













**Transactions**

**American Laryngological Association**

---

SEVENTY-SEVENTH VOLUME

"DOCENDO DISCIMUS"

TRANSACTIONS  
OF THE  
SEVENTY-SEVENTH ANNUAL MEETING  
OF THE  
**American**  
**Laryngological Association**

1956

HELD AT THE SEIGNIORY CLUB  
SEIGNIORY CLUB, P.Q., CANADA  
MAY 13 AND 14, 1956

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ST. LOUIS, MO.

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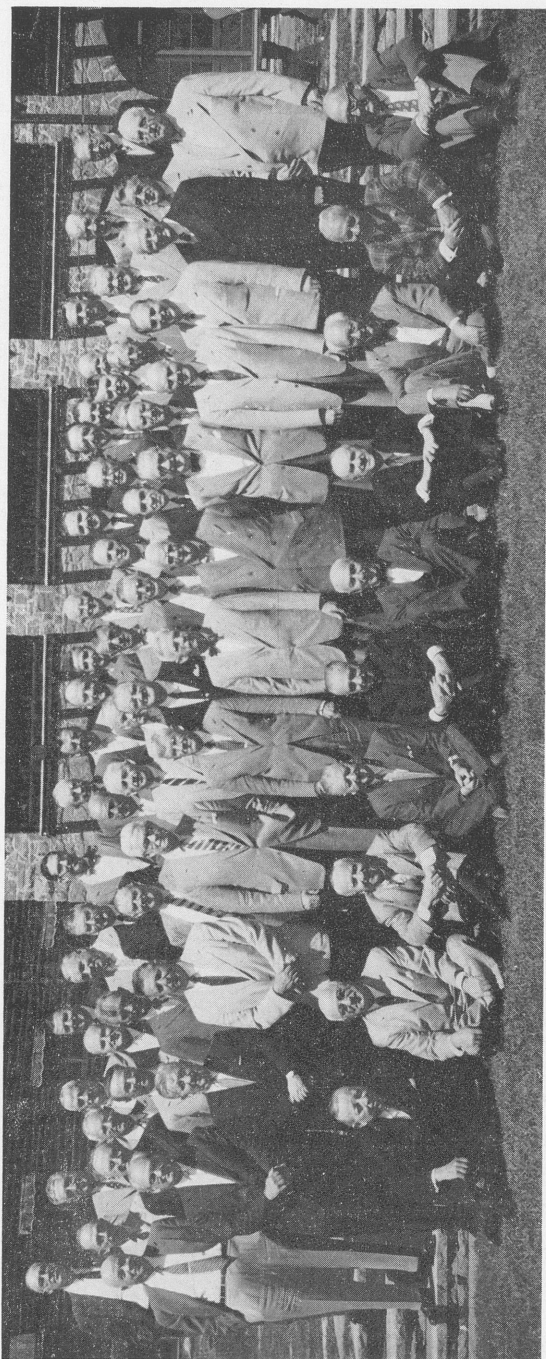
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*Top Row:*

Davison, Lindsay, Wishart, Norris, Ireland, Richardson, Friedberg, Maxwell, Work, Smith, Moore, Engler, Baker, Rawlins, McNally, Eagle, Lederer, Sooy, Robb, Roberts.

*Third Row:*

McCall, Brown, Broyles, Woodman, Hilding, Hilger, Goodale, Tremble, Van Alyea, Kline, Schall, Clerf, Ballenger, Priest, Weille, Galloway, Erich, Hill, Ashley, Campbell.

*Second Row:*

Jackson, Boies, Lierle, McMahon, Coates, Havens, Orton, Cuning, Furstenberg, Holinger, Williams, Harkness.

*First Row:*

Proetz, Lejeune, Theobald, Dixon, Schenck, Meltzer, Murtagh, Salinger, L. F. Morrison, Martin.



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TRANSACTIONS OF THE  
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MAY 13 AND 14, 1956

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The Seventy-Seventh Annual Meeting of the American Laryngological Association was called to order by the President, Dr. Bernard J. McMahon, at the Seignior Club, P.Q., Canada.

REGISTRATION

The following Fellows were in attendance:

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| HILDING, ANDERSON C. | PRIEST, ROBERT E.       |
| HILGER, JEROME A.    | PROETZ, ARTHUR W.       |
| HILL, FREDERICK T.   | PUTNEY, F. JOHNSON      |
| HOLINGER, PAUL H.    | RAWLINS, AUBREY G.      |
| HOOPLE, GORDON D.    | RICHARDS, LYMAN G.      |
| IRELAND, P. E.       | RICHARDSON, JOHN R.     |
| JACKSON, C. L.       | ROBB, JAMES M.          |
| KLINE, ORAM R.       | ROBERTS, SAM E.         |
| LEDERER, FRANCIS L.  | SALINGER, SAMUEL        |
| LEJEUNE, FRANCIS E.  | SCHALL, LEROY A.        |
| LIERLE, DEAN M.      | SCHENCK, HARRY P.       |
| LINDSAY, JOHN R.     | SMITH, AUSTIN T.        |
| MARTIN, ROBERT C.    | SOOY, FRANCIS A.        |
| MAXWELL, JAMES H.    | THEOBALD, WALTER H.     |
| MCCALL, JULIUS W.    | TREMBLE, G. EDWARD      |
| MCCART, HOWARD W. D. | VAN ALYEA, OLIVER E.    |
| MCCASKEY, CARL H.    | WALSH, THEODORE E.      |
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| MELTZER, PHILIP E.   | WISHART, D. E. STAUNTON |
| MOORE, PAUL M.       | WOODMAN, DEGRAAF        |
| MORRISON, LEWIS F.   | WORK, WALTER P.         |

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Joseph C. Barnes, New York, N.Y.  
Shirley H. Baron, San Francisco, Calif.  
William T. K. Bryan, St. Louis, Mo.  
C. J. Campbell, Hanover, N.H.  
Edgar P. Cardwell, Newark, N.J.  
Arthur J. Cracovaner, New York, N.Y.  
James E. Croushore, Detroit, Mich.  
John F. Daly, New York, N.Y.  
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S. J. Melony, Toronto, Canada  
H. James Hara, Los Angeles, Calif.  
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Lawrence McStravog, Upper Darby, Pa.  
Edwin A. Stuart, Montreal, Canada  
J. Sullivan, Toronto, Canada  
G. Dekle Taylor, Jacksonville, Fla.  
J. S. Wendel, Detroit, Mich.  
Heston L. Wilson, Seattle, Wash.  
Earl L. Wood, Newark, N.J.

## REMARKS BY THE PRESIDENT

BERNARD J. McMAHON, M.D.

ST. LOUIS, MO.

It is my privilege and a great pleasure to welcome you to the Seventy-Seventh Annual Meeting of the American Laryngological Association. We are most fortunate in being offered the splendid facilities of such a delightful setting as the Seigniori Club.

In addressing you today I am keenly aware of the signal honor which has been bestowed upon me by my fellow Members of the American Laryngological Association in being selected to succeed in the long line of illustrious Oto-laryngologists who have glorified this office, and it is with mixed feelings of trepidation and humility that I assume this responsibility. As you know, the American Laryngological Association is the oldest Laryngological Society in the World, having been founded in New York in 1878.

The record shows that our last Meeting in Montreal was in 1926. Co-incidentally, Dr. Greenfield Sluder from St. Louis was to have presided, but was prevented from doing so by a sudden illness; and Dr. George Coates, our guest of honor today, was the Secretary.

In retrospect we recall such illustrious Canadians as Dr. H. S. Birkett, who was elected to the Presidency in 1907, Dr. David J. G. Wishart and Dr. Perry Goldsmith. Their valuable contributions to the Science of Otolaryngology have kept them ever fresh in our memories. Today, we are proud to number among our Members, Dr. McNally, Dr. Staunton Wishart, Dr. Ireland, Dr. Tremble and Dr. McCart to carry on the high traditions of their Canadian predecessors.

On the fly leaf of our Transactions is inscribed "Docendo Discimus." This motto of the American Laryngological Association has ever been the guiding principle in our scientific presentations; but always tempered by the straight-forward honesty of "Errando Discimus." Truly, we learn by our teaching, but also by our mistakes.

We are all engaged in the teaching of Otolaryngology and in the training of Otolaryngologists per se. Yet, are we gaining the proper knowledge of the type of physician we have trained? We



feel confident that he is a 'safe' man with a certain amount of diagnostic ability and surgical skill derived from assisting at and performing a wide variety of operative procedures under our own supervision or that of capable members of our Staffs. But, are we just as certain that he is a good 'Doctor'? Do we teach him to know his patient as a human being who is seeking relief not only from a current malady but who is also hoping for understanding, assurance and advice about the causes of his malady, the possibility of recurrences and the ways to protect himself from such recurrences?

While Otolaryngology has advanced tremendously as a Science in the past thirty-five or forty years as a result of the formal training available in the excellently planned courses of the Graduate Schools throughout the country, well controlled Residency Training Programs, and the persistent efforts of the American Board to maintain the highest standards, has it advanced at the expense of the stimulus, the idealism in the service to humanity and the perspicacity which the student acquired under the aegis of a preceptor?

We are all familiar with innumerable instances in the past in which such methods of training have proved most effective. I am not advocating a complete return to preceptorships, nor do I mean to criticize our very efficient systems of education, but I do think that there should be some place in the training of the young Otolaryngologist for a personal association outside of his institution with one of the Senior members of his teaching Faculty for a sufficient length of time to enable him to fill this dehiscence in his otherwise stereotyped and formal Otolaryngologic training.

Over the years, Otolaryngology has become categorized as a Surgical Specialty. I presume that this was because of the fact that the earlier American Otolaryngologists received their training according to European tradition in which Anatomy, Pathology and Surgery were stressed. In recent years the physiology and function of the entire respiratory tract has become so much better understood through the enlightening researches of Proetz, Hilding, Hilger and others, together with the advent of the widespread use of the Antibiotics and the significance of allergic factors, that the inclination has been more and more to consider the patient as a whole, one in whom otolaryngologic symptoms may often be considered as local manifestations of constitutional dyscrasias; in short, the medical aspects of Otolaryngology have become equally as important as the surgical.

This trend has been best exemplified by the insistence by our American Specialty Board that Candidates be well qualified in the Basic Sciences in order to meet the requirements of this Board. This demand has been met by the offering of such Courses of instruction in the Graduate Schools and the Residency Training Programs.

It frequently happens that after a young man has finished his Residency Training he is imbued with the idea that his training as a Doctor, fully qualified to practice his Specialty, is complete. This is too often manifested in his attitude towards the acceptance of appointments on the Faculties of Medical Schools and Staffs of Hospitals in which there are demands made upon him for gratuitous services in teaching and in services in Clinics. He is interested only until he receives an appointment which gives him access to a certain hospital, and then he becomes too busy about too many things to fulfill the other obligations which are necessarily predicated by the appointment. He seems to evidence a lamentable lack of awareness of the debt that he owes to his teachers and to the Institutions in which he was trained, for the sacrifices which they gladly made to educate him, and to the students who are dependent upon him for their education.

If we are failing to recognize this attitude in the neophyte Otolaryngologist, even if it may only be a trend, are we learning by teaching? And furthermore, if this same attitude is being recognized by the patient and by our fellow physicians in other branches of Medical practice, may this not be one of the weak links which has given rise to the impression that Otolaryngology is a dying Specialty? By combining our efforts to correct this attitude, not only in the Graduate but also in the Undergraduate students, we would be taking a long step towards the solution of many of the actual and fancied abuses of 'Doctor-Patient' relationship which have assumed ominous proportions in the present day viewpoint of the critics of our Profession. To err is human, yet "Errando Discimus" is most salutary!

Specialization in medical practice is the privilege and the duty of the physician who appreciates his ability and limitations and best applies this knowledge to the relief of suffering humanity. By the same token, I have the greatest respect for the general practitioner who realizes his professional limitations and advises the patient accordingly.

The charge of over-Specialization can never be substantiated while competent physicians prefer to practice one or the other branches

of medicine in which they may have a greater aptitude and training. When this is well and conscientiously done, the hue and cry which emanates from certain definite sources about 'neglected humanity' can be nothing more than a weak subterfuge to mask the ulterior motives of the self seeking.

The challenge of politically minded individuals can be well and successfully met by every effort being made to influence this attitude of the younger physician during his formative period of training. Much can be done, but it is the duty of all of us who are responsible for teaching at the student level to impress most strongly upon the minds and hearts of our students that the good of the patient and of the community, and the integrity of their profession are paramount.

I hope that these remarks will be taken in the spirit in which they are made, as constructive suggestions and not as criticisms, for, who am I to throw the first stone? My purpose in presenting these thoughts was threefold, to find a way to improve the attitude of the younger Otolaryngologists toward their teaching obligations, to accentuate our responsibilities towards the fostering of better public relations for our Specialty, and to jealously guard our professional prerogatives against the destructive inroads of pernicious influences and practices. These objectives cannot be accomplished by an attitude of complacency and 'laissez faire', but by a militant realistic approach to our problems.

I would like to express my appreciation to Dr. Schenck, our most capable Secretary, and to the Members of our Council for their invaluable assistance and advice in planning our Meeting and preparing this program of excellent papers. It is my sincere hope that you may enjoy them, and participate freely in the discussions, to the benefit of all of us.

8230 FORSYTHE BLVD.

CLAYTON 24, MISSOURI

## HUMIDITY, A PROBLEM IN AIR-CONDITIONING

ARTHUR W. PROETZ, M.D.

ST. LOUIS, MO.

It really *is* the humidity—as you may have heard—and not the heat. Since the advent of central heating it has been a bug-bear, which air-conditioning now aggravates in several respects. In the struggle between the nose and the machinery in the basement, sometimes the heater wins, and sometimes the cooler, but seldom the nose. When the doctor says to his patient, “Keep your living quarters reasonably humid in the winter months,” it is likely that neither of them has a clear conception of what this entails or how ineffectual the attempt may be.

The importance of the respiratory mucous blanket has been so vigorously stressed that we may skip it and come quickly to the point. We deal here first with the water requirements of the mucosa; second, with the behavior of air-vapor mixtures at various temperatures; third, with certain characteristics of houses; and fourth, with the apparatus now available for controlling the humidity in a room.

### REQUIREMENTS OF THE MUCOSA

The nasal mucus contains some 96 per cent water. To begin with, it is more viscous than mucus elsewhere in the body and even slight drying increases the viscosity enough to interfere with the work of the cilia. Demands on the nasal glands are great, even under usual conditions, and they cannot cope with extreme dryness indoors, in winter. Authorities differ as to the “comfort range” of temperature; however both physiologists and engineers regard it as closely approximating thermal equilibrium between the ambient atmosphere and the heat production and retention in the subject. Much of the

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Read before the American Laryngological Association, Montreal, Canada, May 13, 14, 1956.



element of comfort has to do with the location, the season and the altitude, and with the subject's age, habits, occupation and clothing. For purposes of this discussion it will be regarded as ranging between 72°F and 74°F.

Optimum relative humidity for these temperatures has been variously given as 40 per cent to 45 per cent, and some tests with human subjects have placed it (with a temperature of 74°) as high as 79 per cent; but even the lower figures will be difficult to maintain in heated houses, as will appear.

Experience has shown that, with approaching winter, the first wave of dry-nose patients appears in the office when the relative humidity indoors falls to 25 per cent. It would seem, therefore that 35 per cent could be regarded as a passing grade, but 40 per cent as something to shoot at.

Every 24 hours the adult human breathes approximately 500 cu.ft. of air. In passing through the nose the air reaches a temperature of almost 98.6°F and a relative humidity of 95 per cent.

Five hundred cubic feet of air in this physiological state contains 1.2 pounds of water. Since this amount of air in a room at 72°F and .40 r.h. holds only .25 pounds water, then even under these ideal conditions the nose must supply daily .95 pounds—approximately a pint. With the humidity down to 20 per cent this becomes 1.08 pounds; at 12 per cent 1.125 pounds. But 500 cu.ft. in the warm lung was only 453 cu.ft. in the cool room at 72°, so that another 10 per cent must be added. However, some moisture from expired air is condensed on the turbinated bodies and re-breathed, partly cancelling this last.

It boils down to this: a pint of water is a lot of water for a small nose to turn out. In disease or old age it simply doesn't deliver; drainage stops and the germs take over.

Excessive humidity in the room is no parallel problem. The nose merely takes a rest. The skin is less fortunate, along with most of its contents.

#### MOISTURE PLUS AND MINUS

In terms of health and human working efficiency in the summer months humidity is the determining factor, and the wet, rather than the dry bulb reading is the index of tolerance.

According to figures of the United States Weather Bureau the average humidity of American cities in August ranges from 16 to 93 per cent but these figures are significant only as they are related to current temperatures.

In the so-called Temperate Zone—a misnomer, if ever there was one—there are many localities with high summer temperatures in the nineties and humidity around sixty-five per cent. In such climates dehumidification in homes and offices, and especially in theatres, stores and other crowded buildings is almost imperative. Unless the air is dried as well as cooled, perspiration fails to evaporate from skin and clothing and people shiver. In dry air evaporating perspiration does its own cooling and higher dry bulb room temperatures are tolerated with comfort. The removal of moisture is not too difficult. Air passing through chilled grids or cold water sprays loses its vapor by condensation.

The actual quantities of water involved in humidifying and dehumidifying are amazing—which is really what this paper is about.

The simplest unit with which we have to deal is a living room, or a bedroom or a sick room. A room 15 feet square, with a nine foot ceiling, contains approximately 2000 cu.ft.—a handy figure to refer to.

Private homes range from cottages without basement or attic, with a content of 6000 cu.ft., to spacious three story houses of 50,000 cu.ft. and more. Public buildings are individual problems; a ten-story office building will contain from 750,000 to 1 million cu.ft.

Take the large residence: in reducing the temperature in a 50M cu.ft. house from 90° to 76°, and the humidity from .65 to .40, 51.9 pounds of water must be got rid of: 26 quarts.

|                                 |                     |                       |
|---------------------------------|---------------------|-----------------------|
| 50M cu.ft. air (3500 lb. @ 90°) | @ .65 r.h. contains | 70 lb. water.         |
| 50M cu.ft. air (3650 lb. @ 76°) | @ .40 r.h. contains | 28.1 lb. water.       |
|                                 |                     | <hr/> 51.9 lb. water. |

I have checked the performance of an average refrigeration system in such a house, under the conditions referred to, and found the water being removed at the rate of six quarts an hour, which would do the job in four hours and twenty minutes, provided no fresh outside air came in.

TABLE I

Showing the effects upon the relative humidity of bringing the air to 72°F in January, in various localities:

| PLACE             | AV. JANUARY<br>NOON TEMP. | AV. JANUARY<br>P.M. REL. H. | REL. H. @ 72°F<br>INDOORS |
|-------------------|---------------------------|-----------------------------|---------------------------|
| Chicago*          | 27                        | .74                         | .14                       |
| Cleveland*        | 30                        | .76                         | .16                       |
| Dallas            | 48                        | .62                         | .27                       |
| Denver*           | 38                        | .50                         | .18                       |
| Detroit*          | 27                        | .80                         | .14                       |
| Iowa City*        | 25                        | .75                         | .12                       |
| Los Angeles       | 62                        | .59                         | .40                       |
| Miami             | 73                        | .74                         | .76                       |
| Minneapolis*      | 15                        | .78                         | .08                       |
| New Orleans       | 59                        | .73                         | .47                       |
| New York*         | 34                        | .66                         | .18                       |
| Philadelphia*     | 37                        | .69                         | .19                       |
| Phoenix*          | 60                        | .38                         | .16                       |
| Portland, O.      | 42                        | .75                         | .26                       |
| St. Louis*        | 35                        | .69                         | .18                       |
| Salt Lake City*   | 33                        | .75                         | .17                       |
| Seattle           | 42                        | .79                         | .27                       |
| Washington, D.C.* | 38                        | .64                         | .19                       |
| Waterville, Me.*  | 25                        | .69                         | .12                       |

\* Cities in which heating to 72°F depresses rel.hum. below 25%.

Not so simple is the addition of water in winter.

Let us select a mild winter day; the temperature is 30° and the humidity 40 per cent. One hundred cubic feet of air on this pleasant day carries approximately two ounces of water (0.134 lb.). Take it indoors, heat it to 74° and see what happens. The two ounces of water is still there but the 100 cu. ft. is now 109 cu. ft. and the 40 per cent humidity is now only 8 per cent. To bring it back to 40 per cent we must supply 1.2 ounces of water, which goes for every 100 cu.ft. in the house.

The 2000 cu.ft. bedroom will require a pint and a half; the 25,000 cu.ft. house two and one-third gallons; the 50,000 cu.ft. house four and two-thirds gallons: a sizable amount. If we are satisfied

with only thirty per cent the figures will be smaller, but not much, and this applies only to the *air* in the house. One reckons also with thirsty carpets, upholstery, draperies and woodwork which may double the amount—and all this on a *mild* winter day, with a barely freezing temperature. At lower temperatures the requirements pile up. What this amounts to in various parts of the country is shown in the accompanying table.

## HUMIDIFIERS

To test the relative efficiency of some familiar humidifying devices a few careful measurements were made, not in the laboratory but under household conditions in sleeping quarters and sick-rooms. Tap water, and not distilled water, was used; doors, windows and vents were closed, but not hermetically sealed. Consequently there were minor errors which would not have been tolerated in a laboratory experiment, but the same variables occur in houses, and we do not sleep—most of us, that is—in our laboratories.

The amount of water evaporated per hour by the various contrivances is as follows:

TABLE II

|  | OZ. H <sub>2</sub> O<br>PER HR. |
|--|---------------------------------|
| Tea kettle on gas flame                                    | 48-60                           |
| Cold water spray (centrifugal atomizer, furnace type, 25W) | 21.03                           |
| Tea kettle on electric hot plate (600W)                    | 19                              |
| Cold water spray, table type (70W)                         | 16.5                            |
| Commercial steam vaporizer, standard make (250W)           | 10.5                            |
| Pan on radiator, per sq. ft.                               | 1.0                             |
| Pan on table, per sq. ft.                                  | .33                             |

According to these figures the pint and a half required in the 2000 cu.ft. room would come from the tea kettle on the gas flame in 30 minutes; the same kettle on a hot plate in one hour, twenty minutes; the table spray in one hour, thirty minutes; and last, if not quite least, the commercial electric steamer in two hours, twenty-



four minutes. Pans of water, even on the radiator, are hardly worth mentioning.

Once the desired humidity has been reached replenishment must be constantly kept going because of losses through window cracks, chimneys, open doors and to an amazing extent even through brick walls when there is any wind. Efforts to humidify individual bedrooms are practically useless unless these losses are overcome by keeping the vaporizer going and what is more important, by keeping the room shut off from the rest of the house. In cold climates double window panes will be required if adequate humidity is to be maintained, otherwise condensation on windows will not only remove moisture from the air but water will run off and ruin the wallpaper.

To arrive at some figures, a bedroom was shut off by closing (but not sealing) doors, windows and registers. A table model centrifugal cold spray with a capacity of one pint an hour was put in continuous operation. At the end of two hours two doors were opened, one leading to a hall, the other to an adjoining room, with the following results:

HOUSE: brick, insulated.

ROOM: capacity 2100 cu.ft., 4 inside doors, 3 windows double panes, weather stripped, 4 vents closed.

OUTDOORS: dry bulb 49°F, wet bulb 38°F, .31 r.h., bar. 29.8 in.

HUMIDIFIER: cold centrifugal spray, capacity 16.5 oz. per hour.

TABLE III

| TIME               | DRY BULB | WET BULB | REL. H. |
|--------------------|----------|----------|---------|
| 0:00               | 71       | 53       | .27     |
| 1:00               | 69       | 53.5     | .33     |
| 2:00               | 67.5     | 55.5     | .44     |
| — 2 doors opened — |          |          |         |
| 2:10               | 70       | 54       | .33     |
| 2:20               | 70.5     | 54       | .32     |
| 2:25               | 72*      | 55       | .31     |

\* Between 0:00 and 2:25 house temperature rose from 71°F to 72°F

It required one hour, 30 minutes to raise the humidity only 13 per cent from a relatively acceptable 27 to a desirable 40; then, having risen to 44 per cent it fell back to 31 per cent only 25 minutes after the doors were opened, although the vaporizer was kept going. This is practically where it was at the start.

(The fall in room temperature during the first two hours was due partly to heat loss from evaporation and partly to radiation, the heating vents being closed and the outside temperature  $49^{\circ}$ .)

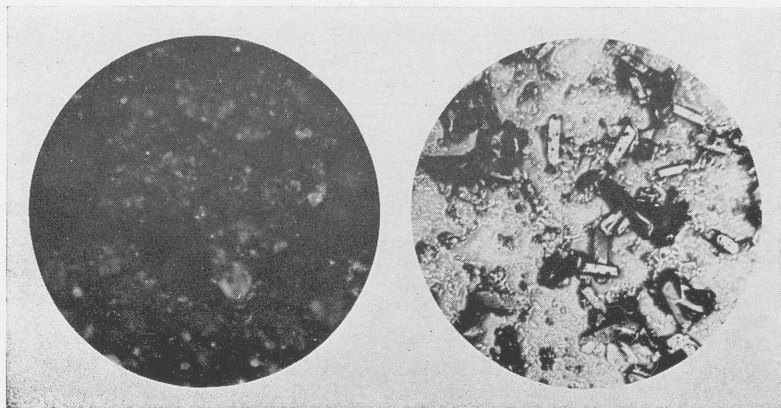


Fig. 1

Fig. 2

It has been contended that steam is superior to the cold spray in humidifying a sick-room because the evaporation from the latter reduces the room temperature whereas the hot steam device raises it. This is true but not of much consequence since the latent heat of water is the same whether the necessary Btu come from a furnace or a hot-plate. The furnace supplies them rather more economically.

A word regarding the furnace type centrifugal cold water spray listed in Table II: it is the successor in this residence plant to 1) an electrically heated water bath, 2) a pair of atomizer-type pressure sprays, 3) two moist-plate evaporators, 40 plates, 4) one home made large same. Of these the centrifugal spray was the most effective.

It has had to be abandoned, at least temporarily, because of one great draw-back: it scatters calcium dust all over the house, and

plays havoc with the nasal mucosa. City water contains solids in various amounts in various localities and until some practical and not too expensive means is found for filtering the dusts which develop on evaporation from cold sprays this type of spray leaves something to be desired. Ordinary furnace filters are only partly effective.

The following figures are indicative of the amount of solid matter in city water in several communities. They were kindly supplied by the water commissioners of those cities.

TABLE IV

|              | TOTAL SOLIDS,<br>PARTS PER MILLION |
|--------------|------------------------------------|
| Boston       | 43                                 |
| Chicago      | 154<br>159<br>199<br>165           |
| Denver       | 104                                |
| Miami        | 105<br>177                         |
| Minneapolis  | 126                                |
| Philadelphia | 250<br>133                         |
| St. Louis    | 298                                |

In grains per gallon this ranges from 2.5 to 17 gr.

Assuming an average solid content of, say, 10 grains per gallon the 50,000 cu.ft. house would receive some 420 grains of white powder every week. In St. Louis it would come to almost an ounce and a half; in Boston only one-seventh as much. Dust gathered from furniture and parts of the heating system was found to contain chiefly calcium and magnesium salts, in both amorphous and finely crystalline form (Fig. 1). Adding a drop of water to the dust on a microscope slide, and allowing the preparation to evaporate slowly, brings out the characteristics of the crystals (Fig. 2).

A final observation on humidity indicators: the sling psychrometer is the instrument of choice for use in the home. Dial instruments

depending upon the expansion and contraction of a thread of sensitive material under the influence of moisture are not very accurate. A strip of paper with graduated spots of cobaltous chloride, which turn color successively with changes in moisture is consistent over all, if not precise. Stationary wet and dry bulb thermometers are singularly misleading even under a reasonable amount of fanning.

Five instruments were tested against one another just as they came from the shop:

TABLE V

|                                 | HUMIDITY<br>READING | ERROR |
|---------------------------------|---------------------|-------|
| Sling psychrometer              | .26                 |       |
| Wet and dry bulb, stationary    | .545                | +28   |
| Wet and dry bulb after fanning  | .39                 | +13   |
| Commercial dial type            | .22                 | — 4   |
| DeLuxe dial type                | .31                 | + 5   |
| Dial type (advertising novelty) | .37                 | +11   |

The adjustable dial instruments were then set to conform to the psychrometer reading and the next day the results were a little worse: —6, +6 and +11 respectively.

From all these dull figures one may gather that the control of humidity in the house is not simple. The fellow with gadgets and contraptions on his furnace is better off than the one with pans of water on the radiator but maybe not so much as he thinks.

Medical programs are packed with brilliant last words on every subject from broad-spectrum antibiotics to the sines and cosines of the nasal angles while here I stand still wondering how to moisten a house. It may be just fortunate that no solution is at hand, for when the solution is found the mid-winter crush in your office and mine will be at an end.



## DISCUSSION

DR. LAWRENCE R. BOIES: Dr. Coates, Dr. McMahon, Members and Guests, Dr. Proetz has the capacity for reducing notions to their mathematical values. I recall the first time that I encountered one of his measurements when a resident over 25 years ago. I learned from his writings that if the average sized maxillary sinus is multiplied by 100, we will have a 10 x 10 ft. room and the natural ostium will be an 8 inch hole near the ceiling. That has been useful information for me chiefly to give to the resident, and an occasional layman, a visual image of the location and size of the drainage outlet.

Today, Dr. Proetz has given us in simple measurements the water requirements of the nasal mucosa, figures on how air vapor mixtures behave at various temperatures, some estimate on what it takes to humidify certain houses and the actual performance of the various gadgets that are used to measure the humidity of a room.

I have to take his word for these figures but I do this with confidence. It is comforting to know the facts, and even though we are left without a practical solution of how to accurately accomplish humidity control in the every day environment in which well people live, the effort will go on. I am also comforted to know that in Minneapolis the mineral content of the water is not nearly as high as it is in St. Louis. (We were low on the list.)

Probably most of us find occasion at frequent intervals to prescribe "humidity" as an item of therapy. The largest use for this used to be for croup and other forms of laryngitis and laryngotracheobronchitis. For this, the old fashioned croup tent (steam tent) with its high temperature has been generally abandoned. I think it was Davison who first effectively promoted the use of high humidity in a controlled temperature. We went from the steam tent to the wet room where in the words of Louis Clerf, the nurse practically had to "wear rain coat and rubbers" to keep dry. Then came the modern plastic tent with its convenient controls for moisture, temperature, and oxygen when needed. This seems to fill the bill and in my observation the mortality has practically been erased in laryngotracheobronchitis when adequate therapy in the form of humidification, antibiotic therapy, etc., is instituted promptly and the occasional need for tracheotomy is promptly met.

Added humidity with controlled temperature is an important factor in lessening morbidity whenever the trachea is opened in other types of laryngeal work.

A patient once asked for a humidifier in her room after I had packed her nose in a plastic operation on her septum. I now prescribe it routinely in all cases where the patient is forced to mouth breathe postoperatively because of nasal packing or for some other reason. It helps.

The facts that Dr. Proetz has given us suggest one of the important factors in our inability to prevent the common cold, in that we cannot control the humidity of the environment in which a majority of people live. We can, however, control the humidity for the sick patient and this we can claim as an important therapeutic advance.

I feel we are indebted to Dr. Proetz for another lesson in fundamentals.

DR. EDWIN N. BROYLES: Mr. President, Dr. Proetz's paper about the nose is a very interesting one, but to those of us who handle patients with laryngectomies it is especially noticed that during the winter months these people have trouble with the dry air and only during the winter months do they have to come in with dried mucus in the trachea and start to bleed when they cough. That worries them. They

think it is perhaps some recurrence when all that it is is the dried mucus on the trachea.

Those of us who handle laryngectomy cases are especially cognizant of the fact that the air in the house is dry in the winter, whereas in the summer time, due to the moisture in the air, they get along very well.

DR. THOMAS C. GALLOWAY: Mr. Chairman, it has always seemed that the very low humidity in the Rocky Mountain area is a very comfortable situation. Low humidity in the Middle West house in winter is a very unhealthy and uncomfortable situation. I have never seen an adequate explanation for that.

Hubbard in his original work on humidity emphasized the factor of dust formation and irritation in the dry house, or atmosphere with low humidity. I would like to hear Dr. Proetz's comment.

DR. ARTHUR W. PROETZ: I should like to express my appreciation for the discussion of the paper, especially to Dr. Boies, who took up the effect of air conditioning on diseases and patient, which time did not permit me to include in my paper.

Dr. Galloway, I don't know why dry air should be healthier in the Rocky Mountains than in a house except that in the latter there are probably more bacteria. It is well known that when Dr. Grenfell's expedition went to Labrador they found no colds among the natives. However, whenever a ship arrived there was an epidemic of colds from which many people died because they were unaccustomed to disease germs and had no immunity.

There is a simple indicator that will tell you just when to get busy with the humidifier. When you begin to draw sparks off in the room, that is the time to turn on the steam.

## HISTAMINE-RELEASING DRUGS

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In recent years observations upon the release of tissue histamine by drugs have been multiplying rather rapidly. Such observations are of interest from a number of points of view. First, because they throw light on some of the side-effects—expected or unexpected, and usually unwanted—that accompany the therapeutic effect for which the drug is given. Secondly, because they afford a new approach to the old problem, the part that histamine plays in the body's normal economy; for it is unfortunately true that in spite of much research the physiological role of this powerful and ubiquitous substance is a matter more of speculation than of knowledge. Thirdly, there is the hope that, since histamine release is undoubtedly a component of certain pathological reactions, especially allergic reactions, we might use histamine releasers to find out more about these phenomena. And lastly, there is the possibility that the pharmacological release of histamine might in some circumstances be useful in therapy. Progress along these various lines has hardly been spectacular; but some of the outlines of the picture are beginning to stand out and a few of the details have been clarified.

Before I discuss the histamine liberators it may be useful to recall some points about the location and action of histamine itself.

As to the distribution<sup>1</sup> of histamine in the organism, most of our information comes from animals; but combining this with the rather scanty data on human tissues it seems safe to guess that the human body contains several hundred milligrams of it, an amount which would certainly produce a very severe reaction or even death if it were all let loose at once. Practically every tissue that has been examined contains measurable amounts; the concentration varies however not only from tissue to tissue, but also from one individual to another. Mucous and serous membranes are often particularly rich in histamine, and substantial amounts are also found in association with striated and smooth muscle and in skin and subcutaneous tissue.

There is no striking sex difference, but in some organs the concentration appears to increase with age. The recent works of Riley and West<sup>2</sup> in Scotland and of Graham and her co-workers<sup>3</sup> in St. Louis have shown beyond any doubt that a great deal of this stored tissue histamine is located in a particular kind of cellular element, the mast cells. These large granular cells were first recognized by Ehrlich<sup>4</sup> because of their affinity for basic dyes, some of which like toluidine blue stain them metachromatically. Metachromasia, as we now know, is characteristic of acidic substances of high molecular weight, particularly sulphated polysaccharides. The strongest naturally occurring acid of this group is heparin; and a group of Swedish workers<sup>5,6</sup> under the leadership of Jorpes showed, about twenty years ago, that heparin is located in the mast cells and accounts for their characteristic staining properties. Riley and West have shown that there is a close correlation between the mast-cell content of a tissue and its histamine content, the most striking example perhaps being the mast-cell tumors occasionally found in elderly dogs, which may contain up to 1 per cent of their dry weight as histamine. Graham and her colleagues have likewise shown that the blood basophils, which may be regarded as circulating mast cells, have a very high histamine content. Histamine and heparin thus inhabit the same cell, a finding that makes sense of many old observations. For instance, in anaphylactic shock in the dog the arterial pressure falls catastrophically, and the blood becomes incoagulable: there is enough free histamine in the blood to account for the circulatory effects, and enough free heparin to account for the absence of clotting. The histamine and heparin have come at least in large part from the mast cells, which in many tissues, especially the liver, are found to have been disrupted. Quite recently another powerful substance, 5-hydroxytryptamine or serotonin, has been found in mast cells.<sup>7</sup>

The mast cells, however, are not the whole of the histamine story. The platelets of rabbit blood and the neutrophil leucocytes of human blood contain stored histamine, and it seems highly probable that there is also a good deal in smooth muscle cells and gland cells, that is to say, in cells which are often themselves very sensitive to free histamine.

The actions of histamine fall into three main groups. Most smooth muscle is contracted, and this is true of smooth muscle of the large and medium-sized blood vessels in most mammals. The smallest



arterioles and the precapillary sphincters, on the other hand, are relaxed in man and the carnivores, so that the over-all effect is vasodilatation unless the local concentration of histamine is high enough to cause spasm of the larger arteries. Gland cells are usually made to secrete, the parietal cells of the stomach being particularly sensitive. Finally, capillary permeability is increased, almost certainly by a direct action on the intercellular cement substance, so that the capillary wall becomes much leakier for plasma protein. Most of these actions are opposed more or less effectively by the antihistamine drugs (the effect on the gastric glands being an exception). If, however, the local concentration of histamine is very high, a correspondingly high concentration of antihistamine will be needed, and this cannot usually be attained when the drug is administered by one of the usual routes. Thus the failure of antihistaminic therapy to prevent an allergic response, for example, does not necessarily mean that the response was not mediated by the release of histamine. The histamine may have been released so close to its site of action that enormous doses of antihistamine would be required to antagonize it.<sup>8-10</sup> In general it appears that the effects of histamine-releasing drugs, if not too great, can be prevented by antihistamines if the dose is sufficiently increased, and that the effects of antigens on sensitized tissues are harder to protect against. But the situation is complicated by the fact that not only are the antihistaminics rather unspecific in high dosage, but they are themselves histamine releasers.<sup>11-13</sup> I do not mean to imply here that antihistamine-resistant allergic phenomena are always mediated by histamine. That certainly would be untrue: besides heparin and serotonin a good many other active substances, including various enzymes and polypeptides, are mobilized or activated in allergic reactions.

We may now focus our attention more directly on drugs that liberate histamine. It has been known for some time that a variety of materials like protein precipitants, proteolytic enzymes and the venoms of snakes and bees have this effect. That this is so is not surprising, since such substances are nonspecific poisons that damage a wide variety of cells. It has also been known for a long time that hydrolyzed protein in the form of Witte's peptone produces in the dog effects remarkably like those of anaphylaxis; and during the thirties it was shown, by Dragstedt<sup>14</sup> and Wilander<sup>6</sup> respectively, that both histamine and heparin are released in peptone shock. The first well-defined chemical compounds shown to be histamine releasers were, however, the curare

alkaloids, whose effect was discovered in 1939 by Anrep and his collaborators in Egypt, in the course of a research with quite a different aim. In 1947 Schild and Gregory<sup>15</sup> showed that strychnine and the related alkaloids can release histamine, and Paton and I<sup>16</sup> made a similar observation with licheniformin, a polypeptide antibiotic. Licheniformin was particularly interesting to us since it seemed to have little toxicity apart from its ability to release histamine. We discovered its activity in a roundabout way. When injected into a cat or dog whose blood pressure was being recorded it produced a kind of effect we had not seen before: at first nothing happened at all, but after about 20 seconds the pressure fell precipitously. Analyzing this phenomenon, we found that blood samples taken after the pressure had fallen produced, on injection into another animal, not a delayed but an immediate fall of blood pressure; and we found that this rapid effect was due to the presence of free histamine in the blood. We thought we ought to look for other substances that acted in this way. The chemical structure of licheniformin had not been worked out in detail, but it was known to give a color reaction for the basic guanidine grouping, so we tried a compound called synthalin which is a guanidine derivative and happened to be on the laboratory shelf. Synthalin produced just the same sort of delayed depressor response we had seen with licheniformin, and proved also to be a histamine releaser. With this clue we tested a great number of compounds of related chemical structure, among which we found several dozen with the type of action we were looking for. Among them was one class of therapeutically useful compounds, the diamidines, stilbamidine, pentamidine and their relatives, which had been widely used in Africa for their trypanocidal properties, and we were interested to learn that their side-effects in man, when given by vein, include "a severe but transient fall in blood pressure . . . a curious puffy suffusion of the face and eyelids . . . and itching, generalized and often very pronounced, the grotesque antics of the patient, trying to scratch every part of his body simultaneously, being a source of great amusement to his companions."<sup>17</sup> There seems to be no doubt that these side-effects are due to histamine release. The therapeutic action of the drug, of course, depends on what it does to the parasite, not on what it does to the host.

All these compounds produced similar effects in a variety of tests. In the dog they released heparin along with histamine, but in the cat, only histamine was released. The histamine released in the

dog came mainly from the liver, but in the cat it came mainly from skin and muscle. The released histamine, besides acting on the vessels, constricted the bronchi, stimulated gastric secretion, and so on. Minute doses injected intradermally in man produced the wheal-and-flare response characteristic of histamine. Rodents like the mouse and rat, which are insensitive to histamine, could tolerate relatively large doses of the liberators.

Chemically the compounds we studied were all bases. Those that produced a "clean" effect (i.e., that had no obvious immediate action that could not be ascribed to the histamine they set free) had a rather characteristic kind of chemical structure. They had the general formula RXY, where R was a basic group (amino, amidino, quaternary nitrogen, etc.); Y was another water-soluble group, not necessarily basic, at the far end of the molecule, and X was a sizeable moiety, not too big or small, of predominantly hydrocarbon structure. The addition of water-soluble groups elsewhere in the molecule either reduced the potency or superimposed activity of a different kind on the histamine-releasing activity. This general formula includes a large number of drugs, but it now appears to be much too restricted. Almost any organic base given in sufficient dosage will release histamine; the simplest aliphatic monoamines,<sup>18</sup> and even ammonia,<sup>19</sup> are fairly potent, though they seem less specific and explosive in their action than, say, the diamines or diamidines. Histamine release is also a feature of the toxic action of bile salts<sup>12</sup> and of certain nonbasic detergents,<sup>20</sup> and in rodents it is produced by rather small doses of the plasma substitutes dextran and PVP,<sup>21</sup> which are nearly nontoxic to man and the carnivores.

The relative potency of histamine liberators covers a wide range, and differs greatly according to the test object on which they are compared.<sup>22</sup> Collier and Macauley<sup>23</sup> suggest that for the evaluation of histamine-releasing potency in man, the whealing activity in human skin may be the best guide. By this and several other tests the most active (and one of the most specific) liberators is compound 48/80, a polybasic aromatic substance whose remarkable activity was first observed by Paton.<sup>24</sup> A molecule of this substance under the most favorable conditions will release more than ten molecules of histamine from the skin.

The effects of histamine liberators in man have received remarkably little study. The side-effects of the diamidines have already been mentioned and incidental observations suggestive of histamine release

have been made with many other drugs known to possess this action in animals. Recently Lecomte<sup>25</sup> has given substantial doses of a new histamine liberator, 1935L, to human volunteers. In addition to the effects already mentioned, he noted erythema limited to the face and upper trunk and headache; the clinical aspect was that of angio-neurotic edema. The effects were transient, were only seen when the injection was made quickly, were partly annulled by antihistaminics, and diminished with each repetition of the dose.

The established drugs that have been shown to release significant amounts of histamine include those on the following list (modified slightly from Paton<sup>26</sup>).

|                         |  |
|-------------------------|--|
| Quaternary salts        | d-Tubocurarine<br>Mytolon<br>Synthetic quaternaries related<br>to the methonium series |
| Morphine alkaloids      | Morphine<br>Codeine<br>Papaverine  |
| Other alkaloids         | Atropine<br>Scopolamine<br>Quinine<br>Dihydroergotamine                                |
| Chemotherapeutic agents | Neoarsphenamine<br>Propamidine<br>Pentamidine<br>Stilbamidine<br>Antrycide             |
| Circulatory drugs       | Apresoline<br>Priscol  |
| Sympathomimetic agents  | Amphetamine<br>Tyramine  |
| Centrally acting drugs  | Demerol<br>Strychnine<br>Amphetamine<br>(Various)                                      |
| Antihistamines          |  |
| Protein hydrolysates    |  |
| Protamines              |  |
| Basic amino acids       |  |



The list is certainly incomplete, and no doubt could be greatly extended by anyone interested enough to do a systematic investigation. Indeed it is likely that any basic drug (and probably half of any list of useful drugs would turn out to be organic bases) will release histamine when given in sufficient dosage. This naturally does not mean that effects ascribable to histamine will be seen whenever one of these drugs is given. When a drug has a powerful specific effect, its subsidiary effects may be neglected, including histamine release unless it is a highly potent liberator. Histamine release in unusually susceptible subjects may, however, be at the root of certain "drug allergies," including vascular collapse, nitritoid attacks, urticaria, etc. These effects, it must be stressed, may be produced without previous sensitization to the drug. It is also easy to think of some minor features of the action of certain common drugs that may involve the same mechanism: such are the poor tolerance of asthmatics for morphine and the itching sometimes seen after that drug, and the flushing of the upper part of the body sometimes produced by atropine.

How and where do the histamine liberators act? Probably mainly or entirely on mast cells; they seem to have little direct action on organs like the intestine, which is sensitive to histamine but whose rich stores of this base are for the most part not located in mast cells. Those who are interested in the details of the mechanism may refer to the numerous papers dealing with this subject in the recent Ciba Symposium. It seems clear that the histamine and heparin of the mast cells is located in intracellular particles. These particles can be isolated by differential centrifugation of tissue homogenates, and their behavior in the presence of histamine liberators can then be studied.<sup>27-30</sup> My colleagues and I<sup>29,31</sup> are attracted by the hypothesis that basic drugs are accumulated within the mast cells by virtue of their affinity for the heparin stored there; when a sufficient concentration has been reached, the histamine-containing granules are lysed or become freely permeable for histamine, or the lytic action may extend to the cell membrane itself. The heparin of the mast cells is also released, but most of it is trapped by the tissues and fails to reach the blood stream.

What is the relationship between the allergic or anaphylactic release of histamine and the release of histamine by drugs? The answer to this question is not clear. In some tissues at least the released histamine comes from the same store, in the mast cells apparently, whatever the mechanism of release. The histamine stores of rat skin

can be almost entirely depleted of histamine by repeated dosage with a histamine liberator,<sup>32</sup> the skin replenishes its histamine only over days or weeks, but remains healthy in appearance and shows normal passive anaphylactic reactions with an antigen to which it is sensitive.<sup>33</sup> It would be tempting to suppose that in the allergic reactions the antigen-antibody reaction triggered some enzymic reaction leading to the formation of basic or polypeptide histamine liberators. But experiments aimed at detecting such a process have so far given only negative results; and, as already pointed out, in some tissues the anaphylactic reaction appears to be independent of the presence of mast cells. It is conceivable that by the local application of a histamine-liberator, a sensitized tissue could be depleted of its histamine stores for a long period, for instance the season for a particular pollen: the antigen-antibody reaction to the allergen would then still take place, but would have fewer unpleasant consequences. The sudden unloading of the histamine depots would produce a severe reaction, but this could presumably be controlled with antihistamines and adrenalin. The idea of using histamine liberators in conditions where a prolonged histamine effect is required, as in Ménière's disease, appears rather unpromising because of the steepness of the dose-response curve with these substances and the brief action of the released histamine. At present the chief interest of these substances lies in their usefulness as research tools and in the light they throw on the subsidiary actions of a number of useful drugs.

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## DISCUSSION

DR. AUBREY G. RAWLINS: Mr. President, Ladies and Gentlemen, histamine is decarboxylated histidine, the latter being one of the essential amino acids. Histamine is present in many cells of the body where it is bound in inactive form. Histamine releasing agents discharge histamine so that it may then produce its physiologic and pathologic actions. This activating phenomenon is known as auto-pharmacology.

The two main actions of histamine are:

1. Stimulation of smooth muscle.
2. Dilatation of capillaries with increased capillary permeability. Histamine is one of the few substances known that will dilate capillaries directly. For example, acetylcholine dilates capillaries but does so by acting on the parasympathetic system.

Adrenalin is the physiologic antagonist of histamine while antihistaminic drugs are only blocking agents. In other words, they prevent histamine from reaching the effector cells.

The pituitary-adrenal axis has something to do with the histamine since Bram Rose has shown that in adrenalectomized animals there is a marked increase in blood and tissue histamine.

To me the most astonishing phenomenon about this drug is that here we have a powerful endogenous agent present in many cells in amounts that can produce drastic physiologic and pathologic effects, and yet under ordinary circumstances is devoid of physiologic significance.

For some time we have known about peptone shock, trypsin shock and the release of histamine by snake venoms. Now Dr. McIntosh has not only shown us what happens when histamine is released but from his research has given us a list of, formerly unknown, histamine releasers.

In the past numerous hypotheses have been presented on the mechanism of histamine release. Dr. McIntosh has also clarified this point.



Until recently little respect has been paid to the lowly connective tissue mast cell. If the physiologists don't stop finding new functions for this cell it would appear that all that is needed for survival is a few healthy mast cells. This cell is a large, granulated, basophilic staining, connective tissue cell which first appears in the connective tissue around the blood vessels. Bensley found that the injection of histamine into the skin produced an increase in the mast cells in the injected area. However, Dougherty states that practically any stress or trauma will result in an increase of these cells. Stuart shows that the shock organs in different species contain more mast cells than can be found in their other tissues. Asboe-Hansen believes that one of the functions of these cells is the production of the ground substance of the connective tissue. It has been known for some time that the granules of the mast cells contain a heparin-like muco-polysaccharide and which when released may act as an inhibitor of hyaluronidase. Now Dr. McIntosh gives us another function for this unusual cell, namely, the storage and release of histamine.

I would like to formulate and ask Dr. McIntosh two questions:

Numerous substances are liberated when tissue is damaged in any way. Among these substances are histamine, heparin, acetylcholine, polypeptids and probably hyaluronidase. Mayer has shown that in allergic and non-allergic inflammation of the skin there is a forty-fold increase of hyaluronidase in the local area. Since little of this enzyme can normally be found in the skin it must be present in an inactive form where it can suddenly be activated and released. Mayer has also shown that the inflammatory spreading effect of hyaluronidase is overcome by antihistamine drugs. Does this not show that antihistaminic drugs are also antihyaluronidase factors and that hyaluronidase as well as histamine are liberated in cell damage and play an active part in inflammation? Also will hyaluronidase liberate histamine?

This Association is indebted to Dr. McIntosh for his very interesting presentation. He is an authority on histamine releasing substances and has presented this difficult and scientific subject in a very interesting and useful manner.

DR. SAM E. ROBERTS: Mr. President, Dr. Coates, I would like to ask Dr. McIntosh a question, either for an answer or a postulation. Is there any synergistic action between heparin and histamine when given concurrently by the intravenous infusion? Is there apt to be any antagonistic action between the two?

DR. F. C. MCINTOSH: I would like to thank the discussers and Dr. Rawlins for his interesting suggestion that activation of hyaluronidase may be a feature of mast cell disintegration, in inflammation or when histamine is released. I don't know of any evidence for or against.

In general the mast cells don't survive very long in any real inflammation although in mild chronic injuries, as Dr. Rawlins pointed out, they tend to be increased in numbers. I don't think that histamine release is of any importance in keeping an inflammation going because this release, as I have said, occurs rather explosively if it occurs at all: it takes a long time for the histamine to be put back in the tissues; and the duration of action of released histamine is so extremely short that the whole thing is over and done with in hours at the most, and then cannot happen again for a long time.

I don't know whether hyaluronidase is activated as a result of mast cell disintegration. I think it is an extremely plausible suggestion that would be well worth investigating. Hyaluronidase itself is not a histamine liberator when given by the usual route.

As to Dr. Roberts' question, there is no appreciable synergism or antagonism between histamine and heparin when both are given intravenously. However, the histamine liberators, or most of them, form insoluble complexes with heparin, and heparin is a moderately active antagonist to the histamine liberators. We have been looking for some heparin analogues that might be more effective in this respect, substances that would perhaps not have too much effect on blood clotting but which would knock out the undesirable effects of histamine liberating drugs; but so far unfortunately we have not come across the right type of compound.

## OTOLARYNGOLOGIC ASPECTS OF HYPOMETABOLISM

JEROME A. HILGER

ST. PAUL, MINN.

The essential regulator of cellular oxidation is a catalytic agent—the thyroid hormone. Normal cell metabolism cannot proceed in its absence. This is true for all the cells in the body.<sup>1</sup> This essential substance is probably in its circulating phase, levothyroxin loosely associated with plasma proteins.<sup>2-4</sup> It is the mobile portion of thyroglobulin<sup>5</sup>—the protein-hormone combine formed by the secreting cells of the thyroid gland and stored in the acini of the gland. The hormone is moved from its storage depot in the gland into the blood stream where it is conveyed to every living cell in the body. It is possible that the fourth iodine atom of thyroxin is spent by degradation process and that triiodothyronine is the transient substance immediately effective at cell level. The mobilization of thyroxin from the thyroid gland is effected by the thyrotropic hormone of the anterior lobe of the pituitary gland. The quantitative release of thyrotropic hormone from the pituitary is influenced by the level of the thyroid hormone in the blood and probably by the level of other hormones as well—notably those from the adrenal cortex.<sup>6</sup> A normal blood level of thyroid hormone, in turn, is necessary for the normal functioning of all the endocrine glands.

The circular chain-like action from pituitary to thyroid and back to pituitary is not independent and self-serving, however. Evidence in recent years demonstrates the commanding influence of basal nuclear aggregates in the anterior hypothalamus over pituitary thyrotropin secretion.<sup>7,8</sup> In any case, the final essential in this sequence is the presence of an adequate level of free thyroxin in the plasma. As stated, it circulates principally in a loose association with plasma proteins<sup>9</sup> and may break down to triiodothyronine before it affects the cell. The cellular phase of metabolism in which thyroxin facilitates oxidation in the individual cell is relatively unexplored.<sup>10</sup> It is reasonable to assume that dysfunctions exist even at this peripheral stage.

Hypometabolism as a systemic problem can result from impaired function at any level in the chain. Four etiologic forms have been postulated:<sup>11</sup> 1) hypothalamic, 2) pituitary, 3) athyreotic, 4) somatic or peripheral. The advanced degree of athyreotic hypometabolism causing widespread mucoproteinous changes in the tissue ground substance is commonly called myxedema. This is but one form and one degree of hypometabolism. Hypothyroidism denotes a state of thyroxin insufficiency in the tissue cells. In this sense it coincides with the clinical state of hypometabolism. Hypometabolism is a common clinical condition. Myxedema is relatively uncommon.

Hypometabolism is characterized by a wide range of nonspecific symptoms headed by fatiguability and cold intolerance. It is probably true that no organ is immune to the lowered tone of this semi-vegetative metabolic state. The symptoms evoked make for a complaining, unwell patient readily buried in the psychoneurotic index. Coarse hair, thick skin, infertility, sluggish mentality, and bradycardia may be uniformly indicative of severe hypometabolism. Unfocalized symptoms like constipation, headache, arthralgia, chronic fatigue, fleeting sensory disturbances, transient swellings of face or ankles and others may individually or in various combinations suggest moderate hypometabolism.

#### DIAGNOSTIC METHODS

Starr<sup>12</sup> suggests that the specific diagnosis of hypothyroidism is best made by determining the level of the serum protein bound iodine. It may be presumed that this is made up almost entirely of the circulating thyroid hormone. The normal human level ranges from 4 to 8 micrograms per cent. The hypothyroid range extends downward from 3.8 micrograms of protein bound iodine per cent. This precise determination avoids the many pitfalls of the basal metabolism test and is applicable where the latter is not—as in children, respiratory and cardiac patients and others unwilling or unable to co-operate. This chemical method is useful not only as a determinant of thyroid function but as a gauge of therapy as well. Its limitations are principally those of availability. Many laboratories do not maintain the facility. This, however, is a question of demand.

It is true that the measurement of heat produced in the process of cellular oxidation is a true measure of the metabolic activity of an organism. The amount of oxygen used to produce the heat is an indirect gauge of the latter. A standard range of normal at basal



conditions exists for the human body. Application of this standard in clinical practice is uncertain because it is difficult to attain and maintain basal conditions for the duration of a test period. The admitted inaccuracy of approximately fifty per cent of basal metabolic determinations is the result. Most of the inaccuracies result in elevation of the range above the patient's true normal. For this reason a reported low rate can be accepted as true hypometabolism. The high side errors exclude many hypometabolics from correct diagnosis, however.

The radioactive iodine ( $I^{131}$ ) uptake of the thyroid gland is probably the simplest of the radioactive iodine methods of estimating thyroid function. The uptake, a specific interval after ingestion of 15 to 25 microcuries of  $I^{131}$ , is measured with suitable apparatus and techniques. The usual interval is 24 hours though this has been modified downward to as little as six hours. In the 24 hour method results are said to be 80 to 85 per cent accurate in their relation to the true thyroid function.<sup>4</sup> Uptake values of 55 per cent or higher are indicative of hyperthyroidism. Values of five per cent and below show the presence of hypothyroidism.<sup>1</sup> The zone from five to ten per cent is suspect.

The use of iodides and thyroid decreases radioactive iodine values as long as six weeks after discontinuance. Estrogens, cortisone and other steroids can also decrease the uptake of the thyroid. Presumably this is effected through pituitary inhibition. False low values may be obtained under these circumstances. Conversely in hypopituitary hypothyroidism uptake values may be in the low normal range and the diagnosis thereby missed.

In most instances the uptake of radioactive iodine and its release from the thyroid gland as hormone run parallel. There are circumstances where there is divergence. In such instances the uptake value does not indicate the quantity of circulating hormone. The measurement of protein bound radioactive iodine (hormone) in the blood a fixed interval after ingestion, or the measurement of the rate at which it appears in the plasma should therefore be a more accurate indicator of hormonal status than the uptake determination. Unfortunately, these plasma methods do not distinguish between the euthyroid and the hypothyroid satisfactorily.

From the foregoing it is apparent that in the laboratory aids to the diagnosis of hypothyroidism there is room for improvement. The

basal metabolic rate method is useful in diagnosing hypometabolism. Unfortunately, it commonly errs to the high side. The radio iodine methods may falsely imply a euthyroid to be low and a hypothyroid to be normal. The chemical determination of the protein-bound iodine of the plasma is the most certain method. Second in value and often useful for confirmation is a properly performed basal metabolic rate determination.

Diagnosis by therapeutic test with thyroid substance is not very satisfying. Hypometabolism is clinically vague and deceiving. A level of hormonal adequacy is not built up over night. Medication has to be persisted with and often increased gradually over long periods of time to obtain optimal results. All of this is difficult to persevere with on a "hunch" indication alone. The price of unrecognized hypometabolism in our field is long-standing disability and sometimes, irreversible pathology. Preventive therapy must be long term, sometimes life-long. Therefore a firmly based diagnosis rather than a presumption should be made at the outset.

#### HYPOMETABOLISM IN OTOLARYNGOLOGY

In otolaryngology hypometabolism is a frequent cause of presenting symptoms.<sup>13,14</sup> The otolaryngologist has an excellent opportunity to recognize it. The larynx, the upper respiratory mucosa, the external and middle ear, the pharynx, the cranial vessels, or the inner ear may be the focal point of symptoms.

#### THE LARYNX

Chronic vocal cord edema not associated with inflammatory reaction is strongly suggestive of hypothyroidism. It is firm, not deeply reddened unless it bears the brunt of excessive vocal trauma, and it is uniform in its involvement of the whole cord. Because of greatly increased cordal thickness the voice is hoarse and of low pitch. Most patients with myxedema grade of hypothyroidism have a low, coarse voice. Advanced degrees of cordal myxedema may result in diffuse polypoid change requiring surgical stripping. The gelatinous character of the submucosa due to the mucoproteinous change in the tissue ground substance makes separation of the edematous masses from the underlying elastic tissues of the cord relatively easy with the usual cupped forceps technique. The focalized myxedema and polyps will recur if the hypothyroidism is unsuspected and untreated.

## REPORT OF A CASE

Mrs. C.S., a white female aged 64, was examined on May 25, 1953. She had been hoarse for more than a year. The hoarseness fluctuated in degree from week to week but was always quite severe. Diagnosis of vocal cord polyps had been made by a competent laryngologist in a distant part of the country some months before. It was his suggestion that the polyps be removed forthwith.

The patient was chronically beset with great fatiguability and cold intolerance. She had a very apparent fluid-holding tendency. There was loose, pouchy edema of the lids. Puffiness of the fingers resulted in indentations from her rings. Her ankles and feet were edematous. Both vocal cords were edematous in the extreme with their medial aspect tensely polypoid.

The patient's internist agreed with the tentative diagnosis of severe hypothyroidism. He found her also to be moderately hypertensive.

The basal metabolic rate proved to be minus 27 per cent. In an apprehensive patient who accommodated to her test inconveniences with less equanimity than was perfectly desirable this was a very significant figure since it probably erred on the high side.

In the course of the next three months with thyroid replacement the patient lost a great deal of fluid and face and extremities took on a healthy tissue appearance. The vocal cord mucosa was loose and floppy along the whole free margin. Though much of the edema was gone there was insufficient elasticity in the membrane to return the lining to normal tonus. To a more limited degree this was also true of the pouchy skin of the lower lids. Her maintenance dose of desiccated thyroid levelled optimally at  $2\frac{1}{2}$  grains a day.

Not until June 13, 1955, did the patient pause in her worldly wanderings sufficiently long to have the vocal cords stripped. Healing thereafter was prompt and her laryngeal function and appearance have remained good.

