

Transactions

American Laryngological Association

NINETY-SECOND VOLUME

1971

PUBLISHED BY THE ASSOCIATION
CHARLES FOSS FERGUSON, M.D., Editor



“DOCENDO DISCIMUS”

TRANSACTIONS
OF THE
NINETY-SECOND ANNUAL MEETING
OF THE
American
Laryngological Association
1971

HELD AT
THE SAN FRANCISCO HILTON
SAN FRANCISCO, CALIFORNIA
MAY 23 AND 24, 1971

PUBLISHED BY THE ASSOCIATION
ST. LOUIS, MISSOURI
CHARLES FOSS FERGUSON, M.D., Editor
1971

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* * * *

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ALDEN H. MILLER, M.D.



FELLOWS IN ATTENDANCE AT THE NINETY-SECOND ANNUAL MEETING

First Row (starting at the bottom of picture): Brown, Tremble, Bordley, Sooy, Frazer, Phelps, O'Neil, Lewy, Keim, Davison. *Second Row*: Hilding, Galloway, MacBeth, A. Miller, Jo Ono, Broyles, Woodman, Lathrop, Putney, Boies, LeJeune, Goodale. *Third Row*: Reed, Pastore, A. Smith, Ogura, van Alyea, Sanders, Weille, Andrews, Ballenger, Williams, D. Miller, Goldman, Baker, Fearon. *Fourth Row*: Cohen, Tabb, Kirchner, Moore, Suehs, Alexander, Friedberg, Tribble, Daly, Atkins, McGovern, Baxter, Munoz-MacCormick, Jones, Pratt, Tucker, Priest, Sisson, Ferguson, Lewis, Alfaro. *Fifth Row*: Harrill, Williams, O'Keefe, Clerf, McHenry, Cody, Bryce, Knight, Hutcheon, Pang, Holmes, Norris, Chandler.

TRANSACTIONS OF THE
NINETY-SECOND ANNUAL MEETING

OF THE

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MAY 23 AND 24, 1971

* * * *

The Ninety-Second Annual Meeting of the American Laryngological Association was called to order by the President, Dr. DeGraaf Woodman, at the San Francisco Hilton, San Francisco, California on May 23, 1971, at 8:15 A.M.

REGISTRATION OF FELLOWS

ACTIVE

ALEXANDER, FRED W.
ALFARO, VICTOR R.
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ATKINS, JOSEPH P.
AUSBAND, JOHN R.
BAKER, DANIEL C., JR.
BALLENGER, JOHN J.
BAXTER, JAMES D.
BOIES, LAWRENCE R.
BORDLEY, JOHN E.
BROWN, LESTER A.

BROYLES, EDWIN N.
BRYCE, DOUGLAS P.
CHANDLER, J. RYAN
CODY, CLAUDE C.
COHEN, SEYMOUR R.
DALY, JOHN F.
DAVISON, FRANCIS W.
DICKINSON, JOHN T.
FEARON, BLAIR W.
FERGUSON, CHARLES F.
FITZ-HUGH, G. SLAUGHTER
FRAZER, JOHN P.

FRIEDBERG, STANTON A.
GOLDMAN, JOSEPH L.
HANCKEL, RICHARD W.
HARRILL, JAMES A.
HARRIS, HERBERT H.
HILDING, ANDERSON C.
HILGER, JEROME A.
HOLMES, EDGAR M.
JONES, EDLEY H.
KEIM, W. FRANKLIN
KING, JAMES T.
KIRCHNER, JOHN A.
KNIGHT, JOHN S.
LATHROP, FRANK D.
LEWIS, JOHN S.
LEWY, ROBERT B.
MCGOVERN, FRANCIS H.
MCHENRY, LAWRENCE C.
MILLER, ALDEN H.
MILLER, DANIEL
MONTGOMERY, WILLIAM W.
MUNOZ-MACCORMICK, CARLOS E.
NORRIS, CHARLES M.
OGURA, JOSEPH H.
O'KEEFE, JOHN J.
O'NEIL, JAMES J.
PANG, LUP Q.
PASTORE, PETER N.
PRATT, LORING W.
PRIEST, ROBERT E.
PUTNEY, F. JOHNSON
REED, GEORGE F.
SANDERS, SAM H.
SAUNDERS, WILLIAM H.
SILCOX, LOUIS E.

SISSON, GEORGE A.
SKOLNICK, EMMANUEL M.
SOOY, FRANCIS A.
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WOODMAN, DEGRAAF

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LIERLE, DEAN M.
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MCCALL, JULIUS W.
MOORE, PAUL M.
PHELPS, KENNETH A.
RAWLINS, AUBREY G.
SALINGER, SAMUEL
SMITH, AUSTIN T.
TREMBLE, G. EDWARD
VAN ALYEA, OLIVER E.

OPENING SESSION

SCIENTIFIC SESSION

SUNDAY MORNING, MAY 23, 1971

The first Scientific Session of the Ninety-Second Annual Meeting of the American Laryngological Association, Inc., held at the San Francisco Hilton, San Francisco, California, convened on Sunday morning, May 23, 1971, at eight-fifteen o'clock, DeGraaf Woodman, M.D., Easton, Maryland, President of the Association, presiding.

PRESIDENTIAL ADDRESS

DEGRAAF WOODMAN, M.D.

Honored Guests, Fellows and Guests. It is my pleasure to welcome you to the 92nd Annual Meeting of our Association. This is the fourth time that we have held it in this beautiful city. In this modern age it is difficult to realize that many of us have been whisked here from other boundaries of this continent on the magic carpet of Jet Flight in a matter of a few hours. This is quite a contrast to what my great-grandfather experienced via a six month voyage around the Horn in 1849.

This is the home of such men as H. L. Wagner, the first member elected from this city in 1890. You may recall others such as Graham, Sewell, Martin, Morrison, and our contemporary, Rawlins, as well as our genial host this year, Francis Sooy.

The state of our association remains healthy. One may witness by the present program that there is no lack of new material related to our specialty both as to clinical and investigative fields. Material was offered in abundance so that the program was filled within six weeks of sending out requests for presentations.

I took the time to go over all 91 annual meetings reported in our Transactions. There appeared fairly regularly the question as to the proper size of our

association and also the manner in which we should choose our Fellowship. The latter has been a perennial problem and I hope that you will follow your Council's urgent request to base your vote on the teaching and hospital affiliations of the candidate, his literary contributions relating chiefly to Laryngology and Rhinology, and, above all, on the probabilities of his carrying on the high standards of the Association of the past and, of course, on his potential of making contributions to future programs.

Our Association, being the senior one of our specialty, has always had the problem of tapping men who have exhibited this ability and who are still young enough to be likely contributors after admission to the Fellowship. I can assure you that there are many men in the wings today who have already shown such abilities. When they come up, bear in mind the suggestions of the council and let us not permit sectional and personal prejudices to keep them out of the Fellowship. This latter plea can be found about every ten years throughout the past presidential addresses.

This is the year of Environmental Awareness, the year that the nation as a whole, as well as the politicians, have seen the light and the need of collective action to give some hope of checking

man's waste of his natural resources. I feel that we, most of whom are leaders in the various teaching institutions, have not only an opportunity but also a duty to pursue the quest of a better environment. The environmental improvement should extend further than just the preservation of forests, pure air and water, and should be carried into the Politico-Social Arena.

Are we going to lie prone and not resist or protest encroachments of government medicine? Or are we going to at least go down fighting against the introduction of third party medicine which interferes with the personal relationship between patient and physician and the contractual remuneration agreed upon between said patient and said physician? There is no substitute for stimulating effort and initiative better than the reward of such effort, whether it be monetary or expressions of gratitude from those unable to pay. This particularly applies to those on the firing line of clinical practice. This gets us involved in the Social and Political fields, a "never-never land" to be avoided at all costs by the busy practitioner and the research man. If accosted on this point and asked to contribute, the reply is generally: "Who? Me? *WHAT COULD I DO? WHO WOULD LISTEN TO ME?*"

Involvement does not mean that you should go out and run for public office, though this is recommended for those thus gifted; but do get involved by first becoming informed and know what is going on in relation to your civic and political life. I have been astounded as to how many of my colleagues are totally unaware of the activities which will affect *THEIR* future *AND* the future of their children. This does not mean that soap box oratory should be brought up with every patient who goes through your office, but a casual remark to one you may evaluate as being responsive, and thus help pass the word along to others, may help build up public opinion.

How might you accomplish the presentation of the advantages of private practice versus government medicine?

First, I would concentrate on your own immediate local environment, both medical and civic groups. Second, do whatever you can to offset the propaganda being put out by the bureaucratic agencies, magazines, and television stations. *PROTEST!* Third, write, wire, or telephone your local representative. Better still make his personal acquaintance and interview him. Give greater force to your actions by frequent repetition. Finally, join and support other groups who collectively will help you with ideas and who have a going machine to work with and for you — such as your own State Political Action program. Another experienced, well-established and effective organization is The Association of American Physicians and Surgeons. This organization has been concentrating on the preservation of private practice ever since its inception in 1943. Let us hope that our American Council of Otolaryngology, taking advantage of its unique geographic position, will lend support. Remember, *YOU* are a key man, *you* are sufficiently articulate to be able to put forth such an effort and you may be surprised, if you try, how many will listen. You have a worthy cause. *FIGHT FOR IT!*

You may recall that during the past year, the Province of Quebec, which joins our northern boundary, by overnight law conscripted 7,000 physicians who could only continue to practice under government dictate. If they refused or left the area, they were subject to a five-hundred-dollar-a-day fine. Is this conscription or slavery? Some of our own bureaucrats have similar plans for us here. We have been and are being conditioned to believe that government control is inevitable. *NEED THIS BE?* For this situation there is an apt saying: "The best way for Evil to Triumph is for Good Men to do *NOTHING!*"

This past year has been most rewarding for me. The great honor of being chosen your President, the support of our Council and loyal officers — all have made this a memorable experience. Please accept my heartfelt thanks for permitting me to have had this opportunity to serve you.

INTRODUCTION OF
GUEST OF HONOR, EDWIN N. BROYLES, M.D.

PRESIDENT DEGRAAF WOODMAN, M.D.

One of the privileges of being President is being able to choose and invite the Guest of Honor. This year he needs no introduction. He has been a member over thirty years. He has been our President. He has been our Historian. He is known well for his teachings and writings, and he has also introduced many special instruments used in laryngology.

He has been so closely knit with the Hopkins family that he now has had the honor of having his name attached to their bronchoscopic unit.

It gives me great pleasure to introduce our friend and colleague, Edwin Nash Broyles!

ADDRESS OF GUEST OF HONOR

EDWIN NASH BROYLES, M.D.

Thank you, Mr. President, for this great honor. In listening to those remarks, I could not help but think about what the little boy had to say about Enoch. Well, this little fellow was supposed to give the "golden-text" to the entire Sunday School and the text was "Enoch was not, for the Lord took him." As the little fellow mounted the platform he became a little flustered, and when they asked for the text, he said, "Enoch was not what the Lord took him for!"

Well, looking back, Mr. President, when I joined this Association in 1940 the President had *his* banquet for the men, and the President's wife had the ladies for a banquet.

As I remember it, there was a kind of "T" shaped table for the men and there were a few remarks from the head table. Then anyone who had a story to tell, or some interesting experience, got up and told it during the dinner, almost like a Quaker meeting.

But then in 1952, Dr. Clerf was elected President. He decided happily that the ladies join us in the banquet and they have been doing so ever since.

Now, if you don't mind I'll go back a few years to 1952, in Toronto. I may be a little wrong about this, but I think there was a formal dinner given to all the members of the three societies by the City of Toronto.

As you know at a formal dinner in the United Kingdom, no one smokes — (the fight against cigarettes hadn't started as severely then in '52 as now) — no one

smokes until after the Queen had been toasted which comes at the latter part of the banquet. Well, during the middle of the banquet, in came a footman with a large silver tray with a smaller silver cup and everybody asked him, "What is that?" "Snuff!"

So they gave us snuff to try to relieve our desire for nicotine; and boy!, if you're not used to it and you take a snuff and put it in the anatomical snuff box, and than take a whiff, oh boy! That silver tray was followed around by an explosion of sneezes!

Then, in 1958 up at the Fairmont Hotel at the top of Nob Hill, our President, Dr. Fred Dixon, was killing time and making a few remarks which he always did wonderfully well. I remember one remark to the effect that the side-walks up to Nob Hill are very friendly. He said, "If you become weary, you can just stop and lean against them!"

Mr. President, I appreciate this honor very much. I know you have a full meeting, and I appreciate very much the many honors that this Association has given me. I appreciate this and I thank you very much.

PRESIDENT WOODMAN: Edwin, we thank you for your reminiscences. I have something to give you.

This is a "Certificate of Merit of the American Laryngological Association and is hereby awarded to Edwin Nash Broyles, the honored guest of the American Laryngological Association at its Ninety-Second Annual Meeting, in recognition of his outstanding contributions to the science of laryngology."

CARCINOMA OF THE LARYNX

DIAGNOSIS AND TREATMENT BY SURGERY OR IRRADIATION

THOMAS P. HAYES, M.D.

GRAND RAPIDS, MICH.

JOSEPH P. ATKINS, M.D.

PHILADELPHIA, PA.

ANTOLIN RAVENTOS, M.D.

DAVIS, CALIF.

Early carcinoma of the larynx is highly curable by either surgery or radiation therapy, and this is well documented in the medical literature.^{2,3,5-7,9,11,14,16,20,21,24-26,29}

There is no agreement, however, on the choice of radiation therapy or surgery in the treatment of early lesions, nor has there appeared a strong trend to support a universal system of staging. In reviewing the recent experience at the Hospital of the University of Pennsylvania, these and other questions will be considered, and our treatment and results will be discussed.

The purposes of clinical staging are to help the physician select the best mode of therapy, to establish a prognosis, and to enable him to compare his results with others. Until recently, the only staging system which has been quite universally accepted is the International Classification of Cancer of the Cervix which was adopted by the World Health Organization in 1950. Continuing efforts are being made to design systems of classification for other neoplastic diseases including carcinoma of the larynx. The problem, it seems, is to design a system acceptable to the majority, reliable in its application, and reasonably simple to use. The TNM system for classifying carcinoma of the larynx published by the American Joint Committee on Cancer Staging and End Results Reporting Act (AJC) in 1961^{10,31} has been used in this hospital for several years.

In 1962, the Union International Contre le Cancer (UICC) published a system which is quite similar, but varies in the "N" categories and in the assignment of some TNM categories to a clinical stage. Since then, at least five other TNM systems have appeared.³⁴ It is apparently not difficult for experienced clinicians to devise clinical staging systems which correlate well with prognosis. Their value as media of communication is largely lost, however, unless there is a common agreement on the definitions. We are pleased that the AJC and the UICC are now discussing revisions of the larynx classifications which may result in a single uniform system.

