microbic invasion. He reminds us that Hellman observed reaction to invasion in the secondary follicles, and that Waldapfel and Grossman confirmed that the tonsil has a protective function. Yoffey believed that the lymphocytes are very likely concerned with the active growth processes of childhood, the lymphoid tissue acting as a storehouse of primitive wandering mesenchyme to supply various tissues as needed.

Wright<sup>20</sup> also reviewed the opinion on the tonsil's function, and attempted to conciliate the evidence for the barrier concept with

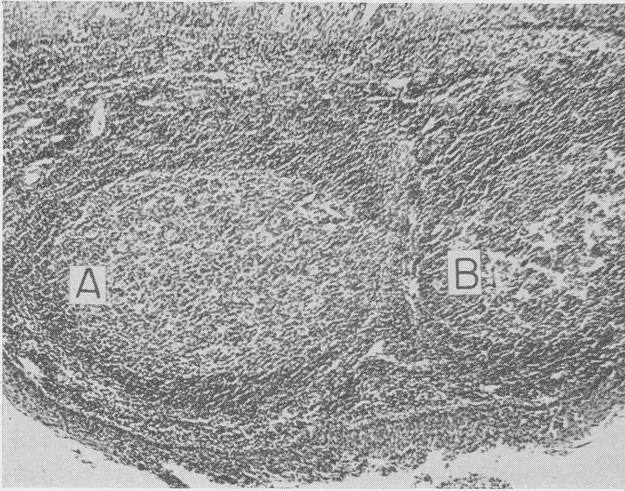


Fig. 2.—Photomicrograph. Lobule containing only two follicles—one dense, one loose; edema? x 60 HE.

the clinical observation that the tonsil often becomes a focus of infection and has to be removed. He suggests that an evolutionary process takes place. The nasopharyngeal lymphoid tissue, originally adequate as a defense against microbic invasion, proves unequal to the assaults that follow in the wake of civilization, which exposes man to all the dangers of crowded urban life.

But Wright himself reminds us of Harry Barnes' warning against "flights of fancy in theorizing on the function of lymphoid tissue," and we might do better to get down to facts.

#### ANATOMY

The most significant characteristic of the nasopharynx as it effects the lymphoid tissue is the great variation in size and conformation. Dickson and McGibbon<sup>2</sup> cite the work of Escat (1901) and of Moure and Lafarelle (1901), who described the many skeletal and other variations of the nasopharyngeal cavity. Escat pointed out that in the dolichocephalic skull, the nasopharynx is narrow transversely, with a long antero-posterior axis; in the brachycephalic skull, the nasopharynx is wide transversely and shallow from front to back.



Adding to these earlier studies their own careful measurements, Dickson and McGibbons concluded that "there is no 'constant' shape and no 'constant' measurement for all pharynges."

One very important factor determining the size and shape of the nasopharynx is the highly variable size of the torus and of the degree to which it projects into the nasopharynx. I was extremely skeptical when I first saw Kostanetsky's pictures showing variations of the tube; I now humbly concede the accuracy of his observations.

Indeed, I have observed the tori to be so hypertrophied as to close off the posterior choanae, completely obstructing the view of the rest of the postnasal space.

The fossae of Rosenmuller also vary widely in conformation. The entry may be a mere slit, measuring only a few millimeters, so that we see the fossa itself only when the torus is retracted laterally; or it may be so deep that one cannot view it in its entirety even on retracting the torus. On the other hand, the fossa may be so shallow that all of it can be seen easily merely by retracting the soft palate.

I have no doubt that the great variation to be found in the local anatomy is what accounts for the indifferent results achieved in the ordinary or blind adenoidectomy.

#### PATHOLOGY

To understand the impact of infection and allergy on nasopharyngeal lymphoid tissue, one must delve a little deeper and investigate capillary permeability. Capillary permeability remains for the most part a mystery. But enough is known about it to clarify what happens when infection or allergy affects lymphoid tissue.

We know that the return of cellular fluids to the circulation takes place either directly into the blood or through the lymph capillaries. According to Dougherty,<sup>3</sup> most of the fluid and the relatively simple compounds of low molecular weight are directly absorbed into the circulation through the blood capillaries. Particles of larger molecular weight, mostly the blood proteins, have to go through the lymph capillaries. When infection is present, not only is capillary permeability increased, but the products of infection bring about stasis, interfering with both blood and lymph flow. With allergy, edema results from the local action of the histamine or histamine-like substance on the tissues.



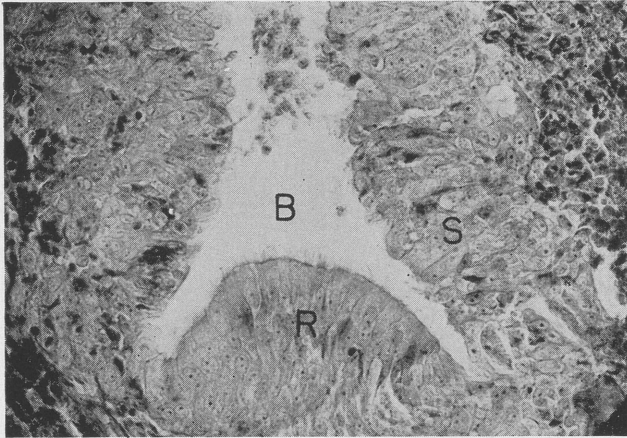


Fig. 3.—Photomicrograph. Respiratory epithelium at bottom of crypt; (B) crypt bottom, (R) respiratory epithelium, (S) stratified epithelium of crypt wall. x 460 Heidenhain.

Drinker's<sup>5</sup> experiments, which have an extremely significant implication for us here, showed that the nasopharyngeal lymphoid tissue need not be overwhelmed by an acute infection to develop edema and hyperplasia. These effects could be marked even when only a few bacteria and viruses of low virulence are present in the mucosa capable of surviving and multiplying. What this means is that the torus and tissues of the fossae of Rosenmuller invaded by these agents become a shock area. There is basis for this hypothesis in Rowe's<sup>12</sup> statement that "... though sensitization of all cells of the body may occur, allergy usually evidences itself in vascular, connective or lymphoid tissue." It could very well be that the tissues harboring these noxious agents become sensitized, become the shock tissues in the sense in which Selye<sup>15</sup> uses the term in his concept of the alarm reaction and the general adaptation syndrome.

How the picture produced by infection can be positively differentiated from what we see in allergy, I do not know. But a clue is to be found in the duration of symptoms, and in their response to treatment. If there is infection, the reaction to it in lymphoid tissues will clear up as the infection subsides. We must remember, however, that the hypertrophied lymph nodules will remain. In an

allergic reaction, the edema will persist much longer; when it does disappear, the remission will be brief, and it will recur.

Allergists, and some otologists, believe that underlying allergy accounts for all alterations in the lymphoid tissue of Waldeyer's ring. Schenck<sup>14</sup> believes this conclusion to be fallacious. Certainly, one cannot blame all lymphoid alteration here on allergy. But I cannot but conclude, after seeing so much recurrence even after the most thorough surgery, that allergy plays a cardinal role. This belief is strongly supported by the number of cases in which improvement is seen following allergic management in these patients.

#### ENDOCRINE FACTOR

I have said that the endocrine aspect of this problem is still to be explored with the help of the endocrinologists. We have learned enough about it, however, to conclude that this is perhaps the most important avenue of research in the study of lymphoid hyperplasia. The work now going forward in exploring the relationships between lymphoid tissue growth and involution and the endocrine glands—the pituitary, adrenal, thyroid—promises to provide us with the key to effective treatment.

It has been shown that adrenocortical secretion brings about lymphoid tissue involution. Some of the involuting effect of irradiation appears to be endocrine-mediated; so, too, the involuting effect of inanition. White believes that all the factors inducing involution—including nutrition, chemical agents like nitrogen mustard, physical factors like heat, cold and atmospheric pressure—may be mediated by the genetic factor.

I do not propose to go into the many ramifications of this highly complex problem here. But I refer to this particular aspect of the subject merely to indicate its complexities and obscurities.

I would like to say one thing more, however. It has already been demonstrated that in allergic rhinitis, startlingly successful results—though too often temporary—can be achieved with ACTH and cortisone. It stands to reason that with further purification of these substances, and with accumulating clinical observation of the results achieved with them, ACTH and cortisone may be successfully applied to nasopharyngeal tissue hyperplasia in the future.

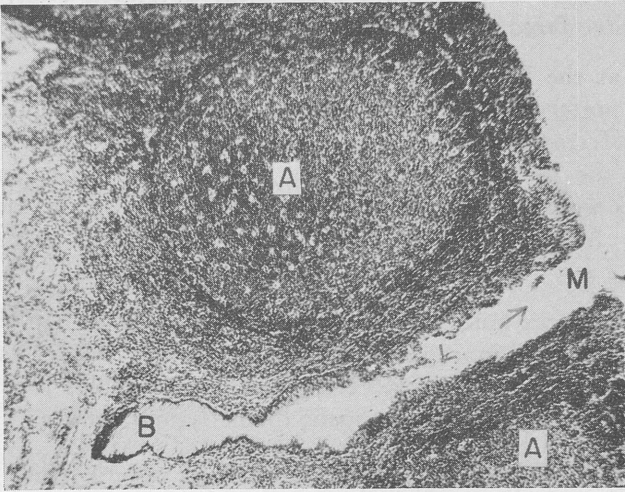


Fig. 4.—Photomicrograph. Opening of gland into crypt bottom flushing crypt content against mouth of crypt. (A-A) follicles, (B) bottom of crypt, (M) mouth of crypt. Arrow shows direction of current of glandular secretion flushing the crypt. x 60 HE.

#### IRRADIATION

The treatment of lymphoid tissue hyperplasia by irradiation goes back to Heineke's discovery in 1903 that roentgen irradiation causes lymphoid tissue to involute. Since then, we have seen the early enthusiasm for reducing the tonsil by x-ray treatment wane, and a new enthusiasm take its place—the use of radium to reduce lymphoid hyperplasia in the nasopharynx. The history of irradiation treatment of the nasopharyngeal lymphoid tissue goes into more than two thousands titles in the literature. It can be summarized in a few sentences.

We know now that the attempt to reduce the tonsil by x-ray failed because irradiation is not effective for encapsulated lymphoid tissue like the tonsil, but, rather for the scattered lymphoid tissue that eludes even the most thorough surgery. Radium treatment for this purpose is still being argued. You are all familiar with the early reports of its results. On the debit side of the ledger is an increas-

ing documentation of the opinion that radium yields less impressive results. Where controls have been used, untreated cases, it is reported, have fared as well, ultimately, as those treated with radium.

We at the Massachusetts Eye and Ear Infirmary, from the beginning, preferred the greater accuracy and safety of x-ray. I have already referred to the Dickson-McGibbon study, in which measurement of the nasopharyngeal cavity made it clear that anatomical variations were too great for accurate positioning of the radium applicator. At the Winthrop Foundation we had come to a like conclusion.

Not that we hold any brief for x-ray treatment. In the course of the twenty and more years of my experience with it, I have never seen a lymphoid mass resolve following its use. This is in keeping with what we know, now, happens to the lymph follicle.

The immediate effect of irradiation is on the secondary nodules, and manifests itself in these three stages: first, cessation of proliferative activity; second, conspicuous scavenger activity during which the destroyed cells are removed; and third, depending on the dosage used, the return of proliferative power. It must return, unless treatment has gone beyond the limits safe for non-neoplastic tissue. Naturally, the greater the dosage, the quicker the ensuing destructive process. But eventually regeneration does occur. Schenck's studies have demonstrated that irradiation by any means—x-ray, radium, ultra-violet rays—produces the same effects.

This is why I believe that irradiation can never replace surgery as a means of removing scattered lymphoid tissue. Not that surgery is by any means the perfect solution. Indeed we know that in some children, operation seems only to increase the tendency of hyperplasia to recur. Then there is the possibility that when it does recur, it will be in an area even less desirable—the lateral bands, the tori or the fossae of Rosenmuller.

But we still operate because we know that in many instances surgery will permanently exhaust the regenerative properties of lymphoid tissue. And quite often this is enough to relieve tubal obstruction. True, the same claim is made for irradiation, and for allergic management. Perhaps the answer is to use all three measures.

Surgery has the advantage over irradiation in that it can actually remove the mass of lymphoid tissue. What irradiation apparently



does is to inactivate the lymphocytes and, as Schenck suggests, to reduce the edema by increasing lymph flow. This result is, only ephemeral, but if normal tubal function can be restored, even temporarily, we are that much ahead.

Our management of lymphoid regrowth resulting in tubal closure depends on the patient's history and what we find at operation under direct vision. If hearing has been impaired as a result of tubal closure following upper respiratory infection but has returned to normal after one or two inflations, we remove the obstructing tissue. No follow-up management is given. Should the tubal occlusion recur after an upper respiratory infection, and nasopharyngeal examination show the vault to be free of lymph nodules, we advise irradiation: 600 r in three treatments; 100 to each side through an intra-oral cone or over the lateral neck.

If, on the other hand, a patient has a history of recurring tubal closure without upper respiratory infection, and we find lymphoid tissue in the areas described, we first remove the tissue thoroughly by surgery and administer x-ray treatment within two weeks of the operation. If this fails to bring about the desired result, we advise allergic studies, regardless of whether the patient shows clinical evidence of allergy or not.

Where the patient is definitely known to have a history of allergy but has not responded to allergic therapy, we remove the lymphoid tissue surgically. If there has been no treatment for allergy, such treatment should be the first step. If the patient does not improve, we resort to surgery. Whether or not irradiation is given depends on his postoperative course.

#### CONCLUSION

What has been said so far adds up to few conclusions. We know this much: When a mass of nasopharyngeal lymphoid tissue interferes with nasal or middle ear function by imposing an obstruction, its removal almost always restores normal function. When tubal obstruction is the result of hypertrophy and edema of the lymphoid tissue on the tori, the fossae of Rosenmuller or the lateral bands extending from the torus to the palate and lateral pharynx, the obstruction can best be coped with by surgical removal of the excess tissue, under direct vision. Not infrequently further treatment such as sup-

plementary irradiation may be necessary to control postoperative recurrence of tubal closure.

Grateful acknowledgment is made of the histological interpretations made by Dr. George Kelemen and the translations and other bibliographic assistance given by him, and to Miss Dorothy Linden, technician at the Massachusetts Eye and Ear Infirmary.

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## DISCUSSION

DR. FRANCIS W. DAVISON: Dr. Harkness, Fellows and Guests, Dr. Meltzer has given us an excellent description of the anatomy and physiology of lymphoid tissue in the nasopharynx.

I should like to mention briefly a few bacteriologic facts recently determined by our Department of Bacteriology. The method of study developed by Professor John Rice of Bucknell University was as follows: the excised adenoid tissue was washed and chewed up with saline in a modified Waring blender known as a disintegrator. Serial dilutions were made and cultured on blood agar plates; thus determining accurately the bacteriologic count per gram of adenoid tissue. Some of the colony counts were extremely high, as high as 14 million beta hemolytic streptococci per gram of adenoid. Some of the patients who were treated before operation with large doses of penicillin by intramuscular injection had extremely low colony counts, thus proving that penicillin can greatly reduce or eliminate, at least temporarily, infection in the adenoid.

MR. TERENCE CAWTHORNE: Mr. President, Members of the American Laryngological Association and Fellow Guests, I would like to express my thanks and pleasure to my friend, Dr. Philip Meltzer, for giving such an interesting, informative and elegant review of what is I think to all of us a very difficult subject indeed.

In England recently the Medical Research Council has been conducting an investigation into the relative merits of local and distant irradiation in cases such as have been described this morning. We started off four years ago expecting to get a very large number of children for the investigation but we found that there were not the number of cases that some of the literature had led us to believe. I know that in my own practice the number of children who after adequate surgical treatment or any other form of treatment require this irradiation is very small. We have toyed with the idea of using some of these new powerful hormones such as cortisone in this condition but I for one have felt rather chary of trying to exchange perhaps a few little nodules of lymphoid tissue for a luxuriant mustache. Nevertheless, in very careful hands and very careful doses it may give us some help. I do think, however, that such papers as Dr. Meltzer's of this morning will help us all to clarify our thoughts in this very difficult subject.

Thank you!

DR. THOMAS C. GALLOWAY: Mr. Chairman and Fellows. This is a very timely paper. It is important to know the train of thought which is going through the pediatrician's mind, based on two conceptions: We have a leading pediatrician at our hospital who no longer has any surgery done on tonsils. He has been persuaded by well-controlled study, by E. M. Uhlman and S. J. Pearlman, a laryngologist at Michael Reese Hospital, of 480 cases followed for three years. Their results seem to be very well reasoned and carry considerable conviction that irradiation has given results comparable to surgery.

In addition three papers which have recently been published make the assertion that tonsillectomy predisposes to poliomyelitis at any time. I have studied that subject intensively and I hold to the conclusion of my paper published in a symposium of the A. M. A., that it is important that tonsillectomy be not done in the presence of the virus of poliomyelitis because it does greatly predispose to the severe form if the disease occurs within one month. But I think these other papers are fallacious in that they deal with hospital populations. However, these statistical studies do raise some question. The facts that Dr. Meltzer has brought for-

ward must be studied. I think he has proved the case for surgery in most instances except in those patients where scattered lymphoid follicles persist after good surgery and especially if we agree that such lymphoid masses may exist within the tube itself.

DR. HENRY L. WILLIAMS: Mr. President and Members of the Society and Guests, I have been extremely interested not only in this paper of Dr. Meltzer but in his previous papers on this subject. I think Dr. Meltzer has brought our attention to a subject which has been grievously neglected. When one considers the history of the operations for removal of lymphoid tissue from the nasopharynx it is striking that the original paper in English, written in 1864 by Wilhelm Meyer, practically assumes the same position that modern advocates of complete adenoidectomy have assumed, that is, that the important structures to be removed are the adenoid vegetations in the nasopharynx, and that the tonsil is of secondary importance. It is most interesting to realize that the anatomical descriptions which Dr. Meltzer mentioned were written I believe in 1862 and that Dr. Meyer's paper came in 1870.

On reading Dr. Meyer's paper I was impressed by the fact that he operated without the advantage of anesthesia. Dr. Meyer removed these adenoid vegetations without anesthesia, having developed a certain resistance in the patient by manipulation of the nasopharynx by finger massage for a few weeks.

Another interesting fact, I think, is that he did these patients in the upright position with the head somewhat forward. In this instance it was for the purpose of allowing blood and secretions to run out of the nasopharynx.

The weight of the dead hand of tradition is demonstrated by the fact that in some areas in this country, in spite of the development of general anesthesia, this type of positioning is still used. It certainly is traditional but I do not think that it is the best position to do an operation of this sort.

DR. WALTER H. THEOBALD: Mr. President, Fellows and Guests, I will be brief because this discussion is getting a little lengthy. Nevertheless this presentation is enlightening because we are again being alerted to the importance of evaluating the nasopharynx. It has been termed the unknown or the neglected field but these titles can be qualified by emphasizing four points: first not seeing the epipharynx; second, not knowing what to look for; third, not evaluating and interpreting correctly what is seen in the epipharynx; and fourth, not looking at all.

I like the term that Dr. Meltzer used when he said pinpoint, for we must pinpoint our examination, our diagnosis just as thoroughly as he pinpoints his operative procedures. I think in that connection for diagnostic purposes and examination that the Yankauer speculum is the choice instrument to get a direct look into the furrows and into the crypts and folds of this lymphoid tissue development and particularly when it involves the lateral folds.

Now if you will permit me I will apologize to my colleagues who may have seen this slide. It may be new to some others. I want to point out one particular thing, however.

(Slide) Because Dr. Meltzer has referred his pathology particularly in connection with the eustachian orifice, this represents a survey, a careful study made of 150 cases, all of which had epipharyngeal pathology. None of this pathology came from the accessory sinuses, from allergies or any other source but primarily from the epipharynx. In these 150 cases we will omit those things at the top,



the post-nasal cough, etc., but I wanted to point out, earaches in which there were 11 cases. Eight cases had fever and recurrent sore throats. In the 150 cases here we had 11 in which the pathology in the ear was directly responsible to the epipharynx.

Thank you, Mr. President!

DR. ROBERT E. PRIEST: I will make this very brief. I thought you might be interested in an experience that I had with a patient who had had irradiation of the tonsils and adenoids. A young man, who was a full time teacher of pediatrics in our medical school and now occupies a similar position in another medical school, brought one of his children to me saying that the boy was a behavior problem and they had recently realized that he did not hear well. He had bilateral serous otitis media and the father said he had extensive radiation for removal of tonsils and adenoids. The Pediatrics Department at our school is a little bit reluctant to accept the concept that the surgical removal of adenoids is necessary. The boy's tonsils were pretty well gone. He was about seven years old and he let me look at his nasopharynx with a mirror. The adenoid was moderately shrunken but a bar remained which went from one tube orifice to the other. That was all that was left. It was beautifully placed. If you had designed the bar to put up in there to occlude the two orifices you could not have done better. I did a removal of the tonsils and adenoids. They boy's hearing came up to normal within a few days and his behavior problem ceased completely and immediately.

We cannot generalize from one case but this experience was interesting to me because we don't have a chance to see too many patients who have had irradiation with the idea of eliminating tonsils and adenoids as an alternative to surgery.

DR. PHILIP E. MELTZER: I know the time is late, therefore I will briefly answer what I think is pertinent.

Dr. Davidson, your technique of determining the degree of infection in what appears to be noninfected adenoid is an excellent one. The important point brought out by Drinker was the reaction in lymphoid tissue that produces edema with closure of the tube and does not require the presence of many organisms. In fact a minimal infection producing a subclinical picture can sensitize the lymphoid tissue.

Mr. Cawthorne, I appreciate very much your remarks and I agree that the number of children involved is limited but they pose a very important problem to the Otologist, the educator and particularly to the parents, and while many of these tubal closures resolve spontaneously there are many that do not. They require an intensive treatment and follow up. Naturally at an Institution like the Massachusetts Eye and Ear Infirmary which draws from a large territory and wide population, we see a greater number of children so involved. Furthermore, having brought this matter to the attention of the pediatricians, I see more of these patients than the average otologist.

Dr. Galloway, it may be that ultimately children who were treated by irradiation and those who were not will show the same picture later in life, and it may well be that the same end result will occur in those who have had secondary adenoidectomies. But during the period where infections are common in children, between three and eight years of age, it is important that middle ear symptoms should not be neglected.

Dr. Williams, as I mentioned in this paper, the success of adenoidectomy in nasal obstruction was due in most part to removal of the mass. As regards the

operative position, I cannot deny that the upright position is more dangerous as regards complications than is the supine position. Dr. Mosher determined that fact for us at the Massachusetts Eye and Ear Infirmary and for many years all tonsillectomies and adenoidectomies were performed in the supine position. However, those of us who have been trained to do this operation in the sitting position are fully cognizant of the danger and take all precaution to prevent blood from flowing into the larynx. With the improved technique of anesthesia, an intratracheal tube can be inserted and there is no problem at all as far as I can see. For the inexperienced surgeon the sitting up position is something he will have to learn, and I repeat that the best view of the torus and the fossa of Rosenmueller as well as the entire nasopharynx can be obtained in the sitting position.

Dr. Theobald is so right about the necessity of examining the postnasal space. The important point is that when the palate is retracted you should be able to see the structures in relationship to one another. You want to see the torus of the tube in its actual relation to the Fossa of Rosenmuller and lateral pharyngeal wall. This cannot be done thru a tubular retractor such as the Yankauer. I prefer the Lathrop retractor. It was originally designed for the tonsillar pillars but it is an excellent instrument for elevation of the soft palate and the posterior pillar. It is somewhat like the Love retractor but preceded it by some years.

I wish to thank the discussors for their kind remarks, and the President and Members of the Society for the privilege of appearing before them.

## SOME PRACTICAL CONSIDERATIONS CONCERNING RESPIRATORY ALLERGY

RICHARD A. KERN, M.D.

PHILADELPHIA, PA.

The 75 years which mark the span of the American Laryngological Association have been the most significant in the whole history of medicine. They have not only witnessed the breath-taking advances in the basic sciences and in the general knowledge of the causes and management of disease, but they have seen the growth and development of this Association's specialty almost from its beginnings to its present high estate. Bacteriology brought asepsis, and anesthesiology made possible new surgical techniques. More recently, antibiotics and bacteriostatic agents made unnecessary many operations that once took most of one's time, but, as Lempert<sup>13</sup> has recently emphasized, they also made possible an effective operative approach in new fields, such as modern labyrinthine surgery.

The word "allergy"<sup>16</sup> was coined within the lifetime of most of the members of this Association. The development of that subject has been known to, and shared in, by many of you, and you all know how great has been its impact upon the practice of your specialty, since the respiratory tract is the site of the commonest, most diversified and some of the severest manifestations of hypersensitivity. The fact that my interest has centered largely on allergy in the respiratory tract has perhaps been responsible for the invitation to appear on this program. I realize keenly how presumptuous it is for me to discuss a topic with whose practical aspects this group is so thoroughly familiar. I can only hope that from my experiences during 40 years in the field of respiratory allergy I have gleaned a few points that may be worthy of your notice.

We are most likely to fall into scientific error when for the moment we lose sight of basic principles as we follow some current enthusiasm in a very proper search for progress. If I may borrow in a limited sense the much broader philosophy in one of Kipling's

poems, we are inclined to heed the glittering promises of the gods of the market-place, rather than the simple truths of the gods of the copybook headings. Yet Kipling<sup>12</sup> makes his point in the opening stanza:

"As I pass through my incarnations in every age and race,  
I make my proper prostrations to the Gods of the Market-place.  
Peering through reverent fingers I watch them flourish and fall,  
And the Gods of the Copybook Headings, I notice, outlast them all."

And so I would like to stress certain principles the slighting of which has been responsible for our commoner mistakes.

The importance of allergy in all phases of clinical medicine needs little emphasis before this audience. Fifteen per cent of the population at some time in their lives will manifest a major form of allergy, such as seasonal and perennial allergic rhinitis, asthma and eczema, and another 25 per cent will show lesser evidences of hypersensitivity. The proportions are even higher in the practice of otorhinolaryngology.

We must be reminded, however, that the allergic patient is a special kind of individual, with an inborn, ineradicable defect. What he has inherited is not a particular disease, such as hay fever, nor a specific sensitivity, such as that to ragweed pollen, but a much greater-than-normal ability to become sensitive to things in his environment; he is born that way and he so remains until the day he dies, and all the medical experiences of his lifetime may be influenced and modified by that fact.

On the other hand, the specific sensitivity to this or that substance may be of limited duration, lasting for months or a few years. The pattern of a patient's sensitivities is therefore not a fixed and constant thing. New sensitivities come and old ones go, so that the same clinical complaint may have different allergenic causes at different times in the patient's life. This is of great importance in solving the problem of recurrences of a complaint after long periods of relief.

Moreover, the part of the body that reacts to the allergen, the shock organ, may change, with or without a change in sensitivities. So a patient may have eczema in infancy from wheat or house dust, nasal symptoms in adolescence, and transient hoarseness, or paroxysmal nonproductive cough, or asthma (depending on whether larynx,



trachea or bronchial tree is the shock organ) in middle years from the same or other causes, and eczema again in old age from yet newer sensitivities. Only one thing does not change: the easy sensitizability of the allergic individual.

It is important to consider how this individual acquires new sensitivities, because this knowledge can make it easier to discover offending allergens, and what is more important, can help the physician to prevent sensitization in the first place. These factors are undoubtedly at work: 1) overexposure of a body surface (skin or mucosa) to allergens; 2) injury to a body surface, whether by chemical irritation, infection or mechanical trauma, rendering that surface more permeable to, and therefore sensitizable by, those allergens which happen to be present. This accounts for the frequent development of respiratory allergy in the course of such severe and prolonged infection of respiratory mucosa as occurs in measles, influenza, whooping cough and various viral pneumonias. Moreover the offending allergens are more likely to be those to which the patient is increasingly exposed during such an illness, namely the things in his bedroom: feathers, wool, house dust.

This principle justifies the restriction to a minimum of exposure to potent allergens in the case of allergic children and also the children of allergic parents. This is particularly true and practical in relation to inhalant allergens, because, to a considerable degree, we can control our environment. In the bedroom, this means rubber-foam pillow and mattress, old hard-weave cotton blankets covered with cotton sheeting, bare floor, with at most a washable cotton rag rug at the bedside, smooth painted walls. It also means no feathers or animal hair in bedding, no upholstery, no unnecessary hangings in the bedroom. Nor should that room be contaminated by the introduction of dust from the rest of the house by a hot-air heating system or by the use of insecticidal sprays. This principle can be extended to include the selection of toys and pets in childhood, cosmetics in adolescence, and vocations in adult years. I have discussed this subject extensively elsewhere.<sup>5</sup>

In 1926,<sup>4</sup> I first called attention to the fact that hay fever often begins in the year in which an operation of election was performed on an allergic individual during a pollen season, and I then urged that such operations in the allergic subject be carried out at least a month before pollen appears in the spring, or be deferred until after frost in the fall. While this suggestion has received fairly gen-

eral acceptance, there are still many unpremeditated exceptions, especially in children, whose tonsils are so frequently removed just after school closes in June, when the grass pollen season is at its height, or else just after they return from summer camp on August 31st and before school opens, when ragweed pollen counts are at the peak.

The next principle is introduced with a modicum of malice aforethought. It applies not only to allergy but to infections in the respiratory tract. I constantly stress it in my teaching of medical students, internes and residents, and I bring it to the attention of physicians whenever opportunity offers, even of those who specialize in disorders of that system, albeit some above, some below the larynx. The whole respiratory tract must be considered as a unit. Disease in one part, whether allergic or infectious or both, is commonly associated with involvement of another part. As you well know, the one may be symptomless, with the result that the other gets all or most of the attention. Yet, regardless of which came first, treatment of the silent lesion must accompany that of the obvious one in order to achieve a lasting result.