Bilateral vocal cord paralysis is caused most frequently by extensive thyroid surgery. A thyroid excision that involves both recurrent laryngeal nerves is likely to be so complete that hypothyroidism results. Protein-bound iodine measurements or radioactive iodine studies are indispensable to this diagnosis. The respiratory apparatus of the metabolometer is not usually applicable to the obstructed or tracheotomized patient.

The respiratory obstruction due to median vocal cord position can be significantly increased by the mucoid edema of the cordal mucosa. The airway can be improved measurably by restoring the normal mucosa with thyroid replacement. This is partially offset by the increased respiratory demand of the improved metabolic state.

## REPORT OF A CASE

Mr. R.D., a 69 year old white male, was examined on February 25, 1955. His history was interesting. In September 1954 he had a near total thyroidectomy for

multiple-adenomatous goiter. A few hours postoperatively an emergency tracheotomy was performed because the patient's condition had deteriorated due to respiratory obstruction, the gravity of which had not been adequately appreciated at the conclusion of surgery. Several efforts were made in October, November and December, 1954, to decannulate this patient without first visualizing his larynx. They were nearly disastrous. In January, 1955, the patient complained of buzzing and roaring in his ears and of fluctuating hearing level. At this time he also had a period in which he had hallucinations and "heard voices." Throughout the winter months he had cold intolerance and increasingly slow cerebration.

The patient's whole physical appearance was that of well established myxedema. A tracheotomy tube was present in high position. Both vocal cords were immobile in the midline. There was firm edema of both arytenoid and ventricular mucosal areas with a reddened inflammatory appearance on the right side. The vocal cords were immobile and in the midline and it was difficult to visualize their whole length because of the edematous supraglottic mucosal overhang. His tongue was thick and clumsy. The upper respiratory and pharyngeal mucosa was thick and edematous. There was bilateral hearing impairment with a sharply downsliding audiographic tracing from the 20 decibel level at the frequency 512 to 80 decibels at 4096.

The radio-iodine uptake was 2.3 per cent and the conversion to protein-bound (plasma hormonal) iodine was low. The interpretation was myxedema level.

One-half grain of desiccated thyroid daily for a week was given and produced no sign of cardiac overload so increase was made to one grain daily.

Preoperative preparation demanded long term replacement to attain optimal metabolic tissue rejuvenation. However, the high position of the tracheotomy tube and reaction in the right cricoarytenoid area were disturbing evidence that cricoid perichondritis was in process. Accordingly laryngoplasty and tracheotomy were effected on March 4, 1955. The cricoid had been violated by the initial "laryngotomy." This area was repaired, the trachea brought from beneath the sternum by traction, and tracheal tube inserted through the second ring.

The soft tissues exposed in this surgical effort were pale and edematous and gave forth a myxomatous ooze when incised. Healing time was very prolonged.

By mid-April, 1955, enough laryngeal edema had cleared that visualization was better. The left vocal cord had sufficient motion to swing past the midline and phonate against the immobile right cord. The left vocal cord had poor abduction power, however. Immediately subcordal there appeared to be a semistenosing bar presumably as a result of the laryngostomy chondritis.

This patient's myxedema is now well controlled. As with most myxedema patients four grains of desiccated thyroid per day caused gradual signs of hyperthyroidism which were unrecognized until auricular fibrillation developed. His family doctor downgraded his overdose to a maintenance level of two grains per day. His physical well being is "normal." For average activity he can keep his tracheotomy tube completely taped. Decannulation has not been done because his previous occupation called for physical exertion beyond average. At present he is determining his obstructive threshold with a full daily work load. It is probable his right vocal cord will need lateral displacement.

Hypothyroid tissues heal poorly. The normal character of the processes of wound healing are grossly altered. To those who have



incised the soft tissues of the myxedematous patient this is not new. One does not forget the mucoid character of the interstitial fluid ooze. To preclude delayed healing after arytenoid surgery the thyroid status should be determined beforehand and the necessary pre-operative weeks be invested in establishing normal metabolic balance. For the hypothyroid patient controlled with replacement therapy, the satisfaction of arytenoid surgery and decannulation can be nullified by a relapse into the hypothyroid state. The return of mucoid edema at the glottic level can revert the postsurgical adequate airway to a condition of respiratory embarrassment. It is difficult for some patients to see the necessity for continuous thyroid therapy. Their sense of well-being when in normal balance suggests that they are too well to be taking medicines. The price of the lapse is not paid for several months. By that time developing symptoms are not associated with lack of a medication long since discontinued. For the patient with an adequate airway and a good voice following arytenoid surgery the gradual return of laryngeal myxedema can vitiate the result.

#### THE NOSE

Mucoproteinous edema of the nasal mucosa is productive of chronic nasal obstruction. A change in the character of the nasal secretions to a more tenacious and mucoid quality creates the additional annoyance of postnasal accumulation. Enough has been said and written of the problem to alert the specialty to its occurrence.<sup>15</sup> It only takes a case refractory to prolonged nasal piddling, surgery, and allergic management but spectacularly responsive to adequate thyroid replacement to enlighten the clinician and broaden his concepts. The patient is usually as grateful for the systemic benefits as for those nasal. His improved vigor, bowel tonus, cold tolerance, arthralgia, etc. when analyzed in retrospect embed these important accessory data in future diagnostic questioning. The frailties of a specialized history are principally those of narrow vision.

#### REPORT OF A CASE

Mr. S.V., a 36 year old male, was first examined in November, 1953. For over five years he had had persistent, severe nasal obstruction and mucoid, tenacious postnasal drainage. His symptoms were always worse in the wintertime. He had had frequent local nasal treatments of various types much of that time. He had a well-developed Privine habit, and used the drops particularly frequently at night. Two months before examination he had a left upper molar tooth removed and coincident

with this or subsequent to it he developed an acute suppurative left maxillary sinusitis. His family physician gave him penicillin by injection.

At the time of examination there was no residual suppuration but edema of the left maxillary sinus mucosa was still present. The rest of the sinuses were clear and normal. The nasal mucosa was exceedingly edematous, pale and wet. It showed very little decongestant response to 2 per cent ephedrine solution. In the week between initial examination and appointment for basal metabolic rate determination there was no significant response to antihistaminics. Basal metabolic rate was reported minus 36 per cent and the blood cholesterol 187 mg per cent. There was slow gradual improvement in the next three months as the daily dose of desiccated thyroid was increased from one-half grain daily to one and one-half grains daily. The latter dose is an optimal daily dose for the patient and has maintained complete nasal comfort regardless of season, for the past two years.

#### THE EXTERNAL AND MIDDLE EAR

One of the usual symptoms of hypothyroidism is a reduction of sweat gland activity. Frequently the function of ceruminous glands suffers the same fate. The dry, itching external ear which results is a frustrating problem unless the diagnosis of thyroid deficiency is made. Improvement is not obtained with local treatment.

This would be a simplified clinical world if some of our vexing chronic or recurrent problems were demonstrated to have single or specific etiologies. Response to a single "standard" treatment could then be expected. If, for example, chronic auditory salpingitis and effusive otitis media would respond to the empiricism of nasopharyngeal irradiation with uniform and lasting results we might be spared the necessity of making an etiologic diagnosis. After the initial flush of irradiation enthusiasm we are returned to the arduous requisite of the diagnosis first. One suspects the dermatologist came to grips with the same harsh reality in his effort to control a rambling range of dermatitides with superficial irradiation.

Some effusive ears are bettered by good allergic management and some by thorough nasopharyngeal surgery and middle ear aspiration. But some have long-standing betterment from nothing at all except correction of a chronic thyroid deficiency.<sup>16</sup> In many of these, concomitant lymphoid hyperplasia can be permanently controlled with adequate thyroid replacement. This is the same lymphoid hyperplasia which defies repeated and adequate removal.

#### REPORT OF A CASE

E.W., a white male aged nine years, was examined on January 28, 1948. He had a chronic problem with nasal obstruction and mucoid drainage in excessive

amounts which had no seasonal pattern. It was his parent's impression that he had a hearing handicap of long standing.

This boy had had tonsillectomy and adenoidectomy performed by his pediatrician three years before.

Juicy, edematous mucosa was the outstanding feature in the nose and nasopharynx. There was diffuse lymphoid hyperplasia centrally in the nasopharynx, in the tubal area and downward into the lateral pharynx. There was effusion in both middle ears and hearing impairment averaging 20 to 30 decibels in a flat tracing for both ears.

This boy was referred for consideration of nasopharyngeal irradiation and I, myopic clinician, concurred. On three occasions at the usually recommended three week intervals the usual 50 milligram nasopharyngeal applicator was placed over the tubal area for the usual twelve minutes to the side. In addition the patient was placed on a diet restricting several of the commoner food allergens.

Three months after the initial examination and treatment the middle ears were ventilating well, hearing was normal and there was diminution in the mucosal edema in the nasopharynx.

A year later the hearing was still almost normal, but the patient's mother was unhappy with his chronic nasal behavior which was diagnosed chronic vasomotor rhinitis. She was advised to bear with it lest the indicated vasomotor or allergic regime be not more troublesome than the disease.

On several subsequent occasions the mother expressed distress with the chronic nasal symptoms and was certain that intermittently the patient had periods of hearing impairment.

By February 28, 1956, the patient—having survived all of the above—was of an age (18 years) to speak for himself. He had had two miserable winters with his nose. The summers had been reasonably comfortable. In spite of the extensive background on our records he volunteered that two years was as long in retrospect as he could recall any nasal problems.

There was no question but that this patient's complaints were justified. His nasal mucosa was juicily edematous to an advanced degree. He breathed through his mouth as a matter of habit and necessity.

There was no ring of true allergy to the problem. There was no additional information gleaned from the history as he gave it. Arbitrarily he was referred for radio-iodine study. His uptake was 8.3 per cent. His conversion to circulating hormone was low. He became asymptomatic in a month with a daily dose of one grain of desiccated thyroid.

At this point this patient's history was pursued further with direct questioning of his mother relative to thyroid deficiency. The fallibility of history-taking and the wisdom of maintaining thyroid-consciousness in our specialty at all ages became evident. At the age of nine months an astute pediatrician diagnosed thyroid deficiency and bettered the infant with replacement therapy. It was later discontinued, presumably through indifference and well-being. The same pediatrician referred him for care and irradiation at the age of 11 years when his nasopharynx became a focal point of distress and recurrent lymphoid hyperplasia. The key to long-standing therapeutic benefit at that point lay in a good thyroid history and evaluation not in fiddling with a radium bomb or elimination diets.

## THE PHARYNX

Mucoproteinous edema of the pharynx initiates symptoms which though vague are persistent,<sup>17</sup> which, though unattended with objective inflammatory signs are pursued with therapeutic vigor ranging from local applications and antibiotics to tonsillectomy. The symptoms are chronic and long-standing and described variously as "sore throat," "throat discomfort," "lump in the throat," etc. True inflammation is absent. A suggestive pale and glossy edema of the pharyngeal mucosa is sometimes discernible. An inquisitive search will usually elicit a history of additional systemic symptoms of hypothyroidism. When the specific diagnostic tests are confirmatory the results with thyroid therapy are very pleasing.

## REPORT OF A CASE

Mrs. C.R., a 34 year old white female, was seen in February, 1955. Her problem was recurrent sore throat and a feeling as though she had something in the hypopharynx which defied clearing. Her symptoms had been annoyingly present for years—sufficiently long so the exact time of onset was not clear in her mind. Though the winter season was the most aggravating period she felt that even in summer her throat was chronically troublesome. She had had tonsillectomy, repeated cycles of local treatment, and numerous antibiotic series. On several occasions she had been told there was nothing remarkable to be seen in her throat.

The patient's general history was not remarkable except for great fatiguability (she was often more tired in the morning than when she went to bed), cold intolerance, constipation and arthralgia. The latter was particularly troublesome in her hands. It took a period of use in the morning to get her fingers limber. She spoke of this problem as arthritis and felt it was in some way related to the "infection" in her throat.

This patient's examination was remarkable for the absence of inflammatory sign in her pharynx and upper respiratory tract. There was a suggestion of pale, glossy edema of the pharyngeal mucosa—particularly of the posterior wall. It was not outstanding.

Her internist reported her general condition as satisfactory other than a mild secondary anemia. Her basal metabolic rate was reported as minus 28 per cent.

Dessicated thyroid grains one daily was given and three weeks later all of the patient's symptoms had improved greatly. After six additional weeks with grains one and a half daily she was free of throat symptoms, her arthralgia was gone, and she had normal vigor and sense of well being. She has had no problem with her throat since.

## VASCULAR HEAD PAIN

The walls of the external carotid arterial tree are amongst the most active and symptomatic tissues in the head region. A wealth



of clinical fact and hypothesis has accumulated around the subject of vascular head pain. No single formula for treatment has uniform success. Again the stumbling block is that of multiple etiology and the necessity in any case of an individual diagnosis. The factors of autonomic dysfunction and stress are well known. The close association with endocrine balance is evident in the cyclic history of some female sufferers. That the edematous substrate in the vessel wall of the hypothyroid sets the stage for some is less well appreciated. The classic myxedema heart undergoes this change to an extreme degree. Headache of vascular character and distribution is not uncommon as a presenting symptom in the hypothyroid patient.<sup>19-22</sup> It is relieved by adequate thyroid replacement. Reasonably enough it is not affected by the vast panorama of agents and techniques accumulated as easy single answers to this problem of multiple causation.

#### REPORT OF A CASE

Mr. R.T., a 27 year old white male, was seen in November, 1954. He had frequent head pains which he referred to as "sinus headaches." He had been troubled with them for several years. The aching was occipital as well as frontal. They were not cyclic in the manner of most vascular head pains. He had them almost every week. He also had moderate nasal congestion and postnasal drainage which clinched the diagnosis of sinusitis in his mind. The only seasonal variation of his headaches was increased severity in the cold weather. He felt worse generally in winter than in summer. He had no family background for his headaches. He seemed to be a well adjusted individual and professed no tension problems. Systemically he had no problems other than chronic fatigue. The latter had prompted him to have a general physical examination. No abnormalities were found.

The patient's ear, nose and throat examination revealed nothing remarkable. He certainly had no sinusitis. His nasal mucosal changes were those of a mild vasomotor rhinitis.

This patient's basal metabolic rate proved to be minus 29 per cent. He was given dessicated thyroid grains one and a half daily. When seen six weeks later he had had freedom from headache for almost three weeks—a quite remarkable achievement by his standards. Recheck two months thereafter confirmed the continuance of general well being and vigor and of freedom from head pain.

#### THE INNER EAR

Endolymphatic hypertension is descriptive of the inner ear phenomenon which became recognized a pathologic entity through the investigations of Hallpike and Cairns.<sup>23</sup> The gross dilatation of the endolymphatic system presented by them accounted for the advanced symptoms and clinical signs, both acoustic and vestibular, which

have been termed variously Ménière's disease, endolymphatic hydrops, labyrinthosis, etc. Study of large groups of cases has demonstrated certain fundamental properties common to all. The disorder is first, (sometimes for many years or for its duration) a cochlear disturbance and secondly, vestibular. It is a disease of variable degree, from mild to destructive. It frequently remits spontaneously or by virtue of treatment. Its mild degrees are as reversible and advanced degrees as disastrous as those of vascular hypertension. Like vascular hypertension, every evidence suggests endolymphatic hypertension is a disorder of multiple etiology. Therefore it is apparent why no single approach to treatment has produced uniformly good results.

Endolymphatic hypertension requires first, an otologic diagnosis. The criteria are clear cut. Diagnosis in the early cochlear phase is a triumph and results in retained function. Aural pressure, broad spectrum tinnitus, distortion with impairment of discrimination beyond the degree of pure tone loss, the early flat or rising audiographic line, and recruitment are diagnostic. When the symptoms are minimal pressure, tinnitus and discrimination loss, tubal nonsense should be abhorred. The eager and repetitious catheter has been a poor substitute for diagnosis much over-long.

Advanced hearing impairment with a downsliding tracing, crippling distortion and frequently cyclic vertigo denotes the late stage of endolymphatic hypertension. Not too late, however, for a specific etiologic diagnosis. The old contention that this is a unilateral disease and not likely to affect the other ear is not true in the hypothyroid case. Bilateral involvement, early or eventual, is common.

The mechanism of endolymphatic fluid production and resorption remains an enigma. The typical clinical course of endolymphatic hypertension indicates that fault lies in excessive fluid production rather than faulty resorption. The dramatic suddenness of some attacks suggests a dynamic vascular or secretory influence. This is typified in the occasional but well documented ingestant allergic case. Clinical parallelisms with vascular autonomic dysfunction elsewhere in the body indicate that for some patients this is the basic mechanism of the endolymphatic hypertensive disturbance.<sup>24-26</sup> Certainly for many patients endocrine imbalance plays a prominent role.<sup>27</sup> In the production of endolymphatic hypertension it frequently loads the gun that stress may pull the trigger. Hence for these patients the etiology is a composite of an endocrine background inadequacy and an immediate precipitating stress.

The relation of hypothyroidism to the inner ear is not a recent observation. Deafness is common in cretinism. In the early writings about myxedema VIII nerve neuritis was cited as a common occurrence. In many illustrative cases of myxedema in the literature hearing impairment was included in the long list of symptoms in the untreated patient. Interestingly enough among the improvements resulting from thyroid medication hearing gain was remarkable in some.<sup>28,29</sup> A summarization of knowledge of the thyroid gland, its function and disorders published more than ten years ago<sup>30</sup> drew attention to the relation of the ear to hypometabolism as follows:

"When deafness is associated with a lowered metabolic rate, either with or without the picture of myxedema, some improvement in audition frequently follows the administration of thyroid. This apparently holds true both in nerve and in middle ear deafness. For this reason it is recommended that the basal metabolism be determined in all cases of deafness.

"The vertigo of Ménière's syndrome may be alleviated greatly by thyroid administration, probably because of its dehydrating action."

These observations are penetrating. The empirical approach to "all cases of deafness" is unnecessary for the otologist. He can separate effusive otitis media and endolymphatic hypertension from the broad generalization of "deafness." To these former he can direct his thyroid investigation.

For the patient with endolymphatic hypertension who is found wanting in thyroid hormone one suspects that replacement therapy has more profound physiologic implications than simple "dehydration." In the minute anatomy of the stria vascularis and the nature of its supporting stroma, its vulnerability to mucoproteinous degeneration can be readily envisioned.<sup>32</sup> Alteration thereby in the control of endolymphatic fluid balance is a tempting hypothesis in the hypothyroid case. At any rate the hypothyroid endolymphatic hypertensive responds well to thyroid adequacy. The stresses of active life which previously precipitated endolymphatic crises are borne with labyrinthine equanimity.

#### REPORT OF A CASE

Mrs. J.D., a 26 year old white female, was seen on August 26, 1952. Ten days prior to examination she noted a plugged feeling in both ears when she awakened.

There was no tinnitus, but sound was distorted. Six days later she experienced a mild vertiginous episode lasting ten minutes. Again on the morning of examination she had a similar attack. History evoked no other pertinent points.

The only physical findings of significance were those related to the inner ear. The patient exhibited recruitment, normal caloric response, inner ear type impairment with audiometric tracing as shown in Figure 1a.

Treatment with vasodilators and blocking agents intravenously and orally, salt restriction and diuretics for a period of three weeks was attended by improvement in the less involved ear to normal (Fig. 1b).

Though there were no suggestive signs of hypometabolism other than fatigability, a basal metabolic rate showed minus 42 per cent with blood cholesterol of 248 mg per cent. One grain of thyroid was given each day for a week. At that time all imbalance and the sense of aural pressure were gone from the ear. The pure tone audiogram had not yet improved. Thyroid was increased to two grains daily. By December 20, 1952, all symptoms had subsided and hearing was normal (Fig. 1c).

Otosclerosis and endolymphatic hypertension sometimes coexist. Here an appreciation of the influence of the hypometabolic state on the inner ear is valuable preoperatively and postoperatively. It can alter the long term results of otosclerosis surgery.

#### REPORT OF CASES

Mrs. R.L., a 28 year old white female, was examined on February 9, 1953, because of hearing impairment. This had started in the right ear shortly after high school age and subsequently involved the left ear as well. There was a mild tinnitus in the right ear which did not annoy her particularly. She had never had vertigo. She was an adopted child and did not know her own family background. She was in good general health, and had not had physical examination in recent years.

Initial audiogram is seen in Figure 2a. Fork tests confirmed the bone conduction tracing. Her diagnosis was otosclerosis with cochlear defection of undetermined origin.

General examination demonstrated no abnormality other than mild secondary anemia. Basal metabolic rate was minus 24 per cent and blood cholesterol 167-mg per cent.

The patient was given two grains of thyroid per day and reexamined three weeks later. Subjectively she was delighted with her hearing improvement and it was borne out by testing (Fig. 2b).

On September 8, 1954, one and a half years later, her well-being and her cochlear reserve were both of a high order (Fig. 2c).

Mrs. C.D., a 34 year old white female, was examined January 16, 1950. She had bilateral hearing impairment which had been increasing since age 18 years. Her diagnosis was bilateral otosclerosis with a cochlear reserve adequate for fenestration. This was performed for the right ear on March 20, 1950. Her result was good as shown by her nine months postfenestration air conduction audiogram (Fig. 3a).

This level was maintained through the following years until the fall of 1955. On December 29, 1955, she gave the following history:



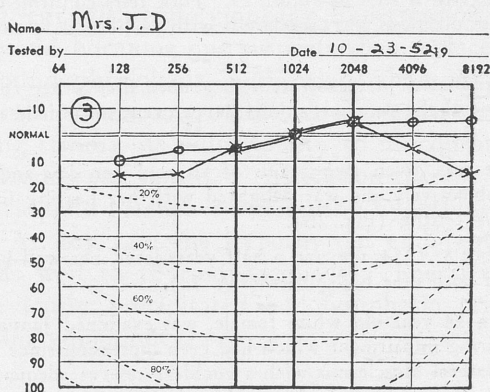
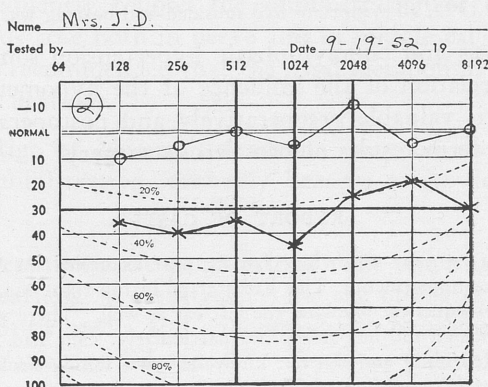
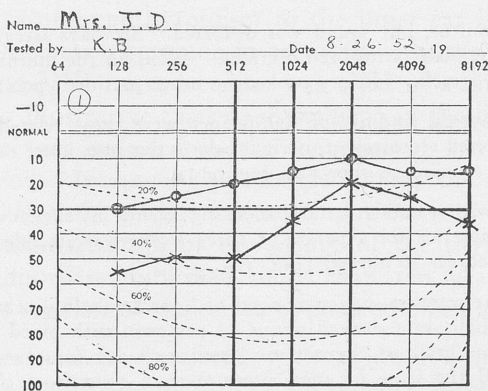


Fig. 1 (a).—Mrs. J.D. Pure tone hearing level at initial examination. Bone conduction and fork testing indicate the loss is purely inner ear. Recruitment is present in the left ear. Caloric responses are normal. Note moderate loss in right ear indicating bilateral involvement. (b) After three weeks of therapy with intravenous and oral vasodilators and blocking agents, salt restriction, and diuretics. Basal metabolic rate minus 42 per cent, blood cholesterol 248 mg per cent. (c) After three months of adequate thyroid replacement.

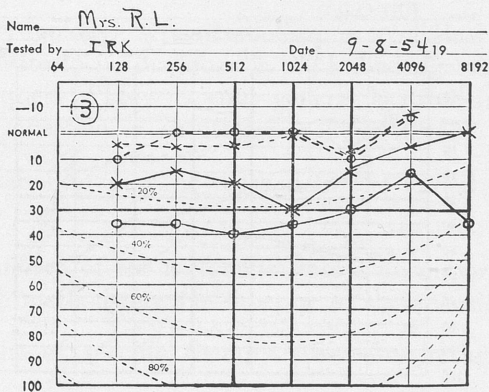
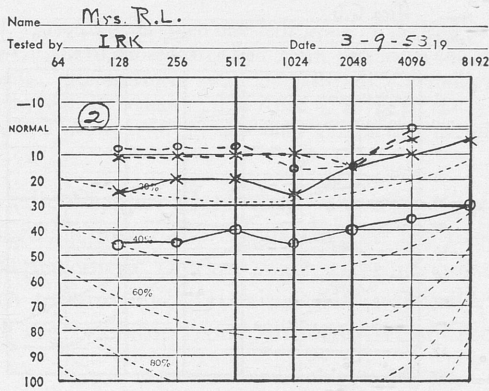
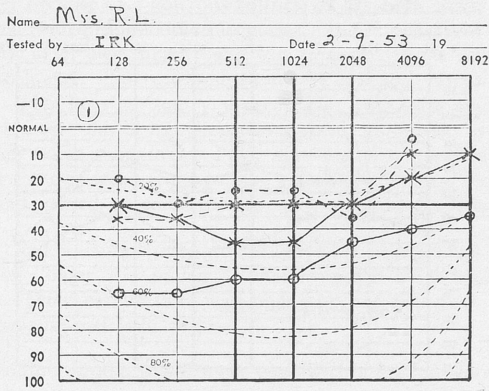


Fig. 2 (a).—Mrs. R.L. Initial audiogram. Cochlear defection confirmed with fork tests. Basal metabolic rate minus 24 per cent, blood cholesterol 167 mg per cent. (b) After two grains of desiccated thyroid daily for three weeks. (c) After one and one-half years of normal metabolic level.

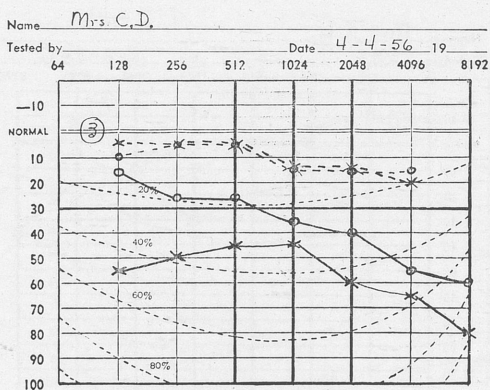
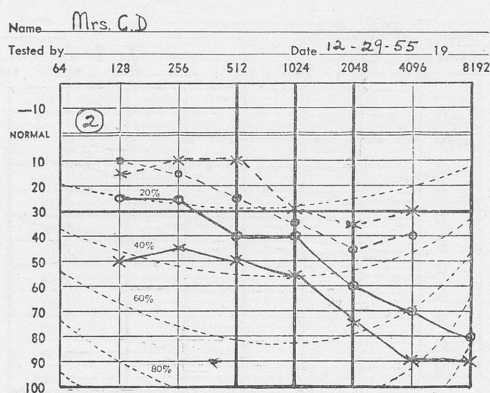
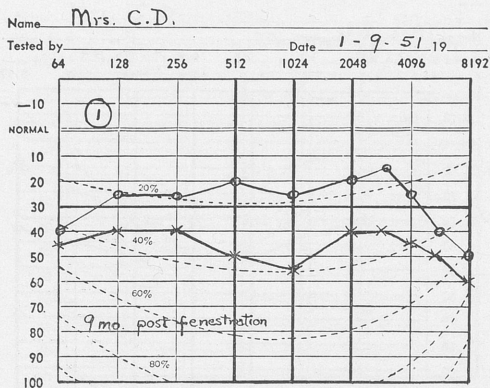


Fig. 3 (a).—Mrs. C.D. Post-fenestration (nine months) air conduction audiogram. (b) Two months after near-total thyroidectomy produced postoperative myxedema. Radioactive iodine uptake 6.5 per cent and low plasma hormone conversion rate. (c) Three months later with normal metabolic balance restored with 0.8 milligrams thyroxin (Squibb) daily (2 grains of thyroid equivalent).

"In September because of a goitrous fullness of her neck she saw a general physician. He diagnosed multiple adenomatous goiter. In October, 1955, she had thyroidectomy. A month after her surgery she was disturbed by a sense of fullness and even pressure in both ears. There was also buzzing low and variable frequency tinnitus in both ears. Her hearing acuity decreased in both ears and she was sure her fenestrum was closing. Mild lightheadedness and giddiness, as she described it, became troublesome some mornings. First movements on arising or sharp changes of position during the day were prone to give a flash of dizziness requiring a moment's steadying. She had gained 10 to 20 pounds in weight in three months."

This patient had a completely altered physical appearance from her normal. She appeared much "older and puffier." Her skin was thick and dry. Her lower lids were pouchy. Her cerebation was slow but not too slow to be exceedingly depressed by her hearing loss (Fig. 3b).

Radioactive iodine uptake was 6.5 per cent with a low conversion rate to plasma hormone.

On January 9, 1956, she was given one-half grain of thyroid per day. Telephone report on January 30, 1956, noted freedom from giddiness and aural pressure and definite hearing gain. Replacement was increased to 0.8 milligrams of thyroxin daily (2 grains of thyroid equivalent). On April 4, 1956, all ear symptoms including tinnitus were gone and hearing for air and bone conduction sharply improved (Fig. 3c).

The old medical school adage that "treatment is easy once diagnosis is established" is never more true than in the problem of hypothyroidism. Replacement therapy with thyroid substance or, more accurately, with sodium levothyroxin itself is specific. The philosophy and dosage are available from authoritative sources.<sup>1,11,31,33</sup>

#### CONCLUSIONS

In otolaryngology hypometabolism is a clinical commonplace. The first requisite to its diagnosis is the knowledge that it is a daily visitor. The query, "How is your general health," is the most important single question in an otolaryngologic history.

Hypometabolism may be masked in the varied symptoms discussed.

The evaluation of the metabolic state is an important diagnostic effort. Precise methods are far superior to therapeutic guessing.

The results of specific therapy are a fine return for accurate diagnosis.



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#### DISCUSSION

DR. FRANCIS W. DAVISON: Dr. Coates, Dr. McMahon, and Fellows, Dr. Hilger has given us a beautiful example of the fact that medical otolaryngology is really important. Dr. Hilger left me very little to say except to mention a few brief case reports to show that the otolaryngologic symptoms of hypometabolism are the same in central Pennsylvania as they are in Minnesota.

One patient, Bessie W., age 65, is a thin person. We sometimes forget that hypothyroid people can be quite thin. Her chief complaints were hoarseness and nasal obstruction. Examination showed pale edema of the vocal cords and nasal mucosa. She in addition had marked subocular edema, thinning of the outer half of the eyebrows and a perceptive type hearing loss more marked for high tones. Her protein bound iodine determination was 2.34 micrograms. For our laboratory the mean PBI value for euthyroid patients is 5.3 micrograms. She was treated with desiccated thyroid, grains 2 daily and her symptoms cleared up in about three weeks.

One of the questions I find helpful in detecting hypothyroidism is: "At what temperature do you keep your house, 70, 75 or 80 degrees?" When they say "80," I suspect hypothyroidism.

Another patient, Edgar L., 55, whose chief complaint was a recurring hearing impairment due to middle ear effusion, I have observed for the last 15 years. He is entirely comfortable if he continues to take two grains of thyroid daily. His middle ear effusion recurs if he discontinues the thyroid for a month. His family physician advised against thyroid on one occasion because his basal metabolism rate was only minus 6 per cent. In many cases I ignore the laboratory data and rely on the clinical response in determining the dose of thyroid. This patient also had sub-ocular edema and a very dry skin when not taking thyroid.

Another patient, Ida K., aged 68, who weighed over 200 pounds, had a chief complaint of recurring hearing loss due to middle ear effusion. The diagnosis of hypothyroidism was easy because this patient had had x-ray therapy for hyperthyroidism some twenty years ago, and thereafter gained considerable weight. I have

observed this patient for the last 12 years. She also has recurrent middle ear effusion whenever she discontinues thyroid for more than a month at a time. Her maintenance dose is 2 grains of thyroid daily. This patient also was told by her doctor to discontinue thyroid because there were to his mind no obvious symptoms suggesting hypometabolism, even though she had the dry scaly skin and marked subocular edema so frequently seen. It is surprising how many people, both doctors and patients, have a phobia about taking thyroid. I agree with Dr. Hilger that in case of doubt it is wise to have a PBI determination to have laboratory confirmation of the hypothyroid state.

DR. LEWIS F. MORRISON: I take this occasion to commend our president for selecting a subject of this type and for his selection of a man to present this subject. No one could improve on either selection. However, there is one bad feature. The author's command of the English language and his mode of presentation is so smooth and so acceptable that for average people such as I am certain of the informative facts could slip by unless they are set apart a bit for attention and emphasis.

First is the matter of terminology. The separation of hypothyroidism and hypometabolism as two distinct clinical entities, in spite of the fact that both have a common etiology and are actually the same thing or at most not more than different degrees of manifestation of the fundamental condition, has proved to be advantageous and has resulted in the saving of considerable time and energy in being able to avoid unnecessary controversy. When talking with one's medical colleagues, it is better to be mindful about semantics or the argument may continue late into the night and still produce nothing of material benefit for the patient.

Another point of information revolves around the laboratory tests. To have a stated normal range from minus ten per cent to plus ten per cent is absurd if not stupid. Almost all the errors that are going to occur will add to the plus readings. Arguments on this subject several years ago resulted in performing a practical experiment at the expense of the taxpayer. More than twenty patients were admitted to the hospital singly or in small groups and were required to remain there for a period of at least five days. The candidates selected were obtained from a group who had no demonstrable reasons for the complaint of a stuffy nose and whose basal metabolic rate determinations were reported to be slightly on the minus side. The majority of these patients had recent basal metabolic rate determinations reported to show a minus 3.0 per cent or 4.0 per cent rate determination. Under ordinary circumstances one would accept such ratings as being within the limits of normal.

It was interesting and informative to watch the changes in readings obtained by subjecting these candidates to daily basal metabolic rate determinations. It was not uncommon to find that although the Monday determination may have been on the minus side not greater than 5.0 per cent, the reading on Friday would range somewhere between minus 23.0 per cent and minus 35.0 per cent. One is permitted to infer from these results that the readings taken during the latter part of the week represented a more true determination of the basal metabolic rate than did those obtained earlier in the week.

I am wholly opposed to the indiscriminate prescription of supplemental thyroid substance. Even though admonitions relative to the necessity for intelligent use of supplemental thyroid substance is presented prior to, during, and following the presentation of the subject matter to general practitioners or general County Medical Society meetings, it was distressing to find that for the next several weeks thyroid tablets were dealt out as though they were bon-bons. Actually, they are not without danger.

Dr. Hilger did not mention the serum cholesterol titre as a metabolic rate indicator.

DR. JAMES M. ROBB: Dr. Hilger and Gentlemen, those who have been on the road in medicine for some time are more likely to appreciate the content of this paper. It is understandable at times when you recommend that a patient have thyroid medication you find that the internist has not followed this suggestion. The facts are he does not take particular interest in the membrane of the nose. He cannot easily see the mucosa of the cavity of the larynx. He cannot interpret audiometric tracings. Consequently, in his mind, he may think it is of no importance. Therefore, in these cases, it is mandatory that the otolaryngologist carry on thyroid treatment.

I recommend this paper to repeated readings.

DR. JEROME A. HILGER: These are warm friends of mine and I appreciate their warm remarks.

Dr. Davison's points are well made. There is much more that can be said on the subject, but time is not sufficient.

In dealing with hypothyroids one learns that they are not, as a rule, putty-faced individuals of the four-plus myxedema level. Rather, they are often asthenic individuals, weary and worn.

The protein bound iodine determination is useful in diagnosis and in guiding one's therapy. The optimum dose for replacement therapy is occasionally difficult to establish by symptoms alone. Protein bound iodine measurement has been used as a guide.

The fallacies of the basal metabolic determination are well labored by Dr. Morrison. In the main, the test is performed indifferently. There is no question that having the patient come off the street for a BMR is a waste of time. It is window dressing. There are less expensive ways to dress patient's windows. If one's local laboratory does not do the PBI determinations, plasma can be sent by mail to laboratories elsewhere in the country at very moderate cost.

The question of serum cholesterol raised by Dr. Morrison was deliberately omitted. It is additive information only and not diagnostically conclusive one way or the other.

Dr. Robb, thank you for suggesting that the paper must be read. Otolaryngologists must be well grounded in general medicine. The endocrine system is exceedingly important in our field. I have tried in the printed copy to include the data that would be useful to you relative to this subject.

The frequency of this disorder in otolaryngologic practice certainly vies with its occurrence in internal medical practice. About half of the patients in whom we are led to suspect the diagnosis because of otolaryngologic symptoms have had some recognition of their hypometabolic state elsewhere, often by the family doctor or internist. About half have the diagnosis made primarily by us. Our radio-isotope laboratory director is an internist and he tells us that from our office he receives more truly hypometabolic patients than he does from the medical men who refer to the laboratory. So one can see that these exceedingly symptomatic tissues with which we deal every day provide us with an excellent opportunity to make the primary diagnosis.



Dr. Morrison made a good point relative to terminology. I chose deliberately to title the discussion hypometabolism. The otolaryngologic disorders discussed appear not to be confined to the primarily thyroid gland-deficient hypothyroid. The hypothalamic hypometabolic and the hypopituitary hypometabolic suffer them too. It is worth while to make these distinctions because in time they may be important therapeutically. As a matter of fact hypometabolism due to fault at the cellular level may eventually come to be recognized. In short, all hypothyroids are hypometabolics, but not all hypometabolics are primary hypothyroids.

## FUNCTIONAL DISEASES OF THE NOSE

C. STEWART NASH, M.D.

ROCHESTER, N. Y.

One of the frequent omissions in our medical teaching today is a proper recognition and understanding of the functional diseases of the nose. When more physicians realize that perhaps 75 per cent of all nasal complaints coming to a doctor's attention are functional rather than organic in nature, then there will be fewer ineffectual nasal and paranasal operations, there will be fewer disappointments from the indiscriminate use of vaccines, vitamins and antibiotics and there will be fewer useless trips to Arizona. I make this statement objectively and impersonally for I continue to perform my share of nasal and paranasal surgical procedures, but I reserve such measures for organic nasal pathology.

When I began a study of this subject in 1931, there were too many ear, nose and throat patients suffering from operative atrophic rhinitis. There were too many people who needed a change in their habits of living, or a study of their endocrine system, or an investigation of their body chemistry, or some other medical exploremment rather than the removal of organically normal but physiologically malfunctioning nasal mucous membranes.

In order to understand the functional diseases of the nose, one must comprehend these affections in terms of their inclusions and exclusions.

To be specific I offer the following definitions and explanations as a guide to our study.

### DEFINITIONS AND EXPLANATIONS

a) A *functional disease* of the nose is a condition of abnormal physiological response in which the mucous membrane of an organically normal nose enters into a state of hypo- or hyperactivity.

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Consultant to the Faculty, Department of Otolaryngology, University of Rochester, School of Medicine and Dentistry.

I concede the difficulty of shaping a definition to conform to such a flexible term as "organically normal," but in this regard I appeal to your liberal interpretation.

In the functional diseases of the nose some agent external to the olfactory organ (but not necessarily external to the body) causes the nasal mucosa to underwork or to overwork, and such a response is always temporary and reversible in character. When the causative agent in an uncomplicated functional disease of the nose is removed, the symptoms it has caused disappear and the patient is well.

#### EXAMPLES OF FUNCTIONAL DISEASES OF THE NOSE:

1. *Hypofunction.* Atropine produces a temporary and reversible hypoactivity of the nasal mucosa.

2. *Hyperfunction.* Mildly irritating inhalants produce a temporary and reversible hyperactivity of the nasal mucosa.

b) An *organic disease* of the nose is a condition of structural change in which the nose presents evidence of destruction or proliferation of tissue or in which it manifests anatomic deformity resulting from developmental or traumatic causes.

May I repeat that everything I say concerning the functional diseases of the nose applies only to those olfactory and respiratory segments that are organically normal. The moment a hypo- or hyper-functioning nasal mucosa develops destructive or proliferating changes that nose is automatically excluded from the classification of a functional disease of the nose.

The therapeutic approach to a functional disease is one thing; the therapeutic approach to an organic disease is another thing.

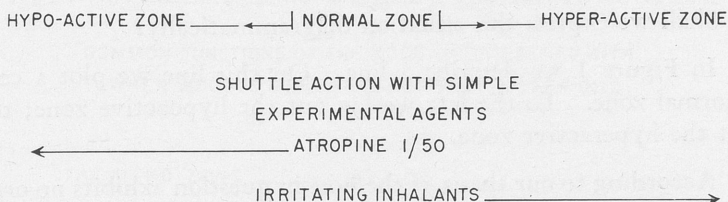
Organic disease produces a change in structure; functional disease produces a change in physiological response.

#### EXAMPLES OF ORGANIC LESIONS OF THE NOSE:

1. *Destructive Lesions.* Lues and tuberculosis are destructive agents that can produce nasal erosions.

2. *Proliferating Lesions.* Chronic sinusitis and malignancies are proliferating agents that can produce polypi or tumors.

FUNCTIONAL DISEASES OF THE NOSE  
ZONES OF MUCOUS MEMBRANE ACTIVITY



SO LONG AS NASAL MEMBRANE ACTIVITY REMAINS WITHIN THE LIMITS OF THE NORMAL ZONE, ALL FUNCTIONS OF THE NOSE WILL BE MAINTAINED. WHEN NASAL MEMBRANE ACTIVITY MOVES INTO EITHER THE HYPO-FUNCTIONING OR HYPER-ACTIVE ZONES, HOWEVER, SOME OF THE FUNCTIONS OF THE NOSE WILL BE INTERFERED WITH.

Fig. 1

In organic nasal diseases the so-called therapeutic response is said to be irreversible not because a luetic or tuberculous lesion cannot heal, but because it rarely heals without some fibrotic change.

c) A mucous membrane is an epithelial tissue, one purpose of which is to produce mucus.

So far as the nasal mucosa is concerned, however, it must produce just so much mucus. If it produces any less or any more than a certain amount, there develops a state of hypo- or hyperactivity in which some of the functions of the nose will be interfered with—and such interferences will produce symptoms for which the doctor will be consulted.

Perhaps we should be more technical and make plain that the amount of mucus produced is simply an index of the mucosa's physiological activity which may be normal, less than normal, or greater than normal.

May I continue to stress the importance of the nose producing just the proper amount of mucus if its functions are to be maintained.

The whole question of nasal mucosa activity presupposes 1) that the mucous membrane under consideration is a normal functioning



epithelium which misbehaves only because some agent external to the nose makes it misbehave; 2) that when such an external agent becomes effective, the amount of mucus secreted decreases or increases in either a hypofunctioning or hyperfunctioning manner.

Shall we express this situation diagrammatically?

In Figure 1 we describe a line. On this line we plot a central or normal zone. To the left we lay out the hypoactive zone; to the right the hyperactive zone.

According to our thesis, if the nose in question exhibits no organic pathology and for no other reasons excludes itself from the classification of functional diseases of the nose, we assume so long as the amount of nasal mucus produced keeps within the confines of the normal zone, that the functions of the nose will be maintained. If, however, the amount of mucus decreases enough to cause this shuttle index of mucosal activity to move to the left and into the hypoactive zone, or if it increases enough to cause this shuttle index to move to the right and into the hyperactive zone, then some of the functions of the nose will be interfered with.

Prosaic as this experiment may be, we present it in all of its self-evident nature. The purpose of the experiment is to cause an organically normal nasal mucosa a) to hypofunction and b) to hyperfunction.

*Hypofunction.* 10 adults given 1/50 of a grain of atropine sulphate hypodermatically developed dry noses within a period of 30 minutes.

By this test the shuttle index moved to the left and into the hypofunctioning zone.

*Hyperfunction.* Ten adults exposed to onion emanations developed a runny stuffy nose within five minutes.

The reversibility of these reactions was noted in the return of the shuttle index to the normal zone after the following intervals: a) the hypofunctioning noses returned to normal in 12 to 14 hours; b) the hyperfunctioning noses returned to normal in 30 to 60 minutes.

#### COMMON FUNCTIONS OF THE NOSE

Figure 2 is Figure 1 to which has been added some of the common functions of the nose.

## FUNCTIONAL DISEASES OF THE NOSE

HYPO-ACTIVE ZONE ← | NORMAL ZONE | → HYPER-ACTIVE ZONE

COMMON FUNCTIONS OF THE NOSE INTERFERED WITH  
WHEN MUCOUS MEMBRANE ACTIVITY MOVES INTO

THE HYPO ZONE ← A  
OR  
B → THE HYPER ZONE

- 1- RESPIRATION
- 2- FILTRATION
- 3- HUMIDIFICATION AND HEATING
- 4- VENTILATION AND DRAINAGE
- 5- CILIARY ACTION
- 6- OLFACTION
- 7- PHONATION
- 8- BACTERIOPHAGE

Fig. 2

We have spoken of the shuttle index of mucosal activity and have hinted that whenever this shuttle passes out of the normal zone, some of the functions of the nose are interfered with.

What then are the functions of the nose that can be interfered with when a normal nasal mucosa enters into a state of hypo- or hyperfunctional activity?

Briefly, the common ones are as follows:

1. *Respiration.* The nose is the peripheral organ of the respiratory system. We grant that a person can live without breathing through his nose but he cannot do so without certain unsalutary results.

2. *Filtration.* The vibrissae (or hairs within the nostrils) act as an inefficient sieve to separate gross particles of foreign material from the inhaled air. Ciliary action, however, has to take care of the foreign substances that pass through the vibrissae.

3. *Humidification and Heating.* A proper amount of mucus in the nose is the means by which inhaled air takes on heat and moisture that is carried throughout the respiratory tract.

4. *Ventilation and Drainage.* The physical and physiological importance of nasal ventilation and drainage is an incompletely explained phenomenon.

One thing, however, is evident, namely that interference with these functions produces some of the more serious nasal complaints.

5. *Ciliary Action.* The cilia act as brooms by which the nose sweeps mucus and foreign material out of the nasal cavity. This process is a procedure which is supplemented by the filtration action of the vibrissae and the physical drainage of the nose.

6. *Olfaction.* The sense of smell depends for its sensory reception, in part at least, on an unobstructed path to its end organ in the superior meatus and to a normal condition of the mucosa which contains it.

7. *Phonation.* The nasal tone accompanying a cold or hay fever is adequate evidence of the importance of the nose as a resonance box for speech.

8. *Bacteriophage.* So far as I know the bacteriophage properties of the nose are inadequately comprehended. Nasal mucus may or may not have antibiotic properties.

#### THE HYPOTHESIS CONCERNING FUNCTIONAL DISEASES OF THE NOSE:

1. All things being equal so long as a nose that is without "organic pathology" continues to secrete a normal amount of mucus, all the functions of the nose will be maintained.

2. If, however, an exciting agent (external to the nose but not necessarily external to the body) causes a hypo- or hyperactivity of the nasal mucosa, then some of the functions of the nose will be interfered with.

3. This interference of nasal function will result in signs and symptoms characteristic of either a hypo- or hyperfunctioning rhinitis.

4. Hypo- or hyperfunctional diseases of the nose are mucous membrane responses that are temporary in nature and reversible in

reaction. Elimination of the cause automatically relieves the abnormal response.

5. If such physiological diseases develop organic pathology they can no longer be classified in our concept of the functional diseases of the nose.

#### INDUCTIVE APPROACH TO THE STUDY

In observing various types of hypo- and hyperfunctioning diseases of the nose, we shall not analyze the agents causing alterations in mucosal activity, neither shall we investigate the mechanisms by which physical, physiological or psychological stimuli produce an alteration of function, but we shall approach this study in an inductive manner and endeavor to correlate a particular syndrome with its nasal response.

#### EXPLANATORY NOTES

1. One should recognize that both the hypofunctional and hyperfunctional diseases may manifest themselves in one of two phases: either a) the transitory form which is reversible in a period of hours; or b) the prolonged form which is reversible in a period of months.

2. Failure to demonstrate reversibility within a reasonable length of time suggests the development of organic changes the result of overstimulation or overexposure.

3. The terms "acute" and "chronic" are avoided because they may connote tissue changes which would exclude them from the functional disease classification.

#### HYPOFUNCTIONING DISEASES OF THE NOSE

Figure 3 is Figure 2 to which has been added an incomplete list of nasal syndromes exemplifying hypofunctioning rhinitis.

Sample types of hypofunctioning rhinitis, as identified by their causative agents, are:

- 1—Atropine
- 2—Physical
- 3—Psychic
- 4—Febrile
- 5—Debilitating (or constitutional)

(There are many others)



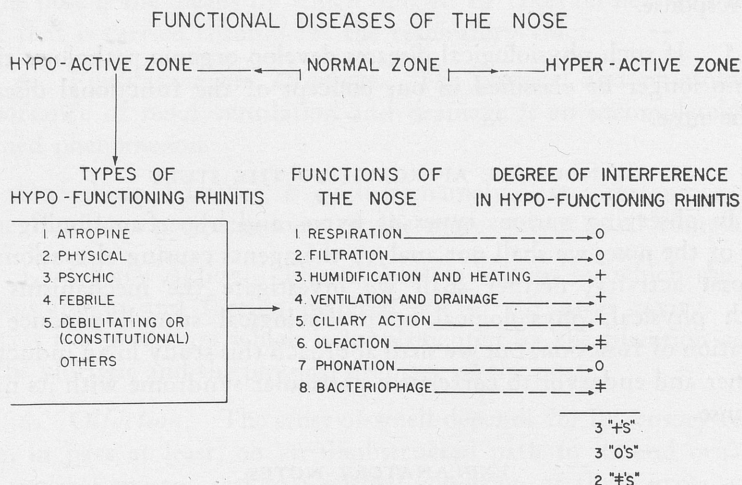


Fig. 3

Collectively, and as shown in Figure 3, the group of hypofunctional diseases has been matched against the functions of the nose and the degree of interference noted. In Figure 3, atropine is the exciting agent against which the selection and degree of interference with nasal function is measured.

Individually we shall discuss each type of hypofunctioning disease in terms of its clinical manifestations and interpretations.

#### DEFINITION

A hypofunctional disease of the nose is a condition in which an exciting agent external to the nose (but not necessarily external to the body) causes an otherwise normally performing mucous membrane to *decrease* its production and thereby to produce a temporary and reversible interference with some of the nasal functions.

*Symptoms of Hypofunctioning Diseases of the Nose:* 1) A feeling of dryness in the nose and throat; 2) often a thick, scanty or scabby nasal or postnasal discharge which may be diagnosed as a sinusitis (usually a sphenoidal infection); 3) occasionally there is a sense of inadequate breathing space, a symptom commonly observed in an organic atrophic rhinitis.

## DIFFERENTIAL DIAGNOSIS

Because both a hypofunctioning rhinitis which is a physiological response and an atrophic rhinitis which is an organic change, have in their early stages the same signs and symptoms, one may have to delay the ultimate differentiation between these two conditions until the reversibility of the functional affection becomes evident.

## DISCUSSION OF TYPES OF HYPOFUNCTIONING RHINITIS

1. *Atropine Rhinitis (Hypofunctioning)* (previously described).

*Transitory Form.* The property of atropine sulphate to arrest the production of bodily secretions is a well-known physiological reaction. Its use as a preoperative medication is contingent upon its ability to induce a hypofunctioning rhinitis.

*Prolonged Form.* Continued use of belladonna, phenobarbital and atropine or atropine eye drops is responsible for this type of nasal dysfunction.

2. *Physical Rhinitis (Hypofunctioning).*

*Transitory Form.* The transitory form of physical rhinitis is a rhinitis sicca brought about by sporadic exposure of the nasal passages to an atmosphere sufficiently dry and warm to cause evaporation of nasal moisture, but not sufficiently so to produce an edematous reaction.

This condition can occur in any of us, but furnace men, dry cleaners, evaporator workers and others similarly employed, whose periods of exposure to such an atmosphere is mild in degree and short in duration, exemplify this form of nasal difficulty.

*Prolonged Form.* A prolonged physical hypofunctioning rhinitis is observed in patients living or working in warm rooms that are inadequately humidified and in persons whose nasal mucosa is laid open to mildly desiccating fumes.

This condition too can occur in any of us. Occupational examples of this type of nasal dysfunction are seen in kiln operators, foundry workers, and bake oven mechanics. Seldom does this condition result in structural damage.

### 3. *Psychic Rhinitis (Hypofunctioning).*

*Transitory Form.* Everyone is subject to a transitory psychic hypofunctioning rhinitis. How often a dry mouth (and nose) precedes a speech or an appearance before the microphone. This exasperating circumstance is a psychic hypofunctioning response. The pitcher of water is on the banquet table not because the speaker is thirsty. He is not thirsty; he is dry!

All too frequently little anxieties (many of which are almost unrecognized) express themselves in a dryness of the mouth and nose.

*Prolonged Form.* As a fleeting anxiety produces a transitory psychic hypofunctioning rhinitis, so enduring anxiety produces a prolonged response.

Our mental hospitals are filled with patients who long before their commitments had an annoying psychic hypofunctioning rhinitis, the significance of which was unrecognized and the treatment of which was empirical or sinus-wise.

### 4. *Febrile Rhinitis (Hypofunctioning).*

*Transitory Form.* Repeatedly I have been called upon to treat a posterior "sinus infection" in a pneumonia patient—one who has maintained a temperature of 105° for several days—only to discover that following the crisis the "sinusitis" disappeared with remarkable rapidity.

Frankly, a febrile rhinitis does present the signs and symptoms of a subacute sphenoidal sinusitis, but experience teaches one to wait for the diagnosis to establish itself.

*Prolonged Form.* So long as a fever lasts, the febrile hypofunctioning rhinitis may continue. In the days of typhoid fever, malaria, smallpox, and other forms of extended febrile states, the prolonged form of febrile rhinitis was common—today it is not so frequent.

### 5. *Debilitating or Constitutional (Hypofunctioning).*

There is no *transitory form*.

*Prolonged Form.* Although the sedentary life of a chronic invalid predisposes him to a dry and scabby nose, even a short period of

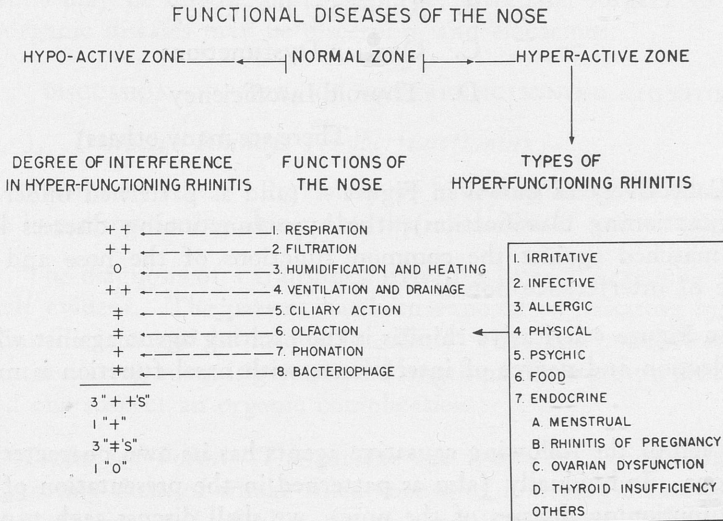


Fig. 4

general improvement can bring about a betterment of the nasal condition.

#### HYPERFUNCTIONING DISEASES OF THE NOSE

Figure 4 is figure 3 in which some of the types of hyperfunctioning rhinitis are substituted in place of some of the types of hypofunctioning rhinitis.

Sample types of hyperfunctioning rhinitis as identified by their causative agents are:

- 1—Irritative
- 2—Infective (Common Cold)
- 3—Allergic
- 4—Physical
- 5—Psychic
- 6—Food
- 7—Endocrine



- A. Menstrual
- B. Rhinitis of Pregnancy
- C. Ovarian Dysfunctions
- D. Thyroid Insufficiency

(There are many others)

Collectively as shown in Figure 4 (and as patterned under the hypofunctioning classification), the hyperfunctioning diseases have been matched against the common functions of the nose and the degree of interference noted.

In Figure 4 irritative rhinitis is the exciting agent against which the selection and degree of interference with nasal function is measured.

Each of the following causative agents has its own characteristic reactions. Individually (also as patterned in the presentation of the hypofunctioning diseases of the nose), we shall discuss each type in terms of its clinical manifestations and interpretations.

A hyperfunctional disease of the nose is a condition in which some exciting agent external to the nose (but not necessarily external to the body) causes an otherwise normally performing mucous membrane to *increase* its usual activity and thereby produce a temporary and reversible interference with some of the functions of the nose.

Symptoms of hyperfunctioning diseases of the nose are: 1) nasal obstruction, 2) rhinorrhea, 3) intranasal pressure, 4) headaches, 5) postnasal discharge (all the signs and symptoms of a common cold or sinus infection).

By way of warning may I counsel the reader against any oversight or indifference in regard to the hyperfunctioning diseases of the nose. The hyperactive nose is one that exhibits signs and symptoms that are so familiar to us, those of the common cold, those of its complicating sinusitis, or those of an allergic rhinitis.

Too many practitioners continue to regard a "runny nose" as a cold, as a sinusitis, or as an allergy, and well they may be the causes too, but failures in the treatment of these ailments have an explanation which is often an unrecognized or unsuspected hyperfunctioning rhinitis, the causative agent of which is evident only after investigation and study.

Intranasal surgery to relieve an uncomplicated hyperfunctioning rhinitis may be unwise and ineffective; intranasal surgery to relieve an organic diseases may be discerning and efficacious.

#### DISCUSSION OF TYPES OF HYPERFUNCTIONING RHINITIS

##### 1. *Irritative Rhinitis (Hyperfunctioning).*

*Transitory Form.* Previously we have mentioned the production of a hyperfunctioning rhinitis by the inhalation of onion emanations.

The diagnosis of a transitory irritative hyperfunctioning rhinitis is self evident. The patient has been exposed to irritating inhalants or instillations and he knows it. Only when the concentration of the stimulating agent is great or the length of the exposure protracted, need one suspect an organic complication.

*Prolonged Form.* The present age is one of noxious fumes, of penetrating dust, of smoke filled rooms, of nonallergic face powders and cosmetics, and of all sorts of nasal instillations. Frankly the prolonged form of irritative rhinitis is common but often unsuspected or unrecognized.

All of us are victims of an irritating rhinitis to some degree. Garage workers, furniture makers, musicians, welders and building painters are but a few of the types of laborers subjected to this sort of malady.

From my experience I am convinced that one of the most common causes of a prolonged irritative rhinitis is the persistent use of nose drops. Any nose drops used long enough will produce the very symptoms they are taken to relieve.

##### 2. *Infective Rhinitis (Hyperfunctioning).* (The common cold). The common cold is a hyperfunctioning rhinitis, the exciting cause of which is a respiratory infective agent or virus. It may or may not be initiated by exposure to cold or dampness.

An uncomplicated cold lasts for 21 days. If someone tells you otherwise, do not believe it.

The course of an infective rhinitis is divided into three stages which follow a rather exact time table. Diagrammatically, we represent this occurrence in Figure 5.

### COMMON COLD (UNCOMPLICATED) STAGES AND TIME—TABLE

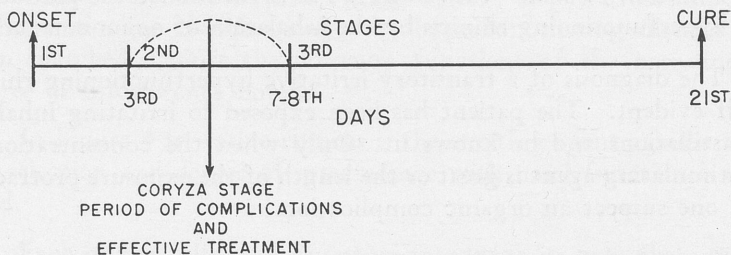


Fig. 5

The first stage of the common cold continues for 48 hours and may or may not be noticed by the patient. Its symptoms, if present, are a slight throat irritation or cough.

An uncomplicated second stage begins on the third day and remains through the seventh or eighth day. Its symptoms are those of a coryza or cold. It is in this stage that the shuttle index of mucosal activity extends far into the hyperfunctioning zone; it is in this stage that so many of the functions of the nose are interfered with; it is in this stage that the symptoms are most severe; it is in this stage that complications occur (most of which are due to an impedance of the function of ventilation and drainage). At some time around the seventh or eighth day following the onset of an uncomplicated cold, the nose opens spontaneously and thereby restores its function of ventilation and drainage. With the attainment of this goal, all functions of the nose return and the cold passes into the final period.

In the third stage of an infective rhinitis the nose is open and draining a mucopurulent material which gradually becomes normal in amount and character. During this time the patient uses three handkerchiefs a day; two handkerchiefs a day, and then one handkerchief a day. Because the cessation of the second stage brings with it a return of nasal functions, the patient insists that at the end of the

second stage the cold is over. The fact that a mucopurulent discharge continues up to the twenty-first day does not interest him for the shuttle index of mucosal activity has returned to the normal zone and so far as he is concerned the nose is well.

From this observation of the stages and the time table of an infective rhinitis, nature appears to teach us that the way to cure a common cold is to establish ventilation and drainage. In my opinion, only those measures that bring about ventilation and drainage have any value in the treatment of an infective hyperfunctioning rhinitis—and this statement applies both to local and systemic therapeutic measures.

The management of the common cold, in accordance with its stages and time table, is a must for every practitioner of medicine, for only by understanding a cold's habits and vagaries can one outwit this commonest of man's afflictions.