FINDINGS

From 1958 through 1966, 209 patients with invasive squamous cell carcinoma of the larynx, representing 2.1% of all cases of malignant disease admitted to the Hospital of the University of Pennsylvania during this period, received their primary definitive therapy at this hospital. All were histologically verified, and were followed for three years or longer. Five year follow-up was available for 159 patients. Of the 209 patients in this study, 189 were male, 20 were female, and the age data are shown in Figure 1. Incidence to sex and age are consistent with other reported series.^{5,18,20,26,31} Distribution by stage (Table I) showed the largest number in stage II,

From the Departments of Bronchology, Esophagology, and Laryngeal Surgery and of Radiation Therapy of the Hospital of the University of Pennsylvania, Philadelphia, Pa.

This investigation was supported in part by PHS Training Grant No. 27102-01-69 from the Cancer Control Program (TPH).

TABLE I
INCIDENCE OF LARYNGEAL CARCINOMA BY STAGE

Stage	Glottic	Supraglottic	All Cases
I	77 (50%)	5 (9%)	82 (39%)
II	67 (43%)	20 (37%)	87 (42%)
III	3 (2%)	7 (13%)	10 (5%)
IV	8 (5%)	22 (41%)	30 (14%)
Totals	155 (100%)	54 (100%)	209 (100%)

and the smallest number in stage III. No palpable nodes were found in 81% (stages I and II), nodes were felt (cancer suspected) in 19% (stages III and IV), and distant metastases were present in one patient when first seen. Supraglottic lesions comprised 26% of the entire series. No tumors originating in the subglottis were identified.

CA OF LARYNX H.U.P.

Age Distribution
Range 24-85
Mean 59

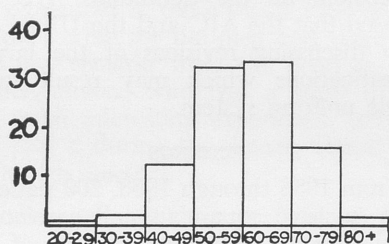


Fig. 1. Age distribution in a series of 209 cases of carcinoma of the larynx.

Three patients were diagnosed as having pseudosarcoma in conjunction with squamous cell carcinoma. One was found with a postradiation recurrence, another following repeated local excisions for carcinoma in situ, and the third was diagnosed with the initial biopsy.

DIAGNOSIS

Good results in carcinoma of the larynx depend on early recognition of the disease and accurate evaluation of the extent of the lesion so that the curative procedure chosen in any instance accurately coincides with the realities of the extent of the tumor. We continue to

insist on careful mirror laryngoscopy supplemented by the detailed information obtained from direct laryngoscopy generally under local anesthesia. Detailed and magnified views of the larynx can be obtained by the use of bronchoscopic telescopes using both the fore-oblique and right-angle instrument. Information as to the location and extent of the tumor in the larynx is more easily possible because it permits one to assess the problem of motility. We feel this method of examination is better than examination of the larynx by means of the operating microscope under general anesthesia. Biopsies should be carefully done so as to preserve the general configuration of the larynx with regard to the extent of tumor invasion, so that at operation one is not confused by an area involved in tumor and an area distorted by unnecessary biopsies.

We have used laryngograms in the study of laryngeal tumors but do not have the confidence in them that we place in direct laryngoscopic observation.

TREATMENT

Forty-five of 82 stage I cases were treated surgically (Fig. 2), 38 by partial laryngectomy, six by total laryngectomy, and one by local excision. Thirty-seven stage I patients received radiation therapy. The tumor dose varied from 5,800 rads in six weeks to 7,000 rads in seven weeks. Kilovoltage x-rays were employed in the first two and one-half years of this study, and 2 MeV x-rays thereafter.

In stage II, 60 of 87 patients were treated surgically — 53 by total laryngectomy and 7 by partial laryngectomy.

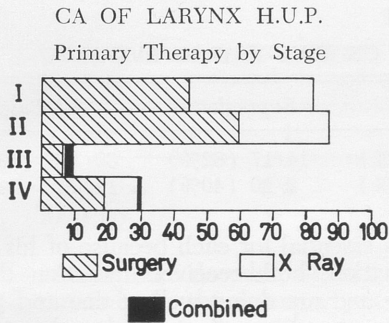


Fig. 2. Choice of therapy in carcinoma of the larynx in Stages 1, 2, 3 and 4.

Sixteen of the 20 supraglottic tumors in this stage were treated surgically and nine also had a prophylactic radical neck dissection. Of the 44 stage II glottic tumors operated upon, only four neck dissections were done. The technique employed in the 27 patients in this stage who received radiation therapy was the same as for stage I.

Seven of the 10 stage III patients had total laryngectomy and radical neck dis-

sections, one received radiation therapy only, and two had planned combined therapy. Nineteen of the 30 stage IV patients were operated upon with some reasonable hope of cure, while radiation therapy for the most part was utilized with palliative intent in this group. The single stage IV patient who received combined therapy had distant metastases, and the objective of treatment was palliation.

RESULTS

Survival curves were constructed by the life table method¹² (Fig. 3). It can be seen that the survival curve for stage I is essentially the same as for 60-year-old males in the mid-Atlantic states.²⁷

Table II shows the five year survival of determinant cases by stage. Our results are quite comparable to those reported by Smith et al.³¹ in 1961 which were 91% in stage I, 72% in stage II, 45% in stage III, and 22% in stage IV. In 1966, separate studies by Heise and Baylis,¹⁸ Caulk,⁸ and Alexander and Casady¹ utilized the AJC system in clas-

TABLE II
FIVE-YEAR SURVIVAL BY STAGE AND TREATMENT
(Lost to Follow-Up — Considered Dead of Disease)

	<i>Alive at 5 Years</i>	<i>Dead of Disease</i>	<i>Lost to Fol- low-Up</i>	<i>Dead N.E.D.</i>	<i>Follow- Up < 5 Years</i>	<i>Survival of Deter- minant Cases</i>	<i>%</i>
<i>STAGE I</i>							
Surgery	36	0	1	2	6	36/37	97
X.R.T.	17	0	3	5	12	17/20	85 (93)
<i>STAGE II</i>							
Surgery	29	10	3	2	16	29/44	66
X.R.T.	7	5	0	5	10	7/12	58 (64)
<i>STAGE III</i>							
Surgery	3	2	0	1	1	5/8	62
X.R.T.	1	1	0	0	0		
Combined	1	0	0	0	0		
<i>STAGE IV</i>							
Surgery	5	7	2	0	5	6/24	25
X.R.T.	1	8	0	1	0		
Combined	0	1	0	0	0		
<i>TOTALS</i>	100	34	9	16	50	100/145	69

TABLE III
INFLUENCE OF PALPABLE NODES ON FIVE-YEAR SURVIVAL

	Glottic	Supraglottic	All Cases
Stages I and II (no palpable nodes)	75/96 (78%)	14/17 (82%)	89/113 (79%)
Stages III and IV (palpable nodes)	3/12 (25%)	8/20 (40%)	11/32 (34%)

sifying a total of 1,296 cases of carcinoma of the larynx and their results were similar.

In stage I, there were seven radiation and two surgical cases which recurred, and all nine were salvaged by subsequent total laryngectomy. All but two of these have now been followed more than three years after surgery. Seven of the recurrences were noted in from two to 24 months. Regrowth of tumor at the original site was seen in the other two cases at 60 and 72 months after initial treatment by radiation therapy.

was essential for each because of his occupation; both received radiation therapy and are doing well at one and two years. Another patient developed a stage III pyriform sinus tumor five years after partial laryngectomy for a cordal lesion and has remained well for 18 months following radiation therapy.

A stage IV patient with recurrent disease in the neck was treated with an ^{192}Ir implant, and has done well for over two years. A stage II patient with local recurrence following partial laryngectomy was treated by x-ray with a 51-month remission before succumbing to distant metastases. In many other instances, x-ray produced worthwhile palliation.

Whereas the incidence of palpable nodes (cancer suspected) was 19% in the entire series, 29 of 54 supraglottic tumors demonstrated this finding (Table I). Survival was comparable to the glottic tumors. The deleterious influence of palpable lymph nodes on five year survival is shown in Table III.

DISCUSSION

It seems reasonable that the objective in treating early carcinoma of the larynx should be to obtain the best cure-rate at least risk to the patient while preserving the voice whenever possible. For tumors involving the mid-portion of the margin of the cord, not extending to the anterior commissure and not impairing cord motility, the partial laryngectomy operation may result in a good voice if normal motility of the arytenoid is maintained. However, preservation of a near-normal voice is more frequently achieved with radiation therapy.

X-ray is a carcinogenic agent, and those using it are always concerned about the possibility of inducing a new tumor. Since the latent interval is likely

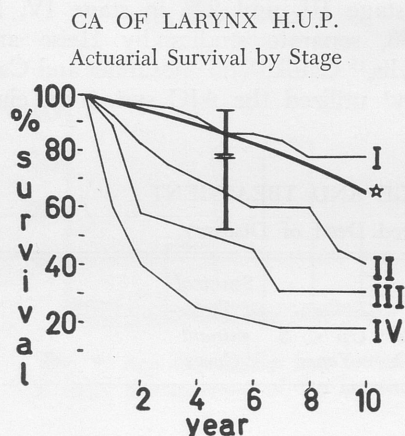


Fig. 3. Actuarial survival in carcinoma of the larynx at the hospital of the University of Pennsylvania, 1958 through 1966.

Although radiation therapy was not employed in treating recurrent stage I lesions, it was used with apparent success in treating three patients who developed second primary cancers. Two patients who had had partial laryngectomy for cordal lesions developed second primaries on the remaining cord seven and eight years later. Voice preservation

TABLE IV
CARCINOMA OF THE LARYNX, H.U.P. SERIES SUCCESSFUL TREATMENT
OF RECURRENCES

Stage	I	II	III	IV	All
By surgery	9/9	8/16	0/2	2/6	19/33
By x-ray	—	0/7	—	1/8	1/15

to be two or more decades, this concern applies mainly to the younger patients who, if cured, have long life expectancies. The actual magnitude of this risk is difficult to quantify. Since x-rays of the megavoltage range are not selectively absorbed by the denser tissues, time may show a decreased incidence of radiation induced sarcomas, and their skin-sparing qualities are well appreciated. However, sincere concern for the ultimate fate of heavily irradiated tissues is clearly reflected in the selection of primary therapy in this series; the average age of stage I and II surgical patients is fully seven years less than those selected for radiation therapy.

We know of no study of the effect of radical irradiation of the head and neck on the survival rate of cured patients, but Sorensen,³² and Kohn et al.²³ concluded that irradiation per se had not materially affected the survival of patients with carcinoma of the cervix cured by irradiation.

Late radiation induced necrosis, requiring laryngectomy, has been reported¹⁵ although it was not encountered in this series. In 1967, Wang³⁵ reported that four of six patients with carcinoma arising in previously irradiated larynges survived from two to four years following radical reirradiation without disease, which suggests that the tolerance of the larynx to irradiation is rather great provided that good technique with proper attention to time-dose-volume relationships is employed.

In cancer therapy, the two established curative modalities, radiation therapy and surgery, frequently are called upon to rescue the patient when one or the other method of primary attack fails, and carcinoma of the larynx is no ex-

ception. Most surgeons prefer to operate in fields not previously irradiated because of the danger of delayed healing or worse.^{4,22,33} Similarly, radiotherapists have long recognized that surgically induced alterations of the tumor bed as well as the possibility of dissemination of tumor cells by surgical manipulation diminish the radio-curability in any situation treated postoperatively. Irrespective of the validity of the arguments on either side, our experience has been that the surgical treatment of recurrences is more often successful than the reverse (Table IV) and to radiation therapists is a good reason to select radiation therapy initially in certain situations.

Preoperative radiation therapy in advanced carcinoma of the larynx has not been extensively employed in this hospital, but the work of Powers and Ogura,²⁸ Henschke et al.,¹⁹ and Skolnik et al.³⁰ suggests that this area should be more thoroughly explored. Similarly, Fletcher et al.¹³ offer evidence of the value of postoperative radiation therapy in advanced supraglottic lesions. At the present time, we feel that prophylactic neck dissections are not worthwhile.

Adjuvant drug therapy is now being evaluated in a cooperative study,¹⁷ and it is hoped that this may provide a more fruitful approach to the advanced head and neck cancers.

CONCLUSIONS

The results of the treatment of carcinoma of the larynx at the Hospital of the University of Pennsylvania are consistent with others and tend to confirm the high degree of curability of early lesions by either radiation therapy or surgery. In this hospital, surgery has been selected more often than radiation therapy in potentially curable early le-

sions in young patients because of concern about late radiation changes including oncogenesis in long-term survivors. Radiotherapists tend to worry less about this than surgeons, and emphasize the importance of voice preservation. However, since patients with carcinoma of the larynx are almost invariably seen first by the surgeon, the preference for surgery in the younger patient in this institution will probably continue until

such time as greater experience may show this hazard to be negligible.

The role of radiation therapy in advanced carcinoma of the larynx has been largely palliative, although combined therapy needs to be more fully evaluated. In our experience, surgery is often effective in the treatment of radiation failures. Conversely, treatment of surgical failures by radiation is usually unsuccessful.

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DISCUSSION

DR. HERBERT H. HARRIS: This subject is very dear to my heart and I think Dr. Atkins has given us a very comprehensive review of his experience.

There is one matter on which I wish to appeal to you, however, and that is the very close follow-up on these patients. I have, I suppose in my recent years, numerous patients with multiple primaries of the oral cavity, the pharynx, the nasopharynx and the hypopharynx. This is striking.

Many patients whom I see today, I go over so thoroughly that sometimes I repeat myself three or four times in one examination, because I have found so many cases who have early primaries, elsewhere than the original primary, which I have treated by surgery or irradiation. In fact, occasionally I find one with a metastatic node in the neck and I say, "how could this happen five years ago; it's been dormant." I came to find out they had a new primary and it was a metastatic lesion.

I do appreciate this opportunity and I do thank Dr. Atkins and his authors for presenting this subject. We are at present moving a little bit towards conservative surgery and trying to preserve as much of the larynx as possible. I believe so far our results are excellent as compared to irradiation because, as you know, we still have recurrences, or incompletely cured cancer from irradiation therapy, regardless of who gives it or how it is given.

DR. JOSEPH P. ATKINS (closing): I would certainly agree entirely with what Dr. Harris has said, that the real salvage of these patients will never be completely realized unless we do have a complete and careful follow-up.

This is one of the reasons why I feel the patient with cancer of the larynx ought to know just what he has. Otherwise, you cannot expect him to follow-up his care intelligently. This is for his own protection, and the results are sufficiently good, so that when you tell him what he has you are not really giving him a great psychological handicap.