The complexity of allergenic etiology is nowhere more marked than in the respiratory forms of hypersensitivity. In the skin, allergic disorders are preponderantly based on contacted substances; in the digestive tract, on substances swallowed. But in the respiratory tract, one must reckon in the same case not only with inhalants, but with things that are swallowed, and also with bacteria. This is true even in many patients with purely seasonal symptoms, due primarily to pollens, but often aggravated by sensitivity to substances that are perennially present. It must be emphasized that this calls for a more extensive allergic survey in many cases of respiratory sensitivity than is now made. Thus, the patient with strictly seasonal symptoms should be tested not only for the pollens that occur in that season, but also for a) the pollens of known significance, in other seasons, in that area, b) the commoner mold spores there prevalent, and c) those animal epidermal substances, cosmetic ingredients, house dust and similar inhaled allergens to which he is perennially exposed in his particular environment. The patient with perennial symptoms of respiratory allergy must be checked routinely also for sensitivity to foods as well as inhalant sensitivities.

The number of potential inhalant allergens is increasing at amazing speed. In modern industry such varied contacts are exceedingly

common, and the products of industry can also contribute to the allergic hazards in the home. It has long since become inadequate to define a patient's occupation in a word or two. A detailed record of his working day may have to be supplemented by a visit to his place of employment to uncover the causes of his trouble. Indeed, it is in this diagnostic phase of our problem that the allergist serves his greatest purpose: skin tests can be done by any technician; it is the allergist's knowledge, based on training and experience, that leads to the proper selection of things to be tested and to a sound interpretation of the results obtained.

In 1921<sup>3</sup> I called attention to the role of house dust in the etiology of asthma. Today it ranks as one of the most important factors in allergic conditions of the respiratory tract. In spite of much intensive study by many investigators over the years, we still have no final answer as to the nature of the allergen or allergens in house dust. Occasionally it is possible to identify a specific component of a given house dust, such as a certain animal epidermal substance, as the thing to which the patient is sensitive. But as a rule this is not true.

The fact that an extract of some house dusts will give positive skin tests in a considerable number of patients with respiratory allergy has led to the view that such dusts must contain an allergen common to them all. It has further led to the view that several dusts from different homes together contain most or all of a few important allergens that any house dust might contain. Above all, it has led to the practice, by many allergists, of testing patients with only the extract of a pooled lot of dust collected, say, from the 24 units of an apartment house, and then of treating with such a so-called stock dust extract those patients who give a positive skin test to it. This, I contend, is fallacious, for the following reasons:

What is house dust? What is hash? Whatever is available to go into it. The dusts of houses are almost as individualistic as the people who live in them. To be sure, some ingredients in the way of furniture and upholstery materials may be common to many houses, and if they are good antigens, can be responsible for many common sensitivities. I once tested with the extract of a house dust that was the cause of a certain patient's asthma a series of 125 other asthmatics. No less than 26 of them reacted positively. But some ingredients (a rug of unusual origin, a mold that grows only in few

cellars, a rare varnish) may occur in only few houses. Thus, I also tested one patient's offending dust in 44 other asthmatics without getting a reaction in any one.

If a stock dust extract from an apartment house produces a positive test in a patient living in Unit Number 6, this could be due to something in the dust of Unit Number 24, but to which the patient in Number 6 is not exposed. To treat him with this stock extract would be an illogical treatment of a falsely positive laboratory test. Or it could happen that a false negative would result in a patient, actually sensitive to the dust of his own home, but not reacting to the extract of a stock dust in which his own dust was too much diluted by the addition of dusts from 23 other dwellings.

Some nevertheless defend the stock dust procedure on the ground that it is more convenient. Now it may be inconvenient, but it is never impossible to get an adequate dust collection from any patient's environment. As for the logic of the convenience of the stock dust extract, I am reminded of the drunk who was seen one night groping about on his hands and knees at a street intersection under the arc light. A passer-by asked: "What are you looking for?" "I losht my watch." "Where did you lose it?" "Over there under the treesh." "Then why are you looking here?" "The light'sh better."

Little need be said about the role of pollens in the etiology of respiratory allergy. Current practice is generally adequate and proper. Patients are no longer tested or treated to any extent with insect-carried pollens that play little or no part in the production of their symptoms. Attention is however called to one important cause for error: the failure to reckon with pyrethrum sensitivity. Skin sensitivity to this substance is almost as common as that to ragweed, and often in the same individuals. Long used as an insecticide, pyrethrum is more frequently encountered than ever before, because of its inclusion in most insecticidal mixtures and sprays, notably the various DDT preparations for use in households, hotels, theatres and the like with heavily carpeted floors.

Mold sensitivity, on the other hand, needs more consideration than it now receives from many physicians, especially along the eastern seaboard. Not only are mold spores present in large concentration in outdoor air in mid-summer, particularly in July, but they can be a source of perennial trouble in individual houses. A damp cellar is the prolific source of such spores, and even more important is the



increasing number of hot-air heating plants which, alleged filter devices notwithstanding, see to their distribution to all parts of the house and in most seasons. Diagnosis and management of mold allergy are analogous to those of pollinosis.

Food sensitivity can play a part in any of the perennial manifestations of respiratory allergy. This fact is generally accepted with regard to asthma, but there are rather frequent oversights as to its role in nasal symptoms.

Bacteria are involved more frequently and more significantly in allergic conditions of the respiratory tract than in those of any other part of the body. In most patients with perennial symptoms and in many who have ragweed pollinosis, we must reckon with their presence as the agents of a complicating infection. The diagnosis and treatment of such infection in sinuses and bronchial tree must be based in part on the same principles as those which apply to any patients with such infections. But in addition they are modified by the important possibility of allergy to the bacteria. Bacterial allergy as encountered in these patients has some important characteristics that differentiate it from the sensitivities to other allergens. It is strain-specific, not species-specific. That is, the patient is sensitive, not to all pneumococci, but to the strain which he himself harbors. Moreover, that sensitivity may take two forms: the usual one is the delayed inflammatory type, comparable to the tuberculin reaction; in rare cases there may be an acute whealing type of reaction. But again this is true for the patient's own strain alone.

These facts have the following practical significance. It is not possible to test for bacterial sensitivity with a stock extract of a species of bacteria. It is not even rewarding to make tests with autogenous killed suspensions of organisms. But the frequent existence of bacterial allergy makes imperative a cautious use of autogenous respiratory vaccines in all patients with respiratory allergy. A rare patient may die in anaphylactic shock from the first dose (I know it to have happened within 5 minutes of the supposed subcutaneous injection of 10,000,000 organisms). A few are precipitated into status asthmaticus with a similar dose. The commonest regrettable event is that a patient is given a few doses of an autogenous vaccine in increasing amounts and he proceeds to get definitely worse after each injection. Both physician and patient then decide that the cure is worse than the disease, and so the vaccine is discarded. Yet in that

vaccine they had the major cause of the patient's trouble and the very key to his successful treatment. What was wrong was the dose. One doesn't begin with large doses of pollen extract in treating hayfever, lest he produce a constitutional reaction; such autogenous vaccines should be given in an initial dose of never more than 5,000,000 organisms, at intervals of not less than a week, and in increments sufficiently small to guard against reactions.

Of particular interest to rhinologists are the structural changes that occur in the nose and sinuses in response to severe prolonged allergic reactions: hyperplastic rhinitis, and its end stage, mucous nasal polyps. Previously attributed to infection, their allergic nature was stressed by Schenck and me<sup>9</sup> in 1933. We were at that time impressed by the very great frequency of such polyps in patients obviously allergic and their absence in non-allergic patients even though they had long-standing extensive infection of various types in sinuses and bronchi. We have felt that bacterial allergy is the commonest factor involved, but that other allergens certainly can and usually do play a part. Indeed, an occasional case is seen in which bacterial allergy may be minimal or even absent, as in a patient whose polyps appear during a severe ragweed hayfever and disappear after frost removes the allergen. The presence of large numbers of eosinophiles in such polypoid and mucosal tissue is a function of the recent exposure to the allergen, not a differentiation from a non-allergic etiology, for as Schenck and I<sup>10</sup> have shown, polyps removed from the same patient a few days apart, first near the end of the ragweed season and again just after frost, showed many eosinophiles in the former and none in the latter. The continued action of allergic factors, we believe, is responsible for the recurrence of polyps after their surgical removal. The logical procedure to prevent such recurrence is therefore adequate attention to the allergic factors involved. This includes a recognition of all participating allergens and their avoidance when possible, or the control of their effects, should they be unavoidable. This, in turn, calls for desensitization with unavoidable inhalants, such as house dust, the drainage, *secundum artem*, of infection where that is necessary, and the use of an autogenous vaccine for bacterial desensitization. In the last connection, I have found it helpful to obtain cultures from polyps and hyperplastic mucosa by grinding the tissue in a mortar with sterile sand. It must be emphasized that this program is of vexing complexity and calls for meticulous care in execution to insure a successful result.

The concept of the allergic cause of polyps has received increasing confirmation and acceptance among rhinologists and allergists. Particularly significant is the effect of those most potent factors in controlling allergic reactions of all sorts, the cortico-steroids, upon polypoid tissue. I shall refer to this later in more detail. Only the pathologists in their textbooks still speak of infection as the only etiology of polyps, and do not even mention allergy—perhaps the penalty of too intent a study of dead rather than living tissue.

While bacteria are under consideration, I wish to refer to an erroneous concept that early gained a foothold and largely still persists in current thinking about the most serious of respiratory allergies, asthma. Chronic respiratory infection and therefore bacterial allergy are frequent in those with perennial respiratory allergic disease. Chronic infection in time causes the ciliated epithelium to be replaced with squamous cells. Especially in the aged do emphysema, mild bronchiectasis and poor circulation accentuate the burden of the disease upon the patient. As a result, asthma in the aged tends to be continuous and chronic, and infection more and more dominates the clinical picture: chronic cough, purulent sputum, low-grade fever, and increase of asthma after every acute cold.

Now it is a well-known fact in all types of clinical allergy that skin reactivity to allergens is greatest in childhood, lessens in adult years, and may often be lost in old age, even though the site of allergic disease, the shock organ, still reacts strongly upon exposure to the allergen. For example, feathers can still cause asthma in a patient whose skin no longer gives a positive test reaction. Therefore skin tests are less and less useful in the diagnostic study of any allergic disease in aging patients.

This poor skin reactivity in the aged, plus the dominance of the clinical picture of their asthma by infection, has been responsible for the erroneous concept that asthma can be classified as either extrinsic, that is, caused by allergens outside the body, or intrinsic, due to causes within the body, notably infection. Indeed, there is a common belief that asthma which begins late in life is practically always intrinsic, infectious and non-allergic. Thus Rackemann<sup>14</sup> has long claimed that extrinsic asthma seldom begins after the age of 45.

This is certainly not true in my experience. Sensitivity to external allergens, if properly and carefully sought for, can be found

in the majority of patients whose asthma begins even in advanced years, and should be suspected in the rest. A further common mistake is to consider the aged patient to be non-allergic because of failure to ask about other evidences of allergy in earlier years. Yet the patient whose asthma began at 70 may have had eczema in infancy, hayfever and hives in adolescence, and typical though mild perennial symptoms of nasal allergy, such as bouts of sneezing or a "wet" nose, for many years, often due to the very same allergens that now affect the bronchi as well as the nose. That infection plays an important role in the asthma of advanced years is freely admitted, but that it is alone responsible except in a small minority of patients is denied.

The role of psychic factors in the production of respiratory symptoms of the types seen in allergic diseases of that tract has had varying emphasis over the years. When this Association was founded, it was the generally accepted view that hayfever was purely a neurosis. Even today most people remember the case cited in the leading text of the late seventies by Beard<sup>1</sup> that told of the patient whose "hayfever" was induced by the sight of an artificial rose. No one paid much attention to Blackley<sup>2</sup> in England and Wyman<sup>17</sup> in this country who in 1873 and 1876 respectively gave convincing proof of grass pollen as the usual cause of hayfever in England and of ragweed in the American late summer variety of the disease. Fifty years later, every one knew that only pollens were responsible. But today, with the increasing recognition of the importance of the psyche as well as the soma in the etiology of all the ills of flesh, the question of the psychic factor in so-called allergic disorders, including those of the respiratory tract, is again being raised. The artificial-rose case has been resurrected, and there are some psychiatrists who are making sweeping claims for the psychic origin of a majority of the diseases under consideration. In presenting specific cases, they point to data on adverse life situations and symptoms, chronologically arranged in parallel columns, showing how the former were promptly followed by the latter. But they ignore such significant dates as the 15th of August when symptoms began; or that, when psychiatric treatment produced such brilliant relief, it had happened when the patient was admitted to a hospital, and so removed from his offending house dust. Even the relapse that came on the day after the patient went home is attributed to his return to an "intolerable situation," meaning his wife, rather than to her allergenic cosmetics.



I do not deny the possible importance of the psychic factors in the production of many and the aggravation of most symptoms of any disease, allergic or otherwise. But I do claim that in the field of allergic disorders they can only pull the trigger, they don't load the gun. I am still looking for an asthmatic patient in whose illness only psychic and no other, including allergic, factors are at work. Moreover, in assessing the relative importance of psychic versus allergic factors in the overwhelming majority of disorders commonly considered allergic, and especially in those of the respiratory tract, I am reminded of the London merchant who was in court on a charge of adulterating his rabbit pies with horse meat. After some damaging evidence had been presented, he admitted that they did contain some horse meat. When further pressed by the judge, he admitted the proportion to be fifty-fifty. "And what is fifty-fifty?" "One horse and one rabbit." I don't deny the presence of a neurotic rabbit in many of these patients, but be sure to find the allergic horse.

Turning now to treatment, the first consideration should always be prophylaxis. In addition to what I have already said on this subject, let me remind you about a preventive measure in respiratory allergy about which all of us could do much more than is being done. Every time a patient with asthma is referred to me, admittedly the most serious of all respiratory allergic diseases, and I get the history of an allergic rhinitis that preceded the asthma by months or even many years, I regret the lost opportunity of preventing the asthma by proper treatment of the rhinitis. Perennial allergic rhinitis is the commonest of respiratory allergies. Even in my practice it is present in 69 per cent of patients whom I see for respiratory allergy.<sup>6</sup> It is the most important respiratory allergy, because it precedes and leads to the asthma in at least 35 per cent of my asthmatic patients. It is the most neglected respiratory allergy. Over half the cases sent to me have existed more than five years, a third for over ten years, and a quarter for over 15 years before an etiologic diagnosis was sought; only six per cent had lasted less than a year. In my practice, many cases of what others have called intrinsic asthma have proved to be the late complication of a long-neglected perennial allergic rhinitis.

The neglect is due in the first place to the patients themselves or to their parents. The symptoms are usually mild, not incapacitating, and persist unchanged over the years. Indeed, some people enjoy their sneezes. Remember the use of snuff by our ancestors.

Children are particularly likely to take their trouble for granted, and their parents, when they realize that the sniffly nose doesn't mean an impending cold, pay little heed. Fully 20 per cent of my cases of allergic rhinitis began in the first decade of life. But general practitioners are also to blame because they fail to inquire routinely about such nasal symptoms in all their patients, or are not impressed by the importance of such symptoms when they do encounter them.

Perennial allergic rhinitis is the worst treated of all respiratory allergies. For years it gets only neglect, or some antihistaminic drugs or a shrinking spray. Finally, when a complicating sinus infection or an obstruction by mucous polyps develops, the mistreatment may continue, now along surgical lines. Not that some operative procedure might not eventually be indicated, but the operations are performed before allergic studies are made and before a two-month trial of allergic treatment is given. Yet such treatment might have resulted in a need for less extensive operative procedures, and occasionally in no operation at all. Above all, proper attention to the nasal allergy might have prevented the far more serious complicating asthma.

From the standpoint of allergy, the first principle of treatment is that complete avoidance of offending allergens will result in complete relief of symptoms. Most patients and doctors now know this, and so they make an effort to take care not only of the patient's bedding, but that of his roommate as well. The commonest current mistake is to believe that a plastic cover over pillows and mattress will completely prevent contact with dust from their contents. There is nothing impervious about a zipper. Such a cover can only reduce the exposure by making the dust take a detour of a few feet in reaching the patient's nose. But what difference do those few feet make to a highly sensitive patient, who, for example, may sneeze and wheeze from the pollen of ragweed plants a few miles away? Plastic covers give partial avoidance, useful while travelling, but not good enough at home. Nor will a spray to coat the fibers of rugs or upholstery produce an effective seal to keep dust particles from leaving them. There can be no compromise in these items: patients must change their bedding to a non-allergic material, preferably foam rubber in pillow and mattress, and the bedroom should be devoid of rugs, upholstery and hangings. Complete avoidance also applies to food allergens. Here, again, patients make two common mistakes: they fail to avoid foods that were not tested (that is why a food test

survey must be sufficiently broad to include the range of what that individual is likely to eat in the course of a year). What is more important, they eat food mixtures of whose ingredients they are ignorant. A basic principle in allergic dieting is this: if you don't know what's in it, don't eat it.

If complete avoidance of an allergen is impossible, then partial avoidance to as great a degree as may be should be the goal. Allergic disease has a quantitative basis: much allergen will cause severe symptoms, less allergen will cause milder symptoms, little allergen may cause few or no symptoms. Partial avoidance alone may be enough to give clinical relief, or when combined with desensitizing procedures, may potentiate such procedures to the point of a satisfactory result. The failure to try for, or to attain a high degree of partial avoidance is an important cause for therapeutic failure. Let me give you two important examples.

Hot-air heating is today the chief cause of failure to control many cases of house dust allergy. We make every effort to render a bedroom allergen-free. But the hot-air heating system, with forced air supply and recirculating ducts, gathers the dust from all the rest of the house, especially from the ground floor with its major supply of heavy rugs and overstuffed furniture (mostly feathers), or from the cellar with its mold spores, and sends it into the bedroom where the patient spends more time than in any other single space. The error is compounded in many instances by using the system to cool the bedroom in the summer. Nor do the filters in use solve the problem: they do not restrain the finest particles, the ones that are the real cause of trouble.

Air-conditioning units are properly recommended to help patients with pollinosis, but they are often improperly used. They rid the air of pollen, not to any degree by means of the rather puny filters which they contain, but chiefly by catching the pollen granules in the water of condensation that forms when the moisture of the air collects on the refrigerating coils. This process is well able to remove the pollen from the air of the room when only room air circulates through the gadget. But it cannot do this for the air of all outdoors. On this one fact has hinged the success or failure of a plan of pollen treatment.

When allergens are unavoidable, and when partial avoidance alone fails to give relief, then desensitization with an extract of the

offending allergens becomes necessary. The results are variable, and never as good as those from complete avoidance, because complete desensitization is never achieved. Nevertheless, the degree of hypersensitization attainable plus the lessened exposure by partial avoidance can give a satisfactory result in most cases, provided we do not make some of the following mistakes. Tolerance is built up gradually; it is therefore unwise to give doses too often or to increase the dosage too fast. Once a week with most antigens and at most twice a week with pollens is the proper interval. Crowding the dosage or the interval will cause constitutional reactions that can be serious and that certainly can make the cure worse than the disease. Nor should patients rush into the office for an injection and then rush out again. The accelerated circulation will help bring on a reaction. The antigens are of protein nature and therefore precipitable by alcohol and various chemicals. Therefore syringe and needle must be sterilized only by boiling or autoclave. The importance of using an autogenous dust extract has already been mentioned.

Symptomatic treatment of allergic disease, useful though it be in giving temporary relief and at times even in saving life, never cured anything. Symptomatic treatment should therefore always be accompanied by definitive and specific measures, when such measures are available. How frequent are the violations of this principle, each one of you knows. I shall only refer to the most common: the careless use of antihistaminics, and the most serious: the improper use of the cortico-steroids.

The antihistaminics are a group of substances, many of them wholly unrelated, which have pharmacological actions that are supposed to be the direct opposite of those of histamine. They were brought into the picture of allergic disease because of the hypothesis that the allergic reaction in body tissue is mediated by the local liberation of histamine. Actually, histamine alone is unable to account for all the local effects in all allergic reactions, and therefore allergists have long thought in terms of H-substances, multiple histamine-like substances, rather than of histamine alone. Moreover, no antihistaminic has exactly the opposite action of histamine. But even with these and other limitations, these drugs are of great practical value, provided we take cognizance of those limitations.

Most antihistaminics have an atropine-like effect in lessening secretion in mucous membranes. In nasal allergy, with its rhinor-



rhea, this is a welcome and desirable effect. But in asthma it is most undesirable, because bronchial secretions are already reduced in amount and more viscid and tenacious in consistency. In the presence of acute infection this is even more the case, and now the further drying effect of antihistaminics can prove disastrous. Many a case of severe asthma has hereby been precipitated into a serious and even fatal *status asthmaticus*.

Antihistaminics in many patients have a more or less marked sedative effect. The first patient in the Mayo Clinic who was given Benadryl® slept for 20 hours, to the considerable discomfiture of the staff. The efficacy of Dramamine® in controlling motion sickness is in large degree a function of this sedative property. A sedative effect is quite desirable when the patient taking the drug is about to go to bed, but it is of serious moment in the daytime when the individual needs to be in full possession of his faculties. A few years ago I incurred the wrath of some manufacturers of these drugs when I<sup>7</sup> pointed out their potential danger in military personnel by impairing the efficiency of the user and so placing him in jeopardy and also those who might be dependant upon his alertness, for example, in piloting a plane or driving a motor vehicle. Here one must bear in mind the frequency with which antihistaminics are self-administered by countless people in seeking relief from the annoyance of the common cold. I am sure that every one of you has knowledge of accidents that were so induced.

Antihistaminics give symptomatic relief from hives. By the same token, they prevent the hives which a diagnostic skin test seeks to elicit. Patients to be tested should therefore be cautioned not to take these drugs on the day when tests are to be made.

Corticotropin and the related adreno-cortico-steroids are the most powerful agents that have come into man's possession since Prometheus stole fire from the gods. But their power can be for evil as well as for good, and we are only beginning to know their possibilities and adapt them to our use. Thus, in infectious diseases they stop the patient's symptoms, that is, his reactions to infection, he looks and feels well for a while, but the bacteria are not affected and the patient dies. Yet this control of symptoms can be turned to life-saving purpose in overwhelming infections, such as meningitis or pneumonia, by staying their progress long enough for the suitable antibiotic to conquer the infective agent.

In allergic diseases these substances have also proved to be by far the most potent means of control of symptoms, almost regardless of their cause or of the shock organ involved. Even chronic lesions, such as nasal mucous polyps, have been caused to disappear. Skin test reactions, both of the acute wheal type and the delayed tuberculin reaction, may lessen and become negative under continued administration. The duration of relief of the symptoms is longer than that from all other medicaments.

Yet the effect of steroids is purely symptomatic; there is no change in the underlying sensitivity. When treatment is stopped, exposure to allergens will again cause symptoms. Polyps recur, and skin reactions resume their former pattern.

What is more important, depending upon dosage and duration of their use, these substances can produce significant and even serious secondary effects that are well known to you, and I have described them elsewhere in detail.<sup>8</sup> But I would like to emphasize the harmful effect of prolonged administration upon the pituitary and the adrenal cortex. Corticotropin given over weeks and months results in partial atrophy of the anterior lobe of the pituitary and marked hypertrophy of the adrenal cortex. But when its use is discontinued, the pituitary picks up only very slowly, while the adrenal cortex atrophies to a size much smaller than it was to begin with. Cortisone, long administered, also results in pituitary atrophy and an even greater adrenal cortical atrophy, and these changes only slowly return to normal after the drug is stopped. In short, for many months after either drug is discontinued, the patient is definitely worse from the standpoint of both pituitary and adrenal size and function than he was when treatment was started.

The use of corticotropin and cortisone in allergic conditions must therefore be formulated with the foregoing facts in mind. The safest indications are those in which short-term (seven to ten days) steroid therapy will relieve the effects of a known antigen that can be avoided in the future. These include: severe drug reactions; contact dermatitis, especially if severe and exfoliating; severe serum sickness; severe ivy poisoning; trichinosis. In this group would also come the preparation of a patient sensitive to Lipiodol® for a necessary bronchogram, or of an asthmatic for an emergency operation. Here also could be listed a few asthmatic patients whose diagnostic studies have been completed, but specific therapy has not yet had time to take hold. This indication should be used cautiously.

Short-term steroid therapy in cases due to unknown allergens is justified in situations that are acute, in themselves of limited duration, but so severe as to threaten life. Severe Loeffler's syndrome would fall in this group. It could also include *status asthmaticus*, but only after all other methods have failed. I have not yet needed to use it in such a case.

Long-term steroid therapy is justified in allergic conditions of unknown cause if they threaten life. Notable examples are periarthritis nodosa, severe allergic purpuras and severe berylliosis.

Long-term steroid therapy is certainly not justified in allergic conditions that in themselves may be annoying but not incapacitating, such as seasonal or perennial rhinitis or urticaria. These can and should be controlled by simpler and safer measures. One doesn't shoot sparrows with 16-inch guns.

The debate now rages as to the justification of long-term steroid therapy in allergic states that can be severe and more or less incapacitating, notably asthma. The opening wedge has been offered by the proponents of steroid therapy who would use it in cases of severe intractable asthma. At first glance this would seem logical. In practice, however, it soon becomes clear that "intractability" is defined very differently by different observers. Men of long experience in allergy find it difficult to recall more than a score or two of really intractable cases over the years. But men of far less experience speak of treating a series of 160 cases of "intractable" asthma. It is clear that these must include patients who certainly have not received proper diagnostic study, and treatment based thereon. As a result, there are being encountered an increasing number of untoward effects of such prolonged therapy in asthmatics: severe gastrointestinal bleeding, osteoporosis and spontaneous fractures, hypertension, vascular disease, psychosis, convulsive seizures and sudden death. It is now known that the minimal amount of cortisone that is required to maintain an asthmatic symptom-free is 50 to 75 mg a day, or more. Even the lowest dose thus represents a functional hypercorticism and is therefore capable of producing in time the more serious manifestations of Cushing's syndrome. I have also seen frank addiction to these drugs. Patients will shop from physician to physician until they find one who is willing to continue to prescribe cortisone for an asthma that should have been treated otherwise, and they become panicky when lowering or cessation of dosage is suggested. Moreover, one must

reckon with transient adrenal insufficiency when treatment is stopped. And, of course, during treatment any attempts at etiologic diagnosis are hampered by the diminished skin reactivity on testing.

It is clear that steroid therapy which may have to be continued indefinitely can lead to serious consequences. It should therefore not be undertaken lightly, or without due regard to its dangers.

In contrast to the many problems raised by the systemic use of steroids, their topical application apparently produces no untoward effects. The amounts involved are small and absorption is negligible. The ophthalmologists have used this method in the conjunctival sac and with good results. An aerosol preparation for inhalation in asthma, on the other hand, has proved of no value. Application of hydrocortisone to nasal polyps by means of a spray has recently been carried out by Silcox<sup>15</sup> with marked reduction in the size of large lesions and disappearance of smaller ones during the continuation of treatment. This should be a valuable adjuvant in the definitive management of this condition.

The treatment of infection is so prominent a part of the daily task of each member of this society that discretion should bid me be silent. But discretion never having been one of my major virtues, I dare to express the thought that in general all too many people are being given all too many chemotherapeutic agents for each and every infection, and so are less and less able to stand on their own immunologic legs. In particular, we are sensitizing all too many allergic individuals unnecessarily to very potent agents given in a trifling illness, so that they are deprived of help in a later serious infection. Penicillin<sup>11</sup> is now the commonest cause of drug allergy, with the most varied and severe reactions, and has replaced foreign sera as the most frequent cause of anaphylactic death. We might prevent much sensitization by not giving penicillin in minor ailments, by avoiding local applications of the drug, by avoiding its depot preparations unless decidedly needed, by giving preference to the oral route of administration, and by avoiding combined parenteral injections with other possible antigens. We could prevent many penicillin reactions in our allergic patients by asking them about previous experiences with the drug, by using oral administration, or if it must be given parenterally, by giving the injection into the arm alone and low enough to permit the use of a tourniquet in case of severe reaction. This thought could



be extended to other of our therapeutic practices with allergic individuals.

I even venture some closing thoughts on surgery in the upper respiratory tract of the allergic patient. It bears repetition that primary and thorough attention to the allergic factors for a two-month period may materially improve the picture and so call for a lesser operative procedure than at first seemed necessary. But those lesions which a clinical and x-ray study now show to remain should be vigorously attacked. The most thorough, seemingly radical operation, such as the Caldwell-Luc for the antrum, will prove in the long run to be the most conservative from the standpoint of the patient's outlook. The golden opportunity to get the best autogenous vaccine is at the time of such an operation. A septum that makes constant lateral contact with the turbinals, or one that obviously interferes with the airway or with the drainage of sinuses, should be straightened. The same is true of a crooked septum which, although not involved in contact or obstruction, offers a considerable surface facing the nostril for the impingement of inhaled particulate matter. Quite as important as the original operation in all of these cases is the regular follow-up and postoperative care by the surgeon *himself*, lest synechiae and other complications arise to nullify the good done by the operation. In the matter of infected lymphoid tissue, one must bear in mind the whole of Waldeyer's ring, not just the faucial tonsils. These are all basic principles that continue to stand as important beacons in the narrow waters of the rhinological sea.

As leaders in your field you know full well that basic principles have been the sure foundation on which were built the great achievements of the past, and, you are the very custodians of such principles. But more than that, you are the teachers of those who will follow and upon whom will rest the responsibility of building the greater glories of the future. You all know how difficult it is to convince the young of the importance of fundamentals. Each new generation insists on making its own mistakes and then hides its confusion behind a false proverb to the effect that "experience is the best teacher." The Spanish version of that saying is more accurate: "experience is a good teacher, but sells its lessons very dearly." And Franklin's Poor Richard was yet more brutally frank: "Experience keeps a dear school, but some will learn in no other, and fools scarcely in that." The wonder is that so many of us have survived and made useful our mistakes; I return to Kipling:

"Then the Gods of the Market tumbled, and their smooth-tongued  
wizards withdrew,  
And the hearts of the meanest were humbled and began to  
believe it was true  
That All is not Gold that Glitters, and Two and Two make Four—  
And the Gods of the Copybook Headings limped up to explain  
it once more."

And so as teachers we try to transmit not only facts, but ideas;  
not merely data, but principles; so that to knowledge may be added  
understanding, and out of understanding may grow wisdom.

BROAD AND ONTARIO STS.