The subject of outwitting the common cold, however, is a treatise in itself.

3. *Allergic Rhinitis (Hyperfunctioning)*. I do not intend to dissertate on an allergic rhinitis but I do wish to emphasize two points, namely: a) that an uncomplicated allergic rhinitis is a hyperfunctioning disease of the nose. Only when the allergy becomes complicated by polypi or hyperplasias does it take on an organic character, and b) an allergic nasal mucosa is pale; all other hyperfunctioning mucosae are red. The swelling of an allergic mucous membrane is caused by distention of intercapillary spaces which contain few red blood cells, consequently the pale color. The swelling of all other hyperfunctioning mucous membranes is caused by a distention of the capillaries themselves, which contain an abundance of red blood cells, consequently the red color.

This point of differentiation is a study that we still have under investigation.

#### 4. *Physical Rhinitis (Hyperfunctioning)*.

*Transitory Form.* This type of nasal dysfunction occurs in the patient who says that he is all right so long as he remains indoors but when he goes out-of-doors, he develops a cold. It also appears in the patient who says that he is all right so long as he remains out-of-doors,



but when he goes into a heated room he develops a stuffy nose. I do not know the answer to this problem.

*Prolonged Form.* Air conditioning, jobs in and out of cold storage vaults, continuous employment in cold or freezing temperatures are only a few of the purported causes of a physical hyperfunctioning rhinitis.

5. *Psychic Rhinitis (Hyperfunctioning).* (See psychic hypo-functioning rhinitis.)

*Transitory Form.* Previously we have noted how anxieties can produce a dry nose and throat. In the same manner, but not always from the same causes, they can produce a rhinorrhea. Examples: 1) For years I had under observation a minister who had a "runny, stuffy nose" that lasted two hours preceding each Sunday sermon. With the onset of his discourse the cold disappeared for another week. 2) I have had several patients who in fits of anger developed a profuse rhinorrhea that continued as long as the emotional disturbance lasted.

*Prolonged Form.* Example: A southern girl married a northern boy and the couple settled in New York State. For more than a year following their marriage the wife had a continuous cold which responded to no treatment. For this difficulty she consulted many doctors. I saw her twice and referred her to a neuropsychiatrist who discovered that the cause of her hyperfunctioning rhinitis was not any condition associated with her marriage, nor was it the result of the inclement weather of the North, but was a drunken father-in-law. In a home of her own she became well.

6. *Food (Hyperfunctioning).* Long have I contended that certain types of food rhinitis are quite independent of and unrelated to an allergy. Such food responses present no lacrymations, not itching of eyes or palate, and no pallor of the mucous membrane or other signs and symptoms characteristic of an allergic rhinitis.

To date I have not been able to isolate nor to identify defensible cases of transitory nonallergic food rhinitis.

*Prolonged Form.* So far as the prolonged type of nonallergic food rhinitis is concerned, however, I consider it a physiological entity. Example: A girl nine years old suffered from a continuous "cold" and sinusitis for six years. During this time she underwent

the following precodures: 1) two tonsillectomies and adenoidectomies, 2) nasal suction treatments twice a week for six years, 3) a subsequent adenoidectomy, 4) an antrum window operation, 5) several other unidentified operative procedures. When a third adenoidectomy was advised, she came to my attention. Objectively this girl presented the signs and symptoms of a chronic pansinusitis. A study of her history revealed that for years she had eaten candy to excess and to the exclusion of other important nutrients. Elimination of the candy from her diet brought about a complete cessation of her symptoms—all of which disappeared within two weeks of the time I first examined her. I saw this child just twice. Twenty years have now passed since she had her food rhinitis and she continues to remain well except when she goes on a candy binge.

Briefly, potatoes, starches, citrus fruits, or an inadequate intake of proteins and vegetables are the common causes of a food rhinitis.

7. *Endocrine Rhinitis (Hyperfunctioning)*: a) menstrual, b) rhinitis of pregnancy, c) ovarian dysfunction, d) thyroid insufficiency.

Endocrine rhinitis does not manifest itself in a transitory form.

a) *Menstrual Rhinitis*. The signs and symptoms of a menstrual rhinitis resemble those of a common cold. It comes on three to four days prior to the menstrual period and disappears soon after its onset. It is not of common occurrence. It has been observed by me only during the first year of menstruation.

It is not prevented or controlled by vaccines, antibiotics, vitamins, or other therapeutic measures usually employed in the treatment of the common cold, but it can be, and often is, corrected by estrogenous substances.

Unfortunately, an endocrine extract that cures one patient may be ineffective with another.

b) *Rhinitis of Pregnancy*. The rhinitis of pregnancy is a hyperfunctioning disease of the nose that is common. It may appear at any time during gestation and it rarely, if ever, disappears until the termination of the pregnancy or shortly thereafter. It does not give the appearance of an allergy. The signs and symptoms are those of a severe infective rhinitis but the response to measures ordinarily used in the treatment of the common cold are of little or no benefit.

Thyroid extract, antihistaminics, antibiotics and even the estrogenous substances that I have used have not relieved this condition. Local vasoconstriction, of course, produces temporary ventilation and drainage.

The subject of rhinitis of pregnancy warrants thorough scientific investigation.

c) *Ovarian Dysfunction.* I have observed many patients in whom there have been associated the signs and symptoms of an ovarian disease and those of a hyperfunctioning rhinitis. In many instances I have seen the correction of the ovarian difficulty bring about a complete relief of the nasal dysfunction. Example: A woman, aged 38, had complained of a stuffy nose since the onset of menstruation at the age of 13. At age 37, she developed a demonstrable ovarian disease which required a bilateral oophorectomy. Following this operation all nasal symptoms disappeared. When, however, her internist prescribed ovarian extract for the essential requirements of her body the rhinitis returned.

d) *Thyroid Rhinitis.* Any patient the cause of whose hyperfunctioning rhinitis has not been determined by proper differential diagnosis should be given provocative doses of thyroid extract. Some hyperactive noses clear completely with thyroid therapy, and this statement holds regardless of the presence of a normal basal metabolic rate and regardless of the concurrence of a complicating sinusitis.

I do not know the explanation of this situation.

We have given no consideration to many other types of hyperfunctioning rhinitis, in particular I mention the dysfunctions due to alcoholism and sexual excitation.

#### COMMENTS AND CONCLUSIONS

1) Years of experience and observation have convinced me that too many doctors are treating noses according to the old adage that every "runny nose" is the result of a cold, an allergy or a sinusitis.

The acceptance of this doctrine leaves many types of hyperfunctioning rhinitis inadequately treated and unimproved.

2) A sinusitis complicating a hypo- or hyperfunctioning rhinitis often receives the attention that the underlying functional disease of the nose should get.

3) Operative procedures for the relief of symptoms of hypo- or hyperfunctioning diseases of the nose in the absence of organic pathology are usually of little value.

4) Operations for the relief of organic diseases of the nose may be of great value.

5) This study of the functional diseases of the nose is not a crusade to reduce the amount of nasal and paranasal surgery; it is merely a remedial enterprise the purposes of which are: a) to recognize the presence, the frequency and the importance of the functional diseases of the nose; b) to discover an underlying functional disease even though it simulates a more common nasal affection with or without a complicating sinusitis; c) to institute the treatment such a dysfunction properly requires.



## UNUSUAL VIRUS DISEASES IN OTOLARYNGOLOGY

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The subject of virus diseases is one with which we in our specialty must be thoroughly familiar. The full gamut of transition from the age of focal infection to antibiotic control of diseases has occurred during our short association with otolaryngology. We are now faced daily, in our clinics and offices, with the patient showing unusual and persistent symptoms which do not respond to the older orthodox procedures or to the most recent broad spectrum antibiotics, even with their exaggerated economic costs to the patient. We must consider whether the virus diseases are about to take over our practices, either because we have not satisfactory defense against them, or that they have become increasingly strong with our apparent conquering attack on the bacterial world. With this in mind I would like to enlist interest in the consideration of a few of the more unusual virus infections that might be of special concern to the otolaryngologist.

The word virus means poison, but it was first applied to any toxic substance that could pass through an earthenware filter which would trap true microbic organisms. Size, therefore, is a primary characteristic, and at first this material was invisible, but with the development of the electron microscope, the shape and size of various strains could be recognized. The next most important characteristic of viruses is that they manifest themselves and multiply only in susceptible living cells. The method of invasion and combination with individual cells is still largely unknown. The importance of this is that living tissue is necessary in any virus study, and one must use either living animals or a live tissue culture for their study. The development of animal inoculation, chick embryo, or tissue cultures either in embryonal

human tissue or the "HeLa" cells of a known cancer stain, have all been used in the study of these diseases.

#### CLASSIFICATION OF VIRUS DISEASES

There is no satisfactory or accepted classification of the virus diseases. Due to so many subgroups in the various broad clinical pictures one only gets more confused by their study. A rough clinical classification seems more to the point and one is presented below.

#### CLINICAL CLASSIFICATION OF VIRUS DISEASES

|                              |  |
|------------------------------|--|
| THE POX AND RASHES           | Small pox earliest recognized and controlled   |
| LIVER VIRUS DISEASES         | a) Infections } Hepatitis, Yellow Fever<br>b) Serum }  |
| RESPIRATORY TRACT            | a) Influenza } Can be proven by Lab. test<br>b) A.R.D. }<br>c) Common Cold—Hodge-podge of many viruses, cannot be grown—know little about it |
| ENCEPHALITIS AND MENINGITIS  | a) Disease of animals—men secondary<br>b) Mosquitoes transfer to men<br>c) Have occurred in epidemics  |
| POLIOMYELITIS & ITS COUSINS  | a) Polio—3 main types—1, 2, 3<br>b) Coxsackie—Group A—Herpes, muscle pain<br>Group B—Bornholm Disease<br>c) E. M. C.—Encephalomyocarditis    |
| INFECTIONS OF SKIN AND BRAIN | a) Herpes Simplex<br>b) Herpes Zoster<br>c) Chicken Pox<br>d) German Measles<br>e) Measles   |

*Rabies* existed 4000 years ago. Pasteur's crowning work.

It would take several tables to show the subclassification of acute respiratory diseases above. These are known in England as the acute febrile catarrh group. It is unfortunate that the large group of the common cold still remains as elusive as before.

## THE ADENOIDAL PHARYNGEAL CONJUNCTIVAL VIRUSES

In the respiratory tract group has been found a relatively new division known as the adenoidal-pharyngeal-conjunctival or A.P.C. viruses. This group has been described by Bell<sup>1</sup> and his co-workers as "viruses of the respiratory tract, nonpathogenic for laboratory animals, ether resistant, heat labile, filtrable, and resistant to antibiotics." There is a group reaction by complement fixation which is not specific as to individual types. There are, however, eight distinct immunological types. Five of these types (1, 2, 4, 5, 6) have been cultured from adenoid and tonsil tissue. Type 3 has been repeatedly cultured from the throats and conjunctiva of the definite disease entity called acute pharyngeal-conjunctival fever.

Acute pharyngeal-conjunctival fever is a mildly epidemic type of disease that has been reported from Washington,<sup>1</sup> San Francisco<sup>2</sup> and Toronto.<sup>3</sup>

In Toronto twenty adults were referred to the laboratory of Dr. Hugh Ormsby, of the Department of Ophthalmology, in the six months following November 1951 with conjunctivitis and other symptoms of the disease. Of these, seven produced a virus which grew in tissue cultures of monkey-kidney epithelium or "Hela" cells. Type 3 A.P.C. group was proven to be present. It is of interest that in adults the clinical symptoms seemed first to be a follicular conjunctivitis in one eye. Followed in three to four days by involvement of the other. Pharyngitis occurred only in two of the patients and catarrhal otitis in one. None had fever, malaise or muscle pains.

The disease in children was more thoroughly studied during a local epidemic, beginning in early August 1955, by Dr. Ormsby and his group. The clinical course was much more like the reported findings in other centers and closely followed the Washington report. Many had a temperature elevation to 102° to 105° F., acute pharyngitis was present, and catarrhal otitis media was a common complication. As the disease was apparently associated with swimming pools, a survey was made within a half mile of a North Toronto pool. It was found that half of the cases had occurred in that area. Seventy-four gave a history of swimming in that pool within ten days of the onset of the disease, six had been in pools elsewhere.

The one significant point is that while most of the work on this disease entity has been largely the interest of the ophthalmol-

TABLE I.

Cases of A. P. C. Fever Occurring Within a Half Mile Radius  
of a North Toronto Indoor Swimming Pool (August, 1955)

(Ormsby and Aitchison)

|   |     |
|---|-----|
| Cases originating in pool (all children)      | 74  |
| Cases originating in pools elsewhere          | 6   |
| Cases originating from direct contact at home | 31  |
| No history of swimming or contact at home     | 1   |
| TOTAL CASES                                   | 112 |

TABLE II.

Symptoms of Pharyngeal-Conjunctival Fever in 112 Patients  
(August, 1955)

(Ormsby and Aitchison)

|                                 | CHILDREN (104) | ADULTS (8) |
|---------------------------------|----------------|------------|
| Sore Eyes                       | 68             | 7          |
| Fever, Malaise and Muscle Pains | 75             | 2          |
| Sore Throat                     | 63             | 1          |
| Sore Ears                       | 42             | 0          |

ogists, the symptoms in Table II show that 67 per cent had sore eyes, 57 per cent had sore throats and 38 per cent had ear symptoms. One would wonder if many of our cases of catarrhal otitis media, aside from this definite group, are not viral in origin. We have hopes that we may have sufficient cases in the near future to present such a group.

#### CAT SCRATCH FEVER

One of the authors (P.E.I.) has had three private cases of cat-scratch disease, so became interested in the work being followed in the Medical Clinic of the Toronto General Hospital by Drs. G. Hart and W. Spaulding. In one year they have seen 40 cases, and it is with



their permission that we quote from their findings which are not yet published.

This disease, which is a benign condition, viral in origin, is important because it may mimic more serious causes of enlargement of lymph nodes. The features have been quite well described by Daniels<sup>4</sup> and Rosenblum.<sup>5</sup> The history is of a minor scratch or contact with a cat. This is followed by a local papule or vesicle at the site of the scratch. This incident may be forgotten, but several weeks later the regional lymph nodes enlarge with malaise and low-grade fever lasting up to several weeks. The lymph nodes become tender and frequently break down and show, on pathological examination, a chronic granuloma impossible to differentiate from tuberculosis, with a caseous type of necrosis and giant cells. The disease usually runs a benign course, but lymph nodes may remain enlarged for two years.

All but three of the 40 cases had close contact with cats. Twenty could recall a scratch from one to three weeks before the lymph nodes enlarged. Only three of the cats had shown any signs of illness, and one child had no contact with a cat, but had scratched his finger on a thorn bush.

*Adenopathy.* In 22 cases the epitrochlear and axillary nodes were involved due to inoculation by hand scratches; ten originated from the feet or legs, eight from pats or scratches of the face or neck giving cervical or preauricular nodes. The nodes become tender and adhere to the skin and adjacent tissue.

In 18 cases there was fluctuation; aspiration of purulent material failed in all to show on smear or culture pyogenic or tuberculosis organisms.

*Differential Diagnosis.* Infectious mononucleosis was considered in two patients, although mild fever, malaise, sore throat and enlarged cervical nodes may occur in this or cat-scratch disease, in the latter there is neither the characteristic changes in the blood smear nor a positive Paul-Bunnell test. Tuberculosis was diagnosed in four patients on the biopsy reports and two of these were admitted to a sanatorium. In each of these cases the tuberculin test was negative, the chest x-ray normal, prolonged fever was absent and no tubercle bacilli was found on the smear, guinea pig inoculation or culture. The findings in five cases were originally attributed to Hodgkin's disease and two others were clinically thought to be tularemia. In

one case the diagnoses considered were syphilitic chancre of the lip, with adenopathy or the malignant pustule of the anthrax. Rheumatic fever was considered early in one case, because of erythema nodosum and a palpable spleen.

*Skin Test.* The cat-scratch skin test is done by injecting 0.1 cc of an antigen of known potency intradermally, noting the local reaction after 48 hours. A positive reaction consists of a papule of at least five mm in diameter and an erythema of at least 10 mm in diameter.

Only 38 of the 40 patients were so tested, and all gave positive responses with potent antigen. Three of these at the time of the test had tenderness, enlargement of lymph nodes or recurrence of discharge from a pre-existing sinus, suggesting a transient exacerbation of the inflammatory process. Fourteen successive healthy controls were all negative. There were also 12 patients tested with conditions such as leukemia, carcinoma, lymphoma and Herpes zoster with negative results. One patient (not included in the series) clinically had cat-scratch disease, but a negative test. He was receiving cortisone therapy.

The test seems to be a very good criterion for the diagnosis of the disease in the general population. McGovern<sup>6</sup> found 4 positive tests in 18 employees in an animal hospital and 10 per cent in healthy control subjects. This latter figure seems much too high for our area. Also one of the present series gave a positive reaction eight years after his illness. The test, therefore, must be associated with the clinical findings and resembles the tuberculin reaction. The big exception is that a very small percentage of the population will react to the cat-scratch antigen.

#### SUMMARY

Cat-scratch disease is not rare, although only recently recognized as an entity. It can present a confusing diagnostic problem if we do not recognize its clinical course. The skin test is a most useful clinical adjunct and is usually negative in healthy people or in other similar diseases. Suppuration in cervical glands in the absence of tubercle bacilli or pyogenic organisms should make us suspicious of this disease especially when the biopsy is reported as tuberculosis. The disease is usually benign and self-limiting and aspiration of fluctuant glands is all that is necessary. A few deaths have been reported from encephalitis so that we must always wonder if epidemic forms of this virus disease in a more severe form is a possible threat. All antibiotics are

ineffective for treatment. Cortisone may have an inhibitory effect, but there seems no need to use this potentially dangerous drug in this benign condition.

#### REPORT OF CASES

CASE 1. Miss X, aged 23. This German immigrant girl was working as a secretary. She developed first a papule, then a larger pustule below the margin of the mandible at the submaxillary gland area. In about eight days glands developed in the submaxillary area, and the anterior cervical chain. Anthrax was thought of but aspiration showed the fluid sterile for organisms. Skin tests were positive for cat-scratch fever. At the onset she had malaise, fever and sore throat. She had been given all the old and newer broad spectrum antibiotics with no effect. At her boarding-house a cat met her each night and jumped from the newel post to her shoulder, patting her face affectionately, but never scratching her. The owner of the house had also a history of pustules on his legs, and had tender inguinal glands. He was positive also for cat-scratch antigen in a skin test.

CASE 2. Miss G., aged 24. This girl was employed as a research chemist with no immediate contact with cats. She was seen by one of the authors last June with a firm mass about 2.5 cm in diameter in front of the right ear. This mass seemed free from the skin, but firmly anchored to the deep structures. With a history of at least a year, it was thought to be a parotid tumor, and arrangements were made to remove it in September. Her father was a doctor, but she was away from home and when she appeared for operation there was a fluctuant mass. This was removed and found to be broken-down lymph gland with parotid tissue also attached to the deep surface. Smear and cultures failed to show pyogenic organisms or tubercle bacilli. Pathologically there was a typical caseous necrosis with tubercle-like formation, and giant cells. No organisms could be found in the tissue by acid-fast stain. She was repeatedly tested and found negative by tuberculin, but positive to cat-scratch antigen. The area healed, by first intention with no recurrences or evidence of other gland involvement. She had visited on a farm where there were cats, but that was the only contact found.

#### VIRUS DISEASE IN ANIMALS AND BIRDS

We were very much enlightened to find, on a visit to our Ontario Veterinary College, the familiarity they have with this type of disease

in animals. It is recognized in the distemper of dogs, epidemic encephalitis of horses, swine rhinitis, turkey sinusitis, psittacosis and many other strains in fowl. They not only have been able to isolate many of these viral strains, but to grow them and transmit them to other animals with the reproduction of the clinical disease.

The most striking of the group, from our point of view, is the turkey sinusitis. This has been mentioned by Dr. C. E. VanRooyen. This disease produces marked distention of the sinuses. The causal agent has been cultivated in embryonal egg media, and the disease reproduced.

We have recently become interested in a local problem of human infections related to fowl disease, but so far the findings are most incomplete. In A——, a town of about 5,000 population, the local practitioner began to find an enormous number of so-called atypical pneumonias of short duration which he chose to call virus pneumonia. There occurred 304 cases in a period of four months. He kindly gave us his records and one of the authors (T.M.) has gone over these carefully. There is no true clinical picture in the symptoms listed that could be called a disease entity. There is a history of a sore throat, varying up to two weeks before the onset of severe general malaise, frontal and suboccipital headaches and patchy rales present on chest examination. On inquiry from a number of these patients they have stated that they felt quite ill for about three days. Unfortunately for the diagnosis of a virus infection, they recovered on the broad spectrum antibiotics—usually aureomycin.

To the local practitioner there occurred the thought of two possible sources of these infections. These were the enormous number of pigeons around the town and the fact that 30,000 turkeys were raised within a five mile radius. On two separate occasions batches of apparently healthy pigeons were submitted to the Provincial Laboratory to check for psitticosis. In the first group 30 per cent showed a positive reaction, the second group showed 60 per cent. This latter group shows a much higher percentage than is usually found in the pigeon population and is certainly suspicious of an outbreak of disease in this group.

From April, 1954, blood was submitted from 102 patients for agglutination tests for virus infections. The method of taking the specimens was 1) at the onset before treatment was initiated, 2) after treatment, or two to three weeks after the onset of the disease. Of



these, 68 agglutinated with influenza A.A.—one or B; 21 agglutinated with psittacosis. Unfortunately most of these were one in ten to one in twenty, which is of no significance. Only two cases showed significant figures—1 in 10 to 1 in 160, and 1 in 160 to 160. One is forced to state that the findings are of no significance in regard to psittacosis as a causative agent.

We can not help but feel that the clinical judgment of an older practitioner must be respected and not turned down by the failure of laboratory procedures. This type of semi-epidemic acute respiratory disease certainly did occur in this town, and not in adjacent towns. The physician has plotted the density of his cases on a map of the area which shows a marked density of the cases in the areas nearest the concentrations of turkeys and it is most striking in appearance. We are hoping to find a virus in the turkeys, perhaps similar to that giving turkey sinusitis, which has been transferred from the turkey droppings to the human to produce the clinical picture.

#### SUMMARY

An attempt has been made to discuss virus diseases as a clinical problem. Too often we are assailed with such technical details of groups, strains and numbers that we become just more confused. In order to avoid this a good deal of exact technical information has been purposely omitted from this paper.

We do know that we have a problem in the general group of A.P.C. viruses. While conjunctivitis is the outstanding symptom we still have sore throats and catarrhal otitis media. We hope to show at a later date that, apart from this epidemic disease, many of our catarrhal otitis cases may also be of viral origin.

Cat-scratch fever is a new entity that should be in our minds in unusual adenopathy and can be diagnosed by a specific skin test.

Many problems of the acute respiratory diseases are a challenge to us. One of these in a local community has been presented. The problem of the common cold is perhaps economically the most unsolved of these challenges which must be met.

100 COLLEGE STREET

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I wish to thank the Department of Ophthalmology, the Department of Bacteriology, and the Department of Medicine for their ever present co-operation.

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# THE VIRUS THEORY OF NASAL POLYP ETIOLOGY AND ITS PRACTICAL APPLICATIONS

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Virus pathology is generally characterized<sup>1</sup> by either proliferation or degeneration of cells, or a combination of the two. In this connection Rivers<sup>1</sup> has remarked: "If the action of a virus is not extremely rapid and explosive, and if susceptible cells are capable of multiplication, the primary effect of the active agent is stimulation leading to cellular hyperplasia." No rhinologist questions the fact that hyperplasia has occurred when polyposis exists in the nose or sinuses. That an alteration in the dimensions of a given polyp is not entirely dependent upon changes in contained edema fluid is evidenced by the fact that a pathologist is unable to determine polyp size from an ordinary microscopic section.

The tendency toward slow or rapid recurrence of nasal and sinus polyps after excision, their spontaneous appearance and disappearance with the onset and subsidence of head colds in certain patients, and their slowness or absence of recurrence after diathermy needle coagulation cauterization of the postexcision stumps, united to suggest the possible viral etiology of these polyps. It was reported in 1950 by Weille<sup>2</sup> that viral infection seems important in certain allergic states. If this observation is correct, then the hypothesis of a viral etiology for polyps merely elaborates upon the present widely accepted relationship in many cases between polyposis and a background of bacterial allergy. There is no real conflict between those insisting upon allergy as a causative factor and the possible virus etiology under discussion.

It was decided to test the hypothesis by an experimental study of nasal polyps excised in routine office or hospital practice for the

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From the Virus Laboratory of the Massachusetts Eye and Ear Infirmary.

possibility of a contained viral agent. It was fully appreciated that the mere presence of such an agent might be of great or of negligible importance in the puzzle of polyp etiology, but its absence, as determined by intensive study, would be strong evidence against the theory herewith proposed.

The clinician who so frequently tells a patient "you have a virus infection" often does so without the use of a virus laboratory. He may not be aware that even highly-trained bacteriologists must specialize in viral techniques before working in this field. The polyp etiology problem, as outlined above, was investigated in the bacteriology laboratory of Harvard Medical School over a two-year period by two workers approved by the chief of that department, Dr. Mueller. The first year was fruitless. Then Dr. Robert S. Gohd of that same laboratory, and later Director of the Virus Laboratory of the Massachusetts Eye and Ear Infirmary, undertook the study and accumulated a wide variety of data, some of which was "positive."

#### POSITIVE EXPERIMENTAL FINDINGS

Since a virus is always a total parasite in that it can multiply only by living a literally borrowed life within a cell, it was necessary to choose an experimental animal, preferably one which is both abundant and cheap. The chick embryo was decided upon because: a) it has been used successfully in the propagation of a large number of viruses; b) it is very sensitive to alterations in its metabolism; c) there is no development of antibody in the egg which would complicate and handicap the isolation of an infectious agent. Therefore an exhaustive study of the effect of nasal polyp suspensions on the fertile egg was undertaken. The first of several procedures to be tried was inoculation of the chorioallantoic membrane of 13 day old embryos with 0.1 cc of 10 per cent polyp suspension. Negative controls consisted of the same material heated to 60°C for 30 minutes. Every second day two controls and two test eggs were examined. The embryonic membranes; the allantoic, amniotic and yolk sac fluids; and the embryo tissues were observed and their appearance recorded. In addition, the fluids were tested for red cell agglutinins. The embryonic membranes were examined for cellular inclusions as were the heart, liver, lung, spleen, stomach, intestines and mucous membranes of the pharynx. A search was also made for gross changes in these structures, as well as for anomalies in the embryos. In addition to these experiments, all classic routes of chick embryo inoculation were



tried, including allantoic, amniotic, and yolk sac routes in 8-14 day old embryonated eggs.

A technique for the non-specific chemical detection of viral alterations of the allantoic membrane has been reported by Kilbourne and Horsfall.<sup>3</sup> This permits the recognition of viral infection by measuring the increased protein concentration of the adjacent allantoic fluid, and seems ideally suited for isolation of an unknown agent. Nonspecific false positives can be circumvented by the use of younger embryos or by dialysis of the test fluids.

Kilbourne and Horsfall found that after infecting the allantoic membrane by influenza virus, large amounts of protein could be demonstrated in the allantoic fluid. This was done by precipitation of proteins in the fluid with 10 per cent trichloroacetic acid and assessing the resulting degree of turbidity in the spectrophotometer.

TABLE I.  
RESULTS

| EGG NUMBER | POLYP | SALINE |
|------------|-------|--------|
| 1          | 320   | 120    |
| 2          | 350   | 60     |
| 3          | 370   | 90     |
| 4          | 400   | 90     |
| 5          | 330   | 160    |
| 6          | 350   | 140    |
| average    | 353   | 110    |

Our early experiments indicated that there were precipitable substances in normal allantoic fluid of 13-day embryos, whether inoculated with saline or uninoculated. This was different in character from a protein precipitate, but could not be differentiated from the latter by a turbidometric method. But by using 9- or 10-day embryos, with fluid for testing available at 11 or 12 days respectively, very little of the nonspecific interfering material appeared. Dialysis against ten volumes of saline removed it, suggesting it to be a small molecule (in contrast to large protein molecules).

Polyp extracts have been inoculated into these younger embryos and a considerable amount of protein has been found in the allantoic fluid 48 hours following the inoculation. The results are clear-cut. In a typical experiment the following values were obtained for the

optical densities of the turbid solutions (the optical density being also directly proportional to the protein concentrations<sup>3</sup> up to 500 turbidity units):

In this experiment there is about three times as much protein or precipitation material in polyp-inoculated eggs as in the controls. In studies with known influenza virus (Lee strain) it has been found possible<sup>3</sup> to centrifuge over 99 per cent of the viral agent out of the infected allantoic fluid with a reduction of only about 20 per cent in turbidity readings. Host reaction to the virus infection is believed to explain the increased protein, and a correlation may be drawn between the toxicity of the viruses studied by Kilbourne and Horsfall and the degree of protein increase.

If the cause of increased protein in allantoic fluid is due to a virus, then this effect should be transmissible. For example, a small amount of the polyp preparation is inoculated into several eggs. After a suitable incubation period, allantoic fluid from each egg is harvested, assayed for protein content and then pooled. This allantoic pool is called the first passage. A small amount of the pooled fluid is next inoculated into a second group of eggs and the eggs handled in a similar manner. Pooled allantoic fluid from the second group of eggs is called the second passage. If after several passages, a small amount of material from a previous passage still induces an effect in a subsequent passage, then the effect is a transmissible one and this property of transmissibility is a property of viruses.

The factor responsible for the increased protein in allantoic fluid appeared to be transmissible. Thus, material from the original polyp was passed in a serial manner five times. A second and a third line were started with two additional polyps. The third line was carried to the ninth passage and the second to the sixth passage. One disturbing feature of the transmission experiments was the loss of virulence of our agent with successive passage in eggs. This was manifest at first in a failure to infect all eggs; and finally in a complete loss of activity. The first line of polyp material went through five passages infecting five out of six eggs in the last passage. The second line became completely inactive for eggs at the eighth passage. Three out of six eggs were infected at the sixth passage, two out of six at the seventh and none of six were active at the eighth passage. Three of six eggs were positive at the level of the ninth passage of our third line.

## COMMENT

Basic experimental research is often fruitless, frustrating, and time-consuming in terms of years of effort. So little progress has been made in the past relative to polyp etiology that any encouraging experimental data deserve thorough exploration.

Our results are encouraging, but can be considered no more than suggestive. Huebner<sup>4</sup> and others have pointed out that the isolation of a viral agent from the nose and throat is insufficient evidence to implicate that agent etiologically with any given disease. Criteria modeled after Koch's postulates are required<sup>4</sup> for etiologic significance; Topley and Wilson<sup>5</sup> quote these as follows:

1. The organism should be found in all cases of the disease in question, and its distribution in the body should be in accordance with the lesions observed.
2. The organism should be cultivated outside the body of the host, in pure culture, for several generations.
3. The organism so isolated should reproduce the disease in other susceptible animals.

Attempts by us to produce gross or microscopic tissue changes in the nasal mucosa of experimental animals or in chick embryos have failed. However, the presence of a specific antibody concentration in the serum of polyposis cases, and its absence in controls, might be considered legitimate and important evidence. We hope to devise a serological test for exploring such a possibility. Immunological neutralization experiments with the Kilbourne-Horsfall chick embryo method may prove illuminating also.

If polyposis be considered of virus etiology, granted that the evidence for such is not fully proven, then certain clinical conditions may be explained at least partially.

1. Recurrence of nasal and sinus polyps and polypoid degeneration after surgical excision would be explained by simple proliferation of residual intracellular viral infection.
2. The gradual slowing down or absence of post-surgical recurrence, when diathermy needle coagulation of residual stumps of polyps or polypoid degenerative membrane is used, would have a rational basis. By such coagulation the tissue is "boiled" by the needle, and an hypothetical intracellular viral agent is destroyed.

3. Virus protein or virus infection in nasal and sinus polyps and polyposis may be important in the etiology of intrinsic asthma, vasomotor rhinitis, and allied conditions. If so, it is suggested that simple loss of virulence of the viral agent may be accompanied by improvement in the clinical condition. This is called a "spontaneous remission" in medical literature.

4. Adrenal steroid therapy in polyposis, as elsewhere, cuts down protein synthesis<sup>6</sup> about 50 per cent. This would strongly restrain a viral agent inducing cell proliferation since the virus and the cell compete with each other for the cell enzyme systems required for building materials. Alterations in extracellular edema fluid probably depend upon the vasoconstrictor action<sup>7</sup> of these compounds, a phenomenon unrelated to the presence of intracellular viral infestation.

5. The work is sufficiently encouraging to warrant experiments along related lines to search for a viral etiological agent in other types of benign tumors, such as laryngeal papillomata; the same may be said of malignant tumors.

Just what agent have we found? The following list enumerates some of the viruses that may be found in the nose or throat at the time acute manifestations of disease are present:

1. Common cold
2. Influenza viruses (A, B and C)
3. Primary, atypical pneumonia
4. Adenoidal-pharyngeal-conjunctival agents
5. Herpes
6. Poliomyelitis
7. Measles (and abortive measles)
8. Rubella (German measles)
9. Mumps
10. Pox viruses: chicken pox, small pox, vaccinia
11. Psittacosis
12. Q fever

It is unlikely that the virus present in nasal polyps will be identified as a member of the above list, but instead is probably a new addition. Kilbourne and Horsfall<sup>8</sup> have said: "It is conceivable that a virus might be recovered and identified immunologically as the



etiologial agent of a disease solely by chemical evidence of infection of the chick embryo."

#### SUMMARY AND CONCLUSIONS

In summary, a virus as the etiology of the nasal polyp is highly plausible. The work cited has shown that polyp suspensions are capable of inciting chemical changes in the allantoic fluid of the chick embryo leading to increased protein in that fluid. This phenomenon is capable of being serially transmitted for at least five passages. The incitant, following filtration, retains almost all of its activity. These factors considered together suggest a possible relationship between virus and nasal polyposis.

However, attempts to produce gross or microscopic tissue changes in the nasal mucosa of experimental animals or in chick embryos have not been successful.

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#### DISCUSSION

DR. LEWIS F. MORRISON: Mr. Chairman, there are two authors and two papers for discussion. Whenever possible, material common to the two papers will be discussed as a single subject.

It is a natural, but not necessarily excusable, reaction for the unprepared or poorly prepared individual, who does not prefer to retreat, to not only attempt to

stand his ground but to actually take the offensive. I intend to be offensive. Under these conditions the individual will be found to be critical and too frequently his criticism is destructive rather than constructive. It is distressing to have to admit that I not only do not possess sufficient exact knowledge on the subject to permit constructive criticism, but was unable to find anyone who could supply the desired information.

I experienced difficulty in finding a common ground for discussion of these two papers except:

A. The term virus is common to both.

B. Both papers indicate that the knowledge of the life habits of the viruses as related to that of man is not only desirable but may be essential.

C. Neither author fully qualifies the reference material. My experience permits the unpleasant inference that even if they are read the net gain will probably be confined to whatever satisfaction comes from becoming awed and confused more quickly but with more effort. Dr. Ireland does intimate that such might be the case.

D. Both authors draw attention to the fact that diffuse laryngeal papillomatosis may have a virus as an etiologic agent.

E. Both authors intimate that the viruses may be an important, if not the sole, inciting factor in causing otherwise normal cells to take on unorganized and uncontrolled characteristics—in other words become malignant.

From this point on the papers will be discussed separately for a time and joined again for summary.

The paper presented under the title of *Unusual Virus Diseases in Otolaryngology* by Drs. Ireland and Maloney will be discussed first. The initial sentence of the first paragraph is much too broad to be accurate. The exact and available knowledge relative to virus diseases is not so sufficient that anyone can be thoroughly familiar with the subject. I agree with the authors in their attempt to arouse interest in the practical clinical phases rather than an attempt to pursue or promote academic discussion on virus groups, their characteristics, classifications, and laboratory characteristics as shown by culture methods.

The Rosetta Stone supplied the key to the ancient Egyptian writings. It might be courteous to leave some similar key so that posterity may correctly interpret the present day trend toward using letters instead of words to designate specific conditions. For example, ARD is supposed to mean acute respiratory diseases. APC is supposed to indicate to even the casual reader of medical literature that it means Adenoidal-laryngeal-conjunctival virus infection. It is wholly possible that some S.O.B. (naturally, referring to senile old buzzard) will disagree with this statement.

The epidemiology of the clinical entities known as cat scratch fever and the virus conjunctivitis, and its association with the swimming pool, was worked out satisfactorily, definitely, and conclusively. The facts as related to cat scratch fever will vindicate some of the reactions of the typical aileurophobe.

I do not feel that the inclusion of the stated incomplete classification is of any value to the paper, and it does unnecessarily lengthen the article and tire the reader. The somewhat detailed differential diagnosis essential to identify the pathology producing the clinical entity of cat scratch fever from other conditions producing lymphadenopathy is very worth while.

The authors are not specific in regard to the details of the material used for the intradermal tests. I would appreciate knowing the details. It is evident that we will all have to pay more attention to the possibility of cross transmission of the virus diseases between man, animals, and birds. The recounting of the high incidence of an atypical pneumonia, first presented by Dr. C. E. Van Rooyen at the Toronto meeting should be of more than passing interest. The general practitioner in this community of 5,000 population should receive credit for his acumen in diagnosis. Although it has not been proved, it is wholly logical to believe that the human infection is directly attributable to the high incidence of virus sinusitis which is so prevalent in the turkey raised in this community.

The second paper, *The Virus Theory of Nasal Polyp Etiology and Its Practical Applications*, by Francis L. Weille and Robert S. Gohd, Ph.D., is more than controversial. There would be trouble enough defending the thesis of the viral theory of nasal polyposis without going into its practical application, or what is more disastrous, bringing in somewhat irrelevant material which distracts as well as annoys the reader. The following are examples:

a) The last sentence of the first paragraph reads in part, "a pathologist is unable to determine polyp size from an ordinary microscopic section." Actually, there is no more reason why a pathologist should have any more idea as to the size of the polyp as judged by the section he is examining under the microscope than the average geologist should have concerning the size and shape of a granite boulder when given only a small portion of this boulder to examine.

b) The statements that polyps occur less often following post-excisional diathermy needle coagulation owing to the fact that this method boils the polyp stump and kills the remaining virus in this tissue. There is adequate data to show that the routine course of the growth of polyps is frequently disturbed or changed as the result of tissue destruction or cellular insult in areas quite distant from the source of complaint. Autochemotherapy, wherein blood is drawn from the arm vein and injected in the donor's buttocks, was based on this phenomenon. The authors make no mention of the fact that polyp formation is not confined entirely to the nose mucous membrane, but is frequently present in the mucous membranes of the gastrointestinal and genitourinary tracts. It would seem evident that all of the mucous membranes participate in whatever condition is capable of producing specific tissue reactions.

c) I could not see any advantages in attempting to relate virus and allergy as etiologic factors and then to excuse this relationship by stating that at least they did not conflict with one another.

d) It seemed quite out of place to criticize the clinician on the basis that a patient is frequently told that his disease is a "virus infection" without having gone through the details of the necessary laboratory identification of a virus to present an exact diagnosis. It would seem that such an individual is less open to criticism than is the practitioner who, also for a fee, takes chances with the patient's life by indiscriminately offering a "shot" of some magic drug in the hope that it will solve all of the problems.

The outline of experimental work leaves no question but what there has been a large expenditure of time and effort in an attempt to defend the thesis that nasal polyposis is of viral origin. It is unfortunate that these workers were not able to have their etiologic agent be a polite, well-mannered, conforming type of large

virus. It is also unfortunate that whatever they have found which is considered responsible for the "phenomenon" is a non-conforming and extremely small agent, for which identification is dependent on a non-specific chemical detection of alterations of the allantoic membrane as demonstrated by an increased amount of protein in that fluid.

The basis for this identification is the work of Kilbourne and Horsfall, who contend that increased protein concentration of adjacent allantoic fluid is "ideally suited for the isolation of an unknown agent." The details of the testing and the increased turbidity of the units leave much to be desired as far as specificity is concerned. It is perfectly true that in this series of tests there is a difference of 353 to 110 as regards the response produced by the suspension made from ground polyps and the normal salt solution used as a control.

The assumption that a virus was a causative agent depends on the turbidity factor as stated and, what is perhaps more specific, the ability to transmit this factor for an average of five passages, and in some instances as high as nine passages.

It is unfortunate that the authors were not able to fulfill Koch's postulates, nor were they able to produce gross or microscopic changes in either the chick embryo or the mucosa of experimental animals.

The authors made no mention of any attempt to substantiate their contention relative to a viral etiologic agent by examining the allantoic fluid under the electron microscope. If such information is available, it should be mentioned. If it is not available, it is suggested that this be done. Also, there is no mention of attempts to centrifuge out the virus and then attempting to transmit the so-called "phenomenon" with the clear supernatant material rather than to use just the turbid allantoic fluid as such.

The authors' statement of hope relative to producing some serilogic test which will permit the presence of the virus or group of viruses, as the case may be, to be determined in a simple manner is ground for speculation.

The analogy between the more or less common papillomatosis in the rabbit and the multiple papillomatosis of the larynx of man is wholly acceptable. The experimental work with the rabbit disease has resulted in the isolation of a large virus with which the investigators had been able to fulfill all of Koch's postulates. Of further interest is the fact that this large virus has been shown to be susceptible to treatment with members of the Tetracycline series of drugs. The application of this form of therapy to man has given encouraging although not completely uniform results.

The authors are making a sincere effort to establish a virus as an etiologic agent which is responsible for the presence of nasal polyps. I do not feel that their contention that a virus is responsible is wholly tenable.

I do want to compliment both of these contributors on their work. The fact that I disagree with much that has been presented is probably based on lack of education and intelligence sufficient to substantiate their presentation.

DR. PERCY E. IRELAND: I would like to say that this paper will be published as a joint paper. As you know, in most institutions a good bit of this work is done by the more junior man, Dr. Terence Molony, a good Irishman who came to us instead of Dr. Schall, is in this position and is present with us. I wish he would stand up so that you could see him.



I am very grateful to Dr. Morrison for his kind discussion. Perhaps it is better to try and talk in simple language. He was much more kind to me than to Dr. Weille. I think it is better not to present too many details of procedures and you can perhaps get away with more.

There is one point that I want to say something about and that is concerning the high potency antigen. Dr. Morrison asked me about that. The antigens are easy to make from an actual case. The material, which is not purulent, is taken from the so-called pustule. When I say a high potency antigen, I can assure you they are all attenuated. There is no doubt about that, but certain of these lesions at stages will not reproduce the skin test. If you are making a new antigen you want to test it from several of the lesions because there is one which will react to another antigen that will not react to certain of the material that is taken from a local lesion.

As far as the turkey disease, I think that represents one of our failures. We have tried and tried to get something specific on this disease entity and I might say there is considerable opposition from a certain large packing company who owns the 30,000 turkeys.

DR. FRANCIS L. WEILLE: In our staff meetings at the Massachusetts Eye and Ear Infirmary we always claim the privilege of being as critical as we please with anybody and now this can work both ways.

In the first place, as regards diathermy and tissue insult, may I point out that Dr. Morrison says that these people get better but he does not propose any alternative explanation. You can say "stress" if you like but he does not tell me why the patients get better any more than I tell you myself. But I have proposed a reasonable hypothesis with a certain amount of scientific backing.

As regards allergy in relation to a possible virus infection in the etiology of polyps, of course you gentlemen know that there are people (and I am sure some of them are in this audience) who believe there is no relationship between allergy and nasal polyps in all cases; that there are cases where the two are not related. Dr. Schenck said years ago about this relationship, that allergy is almost invariably present. I am willing to avoid any battle on that point because I myself believe there is no conflict between the idea of virus infection (if you want to call it an idea rather than an hypothesis or theory) and allergy to ordinary pathogenic bacteria. The basic immunological response to a virus is the same as to hemolytic streptococcus, for example. I know that allergists are in the habit of freely using vaccines in some of their patients. Let us say, to get out of Boston, that a New York group, Cooke's group, certainly is enthusiastic about bacterial allergy. Just because a virus happens to be measured in millimicra instead of being a larger agent is not important immunologically; the virologists believe that the immunological responses to viruses are similar to those of ordinary bacteria. An example is small pox. There you can get a vaccination scratch and you are supposed to be immune for a long time if a "take" occurs.

As regards the electron microscope, when I started being interested in this problem I thought one to be essential. Since then I think it will surprise Dr. Morrison to know that this is not so. We have just built a very well equipped new virus laboratory. In the course of construction the hospital virologist was able to ask for and receive whatever equipment he wanted. We already had an electron microscope in our Retina Foundation and another in the Massachusetts General Hospital. It was not elected to put one in the virus laboratory because this worker thinks he

can borrow one occasionally if necessary. Usually the electron microscope in the methods of virology is not essential.

In attacking the Kilbourne and Horsfall method, the method may mean nothing to Dr. Morrison. It seems unreasonable to him to take a non-specific chemical reaction and say that means the presence of a virus. May I point out that the people who published this technique are very highly regarded workers in the Rockefeller Institute for Medical Research in New York and that the article is published in the proceedings for the Society for Experimental Biology and Medicine, and that my virus worker, our virus worker in Boston, the co-author of this paper, is willing to stack his reputation with other virologists that this work will stand up against anybody saying that we did not find the virus.

About skin tests: we tried them. I assume that, because in allergy cases the antibody is recognized as a reagin. Dr. Morrison believes we ought to look in the skin for polyp virus antibody. We did with human beings and could not find anything. I think myself a much better search could be made by serological studies. We hope to look into this possibility.

## MANAGEMENT OF NON-MALIGNANT GROWTHS IN THE MAXILLARY SINUS

O. E. VAN ALYEA, M.D.

CHICAGO, ILL.

The list of non-malignant growths found in the maxillary sinus is a large one and includes the rarely encountered tumors, such as papilloma, adamantinoma, fibroma, adenoma, angioma, lymphoma, plasmocytoma, lipoma, osteoma, chondroma, neuroma and the more common types of growths, polyps and cysts.

It is my purpose in this paper to deal largely with the two growths most frequently encountered in clinical practice, namely, polyps and non-secreting or mesothelial cysts.

These growths are entities in the cause and maintenance of many cases of maxillary sinus disease, but because they have a tendency to exist quietly in the antrum producing few symptoms traceable to them, rhinologists are inclined to minimize their importance.

In not all instances, however, should the presence of these growths be dismissed so lightly. Too often the identification and elimination of an apparently inoffensive antral cyst or polyp has been followed by a cessation of symptoms which previously had resisted all diagnostic and therapeutic efforts.

During the past several months at the Research and Educational Hospitals of the University of Illinois and the Illinois Eye and Ear Infirmary, we have developed considerable interest in these growths. They have been observed in the routine x-ray examination of patients presenting symptoms referable to the maxillary sinus and occasionally have been unexpectedly revealed in films taken following the installation of iodized oil.

In a careful analysis of any large series of cases presenting symptoms suggestive of sinus involvement the influence of these growths soon becomes apparent.

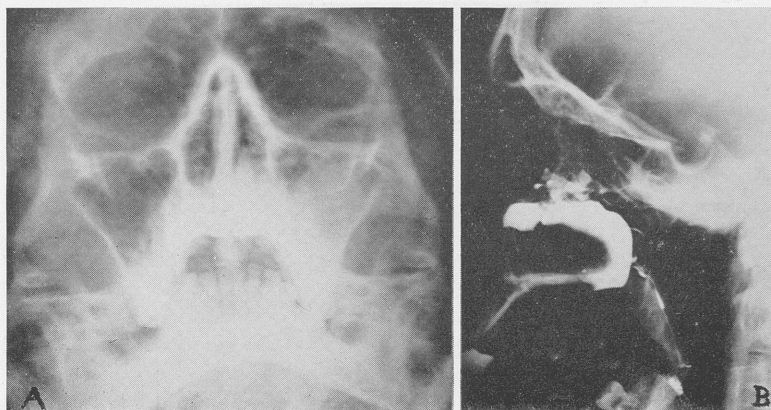


Fig. 1.—Case I. A) X-ray (Waters view) reveals a large cyst almost completely filling left maxillary sinus. B) Lateral view depicts the filling defect.

In our study they have proven to be of extreme interest because of the influence they have on maxillary sinus disease and because of the unique symptoms they cause.

#### PATHOLOGY

*Polyps.* Antral polyps may appear singly or in groups, existing as independent growths or along with generalized nasal hyperplasia or polyposis. They develop as a result of irritation of the sinus mucosa, the irritant being an allergen, a pathogen or a toxic exudate from a long-standing draining, infected sinus.

The histopathology is essentially that of edematous respiratory epithelium. A frequent site of polyp origin is the margin of the maxillary ostium, the growths developing from prolapse of an overloaded mucosa. If the prolapse is in the direction of the nasal cavity the polyp may remain in the middle meatus or, as is occasionally the case, extend to the posterior nares where it forms choanal polyp. Polyps remaining in the vicinity of the ostium, whether within the sinus or in the middle meatus, are usually a strong factor in the etiology and continuation of attacks of purulent sinusitis.

*Cysts.* Cysts common to the antrum appear on the walls singly or in multiple formation. They vary in size from a few millimeters



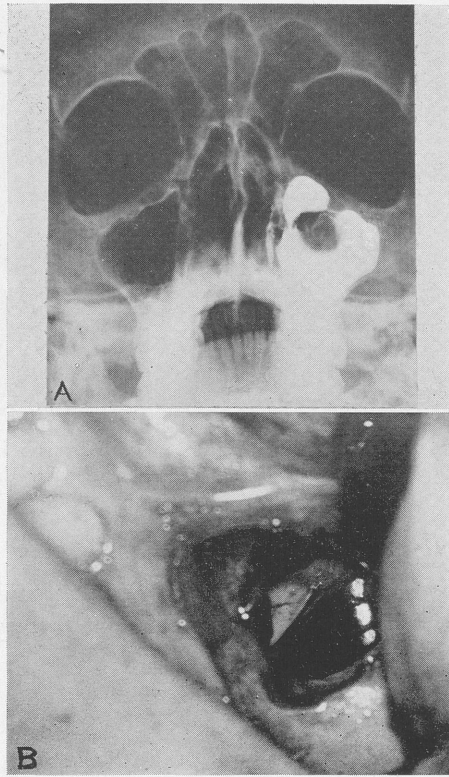


Fig. 2.—Case II. A) Round central filling defect left maxillary sinus caused by mesothelial cyst. B) Cyst exposed through sublabial opening before removal. 4X.

in diameter to one which expands to fill the sinus completely. The non-secreting cysts presumably of allergic origin develop in edematous sinus mucosa as distentions of the subepithelial areolar spaces and contain a clear pale or straw colored fluid.

The small mucous secreting cysts are of less clinical importance. They are, in effect, dilated glands from the sinus mucosa, the ducts of which are obstructed, probably on an inflammatory basis.

To be mentioned, but not considered in this paper are two types of odontogenic cysts, radicular and follicular. Both are of less frequent occurrence than the membranous cysts.

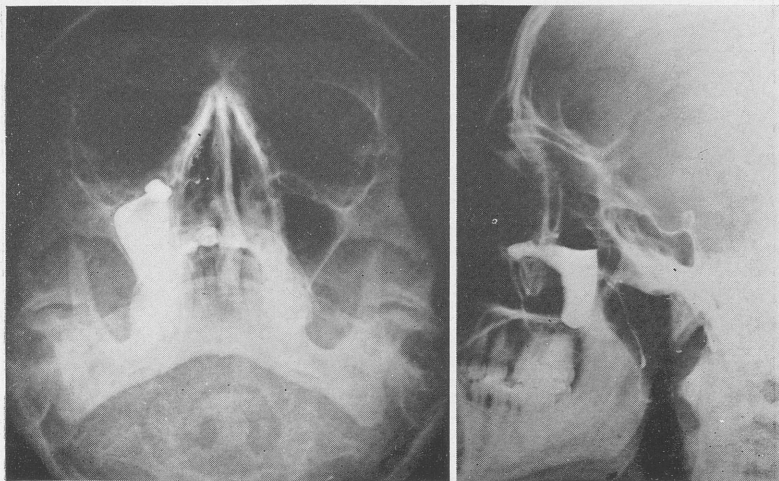


Fig. 3.—Case III. X-rays, Waters and lateral views, showing large filling defect in right antrum. At operation, this proved to be a cyst.

#### SYMPTOMS

Typical of the symptoms of antral cysts are a feeling of fullness or numbness in the cheek, with nasal blockage and frontal headache on the affected side. In many instances, however, no symptoms referable to the sinus are elicited although in some, bizarre and remote symptoms are present which may be attributable to these growths. Similar symptomatology is characteristic of antral polyps with the added one of suppuration which is present in most cases as a chronic or recurrent sinusitis.

Some of the unusual complaints which have been attributed to or at least appearing coincidental to growths in the antrum are occipital or temporal headache, serous otitis media, areosinusitis, postnasal discharge, recurrent sore throats, generalized body aches and pains, nervousness, irritability, fatigue, optic neuritis and other ocular complaints.

#### DIAGNOSIS

The presence of these growths may be determined by x-ray examination with and without the use of opaque media. Smooth

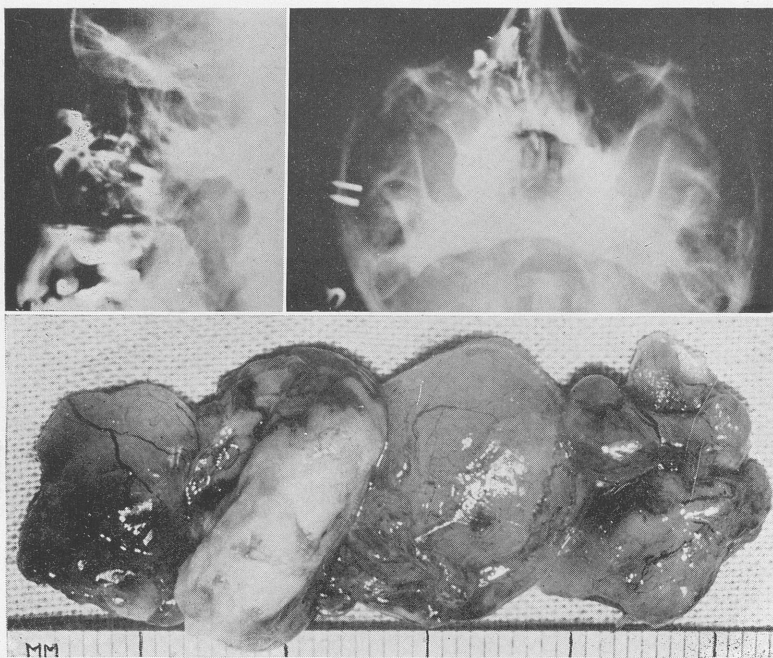


Fig. 4.—Case IV. X-rays, lateral and Waters views, right side taken with iodized oil in the right antrum showing large filling defect. Photograph showing group of 4 polyps removed from sinus.

round dome-like shadows in the floor with uniform density throughout are as a rule indicative of cyst formation. Irregular filling defects are more than likely caused by polyps, although one should always consider the possibility of other types of neoplasia, including malignancy. Smooth, thickened mucosa lining the sinus walls is significant of inflammatory sinusitis, frequently allergic in character. Cysts are not always located in the sinus floor. They may arise from any of the sinus walls and be revealed as rounded, central filling defects. In advanced cases there may be little discernible lumen in the cavity, this being occupied almost entirely by growths or thickened, edematous mucosa, or by various combinations of cysts and polyps. Cysts are less likely than polyps to be associated with sinus infection for seldom are they located near the sinus ostium, although this is a common site for the location of polyps. In cases of nasal polyposis, polyps may also be present in the antrum, this being especially true



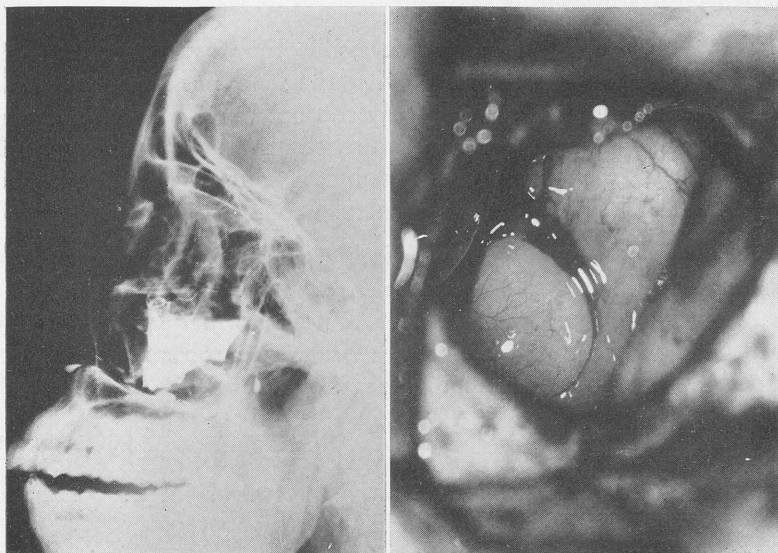


Fig. 5.—Case V. A) Lateral view showing filling defect in the right antrum caused by polyps. B) Polyps in right antrum seen through sublabial exposure. One large and two small polyps were removed. 4X.

in long-standing cases of recurrent nasal polyps when associated with suppurative sinusitis.

#### TREATMENT

*Cysts.* Cysts in the antrum should be considered as of clinical importance even though symptoms attributable to the growth are absent. When recognized, their removal should be seriously considered if for no other reason than because of the carcinogenic possibilities of all apparently innocent growths. More classical indications for removal appear as the sac reaches such proportions as to cause discomfort in the cheek or if symptoms are present near or remote which possibly could be attributed to the cyst or additionally if the sinus is associated with an inflammatory process.

The sacs are on occasions punctured at the time of sinus lavage, occurring as the antral needle penetrates the wall of the cyst. The cyst may also be reached and obliterated in the course of a nasoantral



window operation. If direct vision is deemed more desirable, a sublabial surgical approach is required.

*Polyps.* Antral polyps are accessible by way of the sublabial route. The polyps are removed with the least possible disturbance of the sinus mucosa and are routinely sectioned and studied histologically. The area of the ostium is carefully scrutinized for small growths which also are removed and the procedure is concluded with the institution of an inferior meatal naso-antral window.

Rarely is it necessary or advisable to remove portions of the sinus mucosa. Thickened membranes, even though polypoid in nature, tend to return to normal or a near normal state following antrostomy and control of allergy. Under such circumstances even polyps have been observed to disappear.

Approximately twenty cases were included in the present study. Some of these were old chronic cases which had defied all manner of treatment, others were of comparative recent origin in which the diagnosis was made on routine study.

CASE 1. G.R., white male adult. Complained of frequent head colds with sore throats, pain under the left eye of two months' duration and bilateral otalgia of several years' duration.

On x-ray a large dome-like structure was revealed which almost completely filled the left maxillary sinus (Fig. 1). On February 22, 1956, the antrum was opened through the canine fossa and a large cyst was exposed. This collapsed at the first touch and a large quantity of clear pale fluid escaped. A naso-antral window was made in the inferior meatus and the sublabial wound was closed with interrupted sutures. His symptoms disappeared following the operation and he has remained symptom free.

CASE 2. Mrs. P.E., white female, age 56. For years has suffered from an annoying recurrent sore throat with mild postnasal drip and at times she was conscious of a feeling in her left cheek. She had consulted various otolaryngologists throughout the country and had been studied from an allergic and endocrinologic standpoint. X-ray films revealed a suspicious looking round area in the left antrum. The sinus was irrigated with a negative return and iodized oil was instilled for further x-ray study. This clearly revealed a round shadow centrally located (Fig. 2). On March 10, 1956, a cyst was removed through the canine fossa. Following this the symptoms referable to the cheek disappeared but the throat symptoms remain.

CASE 3. L.B., white male, age 41. Complained of stuffiness in nose for three years with postnasal drip and pain in right cheek for the past two months.

At operation, March 6, 1956, a large bluish colored mesothelial cyst was removed from the anterior wall of the right antrum (Fig. 3). Symptoms greatly relieved since operation.

CASE 4. O.C., white male, age 26. Complained of nasal discharge from the right side for ten years. Had polypectomies in 1945 and 1953.

X-ray studies revealed large filling defect in the right antrum, which on exposure on August 12, 1955, through the canine fossa proved to be caused by a collection of four polyps (Fig. 4). A naso-antral window was inserted. The nasal discharge of many years' duration has subsided.

CASE 5. H.H., colored female. Complained of blocked nose, frequent colds, nasal discharge often.

Operation March 26, 1956, revealed three polyps on the medial wall of the right antrum near the maxillary ostium (Fig. 5). Irrigation through the window has been followed by a remission of symptoms.

#### COMMENT

In numerous instances, cases presenting symptoms referable to the maxillary sinus and many others with unusual complaints, fail of correct diagnosis because the basic cause of the difficulty, namely, benign growths in the sinus, have been overlooked or their importance minimized. In some of these, efforts have been directed toward diagnosis and treatment of the maxillary sinuses but have failed because routine films of the sinuses without the employment of contrast media revealed no filling defects and on irrigation of the sinus there was a negative return and no information was gained. In other cases, irrigation therapy and even intranasal antrostomy have failed to produce a termination of the infection. These are regarded by many writers as cases with irreversible mucosal change until a careful study results in establishing a correct diagnosis and the elimination of the antral growths.