POSTIRRADIATION CARCINOMA OF THE LARYNX

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Postirradiation carcinoma of the larynx, laryngopharynx and thyroid gland has been reported by otolaryngologists, radiologists and pediatricians. Holinger and Rabbett⁵ in 1953 reported a small series of cases that had developed carcinoma of the larynx and hypopharynx. Their review of the literature at that time showed nine similar cases. Goolden³ has studied the subject of radiation cancer of the pharynx over a period of years. In order for a case to be considered in this group, he listed the following criteria: 1) mucosal evidence of malignancy; 2) extensive irradiation administered for disease not of neoplastic nature; 3) a considerable lapse of time between the administration of the irradiation and the occurrence of the tumor; and 4) the location of the tumor in the area of most concentrated exposure to the irradiation.

Goolden⁴ in 1957 reported on 25 cases of radiation cancer of the pharynx and larynx with five occurring in the larynx and 20 in the hypopharynx. Many of his cases were patients who received radiotherapy for thyrotoxicosis. The average time interval for the development of the carcinoma was 25 to 30 years with none less than ten years. The patients had reactions ranging from light pigmentation to actual necrosis of the irradiated area. He postulated that severe damage to tissues is a prerequisite for the induction of radiation cancer.

Lane⁶ and Baker¹ reported on pseudo-sarcoma of the larynx occurring many

years after radiotherapy for a nonepithelial lesion. In their cases, the pseudo-sarcoma was associated with a squamous cell carcinoma.

Wlodyka⁹ reported on the carcinogenic effects of x-ray on the larynx. In his series of cases there were four malignancies that developed in patients who received radiation therapy for benign papilloma of the larynx.

Rabbett⁷ reviewed the literature and added a case of juvenile laryngeal papillomatosis in a child two years of age. The patient had received radiation therapy on three different occasions. The radiation therapy did not control the papillomata, and squamous cell carcinoma of the larynx developed in the papillomatous lesions of the larynx and trachea. The patient had a total laryngectomy and radiation therapy to the trachea. He died of the disease at 23 years of age.

An experimental study by Bonte et al.² on the effects of ionizing radiation upon the immature rabbit larynx showed that a single dose of 800 r was sufficient to cause metaplasia of the epithelium, chondromalacia, and immaturity of cartilage cells. The gross effect was a reduction in the eventual size of the larynx. Laryngeal hypoplasia and stricture of the larynx in children following irradiation therapy has also been reported by others.

The evidence is thus abundant and universal that irradiation therapy to the

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region of the larynx is carcinogenic and may lead to the development of carcinoma in the larynx and laryngopharynx after an interval of many years.

The present study is not to add to previous case reports but to review a group of cases who had carcinoma of the larynx and had received irradiation therapy, and who developed a second carcinoma in the larynx. Som and Peimer⁸ reported on postcricoid carcinoma developing in a group of patients who had received radiotherapy for laryngeal carcinoma.

There are two methods of treatment for intrinsic carcinoma of the larynx — surgery or irradiation. The choice is dependent on the experience and skill of the laryngeal surgeon and the experience and skill of the radiotherapist. Results in institutions will vary, with radiotherapy surpassing surgery and vice versa.

At any large medical center many patients are seen who have been cured by either modality. Unfortunately, many patients are also seen and surgery or irradiation therapy have failed to effect a cure. Many patients who have been subjected to both x-ray and surgery succumb to the disease.

In recent years, more patients are being seen who have survived five or more years after having been treated by surgery or irradiation therapy for cancer of the larynx, only to develop a regrowth of their carcinoma or a new epithelial lesion.

A laryngeal surgeon who performs a laryngofissure or partial laryngectomy for carcinoma of the larynx has the help of the pathologist to determine if the lesion has been completely excised. If the margins of resection show that the tumor has been incompletely removed, the surgeon has the choice of doing more extensive surgery or to refer the patient for irradiation therapy.

The radiotherapist who treats carcinoma of the larynx does not have this advantage and curability rests upon care-

ful follow-ups by mirror laryngoscopy and direct laryngoscopy. Careful histologic examination of the irradiated tissues is not possible nor desirable at the completion of therapy. In the patients who have not been cured of their disease by irradiation therapy there is often a delay of several months or a year before the failure can be documented by histologic study. This can also be true to a lesser degree of a surgically treated patient if the patient has a multicentric lesion. The failure to cure the patient by either surgery, irradiation therapy or by a combination of both modalities results in death of the patient. The curability rate diminishes markedly, the longer the disease is present in the patient.

One of Goolden's criteria for radiation cancer of the pharynx is that there be extensive irradiation administered for a disease of nonneoplastic nature should probably be extended to include some cases of carcinoma of the larynx. The case reports of Som and Peimer would appear to justify this conclusion.

Over a period of years patients have been seen who have had irradiation therapy for carcinoma of the larynx and who have appeared to be cured for more than five years, but who subsequently developed carcinoma of the larynx.

Seven patients were seen who were in this group. The interval between irradiation therapy and the development of either a regrowth of their laryngeal carcinoma or new lesion was five to 25 years. In each instance the lesion appeared to be a new one although this cannot be proven. The findings of mirror laryngoscopy and direct laryngoscopy were that of an early lesion limited to one vocal cord or the anterior commissure, without disturbance in other parts of the larynx. Five of the patients were treated by conservative procedures such as laryngofissure or hemilaryngectomy. In this group one patient had x-ray therapy to the larynx at 33 years of age. He subsequently had two laryngofissure procedures at five years and at

eight years after irradiation therapy and he has remained well at 11 years after the onset of his disease. One patient who continued to smoke after irradiation therapy had a hemilaryngectomy for a second carcinoma ten years after the irradiation therapy had been completed. He is well, two years after the surgery, and he has not smoked since.

Two patients in this group developed evidence of carcinoma at 10 and 12 years after irradiation therapy and had a total laryngectomy. It is not always easy to detect either persistent carcinoma or a new growth in the postirradiated larynx. The tissues as a rule show a marked reaction to inflammation and to tumor formation. Edema obscures the true nature of the disease interfering with examination, biopsy and diagnosis.

Experience has shown that the majority of patients who have persistent carcinoma of the larynx following either surgery or irradiation therapy have evidence of the disease within the first year. The five-year survival period used for statistical purposes, as it applied to the larynx, is probably more satisfactory than with some other forms of carcinoma, notably glandular forms of the disease. It is most unusual to find persistent laryngeal carcinoma after five years of apparent freedom of the disease in patients who have had surgery as the initial form of treatment. The finding of active disease in the larynx of patients after five years of freedom of the disease and who have had irradiation therapy, suggests that a new growth

has developed and that the irradiation therapy was an important carcinogenic factor.

A discussion of the patients who have had one or more courses of irradiation therapy within a five-year period from the onset of the disease, and who have persistent carcinoma, would only add to the confusion of this report and would best be discussed in a future presentation.

CONCLUSIONS

The carcinogenic effect of irradiation therapy on the larynx has been slowly established over a period of years. There is conclusive evidence of its effect on children who had irradiation therapy for papilloma of the larynx.

The question of the probability of carcinoma of the larynx developing again as a new lesion in patients who have had irradiation therapy for a similar disease is raised.

It would appear that an adult, 45 years of age or under who has carcinoma of the larynx, would probably be better treated by surgery than by irradiation therapy because of the persistent carcinogenic effect of the irradiation.

The number of patients who develop postirradiation carcinoma is small compared to the total number treated.

It is in the latent period, i.e., after five years or more that irradiation continues to have an effect that may produce a new epithelial malignant growth.

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DISCUSSION

SHIRLEY H. BARON, M.D.: I have a special awareness of irradiation-induced cancer. In 1958, I presented a paper entitled, "Does Irradiation of Infants and Children Cause Cancer of the Thyroid?" Seven cases of cancer of the thyroid in children were reported. Six had had previous irradiation of the thymus in infancy and one had had irradiation of the nasopharynx for the reduction of nasopharyngeal lymphoid tissue at the age of five. It was concluded that it was unwise to prescribe irradiation for non-malignant conditions in infants and children. This conclusion certainly is supported by the five cases, mentioned by Dr. Baker, in which cancer of the larynx developed after irradiation therapy for laryngeal papillomas.

Dr. Baker suggests that delayed onset of recurrent laryngeal cancer could be considered evidence of irradiation-induced cancer. One of my patients with a T1 lesion of the right vocal cord was treated with linear accelerator irradiation. After a nine year period of uninhibited smoking and drinking, he developed cancer of his other vocal cord. Was this irradiation-induced or the result of irritative habits in a cancer-prone individual?

In 1968, Lathrop reported a ten year follow-up of 71 patients with laryngeal cancer treated with supravoltage irradiation. One patient had cancer of the epiglottis at the start, and 9 years later developed recurrent cancer at the same site. Could *this* be considered irradiation-induced?

In Lathrop's series, no patient classified as "stage I" died as the result of his laryngeal cancer, but it was interesting to note that of the patients who died of other causes, seven (58 percent) succumbed to another primary cancer in a tissue not previously irradiated. One wonders, then, if delayed recurrent cancer is irradiation-induced, or if it occurs because of a special cancer predilection.

Dr. Baker suggests that it would appear that an adult, age forty-five or below, who has carcinoma of the larynx would probably be better treated by surgery than by irradiation therapy because of the persistent carcinogenic effect of irradiation. While the carcinogenic effect of irradiation is indeed a possibility, I personally would be reluctant to follow this suggestion because: 1) modern irradiation is very successful in the treatment of Stage I and II laryngeal cancers. 2) The number of patients who develop postirradiation cancer is small compared to the total number treated. 3) The post-treatment voice quality is much better after irradiation than after conservative surgery. 4) The surgical salvage rate of post-irradiation recurrences is high. Many of these are saved by conservative

surgery with good healing in spite of previous irradiation.

This has indeed been an interesting presentation, Dr. Baker. I appreciate having had the opportunity of discussing it.

DR. DANIEL MILLER: My remarks are directed to both speakers who have produced a most provocative subject this morning.

I should like to echo Dr. Baron's remarks about the morbidity of X-radiation. The incidence is still so low biostatistically that the gains for the "T-1" lesion particularly make it almost mandatory to use it despite the delayed effects of radiation. Our radiation therapy department speak of ten years as a rough time period when a second primary may occur. It is interesting that the palatine arch has a high incidence of multiple primary lesions as does the entire upper respiratory tract. For example, we have in our series about a ten per cent second primary in the palatine arch, and that if a second primary has developed, there would be anywhere from a 15 to 20 per cent chance for a third primary; so we really do not know whether these are irradiation-induced or not.

My remarks are directed mainly to the problem of the recurrent "T-1" lesion. As Dr. Atkins has reported, he had a recurrence rate in his "T-1" lesions. Nine out of nine were taken care of with total laryngectomy. He had a 93 per cent five year cure-rate in the "T-1" lesion. We have about the same 93 to 95 per cent five year cure rate with radiation. Is laryngectomy absolutely necessary for a recurrent "T-1" lesion?

From the standpoint of what we have been doing of late, I'd like to present a few slides showing the effect of the use of cryosurgery in the "T-1" lesion which has failed to be cured by irradiation therapy. We have now done about six. The oldest is about three years or more. It is a simple measure, and the patient leaves the hospital one day after the surgery. What have we done with these "T-1" lesions? Two to three months following the use of cryosurgery we have not depended on our vision; we have depended on a stripping of the vocal cord, thereby having microscopic proof that there is no residual cancer. None of the patients to date who have had this procedure have recurrence of the "T-1" lesion.

DR. DANIEL C. BAKER, JR. (closing): I thank Dr. Baron and Dr. Miller for their discussions. It was my purpose to raise this point, and I know of no way in which one can talk in positive terms as to whether or not the lesions I have encountered represent irradiation-induced carcinoma, or whether or not they rep-

resent a new primary the patient would have had whether or not he had had irradiation therapy. But one thing is certain. After reviewing the literature, having picked up no less than 60 cases of carcinoma of the pharynx and larynx in patients who had had irradiation therapy for a lesion not even connected with

the larynx (for diseases such as tuberculous lymph nodes, and so forth) with a late development of carcinoma, I feel there is a very definite relationship. When I discuss the question of therapy in an individual case — if I have a young adult, certainly in her thirties, I strongly recommend surgery.

CLINICAL AND HISTOLOGICAL OBSERVATIONS ON SUPRAGLOTTIC CANCER

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The concept of a barrier to downward extension of supraglottic cancer has been advanced by both clinical and laboratory observations. Coutard and Valat⁴ observed that these growths do not involve the glottis and that they are often bilateral or horseshoe in distribution across the anterior larynx while remaining entirely above the ventricle.

Leroux-Robert⁹ confirmed these observations in a study of serial sections of the entire larynx, showing that supraglottic lesions remain confined above the ventricle and that it is only ventricular growths that spread to both the supraglottic and infraglottic larynx.

Baclesse² pointed out that supraglottic cancer usually begins at the junction of the epiglottis and false cords, and described large supraglottic growths that hung over and occluded the glottis but in which the base remained well above the ventricle.

Bocca et al.,³ in a study of 160 laryngectomy specimens for supraglottic cancer, observed that "even in the most advanced stages of evolution primary vestibular growths never invaded the floor of the ventricle or the vocal cords." They attributed this to the fact that the supraglottic portion of the larynx develops from the buccopharyngeal anlage, whereas the glottic and subglottic portions develop from the tracheobronchial anlage. Each portion, then, can become

invaded by cancer that is confined within the boundaries of embryologic demarcation.

These clinical observations agree with experimental studies of the spread of fluids and dyes injected into various parts of the larynx. Hajek performed a series of injections into the human larynx and demonstrated, among other things, a sharp boundary along the inferior edge of the false vocal cord below which edema did not spread (Table II, Fig. 4 in Hajek's paper). Even in the most extreme degrees of supraglottic swelling the vocal cords were not involved in any way. This is shown dramatically in Table I, Figure 7 of Hajek's original publication.⁷

Similarly, Pressman et al.,¹² using dye injections, demonstrated submucosal compartments with their regional lymphatics. These studies showed that the ventricle is a compartment completely isolated from the false cord. Injections along the epiglottis spread to the false cord and were sharply terminated along its inferior border.

Tucker and Smith's studies¹⁵ demonstrating anatomic compartments bounded by elastic tissue membranes offer an anatomic basis for these observations.

LOCATION OF SUPRAGLOTTIC CANCER

Baclesse² described five subdivisions of the supraglottic larynx in which can-

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cer can develop, each differing from the others in prognosis and in regional node metastasis. In the more posterior sites of origin regional nodes were involved more than twice as often as in those arising anteriorly.

The present study is an assessment of the pathologic and anatomic features of supraglottic cancer as observed in serial sections in 30 surgical specimens. How is the surface appearance of supraglottic cancer as determined by laryngoscopy and laryngography related to deep infiltration, thyroid cartilage invasion and other features of clinical importance? Further, which supraglottic tumors removed by total laryngectomy could have been removed adequately by horizontal partial laryngectomy?