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# VOICE PRODUCTION IN THE LARYNGECTOMIZED PATIENT

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AND

DAVID W. BREWER, M.D.

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Every laryngologist has often tantalized his inquiring mind with this question: "How can some laryngectomized patients develop highly intelligible postoperative voices while others are unable to speak at all without mechanical aid?" No single answer is readily available.

In reviewing the accomplishments of a small local group of laryngectomized patients, several of whom had been operated upon in other cities, our impressions can be summarized as follows: It appeared that the better educated patients, or those whose financial programs were most demanding, or those whose native ability to imitate was highest, or those whose hearing ability was normal spoke much earlier. On the other hand, those who had sustained a severe hearing loss were unable to hear their own attempts to eructate air. Some surgeons felt that removal of the strap muscles changed the intrinsic mechanics so that voice was developed at a later date; others felt that removal of varying portions of the esophagus had a similar effect; some felt that preoperative or postoperative irradiation, especially the former, had a definite deleterious effect on the patient's ability to speak. In contrast to the clinicians' impressions were those of the esophageal voice teachers, the majority of whom were laryn-

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DR. GORDON D. HOOPLE: Mr. President, Fellows and Guests, many of you in this room knew Dr. Irl Blaisdell. For those of you who did not I would like to say that he was associated in practice with Dr. Brewer and me. He was killed last summer in an automobile accident. He was a brilliant laryngologist. The work that we are presenting is in essence his. Dr. Brewer has added the finishing touches. I can claim only a fatherly interest. We would like with your permission to dedicate this presentation to Dr. Blaisdell's memory. Because Dr. Brewer completed the study he should and will read the paper.

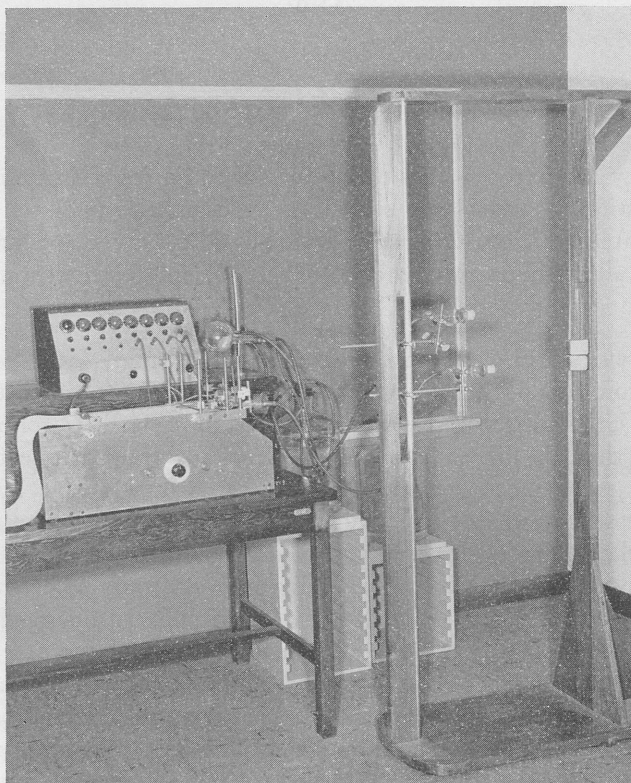


Fig. 1.—Custom built variable speed pentapneumodeik kymograph.



gectomized patients. Certain teachers felt that postoperative laryngectomized voice was entirely esophageal, and others that it was entirely pharyngeal. New patients seeking the help of both types of teachers were immediately confronted with a dilemma. After only partially successful attempts with one teacher they came into the hands of another who assured them they were doing everything wrong. The patient then went home and wrote on paper to his family that he would never speak again.

#### METHOD OF STUDY

In an attempt to shed light on this problem Dr. Irl H. Blaisdell's enthusiastic interest was imparted to Dr. Walter Amster, of the Hearing and Speech Center at Syracuse University. Dr. Amster chose this problem as his candidate's thesis for his Doctor of Philosophy degree. This was completed in 1953 and dedicated to Dr. Blaisdell. Most apropos at this point, a brief review of his hypotheses, methods and findings are as follows: He proposed that: (1) laryngectomized speakers essentially continue normal breathing and speech co-ordinations, and (2) that speakers judged more intelligible would approximate more closely normal co-ordinations than those judged less intelligible.

Fifteen laryngectomized patients were studied. The necessary data was obtained by using a variable speed kymograph, as shown in Figure 1. With this apparatus tracings of movements of the body wall at the lower sternum and mesogastric areas both during silent breathing and speech were taken. Syllable pulse during speech was also recorded (Fig. 2).

In addition tape recordings of speech were made and submitted to auditing by speech pathology and audiology students for intelligibility ratings (Fig. 3).

In Figure 4 two normal and two laryngectomized patients are recorded during silent breathing. The negative pressure lead presents as a straight line since no syllable pulse is produced without speech. Slightly faster respiration and greater chest expansion per minute are indicated.

Figure 5 represents tracings from two normal speakers and two highly intelligible laryngectomized speakers. Note the increased phrasing necessitated by the last speaker.

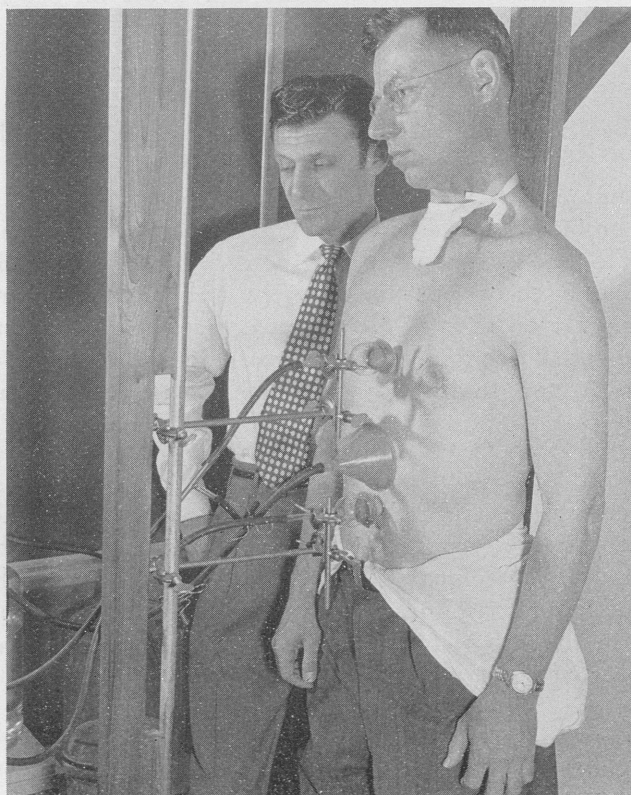


Fig. 2.—Lower sternal lead, negative pressure lead and mesogastric lead in recording positions.



Fig. 3.—Recording speech on magnecord tape recorder.

TABLE 1.  
SURGICAL DATA DIVIDED ACCORDING TO INTELLIGIBILITY GROUPS.

	+1 Group N = 8		-1 Group N = 7	
	Yes	No	Yes	No
Deep x-ray therapy previous to or following surgery	0	8	1	6
Radical neck dissection	0	8	2	5
Removal of epiglottis	7	1	6	1
Removal of strap muscles	7	1	5	2
Resection of any portion of esophagus	1	7	1	6
Postoperative fistula	5	3	2	5

In Figure 6 four low intelligibility laryngectomized speakers demonstrate irregular chest wall movements, out of phase movements and dysynchrony.

Those most easily understood approached the speech co-ordination of normal subjects most closely. This co-ordination consisted primarily of rhythm, time and phrasing.

Surgical data revealed no direct relationships between the extent of surgery pre- or postoperative irradiation or fistulization with voice intelligibility (Table 1).

It was concluded that there was a definite dissynchrony between respiration and voice production in the poorly intelligible speakers as compared with essentially normal movement of the diaphragm, thoracic cage and voice production in the highly intelligible speakers.

It must be remembered, however, that an individual's potential is difficult to judge on any one single examination and that after subsequent training some of the poorly intelligible speakers are now highly intelligible. The changes we attribute to the difference in technique of voice production which these patients have acquired either by their own efforts or after training with a teacher.



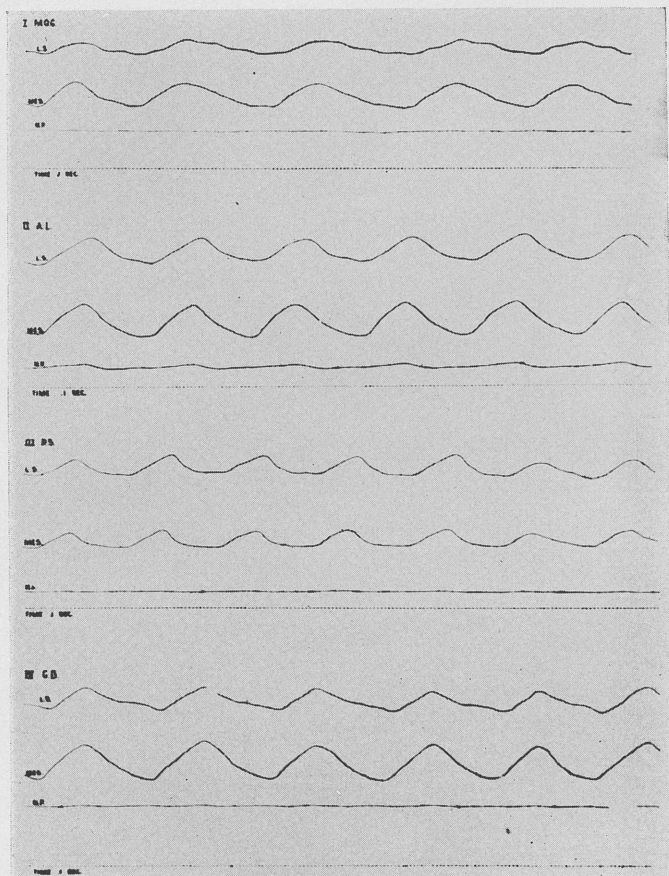


Fig. 4.—L.S., lower sternal lead; Mes., mesogastric lead; N.P., negative pressure lead to record syllable pulse.

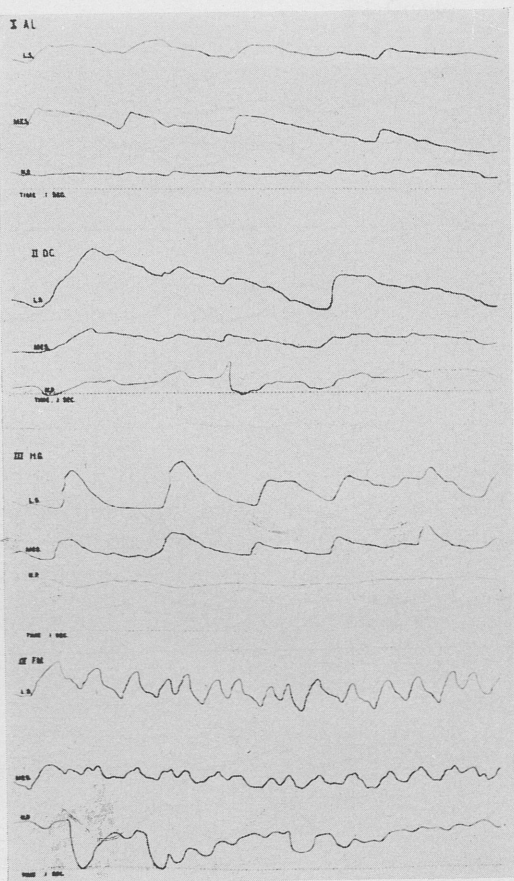


Fig. 5.—L.S., lower sternal lead; Mes., mesogastric lead; N.P., negative pressure lead to recording of syllable pulse.

In order to visualize the mechanics a normal speed cinefluoroscopic study was made through the facilities and highly cooperative efforts of the Department of Radiology at Strong Memorial Hospital in Rochester, New York, under the direction of Drs. Ramsey, Watson and Gramiak. Ten laryngectomized patients were studied by giving thin barium and recording the movements of the hypopharyngeal and esophageal walls during voice production as well as by studying the intercostal and diaphragmatic movements during verbalization of the same material. It soon became evident that the motion at normal speed was too fast for accurate study.

However, the kymographic and taped recordings were submitted to statistical analysis and upheld the findings of the preliminary cine-fluorographic studies which were examined by Dr. Blaisdell and reported as follows: 1) High intelligibility speakers did not appear to employ swallowing behavior. Their intake of air was limited to the proximal portion of the esophagus in the approximate region of the reconstructed crico-pharyngeal sphincter, 2) low intelligibility speakers revealed decided swallowing behavior which propelled air into the medial portion of the esophagus with some escape into the stomach. In these patients the pseudo-glottis appeared to occupy a relatively larger vibrating portion of the esophagus extending below the reconstructed crico-pharyngeal sphincter.

It was proposed that by employing a slow motion technique with a sixteen millimeter Fastax camera set at its slowest speed of 150 frames per second, it would be possible to photograph the fluoroscopic image provided by a television image amplifier so that the intrinsic motion of the pharynx and esophagus could be more carefully studied. This was attempted, but only after a delay of several years necessitated by additional time required for perfection and delivery of the television image amplifier. Test runs recorded no image and it was concluded that the brilliance of the image was still insufficient to allow ultra-slow motion cinefluorography.

However, utilizing normal speed as well as conventional slow motion the last brief section of the motion picture was obtained. They may be identified by the circular mirror image employed, and are silent since sound recorded at slow motion movie speeds cannot be reproduced accurately when played back at normal motion picture film speed.

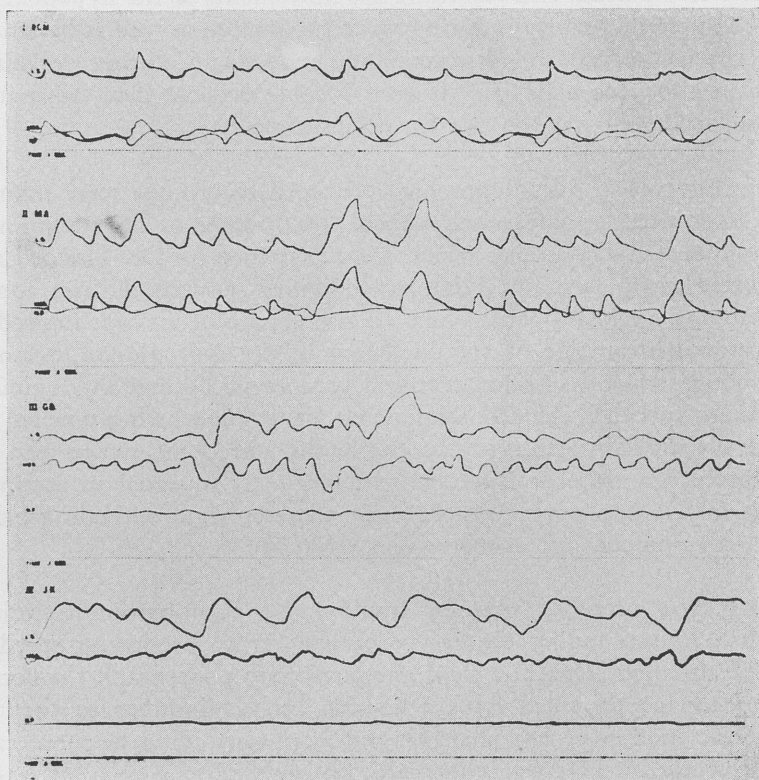


Fig. 6.—L.S., lower sternal lead; Mes., mesogastric lead; N.P., negative pressure lead recording syllable pulse.



Repeated fluoroscopic and indirect laryngoscopic examinations of laryngectomized patients led to the impression that the folds of the reconstructed crico-pharyngeus form the only primary vibratory mechanism. The ease with which air escapes past this sphincter determines whether the patient was previously classed as an esophageal or pharyngeal speaker.

These findings appear to support partially the recently reported studies of Dr. Annie Moolenaar-Bijl<sup>1</sup> of the Netherlands who used a single exposure plain film technique in her investigation. She referred to three methods of trapping air—by inhalation, by injection and by deglutition. In our acquaintance those non-laryngectomized teachers of this type of voice have used either inhalation or injection. To inhale and phonate is relatively simple for many. In pronouncing the phrases containing the consonants p, k, and t, "pah" and "mah" the individual's lips or tongue appear to compress air in the mouth and pharynx and allow it to escape into the upper third of the esophagus through the loose folds of the reconstructed crico-pharyngeus muscle, and no audible or visible swallowing takes place. Other syllables, such as "dah," can be produced only after deglutition of air although in many of the highly intelligible speakers the majority of the air stays in the upper third of the esophagus and only small portions of it reach the lower esophagus and stomach. Barium swallow examination postoperatively may be very helpful in evaluating the patient's potential.

#### CONCLUSIONS

A study of voice production in the laryngectomized patient utilizing kymographic and tape recordings of the phonatory apparatus, when checked by statistical analysis, showed a definite difference in the mechanics by which air was trapped in the pharynx and esophagus by high intelligibility speakers as compared with low intelligibility speakers. Cine-fluorographic studies during phonation, confirmed by repeated fluoroscopic examinations demonstrated that in the patients studied crico-pharyngeal sphincter control was the deciding factor as to where and when the air was trapped. Furthermore, the folds of this sphincter were seen to represent the primary source of sound vibration for voice production. When initial laryngectomy voice training is designed so that the patient can trap air in the upper third of the esophagus without deglutition the highest intelligibility is obtained, though excellent intelligibility can be secured by utilizing the full act of swallowing.

Finally, barium swallow examination should be highly useful postoperatively for evaluation of voice production potential.

1100 EAST GENESEE STREET.

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#### DISCUSSION

DR. JULIUS W. MCCALL: Mr. President, Fellows and Guests, certainly this is a very interesting demonstration of what happens in the pharynx and esophagus as shown by Dr. Hoople and Dr. Brewer. Dr. Brewer brings up an interesting question, why some laryngectomized patients are able to talk and some do not develop an esophageal voice.

I hope to give you a partial solution to this problem. The esophageal voice is an interesting phenomenon. Cermak in 1859 recorded the first case of the esophageal voice. Stoerck and Gersne report a case in which a voice was produced after total laryngectomy. No organized effort to teach the esophageal voice was made until 1943. The Lost Chord Club was formed at the Cleveland Hearing and Speech Center under my supervision. I am more convinced as time goes on that the time to begin training these patients is before and not after laryngectomy. The esophageal voice is the result of timing and synchronization of movements of the lips, tongue and teeth with an adequate expulsion of air from the esophagus and pharynx. Therefore the development of an esophageal voice is dependent on the patient's ability to expel forcibly an adequate volume of air from the pharynx and esophagus, in other words, the act of belching. The laryngectomized patient's ability to develop an esophageal voice is dependent therefore on mastering the technique of belching. It is much easier to learn to belch before the larynx is removed than afterwards. Before laryngectomy he still has the glottic valve to stop the respiratory act. Belching also helps him to disassociate the respiratory mechanism from the pharyngo-esophageal effort. If the training is delayed until after the laryngectomy the check to respiration offered by the glottis is gone and the patient will explode air from the trachea instead of the esophagus. Most patients who must submit to laryngectomy desire a few days to arrange their affairs and if this time is utilized in learning to belch it will greatly shorten the post-operative period of training in the development of the esophageal voice.

(Slide) In a recent review of 149 laryngectomized patients done at St. Luke's Hospital in Cleveland, 99 had preoperative voice training. Of these 99, 67 developed a good voice and 16 had a fair voice, 13 had no voice; 27 had no preoperative voice training of those only 13 or 48 per cent had a good voice, one had a fair voice and 13 or 48 per cent were poor to no voice. In fairness to your patients who are to have laryngectomy, start them belching before operation.

Thank you!

DR. LOUIS H. CLERF: Mr. President, I heartily agree with Drs. Hoople, Brewer and McCall that removal of the larynx in carcinoma does nothing toward

rehabilitating the patient. Since voice production is very important and a large proportion of laryngectomized patients can be rehabilitated almost 100% this problem becomes an obvious part of the pre- and post-operative preparation and care. I refer to have a laryngectomized patient with a good voice talk to these patients.

There are three types of voices observed in laryngectomized patients, the buccal voice which sounds like a quacking duck, the pharyngeal voice which is not much better, and the esophageal voice which is the best.

There is considerable difference of opinion as to how to get air into the esophagus. One does not swallow it and use the stomach as a reservoir. I have been able, by relaxing the cricopharyngeus to draw air into the upper esophagus and then can belch it at will. Radiographic studies revealed the air in the upper esophagus. I cannot agree with those who say that one ought to swallow some effervescing drink and then belch, except merely to demonstrate that a belching sound can be converted into pleasing speech. It is better to inhale and draw the air down into the upper esophagus and then forcing it out, cause mucosal folds to vibrate.

DR. HENRY B. ORTON: Mr. President, Members of the Association and Guests, I have enjoyed this presentation very much. However, I am more inclined to believe that this is a pharyngeal voice rather than an esophageal voice. I agree that the constrictor muscles play a considerable part. In order to get air into the pharynx we open the mouth and air rushes in which will act in the same way as a bolus of food in the simple act of swallowing. The first motion of which is the elevation of that bolus of air up to the roof of the mouth by the tongue, the second motion is just pushing it a little bit farther back, and just at the point where you are going to lose control of this air (as a bolus of food) you burp. In other words, you do not swallow the air.

Regarding the quality of voice; I feel the intelligence of the laryngectomized patient plays a very small or minor part in this. I remember very well one individual, an Italian who could speak the English language but he could neither read nor write it, naturally he was in a bad way, but that man was talking freely before he left the hospital. Then there is the other side of the picture, that of a very intelligent man, an executive whom I operated on almost 20 years ago, and he will not say a word. Why? Because he has a non-cancellable life insurance policy. This might be construed as super intelligence!

Thank you very much.

DR. DAVID W. BREWER: I wish to thank Dr. McCall, Dr. Clerf and Dr. Orton very much for their excellent discussion. I think Dr. Orton's patient who does not wish to cancel his life insurance by speaking is highly intelligent.

## PHARYNGOESOPHAGEAL DIVERTICULUM AND THE OTOLARYNGOLOGIST

FRANCIS E. LEJEUNE, M.D.

NEW ORLEANS, LA.

The expanding field of otolaryngology has focused attention on conditions heretofore shared, in many instances, with the general surgeon. Pharyngoesophageal diverticulum is but one of these many conditions whose management rightly falls into the domain of the well trained otolaryngologist. Too frequently, articles on this subject have appeared in our journals stating that the otolaryngologist in the role of endoscopist is teamed with the general surgeon in the surgical correction of this condition. Except in those cases in which the diverticulum has invaded the thorax, the well trained otolaryngologist is thoroughly capable of performing this type of operation.

Attention was first called to esophageal diverticula in 1816 by Bell.<sup>1</sup> Zenker's<sup>2</sup> description of pharyngoesophageal diverticula in 1877 was so classic that the lesion is often referred to as Zenker's diverticulum. This presentation will be limited to a discussion of this latter type and a review of our experience with 73 cases encountered at the Ochsner Clinic in the ten year period ending December 31, 1953.

Contrary to indications in the literature, the incidence of pharyngoesophageal diverticula is not increasing but the diagnosis is merely being made with greater frequency. The condition is usually encountered in older people. The youngest patient in our series was 38 years of age and the oldest 78, the average age being 60.6 years. Probably the reason for this is that the patient may have the condition for years before it becomes troublesome enough to make him seek medical attention. The lesion also seems to be somewhat more common in men than in women, 44 of our 73 patients being men.

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Because of their location at the junction of the hypopharynx and esophagus, pharyngoesophageal diverticula are of great clinical interest to the otolaryngologist. They always occur on the posterior pharyngeal wall at the junction of the hypopharynx and the esophagus directly behind the cricoid cartilage where the oblique fibers of the inferior constrictor of the pharynx and the orbicular fibers of the cricopharyngeus muscle meet. In this location is a small triangular space, varying in size with the individual and unprotected by muscle. This area was first described by Lannier Hackerman and is frequently referred to by his name. A slight herniation develops in the posterior wall in the midline. As long as it is small, it will remain in the midline, but as it enlarges it will extend to one side or the other, usually to the left. In 46 of our patients the pharyngeal pouch deviated to the left side, 20 were in the midline and in only 7 was the deviation to the right side.

Because of no history of trauma or difficulty in deglutition, development of a diverticulum in this particular area is thought by some to be due to weakness of the triangular space between the muscle fibers. However, Jackson and Shallow<sup>3</sup> did not consider the theory of congenital weakness "a reasonable one." They believed the chief factor in their formation to be the pinchcock action of the cricopharyngeal muscle. The powerful action of this muscle keeps the mouth of the esophagus closed except when it momentarily opens to permit passage of food. If it fails to open, or opens tardily, such tremendous pressure is exerted in the walls of the hypopharynx that pouching may occur in a congenitally weak spot. This hereditary tendency to exceptional weakness in the posterior wall of the hypopharynx in four members of one family was recently reported by Bjork.<sup>4</sup> The association of pharyngoesophageal diverticula with goiter, which has been reported,<sup>5</sup> has not been observed by us.

Regardless of the etiology, once invagination begins, it gradually enlarges thereafter and the patient eventually becomes conscious of certain discomforts pathognomonic of this condition. The onset is so insidious that the patient may have symptoms for a long time before he seeks relief. On the other hand, some patients may have no manifestations to suggest the presence of the lesion. This was the case in nine of our patients, whose diverticula were discovered on routine examination. Gradually, mild dysphagia and a sensation of fullness and choking develop, and the patient is bothered with unpredictable regurgitation, which usually seems to be more pronounced

at night. Occasionally, patients will relate that they have a pocket in their throat, and pressure on the side of the neck results in regurgitation. Some patients complain of a sensation of smothering and an accumulation of fluids in the throat, particularly at night, which is frequently followed by severe bouts of coughing. Dysphagia progresses until loss of appetite and weight forces the patient to seek medical consultation. As the diverticulum increases in size and food accumulates within its lumen, the esophagus becomes more compressed and the patient becomes conscious of retrosternal pressure and discomfort. Gurgling noises are frequent and annoying.

Pharyngoesophageal diverticula may at times assume large proportions and retain food or fluids for a long time. Peikoff<sup>6</sup> reported the case of a patient who considered his diverticulum advantageous. He discovered that at cocktail parties the first few swallows of liquor lodged in the pouch, which distended considerably with each drink. Later in the evening, he could have an extra drink when he needed it by pressing the pouch with his thumb.

The diagnosis of pharyngoesophageal diverticula is easily suspected from the symptoms and quickly established by barium visualization of the esophagus. However, roentgenograms should be made in the anteroposterior and oblique positions. Esophagoscopy should verify the roentgenographic observations and eliminate the possibility of strictures or early malignancies. Esophagoscopy is not always easy; the location, size and pressure exerted by the diverticulum tends to push the esophageal opening anteriorly making it at times difficult to find and visualize satisfactorily. The mouth of the diverticulum remains open more or less at all times; hence food and liquids find their way into this sac more readily than into the esophagus proper.

The treatment of pharyngoesophageal diverticulum has long been recognized as essentially surgical, preferably in one stage, although some advocate two stage diverticulectomy in which the sac is first exteriorized and anchored in the wound near the skin, and ten to fourteen days later it is excised and the wound permitted to close by secondary intention. Welty<sup>7</sup> championed the two stage procedure because of its innocuousness, the tranquility it gives the surgeon and the complete cure it provides. Knight,<sup>8</sup> on the other hand, is of the opinion that this method invites infection and other complications. Recently, Crile and Robnett<sup>9</sup> reported the successful

treatment of pharyngoesophageal diverticula in 11 patients by employing the usual incision, dissecting up the neck of the sac, placing a purse-string suture around the neck and then inverting the sac into the esophagus. With modern asepsis and antibiotics this procedure would seem to have no particular merit. We have always employed the one stage operation because it is technically simple and gives excellent results. A physical examination determines the length of the preoperative hospitalization for rest, preparation and administration of fluids.

#### TECHNIQUE

An incision extending from the middle portion of the thyroid cartilage up to the clavicle is made along the anterior border of the sternocleidomastoid muscle, preferably on the left side except for diverticula located on the right side. The anterior border of the sternocleidomastoid muscle is retracted outward to expose the deep cervical fascia, which is divided parallel with the cutaneous incision. The carotid sheath and its contents are carefully retracted outward while the sternohyoid and larynx are retracted inward. If the omohyoid interferes too much with exposure of the sac, it may be divided. The thyroid vessels should be ligated. Exposure of the thyroid gland and rotation inward provide a view of the cricoid and adjacent tracheal rings. Posterior to these structures lies the esophagus, and if the sac is small, it will be found just below the level of the cricoid cartilage posterior to the esophagus. A large sac will be encountered before the esophagus proper is exposed. When the sac is found, it must be cautiously dissected free of its attachments. If any difficulty is experienced in localizing the sac, the esophagoscope is introduced directly into the diverticulum. Retained secretions are aspirated and a piece of merthiolated gauze to which a string has been attached, is packed into the diverticulum. Regardless of any difficulty, it has become routine for us to introduce an esophagoscope into the esophagus prior to excising the sac. This serves as a guide to excision of the neck of the sac. Before this was done routinely, too vigorous traction on the sac in two of our cases resulted in excessive removal of tissue, which in turn caused strictures necessitating dilation over a long period of time. When the sac is ready to be incised, the merthiolated gauze pack is removed perorally and the neck of the sac is then carefully incised. Subcuticular interrupted sutures are used to approximate the edges of the delicate pharyngoesophageal mucosa and the muscular layers are carefully brought together and sutured

tightly. A dermal drain is placed in the lower portion of the incision, and after the cervical structures are approximated, the wound is closed. A Levin feeding tube is used for four or five days.

#### RESULTS

In our series the 9 patients whose diverticula were discovered on routine examination refused surgical treatment. In spite of definite symptoms and large diverticula, 23 other patients were not operated upon. Nine of these latter patients had severe heart conditions making them poor surgical risks, several others were of advanced age and in poor physical condition and the remainder refused to undergo any type of operative procedure. Diverticulectomy in one stage under general anesthesia was done in the remaining 41 patients. Results were successful in 36 of these. Complications developed in the other 5 patients. Paralysis of the right recurrent nerve occurred in one; paralysis of the left recurrent nerve in another; a persistent fistula necessitating re-operation developed in the third patient and strictures necessitating prolonged dilatation developed in the other two patients but both eventually completely recovered.