## SUMMARY AND CONCLUSIONS

1. Antral cysts and polyps may be present themselves in the maxillary sinus and be responsible for the symptoms, yet escape recognition.

2. These growths appear more frequently than is generally recognized. They may appear as single or multiple formations. They may be present with or without nasal polyposis, and in some instances, both cysts and polyps are present.

3. These growths may be revealed on routine x-ray examination but are better delineated following instillation of an opaque medium.

4. Smooth round shadows on the floor of the sinus are as a rule indicative of cysts. Irregular filling defects are more than likely caused by polyps, although other non-malignant and malignant growths may present a similar clinical picture. Smooth, thickened mucosa lining the sinus walls is significant of allergic or inflammatory sinusitis.

5. Cysts are often eliminated by puncture at the time of sinus lavage or they may be obliterated through an intranasal antrostomy opening. Polyps are removed through a sublabial surgical approach.

6. Choanal polyps commonly arise in the maxillary sinus. Investigation of remaining neoplasia, even though the polyp and its stalk have been removed, is essential. This applies especially to cases of recurrent nasal polyps.

7. All growths recovered from the sinus should be carefully studied microscopically.

135 SOUTH LASALLE STREET

## DISCUSSION

DR. R. L. GOODALE: I entirely agree with Dr. Van Alyea that the common conditions of polypi and cystic disease of the maxillary sinuses merit more consideration than they are frequently given. One frequently thinks of benign tumors in terms of benign neoplastic disease, overlooking the fact that inflammatory and other tissue swellings, which are not neoplastic, may also be classified as tumors in the general sense. I have always felt that there is too much tendency to belittle the role of cysts of the maxillary sinuses. Certainly I do not agree with the often-heard statement that simple cysts of the sinus are of no consequence at all. To be sure many cases, in which a cyst is accidentally discovered in a routine x-ray, are

symptom free. But I am not willing to dismiss a cyst as of no significance. I feel that quite as important as the x-ray finding is the clinical history. Where a patient has persistent evidence of nasal infection which seems to localize on the same side as the cyst, I feel that we must give it more emphasis. In some quarters there is a definite swing away from this opinion and at times I have been led to minimize a maxillary cyst. An illustration is a case which I saw four years ago where a patient was referred from an ophthalmologist for focal infection to account for uveitis. As the uveitis was progressing it was quite important to eliminate all infections as rapidly as possible. An x-ray showed a cyst in the right maxillary sinus. Examination showed also some purulent discharge on this side, and chronic infected tonsils. I felt that the tonsils were more likely to be a focal infection and consequently removed them and decided that the sinus could wait. The uveitis improved slightly but did not clear up and subsequently I decided to remove the cyst. I was surprised to find that the cyst was not a simple cyst but contained frank pus and on culture this proved to be pneumococcus. Following this procedure the uveitis definitely cleared up. From an x-ray point of view I think it is very difficult to distinguish this type of infection from a simple mucus cyst. For this reason I feel that where there is a threat to the patient's health, one should not neglect the possibility that a cyst is at fault. So many of our recent cases, which have been complicated by infection, have responded to the newer drugs that we have swung definitely away from the surgical approach to this type of sinusitis. I think we should be on our guard lest we take it for granted that the drugs can supplant surgery. In my own practice polypi and cysts are quite common findings. I think that in the case of a polyp of the middle meatus or one that has developed within the sinus, surgical removal is the procedure of choice. In the latter case the canine fossa approach is the most satisfactory. In a case where the disease is localized to a small area and where the rest of the sinus cavity seems to have a normal mucous membrane, I would merely remove the pathology as Dr. Van Alyea suggests, leaving the normal mucous membrane intact. At times I have felt that one need not provide additional drainage into the lower meatus. This would be a matter of judgment at the time of the operation. Where infection is a serious factor, I would definitely make such an opening.

Dr. Van Alyea's suggestion in regard to unsuspected malignancy in such cases is an interesting one. Certainly in any operation, one should send his material to the pathologist for confirmation. I think it would be a rare case, however, that would escape the eye of an expert clinician or radiologist. Such things can happen as we all know, and once in a while a middle turbinate or what appears to be a nasal polyp may, under the microscope, prove to be malignant.

DR. LEWIS F. MORRISON: Dr. Goodale presented one answer.

I would like to ask Dr. Van Alyea to answer questions in regard to whether or not the fluid in the cysts had been sent for bacteriological examination and, if so, what were the results of the examination.

A review of the literature shows that a majority of the operators make no endeavor at all to determine whether or not the cystic fluid contains viable organisms. The minority who have been sufficiently interested to send the fluid for bacteriological examination are almost equally divided into one group who maintain that the fluid is always sterile and the other who intimate that the failure to obtain a positive culture should almost be considered as being a technical error rather than any conclusive demonstration that organisms were not present.

Would you please give us a report on your findings, sir?



DR. ROBERT E. PRIEST: I would like to ask Dr. Van Alyea, if he feels that surgical exploration of all antra showing cysts is justified? I base this question on a case in which I and several other people passed up a benign appearing smooth appearing structure at the bottom of the antrum and the patient finally came back to me with pain and I biopsied the lesion and it was a mixed salivary tumor. It looked exactly like any number of benign cysts that I have seen and passed. Within the last two weeks I was approached by a general practitioner who had a film of a cyst in the bottom of the antrum with minimal symptoms which looked exactly like the one I just described. Frankly, I don't know where to draw the line between the ones you look at surgically and the ones you don't.

DR. OLIVER E. VAN ALYEA: I wish to thank Dr. Goodale, Dr. Morrison and Dr. Priest for their discussion.

There does not seem to be a great deal of argument here on this paper, Dr. Morrison. We culture the cyst material obtained at operation routinely but I have nothing to report concerning the results. It is not always necessary to do a surgical operation through the canine fossa to remove cysts when identified. This was done in this study primarily for the purpose of observing and studying them in situ. I find that these cysts are often ruptured during irrigation. On other occasions they may be reached and obliterated by the insertion of a needle through the naso-antral wall. The day I left my office to come here I irrigated a chap's antrum, the first time I had seen him. This was accomplished through the natural opening. I am sure the tip of the cannula did not come anywhere near any cyst, yet out poured a straw colored, watery fluid and I feel that just the force of the irrigation probably ruptured the cyst wall. I know in operating these cases, while we were trying to get the blood out of the way for the photographer the cysts often ruptured, merely because a little gauze was brushed over the top. They are very delicate.

Probably that also accounts for some of the phantom shadows that have been reported. X-ray films reveal a round shadow in an antrum whereas at operation it is not there. I think the chances are that the cyst probably ruptured while the surgeon was approaching it.

# EXPERIMENTAL TREATMENT OF RECURRENT CARCINOMA OF THE NASOPHARYNX WITH ELECTRODESICCATION, RADIOACTIVE COBALT AND X-RAY RADIATION

FRANCIS A. SOOY, M.D.

SAN FRANCISCO, CALIF.

The work on this problem was undertaken in an effort to provide additional relief for six patients with recurrent carcinoma of the nasopharynx.

In each instance the patient had been given external x-ray radiation or reradiation to skin tolerance and the presence of recurrent carcinoma was proven by biopsy and nasopharyngeal smears.<sup>1</sup>

Intracavity radiation and electrodesiccation had been previously used in the treatment of primary tumors of the nasal septum<sup>2</sup> and from this study it was felt that a partial septectomy was a practical method of providing access to the nasopharyngeal vault both local treatment as well as to facilitate adequate follow-up inspection.

The technique used was the same in each case and consisted of:

1. *Intranasal Septectomy* (Fig. 1). Under orotracheal anesthesia the entire posterior three-fourths of the nasal septum was resected leaving only the columellar portion for support.

The margins of the defect were electrocoagulated to control local bleeding which was vigorous in some instances.

This portion of the procedure was carried out solely to obtain exposure of the tumor area in the nasopharynx.

2. *Electrodesiccation of the Tumor Area* (Fig. 2). By means of a special intranasal tip, the tumor area and adjacent mucous membrane was thoroughly electrodesiccated. Care was exercised not to

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From the Sub-Department of Oto-Laryngology, Department of Surgery, University of California Medical School.

Read before the American Laryngological Association, Montreal, Canada, May 13, 14, 1956.

apply the current too long at one time for fear that the sphenoid bone would conduct sufficient heat to the pituitary and optic nerve areas to cause secondary degeneration. However, at the conclusion of the procedure all the mucosa in the area should be thoroughly charred.

3. *Insertion of Radioactive Cobalt* (Fig. 3). Morrison and Low-Beer<sup>3</sup> first reported the use of radioactive cobalt<sup>60</sup> in the nasal cavity and sinuses, and it is their method which was employed.

Cobalt<sup>60</sup> which has been rendered artificially radioactive has a half life of 5.3 years, and differs only slightly in its radiologic and therapeutic characteristics from filtered radium.

The fact that it can be worked to an appropriate size and shape in its inactive state, and then "heated" or "reheated" to the desired degree of radioactivity is a significant advantage.

The Foley urethral catheter is an excellent retainer since the bag can be distended with a radiopaque solution (diodrast) and it is then possible by means of x-ray films to measure the exact distance between the head and the surrounding tissue. This permits an accurate calculation of depth dosage. The bag further serves to maintain the cobalt at the correct distance from the tissue to assure uniform radiation.

The catheter is held in position by means of anterior and posterior nasal packing.

Radioactive cobalt emits almost pure gamma radiation and the treatment time was calibrated so that the patient received 4000 to 6000 gamma roentgens at one centimeter distance divided in two applications one week apart.

The use of additional radiation in an area which has already been heavily radiated was approached with some caution. However, the tolerance of the nasopharynx to radiation can be enormous, e.g., Case 5 in which the patient was subjected to 36,000 roentgen and 1,400 milligram hours of radium to the nasopharynx over a four year period with no lasting harmful effect to his nasopharynx, or unfortunately, his tumor.

Postoperatively there is initially considerable crusting and local radiation mucositis in the nose. This gradually subsides after 6 to 8 weeks and the nasopharynx begins to assume a normal appearance except for local small areas of sequestering bone principally in the anterior sphenoid and posterior ethmoid area.

This process may produce a chronically granulating area which is easily confused with recurrent tumor. The nasopharyngeal smear has been of considerable help in differentiating these lesions.

The patient should be entirely comfortable after the first two months but may require several months for complete separation of the necrotic bone.

Follow-up examinations were made at monthly intervals for the first year and then once every three months after this.

#### RESULTS

All six patients were considered to be hopeless prior to carrying out the procedure.

Three of these patients are well and have been entirely free from any evidence of recurrence for periods of eight years, six years nine months, and six years after the procedure.

One patient died eight months after the procedure from extension of her cervical metastasis. There was no evidence of nasopharyngeal tumor at the time of death. The other two expired within 13 months of their procedure.

Both of these cases had early x-ray evidence of involvement of the base of the skull.

In Case 1 a radium plaque was used instead of cobalt for technical reasons.

We have subsequently accumulated ten additional patients where this method has been applied and of these five are dead while five are living and well for periods varying from six months to four years.

In some of these cases this method of treatment was carried out as the primary treatment but always in combination with external x-ray radiation.

The over-all results can be summarized as follows:

|             | DIED | LIVING |
|-------------|------|--------|
| 0-6 months  | 2    | 1      |
| 6-12 months | 2    |        |
| 1-2 years   | 4    | 1      |
| 2-3 years   |      | 2      |
| 3-5 years   |      | 1      |
| 5-8 years   |      | 3      |



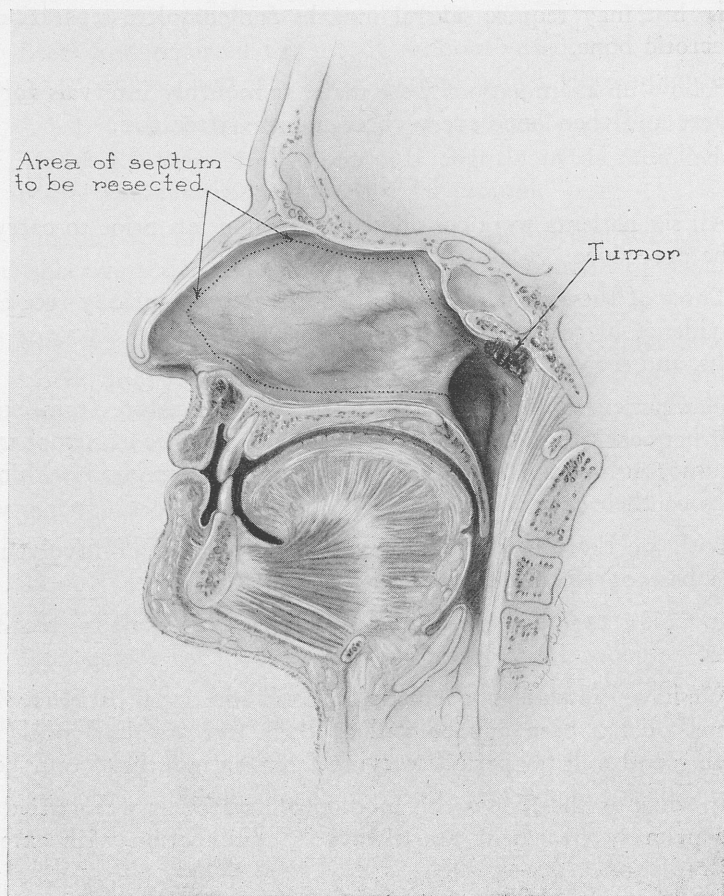


Fig. 1

## COMMENT

Since the series of cases presented is so small it was hoped that a longer follow-up might help evaluate the results.

It is felt at the present time that intensive external x-ray radiation is still the treatment of choice but that there are very useful adjuncts to this therapy and these include:

1. Intranasal radiation with either radium or radioactive cobalt in dosages approximating 6000 r at 1 cm distance. This should be done in every case.

2. Electrodessication can be effectively used in this condition as evidenced by Case 3 in which there was proven recurrence after external radiation and cobalt radiation which was controlled by thorough fulguration of the nasopharyngeal lesion and has remained under control for six years.

3. Septectomy is indicated only when the primary tumor is large and local removal combined with fulguration is desired or when no further local or external radiation can be given and electrodessication is used in the hope of bringing the tumor under control.

*Complications.* Initially it was feared that a severe atrophic rhinitis and ozenae would result from the procedure but this has not been the case and the patients are quite comfortable with steam inhalations during dry weather.

We have, however, been troubled by a persistent dry adhesive otitis media in two cases and one (Case 3) will soon require a hearing aid to continue working.

In one patient of this series and two others a unilateral prophylactic neck dissection was carried out by the referring surgeon in spite of the absence of any palpable nodes on the side of the lesion. In each specimen microscopic metastases were present. Clinical or x-ray evidence of extension of the primary tumor to the base of the skull or to the brain precludes successful therapy in our cases.

Eustachian congestion which persists and particularly serous otitis in a Chinese patient should be regarded with the utmost suspicion.

In three cases an early diagnosis of carcinoma of the nasopharynx was made by means of a prophylactic biopsy into what appeared to

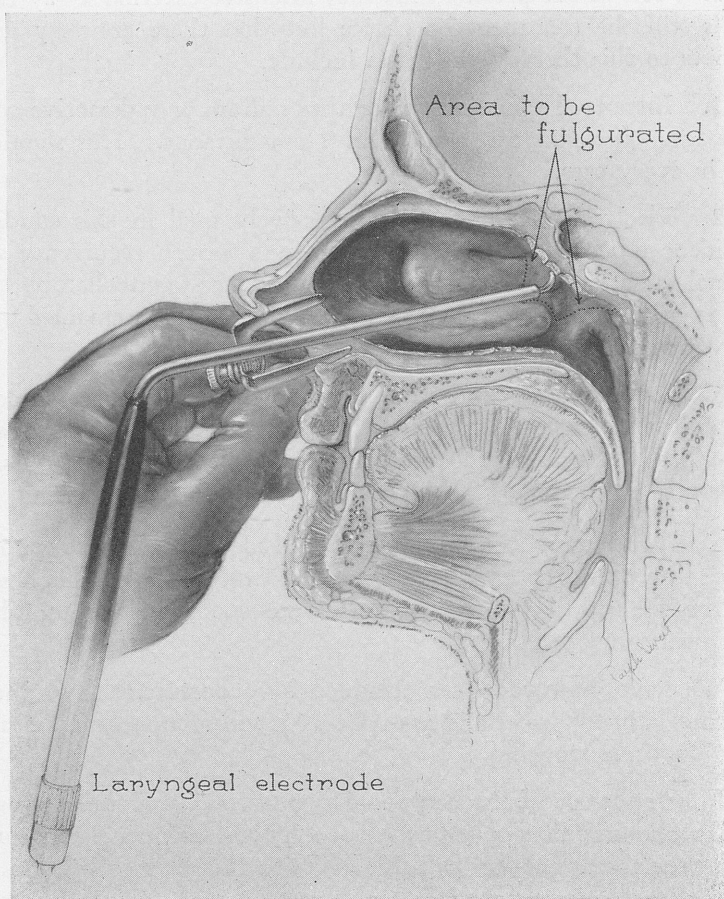


Fig. 2

be a completely normal nasopharynx, when the eustachian symptoms did not respond to adequate therapy.

#### SUMMARY

1. An additional treatment of carcinoma of the nasopharynx is described. This consists of:

- A. Intranasal septal resection.
- B. Intranasal electrodesiccation of the tumor area.
- C. Local intranasal radiation with radioactive cobalt.

2. Initial studies indicate that 50 per cent of the patients were benefited. A larger series of cases with a prolonged follow-up is necessary before any valid conclusions can be drawn.

3. The procedure seems to have little effect when the tumor involves the base of the skull.

4. Six illustrative cases are presented.

#### REPORT OF CASES

CASE 1.—E.M.H. This white male druggist aged 59 was first seen at the University of California Hospital Out-Patient Department on October 12, 1945, complaining of left nasal obstruction, bleeding and left fronto-occipital headache.

Examination revealed a large, fungating mass in the left nasopharynx involving the base of the septum.

Subsequent investigation revealed he had undergone a biopsy two years and four months previously which showed: "Anaplastic carcinoma, grade IV."

This was treated with "radium applications" and his symptoms subsided.

Four months later he received 600 mg hours contact radium therapy for a recurrence.

Rebiopsy two years and four months after his initial diagnosis showed no malignancy; however, his symptoms continued and one year and nine months later he received an additional 1250 r of 200 kv x-radiation to his left posterior nasal area.

The patient did not appear again until March 10, 1948, four years and nine months after his initial diagnosis, at which time he again suffered from left nasal obstruction, bleeding and headache. Biopsy at this time revealed "transitional cell carcinoma of the nasopharynx," and nasopharyngeal smears were also positive.

Two months later a cautery resection of the entire three-fourths of the nasal septum was carried out under general anesthesia and the tumor site fulgurated with



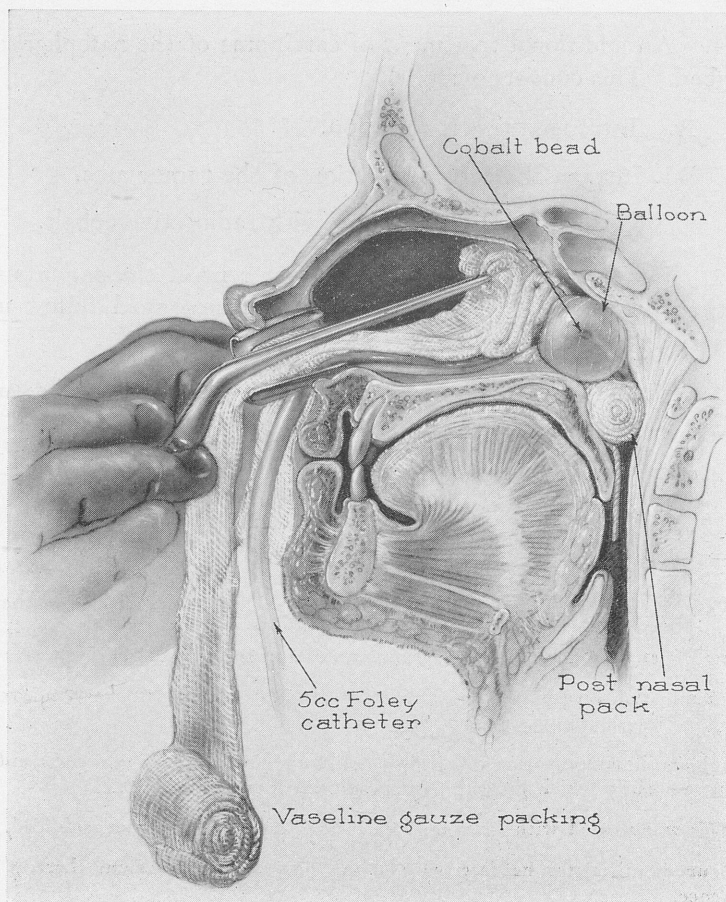


Fig. 3

the bovie unit. Following this procedure the nasopharyngeal smears became negative but his severe headache persisted, and two months postoperatively malignant cells were again found in his smears.

He was again admitted and more complete cauterization of the nasopharynx was carried out under general anesthesia. Preliminary biopsy failed to reveal any malignancy.

He again remained comfortable for two months when he noticed a recurrence of bleeding and pain and malignant cells were found in his smears.

He was again admitted and under general anesthesia a radium plaque consisting of four 10 mg needles was inserted over the tumor area at the base of the septum and held in position by anterior and posterior nasal packing. He received a dosage of 1020 mg hours.

Following this treatment his headache entirely disappeared and no further malignant cells have been found in his smears to the present time, eight years after his last treatment.

There is a slight crusting of the nasal mucosa in dry weather which is controlled by steam inhalations and he did not have any difficulty with a mild diabetes mellitus during his many procedures.

No cervical nodes have been demonstrated at any time and repeated skull and sinus x-rays failed to show evidence of bone invasion at the base of the skull.

This patient subsequently developed a pea sized new squamous carcinoma of the nasal vestibule which was resected locally and not connected with his original lesion.

CASE 2.—J.G.R., a 52 year old Portugese trucker, was first seen at the University of California Hospital Out-Patient Department on March 5, 1948, complaining of progressive right nasal obstruction, occasional bleeding and foul discharge of two years' duration.

Examination revealed a large, fungating mass filling the right nasopharynx and involving the base of the nasal septum. The lesion was biopsied and reported as "lymphoepithelioma of nasopharynx." Nasopharyngeal smears also showed malignant cells.

He was then given a full course of external radiation to his nasopharynx to a total tumor dose of 4000 r.

No cervical adenopathy was present.

The patient remained well and his smears were negative until July 29, 1949, when the mass in his nose recurred and his smears were again positive, one year and four months after his initial treatment.

He was admitted to the hospital and under general anesthesia the recurrence was biopsied, found to be positive, and the lesion thoroughly fulgurated with the bovie unit intranasally. Following this a 6 mm radioactive cobalt<sup>60</sup> bead in a Foley catheter was placed in the right side of the nasopharynx and packed in position. A total of 5000 gamma roentgens were administered at 1 cm distance over 58 hours.

On October 19, 1939, three months later, a recurrence was noted in the vault of the nasopharynx on the right side. Both smear and biopsy were positive.

The patient was re-admitted and the entire posterior three-fourths of the nasal septum was resected under general anesthesia and the tumor site thoroughly cauterized with the bovie unit.

Three months later he was clinically well but his nasopharyngeal smears showed evidence of malignant cells.

He was re-admitted and radioactive cobalt<sup>60</sup> was again applied in a Foley catheter to the nasopharynx for a total dosage of 4000 gamma roentgens at 1 cm over 31 hours.

Since this treatment he has remained clinically and cytologically well to the present time, some six and one-half years after his last treatment and over seven years after his initial diagnosis.

He has only a minimal crusting in his nose and does not feel that it is bad enough to require steam inhalations or douches.

Repeated x-ray examination of the base of the skull failed to reveal any evidence of bone involvement.

A 1 cm area of necrotic bone over the anterior face of the right sphenoid was finally extruded leaving the sphenoid sinus open.

CASE 3.—P.Q. This 35 year old Chinese housewife was first seen on August 8, 1949, complaining of a large cervical node of several weeks' duration.

Examination revealed a small pea sized mass in Rosenmueller's fossa on the right side of the nasopharynx. There was a single large, hard node in the middle of the right posterior cervical chain. Biopsy from the nasopharynx showed a "transitional cell carcinoma." Nasopharyngeal smears were positive.

X-ray examination of the chest revealed no metastasis and x-rays of the base of the skull were normal.

X-ray treatment was given to the nasopharynx and neck area for a total tumor dose of 4700 r.

She remained clinically well for seven months at which time her nasopharyngeal smears again became positive. A rebiopsy again revealed lymphoepithelioma of the nasopharynx.

On April 8, 1950, under general anesthesia, the entire posterior three-fourths of the nasal septum was resected intranasally and the edges cauterized.

The tumor site was thoroughly desiccated and a radioactive cobalt<sup>60</sup> bead in a Foley catheter was packed in place in the right nasopharynx.

A total of 6000 gamma roentgens was administered at 1 cm distance over 45 hours.

Postoperatively there was considerable crusting for eight weeks at which time the nasopharynx was well epithelized except for a small area of exuberant granulation tissue which was further cauterized under general anesthesia.

There was subsequent sequestration of the anterior face of the right sphenoid and the nasal wall of the large right posterior ethmoid cell leaving these sinuses widely open.

Repeated nasopharyngeal smears and biopsies at three month intervals have failed to reveal any evidence of recurrence.

The patient is almost entirely free of nasal crusting and there has been no evidence of recurrence of her cervical nodes to date, six years after her last treatment and six years and nine months after the initial diagnosis was made.

CASE 4.—O.B.Y. This 39 year old white female was first seen at the University of California Hospital on August 11, 1949, complaining of a mass in the left side of her neck and stuffiness in the left ear and left nostril of two months' duration.

Examination revealed a chain of large, hard, fixed nodes along the left sternomastoid muscle, fluid behind the left drum, and a large, fungating mass in the left nasopharynx. This was biopsied and reported as lymphoepithelioma of the nasopharynx. Her chest x-ray was clear.

She was given a full course of external x-radiation to her neck and nasopharynx. The total tumor dosage was 4187 r.

This treatment was tolerated poorly and she was given supportive therapy in the hospital for two and one-half weeks.

She was subjectively improved but her smears remained positive consistently and a small amount of tumor could be visualized high in the nasopharynx.

She was re-admitted four months after her radiation and under general anesthesia the entire posterior three-fourths of the nasal septum was resected intranasally and the margins cauterized. The tumor site was thoroughly cauterized and a cobalt<sup>60</sup> bead in a Foley catheter was inserted and packed in place in the nasopharynx. A total of 4000 gamma roentgens were delivered at 1 cm over 31 hours.

Following this she developed considerable crusting in the nasopharynx and several small sequestra were sloughed out. Three months later she developed a cellulitis in her right neck and subsequent biopsy of this area revealed metastatic carcinoma.

Her last nasopharyngeal smear was negative but she continued to develop more tumor in her neck and after a downhill course, expired some eight months after her last treatment, and 13 months after her initial biopsy.

Repeated x-ray examinations of the base of her skull failed to reveal any evidence of bone involvement.

CASE 5.—H.C.W. This 41 year old Chinese male was first seen in the University of California Hospital Out-Patient Department on March 27, 1950, complaining of recurrent nasal bleeding, obstruction, decreased hearing and a numbness of his right cheek of two months' duration.

Investigation revealed he had received the following treatment for a microscopic diagnosis of "lymphoepithelioma of the nasopharynx."

February 1946—5100 r to nasopharynx.

February 1946—1200 mg hours of radium locally to the nasopharynx.

December 1946—Lesion recurred and he was given 3200 r to the nasopharynx.



January 1947—200 mg hours of radium locally to nasopharynx.

October 1947—Biopsy again positive and patient received 5400 r additional x-radiation to nasopharynx.

March 1948—Recurred again and 3600 r of x-radiation were given through and intraoral cone and 3780 r externally to the nasopharynx.

August 1948—Again recurred and 6000 r were given through three neck fields to the nasopharynx.

September 1948—3172 r were given intraorally.

July 1949—1200 r were given to base of skull for severe pain.

Initial examination revealed a large, flat, bleeding mass occupying the right nasopharynx. There was partial ankylosis of the jaw and marked skin changes from radiation. No cervical nodes were felt. Biopsy revealed "transitional cell carcinoma—nasopharynx," and the smears were positive. Chest x-rays showed no metastases but x-rays of the base of the skull revealed some asymmetry but no definite invasion. The patient is a known diabetic.

The patient was hospitalized for a severe spontaneous nasal hemorrhage which subsided after postnasal packing and antibiotic therapy.

He was re-admitted and under general anesthesia on June 5, 1950, an intranasal resection of the posterior three-fourths of the nasal septum was carried out and the margins cauterized. The tumor area was thoroughly desiccated and a radioactive cobalt<sup>60</sup> bead in a Foley catheter was inserted into the nasopharynx and fixed in place with packing. 500 cc of whole blood was given on the table because of the profuse bleeding encountered.

A total of 6000 gamma roentgens were administered over a treatment period of 51 hours at 1 cm distance.

Postoperatively he developed the usual crusting and required additional antibiotic therapy because of his diabetes.

A complaint of blurring of vision was attributed to his marked diabetic retinopathy.

Additional skull x-rays a month later now revealed evidence of destruction of the base of the skull on the right side.

Two months later biopsy of a cervical lymph node on the right side revealed no tumor.

One month later his nasopharyngeal smears and biopsy were again positive.

His course from this point was steadily downhill, but the time of his expiration was not obtained.

CASE 6.—B.L. This 48 year old Korean male was first seen in July 1950 with evidence of recurrent carcinoma around the right eustachean orifice.

Investigation revealed that he had been seen two years previously at which time he complained of glands in the right side of his neck of one and one-half years' duration. No primary lesion could be found but biopsy of his cervical nodes revealed "lymphoepithelioma."

He was given external x-ray treatment until a tumor dose of 4500 roentgens was reached and was well until August 1949 when he showed evidence of a mass in the right side of his nasopharynx. An additional 4355 roentgens was given partly intraorally to the nasopharynx.

He was free of further symptoms until July 1950, some ten months later, when he again developed a mass in the right side of the nasopharynx and was referred for septal resection and cobalt irradiation.

Chest x-rays revealed no evidence of metastasis and skull films showed the petrous bone to be intact.

On direct examination of the nasopharynx there was a small nodular mass in the area of the right eustachean orifice which partly occluded the tubal orifice.

The right middle ear contained dark fluid and the drum was retracted.

The skin on the right side of the neck showed some radiation telangiectasia but no nodes were palpable.

Biopsy of the right tubal area revealed "transitional cell carcinoma, nasopharynx."

On July 13, 1950, the entire posterior three-fourths of the nasal septum was resected intranasally and the margins cauterized. The tumor site was thoroughly fulgurated and a bead of radioactive cobalt<sup>60</sup> in a Foley catheter was packed in place in the nasopharynx and a total of 8000 gamma roentgens was administered over a period of 35 hours at 1 cm depth.

Postoperatively he did poorly with continued right V nerve pain.

Three months later his nasopharyngeal smear was again positive and examination of skull x-rays showed evidence of definite extension into the petrous area.

Minimal crusting was present in his nose but the tumor site again showed evidence of activity.

He expired 14 months after his septal resection, after receiving one of the experimental cancerocidal chemicals with some subjective alleviation of his symptoms.

Biopsy of an ear polyp on the right side showed carcinoma.

#### 490 POST ST.

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I am deeply grateful to Drs. Lewis F. Morrison and the late Bertram V. A. Low-Beer for the privilege of using radioactive cobalt in these cases.

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## DISCUSSION

DR. HARRY P. SCHENCK: This is a very important clinical paper. I believe it teaches us not to be too pessimistic about some of the malignancies that we encounter. I have been impressed for a long time with the fact that we do not use ordinary electrocoagulation widely enough in otolaryngological practice.

Many instances of epistaxis which formerly required extensive surgical procedures in the neck are frequently controlled quite adequately by the use of electrocoagulation. The reason failure occurs quite frequently is because we attempt to use electrocoagulation under local anesthesia. It is obvious one must use general anesthesia so you can carry out the electrocoagulation with precision and with a certain degree of thoroughness.

I think the climate in California must be more salubrious than in Philadelphia because when I destroy a septum they get horrible crusting for six months. After that interval, strangely enough, there is some adjustment of the organism so that the crusting disappears and thereafter there is little difficulty with crusting. Perhaps that is a peculiarity of the local geography.

I feel that Dr. Morrison and Dr. Sooy have been particularly fortunate in finding so few metastases. These look like fairly large tumors to me. Unfortunately most of ours have metastases to the neck frequently, sometimes on the opposite side of the neck rather than on the same side.

The advantage of their technique is obvious. One can reach all of the nasal pharyngeal areas and the destruction of the large amount of tumor tissue enables them to concentrate their radiation more effectively.

I would like to make one more point and that is that we should not limit the attack to carcinomas. I know of two cases of chordoma, one of which I inherited from the neurosurgeons four years ago because they were about to send him home to die and he had complete destruction of his sella turcica, complete nasal pharyngeal obstruction. I largely carried out an extensive procedure of electrocoagulation, because I thought I would give him an airway. We did not destroy the septum. I used the electrodes through the nostrils alternately and in part up behind the soft palate. That case is still living. He has no symptoms except about once in two months he has a temporary diplopia. I don't know what we have done but in my experience all chordomas of that extent have expired in that interval. It may be that we have been fortunate enough to secure a certain amount of regression.

This is a very instructive paper and one from which we can all derive a great deal of benefit.

DR. THOMAS C. GALLOWAY: Dr. Coates, Mr. Chairman and Fellows, you know that for many years I have been very enthusiastic about electrosurgery when there was no proper irradiation available.

There are some things we should know about: first about the type of tumor—was it lymphepithelioma or transitional carcinoma, which we know are very malignant? Early they are not sharply localized and I doubt that such a procedure as this would be sufficient in most of the cases. I notice that they did have x-radiation and how much of the result was due to that and how much to the cobalt bomb? This procedure of taking out the septum to get the bomb in there impresses me a little like Charles Lamb's story of the Chinaman who burnt down a house every time he wanted roast pig.

By nasal approach to these tumors one did not get the proper exposure. If you are going to treat such a tumor with electrocoagulation you must completely expose it and see it and that can be accomplished by Dr. Mosher's old incision here (indicating) or as I have done by a trap door incision from which I removed carcinoma from both frontals, both antra and the sphenoid successfully. I have done it transpalatally and it seems to me if you are going to take out that much tissue you are entitled to have your tumor right where you can see it directly under your eye.

I think these tumors which arise from the septum arise from mature cell squamous epithelium, at least mature type of respiratory epithelium, and are a little different tumor. They partake of the character of the parent cell and they are much less malignant and they can be effectively destroyed. But I still would wonder if it isn't worth while to make these incisions which are not necessarily non-mutilating.

There is no doubt that all x-radiation and cobalt, as well as all these modalities, are being used much more intelligently. I can remember when Dr. Furstenberg—I don't think he is here—stated that he had not seen one of these transitional cell tumors of the nasopharynx cured. I have had the good fortune in the last few years to see more of them controlled and I thought when the glands were involved and when they were the malignant type that one accomplished not much by electro-surgery; if the tumor involved bone and thus could not directly be taken care of by the radiation, in that case electrosurgical destruction is undoubtedly a very valuable thing. I am glad that Dr. Schenck emphasized that we should re-educate ourselves in the use of diathermy.

Incidentally I take it that the doctor is not using a desiccating current which is a very superficial, non-penetrating current but is using probably a biterminal, deeper electrocoagulation which one should use with a really malignant tumor.

DR. FRANCIS A. SOOY: I would like to thank both Dr. Schenck and Dr. Galloway for their discussion.

In answer to Dr. Galloway's question, our desiccating unit is adjusted so that we plan to achieve a slough of one centimeter of bone and mucosa. In other words, we plan to dessicate it deeply enough so that when the final sequestration occurs there will be a slough of about one centimeter of tissue which may leave the sphenoid sinus wide open and occasionally the posterior ethmoid cells.

The matter of a transpalatal approach was considered but it was felt that in order for us to follow these patients and to be able to watch the area where the tumor might recur we still wanted a posterior septectomy. One last remark, in the overall series of our cases, the initial symptom you will notice —

(Slide) Is most frequently either cervical metastases, nasal obstruction, or bleeding, but I would like to call attention to the ear symptoms, which though small in percentage represent in our opinion the earliest symptom relative to these tumors and to emphasize that, an unresolved sero-otitis in a young person which defies treatment longer than an average period of time should be considered to be due to a carcinoma of the nasopharynx until proven otherwise. We have gone so far in three Chinese patients to do a simple prophylactic biopsy of Rosenmüller fossa on the side of an unresolved serous otitis and in each of these three instances have obtained a positive biopsy. Incidentally, all of these cases were in the transitional cell lymphoepithelioma group.



ON CIGARETTE SMOKING, BRONCHIAL CARCINOMA  
AND CILIARY ACTION  
IV. CILIARY STREAMING THROUGH THE LARYNX  
AND DISTRIBUTION OF LARYNGEAL  
EPITHELIUM

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Recently I have been exploring the possibility of the relation between ciliary action and carcinoma of the respiratory tract.<sup>1,2</sup> This involves a series of studies, one of which, of necessity, is the mapping of the ciliary streaming through the larynx. When first planned, it seemed simple enough. It seemed sufficient to put some indicator on living ciliated epithelium in the larynx and then sit back and record what happened. Unfortunately, it proved not to be so simple. Many unexpected difficulties arose, hence the study has not been completed and this must be in the nature of a progress report.

Closely related to the matter of ciliary streaming, of course, is the distribution of ciliated columnar and squamous epithelium within the larynx. It seemed desirable to study this facet of the subject as well.

A search of the literature failed to reveal any description of ciliary streaming through the larynx.

A number of good descriptions of the distribution of epithelium in the larynx were found. Low-Beer and Morrison<sup>3</sup> have written one and Hopp<sup>4</sup> another. Hopp found that the true cords are covered with stratified squamous epithelium, which has small papillae. This squamous epithelium is predetermined before birth. During the first year of life, the ventricular folds also become covered with squamous

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epithelium. The interarytenoid area is lined with squamous epithelium but he states that, although there are occasional islands of squamous epithelium in the adult larynx, he believes these are rare. The margin of the epiglottis is also covered by squamous epithelium; otherwise by and large the larynx is lined with ciliated columnar epithelium.

#### MATERIAL FOR STUDY

The material used for the study of the distribution of epithelium consisted of five human larynges removed at autopsy. For the study of ciliary streaming forty-three calves' larynges were used. These were obtained on the killing floor at a packing plant very soon after death. Most of them were brought to the laboratory, but some were used right at the packing plant.

#### STUDY: DISTRIBUTION OF LARYNGEAL EPITHELIUM

Of the five human larynges studied for the distribution of the epithelium, the best preparations were obtained from two men, one 61 and the other 81 years of age. The interior surfaces of both of these larynges were stained with one per cent methylene blue and studied under a Greenough dissecting microscope by means of reflected light.

The study on the 61 year old man yielded these findings: The squamous and ciliated cells were fairly well stained and readily differentiated. The line of transition from the columnar ciliated epithelium of the subglottic area and the squamous epithelium on the cord was a very sharp one and followed along the margin of the cord. No squamous cells were found below this line. Following this line of squamous epithelium anteriorly it was found to be continuous until the anterior commissure was reached. There, in the anterior commissure, directly in the midline, it was interrupted by a thin line of ciliated columnar cells extending from the subglottic area below the cords to the ciliated regions in the vestibule. So, anteriorly, in this specimen there was continuous columnar ciliated epithelium in the bronchial tree through to the anterior commissure, upward between the cords to the ciliated epithelium on the vallecule of the epiglottis. Going posteriorly, the line of squamous epithelium continued as far as the arytenoids. The area over the arytenoids was mixed but mostly squamous. The interarytenoid area contained both types of epithelium; the lining was folded into ridges, the tops of which tended to be squamous while the valleys between were ciliated columnar. The ventricle was found to be lined by ciliated columnar epithelium.

The larynx from the 81 year old man proved to be somewhat different. This larynx was split a little to one side of the midline anteriorly and posteriorly and the halves stained and laid out for examination. As in the previous specimen, all of the cells below the line of squamous epithelium on the cords down through the subglottic area into the trachea were of the columnar ciliated type. Many glands were seen in this region just below the cords and they seemed to be arranged more or less in parallel curving lines, directed upward and backward toward the interarytenoid area. Squamous cells were found to begin on the margin of the cords along a very sharp line running parallel with the axis of the cords. In this instance, this line of squamous epithelium extended all the way to the midline anteriorly. There were no ciliated cells going through the commissure. It extended also to the process of the arytenoid cartilage posteriorly and across this into the interarytenoid space to the midline. Again posteriorly, I could trace no ciliated cells continuously all the way from the subglottic to the supraglottic region. There may have been some where the scissors crushed the epithelium. In studying the folds in the interarytenoid area, it was found that some were squamous on the ridges and ciliated in the valleys. Some, on the other hand, were found to have the reverse arrangement. (It may be that the normal premortem folds were distorted and shifted during the dissection.) Excepting for that portion formed by the superior surface of the true cord, the ventricle was lined entirely by columnar ciliated epithelium. The squamous band extended from the margin of the cord a little distance over the upper surface toward the ventricle. The inferior aspect or margin of the false cord also was covered with squamous epithelium. Most of the medial and upper surface of the false cord was found to be ciliated, as was the area above the false cord in the anterior portion of the larynx, until, going outward, it became squamous at the aryepiglottic fold. Toward the posterior end of the false cord, there were many islands and peninsulas of squamous epithelium surrounded more or less completely by columnar ciliated epithelium. As stated, squamous epithelium was encountered on the aryepiglottic folds but even much of this region was ciliated. The epithelium over the arytenoids contained a mixture of squamous cells and ciliated cells. The epiglottis on its laryngeal surface was very largely ciliated with squamous epithelium on the margin.

In a word, the distribution of epithelium, as found in these studies, agreed well with that which is commonly accepted, with the

exception that above the cords the squamous epithelium was not sharply outlined. There were numerous islands and peninsulas of squamous epithelium which were surrounded by ciliated epithelium and also there were numerous folds, the tops of which tended to be squamous epithelium and the depths ciliated. As the aryepiglottic folds and the margins of the epiglottis were approached, an irregular mixture of squamous and columnar epithelium was found. On the very margin of both, it was all squamous.

In these specimens, it was found that there were threads of mucus protruding from the mouths of the glandular ducts. When these threads had been divided from the mucous blanket, they retracted forming small, globular or mushroom-like masses at the opening of the glandular ducts. When they still remained attached to the mucous blanket, they stretched out for considerable distances and were seen in other specimens to act like anchors for the mucous blanket, holding it stationary against the pressure of the cilia. The significance of this will be mentioned later.

#### ANIMAL EXPERIMENTS: CILIARY STREAMING THROUGH LARYNX

Most of the tests of ciliary action were made in the larynges of calves as soon after death as it was possible to get the specimens. India ink was used to make the ciliary streaming visible; small drops were placed on the mucosa at the level of the upper trachea or lower larynx. The specimen was then placed upright and progress of the ink through the subglottic area to the cords and into the upper larynx was observed. Great difficulty was encountered in obtaining adequate ciliary streaming. It was first thought that the specimens became too cold, so they were placed in an incubator in which the air was well laden with moisture, but this did not remedy the difficulty. Then, to shorten the time-lapse after death, experiments were carried out at the packing plant, but the results were disappointing. There was some flow but nothing to compare with that which is commonly found in the trachea. Three different types of insulated containers, providing warm, moisture-laden air, were used but did not solve the problem. It was then discovered that by placing portions of the bronchial tree in plastic bags and leaving them at room temperature, the cilia would continue beating for upwards of 36 hours without any special attention. This was tried on the larynges with only a measure of success. Despite these difficulties, some fairly satisfactory observations were made. From the 43 specimens, enough information was



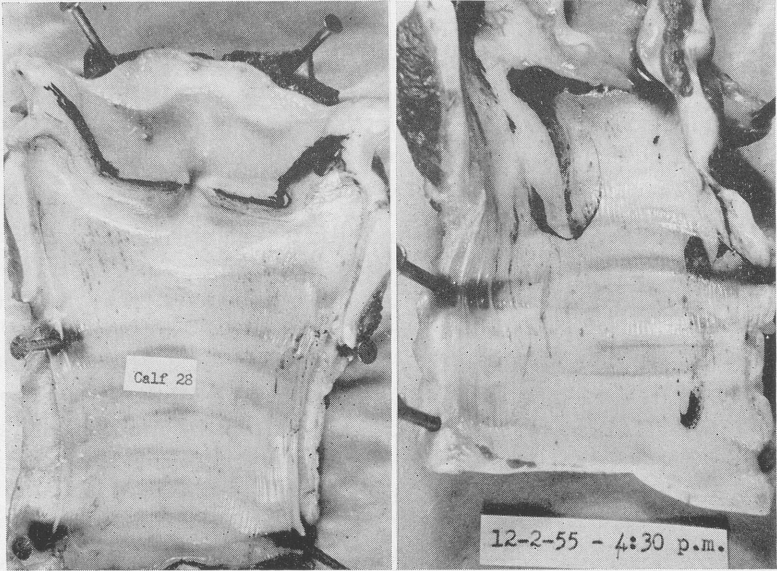


Fig. 1.—Larynx split in the midline posteriorly and spread open. The ciliary flow posteriorly along the cords from the anterior commissure is clearly indicated by the distribution of the ink.

Fig. 2.—One-half of a calf's larynx showing the ciliary streaming in the subglottic area. On the lateral wall, it passes backward and upward in a smooth curve to the interarytenoid space.

gleaned so that a general idea was obtained of ciliary drainage through the larynx. In some, the progress of flow could be followed from the trachea to the pharynx although it was sluggish (Figs. 1 and 2).

Drainage through the larynx in these animals actually begins down at the bifurcation where the streaming from the right and left main bronchi comes together at the coryna. On the coryna, about at the middle, the streams from the medial halves of both the main bronchi divide and pass anteriorly and posteriorly, making two main streams in the trachea, one on the anterior surface and one on the posterior (Fig. 3). It does spread out somewhat before reaching the larynx, but, in general, these two main streams seem to be maintained. The stream along the posterior tracheal wall passes directly up into the interarytenoid space and up to the edge of the larynx where, in

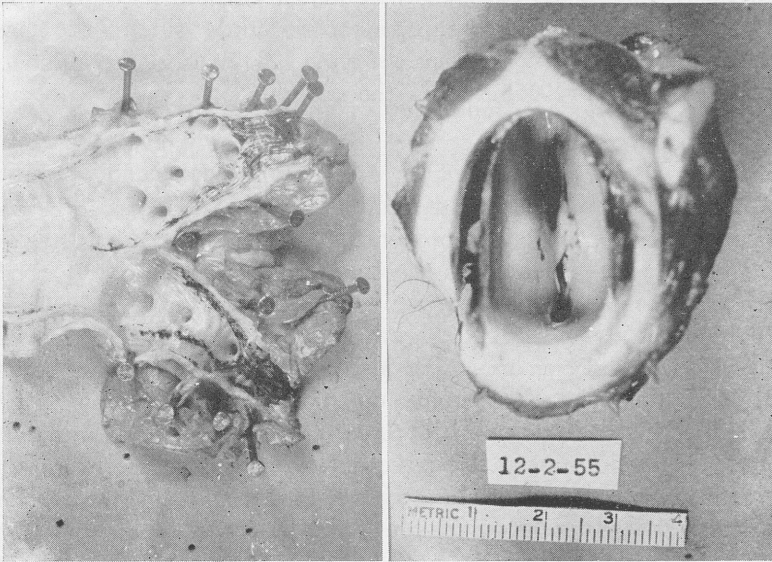


Fig. 3.—Bifurcation of a cow's trachea with India ink indicating the two main streams of flow on the anterior and posterior walls of the trachea and how they are formed at the coryna.

Fig. 4.—Calf larynx seen from below with India ink marking the flow on the inferior surface of the cords. Anteriorly (below) there is a pool at the anterior commissure from which the ink streams backward (up) into the interarytenoid space.

the living state, it undoubtedly is carried over into the pharynx and thus down into the gastro-intestinal tract. The stream on the anterior wall passes up through the trachea and through the lower subglottic region until it approaches the cords. Here a portion of it bends posteriorly, in more or less of an even arc, to the arytenoids and over the arytenoids and out into the interarytenoid space. The very central portion passes directly upward until it reaches the margin of the cords and the anterior commissure. A little pool tends to form at the anterior commissure, the rest turns and follows the margins of the cords posteriorly—some on each cord (Fig. 4) to the arytenoid and thence over the arytenoid, up to the interarytenoid margin of the larynx. In some specimens, the ink did not reach the anterior commissure or the anterior third of the cords. Instead, it described a

circle surrounding these parts, as though a blob of mucus had come from above and passed between the anterior extremities of the cords and through the anterior commissure to the under side of the cords. If the anatomy of the ventricle is the same as in man, such a discharge of mucus could have come from the sacculus.<sup>5</sup>

#### DIRECTION OF BEAT OF CILIA

Attempts were made to determine the direction of the beat of the cilia. As the cord is approached, the ciliary stream turns and passes backward. There seems to be little doubt but that the cilia of the trachea generally beat in the axis of the trachea. The question arises, do they keep beating directly upward as they approach the cords or do the cilia on the cords just inferior to the margin beat at right angles, toward the posterior portion of the larynx? I have watched the cilia in action and have tried several experiments to establish this point but so far have not succeeded in determining if the direction of beat just below the margin of the cords is different from that in the subglottic region or in the trachea.

#### COMMENT

The distribution of epithelium in the few human specimens obtained was found to correspond closely with that which is generally accepted, except that above the cords the pattern of squamous epithelium was varied and inconstant. The whole of the margin of the cord was found to be covered with squamous epithelium as was the anterior portion of the false cord and the rim of the epiglottis and the very edge of the aryepiglottic fold. The arytenoid area and the interarytenoid area, the posterior half of the false vocal cord and the lateral portions of the upper larynx contained a mixture of squamous and ciliated epithelium. There were numerous squamous islands and peninsulas. In one of the specimens examined (an 81 year old man), squamous epithelium seemed to extend entirely around the glottis, i.e., on both vocal cords, across the anterior commissure and over the arytenoids and across the posterior commissure. (How was mucus drainage accomplished in this man? Perhaps he had difficulty. Some elderly people must "clear the throat" very frequently.) There was ciliated columnar epithelium both below and above this ring; there did not seem to be any continuity through from that in the subglottic area and that above the cords. In one specimen continuous ciliated columnar epithelium was found to con-

nect the ciliated epithelium above and below the cords both through the anterior commissure and in the interarytenoid area—although that through the anterior consisted only of a very thin line.

It is difficult to demonstrate ciliary streaming well in the larynges of newly killed calves. The reason is not entirely clear. Stomach content is frequently seen in the larynges and, since acid tends to stop ciliary action, it was thought this might be the reason it was so difficult to demonstrate it and undoubtedly this is one factor. But, obviously, there is something else that has nothing to do with this or with drying from evaporation or with cooling. Many times it was noted that the ciliary action was vigorous but that the mucous blanket did not move. In several specimens, it was noted that fragments of the mucous blanket were sometimes held stationary by long strands of thread-like mucus which came out of the mouths of the glands and acted like anchor ropes. A probable explanation of this difficulty might lie in cessation of secretion of viscid mucus by the glands soon after the time when circulation ceases. It is quite possible that that mucus which at the moment is being produced in the cells fails to be completely extruded and remains attached within the gland. Since it extends through the duct into the lumen and is attached to the underside of the mucous blanket, it could act as an anchor. If enough such anchors are present, the mucous blanket could remain stationary even though the cilia beat vigorously beneath.

Be that as it may, a fair idea has been obtained as to the course of ciliary streaming from the trachea through the larynx. The ciliary streams flowing upward from the main bronchi are divided at the coryna in such manner that there is a special concentration along the anterior and posterior walls of the trachea. The flow in the trachea is usually more or less vertical. As the streams approach the larynx, those on the posterior wall ascend vertically between the arytenoids to the posterior margin of the larynx; those in the anterior portion of the trachea ascend vertically until they approach the vocal cords, then the most lateral deviate, passing obliquely upward and posteriorly, cross the arytenoids, thence course into the interarytenoid space to the upper margin of the larynx. The central portion of the heavy anterior stream flows directly to the margin of the cords, then turns posteriorly and follows the cords back to the arytenoid and thence out with the other stream. At the anterior commissure, the mucous blanket must divide. This means that at one point or line just at the commissure, movement would come to a standstill as the blanket is torn asunder



to pass in two opposite directions. How much the blanket is slowed up in the anterior part of the cords as it continues to tear is probably variable. The slowing would depend a great deal upon the viscosity of the blanket. If the viscosity were low, there might be almost no change in the velocity of flow; if the viscosity were high, the velocity might be sharply reduced until the blanket had been torn apart and no longer offered a pull across the midline.

It is important to know if the direction of ciliary beat alters as well as the direction of flow. This I have been unable to determine as yet. It may be that the cilia beat parallel with the axis of the trachea all the way up to the very margin of the cord, where squamous epithelium is encountered. In that case, the change of direction of the flow of the mucous blanket would be due to the traction of the cilia in the posterior part of the larynx. One of the experiments I tried would seem to indicate that this is the case. Traction from the ciliated surfaces farther back is surely a factor but, if this is the main force, I do not know. It could be that the ciliary beat on the under surface of the cords is in an anteroposterior direction. If this is the case, the dynamics of the ciliary streaming in this portion of the larynx would be very different than if the ciliary beat continues to be in the axis of the trachea. Further experiments are under way to determine this point. In none of the specimens was there any streaming across the cords. I have no doubt that under certain circumstances this could happen but these experiments would seem to indicate that it is not the usual manner of drainage.

In a number of specimens, a little pool of mucus was found, before any testing was done, right at the anterior commissure and, in these, the ciliary streaming from the trachea did not quite reach the anterior portion of the cord. The source of this mucus is not known. It could have been from the ventricular sacculus or it could have been from mucus previously carried up from the trachea and left more or less stagnant at this point.

#### RELATION OF CILIARY STREAMING TO CARCINOMA

Could ciliary streaming have anything to do with carcinoma, either as to genesis or distribution? Two possibilities suggest themselves:

- 1) If carcinogenic substances (be they from cigarette tar, black-top highways, gasoline, mine dust or of whatever nature) which

have been inhaled and deposited on the mucous blanket cause cancer in the larynx,<sup>6</sup> it would likely occur first where the application of the substance to the epithelium is longest and most intimate. These experiments would indicate that that region would be at the anterior commissure and the anterior portion of the cords where the mucous blanket comes to a standstill, divides and passes backward along the two cords. From all the information I have been able to glean from laryngologists, it would seem that 80 to 85 per cent of all of the carcinomas occurring in the larynx occur on the cords and the majority of these occur on the anterior portion or right at the anterior commissure. It is possible that the characteristics of the ciliary streaming in this area might account for this distribution of carcinoma. On the other hand, one could not so conclude on present evidence. There are other factors also to be considered, such as the impact of air on the upper surface of the cords, air eddies, as well as a comparatively large amount of mucus which is discharged from the ventricle over the anterior portion of the cords.

2) Another relation of ciliary streaming to the distribution of carcinoma might lie in the field of transplant. Ormerod<sup>7</sup> and Bryan<sup>8</sup> have both pointed out that carcinoma cells are easily knocked loose from the surface of carcinoma in the larynx. These undoubtedly spread through the larynx and trachea. What becomes of them? In the nose, desquamated epithelial cells pass with the secretion back into the pharynx and into the gastro-intestinal tract. Might not the same be true in the larynx, that carcinoma cells are carried away on the mucous blanket in the mucous stream and taken down into the stomach where they may be destroyed? Another point suggesting this is the statement by Ormerod that recurrences following laryngectomy are very apt to be found just below the tracheotomy opening. A tracheotomy opening would act as an obstruction in the ciliary stream along the anterior wall of the trachea and it could be that detached cancer cells are carried to this point and deposited in such manner that they become transplanted and grow.

#### CONCLUSIONS

The distribution of squamous epithelium within the larynx as found in the specimens studied corresponds closely to that which is commonly accepted, with the exception that above the cords it is apt, in certain areas, to occur in islands and peninsulas surrounded by ciliated columnar epithelium.

The coryna divides the ciliary streams from the two main bronchi in such a way that there is a special concentration of flow on the anterior and posterior walls of the trachea. Both of these stream directly upward to the larynx with some tendency to spread. The posterior stream passes directly between the arytenoids to the rim of the larynx. The anterior stream passes up to the cords where the squamous epithelium acts like a barrier and the stream is divided in the midline in two parts, one passing backward under each cord and thence over the arytenoids to the interarytenoid space.

Possibilities are suggested for the relation of ciliary streaming to carcinogenesis involving the cords and to postoperative implantation in the trachea.

#### ST. LUKE'S HOSPITAL RESEARCH LABORATORY

I am indebted to the Elliot Packing Company of Duluth for providing the material used in these experiments.

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#### DISCUSSION

DR. G. EDWARD TREMBLE: It is indeed a great pleasure and privilege to discuss this paper of Dr. Hilding. I only received it a few days ago so I did not have time to digest the whole of it.

With his usual modesty, Dr. Hilding has described some careful observations he has made in the laryngeal mucosa in man and ciliary streaming through the larynges of freshly killed calves.

Most standard textbooks state that the mucous membrane of the larynx in humans varies in relation to the functions performed by its different parts.

The true vocal cords, which are subjected to considerable wear and tear, are covered by stratified squamous epithelium. Below the vocal cords the epithelium is ciliated and columnar in character with numerous goblet cells and this of course extends throughout the whole bronchial tree. Most of the lining above the vocal cords is high columnar epithelium covered with cilia, although patches of squamous epithelium are present over the ary-epiglottic folds and the arytenoids.

Here again one feels that where there is friction such as occurs when food passes into the esophagus the mucosa reverts to pavement epithelium without cilia.

One important point to my mind, which should be mentioned, is that the interarytenoid area contains both types of epithelium. When the arytenoids come together on phonation, this loose tissue folds upon itself like an accordion forming ridges and valleys as the space is closed. The ridges on the surface according to Dr. Hilding showed squamous epithelium, again due to wear and tear while the protected valleys or crypts showed ciliated columnar cells.

This, of course, is very similar to the nose where ridges and spurs on the anterior part of the septum, which are subjected to blasts of inspired air, often have squamous epithelium on the upper exposed surface, while the underneath protected areas of septal ridges show ciliated columnar cells.

Another point worth mentioning is the drainage upward from below in the experimental animals.

At the carina the streams from the medial halves of both main bronchi divide and pass anteriorly and posteriorly, making two main streams in the trachea, one on the anterior and one on the posterior surface. The streams spread out slightly before reaching the larynx but in general they appear to run in parallel lines until they approach the cords. The stream on the posterior tracheal wall tends to go directly up into the interarytenoid space while the stream on the anterior wall curves posteriorly just below the cords and also reaches the interarytenoid area. Mucus remains here until it is either coughed up or swallowed.

The convergence of these streams posteriorly in humans brings all infected material from the lungs to this loose mucous membrane and no doubt is the reason why tubercular infections are so prevalent in the posterior half of the larynx when the sputum is loaded with tubercle bacilli.

In a drawing such as this (mirror view of the larynx), one cannot help but think that the delay of infected sputum in the interarytenoid area is the reason why over 70 per cent of the cases of laryngeal tuberculosis are found in this location.

Finally, I would like to mention a clinical observation. I have been experimenting for the last year and a half with a harmless powder called dicalcium phosphate. It is a dusky pink powder and quite innocuous. If it is placed in various areas, as soon as it comes in contact with moisture it turns a brilliant orange color, so one can follow the streaming very easily. I would like to mention a clinical observation: innocuous powder placed in the trachea does not appear to move in such parallel lines as Dr. Hilding showed in his picture of the extirpated calf larynx. In the cases



I have seen the ciliary streams in humans seem to take a wavy or slightly spiral course instead of going straight up in parallel lines as he mentioned. I think it is due to the elongation of the trachea and the contractions with respiration and on coughing. This can be appreciated when one sees Dr. Holinger's pictures of the trachea when subjected to an asthmatic attack. There is a marked narrowing of the trachea from above and below. Undoubtedly this occurs to a less extent in normal breathing. If the lines went straight up the contraction and elongation of the trachea would tend to break the streams forming gaps, one might say in the normal ciliary streaming. This does not seem to occur with the little wavy appearance noted. This allows for a little stretch and contraction without any break in the streams.

It might also be mentioned that streaming in the trachea seems to be much slower than that seen in the active posterior two-thirds of the nose. Unfortunately time does not permit discussing all the points in this paper.

I would like to thank Dr. Hilding very much on his excellent presentation.

DR. ARTHUR W. PROETZ: My interest in Dr. Hilding's work and my respect for his scientific ability and honesty grows with the years. I don't know of anybody who says more things that I wish I had said.

For some reason those of us who have been interested in cilia have taken up the study from the nose or from the trachea and doing very little about the larynx. Now we are hearing about the place which really counts the most: the place where the lesions appear.

One of the most interesting points which Dr. Hilding made is that the material accumulates in the center below the cord, stays there and later spreads to the sides in just the places where we can't see subglottic lesions on routine examination. The concentration of the matter there seems to me very important.