SERIAL SECTION STUDY OF LARYNGEAL SPECIMENS

In a previous publication, the histologic findings in ten epiglottic and ten other supraglottic cancers were reported.⁸ Since this original report, additional cases have brought the number of supraglottic specimens to 30. Further study of these specimens, 25 of them removed by total laryngectomy, revealed that the lower edge of the tumor extended downward below the anterior commissure in eight. Serial sections showed that in five of these eight cases, the thyroid cartilage was invaded by cancer. In none of the remaining 25 cases was there any invasion of the thyroid cartilage (Figs. 1, 2 and 3).

Of the five growths which invaded the thyroid cartilage, four had been described as deeply ulcerative. The fifth crossed the ventricle posteriorly and was poorly differentiated histologically.

Of the remaining cases, in which no invasion of the cartilage was found, 16 had been described as exophytic, one as ulcerative. The gross appearance of the remaining eight was not recorded.

Our material suggests then, that exophytic lesions of the supraglottic larynx are the kind that tend to remain confined above the ventricle and the an-

terior commissure and that these do not invade the thyroid ala.

Conversely, ulcerative lesions may extend downward across the anterior commissure or anterior part of the ventricle and, in this case, frequently invade and destroy the anterior portion of the laryngeal framework. Regardless of the surface appearance, however, or of the depth of soft-tissue invasion above the glottis, we have not observed microscopic invasion of the thyroid cartilage unless the inferior edge of the growth was visible below the level of the anterior commissure.

In a study of 28 supraglottic cancers, Ogura¹⁰ observed that none showed invasion of the anterior thyroid cartilage. He further observed that in other types of growths showing erosion of the thyroid cartilage the invasion began in the tendon of the anterior commissure. This agrees with our experience in the serially sectioned material, these latter growths being classified as transglottic, rather than T3 supraglottic.

In three of our cases which had been considered suitable for horizontal supraglottic laryngectomy, and although exhaustive preoperative evaluation did not show anterior commissure involvement, the thyroid cartilage was found to be involved as the perichondrium was being elevated. Total laryngectomy was performed in these three cases and the growth found to have crossed downward below the anterior commissure in all three. The invasion of the thyroid cartilage was found before the larynx was opened.

CLINICAL APPLICATIONS

One of the objectives of this study was to evaluate horizontal supraglottic laryngectomy in the treatment of large supraglottic cancers. More specifically, which of the 25 tumors removed by total laryngectomy could have been encompassed by a horizontal supraglottic resection?

In this group, total laryngectomies were performed for the following reasons:

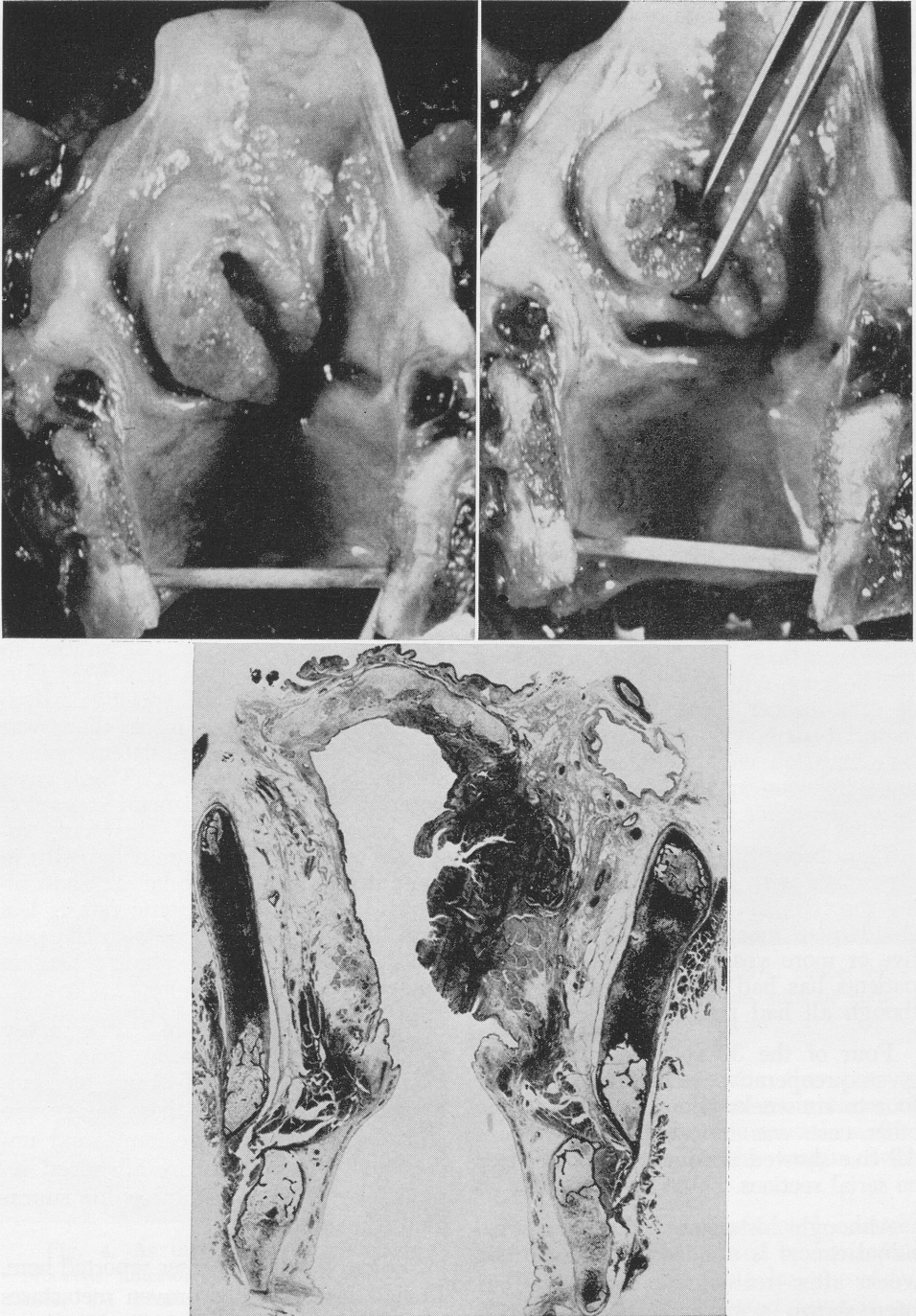


Fig. 1. Large supraglottic cancer obstructing the laryngeal airway.

Fig. 2. Lower edge of the growth shown in Fig. 1, well above the ventricle and anterior commissure. The true vocal cords are free of gross involvement.

Fig. 3. Coronal section of growth shown in Fig. 1. The ventricle and true cord are not involved, and the deep part of the tumor does not reach the thyroid ala.

8 — the lower edge of the tumor was found at operation to lie below the level of the anterior commissure or ventricle.

8 — the tumor approached or involved the arytenoid cartilage.

3 — questionable or definite invasion of the base of the tongue.

2 — pulmonary cripples.

1 — radiation failure.

3 — in these, the extent of the tumor was overestimated clinically, and the growth could have been resected adequately by horizontal supraglottic laryngectomy.

If the mucosa over the arytenoid cartilage is involved with tumor, this is not a supraglottic tumor but one of the laryngopharynx. If, however, tumor merely approaches the arytenoid, and if an adequate margin around the tumor can be obtained by including the arytenoid cartilage in the specimen, this can be done without violating the rules of good cancer surgery. Our histologic material demonstrates arytenoid cartilage invasion only in those cases with gross involvement of the overlying mucous membrane.

This observation is supported by Som's experience with eight such cases requiring resection of the arytenoid to obtain an adequate margin. With a follow-up of five or more years, none of these eight patients has had a local recurrence, although all had positive cervical nodes.

Four of the 30 specimens had been given preoperative radiation of 4,000 r four to six weeks before operation. One other case was a radiotherapy failure. All five showed residual areas of cancer on serial sections.

Although histologic evidence of residual cancer is unreliable at four to six weeks after treatment, radiotherapy has been shown statistically to give lower control rates for supraglottic cancer than does surgery.

While small Stage I lesions of the supraglottic larynx can be controlled

equally well with radiotherapy or surgery (over 70%)⁵ it is in the treatment of larger primary growths that the superiority of surgery over radiotherapy becomes striking. In a survey of 544 supraglottic cancers classified as T2, T3 and T4 and without clinical lymph node involvement, Vermund¹⁶ reported a relative five-year survival of 32% for primary radiation therapy and 64% for primary surgery.

Surgical salvage after radiation failure may require total laryngectomy because of the likelihood of chondritis after horizontal supraglottic resection in the irradiated larynx. The possibility of preserving laryngeal function by supraglottic partial laryngectomy is considerably decreased and the complication rate markedly increased by a curative dose of radiation delivered to the larynx.

Lymph node metastasis, the single most important factor in determining prognosis, was found in 32% of the 75 cases reported by Som in 1970.¹⁴ Further, in 14 patients with positive palpable nodes of 2 cm or more, there was a 42% incidence of contralateral microscopically positive nodes. These cases were all treated by horizontal supraglottic laryngectomy, and, therefore, included a large percentage of growths in the anterior supraglottic larynx and epiglottis, where the metastatic rate is less than half of that for lesions in the posterior portion. (32% versus 73% in Baclesse's series.)

By contrast, Smith et al.,¹³ in a survey of 253 supraglottic cancers from seven institutions, reported a 54% rate of cervical metastasis. Most of these cases were treated by total laryngectomy, and undoubtedly included many advanced lesions that were not suitable for supraglottic resection.

Similarly, in the 30 cases reported here, 18 had histologically proven metastases in the cervical nodes (60%). We have not observed a relationship between the tumor's gross or histologic appearance and the incidence of cervical metastasis. Of the 18 cases with metastases, 12 oc-

curred with exophytic lesions and six with ulcerative lesions.

DISCUSSION

The technique and results of horizontal supraglottic laryngectomy as performed in 93 cases of supraglottic cancer between 1951 and 1966 were reported recently.¹⁴ The operation is based upon Alonso's original technique,¹ but with primary closure (Fig. 4). It proceeds in such a way as to allow exposure of the growth well ahead of the point of resection. In this way the supraglottic nature of the growth is constantly verified as the specimen is freed by cutting along normal tissues. At any point the supra-

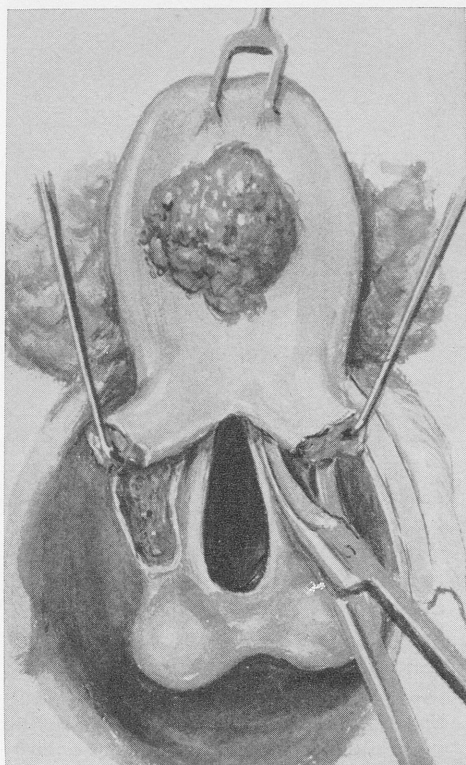


Fig. 4. As the supraglottic resection proceeds anteriorly through normal tissues, the inferior edge of the tumor gradually comes into view, so that its supraglottic nature is constantly verified. At any point this partial resection can be terminated in favor of total laryngectomy if the tumor is found to cross the ventricle or anterior commissure.

glottic resection can be terminated in favor of a total laryngectomy if the tumor is found to cross the ventricle or anterior commissure.

Since horizontal supraglottic laryngectomy results in five-year controls that are virtually the same as for total laryngectomy (68%),^{11,14} it appears to be the treatment of choice for cancer of the laryngeal surface of the epiglottis including the infrahyoid portion,⁶ and for the anterior portion of the false cords and medial surface of the aryepiglottic folds.

Our histologic material supports the concept of a tight barrier at the level of the anterior commissure and ventricle which prevents the downward extension of cancer of the epiglottis and false cords (Figs. 5 and 6). This concept

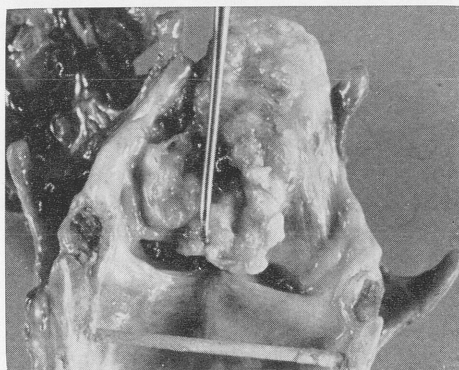


Fig. 5. Large supraglottic cancer extending downward to the anterior commissure but not crossing it.

gains further support from the observation that of the six local recurrences of cancer in 93 supraglottic resections, five were in the base of the tongue, with normal vocal cords.¹⁴

Preepiglottic space invasion by cancer is just as well encompassed by horizontal supraglottic as by total laryngectomy, because most of the hyoid bone is removed with the specimen (Fig. 7). Nevertheless, cancer of the lingual surface of the epiglottis, or cancer extending into the vallecula or base of the tongue is less suitable for horizontal

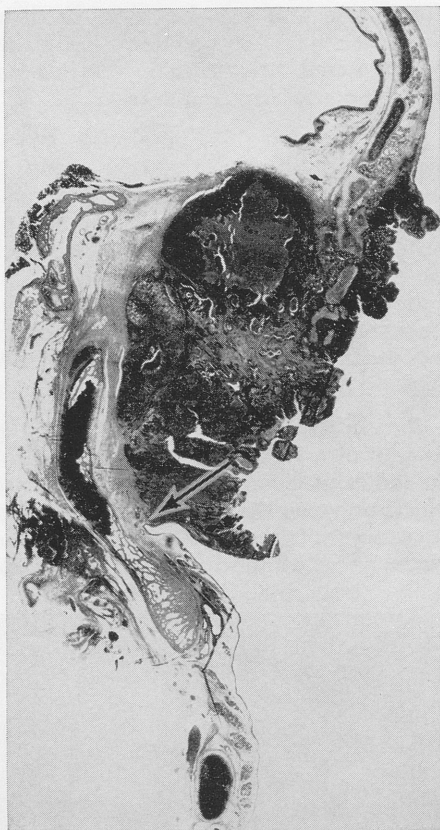


Fig. 6. Midline sagittal section of the tumor shown in Fig. 5. The arrow indicates the lowermost extension of the growth, just above the anterior commissure. Sections made more lateral to the midline showed normal vocal cords and ventricles. These findings are characteristic of the supraglottic growths we have studied by serial sections, and support the observation that gross examination after pharyngotomy is sufficient to define its limits of infiltration. This section also demonstrates the characteristic freedom of the thyroid cartilage from invasion by cancer in supraglottic growths. The lower half of the thyroid cartilage in this section is ossified. The dark portion in the upper half stains blue with hematoxylin and eosin, and represents normal cartilage.