#### COMMENT

Even though every precaution is taken to avoid injury to the inferior laryngeal nerves, occasionally such complications will occur resulting in paralysis of one vocal cord. This does not always mean that the nerve has been sectioned, as paralysis may result from traumatization of the nerve with forceps or from pinching of the nerve by scar tissue produced by postoperative infection. Also, infection may cause disruption of the line of sutures resulting in a fistula, which heals in due time. The two cases of stricture in the present series were due to removal of too much pharyngoesophageal mucosa which could have been avoided by insertion of an esophagoscope into the esophagus. Since doing this routinely, we have never removed too much mucosa. For this reason, we believe the esophagoscope should always be used in the performance of pharyngoesophageal diverticulectomy.

#### SUMMARY

Pharyngoesophageal diverticula are being recognized with increasing frequency. Because of their location at the junction of the hypopharynx and esophagus, they are of considerable interest to



otolaryngologists. They are thought by some to be due to congenital weakness of the triangular space between the muscle fibers and by others to the pinchcock action of the cricopharyngeal muscle. Patients become aware of the anomaly by gradual development of dysphagia, a sensation of fullness and choking and unpredictable regurgitation. These clinical manifestations suggest the diagnosis, which may be readily established by barium visualization of the esophagus. Treatment is surgical excision of the diverticulum, preferably in one stage. Of 41 patients with pharyngoesophageal diverticula treated in this way, successful results were obtained in 36; complications developed in the remaining 5.

Because of the exposure to excellent training in neck surgery, and because of the intimate knowledge and daily experience with the pharynx, hypopharynx, larynx and esophagus as well as with cervical anatomy, the otolaryngologist occupies an enviable position when surgical correction of pharyngo-esophageal diverticula is considered.

OCHSNER CLINIC.

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## DISCUSSION

DR. LOUIS H. CLERF: Mr. President, I am particularly interested in this subject as I have aided in the surgical treatment of many cases and I wish to compliment Dr. LeJeune on his presentation.

I am heartily in agreement with him concerning the importance of a pre-operative esophagoscopy. While it is true that a majority of these patients can be diagnosed by roentgen ray studies we do encounter an occasional carcinoma of the pouch or of the subdiverticular esophagus.

I agree that a one-stage operation is the most desirable plan of treatment irrespective of reports of mediastinitis. We have had none. There is no substitute for surgical treatment. There commonly is progressive enlargement of the pouch particular in elderly patients with increasing dysphagia due to compression and respiratory symptoms resulting from aspiration, particularly while asleep in a prone position.

With regard to inversion of the sac, if it is small I can see no serious objection. If the sac is large and extends into the upper mediastinum inversion could be hazardous.

We have had six cases of paralysis of the larynx in a series of more than 200. The paralysis was incomplete and we ascribed it to compression of the recurrent nerve by the retractor. All of these recovered within six months. The occurrence of fistula usually can be avoided if the neck of the pouch is transfixed with a suture ligature before amputating it.

Atresia or stenosis can be prevented by passing an esophagoscope into the subdiverticular esophagus. We do that routinely and have had no cases of stenosis. We have had opportunity to see 2 patients with atresia who had a gastrostomy done because they could not swallow. One had the esophagus incorporated in the suture ligature and it was tied off very effectively. The other had a portion of the esophagus removed with the neck of the pouch, so it was necessary to take care of that. If one introduces the esophagoscope before amputating the pouch there will always be a food passageway.

DR. FRANCIS E. LEJEUNE: I just want to thank Dr. Clerf for his courtesy in discussing my paper.

I have nothing else to add, Mr. Chairman.

## A THIRTY-YEAR REVIEW OF FRONTAL SINUSITIS TREATED BY EXTERNAL OPERATION

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The pioneers of surgery advocated the external drainage of the frontal sinuses. One of the first such operations was what is known today as the trephine operation. This consisted of an external opening and drainage of the frontal sinus without enlarging or opening the nasofrontal duct. A later operation was that advocated by Killian, and this became the most popular operation at the turn of the century. It consisted of a partial removal of the anterior wall of the frontal sinus and a partial removal of the floor, leaving the supra-orbital ridge as a bridge between these two openings. Drainage was established through the anterior ethmoid cells into the nose. This operation failed because the duct frequently closed and because the ridge of bone often sequestered.

*Intranasal Surgery.* Because of failures, complications, and disfigurements, these external operations fell into disrepute in the early 1900's. They were followed by intranasal operations. Because of the inherent dangers and indiscriminate use of intranasal operations, there was a return to external surgery. Mosher<sup>13</sup> stated that intranasal ethmoid surgery is not easy and may be dangerous. Fenton<sup>1</sup> stated that the attempt to carry out excessively radical sinus surgery blindly through the narrow nasal pathway is a source of danger. Howarth<sup>7</sup> stated that he thought the pendulum had swung too far to the side of purely intranasal surgery. Patterson<sup>15</sup> stated that, because intranasal procedures had so often proven unsatisfactory and not free from danger, the external operation was again coming into favor.

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From the Department of Otolaryngology, Royal Victoria Hospital, and the Department of Neurosurgery, Montreal Neurological Institute.

In our hospital, intranasal surgery was never widely practiced. The records show that only 15 of the 77 patients under consideration in our series had had intranasal sinus surgery. Thirteen other cases in the chronic allergic group had polypi removed on one or more occasions.

*Modern External Surgery.* According to Hajek<sup>5</sup> the modern external frontal sinus operation gradually developed from an operation proposed by Jansen, in 1893. It was modified by Howarth,<sup>7</sup> Lynch,<sup>10</sup> Sewall,<sup>17</sup> Ferris Smith,<sup>18</sup> and Negus.<sup>14</sup> In America, this operation is known as the Lynch operation. In Britain, it is known as the Howarth operation. Most of the external frontal sinus surgery done today is some form of these operations, i.e., removal of the floor of the frontal sinus, preservation of the anterior wall, together with the complete removal of the anterior ethmoid cells, and probably the posterior ethmoid cells and sphenoids, together with infected mucosa. In addition, any infection in the antra, or any intranasal obstruction, is promptly treated. This is what we call a complete Howarth-Lynch operation.

*Modifications in External Surgery.* Goodale,<sup>4</sup> in 1945, reported four cases in which he placed tantalum foil in the nasofrontal duct. In 1948, Harris<sup>6</sup> described the use of a tantalum tube in the nasofrontal duct in three patients in whom the tube was left in position for three weeks. Scharfe,<sup>16</sup> in 1953, also described the use of a tantalum tube in the nasofrontal duct.

*Differences of Opinion.* As always, in an unsettled problem, one is impressed by the variability amongst the masters about fundamental points. Lynch regarded as dangerous the stripping off of the periosteum from the anterior frontal sinus wall because it may cause periostitis and subsequent osteomyelitis. Furstenberg<sup>2</sup> stated, on the other hand, that the blood supply for the cranial bones comes from the internal periosteum, and that very little harm is done by elevating the periosteum from the anterior wall of the frontal sinus. In a few cases in our series, the periosteum was stripped off the anterior wall of the frontal sinus in order to examine the bone when the x-rays suggested disease. In these cases this procedure was not followed by definite osteitis or osteomyelitis.

Lynch regarded as absolutely essential the stripping away of every shred of mucous membrane from the sinuses, as did Ferris Smith, but Howarth and Negus recommended leaving the mucosa if it is at



all possible, and Negus even suggested skin grafting within the sinus itself, this in addition to skin grafting the nasofrontal duct.

Lynch insisted on removing all of the floor of the frontal sinus, as did Sewall and Ferris Smith. Howarth<sup>9</sup> did at first, and then in 1936, when reviewing his cases, stated that it is not essential to remove all of the outer part of the floor. Negus,<sup>14</sup> in 1947, supported this later idea of Howarth.

Lynch insisted on intranasal treatment of the antra, as did Negus, but Howarth did not stress the importance of this. Lynch, Sewall, and Ferris Smith stressed the importance of including drainage of the sphenoids in the operation; Howarth did not.

Lynch stated that complete removal of the anterior ethmoid cells would provide for an adequate and permanent nasofrontal duct. On the other hand, Ferris Smith, Howarth, and Negus recommended the insertion of a skin graft in the nasofrontal duct, and Sewall advocated the use of a mucosal flap from the middle turbinate to line the nasofrontal duct.

#### DESCRIPTION OF MATERIAL

This is a survey of the records of cases of frontal sinusitis covering a thirty-year period (1923-1953) in the Royal Victoria Hospital and the Montreal Neurological Institute. The cases considered had inflammatory disease of the frontal sinus. As a rule, there was infection in other nasal accessory sinuses. Excluded were all primary cases of neoplasm, trauma, and mucocele.

Only those cases of frontal sinusitis which had been subjected to external operations were reviewed. During the interval under consideration, the number of acute cases involving the frontal sinuses admitted to hospital was estimated at approximately 425. The number of cases of chronic frontal sinusitis was estimated at approximately the same, bringing the total number of cases to about 850. Other hundreds of patients with less severe frontal sinus infection had been treated on an ambulatory basis. The total number of patients admitted to the Departments of Otolaryngology and Neurosurgery during the period under review exceeded 87,000. We have reviewed the records of about one hundred of these patients who were subjected to external operations, and we have selected 77 in which the records were sufficiently complete and in which the follow-up information permitted the drawing of conclusions. Eleven of the 77

patients could not be located for a recent report, but even in these there was valuable information which justified their retention in the series. The patients were operated upon by the various members of the Departments of Otolaryngology and Neurosurgery.

The indications for external sinus surgery are fairly generally agreed upon.

Negus, in 1947, stressed the importance of the differential diagnosis and elimination of all other causes of headache before resorting to external frontal sinus surgery.

Lynch, in 1924, advocated the prior treatment of antral infection and of intranasal obstruction. This was re-emphasized by Negus. Lynch, in 1929, divided indications for external sinus surgery into two classes: a) the acute manifestations of extension of infection from the sinuses to the orbits and to the brain; b) the chronic cases, those in which the pathology has resisted all other methods of treatment.

In the first class he described the surgery as "emergency" surgery. The surgery of the chronic group he called "last resort" surgery.

Negus stressed as indications for external operations: chronic sinusitis with headache, difficulty in concentration, and depression.

Evidence that the patient is threatened with a serious complication is generally considered as an indication for external frontal sinus surgery.

#### DIVISION OF MATERIAL

The cases have been classified according to whether the onset of the illness was considered to be acute, acute fulminating, chronic, or chronic in association with gross manifestations of allergic disease (see Table 1 and 2).

We have considered as an "Acute" case a patient who had no history of previous sinus infection and in whom the symptoms requiring external drainage of the frontal sinus began from two weeks to six months before examination. This class of patient usually had a normal or only slightly elevated temperature, and the white blood count was usually below 12,000. Frontal or periorbital swelling was present in five out of the 14 cases. In two cases there were signs of involvement of the central nervous system. Headache and local

tenderness were common symptoms and all had x-ray evidence of frontal sinus disease.

In those patients considered to be "Acute Fulminating," the symptoms and signs reached a climax within one week. There was marked elevation of temperature, 103° to 106° F., and the white blood count rose to 28,000. There was a rapid onset of severe headache and local tenderness. There was external frontal or periorbital swelling in all cases. There were signs of involvement of the central nervous system in 14 out of the 22 patients. There was a history of swimming prior to the onset of the symptoms in at least 50 per cent of the cases. There was definite x-ray evidence of frontal sinus disease in all the cases.

In cases considered to be "Chronic" all patients gave a history of recurring attacks of sinusitis over a period of from one to forty years. On admission there was, as a rule, a normal temperature and white blood count. Signs of central nervous system disease were found in only two out of the 15 cases. Headache and local tenderness were common symptoms. Frontal or periorbital swelling was present in seven cases, occasionally with external fistulae. There was x-ray evidence of frontal sinus disease in practically all cases. The decision to operate was based on the persistence of headache, together with frontal swelling, external fistula or local tenderness, as well as the positive x-ray findings.

In the group of cases listed as "Chronic with Allergy," the signs and symptoms were the same as in the "Chronic" group, except that headache was present in only 50 per cent of the 26 allergic cases, and periorbital swelling and tenderness were unusual. In addition, there were gross manifestations of allergy, such as recurring nasal polypi, persistent asthma, and vasomotor rhinitis, in addition to the sinus infection. In this group the decision to operate was frequently influenced by the severity and persistence of the allergic state.

The cases under review had been treated according to the usual conservative methods of astringent medication, et cetera, with the exception that some of the fulminating cases developed their signs and symptoms so rapidly that surgical intervention had to be undertaken almost immediately. In some of the chronic cases the early treatment included intranasal operations, such as turbinotomy, removal of polypi, intranasal ethmoidectomy, and submucous resection.

## TYPES OF EXTERNAL OPERATIONS

Many of the operations in this series were done by different surgeons. It is obviously impossible to say how thorough or how perfect was the technique in each case. It is fair to point out, however, that all of the operators were competent and well-trained. We have estimated that, of the 77 cases, there were 177 external operations performed on the frontal sinuses. With the exception of ablation operations, if both frontal sinuses were operated upon in the same patient these were recorded as two operations. We have restricted the terms "repeat" or "multiple" operations to those cases where the same frontal sinus was operated on more than once.

*Howarth-Lynch Operation:* A complete Howarth-Lynch operation was performed 58 times. A modification of this operation was performed 53 times.

*Trephine:* The external trephine operation was performed 37 times. In this operation the frontal sinus was opened externally without enlarging the nasofrontal duct, and by the use of a rubber tube the wound was kept open for several days or until there was evidence that the nasofrontal duct had opened. In nine cases of this series, this was the only operation necessary.

The trephine is especially valuable because it readily establishes the diagnosis when there is doubt, and, at the same time, it provides free drainage of the frontal sinus without any interference with nasal physiology. If it is the only operation required, the external scarring is negligible. If further operation is required, the trephine has been a justifiable preliminary step.

*Ablation:* Ablation operations on the frontal sinuses, either partial or complete, were carried out on 26 patients. The operation was bilateral in 12 and unilateral in 14. The operation was a partial excision of the frontal sinuses in 16 cases, and complete in ten. We have described as a partial ablation operation the removal of the anterior wall, the floor, and mucous membrane of one or both frontal sinuses, together with the ethmoid cells, and this was performed in 13 cases. In three other patients the partial ablation consisted of the removal of the anterior and posterior walls of one frontal sinus. In three cases a partial ablation operation was performed for suspected osteomyelitis, which was not confirmed at operation.

A technique of obliteration of the frontal sinuses, practiced by Doctor W. V. Cone, of the Montreal Neurological Institute, we have



referred to as a complete ablation. In this operation, all that bone which contains the frontal sinuses is removed, together with a complete exenteration of the ethmoid cells, including, if necessary, the entire roof of the orbit, and even the cribriform plate on one or both sides. This operation was performed on ten patients.

*Killian:* Only three patients of the series had had a classical Killian operation performed. Two of these were not done in our hospital.

Forty-four patients had had a single external frontal sinus operation. The remaining 33 had had multiple external operations. These numbers do not apply to intranasal operations, such as the removal of polypi—one patient had had removal of polypi on 23 occasions.

Of the 44 patients who had but one operation, 21 had had the complete Howarth-Lynch operation (Table 14).

#### ASSESSMENT OF TREATMENT

When considering the results of surgery on these 77 patients, we have had some difficulty in setting up standards of comparison. Frontal sinusitis may be a serious illness, and in the series under consideration complications occurred in 46 of the cases.

In the uncomplicated case, it is a relatively simple matter to determine whether the patient has been improved by the treatment. One is dealing only with the sinusitis. However, this is an oversimplification because none of these patients have infection confined to one frontal sinus, with all other accessory nasal sinuses remaining normal. In many of these cases there is a bilateral involvement. In trying to assess the value of treatment in a patient suffering from frontal sinusitis, the treatment may have cured the frontal sinusitis, yet the patient may not notice the improvement if one or more of the other sinuses remain infected. This was a difficulty which became very apparent in trying to analyze the answers which we received from our questionnaire.

So far we have considered only the difficulties that arise because there are a number of sinuses. There are even greater difficulties because of the nature of the complications, e.g., a patient who had cerebral venous thrombosis or brain abscess, from which he recovered, may be left with epilepsy. Such a patient might be cured of his initial sinusitis, but he is still suffering from a complication of the sinusitis: recurring seizures.

In the cases considered under the heading, "Chronic with Allergy," the confusion is more marked because of the tendency for nasal polypi to recur, because of the variability of vasomotor rhinitis and because of the persistence of asthma. All of these conditions may frequently be resistant to any known form of treatment.

In trying to determine as accurately as possible the results of treatment of the initial sinus infection, we have listed as improved a patient who died from asthma six years after having had a bilateral modified Howarth-Lynch operation. One year after her operation, the nasofrontal ducts were patent, and subsequent examinations showed the anterior nares to be free from discharge. There was evidence of purulent discharge in the sphenoids. Six years following external operation there had been no gross recurrence of polypi, whereas prior to her external operation she had had six intranasal operations for removal of polypi. To further emphasize the difficulties of arriving at a final assessment, when this patient died of asthma the report of the post-mortem examination mentioned disease in the sphenoids and the ethmoid cells, but the reporter failed to mention anything about the condition of the frontal sinuses.

Another important question that was raised, when assessing the results, is the matter of how long should the symptom-free period be after treatment before one can talk about a cure. Some of the patients in this series have been under observation for over thirty years. One patient had had a Killian operation in 1902, and was well until 1942. Forty years later she had a recurrence of infection in that frontal sinus. The result following the original operation might be called a cure. In 1942 the same frontal sinus was re-opened by the Howarth-Lynch method, and she has had no further trouble for 12 years, again a cure by most standards.

We are impressed with Goodale's statement of 1942: "If the operation has saved a person's life, his health is improved, and he is able to return to his normal life even for a limited period of time, we cannot say that the operation has been a failure or that there has been no improvement." Yet, as Goodale states, "when the frontal sinus again becomes the site of infection requiring re-operation, it is natural to feel that the first operation has failed."

When considering external frontal sinus surgery, Ferris Smith,<sup>18</sup> in 1933, discussed the question of a cure following an external operation on the sinuses. He defined cure as restoration to normal. He

said that such could not always be attained, but it could be approximated. He regarded as a clinical cure the thorough removal of all pathological conditions with a minimal disturbance of function and appearance. He further pointed out that the proper attack on all diseased tissue had been termed radical, and that this is an unfortunate term.

#### CLASSIFICATION OF RESULTS

The accepted standard of cure, for example, in cancer surgery, is a five-year symptom-free period. Instead of attempting to consider our material on this five-year basis we have grouped our symptom-free patients in five-year periods (Tables 3 and 4). Our conclusions are based on the results of the frontal sinus infection insofar as the case can be assessed, regardless of the results of the more remote complications. We have found it almost impossible to assess the results of treatment of a specific frontal sinusitis without taking into consideration the general state of the nose, as may be found in chronic vasomotor rhinitis or hypertrophic rhinitis. In such patients, when there was a doubt as to the results, the case was listed as improved or not improved (Table 5).

Under "improved" were listed all those cases in which the evidence was not sufficient to justify listing them in the symptom-free class. Yet, in some of these cases there were no definite signs of recurrence or persistence of frontal sinusitis.

Referring to Table 4, it is interesting to note that in this series of 77 patients, 11 have been classed as symptom-free for five years or under, and ten of these were acute or acute fulminating cases. There were 24 patients who have been classed as symptom-free from five to thirty years, including 18 in the acute or acute fulminating group. It might be stressed that of the 41 chronic and chronic allergic patients, only seven were symptom-free, and of these six were in the 5 to 30-year class.

*Group One: Acute Frontal Sinusitis.* In this group of 14 patients, the majority were under 21 years of age. The symptoms and reasons for external operations have previously been described. Ten of the patients in the group had only one external operation. Two of these were complete ablations. Five of the single operations and the primary operation of three of the multiple operations were trephines. There were four patients with multiple operations. In two

of these the final operation was a partial ablation, and in the other two, modified Howarth-Lynch operations were performed.

Four of the five ablations were performed because of osteomyelitis. In the fifth, the frontal sinus extended beyond the midline. There had been a subperiosteal abscess, and a modified Howarth-Lynch operation had not controlled the symptoms. In addition to the four patients with osteomyelitis, one had meningitis and one had cerebral venous thrombosis. Two of the patients with osteomyelitis had brain abscess. Eleven of the 14 patients in this group were symptom-free from one to 15 years (Table 4). One was improved, and two were not traced.

The predominating organisms in this group were the Staphylococcus and the Streptococcus hemolyticus, each of which occurred in five patients. Other organisms were *H. influenzae*, Micrococci, and Proteus. In one case, *B. Freidlander* occurred as a later complication.

All but one of the cases were in the chemotherapy era, and received what we considered to be adequate treatment. This may have been a contributing factor in the results. That it was not the all important factor is shown by comparing the results in this group with those in the chronic allergic group, in which many of the patients had had adequate chemotherapy, but in which the results were generally poor.

*Group Two: Acute Fulminating Sinusitis.* The majority of the 22 patients in this group were under 14 years of age. Only five of the patients were females. Three-quarters of the patients were operated upon within two weeks of the onset of symptoms. One-third of the cases occurred in the months prior to July, one-third in the months after July, and one-third during the month of July. The infecting organism was the Staphylococcus in 13 out of the 22 cases.

Chemotherapy does not appear to have altered the incidence or the pattern of complications in this type of case (Tables 6 and 7). Chemotherapy has, however, had a beneficial effect upon the results of the treatment of the complications. Complications followed in cases in which treatment had been most prompt and also followed various kinds of operations. Twelve of the patients had single operations. In seven of the ten cases which had multiple operations, the original operation was a trephine. Seven of the final operations were ablations. The results have not favored any one type of operation.



The type and virility of the infecting organisms and the state of the host must be important factors in determining the pattern of the disease. Eight cases occurred before the advent of chemotherapy and 14 after. In 21 of the 22 cases there were serious complications, eleven having had osteomyelitis (Tables 10 and 11) and intracranial complications. Seven other cases had subperiosteal abscess with fistula. In spite of this, 17 of the group are reported as symptom-free. There were only two deaths. Two were not traced.

*Group Three: Chronic Frontal Sinusitis.* In the chronic group there were 15 patients. The average age was over 25 years. The history of symptoms was present from one to 40 years, with an average of about eight years. Four of the patients had but one external operation. In the other 11 cases, the number of operations ranged from two to seven.

Of the original operations, there were three complete Howarth-Lynch operations, six modified Howarth-Lynch operations, two Killian operations, three trephines, and one partial ablation.

The final operation in five cases was a partial ablation. There was a complete Howarth-Lynch operation in one case and a modified Howarth-Lynch operation in three cases and a complete ablation in two cases.

Osteomyelitis occurred seven times and subperiosteal abscess twice (Tables 10 and 11). Postoperative meningitis occurred in one patient before the advent of chemotherapy. That patient died four days after operation. A second patient with osteomyelitis and a subdural abscess died in spite of adequate chemotherapy. He had had five external operations in the course of about two months prior to his death. The last operation was a complete ablation and preceded his death by only a few days. A third patient, who also had adequate chemotherapy, died about ten days after a complete ablation from osteomyelitis, meningitis, and brain abscess.

In this group there were only three patients classified as symptom-free. This is in marked contrast to the results in the acute and acute fulminating groups (Table 3). There were more complications in this group than in the acute group, but fewer than were present in the acute fulminating group.

We were impressed by the fact that this was an older group of patients in whom the infection had lasted for a long time. Eleven

of the 15 patients had two or more external operations besides intranasal operations.

We have not been able to find a satisfactory explanation for the failure of the first operations in these cases, unless it be that it was due to lack of completeness in the technique. Only in three of the 15 operations was a complete Howarth-Lynch operation done, but one of the 15 was a partial ablation operation, which resulted in the patient being symptom-free.

Of the four patients who each had a single operation, one died from meningitis, as mentioned above. One other case had a complete Howarth-Lynch operation, and has been classified as improved for 15 years. Another had a partial ablation because of osteomyelitis, and has been symptom-free for 17 years. The fourth had a modified Howarth-Lynch operation, and has been improved for nine years.

In this group was one patient, already referred to, who had an original Killian operation. She was symptom-free for forty years before she had a recurrence.

The majority of these patients required several operations, and the results over the years have not been encouraging.

The bacteriology in this group showed more variety than did that of the acute group. In most cases there was a multiplicity of organisms. Staphylococci were present in five cases, Streptococci were present in six cases, pneumococci were present in three cases, H. influenzae in two cases, diphtheroids in three cases, and Micrococci in three cases. In addition to these, there were Proteus, B. coli, and E. coli. In only one case was there no growth, and another was not reported.

There were eight cases in the chemotherapy era, and seven were judged to have had adequate dosage (Table 12). There does not appear to be any obvious correlation between the type of organisms and those conditions which responded favorably to one operation, or between the infecting organisms and the conditions which were ultimately controlled by chemotherapy and multiple operations.

*Group Four: Chronic Frontal Sinusitis with Allergy.* The average age of the patients in this group was over 25 years. The duration of symptoms, prior to the primary external operation, ranged from one to thirty years, the average being over ten years. Four-

teen of the patients had asthma, 21 had nasal polypi. These conditions were alone or in combination, and several were complicated with vasomotor rhinitis and hay fever. In two patients who have been symptom-free for 17 and 18 years following complete Howarth-Lynch operations, for some unknown reason, in both, the polypi have not recurred, but in one the asthma has persisted.

This has been the most difficult group of all in which to judge results. This may be due to the fact that neither the surgeon, the allergist, nor the patient can adequately distinguish between the results of the sinus treatment on the one hand and the results of the allergic management on the other.

The majority of these patients had been under the constant care of competent allergists, and yet in spite of this, in 15 of the 21 cases traced the allergy had not been arrested. The allergic management consisted of bacteriological studies, the use of vaccines, the elimination of, and desensitization to, known sensitivities.

Many of the sinus operations in this group were carried out with a view to aiding the control of bacterial sensitivities. Bacteriological studies revealed the presence of multiple organisms in most cases, with Staphylococci, Streptococci, and pneumococci predominating. The *H. influenzae*, Micrococci, *Neisseria catarrhalis*, and *Proteus* occurred frequently.

The great majority of the patients had multiple intranasal operations, chiefly for the removal of polypi. We know of only three patients in whom polypi did not return, after external operation, for six, 17, and 18 years. Eighteen of these patients had a single external operation on the frontal sinus. Eight patients had multiple external operations. The repeat operations were performed within a year of the original operation in three cases. In five, the second operation followed the original operation by from five to ten years. Tantalum tubes were inserted in two cases, and in both instances the tube had to be removed after two or three months. Tantalum foil was inserted in three cases. In one it was accidentally removed after two months, and it was necessary to obliterate the sinus. In two other patients the foil remained in position for six years, and though the patients are classed as not improved, the ducts can be probed.

There were three cases of osteomyelitis, three cases of cerebral venous thrombosis. One patient had meningitis in conjunction with

osteomyelitis. Three patients had subperiosteal abscess, one in conjunction with cerebral venous thrombosis. In the patient who had meningitis, the organism was a pneumococcus type 3, and the infection responded to sulphapyridine. There were three cases with cerebral venous thrombosis, of which two were in the prechemotherapy era, and of these one died. The third case, which was in the chemotherapy era, survived four years after the thrombosis. Four patients in this group died. In two, the deaths were directly attributable to asthma. One patient died from intracranial complications. The cause of death in the fourth case was unknown.

Four patients were classified as symptom-free, eight as improved, and eight were regarded as unimproved. Five of the patients could not be traced. For relation to chemotherapy, see Table 13.

Twenty of the 26 primary operations performed in these cases were complete Howarth-Lynch operations. This represents a higher percentage of complete primary operations than were performed in any of the other three groups. In spite of this, and in spite of the fact that only eight of the cases required repeat operations, the results as recorded by us have been less satisfactory in this class of case than in those in the other groups. This may be largely due to the special difficulty of assessment in this group with allergy. We suspect that the actual results of treatment of the frontal sinusitis may be better than our records indicate.

#### CHEMOTHERAPY

Twenty-eight of the 77 patients in this series were treated prior to 1938. This date was arbitrarily chosen to mark the advent of modern chemotherapy, because sulphanilamide was used in only three patients of the series in 1937, and the largest dose was 45 gr per day for seven days. From 1938 on, the sulphonamides, and later the antibiotics, were used adequately in the presence of serious infections. Due attention was paid to the determination of bacteriology, and as early as possible the choice of drugs was checked with the organism's known sensitivities.

When the 28 cases treated prior to 1938 were compared with the 49 cases treated after 1937, we found that there was no appreciable difference in the incidence of complications between the groups. In sixty per cent of the cases in each period there were complications (Table 7).



The incidence of the acute fulminating cases in the two periods was also practically the same. There were eight cases out of 28 prior to 1938, as compared to 14 cases out of 49 after 1937, representing in each period 28 per cent of the total.

No comparisons can be made in the acute cases. There was only one acute case in this series which occurred prior to 1938, and the patient has not been recently examined.

When the results of treatment of the chronic cases are compared, there is no striking difference in relation to chemotherapy (Tables 6, 10, 11 and 12).

Of the 26 patients in the chronic allergic group, there were 12 prior to 1938, and 14 after 1937. Of the cases that have been traced, about 50 per cent of those occurring in each period have been regarded as improved (Table 13).

In considering chemotherapy in relation to chronic frontal sinusitis with allergy, our analysis has shown that in 21 of the 26 patients, the treatment was carried over into, or was begun in, the chemotherapy era. Five of the patients were not traced. Of the 21 patients in the chemotherapy era, only 14 received adequate chemotherapy. The over-all results in this group have not been encouraging in spite of careful anti-allergic management, radical and fairly thorough surgical treatment, and adequate chemotherapy in the majority of cases.

One might be justified in concluding that the results in this group of cases illustrate what is generally recognized as being true—that sinus infection and allergic manifestations are so intimately connected that their treatment must be considered conjointly, and better results cannot be looked for until there is a more effective anti-allergic treatment available.