One must keep in mind that the specimens which Dr. Hilding tacked down on his board did not cough, so we don't know how long accumulations lie there in vivo but we do know that lesions form there, and I would like to see that portion of his investigation carried further. The anchors of tough mucus coming out of the openings of glands and lying there to hold the mucus blanket while the cilia beat futilely, I think are very important. It emphasizes what I said the other day about great changes in viscosity of mucus accompanying small changes in humidity. It also calls to mind what Dr. Hilger said about the importance of thyroid activity. I believe that those anchors may be controlled to some extent by supplying thyroid extract when the activity of the thyroid gland (which in turn controls the activity of the tracheal glands) is deficient.

I am wondering what happened to the spiral tracheal ciliary pathways described by Barclay and Franklin of London in 1937. No lesser authority than Hilding himself found out a few years later that a similar spiral streaming took place in the interior of the sphenoid sinuses and I remember some very beautiful photographs he made showing it. I should like to ask Dr. Hilding, whether anywhere in his tracheas he found an indication of spiral streaming because from a philogenetic standpoint it is important.

DR. THOMAS C. GALLOWAY: How persistent and fixed is this pattern of ciliated and squamous epithelium, and how much does metaplasia due to trauma, chemical irritation, etc., change the pattern and possible indications?

DR. ANDERSON C. HILDING: It is rather striking that Dr. Tremble points out that 70 per cent of the tuberculous lesions occur in the posterior of the larynx. It

was pointed out carcinoma occurs in the anterior. It just shows how careful one must be in drawing conclusions before all the facts are available. Probably most of the factors in this instance are unknown. My tests were made on dead tracheas and dead animals and it is quite likely that as the accordion effect of the trachea in the living moves the trachea up and down these straight lines would be disarranged. As to spiral, I have seen no sign of spiral in these animals. In the sinuses there is a very definite spiral with an increase in the rate of flow as the ostium is approached. In one human trachea, which I obtained a couple of months ago, there was a very definite spiral. I took this to be artifact but may have been mistaken. It described almost a full turn in coming up the length of the trachea.

As to Dr. Galloway's question, how fixed is the pattern, I don't know. I can only answer from a few little pieces of information in experimental animals long ago when I stitched up one nostril and made an animal breathe twice as much on the open side. The squamous-like epithelium seemed to extend backward and, as Dr. Tremble has pointed out, the anterior surfaces of polypi and spurs in the nose are very apt to be covered with squamous epithelium. One would suppose that the pattern changes according to the circumstances of exposure to air flow. This might explain why there was more squamous epithelium in the 81 year old man than in the 61.

One would suspect that the squamous epithelium increases somewhat throughout life.

# ELECTRICAL MANIFESTATIONS OF RECURRENT NERVE FUNCTION

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The function of the various fiber groups in the recurrent laryngeal nerve has long been a subject of dispute. It is frequently maintained that the nerve carries efferent or motor fibers only. The question of the existence of afferent (sensory) fibers has been raised by Kuttner and Meyer<sup>20-21</sup> and, on the basis of clinical observations, a negative answer given. Broeckaert<sup>4</sup> considers that in man the recurrent trunk is a mixed nerve carrying motor fibers for the larynx and motor and sensory fibers for the esophagus and trachea. Thus from his point of view the human nerve resembles that of the rabbit rather than that of the dog. The importance of species differences cannot be too much emphasized. The earliest experimental work with which we are familiar which bears directly on our problem is that of Sherrington<sup>38</sup> dealing with the cat and monkey. Some (the first seven) of his conclusions are given below.

1. "In a muscular nerve trunk from one third to one half of the myelinate fibres are from cells of the spinal root ganglion.
2. "The fibres range in size (in fresh preparations) from  $1.5\ \mu$  to  $20\ \mu$  in diameter.
3. "The largest of them are not the largest fibres in the muscular nerve trunk; the largest in the nerve trunk come from the ventral (motor) spinal root.
4. "The largest root-ganglion fibres in the muscular nerve are larger than any fibres in the cutaneous nerves of the limb, and than in any articular nerves examined.

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5. "The smallest myelinate fibres in the muscular nerve are for the most part, perhaps entirely, root-ganglion fibres.

6. "Macroscopic nerve-trunks are as regards their myelinate fibres in no case purely motor, all are sensorimotor or purely sensory. Such nerves as phrenic, hypoglossal, recurrent laryngeal, posterior interosseous contain abundance of fibres from sensory ganglia.

7. "For the root-ganglion fibres in muscles a special end organ exists, this is the so-called 'muscle-spindle'." (Kuhne)  
In this same article there is reference to an amyelous fetus which is apropos:

"It is sufficient to make the general statement that no muscular nerve in the limbs, trunk, or neck was found wanting, although there existed not a single motor (ventral) spinal root in any segment. [Brain and spinal cord are completely wanting.] In view of present knowledge of the growth of the peripheral nervous system this must mean that all and each of the muscles of the limbs, trunk and neck receive abundance of myelinate fibres from the sensory root-ganglia."

Schultz and Dorendorf<sup>37</sup> show a tabulation of their review of the experimental work to that time which had been done on the rabbit, dog, cat and monkey in addition to their own work. The list shows 17 No's and 14 Yes's and one qualified answer to the question, "Does the recurrent contain afferent fibers?" They obtained evidence of afferent fibers in the rabbit and the cat and of no afferent fibers in the monkey. The afferent fibers in the peripheral recurrent of the dog and goat are considered to run through the anastomotic branch from the superior nerve. Boenninghaus<sup>2</sup> considers that there are afferent fibers in the human recurrent. Murtagh<sup>27</sup> reported no recorded responses when the central end of the cut recurrent nerve was stimulated (goat) by faradic current (induction coil) and considered that it appears that there are no afferent fibers in the recurrent. Later, Murtagh and Campbell<sup>28</sup> noted that with high intensity stimulation of the recurrent (goat) in its course respiration was stopped. It was considered that this might be due to spread or escape of stimulus to the vagus itself. In some experiments on laryngeal spasm Murtagh and Campbell<sup>30</sup> noted that section of the left recurrent nerve (goat) produced a transient inhibition of the right cricothyroid muscle. An explanation was not attempted, but it is not unreasonable to consider that the inhibition was central and due to



information carried by afferent fibers in the left recurrent which were stimulated by the cut.

#### ANATOMY

Chase and Ransom<sup>5</sup> investigated the structure (roots, trunk and branches) of the vagus in the dog. They consider that the myelinated fibers of the recurrent fall into two parts: one of large fibers, the other of medium and small fibers. Most of the smaller fibers are distributed in the cardiac, esophageal and tracheal branches. The nerve as it enters the larynx is made up entirely of larger fibers. This should emphasize that the recurrent in the neck is not strictly a laryngeal nerve. It becomes the "recurrent laryngeal" in the neighborhood of the larynx only. They also recalled to us that there are two root ganglia in the vagus, the jugular and the nodosum. This is also well brought out by Lemere<sup>23,24</sup> in his description of the innervation of the larynx in the dog. Lemere considers that the motor components of the laryngeal nerves are derived from the bulbar rootlets of the accessory (XI cranial). He has experimental evidence of afferent fibers though he is uncertain of the location of the cells of origin of these nerves. He calls attention to the anastomosis between the superior and inferior laryngeal nerves. Howell and Huber<sup>17</sup> report that in the dog the communicating branch probably contains only sensory fibers distributed mainly to the trachea and esophagus by way of the recurrent trunk. They also state that in their experiments stimulation of the central end of the cut recurrent never gave any effect on respiration or blood pressure. The cat was considered to resemble the dog. Rabbits did not.

Ransom, Foley and Alpert<sup>35</sup> report on the structure of the vagus nerve in the cat. DuBois and Foley<sup>6</sup> have made a detailed study of the vagus and accessory in the cat. They report that large medullated fibers degenerate after section of the bulbar accessory roots and that the evidence is that the cells of origin of many of the small fibers are in the nodose ganglion. Many of these small fibers go to the trachea and esophagus. They also note an anastomosis between the superior and inferior laryngeal nerves.

Brocklehurst and Edgeworth<sup>3</sup> considered that the recurrent nerve is made up of two components, laryngeal and sensory. They examined the fiber components in the macaque before and after section of the vagi at levels central to the jugular or to the nodose ganglion. Following section at either level most of the laryngeal component de-

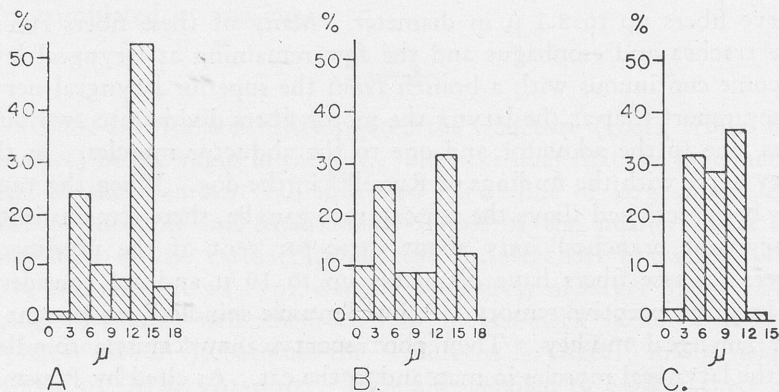
generates. The sensory component makes up about one third of the nerve fibers up to  $8.5\ \mu$  in diameter. Many of these fibers run to the trachea and esophagus and the few remaining at laryngeal level become continuous with a branch from the superior laryngeal nerve. They report that at the larynx the motor fibers divide into two bundles, one to the adductor and one to the abductor muscles. In this they agree with the findings of Russell<sup>36</sup> in the dog. When the vagus has been sectioned above the sensory root ganglia, there remains in the "laryngeal branches" only about three per cent of the myelinated fibers. These fibers have diameters up to  $10\ \mu$  and are considered to be proprioceptive sensory. A few "muscle spindles" are present in the laryngeal muscles. They also report scanty "muscle spindles" in the laryngeal muscles in man and in the cat. As cited by Pressman and Kelemen,<sup>34</sup> Rethi has shown that in the dog the efferent laryngeal fibers of the recurrent have their exit from the medulla via the bulbar rootlets of the accessory XI; and that the efferent fibers of the superior laryngeal nerve come from the vagal, X, rootlets.

Sunderland and Swaney<sup>39</sup> have carefully dissected the recurrent nerve in 65 human subjects and have found considerable variation in the peripheral arrangement of the branches and have also shown that if the various bundles are followed centrally they undergo considerable rearrangement and, in the human at least, do not follow the simple course that Russell<sup>36</sup> postulated in the dog.

Feindel, Henshaw and Weddell<sup>11</sup> raise the question of a dual innervation of the cricothyroid muscle and consider that each nerve (superior and recurrent) "gives rise to terminals which are distributed throughout the muscle rather than to any particular zone of it."

#### FIBER DISTRIBUTION

Eccles and Sherrington<sup>7</sup> report on the fiber spectra of "deafferented" motor nerves, of "demotored" motor nerves, and of sensory nerves. The results of their observations are perhaps most readily seen by looking at Figure 1, which has been redrawn from their data. They show that the efferent fiber spectrum of many skeletal muscles shows two maxima, one at about  $6\ \mu$  and the other in the neighborhood of  $15\ \mu$ . The afferent fibers from the muscles show no prominent maximum. (This is true as they plotted it. When the abscissa is rearranged to agree with our plots in Figures 3 and 4, this no longer holds.) Afferent fibers in sensory nerves may show a single maximum at about  $10\ \mu$ . O'Leary, Heinbecker and Bishop<sup>32</sup> have



## LEGEND:

Redrawn from Eccles and Sherrington (1930)

A. Efferent fibers only, ("Deafferented")

B. Afferent fibers only, ("Demotored")

C. Afferent fibers only, (Sensory nerve)

Fig. 1.—Fiber distributions in peripheral nerves. Redrawn from Eccles and Sherrington (7).

made an analysis of the function of a nerve to a muscle (cat gastrocnemius) with the intact nerve, the nerve after section of the dorsal roots, the nerve after section of the ventral roots, and after sympathectomy. Their conclusions are that about equal numbers of afferent and efferent fibers are present in both large and small fiber groups; that the large fiber group (efferent) is responsible for muscle contraction and that the small fibers innervate "muscle spindles"; that stimulation of large afferent fibers results in a release of tonus (if present).

A more recent work, Fernand and Young,<sup>12</sup> covers a much wider range of muscles. They classify the fiber spectra of muscle nerves (rabbit) as bimodal (two peaks) and as unimodal (single peak). The bimodal nerves contain many large fibers (14 to 20+  $\mu$ ), a few fibers in the 10 to 12  $\mu$  range, and a group of smaller fibers with a mode at 6 to 8  $\mu$ . This distribution is found mainly in the muscles of the limbs. Unimodal muscle nerves contain no large fibers. Among others, these include the phrenic and the recurrent laryngeal and the nerves to the thyrohyoid and the sternothyroid. They consider that the unimodal nerves contain a very few proprioceptive fibers. They

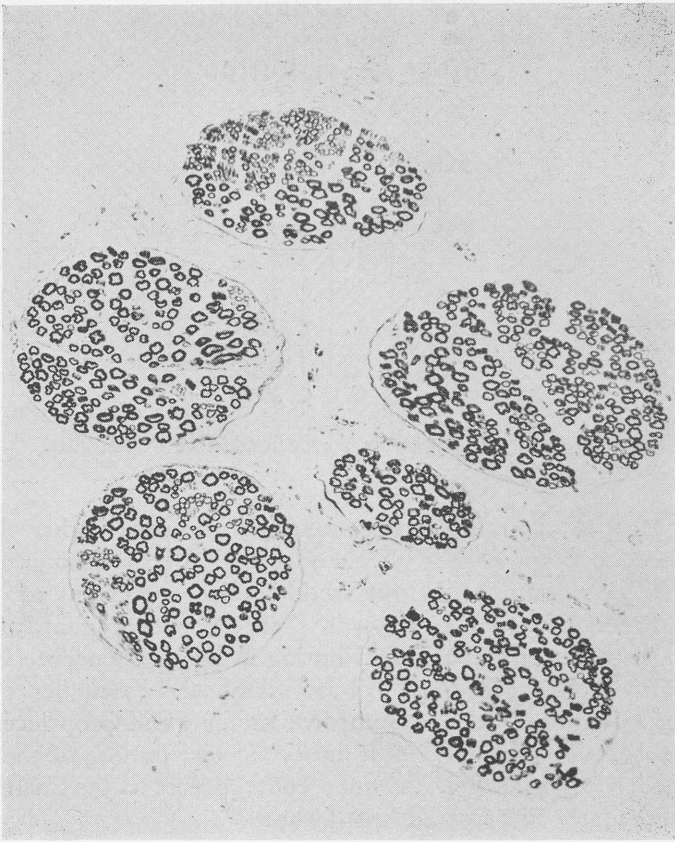


Fig. 2.—An osmic acid preparation of the recurrent nerve of the goat.

state, "We have definite evidence for the absence of afferent fibers from one nerve of this sort, namely, the part of the recurrent laryngeal that innervates the muscles of the larynx. Evans has found that following section of the vagus central to its ganglia all the fibers of this nerve degenerate." We have to date been unable to locate Evans<sup>10</sup> after a search of the literature.

Murtagh and Campbell<sup>29</sup> report the spectra found in the human, the goat and the cat recurrent laryngeal nerves. We now consider that perhaps these should have been reported as of the recurrent



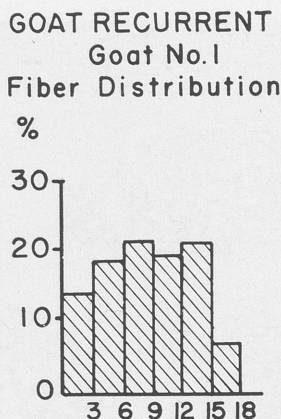


Fig. 3.—Fiber distribution in a recurrent nerve of the goat.

nerve. Most of the samples were taken in the neck rather than in the immediate neighborhood of the larynx. All the histograms are unimodal. An osmic acid cross section of the recurrent of a goat containing six fasciculi is shown in Figure 2. Histograms of a six bundle nerve (goat) give the distribution in the entire nerve (Fig. 3) and in each bundle (Fig. 4). All are unimodal. Fasciculi 5 and 6 may contain most of the presumptive motor (and proprioceptive) fibers. Fasciculi 4 resembles most nearly the distribution of the entire nerve (Fig. 4). Fasciculi 1, 2 and 3 contain most of the small fibers. (Cf. particularly Sunderland and Swaney<sup>40</sup>).

The recurrent nerve in the goat contains from four to sixteen bundles. This particular sample containing only six was chosen for convenience.

#### ACTION POTENTIALS

The foundation for the observation, using the cathode ray tube, of action potentials of peripheral nerve was established by Erlanger and Gasser.<sup>8</sup> Their fundamental work and that of others in the field for the next fifteen years has been collected and published in the Johnson Lectures, Erlanger and Gasser.<sup>9</sup> The action potentials from the vagus and the sympathetic nerve trunks (cat and turtle) have been examined by Heinbecker.<sup>15</sup> Heinbecker and O'Leary<sup>16</sup> have examined the mammalian vagus (cat); have recorded action

GOAT RECURRENT (Goat#1)  
Fiber distribution by bundles

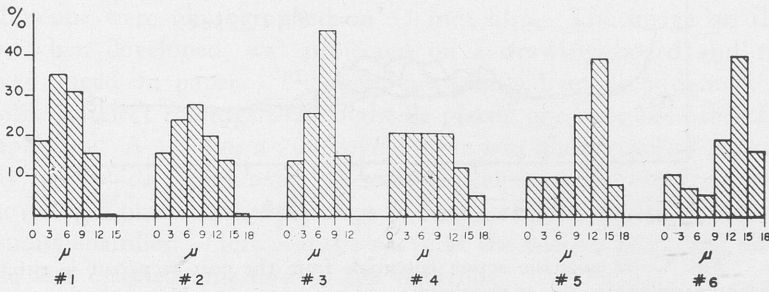


Fig. 4.—Fiber distribution in the individual bundles of the recurrent nerve shown in Figure 3.

potentials; have noted reflex responses to stimulation; and have done histological studies. They also examined in particular the recurrent nerve, which they found to be made up of two groups of medullated fibers. One group contains large fibers, 7 to 13  $\mu$ , which mainly degenerate when the vagus is sectioned above the nodose ganglion. Degeneration experiments appear to demonstrate that most of the cells of origin of the small fibers (2 to 6  $\mu$ ) are in the nodose ganglion. There were no action potentials recorded from the recurrent nerve.

O'Leary, Heinbecker and Bishop<sup>32</sup> previously cited also recorded and measured action potentials and plotted them against various quantities obtained by histological measurement and found a reasonable correspondence.

The superior laryngeal nerve has been stimulated in its course in conscious humans, and examined histologically. Following laryngectomy, the actions potentials of the excised nerve have been recorded by Ogura and Lam.<sup>31</sup> The potentials obtained were all those of the A group (Erlanger and Gasser<sup>9</sup>), probably in the subgroups alpha, and either gamma or delta. No potentials from nonmyelinated fibers were observed.

The physiology of the nerve endings (sensory) in mammalian muscle is exposed in detail by Matthews.<sup>26</sup> Katz,<sup>18-19</sup> using later and

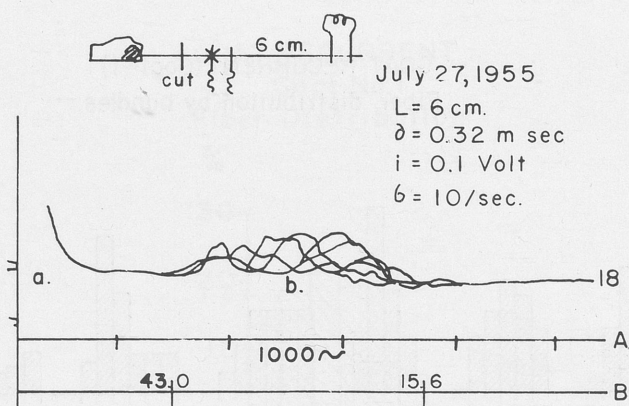


Fig. 5.—Monophasic action potentials from the goat recurrent in situ. Stimulus intensity just at threshold.

improved methods, records and discusses the action potentials from a single sensory nerve ending. The large number of small medullated fibers in the recurrent made the thesis of Leksell<sup>22</sup> particularly interesting. His records and analysis of the action potentials are especially good.

Paintal<sup>33</sup> has recorded action potentials in the cat from the central end of the vagus cut high in the neck, and from single fibers of the nerve in the same region. His interest was in the afferent fibers coming from the lungs and cardiovascular system, but the methods could be applied to the recurrent nerve. Unfortunately we were not aware of this work when we began our experiments.

Andrew,<sup>1</sup> interested in aortic baroreceptors and working with the rat, traced the afferent fibers from these receptors to the recurrent nerve, the communicating branch, and the superior laryngeal nerve. He used a single fiber technique and recorded action potentials.

#### METHOD

Goats were used throughout. The animal was given an initial dose of .045 to .060 gms per kilo of pentobarbital sodium and a continuous drip of 0.6 per cent solution of the same material to maintain the anesthesia. A tracheal cannula was inserted; the esophagus tied with a broad tape, after identifying the recurrences; and a recurrent

nerve then dissected for the greater part of its length in the neck. Stimulating electrodes, connected to a Grass square wave stimulator, and recording electrodes leading to a pre-amplifier, amplifier, and cathode ray oscilloscope were placed on the nerve. The nerve was crushed under the remote recording electrode to make the response monophasic. Repetitive stimulation was used. The traces on the oscilloscope were photographed on 35 mm film. The image on this film, when developed, was projected on a drawing board and the curves traced on paper. This gave an enlarged image and made it possible to select tracings that might be placed one over the other for comparison. A 1000 or a 2000 cycle wave was photographed periodically to provide a time axis. In some experiments the recurrent was removed and placed on stimulating and recording electrodes in a plastic moist chamber. Here also the nerve at the remote recording electrode was crushed and tied to the electrode.

In a third type of experiment the nerve and electrodes were left in place and additional electrodes were placed in the laryngeal muscles. Records were then obtained on a Grass electroencephalograph using the technique employed by Murtagh and Campbell.<sup>30</sup>

#### RESULTS

*Measurement of threshold and of conduction rate.* The possible variability of threshold for fibers of varying sizes conducting at various rates or the varying rate of conduction for the same fibers is shown in Figure 5. Actually this record was obtained when using a prolonged stimulus of a very low intensity in a very sensitive preparation with the central connections of the nerve intact. It should be noted, however, that the action potentials are synchronous with the sweep of the cathode spot (controlled by the stimulator); that no random responses are seen outside a rather limited time range; and that the nerve was cut beyond the crush at the distal electrode. It would appear that none of the recorded potentials can be of central origin or represent afferent pulses or back potentials from the muscles. Apparently the threshold for a few fibers with conduction rates of from 15 to 43 meters per second with a stimulus of 0.32 milli-seconds duration, repeated ten times per second, is in the neighborhood of 0.1 volt. These fibers are medullated, members of the group usually designated as A, and belong to the subgroups  $\alpha$  to  $\gamma$  (Fig. 6). When the intensity of the stimulation was increased the repeated action potentials all followed a single trace and exhibited none of this variability.



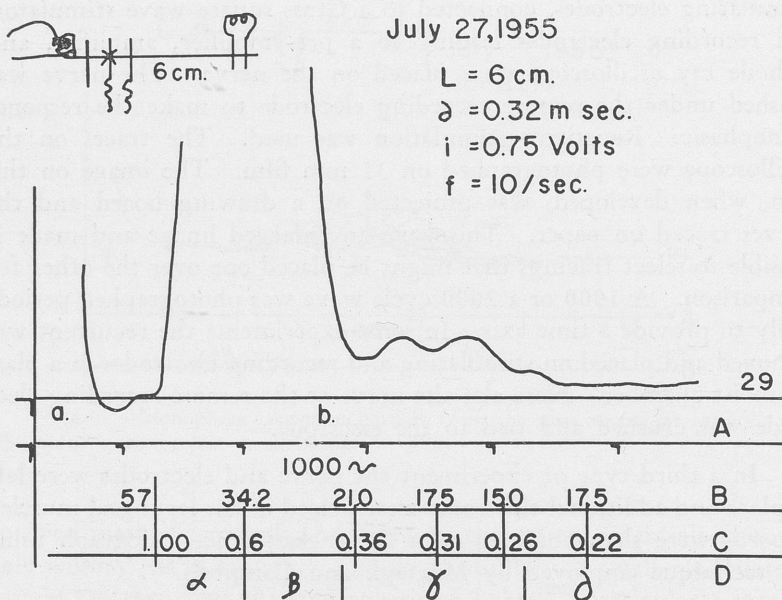


Fig. 6.—Monophasic action potentials from goat recurrent in situ. Stimulus adequate to show complexity of the potential changes.

ty. A record (Fig. 6) from the same nerve eleven observations (a few minutes) later is shown as the more usual occurrence. In Figure 5 (as in Figure 6) the upper trace is the action potential. The break in the line, a, is the stimulus artifact and here as in part b, the action potential, the spot is moving so fast that no record is obtained of the upper portions of the trace. The line A shows a series of dots spaced one-thousandth of a second apart. The figures along line B give the conduction rate in meters per second in the fibers contributing to the action potential at that point. Along C is shown the relative conduction rate of the various components. The small Greek letters in D show the subgroups to which the fibers belong, using the notation and values given by Erlanger and Gasser.<sup>8</sup> In Figure 7 are shown two action potentials from a nerve using different sized stimuli. The nerve has not been sectioned and the peak of the second component may contain a "back response" Lloyd<sup>25</sup> and Leksell.<sup>22</sup> This possibility has been indicated as perhaps accounting for the area above the dotted line. The muscles were not under observation at the time. The im-

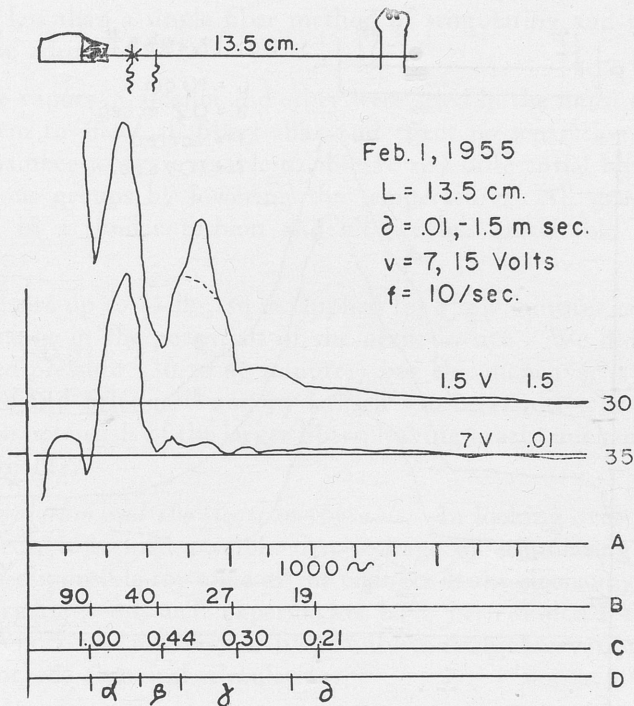


Fig. 7.—Monophasic action potentials from goat recurrent in situ. Extreme changes in duration and intensity of stimulation.

pulse may have jumped the small block presented by the crushing tie on the electrode and caused a contraction. The record is presented as further evidence for the occurrence in the recurrent of three and perhaps all of the subgroups of medullated fibers usually found in a mixed motor nerve.

Since the variety of response of the  $\alpha$  and  $\beta$  fibers is not well shown in the records presented, Figure 8 is included to show the  $\beta$  response and the spread of the  $\alpha$  pattern as the intensity is increased, using a stimulus of moderate length. These records were obtained from the peripheral end of an isolated nerve mounted in a moist chamber at a temperature of  $16.5^{\circ}\text{C}$ . This lowered temperature slows things down so that differences are exaggerated. Here it should be noted that some of the faster (larger) fibers require an intensity greater than that needed for their slightly slower (smaller) neighbors.

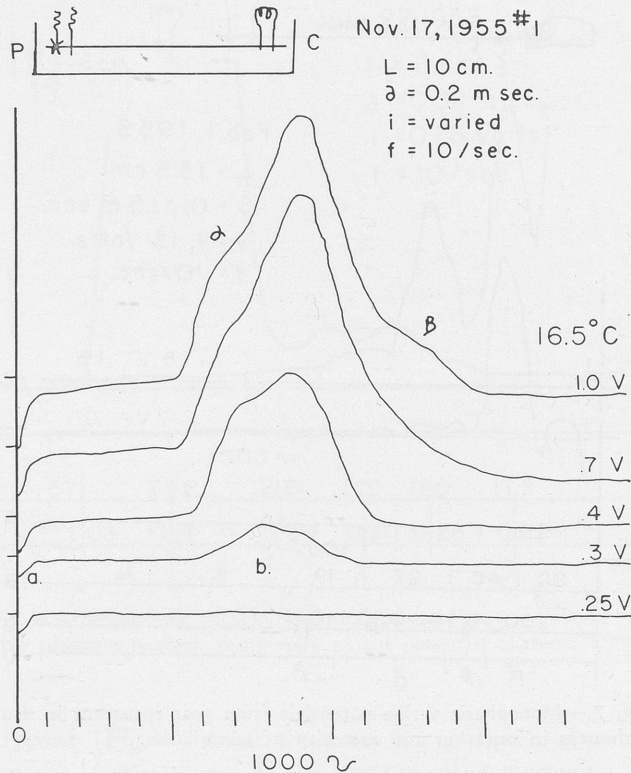


Fig. 8.—Monophasic action potentials of isolated goat recurrent. Constant duration of stimulus. Intensity varied from sub-threshold to near maximum for fastest fibers.

A tabulation of the threshold voltages at various durations gives data which when plotted does little other than emphasize the scatter. The fastest ( $\alpha$ ) fibers have thresholds at 0.01 msec duration of from less than 1.0 to 4.0 volts and conduction velocities of from 45 to 90 M/sec. Slower ( $\beta$ ) fibers at the same duration are excited by voltages of 2.0 to 4.0. The conduction velocity is somewhat difficult to determine but varies from 20 to 30 M/sec. The gamma ( $\gamma$ ) fibers show less variation in threshold, 7.5 volts at 0.01 msec, and exhibit velocities of from 11 to 30 M/sec. These variations, although all were measured with the nerve in place (after dissection), are probably due to the relative position of the particular fibers with relation to the stimu-

lating electrodes and to variations in nerve temperature. Probably nothing less than a single fiber method of stimulating and recording would be adequate.

The vapors of alcohol and ether were tried in the moist chamber. They seem to affect all fibers alike and effect no separation. In the moist chamber we were unable to observe any differential blocking of the various groups by lowering the temperature. All groups seem to react in a similar fashion and all become inexcitable at about  $10.5^{\circ}\text{C}$ .

Pressure up to 25 lbs./sq.in. applied for a few minutes cause little or no change in the potentials of the nerve in situ. We did not try prolonged pressure (30 to 60 minutes) but this maneuver is reported by Gasser and Erlanger<sup>13</sup> and by Leksell<sup>22</sup> to be effective in blocking the action potentials of the larger fibers, leaving nearly unchanged that of the smaller.

*Stimulation and the electromyograph.* In looking over many of the records, it seemed possible that leakage of stimulating current might be responsible for some of the changes in the electromyographs. All observations on such experiments have been omitted from the present report and reference is made only to such observations which do not include electrical stimulation.

In a previous experiment cited earlier (Murtagh and Campbell,<sup>30</sup> Figure 5), we obtained a record which seemed to show that there must be some afferent fibers in the recurrent. Electromyograms were recorded from the cricothyroid and the posticus on each side. The right recurrent nerve had been blocked with alcohol. The electromyogram from the right posticus was quiet. The other three muscles were showing a moderate, continuous background activity which, after an initial flurry on the left only, stopped when the left recurrent was cut. This inhibition lasted about a second after the section. No changes were recorded in respiration.

Since that time a number of electromyographic records have been secured following section or crush of the recurrent nerve.

All of them show increased activity of the cricothyroid, posticus and vocal cord on the same side during the cut. Usually this activity is very brief. One more prolonged than most is shown in Figure 9. Changes in ventilation and heart rate are usually slight. Occasionally there is apnea lasting for a few seconds. During the more recent



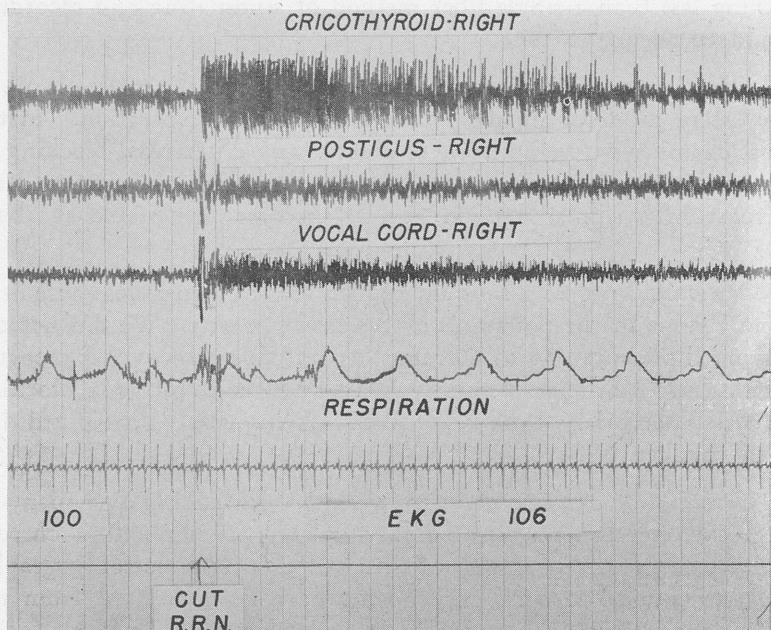


Fig. 9.—Simultaneous electromyograms from the musculature of the right larynx along with a record of respiration and an electrocardiogram. The right recurrent nerve was cut at the signal.

experiments we have recorded the electromyograms from one side only. This problem requires further work.

#### COMMENT

On a purely rational basis there must be some proprioceptive supply from the musculature of the larynx. The reasoning of Sherrington<sup>38</sup> calls for both afferent and efferent fibers in a motor nerve. The weight of the physiological and histological evidence presented is in favor of such a view. It is difficult to imagine a group of muscles which give such clear evidence of nice reciprocal inhibition as that shown in the electromyograms of Figure 10, operating without proprioceptive input. To be sure, reflexes other than simple proprioceptive are involved. The pulmonary stretch receptors (Green and Neal<sup>14</sup>) and perhaps also the pressure receptors from the great vessels (Andrew<sup>1</sup>) certainly act on the medullary centers to alter ventilation.

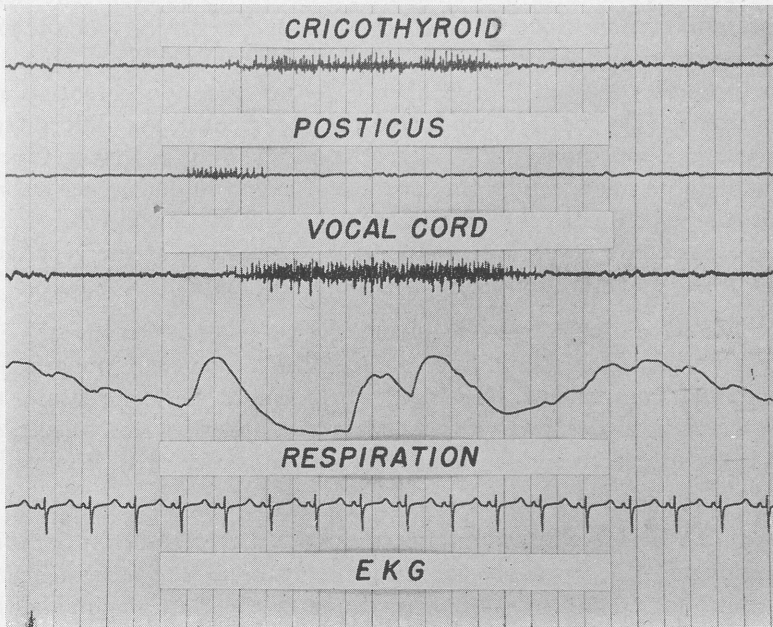


Fig. 10.—Reciprocal inhibition as shown in the electromyograph from the muscles of the right larynx. Air movement in the tracheal cannula and the electrocardiogram are shown below. Respiration was depressed by thioamylal.

The glottic muscles then take their proper part in the coordinated respiratory cycle. The nicety of performance must be due to information from the individual muscles.

The nature of the receptors within the muscle fibers has not been fully established. "Muscle spindles" have been reported in the macaque, man and the cat.<sup>3</sup> We have made preliminary examinations using a methylene blue technique for spindles. They were not found in the laryngeal muscles of the goat. Neither did we find any in muscles admitted by all to contain spindles. So far we have then not been technically adequate. We had hoped to show these (spindles) and thus make rational the large supply of small medullated fibers found in the recurrent as gamma efferents to the spindles reported by Leksell<sup>22</sup> and others.

In recording action potentials (as in the histological work) we have paid attention to medullated fibers only. The recurrent nerve appears to contain fibers in all of the groups ( $\alpha$ ,  $\beta$ ,  $\gamma$ ,  $\delta$ ) of the rapidly conducting fibers classed A. No attempt was made to secure at this time records from B or C fibers. As far as action potentials and conduction velocities are concerned the recurrent nerve differs from the mixed nerve supplying any skeletal muscle only in that it appears to contain no fibers conducting at rates of over 100 meters per second. This might be due to the experimental procedure (anesthesia, cooling of the dissected nerve), but the nerve contains none of the very large (18 to 20  $\mu$ ) fibers found in many "motor nerves."

We have always recorded from the peripheral (laryngeal) end of the nerve. This should have reduced the influence of tracheal and esophageal components. Further work should be done using the peripheral branches just as they enter the muscle. Microelectrodes have been prepared and it is planned to try single or small group fiber recording.

So far we have talked only of the A  $\alpha$  (motor and proprioceptor fibers) and A  $\gamma$  (possible gamma efferents). There is evidence of other groups ( $\beta$  and  $\delta$ ). These might carry other sensory modalities, pain, for instance. These fibers may also be involved in some of the reflex responses noted when cutting or crushing the recurrent.

The response of the ipsilateral cricothyroid to stimulation of the recurrent appears to confirm the thesis of Feindel et al.<sup>11</sup> that this muscle has a double nerve supply.

#### SUMMARY

All the experimental work was done on the anesthetized goat.

The nice reciprocal inhibition shown by antagonistic laryngeal muscles argues for a proprioceptive supply from these muscles.

The action potentials and the conduction velocities observed indicate that the recurrent nerve in its peripheral (laryngeal) portion resembles the mixed motor nerves of other skeletal muscle.

The cricothyroid muscle may have a double (superior and recurrent) nerve supply.



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## DISCUSSION

DR. LOUIS H. CLERF: Mr. President, Fellows, I wish to compliment Drs. Campbell and Murtagh on this interesting study. I question my competence to discuss it intelligently. If we accept the work of previous investigators we must assume that not only the superior but the recurrent laryngeal nerve too is a mixed nerve carrying both efferent and afferent fibers.

The question always arises whether one can translate one's findings in studies of experimental animals to man. A case in point is the finding by one investigator of motor fibers distributed to the arytenoid muscle from the superior laryngeal nerve in man whereas other investigators who, finding no fibers in the dog, applied these

findings to man. As our knowledge of the various species of animals now studied increases we may find that assumed differences will diminish.

I am interested in the effects of pressure applied briefly to the recurrent nerve without interference with its conductivity. I suspect that remote effects will occur, however, as shown in several patients operated for pulsion diverticulum who developed unilateral paralysis apparently due to pressure produced by a metallic retractor applied for 10 or 15 minutes. I suspect that the larger fibers bore the brunt of this trauma.

I am in agreement that one cannot rule out reflexes as a possible explanation of some of the results secured in this and similar studies. They have shown that the amount of stimulation used greatly influences reactions and have taken this into account in the final interpretation of their results.

While the question of some proprioceptive elements in the muscles of the larynx has not been proven, when one recalls the remarkable reciprocal relationship of the external ocular musculature and its similarity to the two halves of the larynx, it would appear that further study of end organs in the laryngeal muscles is necessary.

DR. LEWIS F. MORRISON: It is a privilege to be permitted to comment on this beautiful piece of work. These authors have been producing work of equal caliber on this and closely allied subjects for some period of time. It is possible that my evaluation is slightly biased owing to the fact that the subject is one in which I am vastly and vitally interested.

I do want to ask a question and also make a suggestion. The question is: is there a rough usable formula for determining the amount of electrical impulse in a duration of time of the stimulation whereby one may anticipate optimal responses without having to go through a series of stimulations on a hit-and-miss basis which carry with it the potentialities for erroneous results due to nerve damage? The results of observation of the use, misuse, and abuse of the average nerve stimulator permits the deduction that a better knowledge of the potentialities of the apparatus would do much to bring about some conformity of results. A stimulation sufficiently strong to cause the entire biologic unit to contract and twitch does not appeal to me as being the proper threshold to attempt to determine muscle action in a rather confined area such as is the larynx.

The suggestion has to do with encouraging those who have the opportunity to perform surgical procedures in, on, and about the larynx and this general region of the neck to devote a little more energy and attention to making and noting the results of certain *vivo in situ* physiological responses. In a number of instances wherein the position of a malignancy demands extirpation of the larynx, the extent of the neoplastic invasion may be such that the uninvolved side may well be considered as being normal for the determination of nerve distribution and muscle response. Limited yet accurate test could be conducted without imposing in any way on the physiologic reserve of the patient.

It is also suggested that those who carry out this type of work might lend a small bit of their time, and less than that in the way of effort, in adding to the present fund of knowledge in regard to division of the recurrent laryngeal nerves in the extralaryngeal spaces.

As a parting suggestion, we might be of some assistance to Dr. Hilding if, while we were doing nothing else, we would instill a bit of india ink or dust in some

suitable powder so that further information relative to the cilia pathways might be mapped out in the larynx containing pathology as well as in the normal larynx and trachea.

DR. C. J. CAMPBELL: Thank you very much, Dr. Clerf and Dr. Morrison.

In the matter of actually stimulating the recurrent in situ, one might very well repeat the sort of thing that was done by Ogura and Lamson on the superior laryngeal nerve. They stimulated the nerve in situ and, following laryngectomy, used the removed nerve to record action potentials. I think that it is advisable to have a square wave stimulator. We use a Grass instrument. Any square wave stimulator would do. A stimulus of perhaps a tenth of a millisecond duration and voltages up to about three volts should get most of the fibers in which one is interested. But if one is going to observe the thresholds of the individual muscles, one should be able to run the whole gamut of strength and duration. One should also keep in mind the confusion which we showed may exist at or about threshold values.

To revert to Dr. Clerf's question of pressure, we applied pressure for only a few minutes. Others have applied pressures for very much longer and have secured the blockade of the function of the larger fibers. In fact, Leksell, in his doctor's thesis in one of the Scandinavian countries, devised an instrument to do this. He finally decided that the cheapest way to block the largest fibers and retain perhaps some, not of the slowest, but of the gamma group, was just to pinch the nerve between thumb and finger for some 30 to 60 minutes. The time element is important. We missed the boat in not using longer periods. The effects that we were noticing were the immediate effects only.

In reference to a matter that Dr. Clerf brought up, we did attempt to examine muscles to see if there were spindles there. We found none but fortunately took the precaution of looking at muscles where everyone admits there are spindles. We did not find them there either. The difficulty is with our present techniques. We hope that these will be improved.

SUBMUCOSAL COMPARTMENTATION  
OF THE LARYNX

JOEL J. PRESSMAN, M.D.

Although unknown to me at the beginning of these experiments, it has since been learned that many years ago Hajek demonstrated that if a liquid dye is injected beneath the mucosa of the larynx, the spread of the dye is limited to specific areas which can be mapped. This is of clinical significance from many points of view, but it is of special importance in the light of our studies which concern the use of radioisotopes injected into the larynx. The experiments consisted of attempts to identify and map these compartments and to study the flow or transmission of liquids from one to another as a preliminary to establishing a technique for the practical use of such radioisotopes.

The first observation resulted from the injection of dye into a living human larynx through a laryngoscope prior to performing laryngectomy. The dye was injected on one side into the true and false vocal cords. The anterior commissure was, of course, intact, since no incisions had been made. The purpose was to observe the fate and route of spread of the dye in the living human larynx. The laryngectomy was intentionally given to a new resident to perform, so that the removal of the organ was an unduly prolonged procedure requiring four hours. At the end of three hours, the larynx was finally removed, and the spread of the dye through the larynx could be observed. A sharp demarcation of the hemilarynx was evident. The dye had spread over only one lateral half of the larynx, while the opposite side contained no dye. There was no spread across the midline although the lymphatic system was functioning and intact.

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We can, therefore, begin by saying that the larynx is compartmented into a right half and a left half, in so far as its submucosal structure is concerned. This has been found to hold true as a general principle for all larynges, either in living human subjects, cadavers, or experimental animals.

Externally, from a posterior view it was observed that this failure of the spread of dye to the opposite side occurred not only on the interior of the larynx but exteriorly as well, so that the posterior face of the arytenoid and adjacent tissues on the injected side were well-stained by the dye, whereas the posterior surface of the opposite arytenoid was not stained at all.

Realizing from this single observation that the problem was by no means a simple one, a technique was developed for injecting cadaver larynges since such material was more readily available. The limitations, as opposed to using living material, were, however, recognized. The first series of cadaver specimens was injected in the aryepiglottic fold above the level of the false vocal cord. By using controlled pressure, the dye was forced through the soft tissues to the border of the cartilage of the epiglottis, but not beyond it or across the midline. Its spread was downward to the level of the false vocal cord, but not below it. Apart from the division into halves, therefore, a compartment exists that extends over the supraglottic area above the level of the false vocal cord. It is of particular importance to note that when a massive injection is made on one side of the supraglottic area, the ventricle of the larynx remains clear of dye and is immune to the spread of fluids injected above it. The ventricle of the larynx is, compartment-wise, isolated from the false cord above it.

On repeating the supraglottic injection, but this time in the midline, the spread occurred to the right and left sides of the midline involving the entire epiglottis, whereas on lateral injection the cartilagenous area was uninvolved. Again there was no filling of the ventricle, so that it remained completely compartmentalized from the structures above it, even when large amounts of dye were used and the injection was made in the midline. The demonstration slide shows dye below the true cord on the opposite side as the result of an independent effort to fill the ventricle from below. This can be disregarded for the purposes of the moment.

We are vitally concerned, of course, with what happens to fluids injected in the region of the true vocal cord. When dye was injected

submucosally below the level of the true vocal cord, it also spread essentially to the midline. The slight intrusion of the dye as a tongue to the opposite side was an artefact, in that after one side filled with well-marked demarcation at the midline, efforts to increase the pressure ruptured the mucosa and a little dye penetrated to the opposite side. The lower limit of spread was at the inferior border of the cricoid cartilage without any evidence whatsoever of spread into the trachea. At the uppermost level, the dye reached the superior surface of the true vocal cord, but like the supraglottic injections did not actually enter the substance of the ventricle above.

From observations following injections both above and below the ventricle, it became obvious that both this structure and the immediately adjacent true and false cords required further detailed study, since the dye did not enter them either from above or below. In the first specimen demonstrated in the study of the region of the ventricle, the saccules were particularly prominent, filling the ventricle, and could easily be injected with dye. When injected, the saccule bulged outward with dye. None passed upward into the false cord or downward into the true cord, so that the larynx or the vestibule of the ventricle represented other compartments which submucosally are entirely independent of the adjacent structures. A trifling amount of dye, however, traversed the lymphatics across the midline, and there was a suggestion of its entrance into the other ventricle. There was, therefore, passage across the midline, not from one true or false cord to the other side, but through the lymphatics from one ventricle to the other.

Apart from the saccule, and not to be mistaken for it, is a prominent pearly-like margin, essentially the free edge of the vocal cord. It has been described and injected by Ottaviani and Sbermini, who called it a "bursa." If one takes a flexible needle on an automatically controlled injector and places it underneath the mucosa in this bursa (if one wants to call it that) a filling takes place which is sharply demarcated above and below. It does not pass across the midline and it can be filled to the point of rupturing without dye entering the saccule which is above it. The dye will spread only over the margin of the true cord, including part of the superior surface, but not into the ventricle. By utilizing the opposite side of this same larynx and placing one injection in the true cord below the margin of this "bursa" and a second injection immediately above the bursa into the saccule, the well-delineated bursal compartment

can now be demonstrated in a negative way by the filling of the tissues above and below it and the complete absence of dye in the bursa itself. Therefore, the bursa represents an additional isolated submucosal compartment along the margin of the true vocal cord.

In no instance, so far demonstrated, has the dye crossed the submucosal division at the mid-line, but since the experiments have been performed for the most part in cadavers, the spread of injected substances through the lymphatics has not been taken into consideration. Therefore, it is far more important to study dissemination of these dyes in the living.

The first of such experiments was performed in the dog. After the larynx was split through the anterior commissure, a small drop of dye was injected below the level of the true vocal cord under the mucosa. The dog was then photographed at hourly intervals.

After one hour, the margin of the vocal cord representing the "bursa" was free of dye. The dye had passed deep to the bursa, which likewise did not fill, and entered the superior level of the larynx. The free margin of vocal cord, which strangely enough is the site of so much pathology, particularly polypoid degeneration, and of many benign tumors as well as epithelioma, remained free of the dye.

After 12 days when the dog was sacrificed, one can see that, although the dye has for the most part disappeared, at no place has it crossed the midline to the opposite side of the larynx. It is true that the larynx had previously been split through the anterior commissure, but at least the posterior commissure remained intact and no cross-over took place across this site even after 12 days. Posteriorly, it can be observed that the dye has left the larynx and infiltrated the pyriform sinuses and hypopharynx on the same side as the injection.

To overcome the problem of having incised the larynx anteriorly and destroyed the continuity of tissues across the anterior commissure, studies were made by splitting the trachea transversely between the third and fourth rings, and by using a good head light the larynx was injected on one side from below, thereby leaving the anterior commissure intact.

With the anterior commissure of the larynx intact, the dye injected below the cords again spread superiorly from below to above.

The spread of the dye in the living dog after four hours again bypassed the free margin of vocal cord, thus eliminating the division of the anterior commissure as a factor in the isolation of the vocal cord margin from the remainder of the larynx.

This same experiment was also performed on a pig using the low tracheal incision without destruction of the anterior commissure and injecting the dye submucosally on one side below the level of the true vocal cord. The spread in the pig was especially interesting. The dye reached the midline, obviously not crossing it subglottically or supraglottically, but a very strange cross-over to the opposite side along the margin of the true cord was noted.

To determine what happens when dye is injected below the level of the cricoid cartilage in a living dog, the larynx and trachea were opened and a small segment of dye injected posteriorly. After five hours, the dye had spread downward into the trachea as far as the midline, but had not spread upward towards the larynx. If the injection had been made above the level of the cricoid cartilage, the dye would have spread upward, leaving the margin of the vocal cord free, and entered the ventricle. There is a vast difference in the distribution of dye when it is injected just below the level of the cricoid and when it is injected just above it. This factor may be of great clinical importance, and should be a matter for subsequent study. After a good many more hours, up to 12, there was still essentially no spread across the midline or, at the most, very little.

When it was massively injected into the aryepiglottic fold, dye spread posteriorly into the hypopharynx towards the region of the pyriform sinus and along the lateral walls of the hypopharynx. Anteriorly, it extended into the larynx along the lateral wall. Therefore, fluids injected into the region of the aryepiglottic fold have a distribution greater than that of injections at any other site.

Observations of cross sections made after the injection of dye show that submucosal injections do not cross the midline of the larynx at any level, even when made from below in an intact larynx with the continuity of the anterior commissure preserved. Also, the dye does not pass beyond the thyroid cartilage into the extra laryngeal space. At the lower most level of the cricoid cartilage, however, the dye does pass into the tissues of the neck making its way through the cricothyroid membrane via the recognized route of the lymphatic vessels. While one can observe a spread to the external surface of the



region of the larynx, even here there has been no spread across the midline.

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#### DISCUSSION

DR. PAUL H. HOLINGER: Mr. Chairman, Members of the American Laryngological Association and Guests: I have found Dr. Pressman's slides extremely interesting and also found that there are some differences one may have in interpretation. Possibly his description and discussion may show these to be differences in interpretation of his subject rather than actual differences in recognition of the manner in which the dye has progressed from one area to another.

I am glad that he changed his title from *Compartmentalization* to *Submucosal Compartments of the Larynx*; I can understand the paper a little better by the change in terminology of the title itself. By pressure injection of various areas one does inflate anatomical regions which have been described. For instance, on the posterior aspect of the epiglottis, the mucosa is tightly bound to the cartilage and actually there are small holes through the cartilage itself. I would be interested in knowing whether the transverse sections of the epiglottis show that the dye does go through the epiglottis in these areas. Probably this would have to be determined by the microscope rather than by the gross appearance because the small holes in the epiglottis are microscopic in size.

The course of the lymphatics is well demonstrated. Dr. Pressman demonstrated certain compartments by pressure injection, and showed how the dye was limited in travel to the borders of those compartments. I cannot quite go along with some of the description, and discussed with him yesterday, briefly, the fact that the dye injection is *not* limited by the midline; it appeared to me in many instances that it went beyond the midline. This may be due to lymphatic distribution or lymphatic pickup rather than a lack of limitation to the compartment itself; possibly by less pressure rather than the maximum pressure one could illustrate the compartments better than one can by maximum pressure. Certainly the course of the lymphatics was beautifully demonstrated in these photographs as well as the actual compartmentalization of the structures. The clinical interpretation of edema of the larynx

that occurs in laryngo-tracheo-bronchitis has been substantiated by this work; the subglottic edema, the edema of the false cords with the lack of swelling of the true cord edges and the supraglottic obstruction are all seen in this disease according to the compartments demonstrated this morning. This is again anatomically understandable when we go back to our dissection of the larynx and find the manner in which the mucosa on the phonating edge of the cord itself is tightly bound down, whereas there is loose areolar tissue in the immediate subglottic region. I wonder, too, in regard to the muscle compartments and whether the distribution of the injected material just follows intramuscular areolar tissue rather than submucosal spaces.

In the patients with carcinoma that Dr. Pressman showed, it seemed to me that the carcinoma extended beyond the midline in the same manner as did the dye; in the first case certainly grossly so, and in the second there appeared to be areas of seeding in the upper portion of the section on the opposite side.

The study has been of interest in that it describes not only mucosal compartments but again brought into clear demonstration the manner in which the lymphatics carry material from one area of the cord to another. The absence of lymphatics on the true cord itself, so important in the study of laryngeal carcinoma, was demonstrated as an area which did not fill with dye.

Dr. Lederer has often stressed the differentiation between intrinsic and extrinsic carcinoma based on the lymphatic drainage of the larynx, considering lesions on the cords as intrinsic when they involve areas with almost no lymphatics. Dr. Pressman has shown this absence of a space directly along the phonating edge of the cord, giving us a similar anatomical basis of limitation rather than simply trying to divide the larynx into areas or zones which have no such fundamental limitations.

So that in summarizing the discussion, as one sees the sections, I think we can argue, first, on the question of interpretation as to the extent of injection and the possibility that lighter injections will more clearly identify these spaces; and second, the interesting additional aspect, namely, the manner in which the lymphatics show a flow from one side of the larynx to the other. This was so clearly demonstrated in the pictures of lymphatic distribution of the dye that was injected. I wish to compliment Dr. Pressman on this presentation.

DR. CHEVALIER L. JACKSON: Mr. President and Gentlemen, I was one of those who asked Dr. Pressman what he meant by his title. It seemed fairly descriptive to me and while it did occur to me to try to pin him down yesterday as to what he was going to say, I refrained from doing so.

I enjoyed his presentation very much, and I would like to say that it is just another example of an anatomical concept that may have considerable clinical importance. Anatomy is as old as any part of medicine, the oldest part—it does not change—yet these new concepts come along from time to time.

For example, I have been very much interested in the concept of the bronchopulmonary segment. That has continued for the past several years to be fundamentally important in the better understanding of diagnosis and treatment of bronchopulmonary disease. In the same way, I feel that perhaps this idea that Dr. Pressman has outlined may be clinically important in more ways than we can imagine at the present moment. I believe it is a mistake to think of this concept only in relation to the cancer problem, because it certainly has other possible applications.

Not too long ago the pathologists treated a lung like a loaf of bread. On the postmortem table the lung was sliced down with no consideration of the anatomy.

I do not wish to cast any aspersions on the pathologist, but I just wish to try to point out the value of a getting together of the clinician, the pathologist and the anatomist!

You may say, in connection with the segments of the lung, that the application to cancer is not very important, because you don't do a segmental resection of a carcinoma, whereas you may do a segmental resection of tuberculosis; but even with carcinoma the segmental concept has been important because we now more often pick up the disease at the segmental stage rather than when it has taken the whole lobe or the whole lung.

So, I would like to commend Dr. Pressman for bringing us this very refreshing new slant on the anatomy of the larynx.

DR. JOEL J. PRESSMAN: I am tremendously grateful to both Dr. Holinger and Dr. Jackson for their astute discussion of this problem.

First, to refer to Dr. Jackson's very wise comment that it is a mistake to apply this to the cancer problem, no one agrees more than I for which reason I said that this was off the record. We were simply talking amongst the family and it is something for us to think about. He is certainly right and any reference to it in the serious way would be premature.

Dr. Holinger's comments concerning the spread of these dyes across the midline are very much in point. However, we know that all anatomical structures which come through the midline cross it a little. Thus there is an overlap of nerves which reach the midline of the opposite side, there is overlap of vessels for a short distance and there is in this case an overlap of the dye. The control that we used would seem to bear out the fact that the spread to the midline was a very real thing, leaving out the millimeter beyond it which was of no significance. The control was the injection of tiny amounts in the living where nature spread it rather than the force of the injector and if you saw anything in the dogs and on the pig, there was even less tendency to spread across the midline in the living. So using those controls or calling them to your attention, I would feel that we were on relatively safe ground in limiting it to the lateral distribution of spaces.

The epiglottis has its pathways in the living and injected dyes go through it to the outer world but not in the cadaver. Apparently these little foramina are too tiny for the injected dye to go through.

While this is a subject dear to my heart, I can only close by telling you at least we have learned where to inject our radio isotopes and realize a mass injection in one space won't do any good. We need to inject it in several spaces.

## CANCER OF THE LARYNX

FERNAND MONTREUIL, M.D.

MONTREAL, CANADA

Since the first laryngectomy was performed in 1873, much has been written on cancer of the larynx. It is not my intention to summarize the voluminous literature that has covered the many aspects of this subject, but only to review some of the cases treated in the Ear, Nose and Throat Service of Notre Dame Hospital in Montreal, from July 1, 1952 to December 31, 1955.

During this three and a half year period, 70 cases of laryngeal carcinoma were diagnosed. The first of these cases dates back but four years; because of the somewhat restricted period of follow-up elapsed since the termination of treatment, it is impossible at this time to give a factual evaluation of the various forms of treatment used. We all know of the good results claimed by various investigators with their different forms of treatment. I believe much can be learned by reviewing less successful cases, and for this reason it is my intention to emphasize on some of our failures.

These 70 cases of carcinoma of the larynx represent a little over 3.5 per cent of all cancers seen at the Cancer Institute, Notre Dame Hospital, during this period of time. This figure is in agreement with the incidence of between 1 and 5 per cent reported by various investigators. Sixty-one or 88 per cent were male, nine or 12 per cent were female patients. The youngest patient was a 22 year old male, and the oldest an 86 year old male who underwent laryngectomy with no ill effect. The incidence of cases in various age groups is seen in Table I. The average age of all patients was 58.27 years. In 1945, Jackson<sup>2</sup> stated that "cancer of the larynx is rare under 40 years of age and that in the experience of many investigators showed predilection for the sixth decade of life." We are seeing more and more cancers of the larynx in the younger age group. It is unfortunate that even in these young patients, the lesions are far advanced by the

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Read before the American Laryngological Association, Montreal, Canada, May 13, 14, 1956.



TABLE I  
CANCER OF THE LARYNX

(Incidence in different age groups at Notre Dame Hospital)

|             |          |
|-------------|----------|
| 20 to 39    | 5 cases  |
| 40 to 49    | 14 cases |
| 50 to 59    | 16 cases |
| 60 to 69    | 25 cases |
| 70 to 79    | 8 cases  |
| 80 and over | 2 cases  |

time diagnosis is made. The importance of an early diagnosis cannot too strongly be emphasized; it is up to us to impress the medical student and the general practitioner on the seriousness of a change in voice which tends to persist. An active program of education of the lay people by cancer societies should eventually show some results.

Numerous classifications of cancers of the larynx have been suggested since Isambert's first attempt to divide these tumors into intrinsic and extrinsic. This subject will remain most confusing until such time as an universal method of classification is adopted. We have classified our laryngeal carcinomas into intrinsic, extrinsic and extralaryngeal.