supraglottic laryngectomy. This is not because the resection would be inadequate, but because resection of the base of the tongue adds tremendously to the

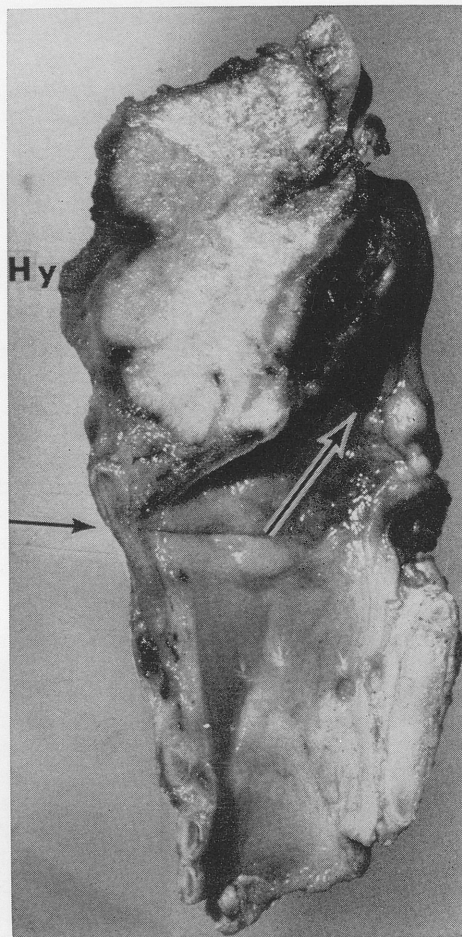


Fig. 7. Gross involvement of the pre-epiglottic space does not contraindicate conservation surgery, since the hyoid bone is removed with the supraglottic structures as a unit. Even with extensive infiltration as shown here, horizontal supraglottic laryngectomy as indicated by the arrows would have allowed generous margins. Because of the upward extension of cancer, however, a large part of the base of the tongue had to be included in the resection. Although the upper margin could have been the same, whether partial or total laryngectomy had been performed, it was considered inadvisable to submit this elderly patient to the additional postoperative swallowing difficulties that partial glossectomy would have added to horizontal supraglottic laryngectomy.

problem of rehabilitating the patient's swallowing mechanism after removal of the supraglottic structures.

CONCLUSIONS

1. Supraglottic cancer does not invade the thyroid cartilage.

2. Cancer of the epiglottis or false cord that extends downward below the anterior commissure is no longer supraglottic, but transglottic, and frequently invades the thyroid cartilage.

3. Downward extension of tumor below the anterior commissure requires total laryngectomy. It can usually be recognized by lateral laryngography before operation, and definitely identified as the larynx is being opened after lateral pharyngotomy.

4. Gross examination of the inferior extent of a lesion after pharyngotomy is sufficient to define its limits of infiltration. In not a single instance was the line of resection violated inferiorly by cancer when subtotal horizontal laryn-

gectomy was performed after adequate exposure. This has been confirmed by our histologic studies.

5. Involvement of perichondrium by cancer at the time of operation is an indication that the growth is transglottic, and not suitable for conservative surgery.

6. Since most of the hyoid bone is removed as part of the horizontal supraglottic laryngectomy specimen, the pre-epiglottic space can be just as well encompassed by this procedure as by total laryngectomy.

7. Extension of tumor along the medial surface of the aryepiglottic fold as far as the arytenoid does not preclude conservation surgery. When the arytenoid itself is involved, the tumor is not supraglottic but laryngopharyngeal.

8. The greatest single cause of failure is cervical node metastasis. If a positive palpable cervical node is present at the time of operation on the side of the tumor, a contralateral elective neck dissection is indicated.

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DISCUSSION

DR. DOUGLAS P. BRYCE: I believe this technique developed by Dr. Tucker followed by Dr. Kirchner and others has undoubtedly been the basis of most of the modern advance in surgery of the larynx. However, in this discussion, John, I'm confused. You say that the supraglottic lesions do not extend beyond the ventricle or below the anterior commissure. If they do so, then they are becoming transglottic. I would like you to clarify.

DR. JOHN A. KIRCHNER (closing): Yes, thank you, Dr. Bryce.

This is a tough one to answer and it gets into the problem of classification of what is a supraglottic cancer, and when does it become transglottic. Now, the point we were trying to make is this — that those tumors that invade the laryngeal framework are those that cross below the anterior commissure or ventricle even if 95 per cent of the tumor is in the supraglottic larynx or false

cord and that this can be determined by visual inspection, or by lateral laryngography. In not a single case in which the lower edge of the tumor by visual inspection (after the pharynx was opened) was above the attachment of the cords was the cartilage invaded. Now, I think regardless of the terminology, whether you call the growth that extends from the supraglottis down below the cord a "T-3" supraglottic or whether you call it a transglottic lesion, its behavior nevertheless is the same and the point that we hope to make is that this can be easily determined by inspection at operation. Not in a single case that we have studied did the gross appearance mislead us.

Further, if one can see that the lower edge has not crossed the attachment of the cord, we have not found submucosal invasion below the cord or into the laryngeal framework in any case.

Does that help?

ELECTIVE NECK DISSECTION FOR PHARYNGEAL AND LARYNGEAL CANCERS

AN EVALUATION

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Laryngeal surgeons have advocated elective (prophylactic) neck dissection for patients with laryngeal and laryngopharyngeal cancers in an effort to improve the survival rate.^{2-5,7,8,10} The assumption that removing an occult node is more curative than waiting for nodes to become palpable is based on clinical experience. Unfortunately, there is no statistical evidence that this clinical assessment is valid.

Reed and Miller⁹ have emphasized the lack of statistical data supporting the elective neck dissection and have suggested that it may be immunologically detrimental.

This paper presents a retrospective study of a large series of laryngeal and laryngopharyngeal cancers, treated by primary resection and neck dissection, in an attempt to evaluate the effect of the elective neck dissection.

One proposition has to be accepted in a retrospective evaluation of the elective neck dissection. An occult node will, in time, become palpable.

The purpose of the elective neck dissection is to remove occult cervical metastases, thereby decreasing the incidence of neck recurrence, when compared with the incidence of neck recurrence following a neck dissection for palpable nodes. This decrease in neck recurrence will be reflected in an increased survival rate, assuming all, or

the majority of patients with neck recurrence following radical neck dissection die. The percentage of survival rate improvement will be dependent upon the incidence of occult cervical metastasis for that specific tumor classification.

If the elective neck dissection for occult nodes does not decrease neck recurrence, or is immunologically detrimental, there will be no change in the survival rate, or decrease in the survival rate, respectively, when compared with patients undergoing neck dissection for palpable nodes.

METHODS AND MATERIALS

Four hundred eleven cases of laryngeal or laryngopharyngeal cancers, treated at McMillan Hospital, Washington University, from 1955 to 1967 were reviewed. The treatment consisted of partial or total laryngectomy with ipsilateral or bilateral neck dissection. In order to obtain a more homogenous sample, 63 patients who either presented with bilateral nodes or in whom delayed contralateral nodes developed were excluded. The remaining 348 patients, who were treated by primary tumor resection with ipsilateral neck dissection whether or not nodes were palpable, were analyzed in this study. One hundred seventy-nine patients received planned low dose irradiation (1500-3000r) prior to surgery.

The entire group of cases is considered homogenous, in that all the cases were examined, classified, and operated upon or the operation supervised by the senior author.

The cancers are classified as supraglottic,

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TABLE I
SUPRAGLOTTIC CANCERS: 110 Cases

	<i>Incidence of Neck Recurrence</i>	<i>Survival Rate</i>
Negative nodes	0/96 (0%)	65/80 (81%)
Occult nodes	0/16 (0%)	10/16 (62%)
Palpable nodes	4/14 (28%)	7/14 (50%)

Effect of elective neck dissection on survival rate — 4%

TABLE II
PYRIFORM SINUS CANCERS: 98 Cases

	<i>Incidence of Neck Recurrence</i>	<i>Survival Rate</i>
Negative nodes	9/29 (0%)	16/29 (55%)
Occult nodes	0/18 (0%)	11/18 (61%)
Palpable nodes	11/51 (21%)	14/51 (27%)

Effect of elective neck dissection on survival rate — 11%

transglottic (true and false cords only), pyriform sinus, base of tongue and vallecula, posterior pharyngeal wall and glottic (fixed cord). Cervical metastases were considered present only if there was histologic evidence of tumor.

The incidence of neck recurrence and the survival rate were determined and compared for occult, palpable and negative nodes, in each tumor classification.

Only patients who had neck recurrence, without evidence of primary recurrence, were considered in the group of neck recurrences.

The influence of neck recurrence in the presence of occult, palpable or negative nodes on the survival rate was determined by the probability model presented in the Appendix.

Patients lost to follow-up or dying within three years following surgery were considered failures for survival rate determinations.

RESULTS SUPRAGLOTTIC CANCERS

One hundred ten cases of supraglottic cancers are included (Table I). No patients were lost to follow-up. Cervical metastases were present in 30/110 (27%); occult nodes 16/96 (16%); palpable nodes 14/110 (12%); negative nodes 80/96 (83%). The incidence of neck recurrence for each group is: oc-

cult nodes 0/16 (0%), palpable nodes 4/14 (28%), negative nodes 0/96 (0%). The incidence of neck recurrence with palpable nodes is significantly greater than with occult nodes ($P < .01$).

The three-year absolute survival rate for the three groups is: occult 10/16 (62%), palpable 7/14 (50%), and negative nodes 65/80 (81%).

The survival rate is significantly greater with occult nodes when compared with survival with palpable nodes ($P = < .05$).

The estimation of the decrease in the survival rate, if the occult nodes were allowed to become palpable before neck dissection was performed is 4%; 78% to 74%.

PYRIFORM SINUS CANCERS

Ninety-eight cases of pyriform sinus cancers are included (Table II). Eleven patients were lost to follow-up (7 with palpable nodes; 1 with occult nodes; 3 with negative nodes). Cervical metastases were present in 69/98 (70%); occult nodes 18/47 (38%); palpable nodes 51/98 (52%); negative nodes 29/47 (61%). The incidence of neck recurrence for each group is: occult

TABLE III
BASE OF TONGUE AND VALLECULA: 34 Cases

	<i>Incidence of Neck Recurrence</i>	<i>Survival Rate</i>
Negative nodes	0/17 (0%)	16/17 (94%)
Occult nodes	0/5 (0%)	3/5 (60%)
Palpable nodes	4/12 (33%)	2/12 (16%)

TABLE IV
TRANSGLOTTIC CANCERS (True and False Cord Only)

	<i>Incidence of Neck Recurrence</i>	<i>Survival Rate</i>
Negative nodes	0/46 (0%)	31/46 (67%)
Occult nodes	0/6 (0%)	2/6 (33%)
Palpable nodes	1/11 (9%)	7/11 (63%)

nodes 0/18 (0%), palpable nodes 11/51 (21%), negative nodes 0/29 (0%). The incidence of neck recurrence with palpable nodes is significantly greater than with occult nodes ($P < .001$).

The three-year absolute survival rate for the three groups is: occult nodes 11/18 (61%), palpable nodes 14/51 (27%), negative nodes 16/29 (55%). The survival rate is significantly greater with occult nodes when compared with palpable nodes ($P < .01$). The survival rate remains significant ($P < .05$) regardless of how the patients lost to follow-up are considered (alive, dead or excluded).

The estimation of the decrease in the survival rate, if the occult nodes were allowed to become palpable before a neck dissection was performed is 11%; 57% to 46%.

BASE OF TONGUE AND VALLECULA

Thirty-four cases of cancer of the base of tongue and vallecula are included (Table III). One patient with palpable nodes was lost to follow-up. Cervical metastases were present in 17/34 (50%); occult 5/22 (22%); palpable nodes 12/34 (35%); negative nodes 17/22 (77%).

The incidence of neck recurrence for each group is: occult nodes 0/5 (0%),

palpable nodes 4/12 (33%), negative nodes 0/17 (0%). The incidence of neck recurrence with palpable nodes is significantly greater than with occult nodes ($P < .05$).

The three-year absolute survival rate for the three groups is: occult 3/5 (60%), palpable nodes 2/12 (16%), negative nodes 16/17 (94%). The survival rate is significantly greater with occult nodes when compared with the survival rate with palpable nodes ($P < .01$).

This group of cases is too small to calculate the change in survival rate due to the elective neck dissection.

TRANSGLOTTIC CANCERS (TRUE AND FALSE CORD ONLY)

Sixty-three cases of transglottic cancers are included (Table IV). Four patients were lost to follow-up (palpable nodes 1, occult nodes 1, negative 2). Cervical metastases were present in 17/63 (26%); occult nodes 6/52 (11%); palpable nodes 11/63 (17%); negative nodes 46/52 (88%).

The incidence of neck recurrence for each group is: occult nodes 0/6 (0%), palpable nodes 1/11 (9%), negative nodes 0/46 (0%). The incidence of neck recurrence with palpable nodes is not

TABLE V
GLOTTIC CANCERS (FIXED CORD) 37 Cases

	<i>Incidence of Neck Recurrence</i>	<i>Survival Rate</i>
Negative nodes	1/32 (3%)	21/32 (65%)
Occult nodes	0/1 (0%)	0/1 (0%)
Palpable nodes	1/4 (25%)	1/4 (25%)

TABLE VI
POSTERIOR PHARYNGEAL WALL CANCERS: 6 Cases

	<i>Incidence of Neck Recurrence</i>	<i>Survival Rate</i>
Negative nodes	0/1 (0%)	1/1 (100%)
Occult nodes	0/2 (0%)	0/2 (0%)
Palpable nodes	2/3 (66%)	0/3 (0%)

significantly greater than with occult nodes.

The three-year absolute survival rate for the three groups is: occult 2/6 (33%), palpable nodes 7/11 (63%), negative nodes 31/46 (67%).

The survival rate is not significantly different when comparing the occult group with the palpable group.

The number of cases with occult nodes is too small to calculate the effect on the survival rate of the elective neck dissection.

GLOTTIC CANCERS (FIXED CORD)

Thirty-seven cases of glottic cancer are included (Table V). No patients were lost to follow-up. Cervical metastases were present in 5/37 (13%); occult nodes 1/33 (3%); palpable nodes 5/37 (13%); negative nodes 32/33 (96%).

The incidence of neck recurrence for each group is: occult nodes 0/1 (0%), palpable nodes 1/4 (25%), negative nodes 1/32 (3%). The incidence of neck recurrence with palpable nodes is not significantly greater than with occult nodes.

The three-year absolute survival for the three groups is: occult nodes 0/1 (0%), palpable nodes 1/4 (25%), negative nodes 21/32 (65%).

The survival rate is not significantly different when comparing the occult group with the palpable group.