#### IMPORTANCE OF THE NASOFRONTAL DUCT

In spite of the widespread use of the Howarth-Lynch operation and the various modifications that have been suggested, there are still many unimproved cases. Goodale reported 30 per cent and Harris 20 per cent of failures in their respective series of cases. It was because of poor results that Goodale, Harris, and Scharfe each recommended the use of tantalum either as foil or a tube in the nasofrontal duct.

It is rather interesting to note that such men as Lynch, Howarth, Ferris Smith, and Negus have not emphasized or stressed the difficulty

of maintaining the patency of the nasofrontal duct, even though they have described methods designed to maintain an open duct.

We have been impressed with Hajek's statement that attention to adequate patency of the nasofrontal duct once or twice yearly should be considered of more importance than any other factor.

Of the 14 cases in the acute group in this series, information on the present state of the duct was available in six. In two, the primary operation had been a trephine. In one of these the duct was closed at the end of six months, and in the other after four years, and both required reoperation. In one case the duct was closed twelve years after a complete Howarth-Lynch operation. Closure followed in two cases with a modified Howarth-Lynch operation. One, in which a rubber tube had been inserted for two months, was closed three months later and required reoperation. In the other case a porex tube was in the duct for six months, and two weeks after its removal the duct could not be probed. The patient has remained symptom-free for six months.

In another of these patients, following a complete ablation of the sinuses, with the preservation of the middle turbinates, it was not possible to probe the ducts seven years after operation. There was no evidence of recurrence of sinusitis in this case, and the patient has remained symptom-free. This illustrates how difficult it is to establish with certainty the patency of the nasofrontal duct, even in normal individuals, because the duct cannot always be probed.

In the 22 fulminating cases, most of the repeat operations were performed because of complications, and in only two of the group do we have any late information about the state of the duct.

In one case the duct was found to be closed ten years after a complete Howarth-Lynch operation, and required reoperation. In the second case the duct could not be probed eleven years after a modified Howarth-Lynch operation, but the patient was symptom-free and there was no evidence of sinusitis.

Of 15 patients in the chronic group, something about the recent state of the duct was known in six. In three of these there had been repeated operations, because the symptoms persisted. All three patients eventually required an ablation operation, and in one of these even following a most extensive operation, tantalum foil had to be

inserted in the duct at a later date. We have no adequate explanation for the persistence of the infection in these three cases, but there were severe complications in all three.

In one case the duct was known to have remained patent for 12 years after a mucosal flap had been used. In seven cases of the series, a mucosal flap, as described by Sewall, was turned up from the middle turbinate into the frontal sinus. Of these latter cases, in four we have no recent information regarding the state of the ducts. One was a failure and a tantalum tube was inserted later. In the two remaining cases the results were satisfactory.

In another case, nine years after a modified Howarth-Lynch operation, the duct could not be probed, but there was no clinical evidence of infection in the sinuses.

We have information about the recent state of the duct in 11 of the 26 patients in the chronic with allergy group.

In one patient, who had had a mucosal flap placed in one duct in the course of a bilateral complete Howarth-Lynch operation, there was recurrence of infection one year later in the sinus which had not had a mucosal flap. The duct on the side of the mucosal flap was patent. The reinfected sinus was reoperated upon and has remained well for 16 years in spite of the fact that a flap was not inserted at the second operation. The present state of the ducts is unknown.

In another case after a bilateral complete Howarth-Lynch operation, the ducts could be probed three years later, but there was pus present and there was periorbital swelling. Another case required ablation one year following a bilateral complete Howarth-Lynch operation because of osteomyelitis in the presence of closed ducts. Another patient had had a Killian operation in 1936 and required reoperation in 1942—a modified Howarth-Lynch operation. The duct remained open for one year. However, when seen in 1950, the duct was closed, and in 1954 the duct was still closed and there was a fluid level in the sinus. In one patient the ducts were found to be closed and reoperation was necessary 19 years after a trephine operation.

Three patients had had a skin graft in the nasofrontal duct, as advocated by Ferris Smith, Howarth, and Negus. Of these, two are dead, but in one of these, a chronic allergic case, the nasofrontal ducts were known to be open for several years after operation.

Tantalum foil was inserted in the duct in five patients and a tantalum tube was inserted in five other patients of the series. One of the allergic patients had the tantalum foil accidentally removed after three months, and the sinus ultimately had to be collapsed. In two other allergic patients the duct has remained patent in spite of the fact that recurring polypi have been removed. Another patient is well, and a fifth has reported by letter that she is not well. The actual state of the duct is not known. Of the patients with tantalum tubes, five in number, one has not been traced; in two the tubes had to be removed after about two months, because of pain and swelling. The two remaining patients are apparently well, but the actual condition of the ducts is not known.

In two patients with allergy, a lucite rod was inserted, this being retained in position for several months. The patients were taught how to change the rod. In one, a rod was kept in position for six months, and in the other the rod was kept in position for two months. In each of these two cases, two weeks after removal of the rod the nasofrontal duct could not again be probed. One would think that the ducts should have epithelized at least within the period of six months.

In the majority of the patients operated upon in our series, exclusive of those having had the trephine operation, a tube was inserted into the nose from the frontal sinus. In the great majority, this consisted of a hard rubber tube kept in position for about two weeks.

In our experience, the crux of external sinus surgery is the establishment of a patent nasofrontal duct. We have not been convinced that any type of operation, no matter how completely it is performed, can be counted upon to re-establish a patent nasofrontal duct in all cases.

The number of cases in this series in which a mucosal flap was used or in which a skin graft was inserted is not sufficient to allow a true assessment of their values. Tubes, whether rubber, metal, or plastic will not invariably maintain the patency of the duct.

The state of the nasofrontal ducts in this series was definitely known in only 25 of the 77 cases, although many of the other patients are known to be symptom-free or improved. In 16 of the 25 cases, the ducts were considered to be closed at varying lengths of



time after the primary operation, from a few months to forty years. In three other cases the ducts could not be probed, but there were no signs of sinusitis, and the patients are symptom-free.

In only six out of the 77 patients were the ducts known to be open. In two of these, there was tantalum foil in the ducts, and these ducts could be probed after six years in spite of recurring polypi in each case. In one case, skin-grafted ducts remained open for six years. In two cases with mucosal flaps, one was known to remain open for a year and one for 12 years. In the sixth and last case, the ducts could be probed at the end of three years, but pus was present and could be irrigated from the sinuses.

#### COMPLICATIONS

Under the heading of "complications" we refer to those major conditions which have been directly attributable to the inflammation in the frontal sinuses. In this series of 77 patients, there were 46 with severe complications (Table 7).

In the acute and acute fulminating groups combined, there were 36 patients, 28 of whom had serious complications. All but one of the 22 acute fulminating cases were complicated. Of the 41 chronic and chronic allergic cases, only 18 had serious complications, and ten of these were in the chronic group.

In spite of the fact that 28, or 60 per cent, of the severe complications occurred in the acute and acute fulminating groups, it was in these same groups that we found the highest number of symptom-free patients, 28 cases, or 77 per cent. On the other hand, in the chronic and chronic allergic groups, whereas only 18, or 40 per cent, had serious complications, only six, or 14 per cent, of the cases in these groups were symptom-free. It would appear from this series that, although the great majority of serious complications occur in acute frontal sinusitis, it is in acute frontal sinusitis that one may hope to obtain the best local results. In chronic disease there is not so much likelihood of a severe complication, but there is a much less likelihood of a satisfactory local result from treatment.

#### OSTEOMYELITIS

The use of the term, osteomyelitis, has given us cause for considerable thought, because we were in doubt as to whether or not we should include under this heading the development of a fistula



Fig. 1.—This 12-year-old girl had acute frontal and maxillary sinusitis. The organism was *Streptococcus hemolyticus*. The symptoms had persisted for one month prior to admission to hospital in 1931. She had a sub-periosteal abscess and was treated by trephine and intra-nasal antrotomy. One month later she again developed swelling above the eye and required a modified Howarth-Lynch operation.

through the floor of the frontal sinus. We decided to include only those patients who were shown at operation to have an osteomyelitis of the anterior or posterior frontal sinus walls or disease located elsewhere in the skull bones. In addition to these cases of osteomyelitis, there were 11 other cases that did not have osteomyelitis but did have fistulae and subperiosteal abscesses. There was frontal and periorbital swelling in all cases of acute fulminating sinusitis, in eight of the 14 acute cases, in eight of the 15 chronic cases, and in six of the 26 chronic allergic cases (Figs. 1, 2 and 3). We have excluded sub-

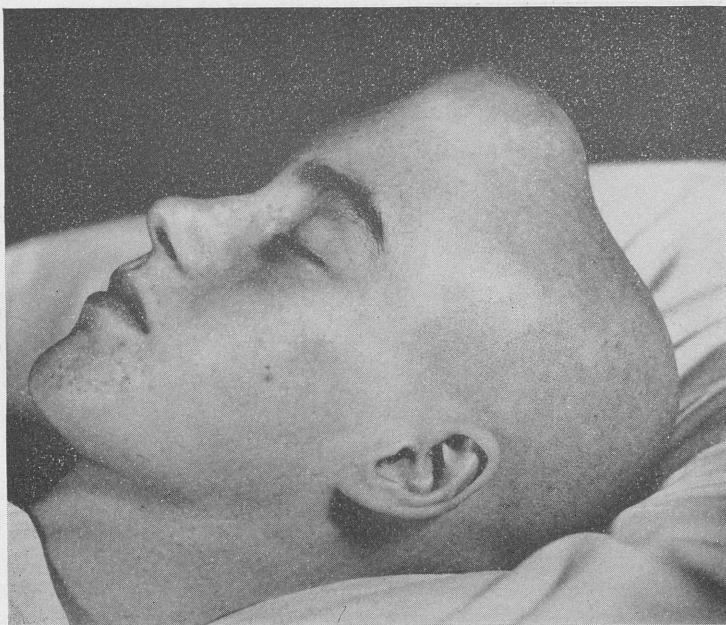


Fig. 2.—This 15-year-old girl, classified as a case of acute sinusitis, was admitted to hospital in October, 1947, with a history of bilateral frontal and antral infection for two months. She had severe headache, and had developed swelling over the frontal bone in the midline during the week previous to admission. She had extensive osteomyelitis of the frontal bone, a large sub-periosteal abscess, and a cerebral abscess in the left frontal lobe. She had an ablation operation without removal of the middle turbinates, extensive removal of diseased frontal bone, and drainage of frontal abscess, with adequate chemotherapy. She has since had epileptic seizures, but she is symptom-free as regards the frontal sinusitis and osteomyelitis.

periosteal abscess or fistula if it occurred in a patient who had a previous Killian operation.

There were 25 cases diagnosed as osteomyelitis. Twelve were found to have a subperiosteal abscess at the time of the first operation, and in nine of these there was a fistula leading into the frontal sinus. In three of the 25 cases there were no periorbital abscesses or fistulae, and in ten others we had not sufficient information about the original operations to know whether there were subperiosteal abscesses or fistulae present.



Fig. 3.—This 18-year-old boy was admitted to hospital in 1942, with acute fulminating sinusitis, and his symptoms had persisted for two weeks prior to admission. He had unilateral pansinusitis, peri-orbital abscess, and retrobulbar abscess. He was treated by complete Howarth-Lynch operation, with adequate chemotherapy. This picture was taken a few hours after operation. He remained well for ten years, when he again had swelling about the eye, and had a tantalum tube placed in the naso-frontal duct.

The osteomyelitis was contiguous to the affected frontal sinus in all but one case. In this instance the bone disease was fronto-parietal, and there was a subperiosteal collection of pus in the region of both frontal sinuses at the time of the original operation.

In the 25 cases of osteomyelitis, the *Staphylococcus* organism was found in pure culture in 14 cases and in combination with other organisms in three cases.

In ten of the cases of osteomyelitis there were no further complications, but in the remaining 15 there were additional complications, mostly intracranial (Table 7).

In assessing the results of treatment in these 25 patients, it was found that 15 were classed as symptom-free. In two patients there has been no recent follow-up. It was rather interesting to find that,



whereas nine cases were treated in the prechemotherapy days and seven were symptom-free, there were 16 treated during the chemotherapy era, and of these only eight were symptom-free.

In reference to these patients being symptom-free, we have assessed the state of the frontal sinuses in addition to the osteomyelitis. As already stated, 15 of the 25 patients had, in addition to the osteomyelitis, intracranial complications, and in the majority of these there were sequelae, such as focal seizures. These have been disregarded in referring to the results.

Of the 25 patients with osteomyelitis, three are dead following intracranial complications. They received what was considered to be adequate chemotherapy, but treatment may have been instituted too late.

Of the 22 remaining cases, in one in which the osteomyelitis was not contiguous to the frontal sinus the infection in the sinus responded to a trephine. Another case, in which the osteomyelitis was limited to the anterior wall of the frontal sinus, responded to a modified Howarth-Lynch operation with partial removal of the anterior wall of the affected sinus. Twenty-three of these patients had ablation operations on the frontal sinuses. Seventeen of these patients had multiple operations. Of the eight patients who each had a single operation, the operation was an ablation in six. Of these eight cases, seven have been symptom-free from two to 17 years, and one has not been traced. The average of these symptom-free periods was over ten years.

Of the 17 patients who had multiple operations, the original operation was a trephine in six cases, a Killian in one, a modified Howarth-Lynch operation in seven cases. Three patients had the complete Howarth-Lynch operation, and in one of these, a chronic allergic case, the osteomyelitis was directly postoperative and was associated with meningitis. In a second case, which was acute and fulminating, the complete Howarth-Lynch operation was carried out too late, since there were already signs of extensive osteomyelitis of the frontal bone. In the third case, also belonging to the chronic allergic group, the osteomyelitis came on about six months after the original operation and the nasofrontal ducts were found to be closed.

Four of the 25 patients with osteomyelitis belonged in the acute group, 11 in the acute fulminating group, seven in the chronic group,

and three in the chronic allergic group. There was a total of 15 cases in the acute and acute fulminating groups, and 13 have remained symptom-free for an average period of 12 years, one was not traced, and one died from overwhelming intracranial infection.

There were ten cases with osteomyelitis in the chronic and chronic allergic groups. One has not been traced and two are dead. The remaining seven cases have been observed for an average period of eight years, and they all have apparently recovered from the osteomyelitis; only two are classed as symptom-free as regards the sinusitis.

All but two of 25 patients with osteomyelitis had ablation operations on the frontal sinuses, together with complete removal of the bone adjacent to the osteomyelitic foci. Fifty per cent of these ablation operations might be described as extreme. This calls to mind the radical treatment of osteomyelitis of the bones of the skull advocated by Mosher, who stressed the importance of early and widespread removal of the bone well beyond the obviously diseased area.

In our analysis of these cases, the osteomyelitis that occurred in association with acute sinusitis did not behave differently from that associated with chronic sinusitis. All the patients who are still alive have recovered from their osteomyelitis. However, in some cases with chronic sinusitis there may be low-grade osteomyelitis, as illustrated by one of our patients who died of brain abscess and meningitis. She was not included in our series as a case of osteomyelitis because there was no description of osteomyelitis having been found at operation. At autopsy, a sequestrum was found in the posterior wall of one frontal sinus.

#### RESULTS

*Ablation Operations.* There were ten complete ablation operations carried out for osteomyelitis. In three instances the operation was terminal because the patients were moribund. Six patients are symptom-free and one is improved. The operation had been carried out as a single and a preliminary operation in four cases, in which there was evidence of osteomyelitis and brain abscess in each instance, in addition to the presence of the frontal sinus disease. It followed one or more previous external operations upon the frontal sinuses in six others.

In the 13 remaining cases, a partial ablation of the frontal sinus had been carried out. In two, in which the partial ablation opera-

tion was carried out as the only external operation, there were signs of osteomyelitis when the patients were first seen, and both of these patients have been symptom-free for six and 17 years.

The remaining eleven partial ablation operations followed one or more trephines, modified or complete Howarth-Lynch operations, and six of the patients are symptom-free.

*Trephine Operations.* In the series under discussion, 22 patients had the trephine operation. The trephine was successful in four acute cases, and in three other acute cases subsequent operations were necessary, in one case because of osteomyelitis. In the fulminating group the trephine was successful in four cases, but in seven other cases subsequent operations were necessary because of such serious complications as osteomyelitis, brain abscess, and cerebral venous thrombosis. In the chronic group, only three trephines were done and all required other operations, in one case for osteomyelitis and meningitis, and in another for subperiosteal abscess.

In the chronic allergic group only one trephine operation was performed, and it was successful in spite of the presence of a subperiosteal abscess and cerebral venous thrombosis.

The trephine operation was successful in nine cases, and failed in 13 other cases chiefly because of associated sinus complications.

The intracranial complications were mostly multiple, with or without osteomyelitis. These complications, for the most part, were treated in the Montreal Neurological Institute. The details of their treatment is beyond the scope of this presentation. The number is probably unusually high because so many such cases are referred to the Montreal Neurological Institute.

*Brain Abscess.* Brain abscess occurred 11 times. In nine instances it was in association with osteomyelitis, and twice it occurred with meningitis. Four cases occurred in the prechemotherapy era, and of these one died. Seven were in the chemotherapy era, and of these two died. The brain abscess always involved the frontal lobe; it was on the ipsilateral side, and was therefore contiguous.

*Cerebral Venous Thrombosis.* In nine patients a diagnosis of cerebral venous thrombosis was made. In one, it was in association with brain abscess and osteomyelitis, and was confirmed at post-mortem. In most cases it was a presumptive diagnosis, because, in

spite of symptoms and signs of a brain abscess, it was not possible to show the presence of a space-occupying lesion.<sup>19</sup>

*Sub-dural Abscess.* Sub-dural abscess was found in five patients, always in association with osteomyelitis and sometimes with another intracranial complication. Death was directly attributable to sub-dural abscess in one instance.

*Meningitis.* Meningitis occurred in six patients. In each instance the organisms were found in the cerebrospinal fluid. In two cases the meningitis was the only complication. In two other cases the meningitis was regarded as an immediate postoperative complication. Of the six cases, two occurred in the prechemotherapy era, and the patients died. Of the four cases which occurred in the chemotherapy era, three survived, including one of the postoperative complications referred to above. This was due to the pneumococcus type 3, and was the first case of type 3 pneumococcal meningitis to recover in this hospital. She was treated with sulphapyridine in 1939.

Of these patients with intracranial complications, the great majority developed sequelae, such as epileptiform seizures.

#### IMPRESSIONS

It is difficult to draw conclusions from a small series of cases, but we have formed certain impressions from this study.

Complications were more frequent in the acute cases of frontal sinusitis, but the greatest amount of local recovery occurred in these cases. Conversely, complications were less common in chronic frontal sinusitis, but satisfactory improvement in the local condition occurred less frequently. We are of the opinion that in the chronic allergic group the prevalence of poor results was due largely to the fact that the allergy had not been controlled in spite of adequate management.

We have been unable to demonstrate any striking difference in the incidence or type of complication in frontal sinusitis in relation to chemotherapy, nor have we noted any striking difference in the results of treatment either of acute or chronic sinusitis before and after the advent of chemotherapy, except in the treatment of meningitis.



We believe that the judicious use of adequate chemotherapy has been a more valuable adjunct to surgery in the treatment of acute and chronic sinusitis than the above finding would appear to indicate.

In this study it was found that the more complete the initial operation, the less likelihood was there of repeated operations on the frontal sinus.

Twenty-three of twenty-five cases of frontal sinusitis with osteomyelitis required an ablation operation on the frontal sinuses.

In this series of cases of frontal sinusitis the osteomyelitis was associated with serious intracranial complications in 50 per cent.

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### DISCUSSION

DR. W. LIKELY SIMPSON: Mr. President and Fellows: At the last annual meeting of the Academy a report was made by me on 325 external ethmo-spheno-frontal sinus operations on 201 patients. These were my own private patients, all being done by myself.

I would like to emphasize that in acute cases only drainage is indicated. Do not do complete operations on acute fulminating sinusitis. In cases of acute frontal sinusitis with osteomyelitis of the skull, open the osteomyelitic area, take out all the bad bone, drain the subdural or brain abscess and do an external drainage of one or both frontal sinuses. Complete surgery should not be done in an acute frontal sinusitis.

I will just mention, Mr. President, the complications on these cases of my own.

(1). Orbital hemorrhage from retraction of the anterior ethmoid vessels which is usually controlled by packing.

(2). Internal carotid injury. I have had two such cases doing the exenteration of the sphenoid at the time of the external frontal operation and both were controlled by packing and both did nicely.

(3). I had no osteomyelitis of the skull as a result of the external-ethmo-spheno-frontal sinus operation.

(4). Double vision. Any surgeon doing this type of work knows there is a small percentage of patients which have some double vision. I had five patients with permanent double vision. One of these was corrected by an operation, one by prisms, the other three persisted.

(5). I had one ptosis of the upper lid, which to me is hard to explain.

(6). Optic atrophy. There was one case of optic atrophy.

(7). Enophthalmos. In very large frontal sinuses occasionally there may be moderate enophthalmos.

DR. ANTONIO BERGARA: Mr. President, Members and Guests: I wish to congratulate Drs. Stuart and McNally for their presentation and as a contribution to their paper I should like to say what we do in our country, Argentina.

When we have a frontal sinus, especially a large one that requires an external operation, we keep in mind three points: 1, the treatment of the disease itself; 2, the way of avoiding relapses; and 3, the cosmetic results.

To achieve the first we deem necessary a wide opening through the anterior wall of the frontal sinus, to remove carefully all the mucous membrane, and the resection of the anterior ethmoid cells.

The only way to avoid relapses is by the obliteration of the cavity. To do so we fill up the sinus with fat tissue taken from the abdominal wall of the patient or from a bank of adipose tissue.

This auto or homograft attaches firmly to the bony walls, completely fills up the sinus and its extensions and finally undergoes a partial fibrosis or bony transformation.

This manner of obliterating the sinus is based on experiments performed on the frontal sinuses of dogs, the results of which we presented to the Argentina Society of Otolaryngology and published in the "Revista Argentina de Oto-rinolaringologia," third part of the year 1951.

The results of the first cases of implantation of fat tissue within a human frontal sinus were presented to the Second Pan-American Congress of Otolaryngology held in Montevideo, Uruguay, in January 1950.

We obtain good cosmetic results with a personal technique that we have performed successfully since 1934, which consists of an osteoperiosteal flap made on the anterior wall of the frontal sinus.

If the Chair will permit me, I shall show you some slides of a dog operated on in both frontal sinuses at the same time, but in one side we did an autograft of fresh fat tissue and in the other a transplantation of boiled fat. The autopsy was done three months after operation.

You will see in the slide a frontal section in which both transplants fill up the cavity and are firmly attached to the bony walls. (Slide) The autograft underwent a little fibrous transformation but appears as normal fat tissue. The boiled fat tissue underwent almost complete bony transformation. That was more evident when we took out the transplants for a histopathological test as is seen in the slides.

I imagine that you will say, "Well, there is a difference between the dog sinus and a human sinus with a chronic suppurative sinusitis." The first is a healthy cavity, but the second is a septic one."

All of us know that the adipose tissue is an easy prey to purulent infections. Then, what is the behavior of the fat tissue transplanted in a septic sinus? The answer is given by many cases operated on with good results and illustrated in the following case:

In July 1949, a man, 46 years old, had a chronic fronto-ethmoidal sinusitis on the left side with an external frontal pneumatocele on the same side. The x-ray examination showed the right frontal sinus suspected of infection. We performed on the left side our technique with an autograft of fat tissue taken from the abdominal wall of the patient. We obtained a good result, both therapeutically and cosmetically.

Four years later the suspected right frontal sinus became an evident suppurative sinusitis. Therefore we performed an external operation and found the inter-

sinusoidal septum partially gone. We enlarged the opening and through the hole we did a biopsy of the fat tissue transplant within the left frontal sinus done four years before. Macroscopically the autograft appeared well vitalized, filling up the cavity and firmly attached to the bony walls of the sinus.

Furthermore, in spite of its contact with the infection of the right frontal sinus, it did not seem to be infected.

The result of the histopathological test may be appreciated through the following microphotograph. (Slide) This is a microphotograph of the biopsy taken from the left side of the graft done four years before. You see the fatty tissue is preserved and on this side there is a partial bony transformation, on this side fibrous.

I think that we have a method that we may suggest for operations on the frontal sinus in cases that are not complicated.

Dr. WILLIAM J. McNALLY: Mr. Chairman and Gentlemen, we would like to thank the discussers and are sorry there was not more time for discussion. We are quite impressed by Dr. Simpson's large number of cases, particularly in relation to our very small series. We dared to present this small number because the cases have been observed over a long time and we thought it was probably fairly typical of the number that would go through an average clinic.

We have been impressed by the difference of opinion among the authorities in the literature. That very difference of opinion bespeaks the fact that the solution is not yet available and certainly we are in no position to offer to you the solution today.

I would also like to thank the Doctor from Argentina for his suggestion about fat implantation. We have had no experience with it but we shall certainly follow the results with great interest.

Dr. Simpson emphasized a point that in an early case of acute frontal sinusitis one would not do an extensive operation, and with that we heartily agree. There have been extensive operations described in this series but there was one case in which there was definite evidence of a complication at the time that the first operation was performed when confronted with an early case of acute frontal sinusitis that is not responding to conservative treatment one is well advised to do a trephine operation. It is an external operation, but it is a very simple one, and it is followed by practically no deformity. It has the advantage of confirming the diagnosis, and of establishing external drainage. An intranasal procedure may not actually confirm the diagnosis; it may not establish free drainage, and in the hands of an inexperienced surgeon it may disturb nasal physiology. The trephine, in many cases will handle the emergency and will allow for time to plan a more deliberate attack. We do feel that the problem is a very serious one and that there is great need for careful diagnosis; for well indicated and technically adequate external surgery one must be on the alert for complications. There is need for competent allergic management, in those cases in which there is evidence of allergy. One should be seeking for the cause of failures in all humility.

Thank you very much!



TABLE 1.  
DIVISION OF CASES WITH REFERENCE TO TIME SINCE  
LAST EXTERNAL OPERATION.

PERIOD OF TIME	ACUTE	ACUTE FULMINATING	CHRONIC	CHRONIC WITH ALLERGY
Under 1 year	2	1	1	0
1 - 5 years	5	4	4	4
6 - 10 years	3	3	2	4
11 - 20 years	2	7	2	8
21 - 30 years	0	3	0	1
Untraced	2	2	2	5
Dead	0	2	4*	4**

\*1 death was due to cardio-vascular disease after 15 years of improvement.

\*\*2 deaths resulted from asthma six and seven years after operation, one improved and one not improved.

1 death of unknown causes four years after improvement.

TABLE 2.  
DIVISION OF CASES WITH REFERENCE TO THE TIME FROM THE ONSET  
OF FIRST SYMPTOMS TO THE END OF OBSERVATION.

PERIOD OF TIME	ACUTE	ACUTE FULMINATING	CHRONIC	CHRONIC WITH ALLERGY
Under 1 year	0	0	0	0
1 - 5 years	6	5	2	1
6 - 10 years	5	3	4	1
11 - 20 years	2	8	0	7
21 - 30 years	0	4	1	4
Over 30 years	0	0	2	5
Not traced	1	0	2	4
Deaths	0	2	4	4

TABLE 3.  
SUMMARY OF RESULTS.

GROUP	ACUTE	ACUTE FULMINATING	CHRONIC	CHRONIC WITH ALLERGY	TOTAL
Symptom-free	11	17	3	4	35
Improved	1	0	3	7	11
Not improved	0	1	3	6	10
Not traced	2	2	2	5	11
Dead	0	2	4*	4**	10

\*One death from cardio-vascular disease after 15 years of improvement.

\*\*Two deaths from asthma. One improved six years, one not improved seven years.

One death of unknown cause; improved four years.

TABLE 4.  
DIVISION OF SYMPTOM-FREE CASES WITH REFERENCE TO  
TIME SINCE LAST EXTERNAL OPERATION.

PERIOD OF TIME	ACUTE	ACUTE FULMINATING	CHRONIC	CHRONIC WITH ALLERGY
Under 1 year	2	1	1	0
1 - 5 years	4	3	0	0
6 - 10 years	3	3	0	1
11 - 20 years	2	7	2	2
21 - 30 years	0	3	0	1
TOTALS	11	17	3	4

TABLE 5.  
DIVISION OF IMPROVED CASES WITH REFERENCE TO  
TIME SINCE LAST EXTERNAL OPERATION.

PERIOD OF TIME	ACUTE	ACUTE FULMINATING	CHRONIC	CHRONIC WITH ALLERGY
Under 1 year	0	0	1	0
1 - 5 years	1	1	1	2*
6 - 10 years	--	--	1	1**
11 - 20 years	--	--	1†	4
TOTALS	1	1	4	7

\*One died, cause unknown.

\*\*Died from asthma.

†Died of natural causes.

TABLE 6.  
DIVISION OF CASES AND RESULTS OF TREATMENT IN RELATION  
TO THE ADVENT OF CHEMOTHERAPY.

CASES PRIOR TO 1938				CASES AFTER 1937		
SYMPTOM-FREE AND IMPROVED	DIED	NO. OF CASES	GROUP	NO. OF CASES	DIED	SYMPTOM-FREE AND IMPROVED
--	--	1	Acute	13	--	12
6	1	8	Acute fulminating	14	1	11
4	1	7	Chronic	8	2	3
5	1	12	Chronic with Allergy	14	--	7
15	3	28	Totals	49	3	33

Four deaths not attributed to frontal sinusitis are omitted from this table.

TABLE 7.  
COMPLICATIONS OCCURRING IN FORTY-SIX OUT OF SEVENTY-SEVEN PATIENTS.