Laryngeal carcinomas are called intrinsic when the tumor is limited to the true cord only. They are extrinsic when the tumor involves structures other than the true cord but remains endolaryngeal. Lastly they are extralaryngeal when the tumor is outside the laryngeal box. Many cases of primarily intrinsic carcinomas are extrinsic by the time diagnosis is made. Ogura<sup>4</sup> has pointed out the importance and the difficulty of determining the exact site of origin of these tumors.

Our total of 70 cases were made up to 26 intrinsic, 34 extrinsic and 10 extralaryngeal carcinomas. Controversial predisposing factors such as syphilis, tuberculosis, bad oral hygiene, industrial fumes, etc. were not prominent in this group of cases. It is to be noted, however, that all these patients made use, to excess, of some form of tobacco.

All 70 cases were squamous cell carcinomas. Sarcomas, which occur more frequently in the younger age group, were not encountered. Neither did we see any intramucosal carcinoma.

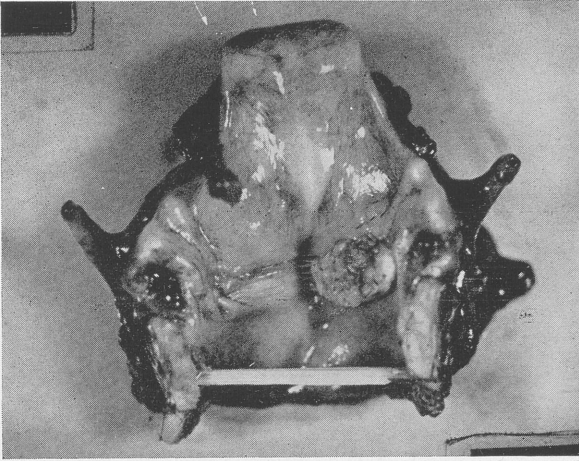


Fig. 1.—59 year old male complained of hoarseness for 1 year. Large papillomatous growth with malignant degeneration is still limited to the cord. Laryngectomy March 1954. Alive and well.

The symptoms of laryngeal carcinoma depend on the site of the lesion, and if a change in voice appears early in cordal lesions, such is not the case in extrinsic or extralaryngeal ones. Hoarseness, or a change of voice, becomes evident at the very onset of a cordal cancer. It may vary from a slightly huskiness to a near total aphonia and is persistent in nature. For a long time, it remains the one and only symptom, and the number of patients presenting this symptom who are treated with gargles, inhalations and what not, is still far too high. Symptoms such as cough, feeling of foreign body in the throat, bloody expectoration may all occur as the lesion progresses. When the tumor presents as a fungating growth and closes the lumen of the glottis, respiratory obstruction ensues. Five of the 70 patients came to the hospital because of dyspnea and required emergency tracheotomy.

Figure 1 shows an endolaryngeal tumor which probably had originated on the true cord but, by the time diagnosis was made, had become extrinsic. The passing of an endotracheal tube in such cases may easily traumatize the lesion and the risk of dislodging cancer cells and implanting them is a real one.

TABLE II  
CANCER OF THE LARYNX

(Treatment of seventy cases—July '52 to December '55  
at Notre Dame Hospital)

|  |    |
|--|----|
| Laryngectomy                             | 25 |
| Laryngo-Fissure                          | 10 |
| Combined Laryngectomy and Neck           | 4  |
| Radiotherapy                             | 20 |
| Radiotherapy and Subsequent Laryngectomy | 3  |
| Thyrotomy                                | 2  |
| Secondary Neck Dissection                | 4  |
| Refused Treatment                        | 6  |

In patients with respiratory obstruction, Hoover and King<sup>1</sup> have recommended immediate laryngectomy instead of preliminary tracheotomy. I cannot agree with this statement and do not believe that one is justified in removing a larynx without histological proof of cancer. Furthermore, the patient is not psychologically prepared to undergo a surgical procedure which leaves him with so great a handicap as the loss of his voice. Extrinsic and extralaryngeal lesions give rise to more indefinite symptoms which are late in manifesting themselves. Dysphagia, lump in the throat, hoarseness, cough, bloody sputum, hypersalivation, dyspnea, otalgia may all appear, but so gradually, that the patient pays little attention to them. Not too infrequently a patient will consult because of a slowly growing mass in the side of his neck. In this group, the initial complaint was hoarseness in 54 patients and dysphagia in 13. In three instances, the presence of a mass in the neck prompted the patient to seek medical advice. The average time elapsed between the onset of symptoms and diagnosis was ten months.

A clinical impression of carcinoma is made by indirect laryngoscopy. A positive biopsy is necessary, however, before instituting treatment, whether it be surgical or radiation.

The advantages of taking the biopsy under direct laryngoscopy need not be emphasized upon. The surgeon should do his own laryngoscopy so that he may properly visualize all laryngeal structures and

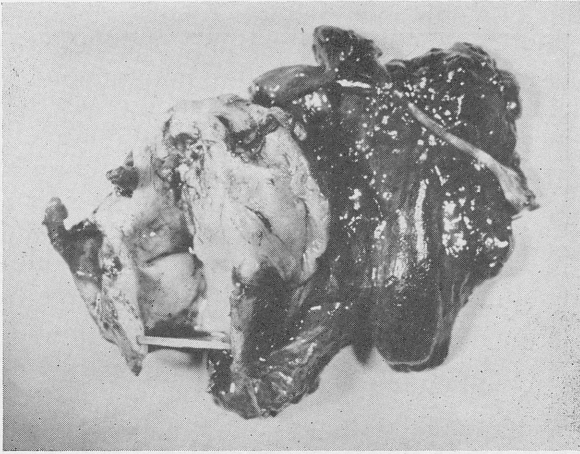


Fig. 2.—51 year old male complained of huskiness for 2 months, sore throat 3 weeks and mass in the neck 1 week. Lesion involved all of the hemilarynx. Combined neck and laryngectomy on January 1955. Alive and well.

appreciate the extent of the tumor mass. Regions which cannot be seen by mirror laryngoscopy can be properly visualized and inspected and unsuspected extensions in the ventricles or in the sub-glottis exposed.

It is beyond the scope of this paper to discuss the indications for the various forms of treatment. The treatment given our 70 cases is shown in Table II.

We believe that surgery offers the best chances of a cure, and in cases other than small cordal carcinomas, wide field laryngectomy has been considered the treatment of choice.

Four patients had a combined radical neck dissection and laryngectomy because of palpable lymph nodes (Fig. 2). One patient had a difficult postoperative course with prostration, elevation of temperature and crusting of the tracheobronchial tree which necessitated bronchoscopic aspirations on two occasions. He did not get out of bed till the fifth postoperative day. On the sixth postoperative day, his condition seemed to be picking up but, on the following morning, that is, on the seventh postoperative day, he suddenly became dyspneic and cyanotic and died within a few minutes. Death was caused by an embolus of the right pulmonary vein. The left



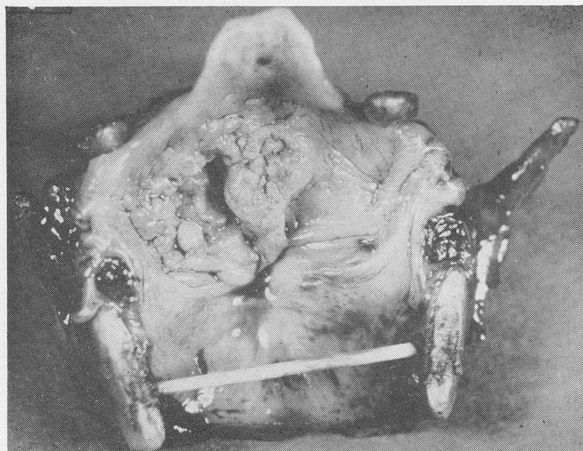


Fig. 3.—39 year old male complained of hoarseness and pain in swallowing for 4 months. Biopsy showed an undifferentiated squamous cell carcinoma with numerous mitotic figures. Patient returned 4 months after laryngectomy because of a node in his neck. Neck dissection in June 1954 and has been well since. This patient could neither read nor write and it was most interesting to see how well he rehabilitated himself after laryngectomy.

post tibial vein was completely thrombosed. Early mobilization of patients is most important to prevent venous stasis, responsible for many complications in the older patients.

Prophylactic neck dissection was not done in this group of cases. Six of the laryngectomized patients subsequently developed cervical metastases and four of them had neck dissections (Fig. 3). Of these six patients, five had presented a large extrinsic laryngeal growth and one, a very small cordal lesion which will be presented later. One of these patients developed cervical metastases, not from his laryngeal carcinoma but from a secondary, an independent lesion appearing on his hypopharynx two years after laryngectomy.

Cordal lesions have been considered as being unlikely to metastasize because of poorly developed lymphatic drainage. Extrinsic and extralaryngeal lesions, on the other hand, have been known to give metastases in a high percentage of cases. Ogura has shown that impalpable metastases were present in 31 per cent of endolaryngeal, 50 per cent of subglottic and in 57 per cent of extrinsic carcinomas.

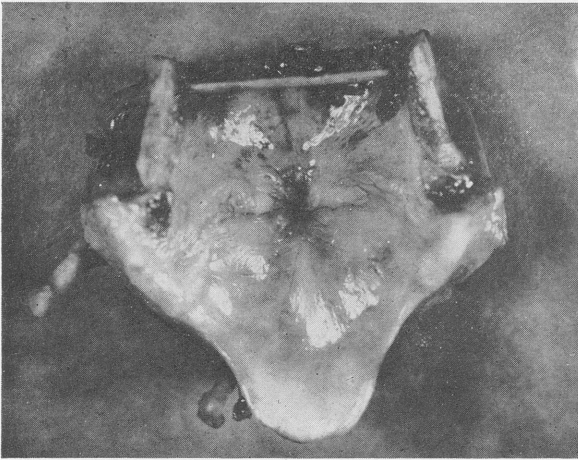


Fig. 4.—L.C., 41 year old male. Leucoplakia of both cords with microscopic area of carcinomatous changes. Most of carcinoma had been removed by biopsy. This patient developed a cervical metastasis 8 months later.

In the light of these recent reports and of the experience gained in our present series, I believe that prophylactic neck dissection is justified and should be done in cases of extrinsic and extralaryngeal carcinomas.

Cancer is most unpredictable at times as the two following cases will illustrate:

A 41 year old male had complained of hoarseness for two and a half months. Mirror examination showed leucoplakia of both cords. Biopsy reported malignant degeneration and laryngectomy was performed (Fig. 4). The examination of the surgical specimen revealed but one small microscopic area of malignancy. Following this pathological report, we considered that the patient had been treated rather radically and we expected to have him among our cured cases. Eight months later, he returned with a small ( $1\frac{1}{2}$  cm) node in the right upper jugular chain which had appeared within a period of one week. A neck dissection was done with much difficulty as the patient bled from everywhere and had abnormally large jugular veins. In the recovery room, he started bleeding in his wound; when brought back to the operating room it was found that the ligature

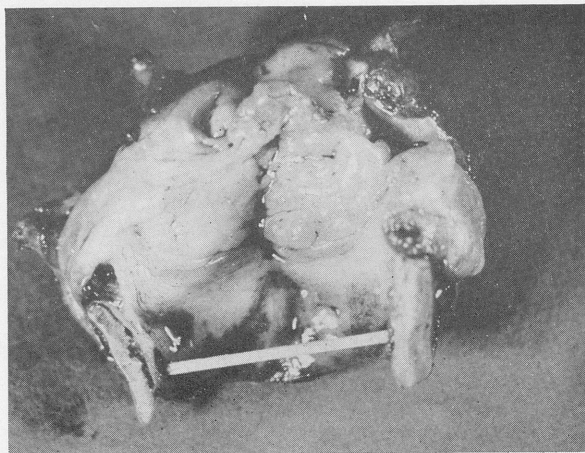


Fig. 5.—Surgical specimen of E.C. treated by radiotherapy and then laryngectomy. Histological examination failed to show persistent carcinoma.

on his internal jugular had come off. This was resutured and the patient's condition appeared fairly satisfactory. He reacted from anesthesia within a short time only to stop breathing one hour later; death came the following morning. Postmortem examination showed that he had died of cerebral edema. There was thrombus of the right cavernous sinus and an undeveloped venous system on the left. Cervical metastases from such a small lesion, limited to the vocal cord, are infrequent.

The next case should, by all standards, have given metastases and yet he is still alive and free from disease. A large fungating mass, involving the epiglottis and the base of the tongue, was first diagnosed as a poorly differentiated carcinoma in 1951. The patient was not given any treatment and came back to the hospital one year later; he was then seen because of dysphagia and some difficulty in breathing. Indirect laryngoscopy showed a large fungating mass involving the epiglottis, aryepiglottic folds and invasion of the base of the tongue. No nodes were palpable in the neck. It was agreed to treat him palliatively by radiotherapy. 7800 r were given in 62 treatments. The regression of the tumor was remarkable but there remained a suspicious irregularity of the epiglottis which was thought to be



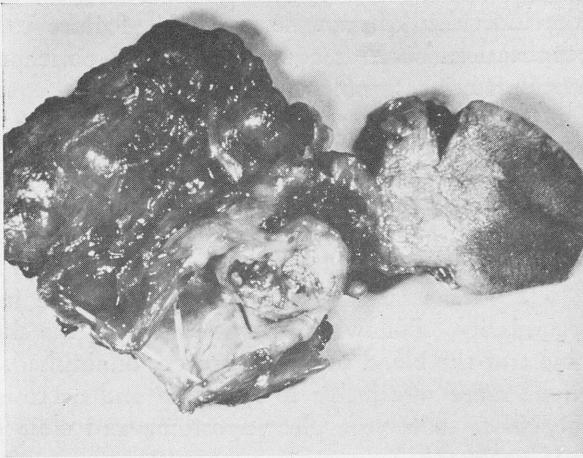


Fig. 6.—Surgical specimen showing fungating growth of vallecule. The endotracheal tube was pushed through the tumor mass between the base of the tongue and the epiglottis.

residual disease. Five months after termination of radiotherapy, a laryngectomy was performed (Fig. 5). The skin of the neck presented marked x-ray changes and our apprehension in regards to closure of the skin proved to be right at the time of the first dressing. The wound had completely broken down and the patient developed a large pharyngeal stoma. Numerous attempts were required before obtaining final closure. During that time a basal cell carcinoma of the lower eyelid was treated and cured by the dermatologists. The operative specimen failed to show residual disease.

Twenty-three cases were treated by radiotherapy, and in three of these a subsequent laryngectomy was performed. The majority of these patients were treated by radiotherapy because of their refusal of operation, and the few other cases were treated palliatively because they were considered inoperable. It has been our good fortune to work in close co-operation with the radiotherapists, who quite agree that surgery is often the best form of treatment. All our patients are seen and followed by members of both services. The last case presented is an example of the effectiveness of radiation in certain instances. Unfortunately, we can not consider radiotherapy an easy



form of treatment. It is a prolonged process, local skin and mucous membrane reactions can be severe and the effects on the general condition of the individual cannot be ignored. Failure to arrest the growth, complications such as radionecrosis and perichondritis still occur and present serious problems.

In one other case, radiotherapy had again sterilized the tumor. Because of severe pain and near impossibility of swallowing due to radiation reactions, a laryngectomy was performed on this patient. In the first two cases where laryngectomy followed radiotherapy, primary closure was attempted and failed. In the third case, the patient was left with a large pharyngostoma with the intention of closing it secondarily. Following intense radiation, the skin becomes quite atrophic and the blood supply is greatly diminished. The skin is also rendered more susceptible to infection and necrosis. I think it is probably wiser to leave a pharyngostoma and close secondarily with grafts if necessary.

Before closing, I would like to add a few words on laryngectomy. A complete medical and blood work-up, including chest and esophageal films and tomographic studies of the larynx, is routine. A most important aspect of the work-up is the psychological preparation of the patient which is done by the surgeon, the social worker, the speech therapist and a laryngectomized person.

Laryngectomy is performed under local anesthesia, except when preceded by tracheotomy or when a combined neck and larynx operation is contemplated. The patient tolerates local anesthesia very well, the bleeding is cut down to a minimum and the necessity of passing an endotracheal tube is obviated.

If for some reason, general anesthesia is used and an endotracheal tube inserted, it should be done under visual control by an experienced anesthetist. Not long ago we did a combined neck, larynx and tongue resection (Fig. 6) and, on opening the hypopharynx, it was realized that the endotracheal tube was not in the glottis, but had forced a passage right through the tumor between the epiglottis and the tongue. The anesthetist had undoubtedly not properly visualized the glottis, and when asked if he had experienced any difficulty with the intubation, he replied in the affirmative, but he did not suspect in the least what had actually occurred.

A number of incisions may be used, but we find the "T" incision most satisfactory. The hyoid bone is completely removed and the

strap muscles are included in the specimen. Closure of the pharynx is done with chromic gut and skin with clips. No gastric tube is used and the patient is fed intravenously for four or five days. In normal postoperative courses the patient is allowed to leave the hospital within ten days following the operation. Rehabilitation of the patient is started immediately.

#### SUMMARY

A brief review of 70 cases of laryngeal carcinoma, seen during the period of July 1952 to December 1955, is given.

Because of the too short follow-up period, no attempt is made to give statistical results.

In the light of recent reports and from experience gained in these cases, neck dissection is considered justifiable in cases of extrinsic and extralaryngeal lesions.

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The author would like to express his thanks to Dr. L. C. Simard, Director of the Cancer Institute, Notre Dame Hospital, for allowing to review the statistical reports and for the photography, all of which was done in his department.

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#### DISCUSSION

DR. HENRY B. ORTON, Newark, N. J.: I feel quite certain that we are seeing more cancer of the larynx in the younger group, especially in males. I have seen it in a boy of 12. The younger the individual the more rapid the growth; this fact we should not lose sight of.

I have, for some time, been very much interested in the classification of cancer of the larynx. The author calls it intrinsic cancer when the tumor is limited to the cord only. This I call the "true cordal" cancer, and in this group the prognosis is very good. I dislike the term extrinsic, for it means very little and gives no identification as to the anatomical site of the growth. I prefer the term epilaryngeal, with specification of the anatomical site of origin: as the epiglottis, aryepiglottic fold,

ventricle, arytenoid, and the piriform sinus. This classification gives us valuable data in formulating our operative procedure and estimating the probable prognosis; for in these types the operative procedure of neck dissection becomes evident with or without palpable cervical lymph node involvement.

It is true that in some of these cases it may be very difficult, or almost impossible to determine the site of origin of the cancer, but with a careful history-taking, valuable information can be obtained, and the operative attack formulated.

In 1954 I presented before the American Academy of Ophthalmology and Otolaryngology a series of cases of carcinoma of the larynx based on the above classification. It shows that my best results following surgery were in the following order for five year cures: Cords, Epiglottis, Ventricles, Arytenoid, Subglottic, Epiglottis and Tongue, Aryepiglottic fold, Piriform sinus. Recurrences approximately 20 per cent; successful cases 70 to 75 per cent; operative mortality 1.3 per cent; deaths other than from cancer 10 per cent.

I have the temerity to suggest that if Doctor Montreuil would reclassify his cases and identify the lesions by their anatomical sites with the operative procedure carried out in each case, that ultimately it will be of the greatest value in his statistical results, and will show him from what anatomical sites he can expect his best results.

We are familiar with the symptoms of laryngeal cancer and it needs no further comment here. However, there is one area often overlooked, and that is in the vallecula where exposure may be difficult due to the epiglottis in close proximity to the base of the tongue. In these early cases a trans-hyoid pharyngotomy will often spare the patient the operation of total laryngectomy.

No one should remove a larynx without histological proof, unless his experience is such as to warrant it otherwise. The specimen should be taken by the operating surgeon. Surgery still offers the best chance of cure with wide field exposure, combining radical neck dissection, if indicated.

The author reports two very interesting cases, which brings up the question of radiotherapy. I am not partial to doing surgery following radiotherapy, or giving radiotherapy following surgery, on account of the complications such as radionecrosis.

Regarding anesthesia—laryngectomy can be done under local anesthesia, and I have done it, but in my experience a general anesthesia is preferable. The psychological effect on the patient coming out of the anesthetic is, in my opinion, not as marked as it is when he suddenly realizes during the operation under local anesthesia that he can no longer talk. With the trachea exposed early in the operation the anesthetic may be at any time transferred to the tracheostomy opening as the surgeon may deem wise. I am convinced that the "U" incision gives a better closure and permits the patient to go without wearing the laryngectomy tube, which psychologically means a great deal to the patient.

The author's mortality in his 44 patients operated upon is slightly over 4 per cent which seems a little high. In my own series of over 450 cases the mortality is 1.3 per cent.

In 1927 while visiting Lionel Colledge in London, I recall he quoted the following which might help to emphasize the above:

"In these pharyngeal operations, individual surgeons have had so little opportunity of operating that their work may be looked on as altogether "Lehrgeld," and

we have not got beyond the educational stage. The "Lehrgeld" is now much less than it was, but nevertheless the price has still to be paid by both patient and operator in his early cases, and in no other way can the necessary experience be obtained which will not only enable him to get the best results, but will teach him which cases are operable, which are inoperable, and what are the limits of his capacity."

DR. FRANCIS E. LE JEUNE: I wish to commend Dr. Montreuil for his splendid presentation and his frankness in reporting an interesting case. A certain amount of stamina is required to report failures and Dr. Montreuil has done this gracefully.

There is one phase he discussed in which I am particularly interested, and I wonder whether any one present has had bad experiences with introduction of an intratracheal tube when a large carcinomatous lesion is present in the larynx. What are the possibilities of carrying a fragment of the lesion with the tip of the intratracheal tube and implanting carcinomatous cells further down the tracheobronchial tree? The general surgeons, as a rule, will discard a knife if an incision has been made through a malignant lesion and frequently new gloves are requested to minimize the possibilities of contamination. Introduction of an intratracheal tube carries with it the possibility of breaking off a piece of tissue and implantation of that tissue into the tracheobronchial tree. I know of no such complication occurring, but it certainly seems plausible that proper precautions should be taken if there is any virtue to the theory of implantation of malignant cells.

DR. EDWIN N. BROYLES: I enjoyed Dr. Montreuil's paper also. I would like to ask one question. He stated that he does not use stomach tube or esophageal tube in feeding the patient after operation but does it by intravenous injection. I would like to ask why he does not use the esophageal tube.

DR. FERNAND MONTREUIL: I would like to thank my discussers. I was a little worried as to what would happen. It wasn't bad at all.

Dr. Orton, this question of classification has me quite puzzled. There are so many different classifications that it is difficult to compare lesions classified in various groups. The groups I mentioned are large groups. We always try to describe the lesion in its exact locality. I quite agree it is very, very important.

Dr. Broyles, I don't know. I started not using a stomach tube when I was in New York and I have followed that line of treatment. I find it works out very well. There is always a certain degree of discomfort to the patient with the tube through his nose and with the old rubber tubes, I think there was a certain amount of irritation of the pharynx. I believe now with the polyethylene tubes, the plastic tubes, the irritation is much less. I found it quite satisfactory. Unfortunately we do have to insert a stomach tube in cases of fistula, which we occasionally do get.



# BENIGN TUMORS OF THE VOCAL CORD

## CLINICAL VERSUS HISTOLOGIC DIAGNOSIS

SAMUEL SALINGER, M.D.

PALM SPRINGS, CALIF.

In the Standard Nomenclature of the American Medical Association, Fourth Edition,<sup>1</sup> one finds 137 items listed under the heading "Diseases of the Larynx." The Michael Reese Hospital Department of Pathology has 24 items in the cross file index under the heading of "Vocal Cord Lesions" including inflammatory conditions. Undoubtedly these lists are incomplete and do not include some of the rarer pathologic entities. Nevertheless, the list is imposing and should cover everything. Yet, despite its wide range or perhaps because of it, confusion often arises in properly designating some of the more common lesions that we see in everyday practice. This applies particularly to tumors or malformations on the vocal cords. In order to clarify my own thinking in this connection, I gathered the reports on 232 consecutive biopsies performed at Michael Reese Hospital during the past three years and compared the preoperative clinical diagnosis with the reports from the laboratory. The result is shown in Table I. I was amazed to note that only exceptionally did the two agree (Chart I). Reflecting on this apparent inconsistency, I concluded that we were either very deficient in clinical acumen or else there was something wrong with our nomenclature. One fact, however, did stand out, namely, that when we were dealing with true neoplasms our batting average was fairly respectable. For instance, of 63 cases diagnosed clinically as carcinoma, 43 were corroborated by the microscopic examination (Chart II). The remainder of this group represented a variety of conditions which might readily be suspect of malignancy from their gross appearance. Chart III shows a group of 17 cases diagnosed clinically as papilloma, in which only eight were so proven. True papillomata, particularly if multiple, have a charac-

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From the Otolaryngologic Service, Michael Reese Hospital, Chicago.

Photographs of microscopic slides by the Department of Photography, Strich School of Medicine.

Read before the American Laryngological Association, Montreal, Canada, May 13, 14, 1956.

CHART I

|                     | CLINICAL<br>DIAGNOSIS | MICROSCOPIC<br>DIAGNOSIS |
|---------------------|-----------------------|--------------------------|
| Carcinoma           | 63                    | 43                       |
| Polyp               | 81                    | 0                        |
| Benign Tumor        | 31                    | 28                       |
| Fibroma             | 19                    | 10                       |
| Papilloma           | 17                    | 8                        |
| Edematous Mucosa    | 7                     | 4                        |
| Keratosi            | 4                     | 2                        |
| Vocal Nodule        | 4                     | 3                        |
| Inflammatory Tissue | 4                     | 1                        |
| Ulcer               | 1                     | 1                        |
| Fibroangioma        | 1                     | 1                        |

CHART II

## CARCINOMA SO DIAGNOSED IN 63 CASES

| MICROSCOPIC DIAGNOSIS     |    |
|---------------------------|----|
| Carcinoma                 | 43 |
| Squamous cell hyperplasia | 4  |
| Thrombosed fibroangioma   | 1  |
| Papilloma                 | 5  |
| Tuberculosis              | 2  |
| Neurofibroma              | 1  |
| Chronic inflammation      | 3  |
| Granulation tissue        | 4  |

CHART III  
PAPILLOMA SO DIAGNOSED IN 17 CASES

| MICROSCOPIC DIAGNOSIS     |   |
|---------------------------|---|
| Papilloma                 | 8 |
| Fibroma                   | 2 |
| Lymphangioma              | 3 |
| Squamous cell hyperplasia | 2 |
| Mucosal fold              | 1 |
| Carcinoma                 | 1 |

CHART IV  
HYPERKERATOSIS - LEUKOPLAKIA CLINICALLY  
DIAGNOSED IN 4 CASES

| MICROSCOPIC DIAGNOSIS |   |
|-----------------------|---|
| Hyperkeratosis        | 2 |
| Carcinoma             | 1 |
| Mucosal fold          | 1 |

teristic appearance which the experienced laryngologist can recognize at the first glance. Solitary lesions, however, especially if small, though appearing grossly like papilloma may be something else, as the chart shows. We had only four cases diagnosed clinically as hyperkeratosis, two of which were so proven (Chart IV). One case turned out to be a carcinoma and the fourth was just a fold of thickened mucosa. There were four cases diagnosed as "vocal nodule," three of which conformed to the usually accepted histologic picture (Chart IV).

Coming now to the group in which the clinical diagnosis was "benign growth" we find a wide variety of conditions, among which there were two cases of malignancy (Chart V). I find no fault with the clinician who sees a nondescript lesion on the vocal cords which is patently not infiltrating and generally circumscribed, and labels it "benign growth." There is nothing characteristic about most of these lesions and I do not blame the laryngologist for not

CHART V  
VOCAL NODULE - SO DIAGNOSED IN 4 CASES

| MICROSCOPIC DIAGNOSIS     |   |
|---------------------------|---|
| Squamous cell hyperplasia | 3 |
| Edematous mucosa          | 1 |

CHART VI  
BENIGN GROWTH - SO DIAGNOSED  
CLINICALLY IN 31 CASES

| MICROSCOPIC DIAGNOSIS    |    |
|--------------------------|----|
| Fibroma                  | 11 |
| Granulation tissue       | 2  |
| Carcinoma                | 2  |
| Edematous mucosa         | 2  |
| Possible Boeck's sarcoid | 1  |
| Cyst                     | 4  |
| Hyperkeratosis           | 3  |
| Angioma                  | 3  |
| Lymphangioma             | 1  |
| Edematous polyp          | 1  |
| Papilloma                | 1  |

wanting to stick his neck out and presume to supplant the microscope with his naked eye. We are fully aware of the importance of doing a biopsy on every lesion of the vocal cord that is not an acute inflammatory process. This is the only way in which an early malignancy may be recognized, as was proven by the two cases discovered in this group.

However, when we look over the group clinically diagnosed as "vocal cord polyp" (Chart VI) we find that so far as the pathologist is concerned there is no such entity. How does it happen, then, that this term is so widely used? The word itself, derived from



two Greek roots, "poly" meaning many and "pes" meaning foot, is described by Dorland<sup>2</sup> as "a smooth pedunculated growth from a mucous surface as the nose, bladder, rectum, etc., the result of hypertrophy of the mucous membrane." Yet one would hesitate to say that a rectal polyp bears any resemblance histologically to a nasal or a vocal cord polyp. All that they have in common is their shape which, after all, is a poor criterion. It is interesting to note how various authors describe this growth. In Thomson and Negus "Diseases of the Nose and Throat"<sup>3</sup> we find this description: "A true polypus originates in Reinke's space as an initial congestion followed by edema and hyalinization." They state further that "a benign laryngeal formation of this type may occur although the term has been used incorrectly in relation to other simple growths or to formations of an inflammatory nature. It is doubtful if it is a true neoplasm." Thus it would seem that they recognize an entity which they term "true polypus" which they attribute to inflammatory reaction, while at the same time calling attention to other formations of an inflammatory nature with which it is not to be confused. What these other inflammatory masses may be is not explained in the text.

Laurens<sup>4</sup> states that under the general term of "polyps of the larynx" one includes benign tumors, liquid or solid developing in the mucosa and skeletal cartilages." Under this heading he describes vocal nodules, fibromas, cysts, lipomas, myxomas, angiomas, chondromas and papilloma, thus piling Pelion on Ossa and confounding confusion in nomenclature!

Ewing,<sup>5</sup> on the other hand, fails even to mention polyps, but describes the fibroma as being the most frequent tumor of the larynx and more than likely the same as the one we call polyp. He says, "it is a small peduncuated tumor on the true or false vocal cord near the anterior commissure; its structure varies; may be hard and mostly fibrous or soft and edematous."

Holinger and Johnston,<sup>6</sup> who reviewed 1197 cases, 43 per cent of which were classified as polyps, agreed with New and Erich that although their clinical appearance may be identical they may vary widely on histologic examination.

New and Erich<sup>7</sup> made it very clear that one must differentiate between neoplastic and non-neoplastic growths, the latter being generally the result of inflammatory process or local trauma. They very correctly pointed out that polypoid tumors may appear the same as

myxomatous laryngeal neoplasms "alike in gross appearance, in mode and time of onset, and in production and duration of symptoms, and are indistinguishable except on microscopic examination. Likewise they showed that true neoplasms such as angiomas fibromas and lymphangiomas are comparatively rare and not to be confused with the "edematous fibromas" or "hemangiomas" or so-called polyps as many of our cases have been designated.

Friedberg and Segall<sup>8</sup> demonstrated that the dominant features of polyps are edema, thrombosis and hyalinization, and that they develop out of the subepithelial space of Reinke because of its peculiar

CHART VII  
POLYP - SO DIAGNOSED  
CLINICALLY IN 81 CASES

| MICROSCOPIC DIAGNOSIS |    |
|-----------------------|----|
| Fibroma               | 30 |
| Papilloma             | 4  |
| Hemangioma            | 10 |
| Lymphangioma          | 6  |
| Neurofibroma          | 4  |
| Edematous mucosa      | 17 |
| Carcinoma in situ     | 2  |
| Granulation tissue    | 8  |

anatomic configuration and that they are in no sense to be considered as neoplasms. Ash and Schwartz<sup>9</sup> also called attention to Reinke's layer in which the mucosa is in close apposition to the deeper tissues at the striae arcuata. Lesions such as polyps, varix, amyloid, etc., are but various stages of the same process. Heavy concentration and intimate application of the elastica over the cord muscle serves as a barrier to deeper extension of these lesions. Glandular elements are comparatively scarce, which accounts for the rarity of cystic formations. Blood vessels and lymph spaces being exposed within these boundaries react to the exciting factors, thus yielding varying pictures resembling true angioma and lymphangioma. They state

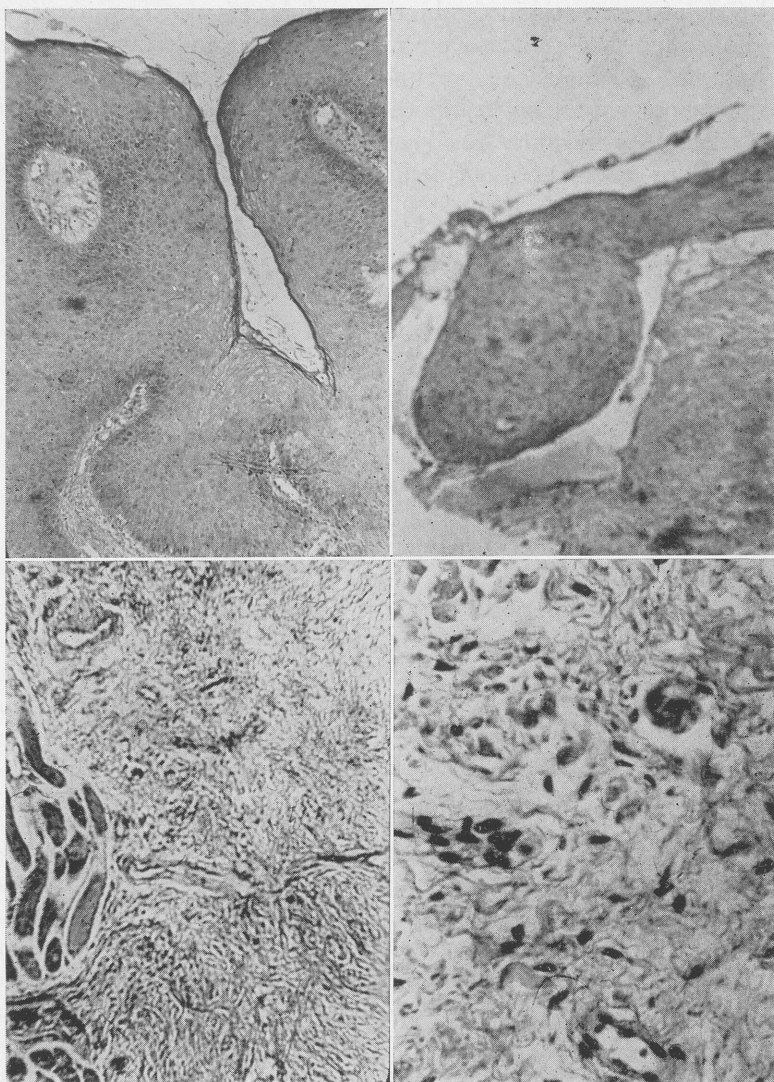


Fig. 1.—Papilloma.

Fig. 2.—Vocal nodule—Clinical diagnosis: Hyperkeratotic vocal cord. Microscopic diagnosis: Submucosal edema, old granulation tissue with overlying squamous cell hyperplasia.

Figs. 3 and 4.—Neurofibroma (53 C1 34)—Clinical diagnosis: Possible carcinoma. Microscopic diagnosis: Neurofibroma—transitional cell hyperplasia.



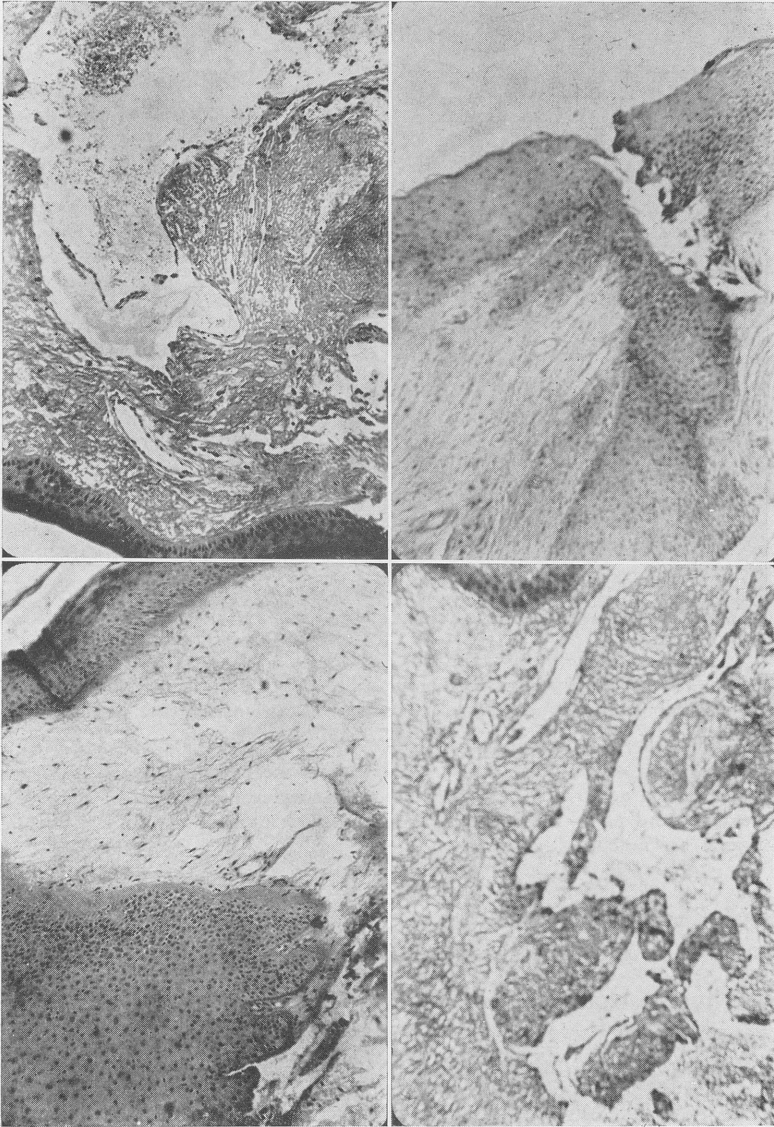


Fig. 5.—Lymphangioma (53P5091)—Clinical diagnosis: Vocal cord polyp. Microscopic diagnosis: Pedunculated lymphangioma.

Figs. 6 and 7.—Edematous fibroma: Papilloma (54P1669)—Clinical diagnosis: Polyps larynx. Microscopic diagnosis: Edematous fibroma and hard papilloma.

Fig. 8.—Hemangioma (52P4173)—Clinical diagnosis: Polyp vocal cord. Microscopic diagnosis: Pedunculated hemangioma.



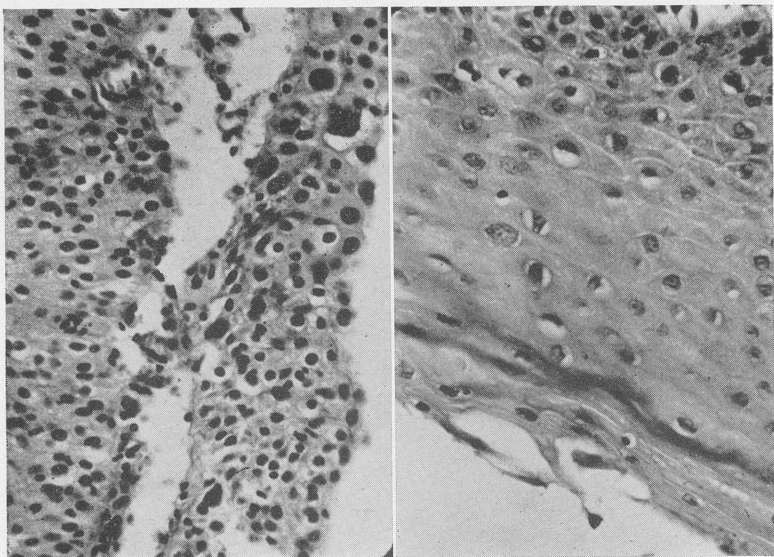


Fig. 9.—Carcinoma in situ (54P2242)—Clinical diagnosis: Vocal cord polyp. Microscopic diagnosis: Tumor cells are stratified squamous cell with large pleomorphic hyperchromatic nuclei. Occasional mitotic figures seen.

Fig. 10.—Carcinoma in situ (54P3397)—Clinical diagnosis: Vocal cord polyp. Microscopic diagnosis: In one section the squamous epithelial layer becomes disorderly and the cells exhibit pleomorphism. The nuclei are partly vesicular and partly hyperchromatic in type. A number of atypical mitotic figures are noted.

further that lesions such as polyps, varix, amyloid, etc., are but variations in the response to the same irritant.

Thus it would appear to be the consensus that the so-called laryngeal or vocal cord polyp is a non-neoplastic tumor-like formation originating out of the subepithelial space of Reinke and capable of assuming various histologic appearances depending on the degree of the irritating factor and the particular histologic elements most involved. Our pathologist seems to agree with this conception, as evidenced by his reports in our 81 cases (Chart VII). According to his analysis the predominance of changes in the fibrous and vascular elements appear to be the most frequently encountered. The diagnosis of "edematous fibroma" runs through a majority of this group. Yet, one would hesitate to call these true fibromas, hemangiomas or

lymphangiomas, since they lack the features commonly associated with true neoplasms. It is interesting to note that in this group, all clinically labeled "polyps," there were four cases of neurofibroma and two cases of carcinoma in situ. I doubt very much that the cases diagnosed as neurofibroma were true neoplasms, although such tumors have been reported. The appearance of elongated neural elements in quasi palisade formation may be only the reaction of these elements to the exciting factor which produced the mass. The true neurofibroma exhibits greater pleomorphism of the cells, occasionally giant cells, and a tendency to recurrence on removal with occasional degeneration into malignancy.

Since there is no acceptable histopathologic picture characteristic of the vocal polyp, I find it disconcerting and frustrating to see the term so widely employed. Even its gross appearance is not uniformly characteristic since it may be pedunculated or sessile, pale or injected, smooth or lobulated.

For myself, I am content to avoid using that term and to designate it a "benign growth of the vocal cord." The fact that the experienced laryngologist can determine from his clinical examination that at least 90 per cent of the lesions which appear benign to him are so proven by the laboratory examination, should be sufficient to justify the clinical diagnosis of "benign growth of the vocal cord" rather than "vocal cord polyp" implying something specific, which it is not.

1260 E. ALEJO ROAD

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NOTE: I am indebted to Dr. Otto Saphir, Pathologist at Michael Reese Hospital, for interpretation of microscopic slides.

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## DISCUSSION

DR. FRANCIS E. LEJEUNE: Dr. Salinger has presented a very interesting and intriguing problem. I thoroughly concur that polyps of the vocal cords do not represent new growths. I am a bit astounded to hear him report that in a clinical diagnosis of 81 cases of polyps of the vocal cords made at the Michael Reese Hospital not one was confirmed by microscopic examination. I cannot believe that the clinician can be wrong 100 per cent of the time—rather I would like to believe that there is lack of unanimity of thought between the pathologist and the laryngologist. A discussion of common problems by the pathologist and the laryngologist and an invitation to see these lesions by direct laryngoscopy would lead to better understanding and mutual benefit. There is evidence, as stated by Dr. Salinger, that lesions such as polyps, fibromas and hemangiomas, merely represent various stages of the same process, dependent upon irritating factors and the stage in which they are removed. The external appearance of a polyp may not show much change as it goes through various stages, whereas the microscopic picture will vary slightly but its basic appearance is that of a polyp.

Dr. Salinger stated that "since there is no acceptable histopathological picture characteristic of the vocal polyp" he finds it "disconcerting and frustrating to see the term so widely used." May I allay his frustration? Fisher, in Coates, Schenck and Miller's recent publication, wrote, "stroma of a laryngeal polyp is quite characteristic in appearance and structure and can usually be recognized without its epithelial covering. . . . A polyp is composed of edematous connective tissue, contains prominent vasculature and is covered by epithelium on all sides but its base of attachment." Morrison stated, "laryngeal polyps develop from Reinke's layer of the vocal cords which is a thin layer of loose connective tissue lying between the epithelium and the elastic muscular tissues of the superior surface of the vocal cords. The polyp has the same basic substances as the tissues of this layer and lacks muscular fibers and elastic tissue." Eggston and Dorothy Wolf are in accord that polyps represent edema of Reinke's triangle and this is in agreement with Friedberg and Segall quoted by Dr. Salinger. Eggston and Wolf further stated, "they are usually of an inflammatory or traumatic nature with frequent thrombosis of the blood vessels accounting for the term frequently used by our pathologist of angiomatous polyp."

Dr. Salinger has suggested using the term benign growths for these types of lesions. It is true they are benign growths, but I believe it would be a mistake to designate them as such as I think we should welcome the challenge to pinpoint our diagnosis.

I have enjoyed Dr. Salinger's informative presentation. It stimulates incentive to more accurate diagnostic ability and it has been responsible for my acquiring added knowledge of the histopathology of lesions of the larynx.

DR. JAMES M. ROBB: Dr. McMahon and Gentlemen, some years ago I sent for publication a paper in which I called the tumor a hemorrhoid of the vocal cord. This choice of word was supported by Dr. P. Morse, the pathologist at Harper Hospital, Detroit, because he said, "In my opinion frequently there is a dilated vessel, a dilated blood vessel that in time undergoes changes, because chronic irritation always produces fibrous tissue which always contracts." Apparently the word "hemorrhoid" was distasteful and varix was finally chosen. I am very glad to find some other way to designate satisfactorily this type of tumor. This helps clarify the confusion that has been going on for years. I had thought hemorrhoid was descriptive.

DR. SAMUEL SALINGER: I am very grateful to Dr. LeJeune for the information he gave me relative to other references and to Dr. Robb for his interesting observation.

The point that has troubled me has been, as I stated before, that our pathologist would not recognize from the specimens we gave him that there is such a thing as a polyp. He never said it was a polyp. He usually called it "edematous fibroma" or "mucosal hyperplasia." In other words, why did he not recognize these sections as polyps?

What is there so characteristic of the polyp when it occurs in the larynx that it fails to be so identified microscopically? The pathologists should say "This is a polyp" when looking at it through the microscope just as they do in the case of a papilloma. They do not say polyp. They describe the various inflammatory reactions that take place in the tissue but do not say that this is a polyp. That sort of thing sticks in my throat. It may be a species of the signs of advancing age and increasing senile sclerosis that affects this thing in my mind. Until somebody gives me a clear microscopic histopathic picture of what a polyp of the larynx is I will still be doubtful about using it.



## PRIMARY MALIGNANT TUMORS OF THE UVULA

FRED Z. HAVENS, M.D.

AND

RICHARD C. YE, M.D.

ROCHESTER, MINN.

Malignant tumors primary in the uvula are very rare. The standard textbooks of diseases of the nose and throat do little more than mention the fact that such tumors can affect the uvula. Because such lesions are so rarely encountered it is not surprising that most articles in the literature on this subject are limited to the report of a single case. Seven cases have been observed at the Mayo Clinic in the period 1919 to 1952 inclusive. The rarity of such lesions is emphasized by the fact that during this period some 1,500,000 patients were admitted to the clinic for examination.

Anatomists have described the soft palate as a backward projection of two layers of mucous membrane from the hard palate, enclosing muscles, an aponeurosis, racemose glands, arteries, nerves, lymph vessels and veins. A conical process known as the uvula projects from the middle of the posterior margin of the palate. It is composed chiefly of a mass of racemose glands and connective tissue covered by mucous membrane and containing slender prolongations of the musculus uvulae in its upper contractile part.

The physiologic function of the uvula was not fully described until 1948 when Richardson and Pullen<sup>1</sup> published their work on structure, function and importance of the uvula. It was considered to be a median-line structure which massages and moistens the posterior pharyngeal wall, aids in the removal of material from it and assists in the passing of this material downward to the hypopharynx and the base of the tongue. In other words it plays a fairly important role in the function of deglutition. These authors expressed the belief that the uvula has little if any importance with reference to speech.

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The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

Read before the American Laryngological Association, Montreal, Canada, May 13, 14, 1956.

TABLE I  
REPORTS OF TUMOR OF THE UVULA COLLECTED BY STOUT

| CASE | TYPE OF LESION | AGE, YEARS | SEX | DURATION OF LESION | OPERATIONS  | RECURRENCE                      | MONTHS AFTER OPERATION |
|------|----------------|------------|-----|--------------------|-------------|---------------------------------|------------------------|
| 1    | Epithelioma    | 81         | M   | "Early"            | Biopsy only | —                               | —                      |
| 2    | Carcinoma      | 66         | M   | 3rd mo.            | 1           | Yes                             | 14                     |
| 3    | Epithelioma    | 37         | F   | 8th mo.            | 1           | No                              | 60                     |
| 4    | Carcinoma      | 49         | M   | 2nd mo.            | 2           | No                              | ?                      |
| 5    | Epithelioma    | 51         | M   | 2nd yr.            | 3           | No                              | 5                      |
| 6    | Carcinoma      | 52         | M   | 3rd mo.            | 1           | No                              | 14                     |
| 7    | Epithelioma    | 62         | M   | "Early"            | 1           | No                              | ?                      |
| 8    | Epithelioma    | 75         | M   | ?                  | 1           | No                              | ?                      |
| 9    | Epithelioma    | 56         | M   | 2nd mo.            | 1           | Cervical nodes<br>10 yrs. later | —                      |
| 10   | Epithelioma    | 41         | F   | "Early"            | 2           | Yes                             | 48                     |
| 11   | Epithelioma    | 64         | M   | 3rd mo.            | 1           | No                              | 5                      |
| 12   | Carcinoma      | ?          | M   | 8th mo.            | 3           | Yes                             | 2                      |
| 13   | Epithelioma    | 37         | M   | ?                  | 1           | No                              | 1                      |

TABLE II  
PRIMARY MALIGNANT TUMORS OF THE UVULA

| CASE | AGE<br>SEX | SYMPTOMS AND<br>DURATION                               | EXTENSION<br>TO PALATE                       | METASTASIS<br>TO CERVICAL<br>NODES  | HISTOLOGIC<br>DIAGNOSIS                      |
|------|------------|--|--|-------------------------------------|--|
| 1    | 70<br>M    | No local symptoms,<br>Enlarged nodes in<br>neck, 6 mo. | Very little;<br>uvula "almost<br>eroded off" | Bilateral                           | Squamous cell<br>epithelioma<br>(not graded) |
| 2    | 63<br>M    | No symptoms;<br>lesion found on<br>examination         | No   | —                                   | Squamous cell<br>epithelioma,<br>grade 3     |
| 3    | 56<br>F    | Pain on swallowing,<br>6 mo.                           | Very little                                  | Left side;<br>extensive<br>and deep | Squamous cell<br>epithelioma,<br>grade 3     |
| 4    | 71<br>M    | Pain in throat run-<br>ning up to ear,<br>9 mo.        | No   | No                                  | Squamous cell<br>epithelioma,<br>grade 3     |
| 5    | 57<br>M    | Persistent soreness<br>on swallowing,<br>3 1/2 mo.     | Little                                       | No                                  | Squamous cell<br>epithelioma,<br>grade 3     |
| 6    | 65<br>M    | Persistent sore<br>throat, 2 mo.                       | Little                                       | No                                  | Squamous cell<br>epithelioma,<br>grade 2     |
| 7    | 59<br>F    | Persistent soreness<br>and pain in throat,<br>6 mo.    | No   | No                                  | Squamous cell<br>epithelioma,<br>grade 2     |

TABLE II  
AT THE MAYO CLINIC, 1919 TO 1952 INCLUSIVE

| TREATMENT   | LOCAL<br>RECURRENCE | OTHER DATA   |
|---|---------------------|--|
| None (inoperable)   | —                   | Axillary metastasis also; death 2 mo. later  |
| Excision, electrocoagulation  | No                  | Patient had also epithelioma of lower lip with bilateral cervical metastasis. Microscopic examination of tissue from lip and cervical nodes revealed squamous cell epithelioma, grade 4, in each region. Death 6 mo. later (metastasis from lip) |
| Electrocoagulation, dissection of neck, x-rays                          | No                  | Death within 1 yr.   |
| X-rays (elsewhere)  | Yes                 | Intracranial metastasis  |
| Electrocoagulation of residual tumor (uvula had been removed elsewhere) | No                  | No recurrence after 4 yrs.   |
| Electrocoagulation  | No                  | Dissection of nodes not done because of active pulmonary tuberculosis; alive after 3 yrs.  |
| Cautery excision, electrocoagulation                                    | No                  | No recurrence 1 yr. after treatment. Lived only 50 miles away; should return if any trouble.   |



Stout<sup>2,3</sup> reviewed the literature from 1858 to 1915 and found reports of 13 cases in which there had been a malignant tumor of the uvula (Table I). In the group there were four cases of carcinoma and nine of epithelioma. The data obtainable from these assembled case reports were not adequate to enable him to come to any definite conclusions regarding the behavior and treatment of the lesion.

In 1939, Robb and Michels<sup>4</sup> reported two cases of primary squamous cell carcinoma of the uvula and concluded that early excision offers a better prognosis than does irradiation alone. Frühwald,<sup>5</sup> in 1950, stated that he believed leukoplakia to be a factor predisposing to carcinoma of the uvula. In 1951, Tremble and Lockhart<sup>6</sup> reported a case of squamous cell epithelioma of the uvula which was excised together with the greater part of the soft palate. This was followed by deep x-ray treatment. Postoperative difficulty in swallowing liquids was only transient. One year later the patient was found to have no evidence of any recurrence.

#### THE PRESENT SERIES OF CASES

The data on our cases are summarized in Table II. Persistent soreness in the throat or pain on swallowing, or both, were the only symptoms encountered. Two patients had no complaints with reference to the throat and the tumor was found while a search was being made for a primary lesion to explain the presence of metastatic growths in the cervical lymph nodes.

The lesion may appear as an ulcer or the uvula may have a granular appearance and look as if it were indurated and stiff rather than flabby as is the normal uvula. In one of our patients the lesion was identified only when the posterior aspect of the uvula was inspected by means of a suitable mirror.

We believe excision of the local lesion, preferably with the electrocoagulating unit, to be the best method of treatment. Surgical dissection of regional lymph nodes was not carried out for any patient who did not have clinical evidence of involved nodes. In one case in which it was considered it was not done because the patient had active pulmonary tuberculosis.

Two of our patients had extensive metastatic lesions in the cervical nodes and a third had an intracranial metastatic lesion. In other words, in this group of seven patients each of whom had a relatively insignificant-appearing primary lesion, there were secondary lesions which led to disaster in 42.8 per cent of the cases. This

emphasizes the importance of careful inspection of the uvula along with other parts of the oral and pharyngeal structures when conducting an examination.

### MAYO CLINIC

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1. Richardson, G. S., and Pullen, E. M.: The Uvula: Its Structure and Function and Its Importance. *Arch. Otolaryng.* 47:379-394 (Apr.) 1948.
2. Stout, P. S.: Tumors of the Uvula With Report of a Case of Papilloma of Uvula. *Laryngoscope* 25:88-89 (Feb.) 1915.
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4. Robb, J. M., and Michels, M. W.: Primary Squamous Cell Carcinoma of the Uvula: Report of Two Cases. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 48:521-524 (June) 1939.
5. Frühwald, V.: Uvulakarzinom bei leukoplakie. *Krebsartz* 5:178 (Aug.) 1950.
6. Tremble, G. E., and Lockhart, H. B.: Carcinoma of the Uvula. *Canad. M. A. J.* 65:50-51 (July) 1951.

### DISCUSSION

DR. LEROY A. SCHALL: The invitation to discuss this paper of Drs. Havens and Ye and the recent article by Drs. Maguda and Bowers<sup>1</sup> sent me into my own files for statistical data on which to base my discussion.

From a careful review of the records of 12 patients seen since 1935, I am in complete agreement both with Drs. Havens and Ye and with Drs. Maguda and Bowers.<sup>1</sup>

I have been impressed by the fact that early malignant disease of the uvula may cause but few symptoms. Patient R.B.C., seen in 1954, had no symptoms; but the lesion of the uvula was discovered by his dentist.

It is unfortunate that so many of these patients come to us too late. It is often difficult to tell whether the primary lesion involved the uvula and spread to the soft palate, or whether it was primary in the soft palate and secondarily involved the uvula.

I agree with the authors in that whenever it is surgically possible, the lesion should be removed. Of 8 patients treated primarily by electro-surgical removal, 6 showed no recurrence for periods of 1, 2, 6, 7, 8 and 10 years. Of 4 patients treated by irradiation, 3 showed no recurrence after 3, 4, and 6 years. Perhaps we were not

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1. Maguda, Thomas A., and Bowers, Ralph F.: Primary Carcinoma of the Uvula. A Report of Seven Cases. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 65:108 (Mar.) 1956.

fair to our radiologists by referring only the more extensively involved cases to them—one of these being an 82-year-old man with extensive disease and bilateral cervical metastasis.

I should like to express my appreciation to the authors for bringing this subject of primary malignant disease of the uvula to our attention and for reminding us again of the extreme importance of a thorough examination of every patient.

#### MALIGNANT DISEASE OF THE UVULA

| DATE     | PATIENT | SEX | AGE | LESION                                  | TREATMENT        | RESULT                        |
|----------|---------|-----|-----|---|------------------|-------------------------------|
| 11- 7-35 | J.S.    | M   | 67  | Ca. Gr. II<br>Uvula &<br>Soft Palate    | Surgery          | Well 1 Yr.<br>Cannot Trace    |
| 1- 3-35  | O.Y.    | F   | 62  | Ca. Gr. II<br>Soft Palate<br>Uvula      | Surgery          | Well 7 Yrs.<br>Dead—No Ca.    |
| 12-18-37 | A.J.G.  | M   | 69  | Ca. Gr. I<br>Uvula<br>Soft Palate       | X-Ray            | Well 6 Yrs.<br>Dead—No Ca.    |
| 4-12-39  | D.S.    | M   | 58  | Ca. Gr. II<br>Uvula                     | Surgery          | Well 9 Yrs.<br>Recurrence     |
| 7-13-48  |         |     |     | Soft Palate                             | Surgery          | Well 8 Yrs.                   |
| 8-17-42  | F.E.D.  | M   | 69  | Ca. Gr. II<br>Uvula<br>Soft Palate      | X-Ray            | Well 3 Yrs.<br>Cannot Trace   |
| 6-15-48  | J.D.N.  | M   | 32  | Ca. Gr. I<br>Uvula                      | Surgery          | Well 7 Yrs.                   |
| 5-16-44  | J.M.    | M   | 82  | Gr. II. Uvula<br>Cervical<br>Metastasis | X-Ray            | Dead<br>1 Month               |
| 1-18-40  | F.P.    | M   | 71  | Gr. III Uvula<br>Cervical<br>Metastasis | X-Ray            | Dead<br>4 Years               |
| 2-15-44  | N.S.    | M   | 74  | Gr. IV Ca.<br>Uvula                     | Surgery          | Dead 10 Yrs.<br>No Recurrence |
| 9-30-54  | J.M.    | M   | 52  | Sq. Cell Ca.<br>Uvula                   | Surgery          | Dead<br>2 Months              |
| 11- 4-54 | R.B.C.  | M   | 54  | Malignant<br>Melanoma<br>Uvula          | Surgery<br>X-Ray | Well<br>1956                  |
| 11- 6-50 | J.C.    | M   | 76  | Gr. I Uvula<br>& Tonsil<br>Pillars      | Surgery          | Well<br>5+ Yrs.               |

DR. JAMES M. ROBB: There is a question I should like to ask Dr. Havens and that is, in his surgical effort, does he remove the soft palate? I have had experience with three patients who had carcinoma of the uvula. With surgery and x-ray treatment, they did not recover. I am sure of this, that any malignant lesion of the uvula is a serious problem.

DR. FRED Z. HAVENS: Dr. Schall, we, like you, found in our records many cases in which it would be impossible to know where the lesion had started and where there was extensive involvement of the palate. We purposely left these out because we wanted to stress the point of the danger of this lesion even though it were limited to the uvula alone. We picked out only those cases in which we knew the lesion had not extended beyond the uvula, in order to emphasize the point that we must not be careless about the lesion that does not extend beyond the uvula.

I like Dr. Schall's emphasis upon careful examination. The case that stimulated us to write this paper had previously passed through the hands of a very competent otolaryngologist and he was extremely embarrassed that he had missed seeing the lesion. In that case it involved only the lower third of the uvula, looked a little bit granular and firm and I think I should have said in my discussion that one should add a finger examination. In this case the minute we touched it we felt very sure that it was a malignant lesion.

Dr. Robb, in answer to your question about the removal of the soft palate: in the case that was the basis of the paper the lesion involved only the lower third of the uvula. This was the only patient I have operated upon. The answer to your question would be to take off what seems necessary. In the case we are discussing we removed palatal tissue for a distance of approximately one and one-half cm proximal to the base of the uvula.



# MECHANICAL APPLIANCE FOR CICATRICIAL STENOSIS OF LARYNX AND UPPER PART OF TRACHEA:

REPORT OF CASE

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On analyzing the results of surgical techniques designed to correct cicatricial stenosis of the larynx, one finds that a satisfactory result can be anticipated in practically all cases in which the laryngeal and tracheal cartilages, and in particular the cricoid cartilage, are intact. However, when all or a large portion of the cricoid cartilage is lost through trauma or perichondritis and sequestration, the possibility of maintaining the patency of a newly constructed lumen through the larynx and upper part of the trachea is remote. In fact, loss of the cricoid cartilage practically always precludes the possibility of eliminating the wearing of a tracheal cannula. Such was the situation in the case which I wish to report in this paper. However, in spite of the fact that the tracheotomy tube could not be removed, a mechanical contrivance was devised to permit normal breathing through the nose and mouth and natural speech. This appliance or some modification of it perhaps might be of value for other patients with laryngeal stenosis in which the surgical treatment fails, and the patient must continue to wear a tracheal tube.