The number of cases with occult nodes is too small to calculate the effect of the elective neck dissection on the survival rate.

POSTERIOR PHARYNGEAL WALL CANCERS

Six cases of posterior pharyngeal wall cancers are included (Table VI). No patients were lost to follow-up. Cervical metastases were present in 5/6 (83%); occult nodes 2/3 (66%); palpable nodes 3/6 (50%); negative nodes 1/6 (16%).

The incidence of neck recurrence for each group is: occult nodes 0/2 (0%), palpable nodes 2/3 (66%), negative nodes 0/1 (0%).

The three-year absolute survival rate for the three groups is: occult nodes 0/2 (0%), palpable nodes 0/3 (0%), negative nodes 1/1 (100%).

The number of cases is too small for calculation.

DISCUSSION

The incidence of occult metastases with glottic cancers (fixed cord) and transglottic cancers (true and false cord involvement only) is 1/33 (3%) and 6/52 (11%), respectively. This low in-

cidence does not significantly decrease the number of neck recurrences, when compared with neck recurrences with palpable metastases. Therefore, elective radical neck dissection in these cancers had no influence on the survival rate.

The incidence of occult nodes with supraglottic cancers, in this series, is 16%. Neck recurrence was significantly less with occult nodes than when nodes were palpable. This is reflected in a significantly increased survival rate. The increase in the survival rate, due to the elective neck dissection is 4%, from 74% to 78%. This is a low estimate considering that the group excludes those patients with ipsilateral nodes who later developed contralateral nodes (24 patients). To increase the survival rate 10%, 74% to 84%, the incidence of occult nodes would have to be 26%. This is the approximate occult metastatic rate for lesions which involve epiglottis and false cords (T_2 supraglottic cancers).

The pyriform sinus cancers with occult nodes exhibit a significant decrease in neck recurrence when compared with those having palpable nodes. This is reflected in a significant increase in the survival rate for patients in the occult group. The effect of elective neck dissection improved the survival rate 11%, 46% to 57%. It would appear if the series of base of tongue and vallecula cancers were larger, similar results might be expected.

The base of tongue and vallecula group does not include eight patients with ipsilateral nodes in whom late contralateral nodes developed. The effect of the addition of these eight patients to the group would increase the incidence of occult nodes.

The posterior pharyngeal wall cancers are too few in number to analyze.

The effect of the elective neck dissection on the survival rate, as determined by the incidence of neck recurrence for supraglottic and pyriform cancers, probably represents a conservative estimate. The incidence of neck recurrence is calculated by the number of neck recur-

rences divided by the total number of occult or palpable nodes. This latter figure includes those patients dying of intercurrent disease, and, therefore, includes patients who did not live long enough to develop neck recurrence.

Even though this series represents a large group of cases, the results must be interpreted with caution. The following factors must be considered in evaluating the study.

One hundred seventy-nine patients received low dose irradiation (1500-3000r) prior to surgery. This probably has minimal influence on the results since prior analysis of this group indicated the incidence of neck recurrence was not influenced by preoperative irradiation.^{1,6}

If preoperative irradiation did influence the cervical metastases, the effect would have been to change the occult node to a negative node. This would make the present results more reliable, since the incidence of occult nodes would be decreased.

The probability model for survival (see Appendix) used in this study assumes that if a patient with an occult node does not have a neck dissection until the node is palpable, the patient's risk of death due to neck recurrence is similar to the risk of death due to neck recurrence in the group which presented initially with a primary and palpable nodes.

This assumption may not be completely valid, for the latter group includes patients with large nodes and multiple nodes. The risk of death due to neck recurrence in this group may be greater than the risk of death due to neck recurrence in a patient with an occult node who has a neck dissection when the node initially becomes palpable.

Although, theoretically, this may be true, it is fallacious to assume that all patients with occult nodes not having an elective neck dissection would return for diagnosis at the first evidence of the smallest palpable node. Experience indicates that patients who are being fol-

lowed for possible neck metastases will return frequently with large nodes, in spite of examination at one to two month intervals. Large fixed nodes occurred in one-third of the patients in whom nodes developed after hemilaryngectomy or in whom delayed contralateral neck nodes developed with laryngeal and laryngopharyngeal cancers.

It is assumed in this study that patients with neck recurrence following neck dissection will die. There are exceptions, but these are rare, and the effect on the present statistical analysis will be minimal. In this study one of

the patients who developed neck recurrence survived with further therapy.

The mortality attributed to the elective neck dissection for negative nodes in this group is less than 1% (2/221 elective necks). This mortality rate applied to the patients undergoing unnecessary neck dissections does not influence the results.

The individual surgeon must determine what percentage increase in the survival rate warrants elective neck dissection. It would appear that increasing the survival rate 5% to 10% is sufficient to indicate elective neck dissection.

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APPENDIX

A PROBABILITY MODEL FOR SURVIVAL IN RADICAL NECK SURGERY AND THE ESTIMATION OF EXPECTED SURVIVAL IN ELECTIVE NECK SURGERY

REIMUT WETTE, D.Sc.

In analyzing the survival over a fixed time interval of patients that have undergone radical neck surgery, as discussed in the main paper, we may differentiate between two primary causes, or risks, of death: 1) Death due to re-

current neck disease. This risk will presumably depend on the degree of node involvement at the time of surgery. 2) Death due to other causes, such as intercurrent disease, complications following surgery, "natural" or accidental

death, and so on. This risk can (and, here, will) be assumed to be independent of the degree of node involvement.

Let us assume that the risk, or probability of death due to other causes is equal to p , that the risk due to recurrent neck disease alone is equal to zero in patients without node involvement, that it is equal to q ($0 < q < 1$) in patients with occult (nonpalpable) node involvement, and that it is equal to r ($0 < r < 1$) in patients with palpable node involvement. Let us also assume that the two risks are nonexclusive and independent, or competitive in the sense that a patient with node involvement is simultaneously subject to both risks. The survival probabilities $P(S | G)$ for the three groups with $G = A$ for uninvolved, $G = B$ for occult, and $G = C$ for palpable node involvement then become

$$P(S | A) = 1 - p, \quad (1)$$

$$P(S | B) = (1 - p)(1 - q), \text{ and } (2)$$

$$P(S | C) = (1 - p)(1 - r). \quad (3)$$

Given a sample of N_A patients with no node involvement of whom n_A died, a sample of N_B patients with occult node involvement of whom n_B died, and a sample of N_C patients with palpable node involvement of whom n_C died, estimates p , q , r for the risks due, respectively, to other causes, occult involvement alone, and palpable involvement alone are obtained by the method of maximum likelihood as

$$p = \frac{n_A}{N_A} \quad (4)$$

$$q = \frac{n_B/N_B - p}{1 - p} \quad (5)$$

$$r = \frac{n_C/N_C - p}{1 - p} \quad (6)$$

where q or r are set equal to zero if the respective numerator is nonpositive.

Let us now consider the hypothetical situation that the patients with nonpalpable nodes receive firstly only resection of the primary tumor and delayed node

excision when and only when the nodes become palpable at some time after primary surgery. If we stipulate that the risk p due to other causes remains numerically the same, and that the risk r due to palpable involvement alone now applies to the patients which had occult node involvement at primary surgery, the survival probability $P(S | G)$ in this group $G = CB$ of patients would simply become

$$P(S | CB) = P(S | C) = (1 - p)(1 - r), \quad (7)$$

the same as in (3). A more conservative approach would assume that the survival probability $P(S | CB)$ should be no greater than the smaller of the two survival probabilities $P(S | B)$, $P(S | C)$. This requirement would be satisfied if we were to assume that the patients in group CB are now, in addition to the always present risk p due to other causes, subject to both the risks due to occult and due to palpable node involvement, viz., q and r . Herewith, the survival probability would become

$$P(S | CB) = (1 - p)(1 - q)(1 - r) = S_{CB}, \text{ say, } (8)$$

and $S_{CB} < \min [P(S | B), P(S | C)]$ for $0 < p, q, r < 1$ as required.

An estimate S_{CB} for this adjusted survival probability S_{CB} is obtained by replacing p , q , and r in (8) by their estimates p , q , r given in (4)-(6):

$$S_{CB} = (1 - p)(1 - q)(1 - r) = \frac{N_B - n_B}{N_B} \frac{N_A}{N_A - n_A} \frac{N_C - n_C}{N_C}. \quad (9)$$

Since it was assumed above that patients with no histologic node involvement have zero node involvement risk, the only group needing adjustment for delayed node excision survival probability is the occult node group B , the survival probabilities for the other two groups A and C remaining the same. An estimate S_T for the total survival experience S_T of the three groups when adjusted for delayed node excision is then obtained as

$$S_T = \frac{(N_A - n_A) + N_B}{N_A + N_B} \cdot \frac{S_{CB} + (N_C - n_C)}{+ N_C}, \quad (10)$$

and an estimate $S_{A,CB}$ for the adjusted survival experience $S_{A,CB}$ of the two non-palpable node groups is, correspondingly,

$$S_{A,CB} = \frac{(N_A - n_A) + N_B \cdot S_{CB}}{N_A + N_B} \quad (11)$$

It should be noted that the estimates (10) and (11) are not only functions of the estimates p , q , and r , but also depend on the distribution of the number of patients N_A , N_B , and N_C , in the three groups. Thus, for example, the estimated survival experience $S_{A,CB}$ depends on the

observed proportion $R = N_B / (N_A + N_B)$ of patients with occult node involvements among the nonpalpable node group:

$$S_{A,CB} = \frac{(1-R) \cdot (1-p)}{1-p-[1-(1-q)(1-r)]} + R \cdot S_{CB} = \quad (12)$$

Equation (12) can be used, for example, to estimate for given p , q , and r values, what values $R < R^*$ would be required to keep $S_{A,CB}$ above a given critical value $S_{A,CB}^*$:

$$R^* = \frac{1-p - S_{A,CB}^*}{1-(1-q)(1-r)} \quad (13)$$

where R^* is equal to zero or unity when the fraction on the right side of (13) is negative or greater than unity, respectively.

DISCUSSION

DR. FRANKLIN KEIM: As usual, Dr. Ogura has come up with a rather impressive bit of evidence on a subject that has bothered a lot of us for a long time. I am glad that he has, because just about ten days ago in Vancouver at the Society of Head and Neck Surgery, the concept of elective neck dissection was challenged by a number of members of that society. They presented the same kind of approach to it that has gone back a good many years to the days of Dr. Hayes Martin when he and others attempted to show that elective neck dissection in patients with lesions that are very prone to metastasize did not significantly alter the survival rate. To my knowledge, this is the first firm evidence that has been offered that such a procedure in these patients favorably influences the result.

I think it is a very significant paper and I would like to congratulate Dr. Ogura on his presentation.

DR. GEORGE REED: I am a bit confused. As I first listened to Dr. Ogura's paper, I thought that he was going to end up seriously questioning the routine value of elective neck dissections, but he ended up, I think, with the tone that his formula and figures show the importance of elective neck dissection.

As some of you may recall, we stuck our neck out a year ago at the Triological meeting to question the routine use of neck dissections. We did this on a rather unscientific basis, by going back into the literature, pooling all of the results in carcinoma of the larynx that were sufficiently detailed to determine the cause of death. This review showed that 72 per cent would never develop a secondary metastasis; ten of the residual 28 would be cured by a secondary radical

neck dissection and another ten would not necessarily be cured, in the usual sense, would die of other causes but without recurrence in the neck and therefore would not have been helped by elective neck dissection.

In essence, when you boil this all down it turns out that you have a maximum of seven patients out of 100 who would be potentially helped by an elective radical neck dissection. These findings fit some of the series that have been sufficiently well controlled to be able to study this, and it is remarkable how closely this comes out with the figures that Dr. Ogura has given us today.

Now, using his formula, which I am glad he can understand, four per cent of supraglottics would be potentially helped by neck dissection, and 11 per cent of pyriform sinus cases. Averaging these two you come up with seven, the same as we did.

Now, even if we accept this, we must accept the fact that you will be doing roughly seventy unnecessary neck dissections to *potentially* save seven, and there is no proof that these seven would be saved by elective neck dissection.

I think we have to stop and think, is the morbidity worth seventy unnecessary neck dissections to save seven.

DR. LOUIS H. CLERF: I was pleased to see on the program Dr. Ogura's paper on elective neck dissection. My interest in this procedure dates back to 1942 when reviewing a series of laryngectomized patients totalling 287, it was found that metastasis to regional lymph nodes had occurred in 67, or 23 percent of the group.

Unfortunately, it is impossible to personally

observe all patients postoperatively at stated intervals. Many referring physicians who examine them often do not fully appreciate the significance of a lump appearing in the neck. As a result patients often return with extensive metastasis. In spite of appropriate treatment in the 77 patients, namely block dissection, irradiation and palliation, there was a salvage rate of but 15 per cent, a mere handful.

I began elective neck dissection in selected cases in 1942. In 17 consecutive cases carcinoma was demonstrated histologically in cervical lymph nodes in six (35 percent). All were free from carcinoma five or more years later. During the five years before I retired I did 34 additional cases of elective neck dissection. Demonstrable metastasis was noted in seven cases (21%). There was no mortality in any of the elective group and I would not consider the morbidity as serious.

Metastasis to regional lymph nodes following laryngectomy is evidence of failure of our diagnostic aids to recognize that the carcinoma no longer is a local lesion. The laryngologist must determine this largely on the basis of the location, extent and duration of the primary lesion plus his clinical judgment. With the present classification of laryngeal carcinoma a more comprehensive picture of this problem probably may be obtained. In my opinion elective neck dissection is a sound surgical procedure in laryngeal carcinoma.

DR. CHARLES NORRIS: This, as usual, has been a very well-documented and thoughtful analysis given by Dr. Ogura.

The problem of determining what would have happened, "if," is always difficult. I think the scientific and statistical approach which Dr. Ogura has outlined certainly is worthwhile.

Others have attempted to grapple with this same problem by comparing groups of cases. In our own series we attempted to find an answer by comparing the group of classified cases treated by elective neck dissection, since elective neck dissection came into vogue, with a similar group treated in the absence of palpable nodes by laryngectomy without elective neck dissection.

Our apparent improvement in survival was somewhat greater than has been documented by Dr. Ogura. There are probably reasons for this which we do not understand.

The question I would like to raise is this — I believe in his conclusion, Dr. Ogura felt that an improvement in survival rate of five per cent or more might justify elective neck dissection. Now, does this mean that since the supraglottic cases apparently according to the statistical analysis showed only an expected improvement of four per cent that the use of elective neck dissection should, in general, be abandoned in supraglottic cases?

And, there are two corollaries to this question:

One is, must not some allowance be made for the extent of the supraglottic lesions? In other words, with a more advanced case you might do an elective neck dissection, or a less advanced one, not do an elective neck dissection.

The other corollary would be in the case of supraglottic "partials," I'd like to have Dr. Ogura tell us what he feels about these two points.