Prior to 1938								
Total Cases:	28							
Complicated Cases:	17							
COMPLICATION	NUMBER OF CASES	SYMPTOM-FREE	IMPROVED	NOT IMPROVED	NOT TRACED	DIED		
Osteomyelitis	5	3	0	1	1	0		
Osteomyelitis with Brain Abscess	3	3	0	0	0	0		
Osteomyelitis with Cerebral Venous Thrombosis	1	1	0	0	0	0		
Meningitis with Brain Abscess	1	0	0	0	0	1		
Meningitis	1	0	0	0	0	1		
Cerebral Venous Thrombosis	*	2	0	0	0	1		
Fistula with Sub-periosteal Abscess	3	0	1	0	2	0		
	17	9	1	1	3	3		



## AFTER 1937

Total Cases: 49

Complicated Cases: 29

COMPLICATION	NUMBER OF CASES	SYMPTOM- FREE	IMPROVED	NOT IMPROVED	NOT TRACED	DIED
Osteomyelitis	5	2	1	1	1	0
Osteomyelitis with Brain Abscess	3	3	0	0	0	0
Osteomyelitis with Brain Abscess and Sub-dural Abscess	1	1	0	0	0	0
Osteomyelitis with Brain Abscess, Meningitis and Sub-dural Abscess	1	0	0	0	0	1
Osteomyelitis with Brain Abscess, Cerebral Venous Thrombosis, and Sub-dural Abscess	1	0	0	0	0	1
Osteomyelitis with Sub-dural Abscess	2	1	0	0	0	1
Osteomyelitis with Meningitis	2	0	1	1	0	0
Osteomyelitis with Cerebral Venous Thrombosis	** 1	1	0	0	0	0
Brain Abscess	1	1	0	0	0	0
Meningitis	1	1	0	0	0	0
Cerebral Venous Thrombosis	3	2	1	0	0	0
Fistula with Sub-periosteal Abscess	† 8	4	1	1	2	0
	29	16	4	3	3	3

\*Fistula with Peri-orbital abscess in two cases.

\*\* Ipsilateral optic atrophy.

† Retrobulbar abscess in one case symptom-free for 10 years.

TABLE 8.  
DIVISION OF CASES. SUB-PERIOSTEAL ABSCESS WITH  
FISTULA. NOT INCLUDING CASES WITH  
OSTEOMYELITIS.

CASES PRIOR TO 1938	GROUP	CASES AFTER 1937
0	Acute	1
2	Acute fulminating	5
1	Chronic	1
2	Chronic with Allergy	1
5	Total	8

TABLE 9.  
ANALYSIS OF DEATHS IN RELATION TO THE ADVENT  
OF CHEMOTHERAPY AND TO THE IMMEDIATE CAUSES.

PRIOR TO 1938	
CAUSE	NUMBER OF CASES
Meningitis	1
Meningitis and Brain Abscess	1
Cerebral Venous Thrombosis	1
TOTAL	3

AFTER 1937	
CAUSE	NUMBER OF CASES
Osteomyelitis, Brain Abscess, and Meningitis	1
Osteomyelitis, Brain Abscess, and Cerebral Venous Thrombosis	1
Osteomyelitis, and Sub-dural Abscess	1
TOTAL	3

DEATHS NOT ATTRIBUTABLE TO FRONTAL SINUSITIS  
OR ITS COMPLICATIONS

CAUSE	NUMBER OF CASES
Asthma	2
Cardiovascular Disease	1
Cause unknown	1
TOTAL	4

TABLE 10.  
FRONTAL SINUSITIS WITH OSTEOMYELITIS. RESULTS OF TREAT-  
MENT WITH REFERENCE TO GROUPS.

PRIOR TO 1938  
Total of 28 Patients

GROUP	ACUTE	ACUTE FULMINATING	CHRONIC	CHRONIC WITH ALLERGY	TOTAL
Symptom-free	0	5	1	1	7
Improved	--	--	--	--	--
Not improved	--	--	1	--	1
Not traced	--	--	1	--	1
Dead	0	0	0	0	0

TABLE 11.  
FRONTAL SINUSITIS WITH OSTEOMYELITIS. RESULTS OF TREAT-  
MENT WITH REFERENCE TO GROUPS.

AFTER 1937  
Total of 49 Patients

GROUP	ACUTE	ACUTE FULMINATING	CHRONIC	CHRONIC WITH ALLERGY	TOTAL
Symptom-free	3	5	0	0	8
Improved	--	--	1	1	2
Not improved	--	--	1	1	2
Not traced	1	--	--	--	1
Dead	0	1	2	0	3

TABLE 12.  
RESULTS OF TREATMENT IN CHRONIC FRONTAL SINUSITIS  
WITH REFERENCE TO CHEMOTHERAPY.

CASES PRIOR TO 1938		CASES AFTER 1937
1	Symptom-free	1
2	Improved	3
2	Not improved	1
1	Not traced	1
1	Died	2
7	Totals	8

TABLE 13.  
RESULTS OF TREATMENT IN CHRONIC FRONTAL SINUSITIS  
WITH ALLERGY IN REFERENCE TO CHEMOTHERAPY.

CASES PRIOR TO 1938		CASES AFTER 1937
3	Symptom-free	0
2*	Improved	7**
3†	Not improved	5
3	Not traced	2
1	Died	0
12	Totals	14

\*One death from asthma.

\*\*One death due to unknown causes.

†One death from asthma.

TABLE 14.  
SUMMARY OF OPERATIONS.  
MULTIPLE OPERATIONS ON THIRTY-THREE PATIENTS.  
SINGLE OPERATIONS ON FORTY-FOUR PATIENTS.

TYPE OF OPERATION	NUMBER OF TIMES PERFORMED	NUMBER OF PATIENTS WHO HAD THE FRONTAL SINUS OPERATED UPON ONCE
Complete Howarth-Lynch	58	21
Incomplete Howarth-Lynch	53	8
Trephine	37	9
Partial Ablation	16	2
Complete Ablation	10	4
Killian	3	--
	177	44



# TREATMENT OF MAXILLARY TUMORS THROUGH THE FERGUSSON EXTERNAL APPROACH

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AND

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(By Invitation)

MINNEAPOLIS, MINN.

This paper considers the surgical approach to tumors of the maxilla, and is not intended as a general review of methods of treatment of maxillary tumors. That subject with comments on recovery rates and prognosis has been reviewed by others.<sup>1-6</sup> This paper deals only with the mechanical approach to the maxilla; its purpose is to call attention to the Fergusson incision, to review the history of that incision, to compare it with others used for the same purpose, and finally to present a few illustrative cases.<sup>7, 8</sup> Rather than write lengthy case reports to present a few salient points, these points are elucidated in the captions beneath the photographs which accompany the paper.

The Fergusson incision consists of two arms placed at right angles to each other (Figs. 1 to 4). The vertical arm lies in the groove between the nose and cheek, goes around the nasal ala and under the naris on the same side and finishes by going straight down from the midline of the columella cutting completely through the lip. The second arm is horizontal lying under the eye and joining the vertical portion at its upper end. In actual practice one can start this incision at either end, and usually does not make the two arms separately. The horizontal arm under the eye is placed in a skin fold if possible. The vertical arm is placed in the groove between nose and cheek. One must divide the soft tissues clear through to the bone and the authors have used sharp dissection for this division. Using the

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From the Division of Otolaryngology of the University of Minnesota Medical School.

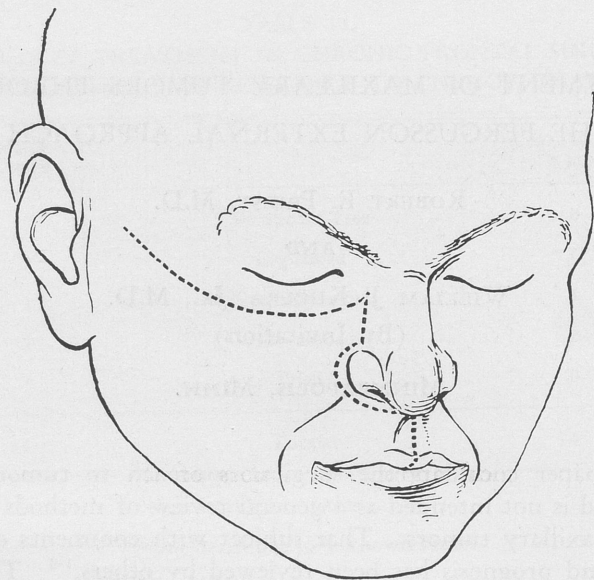


Fig. 1.—Diagram showing external limits of Fergusson incision. (From Tumors of the Head and Neck by Ward and Hendricks, Williams and Wilkins publishers, Baltimore, 1950, see bibliographic reference.<sup>8</sup>)

electric knife would make this much easier because it would control most small bleeding vessels immediately. To the authors it seems disadvantageous because it does not permit the recognition of abnormal tissue with the ease that exists when one uses sharp dissection. After one has accomplished the division of the soft tissues down to the bone, and has secured hemostasis, he is ready to reflect the flap laterally to expose the maxilla. The cheek is very vascular and one must allow himself considerable time for the making of the incision and the securing of hemostasis; he must put on many clamps, cauterize or tie the vessels, and then do the whole thing over again perhaps three times before he gets through the tissues of the cheek down to the bone itself.

The reflection of the cheek off of the maxilla can be done with sharp or electric dissection. Again the authors have the same objection to electrocoagulation voiced previously. Speed can be a disadvantage if it prevents recognition of neoplastic tissue. One picks up the cut lip and makes a horizontal incision in the mucous membrane similar

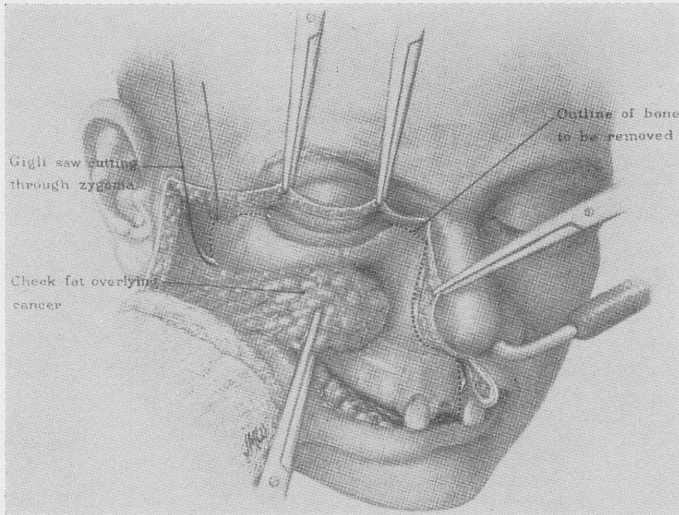


Fig. 2.—Operation for resection of superior maxilla, cheek flap turned back showing growth perforating anterior antral wall and involving subcutaneous fat. Note lines of bone incisions through the midline of the upper jaw to the floor of the nose across nasal bone and through the zygomatic arch and lateral antral wall. (From *Tumors of the Head and Neck* by Ward and Hendricks, Williams and Wilkins publishers, Baltimore, 1950, see bibliographic reference.<sup>8</sup>)

to that of a Caldwell-Luc incision but extending to the midline. This liberates the lower end of the flap. Then one gradually accomplishes the lateral reflection securing his blood vessels as he goes. When one has completed the lateral reflection of the soft tissues the midline of the lip on the diseased side lies adjacent to the ear. The blood supply of this flap is secured through the external maxillary artery. One divides the infraorbital nerve and artery where they come out of the infraorbital foramen. As the reader will see when he inspects the picture taken from Fergusson's book this is exactly the opposite of Fergusson's original principle (Fig. 5). In his original flap the blood supply came from the infraorbital artery and the external maxillary artery was cut.

One then proceeds to remove the maxillary bone. Using a mouth gag of the Crowe-Davis type to expose the roof of the mouth,

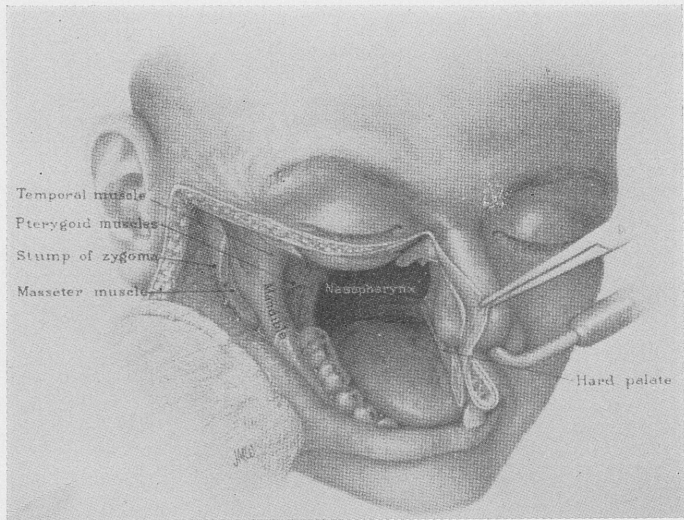


Fig. 3.—Superior maxilla has been resected. Hemostasis secured. (From *Tumors of the Head and Neck* by Ward and Hendricks, Williams and Wilkins publishers, Baltimore, 1950, see bibliographic reference.<sup>8</sup>)

and having removed the first incisor tooth on the diseased side, if it is present, the electric knife is used to divide the soft tissues on the roof of the mouth back as far as the posterior edge of the maxilla; this incision is then carried laterally behind the posterior end of the processus alveolaris of the maxilla. Next, using mallet and osteotome, the maxilla is cut free along the side of the nasal bone upward from the nasal aperture, and laterally under the eye leaving the infra-orbital ridge if possible, then downward either medial to or lateral to the front end of the zygomatic arch depending upon the extent of the disease. Even if the external carotid artery has been ligated there will be considerable bleeding when the maxilla is detached and removed. It is our custom to ligate the external carotid artery, and it seems reasonable that there is less bleeding than with the artery intact. However, there are people who think that ligation of the artery is not useful.

In preparation for the surgery the extent of the tumor has been estimated as accurately as possible. It is our custom to prepare draw-



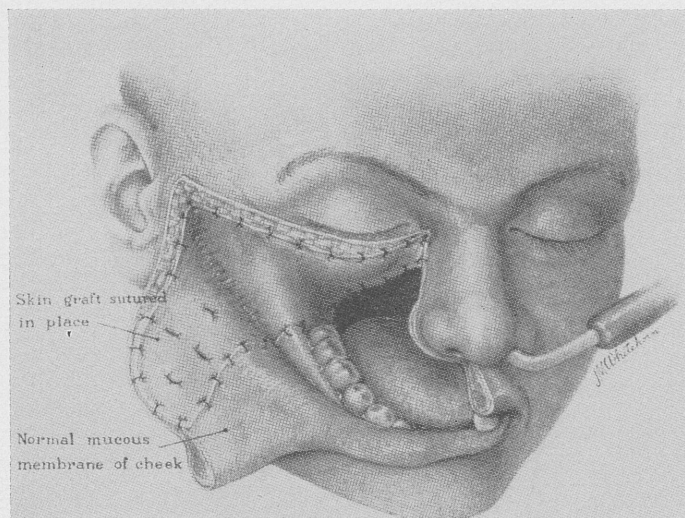


Fig. 4.—Split-thickness graft sutured over all raw surfaces of the cheek, masseter muscle, and pterygoid fossa. Wound closed. (From *Tumors of the Head and Neck* by Ward and Hendricks, Williams and Wilkins publishers, Baltimore, 1950, see bibliographic reference.<sup>8</sup>)

ings of the area expected to be exposed. The drawings are numbered at points around the periphery of the area where biopsies may need to be taken. Twenty or so small containers are labeled and sterilized for the various biopsies. The tumor is removed grossly until one thinks that he has completely eradicated it. Biopsies are then taken from the periphery of the wound and frozen sections are made immediately. If tumor is found in the frozen sections one then goes beyond the original periphery of the wound and removes further tissue and makes second biopsies. It is convenient to number the first biopsy with the Arabic digit and the second biopsy from the same site with the same digit followed by the letter, A, B or C depending upon whether one is dealing with the second, third or fourth biopsy from the same site. Thus the first biopsy might be numbered 2; the second biopsy from the same site but further out from the center of the excised area would be numbered 2A; and if 2A proved to contain tumor the third biopsy would be numbered 2B. When all biopsies are free from tumor on frozen section, if one is



Fig. 5.—Incision used for "Removal of a Tumor of the Maxilla in a Woman." (From Ferguson, "A System of Practical Surgery," published 1845, page 530, see bibliographic reference.<sup>7</sup>)

fortunate enough to be able to accomplish this, it has been our custom to coagulate the wound thoroughly all over with the electrocoagulating current. This results in sequestration of bone later because the bone is devitalized and undoubtedly destroys some occult tumor tissue. The wound is packed with gauze impregnated with petrolatum, and the large flap of cheek is brought back and carefully sutured into its normal position. It is necessary to unite the subcutaneous tissues very thoroughly so that minimal tension will exist on the skin sutures. It is necessary to be particularly careful in the apposition of the lip. One should make this union very strong and one should be very careful to appose the two points of the cut vermillion border of the lip with great delicacy. The use of a feeding tube postoperatively for a few days is desirable.

Removal of the packing from the cavity is done several days after the operation. Usually we have reanesthetized the patient, introduced an endotracheal breathing tube and packed off the pharynx just as we did for the first operation. Then we have removed the packing carefully. By the time one is ready to remove the packing, the permanent paraffin microscopic sections of the tissue examined by frozen section at the time of operation have been prepared. Be-



Fig. 6.—(Left) Photograph of a male patient three months after a right-sided Fergusson incision for carcinoma of the maxilla. In this case the entire maxilla including the infraorbital ridge, and the floor of the orbit was removed.

This patient illustrates two complications. He has the marked edema of the lower eyelid present in most of these patients but which, in our experience, subsides after approximately six months. He has a second complication in the form of a breakdown of the incision through his lip. This was the result of inadequate surgical repair, plus the pull of a very strong orbicularis oris muscle, plus trauma due to an attempt to get a denture into the mouth before the incision had a chance to heal. Secondary repair of the lip has been carried out since this photograph was made and the result is satisfactory.

Fig. 7.—(Right) Photograph of a male patient aged fifty years who was two and one-half years post-operative at the time this picture was made. The patient had a moderately low-grade carcinoma situated in the lower portion of his antrum and one which should be favorable for ultimate cure. Right maxilla was totally removed except for zygomatic arch using incision and procedures illustrated in Figures 1 through 4.

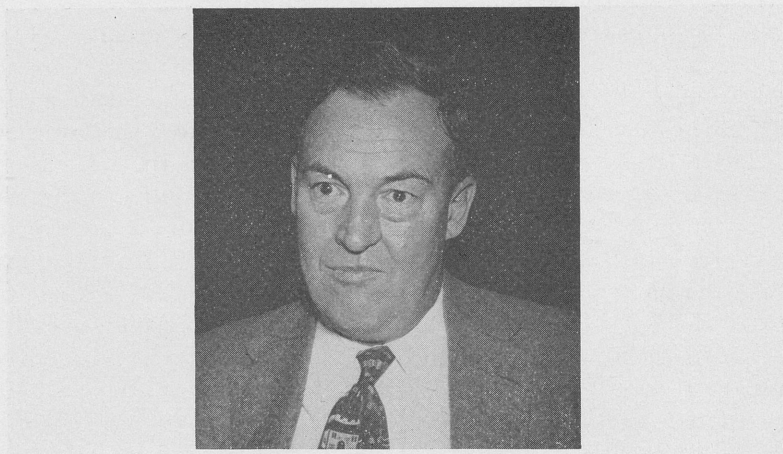


Fig. 8.—Photograph of a forty-four year old male eight months post-operative after removal of the left maxilla because of a cylindroma. Frozen sections showed tumor tissue high up in the antrum on the anterior wall, although all other indications seemed to show complete removal of the tumor. We feel that this man stands a much better chance of cure because of the accuracy with which frozen sections, made from tissue removed high up in the antrum, could be utilized to delimit the outer margins of the tumor.

cause the pathologist can be more accurate with these sections than with the frozen sections, one may have learned that some areas which appeared to contain no tumor when the frozen sections were made, in fact did contain tumor. By removing the packing under general anesthesia one can cauterize these areas further, or even do secondary removal of tissue at this time. Furthermore removal of the packing under anesthesia is a humane procedure compared with doing it without anesthesia. It is easier on the surgeon as well as on the patient. It is safer because it allows command of the situation if there is significant bleeding from any point within the operative cavity.

Patients who have had the Ferguson incision made have all of the usual difficulties following maxillectomy. The cavities crust a good deal and the odor is fairly unpleasant and strong. If the patient is sensitive it is necessary to build up his morale and self-confidence by constant reassurance and by insisting that his family help him in



this regard. The patient may need narcotic drugs at first after the operation and may become dependent upon them. Sometimes rather heroic measures are necessary to get the patient to be dependent upon himself and not upon the drugs. It is common to have very marked edema under the eye above the horizontal arm of the incision (Fig. 6). One evidently interrupts the venous return from the lower eyelid and a large quantity of fluid is imprisoned in the lid. In our experience this condition has corrected itself about six months after the operation. At times it has been necessary to support the edematous lower lid using a piece of adhesive tape fastened to the cheek above the outer canthus of the eye to form a sling. This has prevented the lower lid from being pulled away from the lower half of the ocular globe with resulting conjunctival irritation. It is possible that the tension of the cheek muscles, and sometimes the manipulation of the dentist in making the prosthesis may disrupt the suture line in the midline of the lip (Fig. 6). This may necessitate secondary repair. In the authors' opinion all of these things are of no great significance when one considers that he has had an adequate opportunity to remove the tumor for which the operation was done in the first place. The final cosmetic results certainly bear out this attitude (Fig. 7 and 8). This author believes that the adequate visualization of the maxilla afforded by this incision far outweighs any disadvantages the incision may have from a cosmetic standpoint.

The history of the Fergusson incision will now be examined. In 1845 William Fergusson described an operation for removal of a tumor of the maxilla in a woman.<sup>7</sup> He said that the tumor was, "Fortunately of a hard description, more in fact like an exostosis than what is usually called osteosarcoma, and although firmly fixed, in consequence of the thickened state of the surrounding processes of bone, was successfully dislodged and months afterwards the girl's appearance was as represented in the other likeness" (Fig. 5). He then said, "Operations for the removal of tumors connected with the upper jaw are by no means so modern as some seem to imagine. Such a proceeding is said to have been accomplished as far back as the seventeenth century." He then discusses several cases of tumors of the maxilla and finally says, "Dupuytren claimed to be the first to propose and execute this operation but beyond a doubt it was first performed in this country (England) by Mr. Lizarres in 1826."

As the reader will see by inspection of the pictures taken from Fergusson's original work he made two incisions, one like the modern

incision often named the "Moure" and a second one extending from the region of the zygoma down to the upper lip just medial to the outer corner of the mouth. As pointed out earlier in this paper, these incisions would leave the flap with only the blood supply from the infraorbital artery and would necessitate division of the external maxillary artery. In fact, Fergusson describes division of this artery in detail in his book. Thus the incision carrying the name "Fergusson's incision" today is in fact not the same incision as that described by Fergusson in 1845. Furthermore Fergusson himself makes very plain the fact that he claims no originality for the incision and that it probably antedates him by a considerable period. However when one acquaints himself with Fergusson's life and accomplishments he is only too happy to see the perpetuation of Fergusson's name, even though there may be some slight inaccuracy in its application.

Sir William Fergusson (1808-1877) of Prestonpans, Scotland is said by Garrison to be the founder of conservative surgery, that is, of the preservation of parts of the body which were needlessly sacrificed by earlier operators.<sup>9</sup> Before Fergusson's time, denuded bones, diseased and painful (even neurotic) joints were regarded as sufficient reason for amputation. Fergusson held it to be "A grand thing when by prescience even the tip of a thumb can be saved." Fergusson's progress in substituting excision for amputations was rapid after he began his practice. His surgical experience was large. Between 1828 and 1864 he is reported to have operated four hundred times for hare lip, with only three failures and one hundred and thirty-four times for cleft palate with one hundred twenty-nine successful cases. He proceeded with such lightening speed and skill that a visitor was advised, "Look sharp for if you only wink, you'll miss the operation altogether." Fergusson is said to have been an indifferent lecturer but beloved by his patients and adored by children. He was highly accomplished, a good violinist, an inventor of many surgical instruments, so expert in carpentry and metal work that he could devise any necessary apparatus out of hand, an enthusiast at fly fishing and at dancing the Scottish reel. He was noted for his great generosity and hospitality to struggling authors and dramatists, and medical students.

The incision which today goes under the name of "Moure's" consists of the vertical arm of the Fergusson incision with certain

minor modifications.<sup>10</sup> The basic Moure incision is carried along the nose and the cheek and encircles the ala and runs beneath the naris. It may include an extension to cut the lip in the midline, and a second extension going up medial to the inner canthus and ending in the eyebrow. Thompson and Negus say, "It is never necessary to make a second incision curving outward below the orbit."<sup>10</sup> Ohngren makes the midline incision in the lip extending it into the naris.<sup>6</sup> He then retracts the lip and makes an incision around the pyriform aperture somewhat in the same position as that which would encircle the alar cartilage if it were made through the skin, and a second incision along the maxilla through the mucosa in the gingival fold over the canine fossa somewhat in the manner of the Caldwell-Luc incision. No doubt there are many other modifications of these incisions which have not come to the attention of these authors.

It would seem that the Fergusson incision is indicated in cases of maxillary tumor in which the overlying skin and soft tissues are not likely to be invaded by the tumor to a degree requiring their excision, and to cases in which paramount importance is attached to the ability of the operator to visualize the entire maxillary cavity during removal of the maxillary tumor. It is the authors' opinion that this set of conditions exists in a great many maxillary tumors, with the exception of the very smallest ones located in extremely accessible positions. If one wants to give his patient the best opportunity for cure in any maxillary tumor he must be able to make adequate visual examination of all parts of the maxilla. It seems that this can be accomplished very well through the Fergusson incision and we believe that it can be accomplished better through this incision than the others described. Furthermore it is easy to do lateral rhinotomy and to excise the zygomatic arch if this proves to be necessary. In our cases illustrated herewith, it seemed to us that we gave our patients better chances for recovery because of our ability to make frozen sections high up in the antrum with the degree of accuracy made possible by the very adequate exposure permitted by this incision.

#### SUMMARY

This paper describes the performance of maxillectomy through the Fergusson incision using frozen sections as a guide the extent of removal necessary. The history of the incision is reviewed by examination of Fergusson's own writings. The incision is compared to

others used for maxillectomy. Complications and good results are illustrated with photographs.

302 MEDICAL ARTS BLDG.

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## Memorials



## RALPH BUTLER, M.D.

1873-1954

Dr. Ralph Butler, an Active Fellow of the American Laryngological Association from 1913 to 1944, and Emeritus Fellow since 1944, died at his home in the Garden Court Apartments in West Philadelphia on March 3, 1954.

Dr. Butler was born in Chester County near the "Eagle"—thirty miles from Philadelphia—on July 24, 1873, the son of James and Rachael M. Butler of a well-known Chester County family, and in this beautiful rural country he spent his early years and there he attended the country schools until the age of sixteen. He then attended the country schools until the age of sixteen. He then attended the West Chester State Normal School (now the State Teachers' College), from which he graduated in 1893, and in medicine from the University of Pennsylvania Medical School in 1900. He served a rotating internship in St. Joseph's in Philadelphia and then began the study of laryngology in the University of Vienna in the noted clinics of Politzer, Chairi, Hajek, Alexander, and the other famous teachers of that period.

Upon his return to America he began his teaching career in the Department of Otology in the University Hospital under B. Alexander Randall, then Professor of Otology in the University, where he advanced to the title of Assistant Professor before his retirement in 1926. About 1904 Dr. Butler became assistant to Dr. George C. Stout in the Department of Otology in the Philadelphia Polyclinic and College for Graduates in Medicine, starting as Instructor and advancing to Associate in the Polyclinic otological service of Dr. Walter Roberts. He shortly afterwards, in 1916, was elected Professor of Laryngology, and previous to that he was Chief of Otolaryngology in the German Hospital (now the Lankenau Hospital) and was for a time on the otolaryngological service of Dr. Walter Roberts in the Philadelphia General Hospital. He was for years Otologist to the Mary J. Drexel Home and Children's Hospital, and Consultant to the Woman's Hospital, where his sister, Dr. Margaret Butler was Chief of Otolaryngology. (She was also Professor of Otolaryngology in the Woman's Medical College of Pennsylvania and a distinguished otolaryngologist and a notable surgeon.)

When the Graduate School of Medicine of the University of Pennsylvania was established in 1918 by the merger of the Medico-Chirurgical Medical School and the Philadelphia Polyclinic, Dr. Butler became Professor of Laryngology in the new institution, the major otolaryngological faculty of which consisted of Edward B. Gleason, Walter Roberts, Ross Hall Skillern, Ralph Butler, Arthur W. Watson, George B. Wood and George M. Coates. Dr. Butler also at this time became secretary to the otolaryngological faculty, of which Dr. Walter Roberts was later chairman, and Vice-Dean for Otolaryngology, a position for which he was eminently fitted.

Always courteous, quiet and understanding, he was a true gentleman, popular alike with students and his faculty colleagues. His service in organizing the new school, the curriculum of which was unique in the history of graduate teaching, was a great factor in its ultimate success. As a teacher he was outstanding, although he could never be induced to give a course of formal lectures. But in the clinic and operating room his great knowledge of his subject and his wide experience in otolaryngology, his patience and kindness, endeared him to his students and made him a most useful teacher. He was indeed an excellent clinician and a skillful and resourceful surgeon. He became Professor Emeritus in 1947.

While Dr. Butler was not a voluminous contributor to the literature of the specialty, his papers were always carefully thought out and well prepared.

In addition to his fellowship in the American Laryngological Association, he became, early in his career, a Fellow of the American Otological Society, of the American Laryngological, Rhinological and Otological Society and of the College of Physicians of Philadelphia, as well as a member of the Philadelphia Laryngological Society. He served as Clerk, and later Chairman, of the Section on Otolaryngology of the College of Physicians in 1930-32, and as President of the Philadelphia Laryngological Society in 1926-28. He was a Fellow of the American College of Surgeons, and, of course, a member of his county and state societies, and of the American Medical Association.

For recreation in his earlier days Dr. Butler played handball; he always kept himself in good physical condition canoeing and fishing. He belonged to the Presbyterian Church, and in politics was a Republican. He was also a Mason.

In 1905 Dr. Butler married Ida Shaw, who survives him.

—G. M. C.