The patient under consideration had a traumatic injury to the larynx and subsequently complete laryngeal stenosis in the subglottic region due to perichondritis. Although there was some limitation of motion of the vocal cords, both of them did move satisfactorily. As will be pointed out later, it was discovered during the surgical repair of this stricture that practically all of the posterior part of the cricoid cartilage had been extruded due to sequestration. The loss of cartilage was associated with a sort of herniated protrusion of the

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underlying esophageal wall. This undesirable circumstance so complicated treatment of the stenosis that attempts to maintain the patency of a newly constructed laryngeal lumen had to be abandoned. However, the mechanical appliance previously mentioned has enabled the patient to carry on his normal activities with an unimpaired voice.

Some of the operative procedures undertaken in this case involve the form of treatment for cicatricial laryngeal stenosis which I employ and which I would like to review briefly before giving the details of the management of the patient.

#### TREATMENT OF CICATRICIAL STENOSIS OF THE LARYNX

The surgical exposure of the scar tissue causing cicatricial stenosis of the larynx or upper part of the trachea can be accomplished with the least amount of difficulty under local anesthesia—that is, infiltration of 1.5 per cent solution of piperocaine hydrochloride (metycaine).<sup>1</sup> An incision is made from the tracheotomy opening upward to expose the thyroid and cricoid cartilages. These are divided in the midline (Fig. 1). By separating the cut edges of these cartilages, the underlying laryngeal mucosa and the cicatrix can be incised in a vertical direction to expose the interior of the larynx and trachea as well as the constricted lumen in the region of the stricture. The scar tissue producing the obstruction then can be excised; this procedure restores the normal dimensions of the laryngeal and upper tracheal lumen. However, surgical removal of the cicatrix leaves a raw surface which, if of appreciable size, should be covered with a thin shave (Thiersch) skin graft. Such a graft offers a rapid method of epithelialization for the freshly denuded surface and tends to reduce the subsequent development of scar tissue.

A shave skin graft is wrapped around a sponge rubber mold which has been cut to fit snugly within the larynx and trachea (Fig. 1). The graft should be large enough to cover the sponge and is tacked down to the latter in several places with fine silk. The stent covered with a skin graft is held in place with a suture passed around the tracheal cannula and with two or three strong transfixation sutures which are passed through the neck and larynx from one side to the other.

After a week to ten days, the mold can be removed and invariably one finds that the skin graft has taken satisfactorily. At this stage, some form of obturator must be prepared to maintain the patency of the newly made lumen for several months. Otherwise, residual scar-

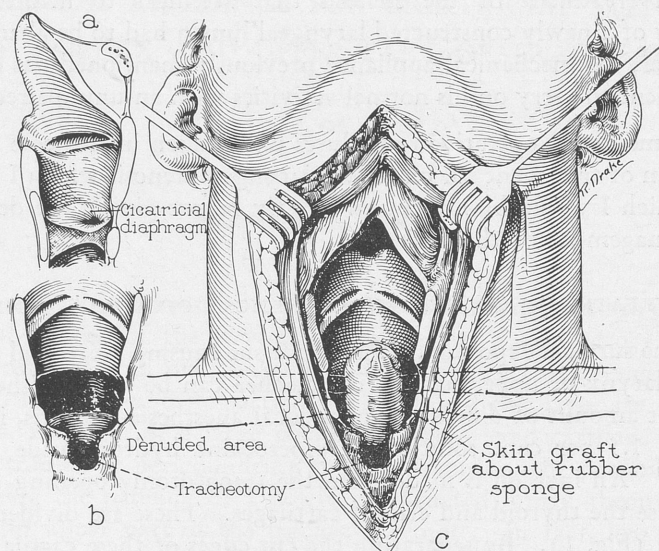


Fig. 1.—Surgical excision of the thickened scar tissue producing laryngeal or upper tracheal stenosis, with subsequent grafting of skin on the resultant raw surface. *a.* The subglottic stricture. *b.* The resultant raw surface after the surgical excision of the scar tissue. *c.* The surgical approach through the midline to the interior of the larynx and the method of grafting skin on the newly formed lumen. The sponge rubber mold covered with a skin graft is visible in the region of the newly formed subglottic lumen. Three silk sutures used for immobilization of this mold are visible.

ring in the walls of the larynx and trachea may contract to reproduce the stricture. Laryngeal obturators have been constructed in various materials. Some surgeons employ hard rubber tubing or sponge rubber for this purpose. Although rubber obturators do maintain patency of the laryngeal lumen, they do have certain disadvantages. They tend to elongate and then must be removed and replaced with one of proper size. Rubber obturators stimulate the formation of granulation tissue within the larynx as well as a discharge which may have a foul odor. Furthermore, there is often some difficulty in maintaining fixation of a rubber obturator in the larynx for a long time.

*The Acrylic Obturator.* I prefer obturators made of the plastic (acrylic) materials. They have the advantage of never changing shape; they are smooth, nonporous, nonirritating and tend to

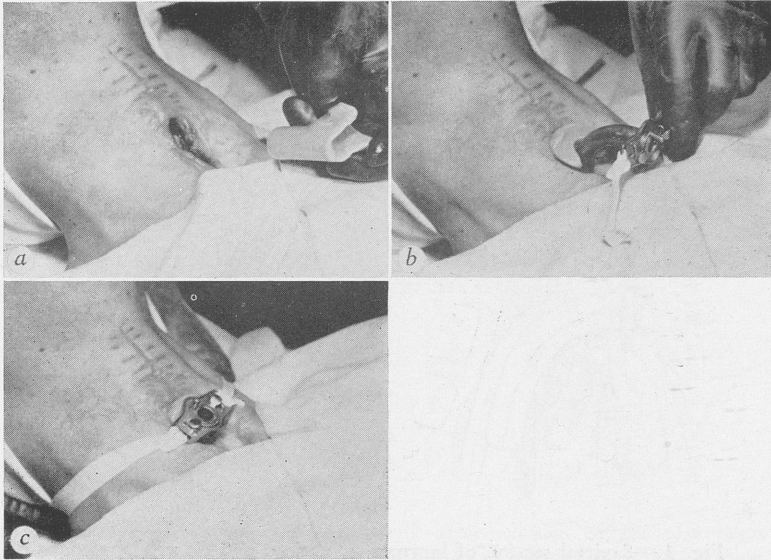


Fig. 2 *a*.—The finished acrylic obturator is about to be inserted through the external tracheal opening into the newly formed and skin-grafted lumen. *b*. The acrylic obturator is in position and the tracheal cannula is being inserted below. *c*. The acrylic obturator and the tracheal cannula in correct position.

inhibit the formation of granulation tissue. They produce no discharge and consequently no odor. The type of acrylic obturator which I employ has one disadvantage: it necessitates a large tracheal stoma. However, it is my opinion that the advantages of this type of obturator outweigh this one disadvantage.

The various steps required in the construction of an acrylic obturator are briefly as follows:

After removal of the sponge rubber mold from the larynx, a section of large-size rubber tubing is cut to equal the length of the sponge rubber mold. This piece of rubber tubing is coated with petrolatum and is introduced in an upward direction through the external tracheal opening into the larynx. If the tracheal stoma is not of sufficient size vertically to permit insertion of the rubber tube, the opening must be enlarged by an upward incision in the median



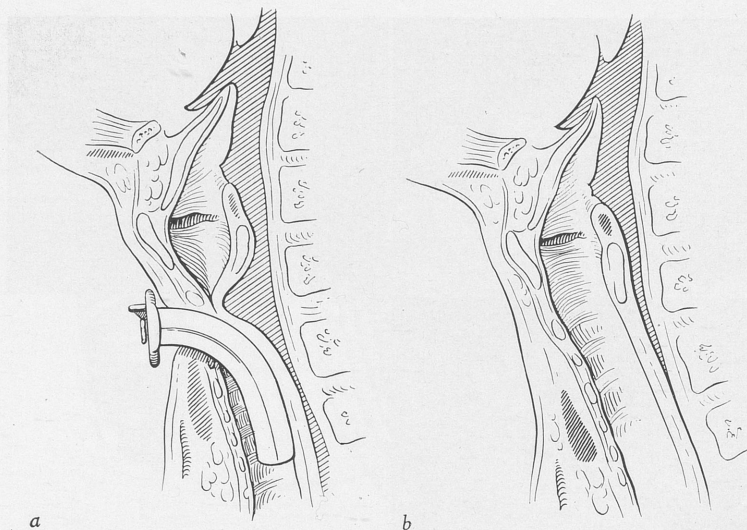


Fig. 3.—Sagittal section of larynx. *a*. Patient had cicatricial stenosis of the subglottic region of the larynx which completely obliterated the laryngeal lumen. This stenosis was associated with a sort of herniated protrusion of the anterior wall of the esophagus into the cicatricial mass in the larynx. *b*. Normal trachea and esophagus shown for comparison with the stenosed larynx in *a*.

line. After the rubber tube has been seated within the larynx, the tracheal cannula is inserted. A piece of silk suture thread which has previously been inserted through the lower end of the rubber tube anchors the latter in the larynx to the tracheal cannula.

A small piece of dental impression compound is softened in a pan of hot water. This soft compound is forced into the tracheal opening above the tracheal cannula and to the rubber tube within the larynx. The suture anchoring the rubber tube to the tracheal cannula is out, thus permitting withdrawal of the tracheal cannula, the extension made of dental compound, and the piece of rubber tubing in the larynx. The extension and the rubber tube are assembled in the same relationship to each other as existed when located within the larynx. These two structures are made to adhere with melted wax. After the two open ends of the piece of rubber tubing have been blocked with wax, the model consisting of the piece of

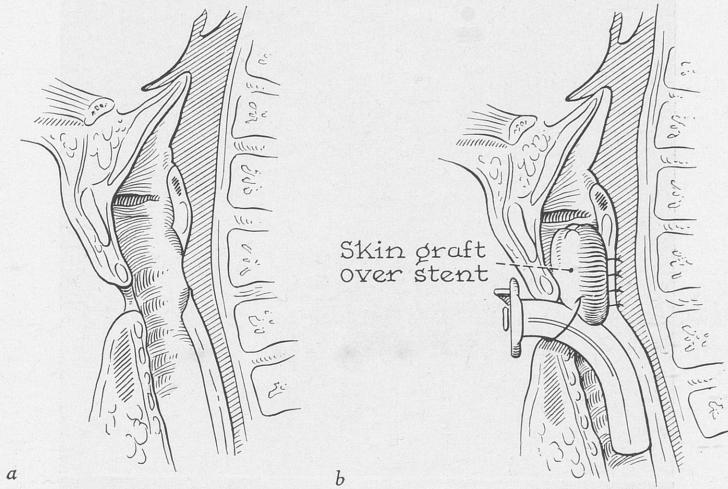


Fig. 4 *a*.—Sagittal section of larynx after excision of the scar tissue forming the subglottic stenosis which resulted in an opening (tracheo-esophageal fistula) from the larynx and trachea into the esophagus as illustrated. *b*. The lateral edges of this tracheo-esophageal perforation were sutured together; the raw surface within the larynx due to excision of scar tissue was skin-grafted by applying a Thiersch graft over a sponge rubber stent as illustrated.

rubber tube and the dental compound extension is sent to the dental laboratory for processing in one of the acrylic materials.

While the acrylic obturator is being made, the original sponge rubber mold should be reinserted into the larynx and anchored around the tracheotomy tube with a silk suture. If this sponge rubber mold were not inserted during the several hours which are required for construction of the obturator, the newly formed lumen within the larynx would become so contracted that the introduction of the obturator would be impossible.

When the acrylic obturator has been finished by the dental laboratory, the sponge rubber mold and tracheotomy tube are again removed. The obturator is promptly introduced through the tracheotomy opening into the newly formed laryngeal lumen (Fig. 2). Subsequently, the tracheal cannula is inserted into the trachea just below the obturator. The forward extension of this obturator rides

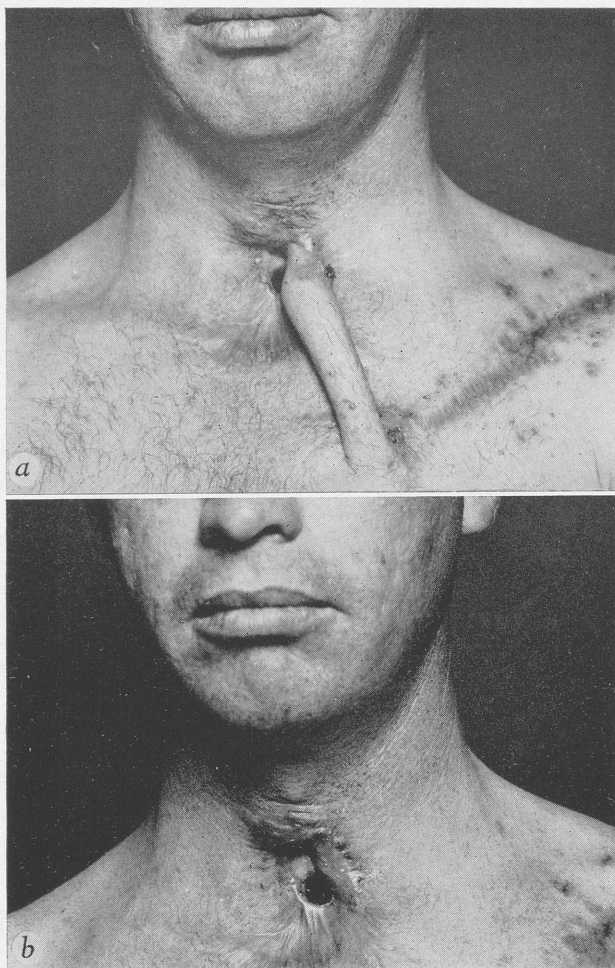


Fig. 5 *a*.—Lateral end of the clavicular tube flap transferred to cover the tracheo-esophageal fistula. All of the laryngeal mucous membrane on the lateral and posterior walls of the larynx in the region of the fistula was excised before the tubed pedicle flap of skin was applied. *b*. The unused part of the clavicular tube flap has been cut off and discarded. The fistulous tract has been permanently closed. However, the use of the pedicle flap of skin within the larynx has so reduced the size of the laryngeal and tracheal lumen that removal of the tracheal cannula is impossible.

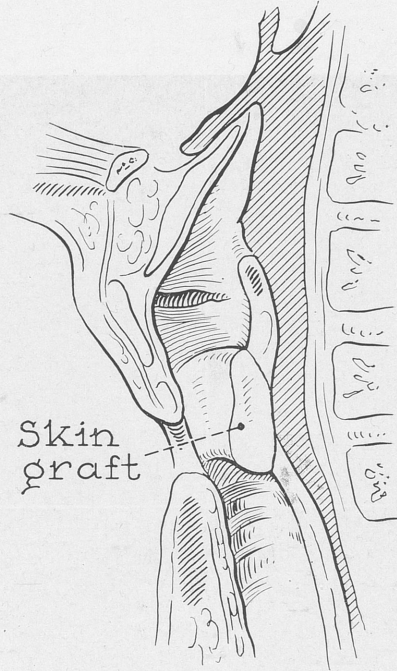


Fig. 6.—Sagittal section of larynx and trachea. The tracheo-esophageal fistula has been covered with skin from a clavicular tube flap. Notice how the use of this flap of skin has reduced the size of the laryngeal and tracheal lumen.

on top of the tracheotomy tube and emerges through the tracheotomy opening. This extension prevents any upward or downward displacement of the obturator whether or not the tracheal cannula is in position. If, at any time, the surgeon wishes to examine the interior of the larynx, the tracheotomy tube and obturator can be removed through the tracheotomy opening and the examination performed as an office procedure.

In my experience, such an obturator must be worn for three to six months if the patency of the newly formed laryngeal lumen is to be maintained. On removal of the obturator several months later, if the laryngeal lumen stays open satisfactorily, the large tracheal stoma can be closed. Several years ago, it was thought that this large opening required a pedicle flap of skin for closure. However,



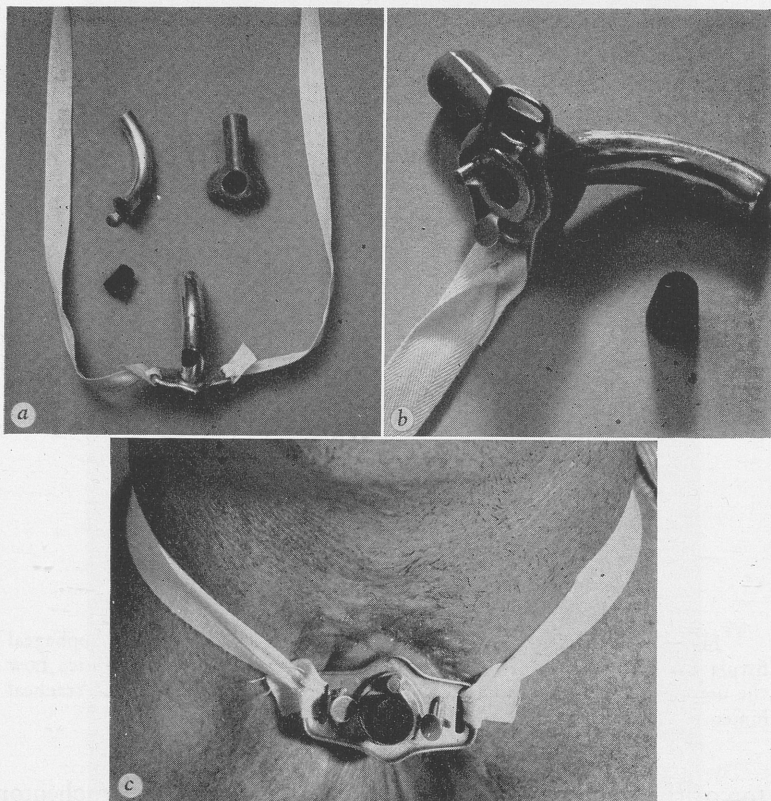


Fig. 7 *a*.—Mechanical appliance used in conjunction with a tracheal cannula to enable the patient to breathe through his mouth and nose and to speak in a normal manner. The acrylic device is shown on the upper right. The inner and outer tracheal cannulae are shown; a hole in the top of each of these cannulae at the site of contact with the acrylic appliance is visible. The rubber cork used to occlude the normal outer opening of the tracheotomy tube is also illustrated. *b*. The appliance has a ringlike extension below, through which the tracheal cannula is inserted. *c*. Appliance and tracheal cannula in proper position.

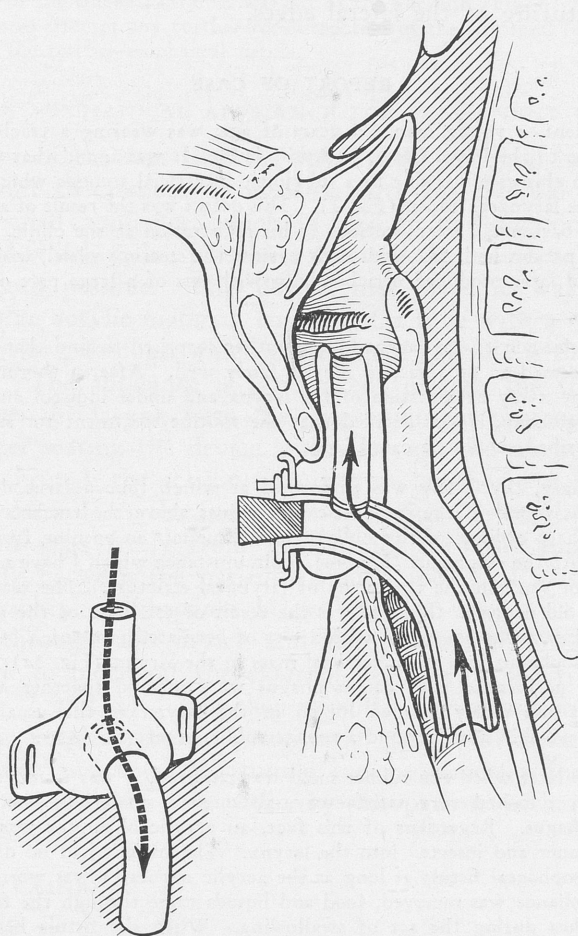


Fig. 8.—Sagittal section of larynx. Tracheotomy tube and acrylic appliance are in place; the arrows indicate the passage of air from the trachea into the larynx when the rubber cork is inserted into the tracheal tube. Relationship of the acrylic appliance to the tracheal tube is illustrated in the inset. When the tracheal tube is corked, the patient breathes normally through the nose and mouth and can speak with no difficulties.

I have found that the tracheal opening will shrink down in size within a few days so that it can be closed by simple approximation, freshening and suturing of the lateral edges.

#### REPORT OF CASE

The patient, a young man, 26 years of age, was wearing a tracheotomy tube when he came to the Mayo Clinic in April, 1953. It was found that the laryngeal obstruction in this case was due to a subglottic cicatricial stenosis which completely obliterated the laryngeal lumen (Fig. 3). This defect was the result of an automobile collision that occurred a year previous to his registration at the clinic. During the accident, the patient had struck his neck against the steering wheel, which fractured his larynx and later produced perichondritis with loss of a large part of the cricoid cartilage.

On examination of the larynx, I was rather surprised to find that the patient's vocal cords seemed to be free and moved fairly well. After a thorough study of this stenosis by x-ray examination of the larynx and under indirect and direct suspension laryngoscopy, it was decided that the routine treatment for stenosis which has been described should be carried out.

Accordingly, thyrotomy was performed at which time a large dense mass of scar tissue was found in the subglottic region just above the tracheotomy opening (Fig. 3a). Sharp excision of this bulk of scar tissue left an opening from the larynx and trachea into the esophagus (Fig. 4a), a circumstance which I have never encountered before or since in the treatment of laryngeal stricture. The reason that this complication did occur in this case was the result of total loss of the posterior part of the cricoid cartilage associated with a sort of herniated protrusion of the anterior wall of the esophagus into the cicatricial mass in the larynx (Fig. 3a). The lateral edges of this perforation into the esophagus were sutured together and skin was grafted about the newly formed lumen in the larynx in the usual manner by applying a shave skin graft around a sponge rubber stent (Fig. 4b).

Ten days later when the rubber mold was removed, it was found that the new laryngeal lumen looked very satisfactory, although a small hole was still present into the esophagus. Regardless of this fact, an acrylic obturator was prepared in the usual manner and inserted into the larynx. The patient had no difficulty with the tracheo-esophageal fistula as long as the acrylic obturator was worn. However, when this appliance was removed, food and liquids came through the fistulous tract into the trachea during the act of swallowing. When the fistula had not healed in a month, I attempted to close this tract with a small flap of mucous membrane which was elevated adjacent to the tracheo-esophageal opening. Unfortunately, the flap did not heal into place satisfactorily because there was too much motion of the esophageal wall during swallowing.

It became imperative that this fistula be closed, and consequently I constructed a tube flap of skin obliquely across the left clavicle. One month later, the lateral end of this tube was elevated and transferred to the laryngeal region (Fig. 5a). All of the laryngeal mucous membrane on the lateral and posterior walls of the larynx in the region of the tracheo-esophageal fistula was excised. This denuded surface, as well as the fistulous opening itself, was covered with the lateral end of the clavicular tube flap. Healing was complete in three weeks after which the unused part of the clavicular tube was cut off and discarded. By this plastic procedure, the

fistulous tract was permanently closed (Figs. 5*b* and 6). However, the use of the pedicle flap of skin within the larynx so reduced the size of the laryngeal lumen that removal of the tracheal cannula was out of the question (Fig. 6). Moreover, it seemed useless to attempt any further reconstruction of the laryngeal lumen for fear of reopening the tracheo-esophageal fistula.

#### THE MECHANICAL APPLIANCE FOR PERMANENT USE

In view of the fact that the constricted laryngeal lumen never would permit the patient to dispense with his tracheal cannula, I devised a small appliance which is used in conjunction with the cannula and which makes breathing through the mouth and nose and normal speech possible. This device, first made in wax and then changed to an acrylic material, consists of a tube with a ring-shaped extension below, through which the tracheal cannula is thrust (Figs. 7 and 8). The appliance is introduced through the tracheotomy opening into the larynx with the ring extension protruding externally. After seating the device in the larynx, the tracheal cannula is inserted through the acrylic ring into the trachea. A hole in the top of the cannula at the site of contact with the acrylic appliance allows air to pass freely from the cannula into the appliance and consequently between the patient's mouth and nasal passages above and the trachea and bronchi below (Figs. 7 and 8). However, breathing through this device and the cannula requires use of a rubber cork to block off the external opening of the tracheotomy tube (Figs. 7 and 8). Air then expelled on expiration passes up between the vocal cords and permits the patient to speak normally. The appliance and tracheal cannula can be removed for cleansing at any time and have been worn by the patient with satisfaction for two years.

MAYO CLINIC

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# USE OF THE LARYNX IN REPAIR OF CICATRICAL STENOSIS OF ESOPHAGEAL INTROITUS:

REPORT OF CASE

JOHN B. ERICH, M.D.

ROCHESTER, MINN.

A cicatricial stenosis of the esophageal introitus usually presents a decidedly challenging problem in therapy. Frequently, correction of such a defect requires laryngectomy, removal of the cicatrix and reconstruction of the upper end of the esophagus. Such management not only becomes extremely involved but not infrequently ends in a repair that is none too satisfactory. In the case which I wish to report in this paper, a complete stricture at the entrance of the esophagus was treated surgically by dividing the trachea from the larynx, cutting across the esophagus just below the stricture and connecting the cut edge of the gullet to the mucosal lining of the larynx. In so doing, the larynx became the upper end of the esophagus and, in this particular case, functioned exceptionally well.

The technique of this operation is somewhat different from that described by Asherson. The patient on whom he reported had undergone pharyngectomy for postcricoid carcinoma; after removal of the thyroid cartilage the laryngeal mucosa was attached to the stump of the esophagus, thereby making a connecting link between the pharynx and the gullet. A recent article by Wilkins outlines a method of extirpating the laryngeal cartilages and using the mucosa of the larynx in reconstruction of the esophagus for patients in whom a malignant neoplasm of the upper end of the esophagus has been excised. In the last-named technique, some difficulty was encountered in keeping the tract open, and dilatations were required.

REPORT OF CASE

The patient was a man, 68 years of age, who came to the Mayo

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From the Section of Plastic Surgery and Laryngology, Mayo Clinic and Mayo Foundation. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

Read before the American Laryngological Association, Montreal, Canada, May 13, 14, 1956.

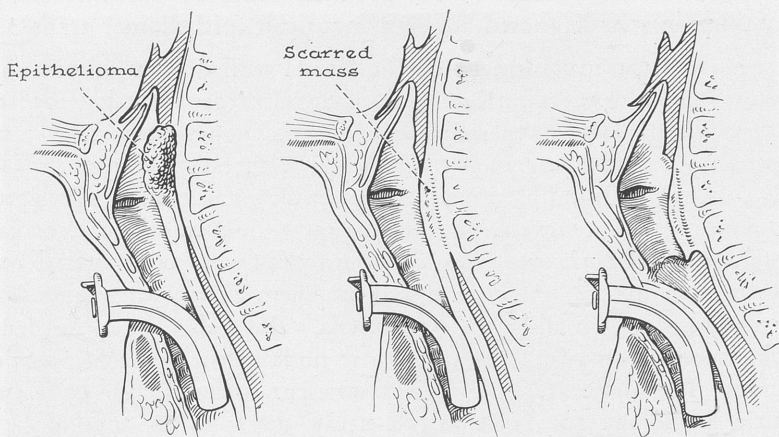


Fig. 1 *a*.—Sagittal section of the larynx. Patient had a huge malignant tumor (squamous cell epithelioma, grade 3) of the hypopharynx arising from a broad base in the right arytenoid region, the right aryepiglottic fold and the vallecula. Tracheotomy has been performed to relieve laryngeal obstruction. *b*. Sagittal section of the larynx after treatment. The epithelioma was treated by fractional x-ray therapy and interstitial irradiation with radon seeds. The treatment completely eradicated the growth but resulted in sequestration of a large part of the cricoid cartilage and almost complete stenosis of the esophageal introitus as illustrated. *c*. Sagittal section of the larynx. Since the vocal cords were fixed in complete abduction with the glottis widely open, the trachea was detached from the lower end of the larynx and brought out through the neck as in the tracheal stoma of a laryngectomy. The esophagus was then opened below the area of stenosis and the cut edge sutured to the lower margin of the laryngeal mucous membrane. By this technique the larynx was transformed into the upper end of the esophagus.

Clinic in August, 1951. On mirror examination of his larynx, I found that he had a huge malignant tumor of the hypopharynx and larynx with a metastatic node in the right midcervical region (Fig. 1 *a*). Due to the size of this growth, the airway through the larynx was markedly obstructed causing noticeable dyspnea. Except for the presence of this tumor, the general physical condition of the patient was satisfactory.

Without further delay, tracheotomy was performed to relieve the shortness of breath. The tracheal cannula was inserted through the second and third tracheal rings. Subsequently, the pharynx and larynx were thoroughly examined under suspension laryngoscopy. The growth was found to be extremely large and pedunculated, arising from a broad base in the right arytenoid region, the right aryepi-

glottic fold and vallecula. A specimen removed for microscopic examination was diagnosed as a squamous cell epithelioma, grade 3.

Due to the involvement of the lateral wall of the pharynx and the presence of a metastatic cervical node, electrocoagulation together with interstitial and external irradiation was thought to offer as good a prognosis as pharyngectomy combined with laryngectomy. With this decision in mind, I proceeded to destroy the tumor by electrocoagulation under suspension laryngoscopy. Eleven radon seeds each worth one millicurie were implanted into the electrocoagulated base, and an attempt was made to distribute them evenly throughout the involved tissue. In addition, seven seeds, valued also at one millicurie each, were inserted into the metastatic node in the right midcervical region. Two days later, the patient was sent to the Section of Therapeutic Radiology for fractionated x-ray therapy. A total dose of 2,250 r was given to each side of the neck over a period of 15 days. Due to the intensity of the treatment, edema in the mucosa of the hypopharynx and larynx developed promptly so that swallowing was impossible. Consequently, a rubber feeding tube was inserted through the nose to the stomach. On completion of the radiation therapy, the patient was permitted to go home with the tracheal cannula and feeding tube in place.

Because of postradiation edema, the patient still was unable to swallow three months after operation. With the passage of another three months, it was evident that the persistent dysphagia in this case was due to perichondritis. As a matter of fact, a large piece of sequestered cricoid cartilage was removed under suspension laryngoscopy at this time. Unfortunately, the perichondritis resulted in complete stenosis of the esophageal introitus (Fig. 1 *b*). In addition, scar tissue in the arytenoid regions pulled the vocal cords widely apart, a complication which later proved to be extremely beneficial. The patient continued to take nourishment through the feeding tube. Eighteen months following the operation, a thorough examination of the patient's throat and neck revealed no recurrence of the carcinoma. Because dysphagia still was complete, I decided that some surgical procedure should be undertaken to correct the stenosis of the esophagus, so that the patient could swallow again.

At first I considered performing laryngectomy, resecting all of the cicatricial tissue producing the stricture and attempting to rebuild the upper end of the esophagus. However, such a reconstruction not only would have been extremely involved but I was fearful

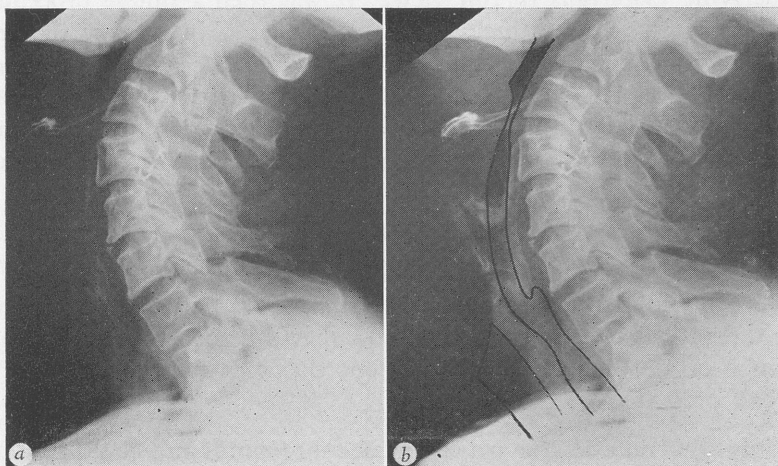


Fig. 2.—Patient whose condition is illustrated in Figure 1. X-rays were taken 4 months after the surgical procedure which attached the larynx to the esophagus as illustrated in Figure 1 *c*. *a*. The lumen through the larynx into the esophagus is visible; also the tracheal lumen which opens externally. *b*. Same as *a* with the tracheal, laryngeal and esophageal lumina outlined for clarity. Since the operative procedure illustrated in Figure 1 *c*, the patient has swallowed satisfactorily through the larynx into the esophagus for the past three and one-half years, and there has been no evidence of recurrence of the laryngeal carcinoma for almost five years.

that healing of the wound might be delayed or unsatisfactory because of radiation changes in the tissues. In view of the fact that the vocal cords were fixed in complete abduction with the glottis widely open, everything which the patient swallowed emptied directly into the larynx. Consequently, I decided to detach the trachea from the lower end of the larynx and to connect the esophagus to the laryngeal mucosa; this would transform the larynx into the upper end of the esophagus (Fig. 1 *c*).

Accordingly, the larynx was opened through a laryngofissure after which the trachea was severed from the larynx by a horizontal incision across the upper end of the windpipe. The trachea was freed downward from the surrounding tissues for a distance of 3 cm. with blunt dissecting scissors. The free edge of the trachea then was sutured to the surrounding skin to make a tracheal stoma similar to that made whenever any laryngectomy is performed. Furthermore,



separation of the trachea from adjoining structures exposed the underlying esophagus to full view. It was then a simple procedure to prepare a horizontal incision in the esophagus just below the area of stenosis. Subsequently, the cut edge of this esophageal incision was sutured to the lower margin of the laryngeal mucous membrane to complete the connection between the esophagus and pharynx. Strap muscles were sutured together in the midline as was the vertical incision in the skin.

Three and one-half weeks after operation, the wound was entirely healed, and the patient could swallow well enough so that the rubber feeding tube could be removed. It was necessary, of course, for the patient to take small bites and chew his food thoroughly, but passage of the bolus of food through the wide-open glottis in the larynx presented no difficulties.

In four months, the patient gained 40 pounds and could swallow any type of well-masticated food. Three years have now elapsed since the larynx was connected to the esophagus. The patient still swallows satisfactorily, and has had no recurrence of the carcinoma (Fig. 2).

#### COMMENT

While this case presents a rather peculiar set of circumstances in that the vocal cords were held widely apart by scar tissue so that food could pass easily through the larynx, I think that the surgical technique employed on this patient might be considered occasionally for other individuals with severe cicatricial stenosis or a carcinoma of the esophageal introitus. If the vocal cords were not abducted, it would be necessary to do submucous resection of tissues forming the vocal cords in order to create a large open glottis through which food could pass. Moreover, excision of that portion of the epiglottis which projects into the pharynx would also be of help in effecting a more direct passage for food from the pharynx into the larynx.

MAYO CLINIC

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## Memorials

## JAMES HARDIE NEIL, M.D.

Dr. James Hardie Neil, corresponding fellow of the American Laryngological Association, nose and throat surgeon, who was widely known for his research work, died January 27, 1955.

He established the knowledge of the tonsil anatomy by private research in 1908 and recently gained international recognition by his work on the anatomy of the lungs.

Following this, he was elected to the Collegium Oto-Rhino-Laryngologicum Amicitiae Sacrum, a distinction shared by only one other member of the British Commonwealth.

Educated at Otago Boys' High School, Dr. Hardie Neil graduated at the University of Otago Medical School in 1898 and gained his M.R.C.S., England, in 1902.

He was appointed to the honorary staff of the Auckland Hospital and served as an ear, nose and throat surgeon for almost 50 years. On his retirement he was appointed honorary consulting ear, nose and throat surgeon to the Auckland Hospital Board, and held a similar position at the Mater Misericordiae Hospital for 20 years.

He was elected a fellow of the American College of Surgeons in 1924 and a member of the American Broncho-Oesophagological Association later. He served as president of the Pan-Pacific Surgical Congress at Honolulu in 1939 and he was a foundation member of the Royal Australian College of Surgeons.

Dr. Hardie Neil's efforts on behalf of the Hard of Hearing resulted in the State subsidizing hearing aids and he was responsible for organizing the Hearing Aid Clinic. To rehabilitate the hard of hearing, he established the New Zealand league, of which he had been president since its inception.

He was a past president of the Auckland branch of the B.M.A. and a foundation member and past president of the Auckland Clinical Society.

Dr. Hardie Neil wrote two textbooks dealing with ear, nose and throat nursing and with field ambulance organization. He was a lecturer in diseases of the ear, nose and throat to students and nurses at the Auckland Hospital.



JAMES HARDIE NEIL

He was closely associated with military affairs throughout his life. He served as a surgeon captain in the Boer War, and as a lieutenant-colonel in the Field Ambulance in World War I in Egypt, Gallipoli, and France. He received several awards.

In World War II he was in charge of Auckland medical boards for six years. He was also officer commanding the 12th Field Ambulance at the Epsom Showgrounds, and was closely associated with the organization and administration of the Emergency Precautions Service.

He was awarded the C.B.E. in 1947.

Dr. Hardie Neil keenly followed a variety of sports.

He is survived by his wife, one son and four daughters.





JOHN G. McLAURIN

## JOHN G. McLAURIN, M.D.

1891-1955

Dr. John Gane McLaurin, an active fellow of the American Laryngological Association, died May 2, 1955. He was born December 8, 1891 and was graduated from Tulane University of Louisiana in 1914. He had retired from the active practice of medicine about three years prior to his death because of ill health.

He practiced Internal Medicine in Dallas, Texas until 1922. After special training in New Orleans and New York, he then limited his work to Ophthalmology and Otolaryngology. In 1946 he further restricted his practice to Otolaryngology.

Dr. McLaurin was on the teaching staff of Baylor University in the Department of Internal Medicine and later in the Department of Otolaryngology. During the first World War, he served overseas as a Captain in the Army Medical Corps.

He was certified by the American Board of Otolaryngology in 1926. He was a member of the American Academy of Ophthalmology and Otolaryngology, American College of Surgeons, Texas Society of Ophthalmology and Otolaryngology, and was an Associate Editor for several years of the Annals of Otolaryngology.

H. L. McLAURIN.

## WILLIAM EMIL SAUER, M.D.

1875-1955

Dr. William Emil Sauer, emeritus fellow of the American Laryngological Association, distinguished service professor of Otolaryngology of St. Louis University School of Medicine, and one of the leading otolaryngologists in the city of St. Louis for over fifty years, died at his home on September 3, 1955, at the age of eighty years.

Dr. Sauer was born and spent the early years of life in Evansville, Illinois. He was the son of Nicholas and Elizabeth Gerlach Sauer. He received his university education at Shurtleff College in Alton, Illinois, and his medical education at Washington University School of Medicine, from which he received his Doctor of Medicine Degree in 1896. This was followed by two years of internship in St. Louis City Hospital. Following this came a period of two years post-graduate study in his chosen specialty in the medical centers of Europe. He returned to St. Louis and began the practice of otolaryngology in 1900. He also began teaching his specialty in the Washington Medical School and was chief of the ear-nose-and-throat clinic of that school from 1905 to 1913. In 1922 Dr. Sauer became chief of the department of otolaryngology and professor of otolaryngology in St. Louis University School of Medicine. He remained in this position until his retirement from active teaching in 1945, at which time he was made Distinguished Service Professor of Otolaryngology of the St. Louis University School of Medicine, in recognition of his long years of devoted service to the Medical School. Dr. Sauer was personally very active in raising funds to construct the University's Firmin Desloge Hospital in 1927, which serves as the principal teaching institution of the Medical School, and he was responsible for establishing the Department of Otolaryngology on a firm foundation in the School.

Dr. Sauer contributed many articles to the Medical literature on a variety of subjects within his specialty. He wrote, among others, papers on various complications of mastoiditis, on fractures of the larynx, an endonasal approach of hypophyseal tumors, on syphilis of the ears, nose and throat, carcinoma of the larynx. He devised a tonsillotome, and brought out a combined intranasal approach to relieve obstruction of the nasolacrimal duct.



WILLIAM EMIL SAUER



Dr. Sauer took an active part in the work of his chosen specialty. He was a member of the American Otological Society, American Laryngological, Rhinological and Otological Society, the Pan-American Medical Society (chairman of the section on otolaryngology in 1935), the American Academy of Ophthalmology and Otolaryngology. He was a member of the honorary medical fraternity, Alpha Omega Alpha. Dr. Sauer was for many years a member of the examining board of the American Board of Otolaryngology.

Dr. Sauer, in spite of heavy demands upon his time from his professional duties, was a devout Christian and took an active role in the Second Presbyterian Church of St. Louis during his entire career in this city. He served as an Elder in his Church for many years, and was a loyal and devoted supporter of the Church. To those of us who knew him well, Dr. Sauer was, in deed, a Christian, in the best sense of the word.

Dr. Sauer was a member of the University Club of St. Louis, of the St. Louis Country Club, of the Masonic Lodge of St. Louis, and of the Ear, Nose and Throat Club of St. Louis. During the First World War he served as a Captain in the Army Medical Corps. Dr. Sauer was on the staffs of the following hospitals in St. Louis: St. Mary's Group of Hospitals (St. Louis University), St. Luke's, DePaul, Missouri Baptist, and Bethesda General Hospital.

Dr. Sauer is survived by his widow, Mrs. Mary Irene (Borders) Sauer and one son, Dr. W. Nicholas Sauer, who continues in the practice of otolaryngology in St. Louis where he had been associated with his father for several years. Dr. Sauer's passing was a real loss to the medical school, to the community and especially to thousands of devoted patients and friends in this area, to whom he had given unselfishly of his skill and time. He was always a skillful and thorough practitioner of his surgical specialty, an enthusiastic teacher of students and younger doctors, and an honorable and true Christian gentleman.

W. B. H.

## H. MARSHALL TAYLOR, M.D.

1881-1955

Dr. H. Marshall Taylor, an active member of the American Laryngological Association, sprang from distinguished lineage and pure American stock. Dr. Taylor was born at Wilmington, N. C., on June 7, 1881. He was the son of a Baptist minister, the Reverend James Barnett Taylor, D.D., and Fannie Callendine Taylor. His father, a native of Richmond, Va., and a graduate of the University of Virginia, served with distinction in J. E. B. Stuart's cavalry during the War Between the States and later taught philosophy at Washington and Lee University. Notable among Dr. Taylor's ancestors were colonial governors of Massachusetts and Connecticut, a signer of the Declaration of Independence, an officer of the Revolutionary War, and a president of Yale College.

Dr. Taylor spent his early life in Lexington, Va., where he lived in the old home of Stonewall Jackson. He received his medical education at Emory University in Atlanta and was awarded the degree of Doctor of Medicine by that institution in 1903. Later he engaged in postgraduate study at the Medical College of Virginia in Richmond. For three years he had special training at the New York Eye and Ear Infirmary, and was associated in practice with the eminent Dr. Cornelius G. Coakley. He also studied otolaryngology in the clinics of Vienna, Munich and Paris. During World War I, he served as a captain in the Medical Corps of the United States Army.

In 1911, Dr. Taylor located in Jacksonville, Fla., where for more than four decades he has continued to engage in the practice of his specialty of otolaryngology. Through the years he has served locally as consulting otolaryngologist to the Duval Medical Center, to Riverside Hospital, and to St. Luke's Hospital where he has served as chief of staff. He is also a member of the staff of St. Vincent's Hospital, Hope Haven Hospital, and Brewster Hospital. In addition, for many years he has been consulting otolaryngologist to the Florida East Coast Hospital and the State School for the Deaf and Blind at St. Augustine. As one of the city's most distinguished citizens and a man of exceptional business acumen and executive ability, he has been and continues to be active in civic affairs and socially in all that promotes the welfare and progress of Jacksonville. For years he served on the Board of Directors of the Atlantic National Bank of Jacksonville. A devout member of the Baptist Church, he has



H. MARSHALL TAYLOR

been most active in bringing to Jacksonville the large Baptist Hospital on which construction began in 1953. He holds membership in the Florida Yacht Club and in the Timuquana Country Club of which he was a charter member. In the care and culture of orange groves he finds an interesting avocation.

Dr. Taylor has also served his adopted state well. He is a former president of the Florida Medical Association and of the Duval County Medical Society. For a number of years he represented Florida in the House of Delegates of the American Medical Association. He was a founder member and first president of the Florida Society of Ophthalmology and Otolaryngology. He has served as head of the Department of Otolaryngology of the Graduate School of Medicine of the University of Florida since its founding and was the founder of its Midwinter Seminar in Ophthalmology and Otolaryngology. Through the years he has been keenly interested in the promotion of welfare legislation in the state. It was at his instigation and through his influence and personal endeavor that Florida became the second state in the Union to pass the lye legislation which has since become widespread.

In Dr. Taylor the Southern Medical Association has had a staunch friend and supporter for some thirty-five years. Doubtless more than any other one physician in Florida he has engaged actively across the years in stimulating interest in the Association and building up its membership in that state. He has served as Secretary and as Chairman of the Section on Ophthalmology and Otolaryngology. He was on the Council for six years, the last two serving as Chairman, and then was elevated to the Association's highest office. It was in 1935 that he served as President. Since that time he has continued to manifest keen interest in the Association and to contribute to its progress.

In addition to serving as a member of the House of Delegates of the American Medical Association for some years, Dr. Taylor in 1938 was chairman of the Section on Laryngology, Otology and Rhinology of that organization. For twelve years he held the chairmanship of its Committee on Otorhinologic Hygiene of Swimming.

Dr. Taylor has long been a Fellow of the American College of Surgeons. In 1950 he was elected to its Board of Governors.

To the national organizations of his specialty Dr. Taylor has given generously of his time and talents. In 1935, while serving as



President of the Southern Medical Association, he was also President of the American Bronchoscopic Society, now known as the American Broncho-esophagological Association. He acceded to the presidency of the American Laryngological, Rhinological and Otological Association in 1944. He became President of the American Laryngological Association in 1952. This society bestowed upon him its highest honors for outstanding achievements—the Casselberry Award in 1939 and the James E. Newcomb Award in 1950. At various times he has served these specialty organizations in numerous official capacities other than the presidency and has been a member of the Educational Board of their official publications.

An indefatigable worker, Dr. Taylor has taken time from his heavy practice to engage in fruitful clinical research and to contribute many articles which have enriched the literature of his specialty. Among his early contributions to scientific progress were studies upon the different types of sand spurs and their deleterious effect upon the membrane of the larynx and lungs. His reports upon the causes and prevention of otologic disease and sinusitis following swimming and diving merited and received wide attention. His motion picture on "The Hygiene of Swimming," demonstrating the potent dangers to the middle ear and sinuses of swimming and diving, had such unique educational value that it has been used extensively in schools and universities throughout the country. Particularly notable also are his articles on prenatal medication in relation to the fetal ear, which have reported from time to time his research in this field in which he has pioneered. He contributed the chapter on "Deafness from Drugs and Chemical Poisons" in Fowler's *Medicine of the Ear* and the chapters on "Congenital Deafness" and "Otorhinologic Hygiene of Swimming" in the *Encyclopedia of Medicine* now in process of publication.

In 1907, Dr. Taylor was married to Miss Pallie Elaine Dekle of Marianna, Fla. They have three children. Their daughter Margaret is Mrs. W. J. Pattison of Santa Monica, Calif. The two sons are Coakley Taylor, a Jacksonville attorney, and Dr. G. Dekle Taylor, who is associated with his father in the practice of otolaryngology in Jacksonville.

While participating on September 11, 1955, in the opening ceremonies of the Baptist Hospital of Jacksonville, for which he had labored arduously, serving in many official capacities, Dr. Taylor was stricken with coronary occlusion and cerebral thrombosis. He died on September 22, 1955.

## SAMUEL J. CROWE, M.D.

1883-1955

Dr. Samuel J. Crowe, an emeritus fellow of the American Laryngological Association and emeritus professor of laryngology and otology at the Johns Hopkins Medical School and an international authority on deafness, died November 13, 1955. He was 72 years old.

He has been hospitalized since October 28 when he suffered a heart attack.

Dr. Crowe was associated with the Hopkins since his graduation in 1908. Prior to retirement in June 1952, he was professor and chief of the ear-nose-throat service, adjunct professor of environmental medicine and otolaryngologist-in-charge at the hospital.

Even after retirement, he had continued his research and teaching. He also traveled extensively to Europe, Africa, Asia Minor and Alaska conducting research for the Air Force.

Dr. Crowe was best known for his development in 1924 of the radon (radium byproduct) treatment for deafness. It proved highly effective during World War II in relieving acrotitis, caused by sudden changes in air pressure, which led to casualties among airmen and submariners.

He also helped found the program for treating children at young ages which became known as the Maryland Program for Conservation of Hearing.

Born in Washington county, Virginia, he was the son of a physician, the late Dr. Walter Andrew Crowe, and Flora Thompson Crowe. He received his bachelor of arts degree from the University of Georgia in 1904.

In 1908 he married Susie Childs Barrow, who survives him, daughter of the former chancellor of the University of Georgia. The couple resided in the Tuscany Apartments on Stony Run lane.

After earning his M.D. here, he was appointed assistant in general surgery to the late Dr. William S. Halsted, first professor and surgeon in chief, and the late Dr. Harvey Cushing, then associate in surgery.



SAMUEL J. CROWE

Dr. Crowe was named director of the ear-nose-throat service in 1912. He also attended graduate medical schools in Berlin, Vienna and Friedburg.

He was cited in 1951 by the Secretary of the Air Force for his contributions to the health of military and civilian occupation personnel in Germany and his work there in teaching and operating clinics.

In recent years he had been honored by the Johns Hopkins with the honorary degree of doctor of science and the presentation of his portrait to be hung in the new building, of the hospital.

Dr. Crowe was the author of many books and articles and just last month completed a history of the development of the department of surgery at the Johns Hopkins under Halsted.

He was a fellow of various medical groups, including the American College of Surgeons, the American Medical Association, the Rhinological and Otological Society, and past president of the American Otological Society.

He was a member of Phi Beta Kappa and the Maryland Club.

Surviving, in addition to his wife, are one son, David, of Asheville, N.C., and five grandchildren. Another son, Samuel James, Jr., is deceased.



## VILRAY PAPIN BLAIR, M.D.

1871-1955

Dr. Vilray P. Blair, corresponding fellow of the American Laryngological Association and Professor Emeritus of Clinical Surgery at Washington University School of Medicine, was born June 15, 1871 and died November 24, 1955. He was one of the pioneers of plastic surgery in the United States. His outstanding achievements in this field brought him many honors.

In World War I he was in charge of plastic and oral surgery for the United States Army, and served as chief consultant in maxillo-facial surgery to the American Expeditionary Force.

Dr. Blair was a native of St. Louis and a descendant of the Papins, one of the original French families here. His parents were Edmund Harrison and Minnie Papin Blair. His grandfather, Timothy P. Papin, two of his maternal uncles and his father-in-law, the late J. B. Johnson, all were physicians. Dr. Johnson had been a classmate of Bigelow.

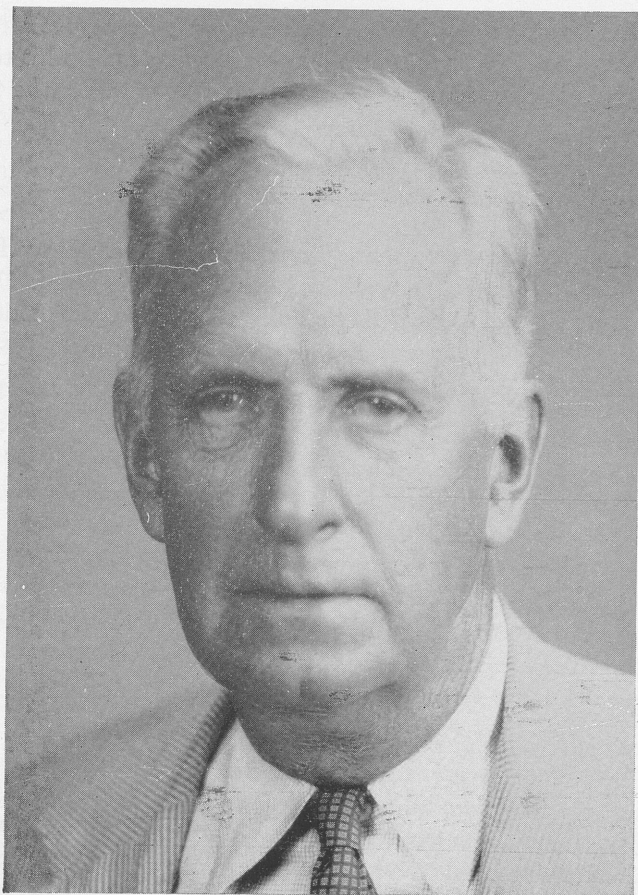
In 1894 he began a teaching career at Washington University, which led from instructor in practical anatomy through half a dozen other positions to professor of clinical surgery in the school of medicine and of oral surgery in the school of dentistry.

When the Boer War broke out in 1899, Dr. Blair obtained a leave of absence from the university to serve as a surgeon in the British merchant marine and as a troop surgeon in Africa.

As a young teacher he had a student who, because of a malformed jaw, seemed destined to go through life severely handicapped. Dr. Blair offered to try corrective surgery. A difficult operation in which sections of jawbone were removed proved successful.

Soon all kinds of bizarre surgical problems were being sent to the surgeon.

Members of the American Association of Oral and Plastic Surgeons met in St. Louis in 1934 to honor Dr. Blair and observe his work. The Mississippi Valley Medical Society awarded him its distinguished service medal in 1938. The St. Louis Medical Society in



VILRAY PAPIN BLAIR

1947 presented him with a gold medal and a certificate of merit for scientific accomplishment.

He was cited as a "sculptor in living flesh" when Washington University conferred on him the honorary degree of Doctor of Science in 1949.

Dr. Blair was the author of textbooks and numerous papers on surgery and diseases of the mouth and jaws. He gave up his teaching in 1942, and retired from active practice in 1946, when he reached the age of 75.

The surgeon married Miss Kathryn Lyman Johnson in 1907. She died in 1950.

He is survived by two sons, John Blair and Dr. Vilray P. Blair, Jr., two daughters, Mrs. George A. Lane and Mrs. James O'Leary; and two sisters, Mrs. Richard Boyle and Miss Marguerite Blair, all of St. Louis.

## JOHN MACKENZIE BROWN, M.D.

1878-1955

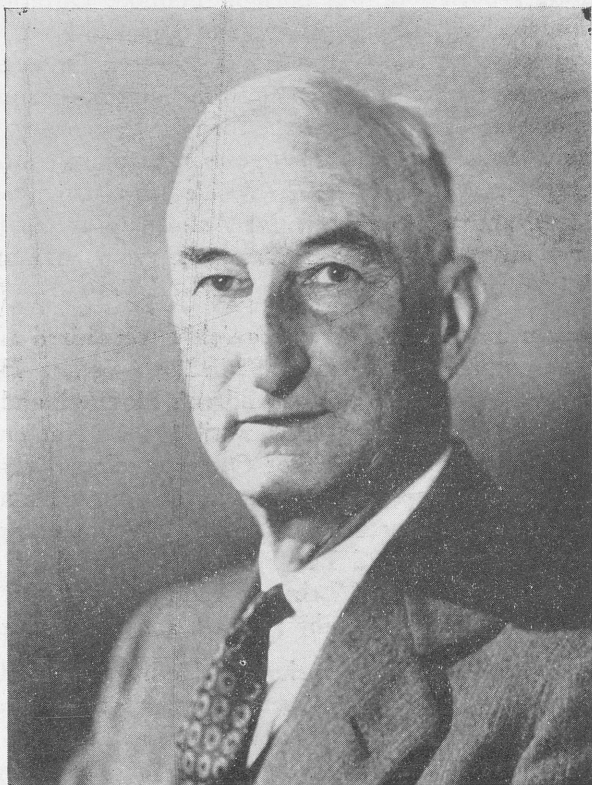
John Mackenzie Brown, an active member of the American Laryngological Association, was born in London, Ontario, Canada, in 1878. He received his preliminary education and was graduated from medical school at the University of Western Ontario in 1899. Shortly thereafter, he decided to come to the Southwest and made his permanent home in Los Angeles at the turn of the century, becoming an American citizen within a few years. Becoming interested in otolaryngology early in his career, he decided to obtain special training in this field and traveled extensively studying at the University of Michigan, University of Pennsylvania, Harvard, Edinburgh, and at several European centers.

His skill in his special field was quickly recognized and he was appointed to a number of hospital staffs in Los Angeles. He was one of the first members of the staff at Children's Hospital, and remained active in that institution from 1909 until 1955. Not only did he serve as Chief of the Department of Otolaryngology, but served as Chief of Staff at that hospital for a number of years. He maintained affiliations with many other hospitals in the city, including St. Vincent's Hospital, the Eye and Ear Hospital, Hospital of the Good Samaritan, and the Cedars of Lebanon Hospital. His services as a consultant were constantly sought at these and other hospitals.

With the re-opening of the School of Medicine at the University of Southern California in 1928, Doctor Brown became vitally interested in the welfare of this institution and became Professor and Head of the Department of Otolaryngology in 1930. Several years later he became Chief of the Department of Otolaryngology at the Los Angeles County General Hospital, and even after his promotion to Professor Emeritus in 1950, he maintained an active interest in the work of both institutions.

The Residency Training Program and the Postgraduate Education in Otolaryngology were greatly stimulated and put on a solid footing during his period of leadership. His Wednesday rounds at 1:00 o'clock became traditional at the County Hospital and were renowned throughout the country for their excellence and stimulation.





JOHN MACKENZIE BROWN

Doctor Brown was deeply interested in the national oto-laryngological societies, and served as an officer in many of them. In addition to serving as President of the American Laryngological, Rhinological and Otological Society, and President of the American Academy of Ophthalmology and Otolaryngology, he was also a member of the Los Angeles Society of Ophthalmology and Otolaryngology, the Pacific Coast Oto-ophthalmological Society, the American Otological Society, and the American Laryngological Society, and Vice-President of Laryngological in 1952. He was a devoted member of all of these organizations and rarely failed to attend a national or local meeting of any society in the field of otolaryngology.

During his many years in the practice of otolaryngology, he contributed much to the advancement of the specialty and his contributions in several fields are world famous. His work on the frontal sinus, including his advocacy of early trephine in acute frontal sinusitis and conservative surgery in chronic frontal sinusitis; his work in branchial-cleft anomalies and his interest in intracranial complications were well recognized throughout the world. He served as Associate Editor of the Archives of Otolaryngology for many years.

But all of these sterling qualities and great accomplishments in the field of medicine do not tell the real story of "J.M.", a truly beloved physician. He will be remembered by many thousands of patients who knew him as a kind and able physician with real devotion to the sick. To his fellow physicians, he was known as an astute diagnostician, and a very able surgeon in the field of otolaryngology. To many otolaryngologists now practicing, not only on the West Coast but in various parts of the country, Doctor Brown will always be the great teacher, the great educator, and the kindly advisor who guided and helped innumerable young men in the specialty of otolaryngology. He was deeply interested in people and in education. He was a strong guiding force in the early days of organization of the School of Medicine at the University of Southern California, and for many years was a member of the executive committee of the faculty. A large group of his former students and friends have for years met at the annual meetings of the American Academy of Ophthalmology and Otolaryngology as the "John Mackenzie Brown Alumni Group," and have awarded an annual recognition to that member of the group who made the best contribution to the literature in that year. This award is considered a great honor. In 1949, a testimonial dinner was given to Doctor Brown by a large number of

otolaryngologists in the Southern California area upon the occasion of his completion of fifty years of practice. At this testimonial dinner, an oil painting was presented to him by his devoted friends, and copies of this painting hang in many otolaryngological offices on the West Coast.

"J.M." was a most humble and modest person who always tried to remain quietly in the background. Nevertheless, his great character and human qualities were so evident that many honors were presented to him during his years in practice.

Remaining actively engaged in practice and in his educational work until the very end, Doctor Brown passed away in his sleep on December 31, 1955. He is survived by his widow, Marian, two daughters, two sons, and six grandchildren.

The John Mackenzie Brown Memorial Fund has been established to perpetuate the name of the man who was known as the dean of otolaryngologists on the West Coast, and who made contributions to the specialty which will be permanent in the archives of the specialty. This fund will be used to build a lecture hall in the new University of Southern California Medical School Center. This will be a fitting memorial to Doctor Brown, who was very anxious to see education in medicine raised to the highest possible level.

A. H. M.

## HAROLD GRANT TOBEY

1884-1955

Harold Grant Tobey, an emeritus fellow of the American Laryngological Association, known to all his friends as "Hal" Tobey, died suddenly and peacefully of cardio-vascular disease on December 26, 1955 at home.