DR. JOSEPH H. OGURA (closing): First, let me thank Dr. Keim for his remarks. You know a retrospective study of this type requires a rigid statistical approach. We knew if we approached it by using and comparing case for case we could not show the right evidence.

To Dr. Reed, let me say this was a homogeneous group that has been studied and the conclusions I feel, for pyriform lesions, are warranted. We have a definite 11 per cent improvement, enough for the proposition of composite elective neck dissection with removal of the primary lesion. The five per cent that we referred to are for the supraglottic lesion. There is an influencing factor in that preoperative irradiation was used. I mentioned that this might influence the changing the node from an occult to a negative node; therefore the actual incidence of an occult node may be higher.

I certainly would not do an elective neck dissection for a glottic lesion with a fixed cord. I have abandoned this some time ago. For a transglottic lesion, I think it is questionable. We do not have enough evidence for that.

It is warranted only for example for the supraglottic lesion and this would probably answer Dr. Norris's question. We perform an elective neck dissection only when the primary lesion is large, T-2, T-3 or T-4 lesions, certainly not for T-1 lesions.

I am very grateful to Dr. Clerf because in my youth back in 1950 before the Academy I presented this work on laryngectomy and elective neck dissection, and I realized at that time how little I knew about the pathology and mixed up "potatoes with carrots." It is only after a few years of experience that you learn something about cancer of the larynx and pharynx and that you find out that the metastatic rate changes depending on the location.

In conclusion, let me say that we have excluded the ipsilateral positive nodes. Ipsilateral nodes which later develop contralateral nodes include a large number of epiglottic lesions. Elective neck dissection on the contralateral side would seem indicated if ipsilateral nodes are positive.

HYPERBARIC PERFUSION OF THE ISOLATED LARYNX

AN AUTORADIOGRAPHIC STUDY

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Previous investigators¹⁻⁶ have shown that laryngeal tumor cell viability can be demonstrated by incubating fresh tissue slices with tritiated thymidine and preparing autoradiographs. It was hoped that by developing a method of laryngeal perfusion, that topographical differences in cell viability might be demonstrated, using autoradiographs of the serially sectioned larynx. It was also hoped the method of perfusion could be used for short term preservation of the larynx prior to transplantation. Three groups of patients have been studied: 1) those that have had surgery without irradiation; 2) those that had a preoperative course of irradiation followed by elective surgery; and 3) those having surgery for recurrence after a previous course of irradiation.

EQUIPMENT

A laryngeal perfusion apparatus was designed and developed in our laboratory. It is a closed circuit system (Fig. 1) and consists of a hyperbaric oxygen chamber, a variable flow masterflex tube pump and a temperature controlled water bath. Oxygen is supplied to the chamber at three atmospheres absolute pressure from an oxygen cylinder. Perfusion pressure is monitored by a differential pressure gauge. The whole apparatus is mounted on a utility cart for mobility (Fig. 2).

Hyperbaric conditions were chosen

for two reasons: first, experiments have shown that pigs could be kept alive by exchanging their blood with plasma at 3 atmospheres pressure,² and second, that under hyperbaric conditions, the depth of the zone of incorporated tritiated thymidine is increased.⁴ Acellular perfusates have been used in all our experiments; first, frozen plasma, and second, a 4:1 mixture of Ringers lactate and rheomacrodex. The pH of this mixture was adjusted to 7.4 by adding 5% sodium bicarbonate and it was heparinized. We changed to a mixture of Ringer's lactate and rheomacrodex because of difficulties in obtaining supplies of plasma.

The apparatus and perfusates have been thoroughly evaluated with a series of rabbit kidney and dog laryngeal perfusions.

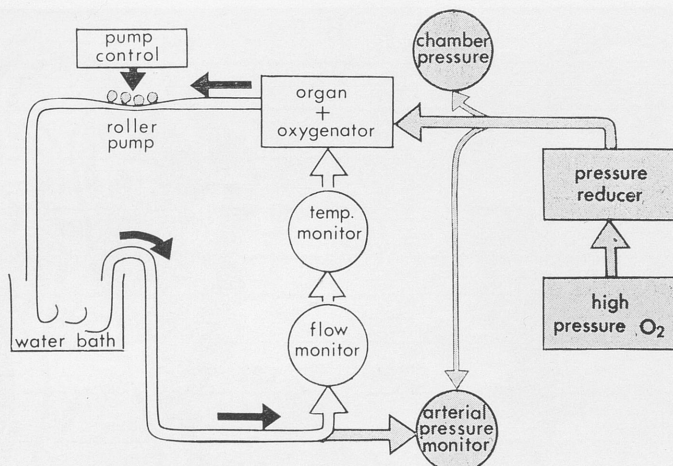
METHOD

TECHNIQUE OF PERFUSION

At surgery, as many as possible of the major vessels supplying the larynx are preserved and clearly marked with long ligatures. Meanwhile, the perfusion apparatus is taken to the operating room and primed with 500 cc of the perfusate. The solution is thoroughly oxygenated, the temperature adjusted to 37°C and 0.5 mc of tritiated thymidine, specific activity 15 curies per mmole, are added to the mixture. The larynx is removed and the marked arteries identified and cannulated with as large diameter polyethylene

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LARYNGEAL PERFUSION UNIT

Fig. 1. Circuit diagram of perfusion apparatus.

cannulae as possible. If it is possible, the right and left superior thyroid or laryngeal vessels are preferred. The cannulated arteries are injected with methylene blue and the laryngeal mucosa observed for staining. They are then washed with a separate portion of the perfusate, not containing tritiated thymi-

dine, at 37°C until clear fluid issues from the veins. At the same time, any major cut vessels in the larynx are ligated. The cannulated vessels are connected to the Y terminal inside the perfusion chamber, the chamber is gradually pressurized and the perfusion pump adjusted so that the differential pressure gauge reads 90 mm Hg. The perfusion is continued for two hours with constant checking of the temperature and arterial pressure. At the end of the perfusion, the specimen is thoroughly washed with Ringers lactate solution and after photography is fixed in formalin.

TECHNIQUE OF AUTORADIOGRAPHY

Theory. The human cell cycle consists of four phases, and it is in the S phase, which is of about eight hours duration, that thymidine, a precursor of DNA, is incorporated into the nucleus.¹⁶ Viable cells will only take up tritiated thymidine during the S phase. The specimen is fixed, decalcified, sectioned and suitable sections are photographically exposed and developed.

Technique. After perfusion the larynx is fixed in formalin, decalcified, embedded in paraffin, and serially sectioned. (The technique was originally used with celloidin embedded material, but the method was fraught with so many technical problems that it was abandoned.) The sections are mounted on specially cleaned lantern slides. Alternate sections are stained with hematoxylin and eosin, and only those that contain histologically

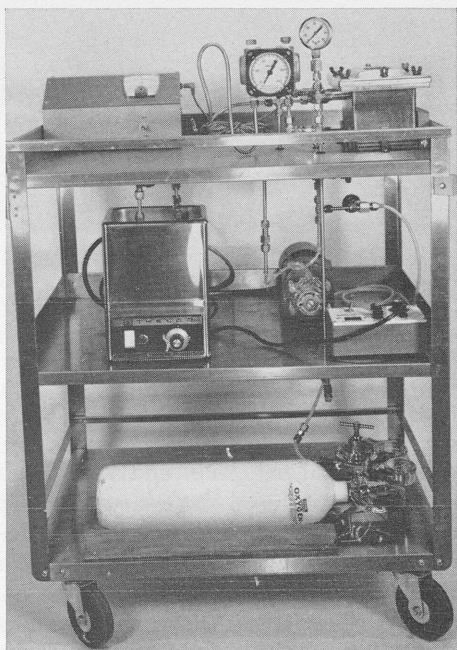


Fig. 2. Perfusion apparatus mounted on utility cart.

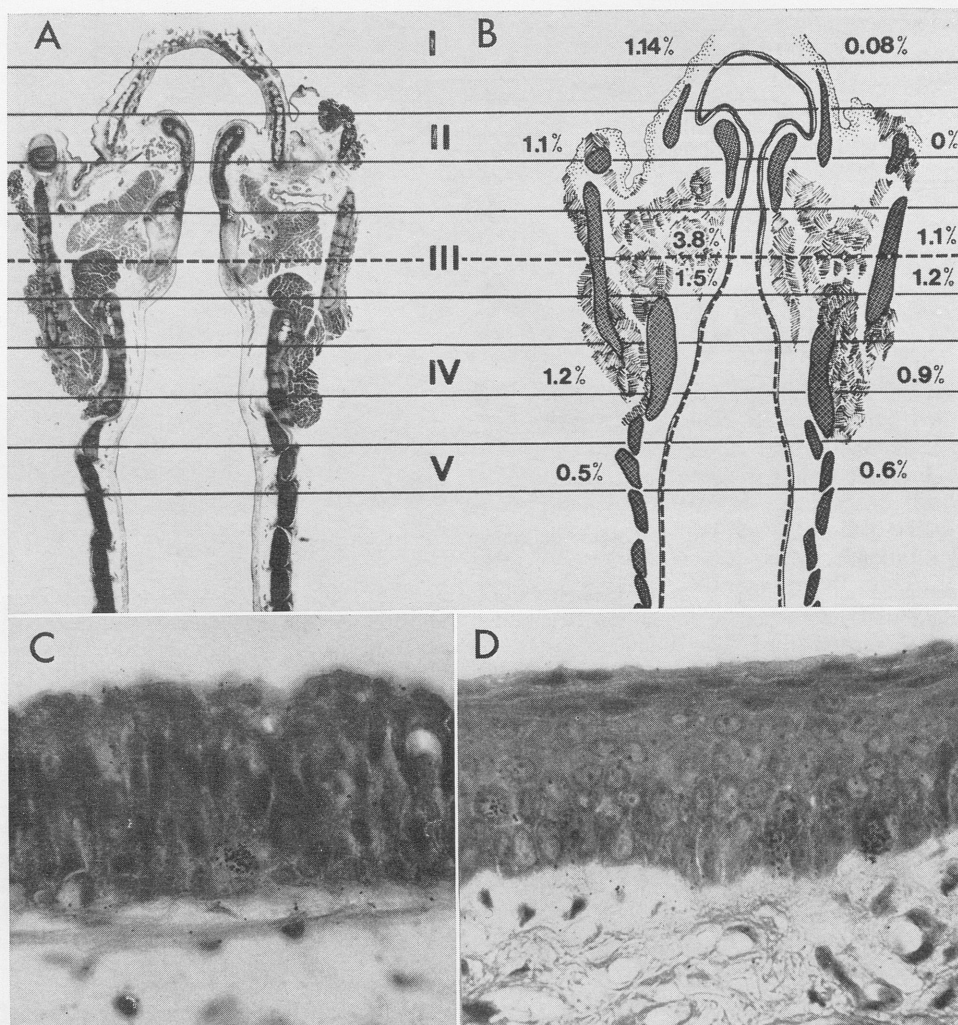


Fig. 3A. Coronal section of dog's larynx at level of the posterior one-third of the vocal cord. B. Labelling index of normal epithelium. C. High power view of labelling of columnar epithelium below the level of the cords. Note labelling in basal layer. X400. D. High power stratified squamous epithelium showing labelling confined to basal layer. X400.

viable tumor are exposed and developed. The liquid emulsion dipping method, for high resolution autoradiography, using Kodak NTB 2 emulsion has been used.^{12,17} Exposure time is three weeks at 4°C using specially designed containers to accommodate the large lantern slides. The autoradiographs are developed using Kodak Dektol developer. The prepared sections are lightly stained with Ehrlich's hematoxylin and eosin and examined for the presence of labelling.

RESULTS

The normal pattern of epithelial labelling in the perfused dog's larynx is seen in Figure 3B. A labelling index was obtained by counting the labelled cells in ten random sites of 100 cells each, in each of the marked areas on the section. The number of labelled cells in each of the areas was then expressed as a percentage, which was the labelling

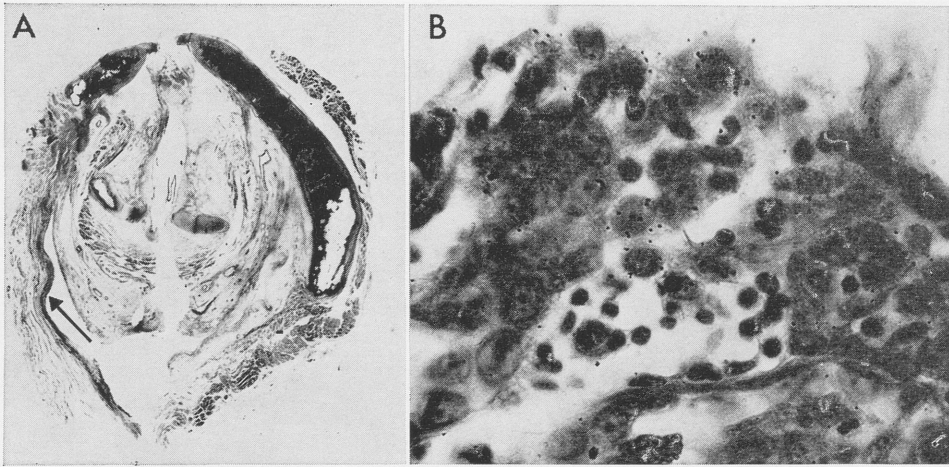


Fig. 4A. Horizontal section through level of vocal cords. B. Labelling in dysplastic epithelium from area marked with arrow in low power view. Note labelling throughout the dysplastic epithelium in contrast to labelling in Fig. 2 normal epithelium. X400.

index of the area under study. It can be clearly seen that the labelled cells are present in the basal layer only of the normal columnar (Fig. 3C) and stratified squamous epithelium (Fig. 3D). The normal labelling index of the epithelium is not high as demonstrated by this method, but was uniform throughout three coronal sections from representative areas of the larynx. Four dog larynxes have been successfully perfused and were autoradiographically positive.

REPORT OF CASES

Case 1. In 1950, a 62-year-old female had a carcinoma of her left pyriform sinus which was treated by lateral pharyngectomy. She remained well until 1966 when she developed a carcinoma of her left arytenoid which was treated with a full course of irradiation. She began to develop soreness and discomfort in her throat and in 1970 a biopsy of her posterior pharyngeal wall and aryepiglottic fold showed carcinoma. She underwent a pharyngolaryngectomy and the specimen was perfused.

Figure 4A is a horizontal section through the larynx and hypopharynx at the level of the vocal cords, and the arrow indicates severe dysplastic epithelium in the pyriform fossa, which is his-

tologically carcinoma in situ. There were also marked dysplastic changes in the laryngeal epithelium. However, no definite invasive carcinoma could be demonstrated in any of the sections. Figure 4B is an autoradiograph of the dysplastic epithelium of the pyriform fossa, and it should be contrasted with the autoradiographic findings in the normal dog larynx. It shows intense nuclear activity which is randomly scattered throughout the whole epithelial surface, and contrasts with the basal activity in the normal epithelium in the dog's larynx.