## HARRY ALDRICH BARNES

1872-1954

Harry Aldrich Barnes, an Active Fellow of the American Laryngological Association since 1915, and an Emeritus Fellow since 1937, died at his home in Kingston, Mass., July 18, 1954. Born in Somerville, Mass. on May 26, 1872, he received his preliminary education in its Public Schools, after which he attended Harvard College for two years. He then transferred to Harvard Medical School, graduating in 1896.

Following graduation he served a Surgical Internship at the Boston City Hospital from 1897 to 1899. He began the study of Laryngology at the Massachusetts General Hospital under Dr. Algeron Coolidge in 1903 and continued his affiliation with that institution until its Laryngological Service was merged with the Massachusetts Eye and Ear Infirmary. He served on the Staff of the Boston Dispensary as Laryngologist from 1905 to 1916 and in like capacity with the Huntington Memorial Hospital until this was combined with the Massachusetts General. His service at the Massachusetts Eye and Ear Infirmary continued until his retirement from active practice. During that period he held an appointment as Instructor in Laryngology at the Harvard Medical School.

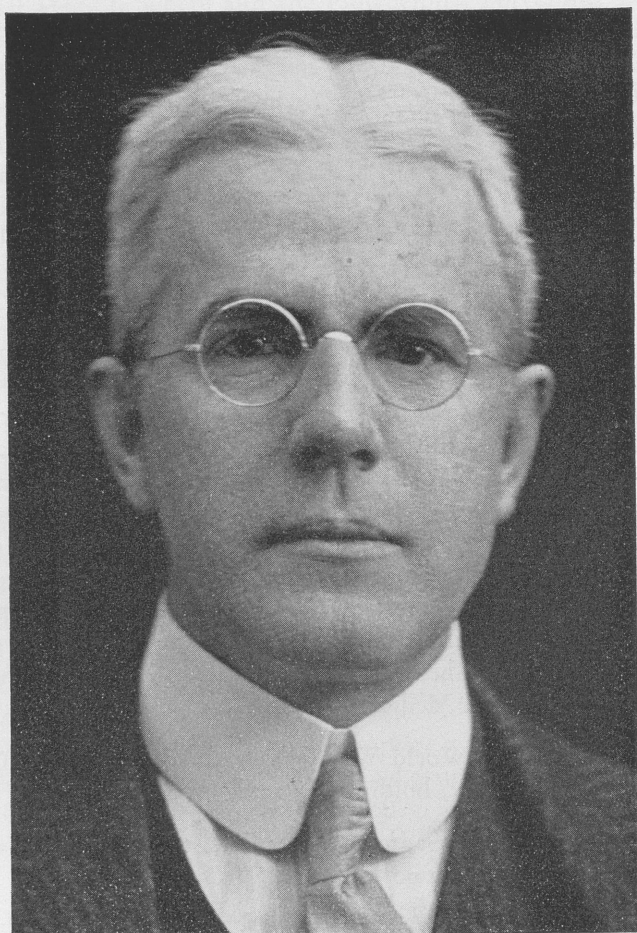
During the First World War he served in the Medical Corps of the United States Army, holding the rank of Captain.

He was married on September 20, 1921 to Helen Copeland who survives him. They had no children.

He was admitted to Active Fellowship in the American Laryngological Association in 1915 and became an Emeritus Fellow in 1937.

He was a member of the American Laryngological, Rhinological and Otological Society from 1911 to 1928. His other medical society affiliations were the New England Oto-laryngological Society, the Massachusetts Medical Society, and the American Medical Association.

Dr. Barnes was best known professionally for his pioneer work on malignant disease of the paranasal sinuses and for his book, "The



Tonsils," published in 1914. This monograph contained a great deal of original material and remains a valuable source of information on the subject today.

While not a prolific writer his contributions were extremely valuable, pertaining to the two subjects in which he was most interested and to which he devoted his studies. These were:

"Relations of Age to Tonsillar Infections," Trans. A. L. A., 1909.

"Treatment of Peritonsillar Abscess by Tonsillectomy during the Acute Stage of the Disease," Trans. A. L. A., 1915.

"The Combined Operative and Radium Treatment of Malignant Disease of the Nasal Accessory Sinuses," Laryngoscope (Sept.) 1920.

"Malignant Tumors of the Nasal Sinuses," Trans. A. L. A., 1927.

"Malignant Disease of the Pharynx, Nasopharynx, and Nasal Sinuses," Jackson & Coates, The Nose, Throat and Ear and Their Diseases, 1929.

"Prophylaxis of Post-operative Diphtheria"

"Some Points in the Applied Anatomy of the Tonsil"

"Hemorrhage after Tonsillectomy"

"Vincent's Angina"

All published in the Medical Clinics of North America (Boston Number).

A staunch Republican, a Unitarian, and a member of the Harvard Club, Dr. Barnes typified the "proper Bostonian" despite his birth and childhood days in suburban Somerville. Rather serious and dignified of mien, he was always courteous and kindly, both to his conferees and to those who might be subordinate to him. An excellent physician and an able and courageous surgeon he was among the first to attempt the radical and rational approach to malignant disease of the Sinuses.

In his passing the Association has lost another of the Old School who did much to make the American Laryngological Association what it is today.

—F. T. H.





## HARRY WEBSTER LYMAN, M.D.

1873-1954

On the sixth of May, in the beginning of his eighty-second year Harry Webster Lyman came to the end of a life as fruitful and, in a number of ways, as rewarding as anyone could wish a life to be.

Equanimity was perhaps his outstanding quality, by which it is in no wise implied that he was not forever doing battle for a principle, or a friend, or most of all for some struggling youth at grips with his early medical training. Many a mid-western otologist owes much of his proficiency to the surgical judgment and the patient ministrations of Harry Lyman in the clinic and the operating room.

In vain his friends tried to induce him to set down in a book the lessons of his large and diverse experience running almost the whole gamut of modern otology. That would have been a memorable book. It was, no doubt, partly because he was an easy-going man that the task did not appeal to him; but not altogether, for labor was no obstacle wherever the welfare of his students was concerned. It was more probably because he was a modest man; in a university atmosphere of aggressive individualism, self-aggrandizement he had none.

Harry Lyman was born in Cedar Rapids, Iowa, March 10, 1873, the son of James and Martha Lyman, both natives of Massachusetts. His early education in the Public Schools of St. Louis was followed by the usual medical training and internship, and, significantly, a preceptorship with Dr. William Porter one of the two original St. Louis members of this Association. This stimulating connection, followed by another of many years' duration with Dr. John B. Shapleigh, Professor of Otology in Washington University provided a graduate course comparable with anything in the country at the time. His connection with the University continued for the remainder of his life. He was associated at some time with a number of private hospitals, and with institutions of the U. S. Veteran's Administration and the Public Health Service.

In 1918 he served with the rank of Captain in the Medical Department of the U. S. Air Service then in its infancy and presenting

for the first time some of the perplexing problems of aviation medicine. His association there with Lewis Fisher and Isaac Jones laid the groundwork for many a stimulating lecture in the years to follow. His Candidate's Thesis for admission to the "Triological" Society in 1919 was titled "Vertigo and Aviation"—a subject which, one may be sure, was not considered too comprehensive in that day.

Besides Fellowship in this Association, Dr. Lyman held others: in the American Otological Society, the American Otological, Rhinological and Laryngological Society (of which he was President in 1947), the American Academy of Ophthalmology and Otolaryngology and the American College of Surgeons.

Surviving him are Mrs. Lyman (the former Sarah E. Long) a son, Dr. Edward H. Lyman, a daughter, Mrs. Allan E. Clark, three grandchildren and a host of colleagues and students who will forever treasure the memory of his warm friendship.

—Arthur Proetz.

## Executive Sessions

1954

## FIRST EXECUTIVE SESSION

MAY 27, 1954

The First Executive Session of the Seventy-Fifth Annual Meeting of the American Laryngological Association held at the Hotel Statler, Boston, Massachusetts, convened on May 27, 1954, at 4:45 P. M., following the First Scientific Session. Dr. Gordon F. Harkness, President, presided.

The following were elected to *Active Membership* in the Association:

DR. MURDOCK S. EQUEN  
DR. PHILIP E. MELTZER  
DR. PAUL M. MOORE  
DR. JOEL J. PRESSMAN  
DR. FRANCIS A. SOOY  
DR. WILLIAM C. THORNELL  
DR. THEODORE E. WALSH

To *Emeritus Fellowship*:

DR. ROBERT M. LUKENS  
DR. DUNCAN MACPHERSON  
DR. GORDON B. NEW  
DR. ERNEST M. SEYDELL  
DR. JOHN D. KERNAN

The following Fellows were elected to serve as a Nominating Committee:

DR. LEWIS F. MORRISON  
DR. LAWRENCE R. BOIES  
DR. DANIEL S. CUNNING  
DR. FRANCIS E. LEJEUNE  
DR. ARTHUR W. PROETZ

The deRoaldes Award was presented to Dr. Louis H. Clerf.



## REPORT OF THE deROALDES AWARD COMMITTEE

The deRoaldes Award is made to "that individual in the Americas who by his outstanding accomplishments and contributions in the field of Rhinology and Laryngology is recognized as worthy of this special honor." This Award is made available once in every three years. This is the seventh Award since the first was made in 1928.

Your Committee has considered the qualifications of outstanding American rhinologists and laryngologists for this Award and announces that its choice for this special honor is Louis Henry Clerf.

Dr. Clerf was born in the State of Washington in 1889 and spent his boyhood there on his father's ranch. He received his elementary and premedical education in that State and then entered Jefferson Medical College in Philadelphia where he earned his degree in 1912.

Dr. Clerf's preparation to be an outstanding clinician in Rhinology and Laryngology was a sound one. He served a rotating internship at Jefferson Hospital for 26 months and then became Chief Resident Physician in the same hospital for a period of 18 months. He then began the study of Laryngology at the Jefferson Hospital with Dr. D. Braden Kyle in 1915. This was interrupted for service in the U. S. Navy in 1917 and 1918, after which he pursued further graduate study at the New York Eye and Ear Hospital and the New York Throat and Lung Hospital from 1919 to 1921.

In 1925, Dr. Clerf was admitted to Fellowship in the American Laryngological Association. This recognition came to him because of his contributions to the fields of laryngology, bronchology and esophagology. By that time he had published 27 papers.

These contributions continued to expand the knowledge of bronchoesophagology, and by 1932 the Archives of our Association recorded that he had made over 60 contributions to the literature of this relatively new specialty.

Despite the limitation of his clinical practice to a narrow segment of Otolaryngology, Dr. Clerf's medical and geographical interests have been anything but provincial. He is a member of the American Association of Thoracic Surgeons, the American Therapeutic Society, the American College of Physicians, the American College of Chest Surgeons, the American Gastroenterology Asso-

ciation, the American Trudeau Society, the Pan American Medical Association, the Societe de Broncho-Esophagoscopie de Langue Francaise, the Collegium Oto-Rhinolaryngologicum, and the Scottish Otolaryngological and Laryngological Society. These are in addition to the memberships that he holds in fourteen other medical societies in this country.

He was our official representative to the Fourth International Congress of Otolaryngology in London in 1949 and the Fifth International Congress in Amsterdam in 1953. Dr. Clerf has held or holds offices in many of the Societies or of the Associations of which he is a member. These include, in addition to having been President, Secretary and Historian and Editor of our Association, the following offices: Past President, Secretary and Editor of the Transactions of the American Broncho-Esophagological Association; Past President and Vice-President of the American Laryngological, Rhinological and Otolological Society, Chairman and Secretary of the Section on Otology, Rhinology and Laryngology of the A. M. A.; Member of the Board of Governors of the American College of Surgeons; Member of the Board of Directors of the American Board of Otolaryngology; Member of the American Board of Chest Surgeons; and Director of the National Tuberculosis Association. In addition he has held numerous offices in the organizations within his state and the Philadelphia area. At present he is President of the New York Laryngological Association.

Dr. Clerf has had a continuous association with his alma mater since he received his medical degree in 1912. He has been Professor of Laryngology and Broncho-Esophagology at the Jefferson Medical College and Hospital since 1930. Villanova College conferred the honorary degree of LL.D. in 1938. He has been honored with the Signum Fidei Medal of LaSalle College and the Clarence E. Shaffrey Medal of St. Joseph's College.

Dr. Clerf has served his country in both World Wars with a six years of total active service in the U. S. Navy. He now holds the rank of Captain, MC, USNR (retired).

Today, he is a willing and tireless worker in the interests of programs concerning graduate teaching in the specialty to which he has devoted his medical life. We know him as a superb clinician and an inspiring teacher. He has proven his qualities as an able administrator and his sterling integrity as an investigator. To these

talents we can add a note on his proven ability as a raconteur and as an international ambassador of good will.

It seems fitting, therefore, that the deRoaldes Award should be made in 1954 to "that individual in the Americas who by outstanding accomplishments in the field of Rhinology and Laryngology is recognized as worthy of this special honor," Louis Henry Clerf.

Signed,

The deRoaldes Award Committee

Lawrence R. Boies, Chairman

John Mackenzie Brown

Edward N. Broyles

Francis E. LeJeune

Lyman G. Richards

There being no further business the session adjourned at 5:00 P. M.

Respectfully submitted,

HARRY P. SCHENCK,

*Secretary.*

## ANNUAL BUSINESS MEETING

MAY 28, 1954

The Annual Business Meeting, the second Executive Session of the American Laryngological Association, was called to order by President Harkness at 12:35 noon following the close of the Scientific Session.

### REPORT OF THE NEWCOMB AWARD COMMITTEE

"The Newcomb Award Committee recommends that the Award for 1954 be presented to Henry Boylan Orton of Newark, New Jersey, in consideration of his scientific contributions to this Association and his outstanding work in Laryngology, particularly in surgery of malignant disease of the larynx, hypopharynx and neck."

Signed,

Newcomb Award Committee.

President Harkness then presented the Award to Dr. Orton.

The report of the Secretary was accepted as circularized.

The combined reports of the Treasurer and Auditors were received with thanks.

The Librarian, Historian and Editor, Dr. Bernard J. McMahon presented his annual report stating that the 1953 volume of the Transactions was mailed to the Active, Honorary, Corresponding and Emeritus Fellows of the Association in January 1954. He pointed out that because of the marked increase in publication costs, the Council has deemed it necessary to discontinue the gratuitous presentation to Honorary, Corresponding and Emeritus Fellows and that such Fellows share in the expense of publication by remitting \$5.00 per volume, which is about one-half of the actual cost per volume. Several complete sets of the Transactions are on file and may be obtained for \$5.00 per volume for the years prior to 1949, and \$8.00 per volume for subsequent years. The names of the following Fellows whose deaths occurred since the last meeting of the Association were reported:



Dr. Ralph Butler, Philadelphia, Pa., Emeritus Fellow since 1944 and an Active Fellow 1913-1944. Died on March 3, 1954.

Dr. Harry W. Lyman, St. Louis, Mo., Emeritus Fellow since 1950 and an Active Fellow 1928-1950. Died on May 6, 1954.

The Membership rose and stood in silence in honor of the departed Fellows.

The report of the Librarian, Historian and Editor was accepted unanimously.

The Nominating Committee recommended the following Fellows to serve as Board of Directors of the Association for the year 1954-1955:

HENRY B. ORTON

JAMES H. MAXWELL

CLYDE A. HEATLY

HARRY P. SCHENCK

FRED W. DIXON

BERNARD J. McMAHON

J. MACKENZIE BROWN

H. MARSHALL TAYLOR

LOUIS H. CLERF

GORDON F. HARKNESS

There were no nominations from the floor and the recommendations of the Committee were approved unanimously and the Secretary was instructed to cast the ballot.

Dr. Frederick T. Hill reported for the Representatives to the American Board of Otolaryngology. The Board held two examinations since the last meeting of the Association. Of 98 candidates examined in Chicago, 22 per cent either were conditioned or failed. Of 58 candidates examined in Boston, 20.5 per cent were conditioned or failed. The next examination of the Board will be held in New York in September 1954 and the following examination will be held in Richmond, Va., in March 1955.

Dr. Arthur W. Proetz reported for the Laryngological Research Fund Committee. He reported that there was no demand for sums of money for the purposes for which this fund exists. It seemed wise to continue to augment the fund and to await the inevitable change in conditions which will render the funds of value in stimulating research.

As President of the Sixth International Congress of Otolaryngology, Dr. Arthur W. Proetz announced that the Congress would be held in Washington, D. C., on May 6 to 10, 1957, with the Convocation taking place on May 5th.

The President and Secretary were empowered by unanimous vote to prepare and send a resolution to the American Medical Association protesting the proposed action in regard to the A. M. A. Directory. This action proposes to determine which medical societies shall have their membership indicated by a special symbol in the physician's data on the basis of numerical membership; a society to be listed must have more than 200 members.

President Harkness suggested that official action should be taken in petitioning the American College of Surgeons to select one member of the Board of Regents from the specialty of otolaryngology.

Dr. Dean M. Lierle stated that the Board of Regents had been reorganized to consist of 19 members. He suggested that the Board of Regents be composed of 12 general surgeons and the other 7 members be selected from among the various surgical specialties. Well over 15 per cent of the Fellows of the American College of Surgeons are otolaryngologists and it seems appropriate that the various otolaryngological societies request Dr. Hawley to bring this question to the attention of the College.

The Secretary was instructed to communicate with the American College of Surgeons and express the desire of the Association that an otolaryngologist be included on the Board of Regents.

There being no further business the meeting adjourned at 1:10 P. M.

Respectfully submitted,

HARRY P. SCHENCK,

*Secretary.*

REPORT OF THE SECRETARY  
AMERICAN LARYNGOLOGICAL ASSOCIATION

MAY 27, 1954

The Seventy-Fourth Annual Meeting of the American Laryngological Association was held on April 26th and 27th, 1953, at the Roosevelt Hotel, New Orleans, Louisiana, under the Presidency of Dr. Louis H. Clerf.

There were 79 Fellows and 220 Guests in attendance.

Fifteen scientific papers were presented.

The following were elected:

*Emeritus Fellowship:*

DR. HAROLD I. LILLIE

DR. HAROLD G. TOBEY

DR. WILLIAM E. SAUER

*Active Fellowship:*

DR. FRANCIS L. LEDERER, Chicago, Ill.

DR. JOHN A. MURTAGH, Hanover, N. H.

DR. SAM E. ROBERTS, Kansas City, Mo.

DR. WILLIAM W. WILKERSON, JR., Nashville, Tenn.

DR. WALTER P. WORK, San Francisco, Calif.

The Newcomb Award was presented to Dr. Frederirk Thayer Hill of Waterville, Maine, for his distinguished services to this Association and to American Medicine.

The following Officers were elected:

President..... Dr. Gordon F. Harkness

First Vice-President..... Dr. Claude C. Cody

Second Vice-President..... Dr. Daniel S. Cunning

Secretary..... Dr. Harry P. Schenck

Treasurer.....	Dr. Fred W. Dixon
Librarian and Historian..	Dr. Bernard J. McMahon
First Councillor.....	Dr. Ralph A. Fenton
Second Councillor.....	Dr. J. Mackenzie Brown
Third Councillor.....	Dr. H. Marshall Taylor
Fourth Councillor.....	Dr. Louis H. Clerf

The Association has lost by death during the past year:

*Emeritus Fellows:*

DR. RALPH BUTLER  
DR. HARRY WEBSTER LYMAN

The Council at its Annual Meeting held in New York City on January 9, 1954, recommended for:

*Active Fellowship:*

DR. MURDOCH S. EQUEN, Atlanta, Ga.  
DR. PHILIP E. MELTZER, Boston, Mass.  
DR. PAUL M. MOORE, Cleveland, Ohio  
DR. JOEL J. PRESSMAN, Los Angeles, Calif.  
DR. FRANCIS A. SOOY, San Francisco, Calif.  
DR. WILLIAM C. THORNELL, Cincinnati, Ohio  
DR. THEODORE E. WALSH, St. Louis, Mo.

*Emeritus Fellowship:*

DR. R. M. LUKENS  
DR. DUNCAN MACPHERSON  
DR. GORDON B. NEW  
DR. ERNEST M. SEYDELL  
DR. JOHN D. KERNAN

Dr. Samuel Salinger was reappointed Abstract Editor.



The present membership of the Association is:

Active	97
Honorary	5
Corresponding	12
Emeritus	19
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	133

Respectfully submitted,

HARRY P. SCHENCK,

*Secretary.*

## EXHIBIT "A"

REPORT OF THE TREASURER AND AUDITORS  
AMERICAN LARYNGOLOGICAL ASSOCIATION

## BALANCE SHEET

April 1, 1954

## ASSETS

	General Fund		Trust Fund
Cash—Commercial Account .....	\$639.53		
Cash—Savings Account .....	91.43	\$ 4,948.57	
Bonds—Cost (Schedule 1) .....		32,104.70	
Total Assets .....	\$730.96		\$37,053.27

## LIABILITIES

Surplus April 1, 1953 .....	\$671.18		
Excess of Income Over Expense— ended April 1, 1954 .....	59.78		
Surplus April 1, 1954 .....	\$730.96		
Trust Fund Equity April 1, 1953 .....		\$36,137.83	
Excess of Income Over Expense— Year ended April 1, 1954 .....		915.44	
Trust Fund Equity April 1, 1954 (Schedule 2) .....		\$37,053.27	
Total Liabilities .....	\$730.96		\$37,053.27

## EXHIBIT "B"

AMERICAN LARYNGOLOGICAL ASSOCIATION  
STATEMENT OF INCOME AND EXPENSE

April 1, 1953 to April 1, 1954

## Income of the Association:

	General Fund	Trust Fund
Dues:		
1952-53—1 Member @ \$40.00 .....	\$ 40.00	
1953-54—92 Members @ \$40.00 .....	3,680.00	
1953-54—5 Members @ \$100.00 .....	500.00	
	\$4,220.00	

Registration Fees (New Orleans).....	1,300.00
Intrest received on Savings Account.....	48.90
Sale of "Transactions" .....	400.00

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\$5,968.90

Interest on Bonds .....	\$ 822.44
For de Roaldes Funds.....	97.00
	<hr/>
	\$ 919.44

## Expense

## Annual Meeting:

Projection Equipment and Operation .....	\$100.16
Reporting Service .....	192.80
Banquet .....	989.46
Programs .....	201.00
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	\$1,483.42

## Council Meeting:

Council Dinner (Annual Meeting)....	\$ 97.49
Travel Expense (Mid-Winter Meeting) .....	846.68
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	\$ 944.17

## General Expense:

Clerical Hire .....	\$ 500.00
Cost of "Transactions".....	2,335.33
Stationery, Printing, Postage.....	367.72
Auditing .....	100.00
Treasurer's Bond .....	62.50
Transfer Charges .....	14.71
Bank Service Charge, Davenport, Iowa.....	4.27
Transfer to de Roaldes Fund.....	97.00

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\$5,909.12

Net Income .....	\$ 59.78
Newcomb Award .....	4.00
	<hr/>
	\$ 915.44

## SCHEDULE 1

## AMERICAN LARYNGOLOGICAL ASSOCIATION

## TRUST FUND INVESTMENTS

April 1, 1954

## Government Bonds

	Par	1953-54 Income
U. S. Defense 2½—G—1954 .....	\$ 1,000.00	\$ 25.00
—G—1956 .....	5,000.00	125.00
—G—1957 .....	1,000.00	25.00
—G—1958 .....	2,000.00	50.00
U. S. Savings 2½—G—1959 .....	5,000.00	125.00
—G—1960 .....	1,000.00	25.00
—G—1961 .....	1,000.00	25.00
2.76—K—1965 .....	7,000.00	193.00
U. S. Treasury 2¼—1956/59 .....	1,000.00	22.50
2¾—1958/63 .....	1,000.00	27.50
2½—1964/69 .....	3,000.00	75.00
—1967/72 .....	1,000.00	25.00
3¼—1978/83 .....	3,000.00	79.24
	<hr/> \$32,000.00	<hr/> \$822.44

## SCHEDULE 2

## AMERICAN LARYNGOLOGICAL ASSOCIATION

## REPORT OF TRUST FUNDS

April 1, 1954

	Principal	Unused Income 4-1-1953	Income 1953-54	Expended 1953-54	Unused Income 4-1-1954	Principal & Unused Income 4-1-1954
Casselberry .....	\$ 5,000.00	\$ 840.53	\$132.92	\$ .....	\$ 973.45	\$ 5,973.45
Johnson .....	5,000.00	350.07	121.79	.....	471.86	5,471.86
de Roaldes .....	3,000.00	544.56	177.66	.....	722.22	3,722.22
Newcomb Trust .....	2,000.00	392.77	54.45	.....	447.22	2,447.22
Newcomb Prize .....	500.00	15.52	11.73	4.00	23.25	523.25
Hourne						
Scholarship .....	20.23	1.70	.49	.....	2.19	22.42
Research .....	16,014.19	2,458.26	420.40	.....	2,878.66	18,892.85
Totals .....	\$31,534.42	\$4,603.41	\$919.44	\$ 4.00	\$5,518.85	\$37,053.27



## COMMENT

In presenting this report I have placed the assets of the Association in one column and the amount of money entrusted to us by the various Benefactors in another column.

The Trust Funds are invested in U. S. Government Bonds. The income from these bonds is pro-rated and returned to the Trust Funds and is not used to defray the expenses of the Association.

In the past five years the income over expenses has averaged about \$500.00. This year, due to the printing of the 10-year index, the cost of printing the "Transactions" was increased about \$450.00. Without this added expense, the net income for the coming year should equal that of previous years.

Respectfully submitted,

FRED W. DIXON, M.D., *Treasurer*

May 10, 1954

## ACCOUNTANT'S REPORT

American Laryngological Association

Gentlemen:

We have examined the balance sheet of the American Laryngological Association as of April 1, 1954 and the related statements of profit and loss for the year then ended. Our examination was made in accordance with generally accepted auditing standards and accordingly included such tests of the accounting records and such other auditing procedures as we considered necessary in the circumstances with the exception that the allocation of equities to the various funds was accepted as shown by the books without verification by us. This report is on a cash basis and does not include income receivable or expenses payable at April 1, 1954, which appear to be of minor consequence.

In our opinion, except that as noted above that allocation of fund equities was accepted by us without verification, the accompanying balance sheet and statement of profit and loss, together with the schedules, present fairly the financial position of the American Laryngological Association at April 1, 1954, and the results of its operations for the year then ended in conformity with generally accepted accounting principles applied on a basis consistent with that of the preceding year.

CHARLES C. BARNES & Co.,

Certified Public Accountants.

## FINAL EXECUTIVE SESSION

MAY 28, 1954

The Final Executive Session was called to order by President Harkness at 4:10 P. M. on May 28, 1954 immediately following the Scientific Session.

On motion from the floor the report of the Nominating Committee was presented and unanimously accepted. The meeting recessed while the new Board of Directors retired and elected the following Officers for the ensuing year:

President.....	Henry B. Orton
First Vice-President.....	James H. Maxwell
Second Vice-President.....	Clyde A. Heatly
Secretary.....	Harry P. Schenck
Treasurer.....	Fred W. Dixon
Librarian, Historian and Editor.....	Bernard J. McMahon
First Councillor.....	J. Mackenzie Brown
Second Councillor.....	H. Marshall Taylor
Third Councillor.....	Louis H. Clerf
Fourth Councillor.....	Gordon F. Harkness

The President-Elect, Dr. Henry B. Orton was conducted to the Rostrum and delivered a brief address of acceptance.

There being no further business the Seventy-Fifth Annual Meeting of the Association was adjourned at 4:25 P. M.

Respectfully submitted,

HARRY P. SCHENCK,  
*Secretary.*

## List of Fellows

1954

## 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*

1942. ASHLEY, REA E., 384 Post Street, San Francisco 8, California, 1946, '47, '48, '49, '50, '51\*, '53, '54.
1936. BALLENGER, HOWARD C., 723 Elm Street, Winnetka, Illinois, 1936, '37, '38\*, '40, '41, '46\*, '47, '48, '51, '52\*, '53, '54.
1949. BECK, AUGUST LEO, 421 Huguenot Street, New Rochelle, N. Y., 1949, '50, '51, '52\*, '54.
1938. BLASSINGAME, CHARLES DECATUR, 414 Physicians and Surgeons Bldg., Memphis 3, Tennessee, 1938, '39, '41, '42, '44, '46\*, '47, '50, '51, '52\*, '53\*.
1944. BOIES, LAWRENCE R., 1631 Medical Arts Building, Minneapolis 2, Minnesota, 1944, '46, '47, '48\*, '49, '50\*, '51\*, '53, '54.
1941. BOWERS, WESLEY C., 17 East 61st Street, New York 21, N. Y., 1941, '42, '44, '49.
1951. BOYDEN, GUY LEE, 1735 N. Wheeler St., Portland 12, Ore., 1952\*, '53, '54.
1932. BROWN, JOHN MACKENZIE, 1136 W. 6th Street, Los Angeles, 14, California, 1935\*, '37, '40, '41, '42, '44, '47\*, '49, '50, '51, '52, '53, '54.
1940. BROYLES, EDWIN N., 1100 North Charles Street, Baltimore, Maryland, 1940, '41\*, '42, '46, '48\*, '49, '50, '51\*, '52, '53, '54.
1930. CAMPBELL, EDWARD HASTINGS, 133 S. 36th St., Philadelphia 4, Pennsylvania, 1930, '31\*, '32\*, '33, '35, '38, '39\*, '40, '41, '47, '48, '49, '51, '52.
1945. CAMPBELL, COLONEL PAUL A., USAF (MC), Office of Air Attache Box 36, F.P.O. 100, New York, N. Y.