He practiced Otolaryngology to the last day of his life and enjoyed his hunting and fishing to the end.

He is survived by his wife and two daughters and two grandchildren. His wife was Anna Marie Sundberg of Boston to whom he was married in September 1919, and his two daughters are Mrs. A. C. Petite of New York City and Mrs. Sanford L. Williams of Boston.

"Hal" was a son of Dr. and Mrs. George Loring Tobey, Sr. of Lancaster, Massachusetts. He attended, along with his late brother, George Loring Tobey, Jr., the public schools of Lancaster, Massachusetts. He graduated from Bowdoin College in 1906, where he was a tennis and track star. Harvard Medical School gave him a degree in 1911. He then interned at the Rhode Island General Hospital and next specialized in Otolaryngology at the Massachusetts Eye & Ear Infirmary.

Soon after becoming a specialist he went with the first Harvard Unit to join the British Expeditionary Forces and afterwards was a Captain in the United States Army with the Massachusetts General Hospital Base Hospital No. 6 in France.

"Hal" Tobey served on the staffs of many Massachusetts hospitals, chiefly the Massachusetts Eye & Ear Infirmary and the Children's Hospital. He taught at the Harvard Medical School and always loved to teach and help the younger men.

He contributed many scientific papers in his field and was an active member of all our national and local societies.

Above all else, "Hal" Tobey loved life and was beloved by all.

C. I. J.





HAROLD GRANT TOBEY

## Executive Sessions

1956

## FIRST EXECUTIVE SESSION

MAY 13, 1956

The First Executive Session of the Seventy-Seventh Annual Meeting of the American Laryngological Association held at the Seignior Club, Montebello, P.Q., Canada, convened on May 13, 1956, at 12:00 noon, following the First Scientific Session. Dr. Bernard J. McMahon, President, presided.

The following were elected to *Active Fellowship* in the Association:

DR. DANIEL CLIFTON BAKER, JR.

DR. STANTON A. FRIEDBERG

DR. CHARLES M. NORRIS

DR. JOHN R. RICHARDSON

To *Emeritus Fellowship*:

DR. WILLIAM HOWARD JOHNSTON

DR. GABRIEL TUCKER

The Newcomb Award was presented to Dr. Dean M. Lierle for his significant contributions to Laryngology and Rhinology and the American Board of Otolaryngology. The citation of the Newcomb Award Committee stated that "the Newcomb Award is made to one who has contributed significantly to the science and the art of Laryngology and Rhinology. The recipient of this year's award was distinguished for scholarship as an undergraduate student. His intellectual curiosity manifested itself early in his graduate training and has persisted during the past twenty-five years. He has enriched the literature of our specialty by more than fifty contributions and he has been honored by membership in all of the special societies identified with otolaryngology. His name is associated with most of the important advances in the graduate training of his specialty in his generation. The department that he heads in his school of medicine is a modern pattern for graduate training. It seems fitting, there-

fore, that the Newcomb Award for 1956 be made to Dean McAllister Lierle."

The following Fellows were elected to serve as a Nominating Committee:

DR. LEWIS F. MORRISON

DR. LOUIS H. CLERF

DR. SAM ROBERTS

DR. JULIUS W. McCALL

DR. ARTHUR W. PROETZ

There being no further business the session adjourned at 12:20 P. M.

Respectfully submitted,  
HARRY P. SCHENCK,  
*Secretary*



# REPORT OF THE SECRETARY AMERICAN LARYNGOLOGICAL ASSOCIATION

MAY 13, 1956

The Seventy-Sixth Annual Meeting of the American Laryngological Association was held on March 13th and 14th, 1955, at the Hollywood Beach Hotel, Hollywood Beach, Florida, under the Presidency of Dr. Henry Boylan Orton.

There were 75 Fellows and 202 Guests in attendance.

Sixteen scientific papers were presented.

The following were elected:

## To *Emeritus Fellowship*:

DR. E. TRIBLE GATEWOOD, Richmond, Va.

DR. KARL M. HOUSER, Philadelphia, Pa.

## To *Active Fellowship*:

DR. JOHN E. BORDLEY, Baltimore, Md.

DR. LESTER A. BROWN, Atlanta, Ga.

DR. JEROME A. HILGER, St. Paul, Minn.

DR. G. SLAUGHTER FITZ-HUGH, Charlottesville, Va.

The Newcomb Award was presented to Dr. Thomas Cyrus Galloway for his many contributions to Laryngology and Rhinology as related to the treatment of poliomyelitis.

The following Officers were elected:

President..... Dr. Bernard J. McMahon

First Vice-President..... Dr. Robert L. Goodale

Second Vice-President..... Dr. Paul H. Holinger

Secretary..... Dr. Harry P. Schenck

Treasurer ..... Dr. Fred W. Dixon  
 Librarian, Historian and Editor..... Dr. Edwin N. Broyles  
 First Councilor..... Dr. Francis E. LeJeune  
 Second Councilor..... Dr. Louis H. Clerf  
 Third Councilor..... Dr. Gordon F. Harkness  
 Fourth Councilor..... Dr. Henry B. Orton  
 The Association has lost by death during the past year:

*Corresponding Fellow:*

DR. VILRAY P. BLAIR

*Emeritus Fellows:*

DR. SAMUEL J. CROWE

DR. WILLIAM E. SAUER

DR. HAROLD G. TOBEY

*Active Fellows:*

DR. JOHN MACKENZIE BROWN

DR. JOHN G. McLAURIN

DR. H. MARSHALL TAYLOR

The Council at its Annual Meeting in New York City on January 14, 1956, recommended for:

*Emeritus Fellowship:*

DR. WILLIAM H. JOHNSTON, Santa Barbara, Calif.

DR. GABRIEL TUCKER, Philadelphia, Pa.

*Active Fellowship:*

DR. DANIEL C. BAKER, JR., New York, N.Y.

DR. STANTON A. FRIEDBURG, Chicago, Ill.

DR. CHARLES M. NORRIS, Philadelphia, Pa.

DR. JOHN R. RICHARDSON, Boston, Mass.

Dr. Samuel Salinger was reappointed Abstract Editor.

The Council has voted to hold an Annual Session in Washington, D.C., on May 3, 1957. This will consist of a one day meeting and will be followed by the meeting of the American Otological Society on May 4, 1957, and Registration for the Sixth International Congress of Otorhinolaryngology on May 5, 1957. Papers presented by Fellows of the American Laryngological Association at the Sessions of the International Congress will be published in the Transactions of the Association for 1957.

The present membership of the Association is:

|               |    |
|---------------|----|
| Active        | 96 |
| Honorary      | 4  |
| Corresponding | 11 |
| Emeritus      | 18 |

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Respectfully submitted,

HARRY P. SCHENCK,

*Secretary*

## EXHIBIT A

AMERICAN LARYNGOLOGICAL ASSOCIATION  
GENERAL FUND—OPERATING STATEMENT

For Period Ending April 1, 1956

## Income:

|   |            |            |
|---|------------|------------|
| Dues—95 members @ \$40.00 .....         | \$3,800.00 |            |
| Dues—4 new members @ \$100.00 .....     | 400.00     | \$4,200.00 |
| Registration Fees—Hollywood .....       |            | 1,180.00   |
| Sale of <i>Transactions</i> .....       |            | 629.00     |
| Interest Received—Savings Account ..... |            | 143.99     |
| Canadian Exchange on Check .....        |            | .30        |
| Total Operating Income .....            |            | \$6,153.29 |

## Expenses:

## Annual Meeting

|                                   |           |            |
|-----------------------------------|-----------|------------|
| Programs .....                    | \$ 201.93 |            |
| Tickets .....                     | 10.42     |            |
| Secretary's Report .....          | 15.41     |            |
| Projection Equipment .....        | 61.80     |            |
| Guest Speaker (Dr. Oschner) ..... | 114.34    |            |
| Universal Reporting Service ..... | 198.47    |            |
| Banquet .....                     | 687.66    | \$1,290.03 |

## Council Expense

|   |          |        |
|---|----------|--------|
| Annual Dinner .....                                 | \$ 47.70 |        |
| Mid-Winter Meeting (including travel expense) ..... | 655.19   | 702.89 |

## General Expense

|                                     |           |            |
|-------------------------------------|-----------|------------|
| Clerical Hire .....                 | \$ 500.00 |            |
| <i>Transactions</i> (1955) .....    | 2,035.20  |            |
| Stationery, Printing, Postage ..... | 415.41    |            |
| Auditing .....                      | 100.00    |            |
| Treasurer's Bond .....              | 62.50     |            |
| Transfer to de Roaldes Fund .....   | 99.00     | \$3,212.11 |

|                     |  |            |
|---------------------|--|------------|
| Total Expense ..... |  | \$5,205.03 |
|---------------------|--|------------|

|  |  |           |
|--|--|-----------|
| Net Profit to General Fund .....         |  | \$ 948.26 |
| General Fund Balance March 1, 1955 ..... |  | 763.05    |

|  |  |            |
|--|--|------------|
| Total General Fund Balance April 1, 1956 ..... |  | \$1,711.31 |
|--|--|------------|



## EXHIBIT B

## AMERICAN LARYNGOLOGICAL ASSOCIATION

## TRUST FUND

For Period Ending April 1, 1956

## Trust Fund Income:

|  |           |             |
|--|-----------|-------------|
| Interest—Government Bonds .....                | \$ 918.30 |             |
| Transfer—General Fund to de Roaldes Fund ..... | 99.00     |             |
| Total Income .....                             |           | \$ 1,017.30 |

## Trust Fund Expenses:

|   |           |             |
|---|-----------|-------------|
| Transfer Transactions to new editor .....                                   | \$ 218.38 |             |
| (Charged to Jas. E. Newcomb Fund as required)                               |           |             |
| Accrued Interest and Excess Cost of Government Bond<br>over par value ..... | 99.66     | 318.04      |
| Net Trust Fund Addition .....   |           | \$ 699.26   |
| Trust Fund Balance March 1, 1955 .....                                      |           | 37,761.51   |
| Trust Fund Balance April 1, 1956 .....                                      |           | \$38,460.77 |
| Trust Fund Balance represented by Government Bonds, par .....               |           | 37,000.00   |
| Remaining Balance represented by Cash .....                                 |           | \$ 1,460.77 |

## Cash Recapitulation:

|                                  |            |
|----------------------------------|------------|
| Cash in Trust Fund .....         | \$1,460.77 |
| Cash in General Fund .....       | 1,711.31   |
| Total Fund Cash .....            | \$3,172.08 |
| Cash in Savings Account .....    | \$1,715.63 |
| Cash in Commercial Account ..... | 1,456.45   |
| Total Actual Cash .....          | \$3,172.08 |

## EXHIBIT C

## AMERICAN LARYNGOLOGICAL ASSOCIATION

## REPORT OF TRUST FUNDS

April 1, 1956

| Fund                     | Principal   | Unused<br>Income<br>3-1-55 | Income<br>3-1-55 to<br>4-1-56 | Expenses<br>3-1-55 to<br>4-1-56 | Unused<br>Income<br>4-1-56 | Principal<br>and Unused<br>Income 4-1-56 |
|--------------------------|-------------|----------------------------|-------------------------------|---------------------------------|----------------------------|--|
| Casselberry .....        | \$5,000.00  | \$1,109.19                 | \$ 148.56                     | \$ .....                        | \$1,257.75                 | \$ 6,257.75                              |
| Johnson .....            | 5,000.00    | 596.22                     | 136.09                        | .....                           | 732.31                     | 5,732.31                                 |
| de Roaldes .....         | 3,000.00    | 785.74                     | 191.06                        | .....                           | 976.80                     | 3,976.80                                 |
| Newcomb Trust .....      | 2,000.00    | 502.83                     | 60.87                         | 218.38                          | 345.32                     | 2,345.32                                 |
| Newcomb Prize .....      | 500.00      | 27.13                      | 12.82                         | .....                           | 39.95                      | 539.95                                   |
| Hourne Scholarship ..... | 20.23       | 2.69                       | .56                           | .....                           | 3.25                       | 23.48                                    |
| Research .....           | 16,014.19   | 3,203.29                   | 467.34                        | 99.66                           | 3,570.97                   | 19,585.16                                |
| Totals .....             | \$31,534.42 | \$6,227.09                 | \$1,017.30                    | \$318.04                        | \$6,926.35                 | \$38,460.77                              |

## EXHIBIT D

AMERICAN LARYNGOLOGICAL ASSOCIATION  
TRUST FUND INVESTMENTS

April 1, 1956

Government Bonds:

|                     | Description | Par Value   | Current<br>Period<br>Income |
|---------------------|-------------|-------------|-----------------------------|
| U. S. Defense       | 2½ G 1956   | \$ 5,000.00 | \$125.00                    |
|                     | 2½ G 1957   | 1,000.00    | 25.00                       |
|                     | 2½ G 1958   | 2,000.00    | 50.00                       |
| U. S. Savings       | 2½ G 1959   | 5,000.00    | 125.00                      |
|                     | 2½ G 1960   | 1,000.00    | 25.00                       |
|                     | 2½ G 1961   | 1,000.00    | 25.00                       |
|                     | 2.76 K 1965 | 7,000.00    | 193.20                      |
|                     | 2.76 K 1966 | 1,000.00    | 27.60                       |
| U. S. Treasury      | 2¼ 1956-59  | 1,000.00    | 22.50                       |
|                     | 2¼ 1958-63  | 1,000.00    | 27.50                       |
|                     | 2½ 1964-69  | 3,000.00    | 75.00                       |
|                     | 2½ 1967-72  | 1,000.00    | 25.00                       |
|                     | 3¼ 1978-83  | 3,000.00    | 97.50                       |
| (Purchased 1-25-56) | 3 1955-95   | 5,000.00    | 75.00                       |
|                     |             | <hr/>       | <hr/>                       |
|                     |             | \$37,000.00 | \$918.30                    |

April 25, 1956

## ACCOUNTANTS' REPORT

American Laryngological Association

Gentlemen:

We have examined the records of the American Laryngological Association for the period March 1, 1955 to April 1, 1956.

All of the receipts and disbursements since March 1, 1955 were verified by us, the cash balances verified, and the U. S. Government Bonds counted and examined.

In our opinion the accompanying financial statements present accurately the position of the American Laryngological Association as of April 1, 1956 and the results of its operations for the thirteen months then ended.

CHARLES C. BARNES &amp; CO.

Certified Public Accountants

## COMMENT

The foregoing report includes income and expenses of the Association from March 1, 1955 to April 1, 1956. This is the first time in several years that we have had a comfortable margin in the General Fund income. It might be timely to suggest that the income from future donations be used to defray the expenses of the Association. I believe it would be more desirable to use such funds for present day needs rather than for research which requires more money than the Association can supply.

Since the Bank Deposits in the Trust Funds had increased to \$7,000.00, it was decided by the Council to invest \$5,000.00 in 3% Government Bonds. These funds now have an Annual Income of approximately \$1,000.00. This fund, however, cannot be used to defray the expenses of the Association, but is to be used for research.

Respectfully submitted,

FRED W. DIXON, M.D., *Treasurer*

## ANNUAL BUSINESS MEETING

MAY 14, 1956

The Annual Business Meeting held in connection with the Seventy-Seventh Annual Meeting of the American Laryngological Association at the Seignior Club, Montebello, P.Q., Canada, on Monday, May 14, 1956, convened at 9:45 A.M., President Bernard J. McMahon presiding.

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. Edwin N. Broyles, presented his annual report and announced the deaths of the following Fellows since the last Annual Meeting:

Dr. John G. McLaurin, Dallas, Texas, an Active Fellow since 1945. Died on May 2, 1955.

Dr. William E. Sauer, St. Louis, Missouri, an Emeritus Fellow since 1953 and an Active Fellow 1921-1953. Died September 3, 1955.

Dr. Hermon Marshall Taylor, Jacksonville, Florida, an Active Fellow since 1924. Died September 22, 1955.

Dr. Samuel J. Crowe, Baltimore, Maryland, an Emeritus Fellow since 1950 and an Active Fellow 1937-1950. Died November 13, 1955.

Dr. Vilray P. Blair, St. Louis, Missouri, a Corresponding Fellow since 1938. Died November 24, 1955.

Dr. Harold G. Tobey, Boston, Massachusetts, an Emeritus Fellow since 1953 and an Active Fellow 1927-1953. Died December 26, 1955.

Dr. John Mackenzie Brown, Los Angeles, California, an Active Fellow since 1932. Died December 31, 1955.

The Membership rose and stood in silence in honor of the departed Fellows.

Dr. Broyles stated that 200 copies of the Transactions for 1955 had been published at a cost slightly greater than ten dollars per



volume. In presenting the itemized sales of the Transactions, he pointed out that during the past year \$827.00 had been sold of which the sum of \$320.00 was outstanding. It was again emphasized that Transactions of past years may be purchased at a very reasonable price.

The report of the Librarian, Historian and Editor was accepted unanimously.

Dr. Paul H. Holinger reported the death of Dr. James Hardie Neil of Auckland, New Zealand, a Corresponding Fellow since 1950. Further details were not immediately available.

Dr. Arthur W. Proetz, reporting for the Representatives to the American Board of Otolaryngology, stated that at the examination conducted by the Board in Chicago in October 1955, 48 candidates passed, 13 were conditioned and 8 failed. At the examination conducted by the Board in Montreal, Canada, in May 1956, 35 candidates passed, 10 were conditioned and 8 failed. The next examination will be held in Chicago, October 8 to 12, 1956.

Dr. Lawrence W. Boies reported for the Laryngological Research Fund Committee and stated that no requests for funds for research had been granted during the past year but that funds were available for the support of worthy projects.

Dr. Samuel Salinger, reporting for the Representatives to the American College of Surgeons, stated that the annual Clinical Congress of the American College of Surgeons held in Chicago last Oct.-Nov. was very well attended and was productive of much interesting scientific material. It was the first time in many years, however, that there was no program in Otolaryngology. This was due, no doubt, to the fact that the session followed closely on the Academy meeting held only a few weeks before in the same city.

Of interest to us were the figures showing the percentage of representation on the Board of Governors in relation to the percentage of specialists in the various categories. Eye, Ear, Nose and Throat numbering 12.7 per cent of the total membership were represented in 13.5 per cent of the total number of Governors.

Among the recommendations offered by the Governors to the Regents were the following:

1. The Junior Candidate Group which in 1954 constituted only 9 per cent of the surgeons in training should be continued for two

more years and efforts be made through hospital staffs and administrators to acquaint their resident staff with the program. Should the membership in the Candidate group drop below one-fifth of the total number of residents in training the entire program should be abandoned.

2. The Board of Regents should be increased to 19 by 1958 and should include continuous representation from each of the specialties. At the present time Dr. Dean Lierle is our representative.

3. The College through its Committee on Cancer can further the aims of the American Cancer Society and the National Cancer Institute by engaging more actively in the educational field. The Board of Regents was asked to define more specifically the scope of such activities for the guidance of the Committee.

4. To assist the Joint Commission on accreditation of hospitals it was recommended that the joint inspecting groups be requested to contact the chairman of the local council for such information as he may be able to gather which would permit a more thorough and intelligent appraisal of the hospital. An appointed representative of the College would be more free to communicate irregularities than the staff's own representative who is too close to the picture.

Dissatisfaction in this respect has already been expressed by other groups. The A.M.A., for instance, stated its desire to control and supervise the determination of requirements for hospital accreditation and to assume greater responsibility in the enforcement of standards.

It was recommended that an annual forum be established to be conducted by representatives of the College with free discussion of problems both from the standpoint of the committee members and the fellows of the College.

In addition to myself, the following Otolaryngologists attended the sessions of the Board of Governors: Dr. Henry Goodyear, Dr. Arthur C. Jones, Dr. Carl McCaskey, Dr. Robert S. Martin.

Respectfully submitted.

PRESIDENT McMAHON: Thank you, Dr. Salinger.

You have heard the report. Are there any comments?

DR. HOOPLE: I move that it be accepted.

DR. HILL: I will second that.

PRESIDENT MCMAHON: All in favor signify by saying "aye"; contrary "no". It is carried.

Dr. Schenck has a couple of announcements to make.

SECRETARY SCHENCK: At the annual meeting of the Council of this Association in New York in January, it was moved, seconded and passed that the annual dues be increased from \$40 to \$50 and that this recommendation be presented for the consideration of the fellows at this annual meeting.

It might be pointed out that the Otological Society has now increased their dues from \$50 to \$60 per annum.

PRESIDENT MCMAHON: You have heard the report of the recommendation of the Council in regard to the increase of the annual dues. Are there any comments?

DR. HILL: I would like to move the acceptance of that recommendation.

DR. BOIES: I second that.

DR. LE JEUNE: I second that.

PRESIDENT MCMAHON: It has been moved and seconded that this recommendation be accepted. Is there any discussion of this motion of Dr. Hill? This is an important step. Just shortly after this was announced, the intention, our Treasurer got busy and got a whole lot of black ink and wrote big figures on his report and he was not very encouraging which surprised me because he likes to get a little more money. But that is the way it stands.

DR. DIXON: The amount of money that we made last year over our expenses was \$42, the year before \$24. But the year before this last year we gave \$500 to the International Congress. The year before it had cost us \$400 more to print the ten year index so that in both years we made less than \$100 for the total of two years. This year after giving the \$500 to the International Congress, which we decided on the night before, we don't have much money in the treasury. We are an old society, rather with genteel poverty, and if we could get just a little more I think it would be highly desirable, because this International Congress that is coming on may make it necessary that we might have to spend a little more. I hate to raise the dues. I fought against it since I have been Treasurer, but it seems to me that we just cannot escape it.

The dinner last night cost us about \$13. In Hollywood last year \$14. We charged \$10 for the registration fee and we just about struggle but if there is an extra expense coming in, then we are in the red and we have no place to borrow any money. If we could raise the dues, I suppose it would be the desirable thing although I hate to do it. I would like to put it back where it was. I see no alternative if we are going to have to contribute to these various projects which seems desirable.

I would like to have discussion from the floor. I think it is timely and surely it is whatever your wish would be.

PRESIDENT McMAHON: Are there any other remarks?

DR. MORRISON: I would like further definition of "genteel poverty."

PRESIDENT McMAHON: I was just looking around here.

DR. MORRISON: It is universal.

PRESIDENT McMAHON: That is the whole thing, for the sake of the income tax collector. Both these words are complimentary.

DR. HOOPLE: Mr. President, I would like to suggest an amendment and the amendment is as follows, that the annual dues of this Association be \$60. That will eliminate collecting \$10 registration fee at the time of the meeting. It is much simpler for all of us to do it that way income tax-wise. We pay our dues to this Association \$60. We come to the meeting. We think we get a dinner free. Of course we don't and the Association makes in that way, makes a little bit of money because all of the 99 members do not come and we get a few extra dollars that way, in case the dinner cost \$15 instead of \$13.

I move that as an amendment to the motion.

DR. CLERF: I should like to second that amendment and support Dr. Hoople's reason for it. I have felt over the years that it is an error that has crept in somewhere along the line that we who attend this meeting must pay for the holding of the annual meeting. It is not merely the shareholders who attend the meeting who should pay for it. All the shareholders participate equally. If they wish to come that is their prerogative. If they don't, nevertheless they must contribute to the support of the meeting.

PRESIDENT McMAHON: Thank you, Dr. Hoople and Dr. Clerf. I will ask Dr. Hill if he will accept the amendment.



DR. HILL: With the greatest of pleasure.

PRESIDENT McMAHON: The seconder of your motion?

DR. BOIES: Yes.

PRESIDENT McMAHON: You have heard the amendment to Dr. Hill's motion. Is there any further comment? You understand it I hope and I think maybe this may be a greater incentive to attend the meetings to get your extra ten dollars' worth. If there is no further comment I will ask those in favor of this motion to signify by saying "aye"; contrary "no". It is so ordered.

DR. BROYLES: That includes registration and the banquet?

PRESIDENT McMAHON: That includes the banquet dinner that you don't pay for at the time. Those in favor of this motion of Dr. Hill say "aye"; contrary "no". It is so ordered.

SECRETARY SCHENCK: The next annual meeting of this Association will have to be held in Washington, D.C., May 3rd. This meeting will be followed by a one-day meeting of the Otological Society. Our meeting is on Friday. The Otological is on Saturday and Sunday morning the registration for the International Congress will begin.

The following year we meet in San Francisco at the Fairmount Hotel. The Otological and Laryngological Societies will be at the Fairmount. The Triological will meet across the street at the Mark Hopkins Hotel. Is it appropriate at this time to mention anything about the room situation in Washington?

DR. HOLINGER: Do you want me to do it?

Mr. Chairman, Members of the Association, the plans for the International Congress are proceeding I think very smoothly. I am extremely encouraged by the support, by the interest that is arising everywhere, the realization that this is a tremendous international function, not only of scientific value but certainly to improve international relations. We could not have a better group to talk to and to be with. I believe that people throughout the country are realizing this fact. The support financially and scientifically is more and more encouraging as time goes on.

Financially, of course, a Congress such as this requires a tremendous amount of help. Everybody has ideas. We want ideas but we

also need the checks as you know. The State Department, the various drug houses, the instrument houses, individual funds and corporations have been approached or are on the list for being approached so that the activities along this line I think are great.

The scientific program is increasing. Dr. Boies has the reports of requests for places on the program. There are some 125 to 150 of them from the areas in Europe, in Japan, South America, in Africa, Australia. They are sending in their requests for places on the program and it truly has an international flavor.

You all know the general setup of the program with the plenary sessions and the sessions throughout the afternoons for the various individual meetings, five sessions running simultaneously on the afternoons and the two mornings on which the plenary sessions are not being held. In addition there will be we hope the Armed Forces are going to have a colored television program piped from Walter Reed Hospital, the Armed Forces Institute of Pathology and some of the other areas.

Dr. Alfaro in his very thorough way has made his arrangements for the various trips in the afternoon to visit these areas, such as the Walter Reed and the Armed Forces Institute, Bethesda, the Public Health Institute. So that the progress along this line is very, very encouraging.

In addition the post-Congress tours are beginning to shape up and the various areas that are being visited, individuals in those cities are helping in presenting programs for their various cities.

Baltimore will be descended upon by a host and the Baltimore group, Dr. Bordley and Dr. Broyles, have already made wonderful progress in a reception there, both scientific and social. The groups will go on to Philadelphia, St. Louis, Chicago and then to the West Coast, Boston and other areas en route, which means that there is a national activity along this line and again these will be post-Congress-wise both scientific and social events that will be participated in, not only by the foreign group but by many of those here in the United States as well.

In regard to the room situation, which was the thing that gave me the lever to get up here, the requests for rooms will be handled by the Convention Bureau in Washington. We have a good many rooms assigned to us and we have gone into that very thoroughly with the

Convention Bureau. However, they say it is necessary for them to handle it totally if they are going to handle it at all and this, as you can realize, is part of the work that is probably most complicated and most difficult to arrange satisfactorily. The reason for that is that unfortunately many people don't read their mail and they will arrive, in spite of the fact that they have had letters requesting, asking whether they want reservations, want to put in requests for a place on the program, and say, "Here I am. I would like to have a room." That is a tragedy that we want to get around in some way or other and avoid. The system that the convention bureau works out is that on certain prescribed dates they will send letters to you, to those who register for the Congress asking you to fill out the enclosed form and asking you to return it to them immediately. Those will be handled in the manner in which they are returned. We cannot get them before they send them out. We cannot send them to you before they send them out but they will be sent out in adequate time for you to get your reservations and we will simply ask you to be sure when your letter comes that you do fill it out and return it because if you wait a month and then return it there will be an additional group probably that will have been sent out and therefore the reservations may be lost. So it is important that this material be returned as soon as you get it.

There are a number who have apparently indicated that they have not received the general announcement with the request form to be returned for registration. I can assure you that these forms have gone to every member of the Laryngological yet there are many that told me they did not receive them. They went out in the regular mail. They have not been returned to our office, although a return address was there, so we know they were delivered. Under these circumstances should anyone still feel that he has not received one, please let me know in order that we can send you a second one or in some way or other get it to you. We will have some at the meeting in Montreal so that anyone who wants to pick up another one can do so.

In addition besides the registration for the Congress, the \$25 fee, there will be the registration of wives or those that accompany the members, the wives or a reasonable facsimile thereof. So those that accompany members also will be asked to register at a charge of \$10. This is customary in International Congresses. Many have returned the registration blanks. Many have not returned the blanks indicat-

ing that their wives are to accompany them and requesting that they be registered for the additional \$10. So that those of you who have not so done, we would appreciate having you do so if you wish to do that.

In addition I do appreciate the opportunity to again present some of these things to you. It is not that we are presenting them to you but it is a report because of course this is your Congress that you have asked us to put on and we need your support, continued both financially and scientifically and eventually at the Congress from the social standpoint as well.

Thank you.

PRESIDENT McMAHON: Thank you, Dr. Holinger!

I am sure that we all appreciate the importance of this coming meeting and the tremendous amount of effort that is being put into the organization, especially the importance of the financial side of the meeting.

Dr. Schenck, will you make another announcement please?

SECRETARY SCHENCK: It might be well to repeat the action of the Council two years ago, that the papers of the one-day session in Washington of the scientific session of our Association will of course be printed in the Transactions, but in addition all papers presented at the International Congress by our members will also be included in the Transactions. This, of course, is necessitated by the fact that we have cut our meeting to one day and there would not be enough papers in one day to make an adequate issue of the Transactions.

The arrangements for 1959 have not yet been made but there is a possibility that the Homestead at Hot Springs, Virginia, will be selected for that meeting.

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There being no further business the meeting adjourned at 10:30 A.M.

Respectfully submitted,

HARRY P. SCHENCK,

*Secretary*



## FINAL EXECUTIVE SESSION

MONDAY AFTERNOON, MAY 14, 1956

The meeting was called to order at 3:30 P.M., Dr. Bernard J. McMahon, President, presiding.

PRESIDENT MCMAHON: Gentlemen, the first order of business is the election of the Board of Directors. That is the report of the Nominating Committee:

LeRoy Schall  
Henry M. Goodyear  
Robert E. Priest  
Harry P. Schenck  
Fred W. Dixon  
Edwin N. Broyles  
Louis H. Clerf  
Gordon F. Harkness  
Henry B. Orton  
Bernard J. McMahon

. . . Dr. Maxwell assumed the Chair . . .

CHAIRMAN MAXWELL: Gentlemen, what is your pleasure with regard to this slate for Board of Directors?

DR. ROBB: I move it be accepted and elected as the Board of Directors for the ensuing year.

DR. SALINGER: I second that.

CHAIRMAN MAXWELL: All those in favor of electing these as members of the Board of Directors of the Laryngological Society for the ensuing year signify by saying "aye"; contrary "no". It is carried. Will this Board of Directors please meet in the corner and elect the respective officers which would include the President; First Vice-President; Second Vice-President; the Secretary; the Treasurer; the Librarian, Historian and Editor, and the four officers of the Council. Gentlemen, we breathlessly await their decision.

. . . President McMahon resumed the Chair . . .

PRESIDENT MCMAHON: The Secretary, Dr. Schenck, will write down the decision of the Board of Directors.

## SECRETARY SCHENCK:

|                                      |                    |
|--------------------------------------|--------------------|
| President .....                      | LeRoy Schall       |
| First Vice-President.....            | Henry M. Goodyear  |
| Second Vice-President.....           | Robert E. Priest   |
| Secretary.....                       | Harry P. Schenck   |
| Treasurer.....                       | Fred W. Dixon      |
| Librarian, Historian and Editor..... | Edwin N. Broyles   |
| First Councilor.....                 | Louis H. Clerf     |
| Second Councilor.....                | Gordon F. Harkness |
| Third Councilor.....                 | Henry B. Orton     |
| Fourth Councilor.....                | Bernard J. McMahon |

DR. HILL: I move the report be accepted.

DR. ROBB: I second that.

PAST PRESIDENT MCMAHON: All in favor of accepting the report say "aye"; contrary "no". It is so ordered.

Will someone please conduct Dr. Schall to the rostrum?

DR. HILL: Mr. President, Dr. Schall.

PAST PRESIDENT MCMAHON: I wish you the best of luck. I can assure you this is the finest decision the American Laryngological has ever made. As I tender to you this scepter of office, it is one of the most prized possessions I have been allowed to keep in my possession for one year.

. . . President Schall assumed the Chair . . .

PRESIDENT SCHALL: Dr. McMahon, Members of the Association, this is the proudest day of my life. Words fail me to express to you my humility and my gratitude in the desire to conduct the office of President of this Association, at least half as good as my predecessor, Dr. McMahon.

I thank you very much for the honor you have conferred upon me and I will do my best to serve you faithfully and well. I can say no more.

PAST PRESIDENT MCMAHON: Mr. President and Members, I want to thank you again for your kindly interest and acceptance of the program which we tried to put together, and thank you for your cooperation, not only for the presenters of the papers but also for the cooperation of the discussers in preparing such marvelous discussions, and also for your spirit of camaraderie in entering into this entire proceedings of our meeting here. I have never enjoyed a meeting as much as I have enjoyed this nor am I as proud at having been permitted to act as your President.

Thank you very much!

PRESIDENT SCHALL: Mr. Secretary, is there any further business to come before the Association? If there is not any further business, the Chair will entertain a motion to adjourn.

DR. ROBB: I so move.

PRESIDENT SCHALL: All in favor of that motion will say "aye"; contrary "no". The motion is carried and the meeting is adjourned.

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There being no further business the Seventy-Seventh Annual Meeting of the Association was adjourned at 3:35 P.M.

Respectfully submitted,

HARRY P. SCHENCK,

*Secretary*

## List of Fellows

1956



## 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, '56.
1956. BAKER, DANIEL CLIFTON, JR., 903 Park Avenue, New York 21, N. Y., 1956.
1936. BALLENGER, HOWARD C., 723 Elm Street, Winnetka, Illinois, 1936, '37, '38\*, '40, '41, '46\*, '47, '48, '51, '52\*, '53, '54, '55, '56.
1949. BECK, AUGUST LEO, 55 Pell Place, New Rochelle, N. Y., 1949, '50, '51, '52\*, '54, '55.
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., University Hospital, Minneapolis 14, Minnesota, 1944, '46, '47, '48\*, '49, '50\*, '51\*, '53, '54, '55, '56.
1955. BORDLEY, JOHN E., Johns Hopkins Hospital, Baltimore 5, Maryland, 1955, '56.
1941. BOWERS, WESLEY C., 2 East 54th Street, New York 22, N. Y., 1941, '42, '44, '49.
1951. BOYDEN, GUY LEE, 1735 N. Wheeler St., Portland 12, Ore., 1952\*, '53, '54.
1955. BROWN, LESTER ALEXANDER, 490 Peachtree Street, N.E., Atlanta 8, Georgia, 1955, '56.
1940. BROYLES, EDWIN N., 1100 North Charles Street, Baltimore 1, Maryland, 1940, '41\*, '42, '46, '48\*, '49, '50, '51\*, '52, '53, '54, '55, '56.

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, '55, '56.
1945. CAMPBELL, COLONEL PAUL A., USAF (MC), Office of Air Attache Box 36, F.P.O. 100, New York, N. Y., 1946, '47, '49, '51.
1942. CANFIELD, NORTON, 175 St. Ronan St., New Haven 11, Connecticut, 1942, '46, '47, '48, '49\*, '50, '51, '56.
1925. CLERF, LOUIS H., ~~5575~~ Eighth Avenue, North, St. Petersburg 2, Florida, 1925, '27\*, '28, '29, '30\*, '32, '33, '34, '34, '36\*, '37, '38, '39, '40, '41\*, '42\*, '44\*, '46, '47, '48, '49, '50, '51, '52, '53\*, '54, '55\*, '56.
1939. CODY, CLAUDE CARR, 1304 Walker Avenue, Houston 2, Texas, 1939, '41, '42, '44, '46\*, '47, '48\*, '49, '50, '51, '52, '53, '54, '55.
1941. CUNNING, DANIEL S., 115 East 65th Street, New York 21, N. Y., 1941, '42\*, '44, '46, '47\*, '48, '49, '50\*, '51, '52, '53, '54, '55\*, '56.
1951. DAVISON, FRANCIS W., Geisinger Memorial Hospital, Danville, Pa., 1951, '52, '53\*, '54, '55, '56.
1941. DIXON, FRED W., 1027 Rose Building, Cleveland, Ohio, 1941, '42\*, '44, '46, '47\*, '49\*, '50, '51, '52, '53, '54, '55, '56.
1947. EAGLE, WATT WEEMS, Duke Hospital, Durham, N. C., 1948\*, '49, '51, '53, '54, '56.
1947. ENGLER, CLARENCE W., 2323 Prospect Avenue, Cleveland, Ohio, 1947, '48\*, '49, '50, '51, '54, '55, '56.
1954. EQUEN, MURDOCK S., 2505 Habersham Road N. W., Atlanta, Ga., 1954, '55.
1952. ERICH, JOHN B., Mayo Clinic, 102 Second Avenue, Rochester, Minn., 1953, '56\*.
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\*.

1955. FITZ-HUGH, GLASSSELL SLAUGHTER, 2003 Spottswood Road, Charlottesville, Virginia, 1955.
1956. FRIEDBURG, STANTON A., 5730 South Kenwood Avenue, Chicago, Illinois, 1956.
1932. FURSTENBERG, ALBERT CARL, Out-Patient Bldg., University Hospital, Ann Arbor, Michigan, 1933\*, '36\*, '38\*, '46, '48\*, '50, '51, '53, '54, '55\*, '56.
1936. GALLOWAY, THOMAS CYRUS, 636 Church Street, Evanston, Illinois, 1936, '37, '38\*, '39, '40, '41, '42\*, '46\*, '47, '48, '50, '51, '52, '53, '54, '55, '56.
1940. GOODALE, ROBERT L., 330 Dartmouth Street, Boston, Massachusetts, 1940, '41, '42\*, '44\*, '46, '47, '49, '50\*, '53, '54, '55\*, '56.
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, '55.
1950. HALL, COLBY, 1136 West Sixth Street, Los Angeles 14, Calif., 1950.
1940. HANSEL, FRENCH K., 634 North Grand Boulevard, 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\*, '55, '56.
1944. HAVENS, FRED Z., 102 Second Avenue S-W., Rochester, Minnesota, 1944, '46, '49, '51, '53, '54, '55\*, '56\*.
1942. HEATLY, CLYDE A., 11 North Goodman Street, Rochester 7, N. Y., 1942, '46\*, '49, '50, '52, '53\*, '54, '55.
1943. HILDING, ANDERSON C., Research Laboratory, St. Luke's Hospital, Duluth 11, Minnesota, 1944\*, '46, '47, '48\*, '49, '50, '51, '53, '55, '56\*.
1955. HILGER, JEROME A., 350 St. Peter Street, St. Paul 2, Minnesota, 1955, '56\*.

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, '55, '56.
1945. HOLINGER, PAUL H., 700 North Michigan Ave., Chicago 11, Illinois, 1946, '47, '49, '50\*, '51\*, '52, '53, '54\*, '55, '56.
1944. HOOPLE, GORDON D., 1100 East Genesee St., Syracuse 10, New York, 1946, '47, '48, '49, '50, '51, '52, '53, '54\*, '55, '56.
1952. IRELAND, P. E., Room 82, Banting Institute, University of Toronto, Toronto, Ontario, 1952, '53, '54, '55, '56\*.
1938. JACKSON, CHEVALIER LAWRENCE, 3401 North Broad Street, Philadelphia 40, Pa., 1938, '39, '40, '41\*, '42, '44\*, '46, '47\*, '48, '49, '51, '53, '54, '55, '56.
1942. KELLY, JOSEPH D., 140 East Fifty-fourth Street, New York 22, N. Y., 1942, '44\*, '47, '48, '49, '51, '53, '54, '55.
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, '55, '56.
1953. LEDERER, FRANCIS LOEFFLER, 1853 W. Polk Street, Chicago 12, Illinois, 1953, '54, '55, '56.
1936. LEJEUNE, FRANCIS ERNEST, Ochsner Clinic Building, Prytania and Aline Streets, New Orleans 15, Louisiana, 1937, '46\*, '47, '50, '51, '52, '53, '54\*, '55, '56.
1933. LIERLE, DEAN M., University Hospital, Iowa City, Iowa, 1933, '34, '35\*, '37, '38, '39, '40\*, '42\*, '44\*, '46, '47\*, '48, '49, '50\*, '51, '52, '53, '54, '55\*, '56.
1949. LINDSAY, JOHN R., 950 E. 59th Street, Chicago 37, Illinois, 1949, '50, '51\*, '53, '55\*, '56.
1939. LUPTON, IRVING MARIN, 806 Medical Arts Building, Portland, Oregon, 1946, '50.
1952. LYNCH, MERCER G., 6262 Prytania Street, New Orleans 18, Louisiana, 1953, '54, '55\*.



1941. MARTIN, ROBERT C., 384 Post Street, San Francisco 8, California, 1941, '44, '47, '48, '50, '51, '53, '54, '55, '56.
1948. MAXWELL, JAMES H., Out-Patient Bldg., University Hospital, Ann Arbor, Michigan, 1948, '49, '50\*, '51\*, '52, '53, '54, '55, '56.
1952. MCCALL, JULIUS W., 610 Rose Building, Cleveland 15, Ohio, 1952, '53\*, '54, '55, '56.
1951. MCCART, HOWARD W. D., Medical Arts Bldg., Toronto, Canada, 1951, '52\*, '54, '55, '56.
1939. MCCASKEY, CARL HEBER, 608 Guaranty Building, Indianapolis 4, Indiana, 1939, '40, '41, '42\*, '44, '46\*, '47, '48, '49, '50, '51, '52, '53, '55, '56.
1951. MCHENRY, LAWRENCE C., 604 Medical Arts Bldg., Oklahoma City, Okla., 1953\*.
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, '55\*, '56\*.
1931. McNALLY, WILLIAM JAMES, 1509 Sherbrooke Street West, Montreal, Canada, 1931, '33, '34\*, '39, '44, '47, '49, '49, '52, '53, '54\*, '55, '56.
1954. MELTZER, PHILIP E., 285 Commonwealth Ave., Boston, Mass., 1954\*, '56.
1952. MILLER, ALDEN H., 500 South Lucas Ave., Los Angeles 17, Calif., 1952, '53\*, '55.
1954. MOORE, PAUL M., 10,515 Carnegie Avenue, Cleveland, Ohio, 1954, '55\*, '56.
1950. MORRISON, LEWIS F., 3536 Jackson Street, San Francisco 18, California, 1950, '51, '52\*, '53, '54, '55\*, '56.
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, '55, '56\*.

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, '55, '56\*.
1956. NORRIS, CHARLES MORGAN, 3401 North Broad Street, Philadelphia 40, Pennsylvania, 1956.
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, '55\*, '56.
1948. PHELPS, KENNETH A., 1137 Medical Arts Building, Minneapolis 2, Minnesota, 1948, '50, '53\*, '55.
1954. PRESSMAN, JOEL J., 10,911 Weyborn Avenue, Los Angeles 24, Calif., 1954, '56\*.
1951. PRIEST, ROBERT E., 79, S. Ninth St., Minneapolis 2, Minn., 1951, '53\*, '54\*, '56.
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, '55, '56\*.
1951. PUTNEY, F. JOHNSON, 1719 Rittenhouse Square, Philadelphia 3, Pa., 1951, '52, '53\*, '54, '55, '56.
1951. RAWLINS, AUBREY G., 384 Post Street, San Francisco, Calif., 1951, '53\*, '56.
1934. RICHARDS, LYMAN GILDER, Massachusetts Institute of Technology, Cambridge 39, Mass., 1934, '35, '36\*, '37, '38, '39, '47, '49, '51\*, '54, '56.
1956. RICHARDSON, JOHN ROBERT, 403 Commonwealth Avenue, Boston 15, Massachusetts.
1945. ROBB, JAMES M., 633 David Whitney Building, Detroit 26, Michigan, 1946, '48, '49\*, '51, '52, '55, '56.

1953. ROBERTS, SAM E., 5964 Overhill Road, Kansas City, Missouri, 1953, '54\*, '55, '56.
1938. SALINGER, SAMUEL, 1260 Alejo Road, Palm Springs, California, 1938, '39, '40\*, '42, '44\*, '47, '48, '49, '50, '51, '52\*, '53, '54, '56\*.
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, '55, '56.
1934. SCHENCK, HARRY P., 326 South 19th Street, Philadelphia 3, Pennsylvania, 1934, '35, '36\*, '37, '38, '39, '40, '41\*, '44\*, '46, '47, '49, '50, '51, '52, '53, '54, '55, '56.
1939. SIMPSON, W. LIKELY, 512 Physicians' and Surgeons' Building, Memphis 3, Tennessee, 1939, '41, '44\*, '46, '47\*, '48, '50, '51\*, '52, '53, '54, '55.
1950. SMITH, AUSTIN T., 1830 Spruce St., Philadelphia 3, Pa., 1950, '51, '52, '53, '55, '56.
1954. SOOY, FRANCIS A., 490 Post Street, San Francisco 2, Calif., 1954, '56\*.
1947. THEOBALD, WALTER H., 307 N. Michigan Avenue, Chicago, Illinois, 1947, '48\*, '49, '50, '51, '52, '53, '54, '55, '56.
1954. THORNELL, WILLIAM C., 2508 Auburn Avenue, Cincinnati 19, Ohio, 1955\*.
1950. TREMBLE, G. EDWARD, 1390 Sherbrooke St. W., Montreal, Quebec, Canada, 1950, '51, '52, '53, '54, '55\*, '56.
1943. VAN ALYEA, OLIVER E., 135 South LaSalle Street, Chicago 3, Illinois, 1944, '46\*, '47, '48, '50, '51, '52\*, '53, '54, '55, '56\*.
1941. VIOLÉ, PIERRE P., 1930 Wilshire Boulevard, Los Angeles 5, California, 1944\*, '46, '47, '50, '52, '53, '55.
1954. WALSH, THEODORE E., 640 S. Kingshighway Blvd., St. Louis 10, Missouri, 1954, '55\*, '56.
1940. WEILLE, FRANCIS L., 247 Commonwealth Avenue, Boston 16, Massachusetts, 1940, '41, '42, '44, '46\*, '47, '48, '49, '50, '51, '52, '53, '54, '55, '56\*.

1948. WHALEN, EDWARD J., 750 Main Street, Hartford, Connecticut, 1948, '50, '51, '52, '54, '55.
1953. WILKERSON, WILLIAM WESLEY, JR., 1028 Bennie Dillon Building, Nashville, Tennessee, 1953, '54, '55.
1948. WILLIAMS, HENRY L., 2nd, Mayo Clinic, Rochester, Minnesota, 1948, '49, '50\*, '51, '52\*, '53, '54, '55, '56.
1942. WISHART, D. E. STAUNTON, 170 St. George Street, Toronto, Canada, 1942, '44, '46, '47\*, '49, '51, '52\*, '53, '55, '56.
1951. WOODMAN, DEGRAAF, 156 E. 37th Street, New York 16, N. Y., 1951, '53, '54, '55\*, '56.
1953. WORK, WALTER P., 384 Post Street, San Francisco 8, California, 1953, '54, '56.

*Honorary Fellows*

1949. (1917) COATES, GEORGE M., 1721 Pine Street, Philadelphia 3, Pa., 1949\*, '51, '52, '54, '56.
1939. (1907) JACKSON, CHEVALIER, Schwenksville, Pennsylvania, 1939\*, '40\*.
1943. (1918) LEWIS, FIELDING O., Providence Road, Media, Pennsylvania.
1937. NAGER, F. B., Freiestrasse 30, 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\*.
1943. EGGSTON, ANDREW O., 653 Park Avenue, New York, N. Y., 1944, '49, '55.
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\*.
1937. PORTMANN, GEORGES, 25 bis Cours de Verdun, Bordeaux, France, 1947.
1924. SCHAEFER, J. PARSONS, 4634 Spruce Street, Philadelphia 39, 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., Musquodoboit Harbor, Nova Scotia, Canada.
1951. (1944) BEATTY, HUGH G., 150 E. Broad St., Columbus 15, Ohio, 1951, '52, '53.
1946. (1933) FOSTER, JOHN H., Eagle Lake, Texas, 1948, '49, '50, '51, '52.
1955. (1940) GATEWOOD, E. TRIBLE, Professional Building, Richmond, Virginia.
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.
1955. (1930) HOUSER, KARL M., 2035 Delancey Street, Philadelphia 3, Pennsylvania.
1951. (1928) JARVIS, DEFOREST C., Quarry Bank Bldg., Barre, Vt., 1952.
1956. (1939) JOHNSTON, WILLIAM HOWARD, 1515 State Street, Santa Barbara 7, California.

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.
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.
1956. (1925) TUCKER, GABRIEL, 429 Montgomery Avenue, Haverford, Pennsylvania.
1950. (1922) WHITE, FRANCIS W., 471 Park Avenue, New York 22, N. Y.
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

DR. BURT R. SHURLY

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. HENRY B. ORTON
- 1955. DR. THOMAS C. GALLOWAY
- 1956. DR. DEAN M. LIERLE

## 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        | 1949. Ralph A. Fenton    |
| 1940. Gordon Berry            | 1950. Gordon B. New      |
| 1941. Thomas E. Carmody       | 1951. H. Marshall Taylor |
| 1942-3. Charles J. Imperatori | 1952. Louis H. Clerf     |
| 1944-5. Harold I. Lillie      | 1953. Gordon F. Harkness |
| 1946. Frank R. Spencer        | 1954. Henry B. Orton     |
| 1947. Arthur W. Proetz        | 1955. Bernard J. McMahon |
| 1948. Frederick T. Hill       | 1956. LeRoy A. Schall    |

*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     |                    |

|                             |                     |
|-----------------------------|---------------------|
| 1904. Thomas Hubbard        | W. J. Freeman       |
| 1905. J. L. Goodale         | C. W. Richardson    |
| 1906. G. H. Makuen          | A. B. Thrasher      |
| 1907. J. P. Clark           | J. E. Rhodes        |
| 1908. E. Mayer              | F. R. Packard       |
| 1909. C. G. Coakley         | H. P. Mosher        |
| 1910. Robert C. Myles       | J. M. Ingersoll     |
| 1911. F. C. Cobb            | B. R. Shurly        |
| 1912. A. W. Watson          | W. Scott Renner     |
| 1913. F. E. Hopkins         | Geo. F. Shambaugh   |
| 1914. Clement T. Theisen    | Lewis A. Coffin     |
| 1915. J. Gordon Wilson      | Christian R. Holmes |
| 1916. Thomas H. Halsted     | Greenfield Sluder   |
| 1917. John Edwin Rhodes     | D. Crosby Greene    |
| 1918. G. E. Shambaugh       | John R. Winslow     |
| 1919. Francis R. Packard    | Harmon Smith        |
| 1920. Harmon Smith          | W. B. Chamberlin    |
| 1921. Dunbar Roy            | Robert C. Lynch     |
| 1922. George Fetterolf      | Lorenzo B. Lockard  |
| 1923. Hubert Arrowsmith     | Joseph B. Greene    |
| 1924. Ross H. Skillern      | Gordon Berry        |
| 1925. John E. Mackenty      | Robert Levy         |
| 1926. Lewis A. Coffin       | William V. Mullin   |
| 1927. Charles W. Richardson | Hill Hastings       |
| 1928. Robert Clyde Lynch    | Francis P. Emerson  |
| 1929. William B. Chamberlin | Ralph Albert Fenton |
| 1930. Harris P. Mosher      | James A. Babbitt    |
| 1931. Joseph B. Greene      | E. Ross Faulkner    |
| 1932. Gordon Berry          | Frank A. Spencer    |
| 1933. E. Ross Faulkner      | Thomas S. Carmody   |
| 1934. Gordon B. New         | Samuel McCullagh    |
| 1935. Edward C. Sewall      | H. Marshall Taylor  |
| 1936. William P. Wherry     | Harold I. Lillie    |
| 1937. Frank R. Spencer      | Bernard J. McMahon  |
| 1938. Ralph A. Fenton       | Frederick T. Hill   |

|                          |                      |
|--------------------------|----------------------|
| 1939. John H. Foster     | Thomas R. Gittins    |
| 1940. Charles H. Porter  | Gordon F. Harkness   |
| 1941. Arthur W. Proetz   | Henry B. Orton       |
| 1942-3. Harold I. Lillie | Dean M. Lierle       |
| 1944-5. John J. Shea     | Thomas C. Galloway   |
| 1946. H. Marshall Taylor | C. Stewart Nash      |
| 1947. John J. Shea       | Frederick A. Figi    |
| 1948. Henry B. Orton     | Anderson C. Hilding  |
| 1949. LeRoy A. Schall    | Fletcher D. Woodward |
| 1950. W. Likely Simpson  | Lyman G. Richards    |
| 1951. Wm. J. McNally     | Thomas C. Galloway   |
| 1952. J. Mackenzie Brown | Edwin N. Broyles     |
| 1953. Claude C. Cody     | Daniel S. Cuning     |
| 1954. James H. Maxwell   | Clyde A. Heatly      |
| 1955. Robert L. Goodale  | Paul H. Holinger     |
| 1956. Henry M. Goodyear  | Robert E. Priest     |

#### *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   | 1935. Charles J. Imperatori |
| 1923. George Fetterolf  | 1939. Frederick T. Hill     |
| 1932. William V. Mullin | 1948. Gordon F. Harkness    |
| 1933. James A. Babbitt  | 1953. Fred W. Dixon         |

*Librarians*

- |                      |                        |
|----------------------|------------------------|
| 1879. F. H. Bosworth | 1930. John F. Barnhill |
| 1883. T. R. French   | 1934. Burt R. Shurly   |
| 1903. J. H. Bryan    | 1935. George M. Coates |

*Librarian and Historian*

- |                        |                      |
|------------------------|----------------------|
| 1936. George M. Coates | 1944. Louis H. Clerf |
|------------------------|----------------------|

*Librarian, Historian and Editor*

- |                        |                          |
|------------------------|--------------------------|
| 1947. Harry P. Schenck | 1952. Bernard J. McMahon |
| 1955. Edwin N. Broyles |                          |



## 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.
- \*1904. Mosher, Harris P., Marblehead, Mass.
- \*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.
- \*1938. Blair, Vilray P., St. Louis, Missouri.
- \*1940. Colledge, Lionel, London, England.
- \*1901. Collier, Mayo, Kearsney Abbey, Kent, England.

- \*1893. Desvernine, Carlos M., Havana, Cuba.
- \*1930. Emerson, Francis P., Franklin, Mass.
- \*1936. Fraser, John S., Edinburgh, Scotland.
- \*1901. Grant, Sir James Dundas, London, England.
- \*1887. Cougenheim, A., Paris, France.
- \*1894. Holden, Edgar, Newark, N. J.
- \*1919. Kelly, Adam Brown, Helensburgh, Scotland.
- \*1881. Labus, Carlo, Milan, Italy.
- \*1931. LasSagna, Francesco, Parma, Italy.
- \*1926. Law, Frederick M., New York, N. Y.
- \*1902. Lermoyez, Marcel, Paris, France.
- \*1897. Luc, H., Paris, France.
- \*1896. MacDonald, Greville, Haslemere, Surrey, England.
- \*1894. MacIntyre, John, Glasgow, Scotland.
- \*1903. McBride, P., York, England.
- \*1920. McKenzie, Dan, London, England.
- \*1919. McKernon, James F., New Canaan, Conn.
- \*1880. Meyer, Wilhelm, Copenhagen, Denmark.
- \*1910. Moure, J. J. E., Bordeaux, France.
- \*1896. Mygind, Holger, Copenhagen, Denmark.
- \*1950. Neil, James Hardie, Auckland, New Zealand.
- \*1919. Paterson, Donald Rose, Cardiff, Wales.
- \*1941. Patterson, Norman, Herts, England.
- \*1919. Rogers, John, Jr., New York City.
- \*1894. Sajous, C. E. DeM., Philadelphia, Pa.
- \*1896. Schmiegelow, Ernst, Copenhagen, Denmark.
- \*1946. Segura, Eliseo, Buenos Aires, Argentina.
- \*1940. Soto, E. Fernandez, Havana, Cuba.
- \*1881. Thornton, Pugin, London, England.
- \*1913. Turner, A. Logan, Edinburgh, Scotland.
- \*1880. Whistler, W. McNeil, London, England.
- \*1901. Wingrave, Wyatt, Lyme Regis, Dorset, England.
- \*1894. Wolfenden, R. Norris, Kent, England.

*Emeritus Fellows*

- \*1915. Barnes, Harry Aldrich, Kingston, Mass.
- \*1928. Beck, Joseph C., Chicago, Illinois.
- \*1921. Berry, Gordon, Worcester, Mass.
- \*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.
- \*1937. Crowe, Samuel J., Baltimore, Maryland.
- \*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.
- \*1922. New, Gordon B., Rochester, Minn.
- \*1878. Porter, William, Ocean Springs, Miss.
- \*1901. Myles, Robert C., New York City.
- \*1923. Newhart, Horace, Minneapolis, Minn.
- \*1903. Packard, Francis R., Philadelphia, Pa.
- \*1942. Potts, John B., Omaha, Nebr.

- \*1903. Renner, W. Scott, Buffalo, N. Y.
- \*1897. Rhodes, John Edwin, Chicago, Ill.
- \*1884. Rice, Clarence C., New York City.
- \*1905. Richards, George L., South Yarmouth, Mass.
- \*1878. Robinson, Beverly, New York City.
- \*1921. Sauer, William E., St. Louis, Mo.
- \*1907. Shambaugh, George E., Chicago, Ill.
- \*1908. Smith, Harmon, New York City.
- \*1927. Tobey, Harold G., Boston, Mass.
- \*1892. Wagner, Henry L., San Francisco, Calif.
- \*1892. Watson, Arthur W., Philadelphia, Pa.
- \*1939. Wilson, J. Gordon, Old Bennington, Vt.
- \*1905. Wood, George B., Wynnewood, Pa.

*Active Fellows*

- \*1880. Allen, Harrison, Philadelphia, Pa.
- \*1917. Arrowsmith, Hubert, Brooklyn, N. Y.
- \*1879. Asch, Morris J., New York City.
- \*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.
- \*1932. Brown, John Mackenzie, Los Angeles, Calif.
- \*1892. Brown, Moreau R., Chicago, Ill.
- \*1933. Buckley, Robert E., New York City.
- \*1915. Canfield, R. Bishop, Ann Arbor, Mich.
- \*1934. Carmack, John Walter, Indianapolis, Ind.
- \*1924. Carmody, Thomas E., Denver, Colo.
- \*1889. Casselberry, William E., Chicago, Ill.
- \*1883. Chamberlain, C. W., Hartford, Conn.
- \*1917. Chamberlin, William B., Cleveland, Ohio.
- \*1896. Chappell, W. F., New York City.
- \*1882. Chapman, S. Hartwell, New Haven, Conn.
- \*1902. Coakley, Cornelius G., New York City.
- \*1913. Coffin, Rockwell C., Boston, Mass.
- 1918. Cox, Gerald H., New York City.
- \*1880. Cushing, E. W., Boston, Mass.



- \*1878. Cutter, Ephraim, West Falmouth, Mass.
- \*1880. Daly, W. H., Pittsburgh, Pa.
- \*1878. Davis, F. H., Chicago, Ill.
- \*1941. Davis, Warren B., Philadelphia, Pa.
- 1926. Dennis, Frank Lownes, Colorado Springs, Col.
- \*1901. Dickerman, E. T., Chicago, Ill.
- \*1878. Donaldson, Frank, Baltimore, Md.
- \*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
- \*1945. Hickey, Harold L., Denver, Colo.
- \*1879. Holden, Edgar, Newark, N. J.
- \*1907. Holmes, Christian R., Cincinnati, Ohio.
- \*1882. Hooper, Franklin H., Boston, Mass.
- \*1893. Hope, George B., New York City.
- \*1938. Hourn, George E., St. Louis, Mo.
- \*1939. Hunt, Westley Marshall, New York, N. Y.
- \*1901. Hyatt, Frank, Washington, D. C.
- \*1925. Iglaue, 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.
- \*1945. McLaurin, John G., Dallas, Texas.
- \*1885. McSherry, H. Clinton, Baltimore, Md.
- \*1914. Mackenty, John E., New York City.
- \*1898. Makuen, G. Hudson, Philadelphia, Pa.
- \*1881. Major, G. W., Montreal, Canada.
- \*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.
- \*1924. Taylor, Hermon Marshall, Jacksonville, Fla.
- \*1903. Theisen, Clement F., Albany, N. Y.
- \*1899. Thorner, Max, Cincinnati, Ohio.
- \*1892. Thrasher, Allen B., Cincinnati, Ohio.
- \*1937. Tobey, George L., Jr., Boston, Mass.
- \*1888. Van der Poel, S. O., New York City.
- \*1938. Vail, Harris H., Cincinnati, Ohio.
- \*1936. Voislowsky, Antonie P., New York City.
- \*1933. Wanamaker, Allison T., Seattle, Wash.
- 1896. Ward, Marshall R., Pittsburgh, Pa.
- \*1879. Ward, Whitfield, New York City.
- \*1886. Westbrook, Benjamin F., Brooklyn, N. Y.
- \*1924. Wherry, William P., Omaha, Neb.
- \*1924. White, Leon E., Boston, Mass.
- \*1939. Williams, Horace J., Philadelphia, Pa.
- \*1922. Wishart, David J. G., Toronto, Ontario.
- \*1940. Wood, V. Visscher, St. Louis, Mo.
- \*1896. Wollen, Green V., Indianapolis, Ind.