1942. CANFIELD, NORTON, 175 St. Ronan St., New Haven 11, Connecticut, 1942, '46, '47, '48, '49\*, '50, '51.
1925. CLERF, LOUIS H., 1530 Locust Street, Philadelphia 2, Pennsylvania, 1925, '27\*, '28, '29, '30\*, '32, '33, '34, '35, '36\*, '37, '38, '39, '40, '41\*, '42\*, '44\*, '46, '47, '48, '49, '50, '51, '52, '53\*, '54.
1939. CODY, CLAUDE CARR, 1304 Walker Avenue, Houston, Texas 1939, '41, '42, '44, '46\*, '47, '48\*, '49, '50, '51, '52, '53, '54.
1941. CUNNING, DANIEL S., 118 East 53rd Street, New York 22, N. Y., 1941, '42\*, '44, '46, '47\*, '48, '49, '50\*, '51, '52, '53, '54.
1951. DAVISON, FRANCIS W., Geisinger Memorial Hospital, Danville, Pa., 1951, '52, '53\*, '54.
1941. DIXON, FRED W., 1027 Rose Building, Cleveland, Ohio, 1941, '42\*, '44, '46, '47\*, '49\*, '50, '51, '52, '53, '54.
1947. EAGLE, WATT WEEMS, Duke Hospital, Durham, N. C., 1948\*, '49, '51, '53, '54.
1947. ENGLER, CLARENCE W., 2323 Prospect Avenue, Cleveland, Ohio, 1947, '48\*, '49, '50, '51, '54.
1954. EQUEN, MURDOCK S., 2505 Habersham Road N. W., Atlanta, Ga., 1954.
1952. ERICH, JOHN B., Mayo Clinic, 102 Second Avenue, Rochester, Minn., 1953.
1923. FENTON, RALPH ALBERT, 1020 S.W. Taylor Street, Portland 5, Oregon, 1923, '25\*, '26, '28, '30, '34, '35, '37\*, '39, '40\*, '42, '44, '46, '47\*, '49, '50\*, '52, '53, '54.
1930. FIGI, FREDERICK A., Mayo Clinic, Rochester, Minnesota, 1930, '32\*, '34, '35, '38\*, '42\*, '47\*, '48, '50\*, '53\*.
1932. FURSTENBERG, ALBERT CARL, 201 South Main Street, Ann Arbor, Michigan, 1933\*, '36\*, '38\*, '46, '48\*, '50, '51, '53, '54.
1936. GALLOWAY, THOMAS CYRUS, 636 Church Street, Evanston, Illinois, 1936, '37, '38\*, '39, '40, '41, '42\*, '46\*, '47, '48, '50, '51, '52, '53, '54.

1940. GATEWOOD, E. TRIBLE, Professional Building, Richmond, Virginia, 1940, '41, '42\*, '44, '47.
1940. GOODALE, ROBERT L., 330 Dartmouth Street, Boston, Massachusetts, 1940, '41, '42\*, '44\*, '46, '47, '49, '50\*, '53, '54.
1928. GOODYEAR, HENRY M., 556 Doctors' Building, Cincinnati 2, Ohio, 1928, '29, '30\*, '31, '32, '33, '34\*, '35, '36, '37, '38, '39, '40, '44\*, '46, '47, '48, '49\*, '51, '53.
1950. HALL, COLBY, 1136 West Sixth Street, Los Angeles, Calif., 1950.
1940. HANSEL, FRENCH K., 634 North Grand Avenue, St. Louis 3, Missouri, 1940, '42, '46, '47, '51, '53\*, '54\*.
1933. HARKNESS, GORDON FOLLETTE, Blackhawk Hotel, Davenport, Iowa, 1934, '35, '36\*, '40, '41, '44, '46, '47, '48, '49, '50, '51, '52, '53, '54\*.
1944. HAVENS, FRED Z., 102 Second Avenue S-W., Rochester, Minnesota, 1944, '46, '49, '51, '53, '54.
1942. HEATLY, CLYDE A., 11 North Goodman Street, Rochester, N. Y., 1942, '46\*, '49, '50, '51, '52, '53\*, '54.
1945. HICKEY, HAROLD L., 1612 Tremont Street, Denver 2, Colorado, 1946, '47, '48, '49, '50, '52, '54.
1943. HILDING, ANDERSON C., 324 West Superior Street, Duluth, Minnesota, 1944\*, '46, '47, '48\*, '49, '50, '51, '53.
1928. HILL, FREDERICK THAYER, Professional Building, Waterville, Maine, 1928, '29\*, '30\*, '31, '32, '33, '34, '35, '36, '37\*, '38\*, '39, '40, '41\*, '42, '44\*, '46, '47, '48, '49\*, '50, '51, '52\*, '53, '54.
1945. HOLINGER, PAUL H., 700 North Michigan Ave., Chicago 11, Illinois, 1946, '47, '49, '50\*, '51\*, '52, '53, '54\*.
1944. HOOPLE, GORDON D., 1100 East Genesee St., Syracuse, New York, 1946, '47, '48, '49, '50, '51, '52, '53, '54\*.
1930. HOUSER, KARL MUSSER, 2035 Delancey Street, Philadelphia 3, Pennsylvania, 1930, '32\*, '33, '34\*, '37, '38, '39, '40, '41, '44, '48.

1952. IRELAND, P. E., Room 82, Banting Institute, University of Toronto, Toronto, Ontario, 1952, '53, '54.
1938. JACKSON, CHEVALIER LAWRENCE, 1901 Walnut, Philadelphia 3, Pa., 1938, '39, '40, '41\*, '42, '44\*, '46, '47\*, '48, '49, '51, '53, '54.
1939. JOHNSTON, WILLIAM HOWARD, 1515 State Street, Santa Barbara 7, California, 1939, '40, '41, '42, '44, '46, '47, '49, '50, '52.
1942. KELLY, JOSEPH D., 140 East Fifty-fourth Street, New York 22, N. Y., 1942, '44\*, '47, '48, '49, '51, '53, '54.
1942. KING, EDWARD D., 10220 Riverside Drive, North Hollywood, California, 1942, '46, '47, '49\*, '50.
1950. KLINE, ORAM R., 514 Cooper Street, Camden, N. J., 1950, '51, '52, '53, '54.
1953. LEDERER, FRANCIS LOEFFLER, 1853 W. Polk Street, Chicago 12, Illinois, 1953, '54.
1936. LEJEUNE, FRANCIS ERNEST, Ochsner Clinic Building, Prytania and Aline Streets, New Orleans 15, Louisiana, 1937, '46\*, '47, '50, '51, '52, '53, '54\*.
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.
1949. LINDSAY, JOHN R., 950 E. 59th Street, Chicago 37, Illinois, 1949, '50, '51\*, '53.
1939. LUPTON, IRVING MARTIN, 806 Medical Arts Building, Portland, Oregon, 1946, '50.
1952. LYNCH, MERCER G., Ochsner Clinic, Prytania and Aline Sts., New Orleans 15, La., 1953, '54.
1941. MARTIN, ROBERT C., 384 Post Street, San Francisco 8, California, 1941, '44, '47, '48, '50, '51, '53, '54.
1948. MAXWELL, JAMES H., 201 S. Main Street, Ann Arbor, Michigan, 1948, '49, '50\*, '51\*, '52, '53, '54.
1952. MCCALL, JULIUS W., 610 Rose Building, Cleveland 15, Ohio, 1952, '53\*, '54.

1951. McCART, HOWARD W. D., Medical Arts Bldg., Toronto, Canada, 1951, '52\*, '54.
1939. McCASKEY, CARL HEBER, 608 Guaranty Building, Indianapolis 4, Indiana, 1939, '40, '41, '42\*, '44, '46\*, '47, '48, '49, '50, '51, '52, '53.
1951. McHENRY, LAWRENCE C., 117 North Broadway, Oklahoma City 2, Okla., 1953\*.
1945. McLaurin, JOHN G., 1322 Medical Arts Building, Dallas 1, Texas, 1946, 49.
1933. McMAHON, BERNARD J., 8230 Forsythe Blvd., Clayton 24, Missouri, 1934\*, '35, '36, '38\*, '41, '44, '46\*, '47, '48, '49, '50\*, '51, '52, '53, '54.
1931. McNALLY, WILLIAM JAMES, 1509 Sherbrooke Street West, Montreal, Canada, 1931, '33, '34\*, '39, '44, '47, '49, '49, '52, '53, '54\*.
1954. MELTZER, PHILIP E., 285 Commonwealth Ave., Boston, Mass., 1954\*.
1952. MILLER, ALDEN H., 500 South Lucas Ave., Los Angeles 17, Calif., 1952, '53\*.
1954. MOORE, PAUL M., 10,515 Carnegie Avenue, Cleveland, Ohio, 1954.
1950. MORRISON, LEWIS F., 3536 Jackson Street, San Francisco 18, California, 1950, '51, '52\*, '53, '54.
1940. MORRISON, WILLIAM W., 140 E. 54th Street, New York 22, N. Y., 1940, '41\*, '44, '47, '49\*, '50, '52, '53.
1953. MURTAGH, JOHN A., 2 Maynard Street, Hanover, New Hampshire, 1953, '54.
1927. MYERSON, MERVIN C., 416 North Bedford Drive, Beverly Hills, California, 1927 '28\*, '29, '30, '31\*, '32\*, '33\*, '34\*, '35, '36, '37, '38, '39\*, '40, '41\*, '42, '44, '50\*.
1937. NASH, C. STEWART, Medical Arts Building, Rochester 7, New York, 1937, '38\*, '40, '42, '44, '46, '49, '50, '51, '52, '53, '54.



1927. ORTON, HENRY BOYLAN, 224 Delavan Avenue, Newark 4, New Jersey, 1927, '28, '29, '30\*, '31, '32, '33\*, '35, '36, '37, '38\*, '40, '41, '42, '44\*, '47\*, '49, '51\*, '52, '53\*, '54.
1948. PHELPS, KENNETH A., 1137 Medical Arts Building, Minneapolis 2, Minnesota, 1948, '50, '53\*.
1954. PRESSMAN, JOEL J., 10,911 Weyborn Avenue, Los Angeles 24, Calif., 1954.
1951. PRIEST, ROBERT E., 79, S. Ninth St., Minneapolis 2, Minn., 1951, '53\*, '54\*.
1924. PROETZ, ARTHUR WALTER, 1010 Beaumont Building, St. Louis 8, Missouri, 1924, '25\*, '26\*, '27, '28, '29, '30, '31, '32\*, '33\*, '34, '35\*, '36, '37, '38\*, '39\*, '40, '41, '42\*, '44, '46, '47\*, '48\*, '49\*, '50, '51\*, '52, '53, '54.
1951. PUTNEY, F. JOHNSON, 255 South 17th Street, Philadelphia 3, Pa., 1951, '52, '53\*, '54.
1951. RAWLINS, AUBREY G., 384 Post Street, San Francisco, Calif., 1951, '53\*.
1934. RICHARDS, LYMAN GILDER, 1101 Beacon Street, Brookline 46, Massachusetts, 1934, '35, '36\*, '37, '38, '39, '47, '49, '51\*, '54.
1945. ROBB, JAMES M., 623 David Whitney Building, Detroit 26, Michigan, 1946, '48, '49\*, '51, '52.
1953. ROBERTS, SAM E., 5964 Overhill Road, Kansas City, Missouri, 1953, '54\*.
1938. SALINGER, SAMUEL, 25 E. Washington Street, Chicago, Illinois, 1938, '39, '40\*, '42, '44\*, '47, '48, '49, '50, '51, '52\*, '53, '54.
1932. SCHALL, LEROY ALLEN, 243 Charles Street, Boston 14, Massachusetts, 1934\*, '35, '38, '40\*, '41, '42, '44\*, '46, '47, '48\*, '49, '50\*, '51, '53\*, '54.
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.

1939. SIMPSON, W. LIKELY, 512 Physicians' and Surgeons' Building, Memphis 3, Tennessee, 1939, '41, '44\*, '46, '47\*, '48, '50, '51\*, '52, '53, '54.
1950. SMITH, AUSTIN T., 1830 Spruce St., Philadelphia 3, Pa., 1950, '51, '52, '53.
1954. SOOY, FRANCIS A., 490 Post Street, San Francisco 2, Calif., 1954.
1924. TAYLOR, HERMON MARSHALL, 111 West Adams Street, Jacksonville 5, Florida, 1924, '25\*, '26, '27, '28, '31, '34, '35\*, '36, '37, '38, '39\*, '40, '41, '42, '44\*, '46, '47, '48, '49, '50\*, '51, '52\*, '53, '54.
1947. THEOBALD, WALTER H., 307 N. Michigan Avenue, Chicago, Illinois, 1947, '48\*, '49, '50, '51, '52, '53, '54.
1954. THORNELL, WILLIAM C., 2508 Auburn Avenue, Cincinnati 19, Ohio.
1950. TREMBLE, G. EDWARD, 1390 Sherbrooke St. W., Montreal, Quebec, Canada, 1950, '51, '52, '53, '54.
1925. TUCKER, GABRIEL, 250 South Eighteenth Street, Philadelphia 3, Pennsylvania, 1925, '26\*, '27\*, '29, '30, '31, '32, '33, '34, '35\*, '36, '37, '38, '39, '40\*, '41\*, '42, '46, '47, '48, '49, '50, '51, '53, '54.
1943. VAN ALYEA, OLIVER E., 135 South LaSalle Street, Chicago, Illinois, 1944, '46\*, '47, '48, '50, '51, '52\*, '53, '54.
1941. VIOLÉ, PIERRE P., 1930 Wilshire Boulevard, Los Angeles 5, California, 1944\*, '46, '47, '50, '52, '53.
1954. WALSH, THEODORE E., 640 S. Kingshighway Blvd., St. Louis 10, Missouri.
1940. WEILLE, FRANCIS L., 247 Commonwealth Avenue, Boston 16, Massachusetts, 1940, '41, '42, '44, '46\*, '47, '48, '49, '50, '51, '52, '53, '54.
1948. WHALEN, EDWARD J., 750 Main Street, Hartford, Connecticut, 1948, '50, '51, '52, '54.
1953. WILKERSON, WILLIAM WESLEY, JR., 1028 Bennie Dillon Building, Nashville, Tennessee, 1953, '54.

1948. WILLIAMS, HENRY L., 2nd, Mayo Clinic, Rochester, Minnesota, 1948, '49, '50\*, '51, '52\*, '53, '54.
1942. WISHART, D. E. STAUNTON, 170 St. George Street, Toronto, Canada, 1942, '44, '46, '47\*, '49, '51, '52\*, '53.
1951. WOODMAN, DEGRAAF, 156 E. 37th Street, New York 16, N. Y., 1951, '53, '54.
1953. WORK, WALTER P., 384 Post Street, San Francisco 8, California, 1953, '54.

*Honorary Fellows*

1949. (1917) COATES, GEORGE M., 1721 Pine Street, Philadelphia 3, Pa., 1949\*, '51, '52, '54.
1939. (1907) JACKSON, CHEVALIER, Schwenksville, Pennsylvania, 1939\*, '40\*.
1943. (1918) LEWIS, FIELDING O., Providence Road, Media, Pennsylvania.
1947. (1904) MOSHER, HARRIS P., 127 Front Street, Marblehead, Mass., 1947, '48, '49, '50\*, '51, '52, '53, '54.
1937. NAGER, F. B., Freiestrasse 3, Zurich, Switzerland.

*Corresponding Fellows*

1946. ALONSO, JUSTO M., Mercedes 1233, Montevideo, Uruguay.
1942. BATSON, OSCAR V., 133 South Thirty-sixth Street, Philadelphia 4, Pennsylvania, 1942\*, '54\*.
1938. BLAIR, VILRAY P., 508 North Grand Avenue, St. Louis 3, Missouri.
1943. EGGSTON, ANDREW O., 653 Park Avenue, New York, N. Y., 1944, '49.
1950. LARSELL, OLOF, University of Oregon Medical School, Portland, Oregon, 1950\*.
1921. LE MAITRE, FERNAND, Avenue Victor Hugo, 120 (XVI), Paris, France.
1930. NEGUS, VICTOR E., 149 Harley Street, London, W 1, England, 1938\*.

1950. NEIL, JAMES HARDIE, 64 Symonds Street, Auckland, New Zealand.
1937. PORTMANN, GEORGES, 25 bis Cours de Verdun, Bordeaux, France, 1947.
1924. SCHAEFER, J. PARSONS, 4634 Spruce Street, Philadelphia 3, Pennsylvania, 1934.
1952. TAPIA ACUNA, RICARDO, Napoles 9, Mexico 6, D. F., Mex.
1936. VIALLE, JACQUES, 27 Rue de France, Nice (Alpes Maritimes), France.

*Emeritus Fellows*

1941. (1923) BARLOW, ROY A., Berryville, Va.
1951. (1944) BEATTY, HUGH G., 150 E. Broad St., Columbus 15, Ohio, 1951, '52, '53.
1950. (1937) CROWE, SAMUEL J., Tuscany Apartments, Baltimore 10, Maryland.
1946. (1933) FOSTER, JOHN H., Eagle Lake, Texas, 1948, '49, '50, '51, '52.
1951. (1928) GITTINS, THOMAS R., 408 Davidson Bldg., Sioux City, Iowa.
1936. (1898) GOODALE, JOSEPH L., Argilla Road, Ipswich, Massachusetts.
1933. (1905) HALSTED, THOMAS H., 3500 West Adams Boulevard, Los Angeles 25, California.
1951. (1928) JARVIS, DEFOREST C., Quarry Bank Bldg., Barre, Vt., 1952.
1954. (1921) KERNAN, JOHN D., JR., 103 East Seventy-eighth Street, New York 21, N. Y.
1953. (1922) LILLIE, HAROLD I., Mayo Clinic, Rochester, Minnesota.
1954. (1927) LUKENS, ROBERT M., 1308 W. Hunting Park Avenue, Philadelphia 40, Pa.



1954. (1928) MACPHERSON, DUNCAN, 2350 Broadway, New York 24, N. Y.
1948. (1939) MYERS, JOHN LLEWELLYN, 1115 Grand Avenue, Kansas City, Missouri.
1954. (1922) NEW, GORDON B., 822 9th Ave., S.W., Rochester, Minnesota.
1953. (1921) SAUER, WILLIAM E., 3720 Washington Boulevard, St. Louis 8, Missouri.
1940. (1923) SEWALL, EDWARD C., 430 El Escarpado, Stanford University, California.
1954. (1930) SEYDELL, ERNEST M., First National Bank Building, Wichita, Kansas.
1948. (1923) SPENCER, FRANK R., 429 Pine St., Boulder, Colorado.
1953. (1927) TOBEY, HAROLD G., 274 Commonwealth Avenue, Boston 16, Massachusetts.
1950. (1922) WHITE, FRANCIS W., 471 Park Avenue, New York 22, N. Y.
1946. (1905) WOOD, GEORGE B., 329 Hathaway Lane, Wynnewood, Pennsylvania.
1951. (1935) WOODWARD, FLETCHER D., 104 E. Market St., Charlottesville, Va.

## BENEFACTORS

DR. ARTHUR W. DEROALDES

DR. WILLIAM E. CASSELBERRY

DR. SAMUEL JOHNSTON

DR. CORNELIUS G. COAKLEY

DR. JOHN F. BARNHILL

DR. JAMES E. NEWCOMB

DR. LEE WALLACE DEAN

DR. GEORGE E. HOURN

BENEFACTORS OF THE LARYNGOLOGICAL  
RESEARCH FUND

DR. JOHN F. BARNHILL

DR. AND MRS. LEE WALLACE DEAN

DR. GORDON BERRY

DR. FREDERICK T. HILL

DR. AND MRS. GEORGE E. HOURN

DR. SAMUEL SALINGER

DR. H. MARSHALL TAYLOR

DR. LOUIS H. CLERF

MR. LESTER T. SUNDERLAND

DR. WILLIAM E. GROVE

DR. BURT R. SHURLY

DR. HENRY B. ORTON



*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



## 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

## 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. HENGY B. ORTON

## 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	1948. Frederick T. Hill
1940. Gordon Berry	1949. Ralph A. Fenton
1941. Thomas E. Carmody	1950. Gordon B. New
1942-3. Charles J. Imperatori	1951. H. Marshall Taylor
1944-5. Harold I. Lillie	1952. Louis H. Clerf
1946. Frank R. Spencer	1953. Gordon F. Harkness
1947. Arthur W. Proetz	

*Vice-Presidents*

1879. F. H. Davis	J. O. Roe
1880. W. C. Glasgow	W. Porter
1881. E. L. Shurly	E. F. Ingals
1882. C. Seiler	S. Johnston
1883. S. W. Langmaid	W. H. Daly
1884. J. H. Hartman	G. W. Major
1885. H. A. Johnson	J. N. Mackenzie
1886. E. C. Morgan	S. W. Langmaid
1887. J. N. Mackenzie	C. E. DeM. Sajous
1888. W. C. Glasgow	C. E. Bean
1889. E. Holden	J. H. Hartman
1890. J. O. Roe	S. Johnston
1891. M. J. Asch	J. C. Mulhall
1892. S. Johnston	W. E. Casselberry
1893. J. C. Mulhall	S. H. Chapman
1894. C. C. Rice	A. W. 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	T. Melville Hardie
1902. G. A. Leland	W. Peyre Porcher
1903. J. H. Lowman	W. J. Freeman
1904. Thomas Hubbard	



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

*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	1935.	James A. Babbitt
1918.	D. Bryson Delavan	1939.	Charles J. Imperatori
1919.	J. M. Ingersoll	1942.	Arthur W. Proetz
1920.	George M. Coates	1947.	Louis H. Clerf
1933.	William V. Mullin	1952.	Harry P. Schenck

*Treasurers*

1912.	J. Payson Clark	1933.	James A. Babbitt
1923.	George Fetterolf	1935.	Charles J. Imperatori
1932.	William V. Mullin	1939.	Frederick T. Hill
1948.	Gordon F. Harkness	1953.	Fred W. Dixon

*Librarians*

1789.	F. H. Bosworth	1930.	John F. Barnhill
1883.	T. R. French	1934.	Burt R. Shurly
1903.	J. H. Bryan	1935.	George M. Coates

*Librarian and Historian*

1936. George M. Coates

1947. Harry P. Schenck

1944. Louis H. Clerf

1952. Bernard J. McMahon

## 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.
- \*1918. Dean, Lee Wallace, St. Louis, Mo.
- \*1881. Delavan, D. Bryson, New York City.
- \*1893. deRoaldes, Arthur W., New Orleans, La.
- \*1890. De La Sota Y. Lastra, Ramon, Seville, Spain.
- \*1879. French, Thomas R., Brooklyn, N. Y.
- \*1880. Garcia, Senior Manuel, London, England.
- \*1903. Harris, Thos. J., New York City.
- \*1878. Johnston, Samuel, Baltimore, Md.
- \*1878. Lefferts, George Morewood, Katonah, N. Y.
- \*1914. Levy, Robert, Denver, Colo.
- \*1883. Mackenzie, John N., Baltimore, Md.
- \*1881. Mackenzie, Sir Morel, London, England.
- \*1910. Masser, Ferdinand, Naples, Italy.
- \*1910. Moure, J. J. E., Bordeaux, France.
- \*1918. Oliver, H. K., Boston, Mass.
- \*1906. Pierce, Norval Harvey, San Diego, Calif.
- \*1909. Semon, Sir Felix, Great Missenden, England.
- \*1878. Solis-Cohen, J., Philadelphia, Pa.
- \*1889. Swain, Henry L., New Haven, Conn.
- \*1914. Thomson, Sir St. Clair, London, Eng.
- \*1903. Tilley, Herbert, London, England.
- \*1914. Wagner, Clinton, New York City.
- \*1890. Wright, Jonathan, Pleasantville, N. Y.

*Corresponding Fellows*

- \*1892. Browne, Lennox, London, England.
- \*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.
- \*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.
- \*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., R. F. D. No. 2.
- \*1928. Beck, Joseph C., Chicago, Illinois.
- \*1921. Berry, Gordon, Worcester, Mass.
- \*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.

- \*1905. Coffin, Lewis A., New York City.
- \*1893. Coolidge, Algernon, Boston, Mass.
- \*1913. Dabney, Virginius, Washington, D. C.
- \*1882. De Blois, Thomas Amory, Boston, Mass.
- \*1892. Farlow, John W., Boston, Mass.
- \*1922. Forbes, Henry H., New York City.
- \*1905. Freer, Otto T., Chicago, Ill.
- \*1880. Gleitsmann, Joseph W., New York City.
- \*1922. Goldsmith, Perry G., Toronto, Canada.
- \*1932. Graham, Harrington B., San Francisco, California.
- \*1906. Greene, D. Crosby, Jr., Boston, Mass.
- \*1917. Greene, Joseph B., Asheville, N. C.
- \*1896. Hardie, Thomas Melville, Chicago, Ill.
- \*1915. Hastings, Hill, Los Angeles, Calif.
- \*1888. Hinkel, Frank Whitehill, Buffalo, New York.
- \*1895. Hopkins, Frederick E., Springfield, Mas.
- \*1927. Hubbard, Thomas, Toledo, Ohio.
- \*1919. Hurd, Lee Maidment, Rowayton, Conn.
- \*1920. Imperatori, Charles J., Essex, N. Y.
- \*1904. Ingersoll, John Marvin, Miami, Fla.
- \*1918. Kenyon, Elmer L., Chicago, Ill.
- \*1929. Kistner, Frank B., Portland, Oregon.
- \*1885. Knight, Charles H., New York City.
- \*1939. Large, Secord H., Cleveland, Ohio.
- \*1894. Leland, George A., Boston, Mass.
- \*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.
- \*1943. McCullagh, Samuel, New York, N. Y.
- \*1923. McKinney, Richmond, Memphis, Tenn.
- \*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.

- \*1907. Shambaugh, George E., Chicago, Ill.
- \*1908. Smith, Harmon, New York City.
- \*1892. Wagner, Henry L., San Francisco, Calif.
- \*1892. Watson, Arthur W., Philadelphia, Pa.
- \*1939. Wilson, J. Gordon, Old Bennington, Vt.

*Active Fellows*

- \*1880. Allen, Harrison, Philadelphia, Pa.
- \*1917. Arrowsmith, Hubert, Brooklyn, N. Y.
- \*1879. Asch, Morris J., New York City.
- \*1923. Babbitt, James A., Philadelphia, Pa.
- \*1906. Ballenger, William L., Chicago, Ill.
- \*1880. Bean, C. E., St. Paul, Minn.
- \*1904. Berens, T. Passmore, New York City.
- \*1924. Bigelow, Nolton, Providence, R. I.
- \*1893. Bliss, Arthur Ames, Philadelphia, Pa.
- \*1895. Boylan, J. E., Cincinnati, Ohio.
- \*1892. Brown, Moreau R., Chicago, Ill.
- \*1933. Buckley, Robert E., New York City.
- \*1915. Canfield, R. Bishop, Ann Arbor, Mich.
- \*1934. Carmack, John Walter, Indianapolis, Ind.
- \*1924. Carmody, Thomas E., Denver, Colo.
- \*1889. Casselberry, William E., Chicago, Ill.
- \*1883. Chamberlain, C. W., Hartford, Conn.
- \*1917. Chamberlin, William B., Cleveland, Ohio.
- \*1896. Chappell, W. F., New York City.
- \*1882. Chapman, S. Hartwell, New Haven, Conn.
- \*1902. Coakley, Cornelius G., New York City.
- \*1913. Coffin, Rockwell C., Boston, Mass.
- 1918. Cox, 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.
- \*1935. Eves, Curtis C., Philadelphia, Pa.
- \*1919. Faulkner, E. Ross, New York City.

- \*1914. Fetterolf, George, Philadelphia, Pa.
- \*1901. Freeman, Walter J., Philadelphia, Pa.
- \*1917. Friedberg, Stanton A., Chicago, Ill.
- 1940. Fuchs, Valentine H., New Orleans, La.
- 1909. Getchell, Albert C., Worcester, Mass.
- \*1897. Frothingham, Richard, New York City.
- \*1907. Gibb, Joseph S., Philadelphia, Pa.
- \*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.
- \*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.
- \*1880. Jarvis, William C., New York City.
- \*1878. Johnson, Hosmer A., Chicago, Ill.
- \*1879. Johnson, Woolsey, New York City.
- \*1944. Jones, Marvin F., New York City.
- \*1879. Kealhofer, R. H., St. Louis, Mo.
- \*1901. King, Gordon, New Orleans, La.
- \*1885. Knight, Charles H., New York City.
- \*1878. Knight, Frederick Irving, Boston, Mass.
- \*1898. Kyle, D. Braden, Philadelphia, Pa.
- \*1880. Langmaid, Samuel W., Boston, Mass.
- \*1878. Lincoln, Rufus P., New York City.
- \*1911. Lockard, Lorenzo B., Denver, Colo.
- \*1913. Loeb, Hanau W., St. Louis, Mo.
- \*1897. Logan, James E., Kansas City, Mo.
- \*1935. Looper, Edward A., Baltimore, Maryland.
- \*1888. Lowman, John H., Cleveland, Ohio.
- \*1919. Lynah, Henry L., New York City.
- \*1915. Lynch, Robert Clyde, New Orleans, La.



- \* 1879. McBurney, Charles, New York City.
- \* 1927. McGinnis, Edwin, Chicago, Ill.  
1936. McGregor, Gregor, Toronto, Canada.
- \* 1913. McKimmie, O. A., Washington, D. C.
- \* 1885. McSherry, H. Clinton, Baltimore, Md.
- \* 1914. Mackenty, John E., New York City.
- \* 1898. Makuen, G. Hudson, Philadelphia, Pa.
- \* 1881. Major, G. W., Montreal, Canada.
- \* 1881. Morgan, E. C., Washington, D. C.
- \* 1886. Mulhall, J. C., St. Louis, Mo.
- \* 1925. Mullin, William V., Cleveland, Ohio.
- \* 1914. Munger, Carl E., Waterbury, Conn.
- \* 1892. Murray, T. Morris, Washington, D. C.
- \* 1881. Mynter, H., Buffalo, N. Y.
- \* 1893. Newcomb, James E., New York City.
- \* 1895. Nichols, J. E. H., New York City.
- \* 1894. Park, William H., New York City.
- \* 1892. Porcher, W. Peyre, Charleston, S. C.
- \* 1927. Porter, Charles T., Boston, Mass.
- \* 1908. Randall, B. Alexander, Philadelphia, Pa.
- \* 1882. Rankin, D. N., Allegheny, Pa.
- \* 1902. Richardson, Charles W., Washington, D. C.
- \* 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.
- \*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.
- \*1922. Wishart, David J. G., Toronto, Ontario.
- \*1940. Wood, V. Visscher, St. Louis, Mo.
- \*1896. Wollen, Green V., Indianapolis, Ind.