Case 2. In 1969, a 71-year-old male was diagnosed with a $T_3N_1M_0$ supraglottic tumor, which was treated by a total laryngectomy and neck dissection. The laryngectomy specimen was perfused.

Figure 5A is a coronal section of the posterior third of the vocal cord showing a large right-sided transglottic tumor. Figure 5B shows the geographic distribution of the tumor (solid black) and a labelling index for the one side of the larynx which was perfused. Figures 5C and D are high power views using light and dark field microscopy and show fairly heavily labelling over the nuclei

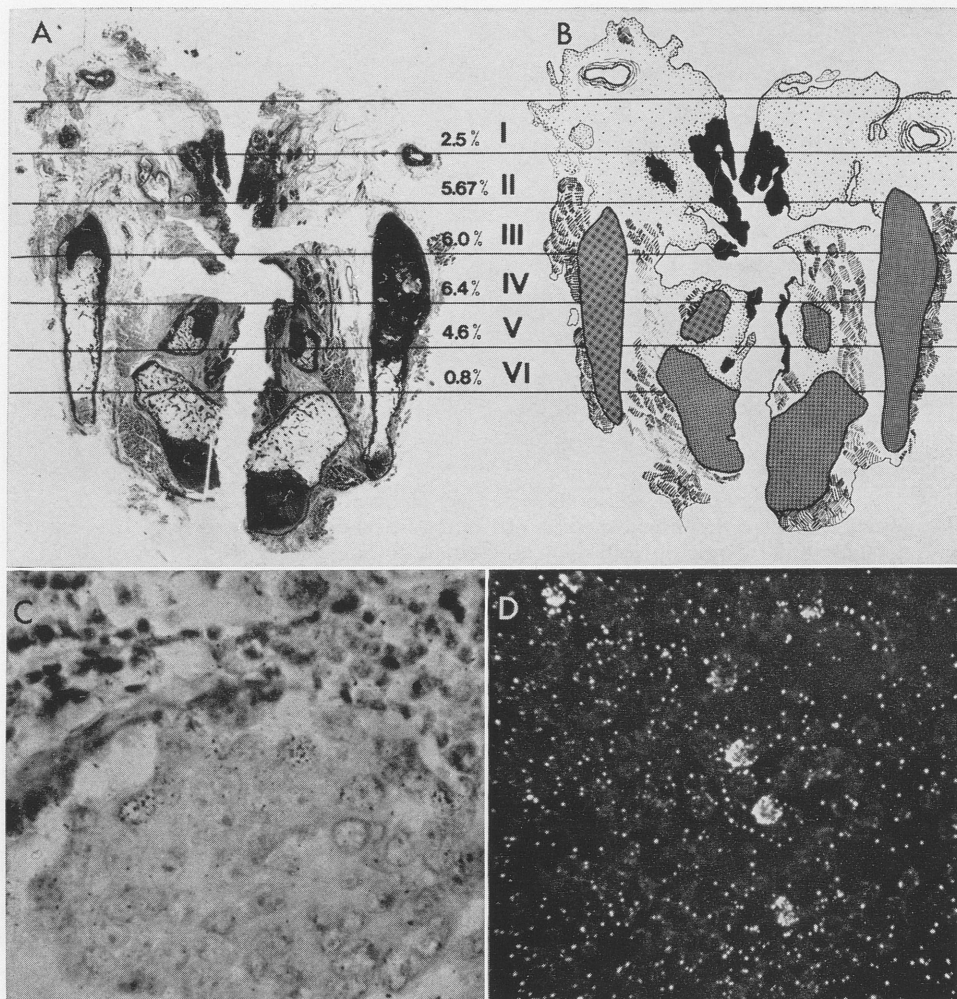


Fig. 5A. Coronal section through posterior one-third of larynx. B. Labelling index of tumor marked in black. C. High power view of nest of labelled tumor cells. X400. D. Dark field view of labelled tumor cells. X400.

of a nest of poorly differentiated squamous cell carcinoma.

This case is an example of a tumor which has not been irradiated and shows a fairly high labelling index. It is not possible to relate any uptake of labelling to the tumor blood supply.

Case 3. In 1970, a 64-year-old male was diagnosed as having a $T_3N_0M_0$ glottic carcinoma for which he received a 5,000 r course of preoperative irradiation followed by total laryngectomy at six

weeks. The removed specimen was perfused.

Figure 6A is a low power view, showing the thyroarytenoid muscle replaced by fibrous tissue in which are scattered nests of tumor cells. These are beneath an intact epithelium (Fig. 6B). Figure 6C shows a nest of tumor cells which have failed to take up tritiated thymidine and nearby is an endothelial cell, in a vascular space showing good labelling (Fig. 6D).

It is interesting to speculate that this

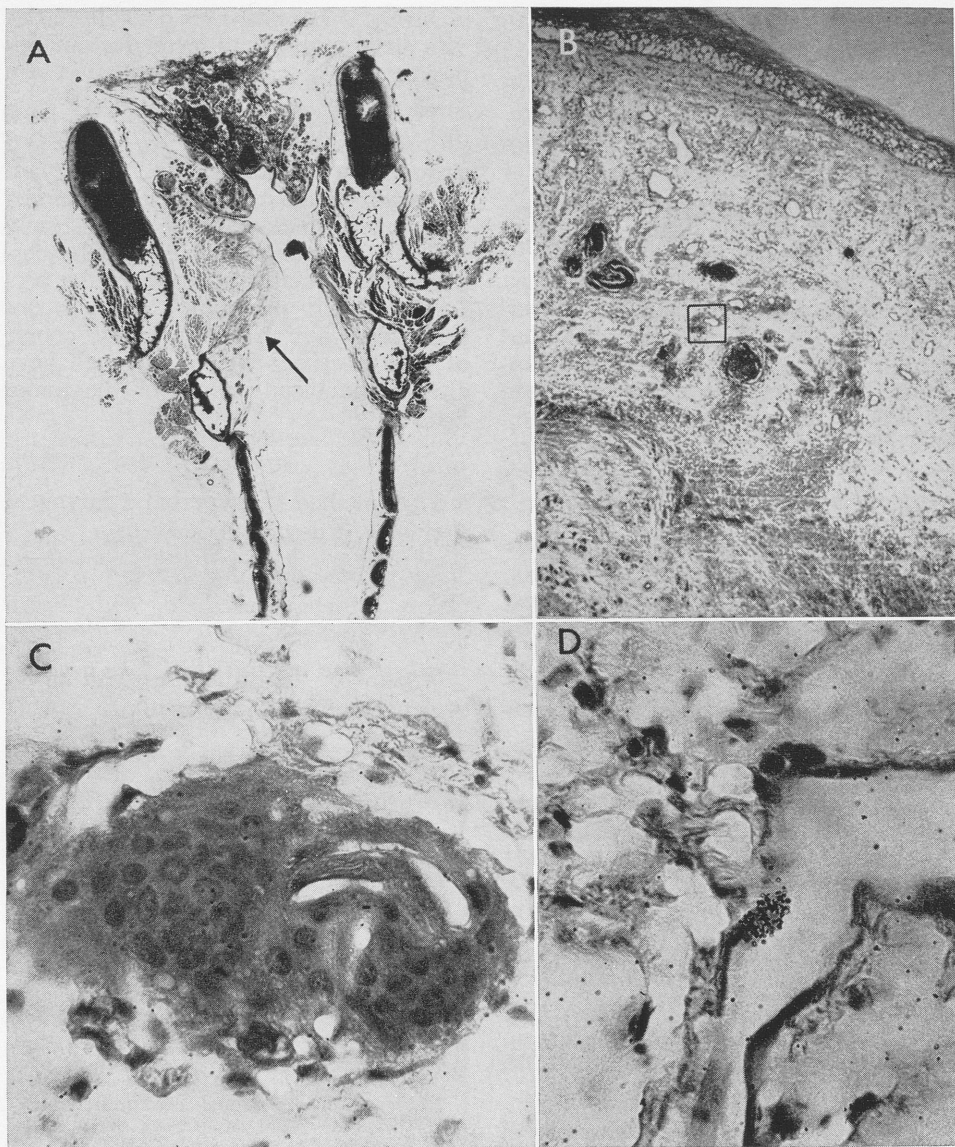


Fig. 6A. Coronal section mid coronal level. B. Low power view showing intact epithelium with nests of tumor cells in scarred thyroarytenoid muscle and vascular spaces. C. Nest of unlabelled tumor cells (inside square in Fig. A). D. Labelled endothelium cell in wall of vascular space (inside square in Fig. B).

tumor was not viable at the time of perfusion and, indeed, recent follow-up has not shown any recurrence of growth. It also demonstrates that it is possible to perfuse the larynx, and that the uptake of thymidine in the basal epithelial layer is not due to incubation with the tritiated thymidine.

DISCUSSION

Autoradiography is a well established research tool to demonstrate nuclear viability. Many experiments have been performed *in vitro*,^{2-4,8,12,17} some *in vivo*^{1,8-11,14} on patients dying with inoperable tumors or blood dyscrasias, but

only one in conjunction with perfusion of the whole organ.¹⁵

This investigation has shown that it is possible to demonstrate cellular activity, by perfusion with tritiated thymidine, in the various tissue components of the human and dog larynx. The development of the paraffin serial sectioning technique,⁷ enabling sections of 5 to 10 microns thick to be obtained, has been largely responsible for this achievement. However, laryngeal perfusion has considerable technical problems. The diameter of the vessels supplying the organ is small and cannulation has often been difficult, adding to the delay before perfusion was established. This time delay averaged about 15 to 20 minutes.

There have been no physiologic studies of the normal human laryngeal blood flow. In the dog, however, normal flow is 6.7 ml/min as measured by directly collecting the venous outflow;¹⁹ so presumably the human blood flow is comparably as small. Because of the low flow

involved, it has not been possible to devise an accurate flow meter for our apparatus.

The laryngeal blood supply is from four vessels which supply the larynx and surrounding structures of the hypopharynx. Unlike the kidney, the larynx does not have a discrete blood supply and it is, therefore, difficult to be certain of the exact distribution of the perfusate during perfusion. Most of our cases have also had a previous course of radiotherapy which might well have altered the blood supply of the tumor bed.

SUMMARY

1. A method of hyperbaric laryngeal perfusion is described.
2. Autoradiographs showing cell viability in the perfused dog and human larynx are demonstrated.
3. Some of the difficulties with laryngeal perfusion are discussed.

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DISCUSSION

DR. MARK N. SABERMAN: I would like simply to say that if a thymidine study such as this is conceivably being used to indicate tumor viability after treatment by radiation, we should look at both sides of the coin, not only from the otolaryngologic point of view in tumor control but also as a radiobiological study.

The radiologists have shown (most notably Gilbert Fletcher of Houston) that despite cells being viable now as indicated by tritiated thymidine tagging, this is not an indication that several generations later these cells are in fact viable.

I simply bring this to your attention, that the fact they are now labeled does not mean that the therapy was of necessity unsuccessful.

DR. JOSEPH GOLDMAN: I would not like this occasion to pass without comment because I think this is a very significant and almost brilliant piece of work in studying the biology of cells which have been treated by radiation.

The work we did originally involved making biopsies of tumor and studying them in tissue culture before radiation, during radiation and after radiation.

I can assure you that this method of perfusion is a very involved and difficult one and the authors should be congratulated on their efforts.

This kind of work is a step forward, and a great step forward, in the study of tissues after radiation. It is only by these kinds of studies that we can arrive at the final truth of what radiation accomplishes before surgery.

I do not wish to preempt the authors' discussion of the last speaker but I believe what Dr. Briant is trying to show is that these cells are viable at that particular time. It does not in any way try to predicate what tritiated thymidine will do to cells three and four generations later. I think this concept must be appreciated.

I rise to congratulate the authors on what I think is a very important step forward in the study of the subject.

DR. T. D. R. BRIANT (closing): I should like to answer the first discussor's comment.

We often have arguments with our radiotherapist as to when has he failed and, as a surgeon, I used to believe that he failed because there was still tumor present after six weeks. Then he showed me that at least two months should elapse before he has failed. He has even convinced me that there may be tumor still present three months following his irradiation. That is really the outside limit that I accept at the present time.

We are really examining this tissue in a very thin "slice of time" and I think his comment is very well taken. However, I would also like to say that this larynx was removed at least six to eight weeks following radiotherapy, and it is my strong feeling that in the majority of cases if the tumor has not gone at this point, then it is likely that this is residual tumor and will have to be dealt with surgically.

I think there are a number of factors that one takes into consideration when one thinks about tumor recurrence or tumor persistence.

Thank you very much for your very kind remarks, Dr. Goldman.

PHARYNGEAL DIVERTICULUM

RONALD MACBETH, F.R.C.S.

OXFORD, ENGLAND

I should like to draw attention to a few points appertaining to the subject of pharyngeal diverticulum in general and its treatment in particular.

SUMMARY OF RESULTS 1950-69

(34 patients)

Sex distribution	Male 18	Female 16
Operated		25
Not operated		7
Primary healing		24
Secondary healing		1
Deaths		2

This is a condition which affects the asthenic, the "lean and hungry" worriers, the people who are irregular with their meals and who often eat in a hurry. In my experience both sexes are affected equally. The symptomatology is so well known that clinical students are often able to correctly make a diagnosis. I would emphasize here, however, that the characteristic history of the double swallow is too little stressed in the textbooks and by teachers.

The general surgical textbooks are indeed guilty of perpetuating three myths about the etiology of pharyngeal pouch. The first is that there is a triangular weak spot at the back of the pharynx called Killian's dehiscence. In fact, nobody has ever found Killian's dehiscence in the dissection of normal specimens. The second is that there is a neuromuscular imbalance as between the thyropharyngeus and the cricopharyngeus in the swallowing act. Wilson has shown that the only imbalance lies in the actual weakness of contraction of the thyropharyngeus.¹ The third is that there is

a spasm of the cricopharyngeus. This story of spasm can be disproved by passing an esophageal speculum on any established case.

We are, therefore, left with Wilson's concept of a weak thyropharyngeus which leads to some degree of megapharynx which contracts poorly and fails to send on the food. The cricopharyngeus opens normally but receives little or nothing, the thyropharyngeus contracts a second time, but by then the cricopharyngeus is also contracting, and so a flabby protrusion forms, in effect producing Killian's dehiscence.

The mythical etiology has led to improper forms of treatment. Dilatation of the cricopharyngeus has sometimes led to temporary improvement, because of actual damage to the circular muscle, but the patient always relapses, and the scarring induced may make ultimate operation more difficult.

Similarly the diathermic destruction of part of the cricopharyngeus (Dohleman's procedure) cannot be regarded as sound surgery. It may have a place in very large pouches in very old and weak patients, but it has always seemed to me to be a piece of unscientific, unjustifiable interference. In the first place, the method is not devoid of risk; cases of mediastinitis following it are not publicized, but they occur. Secondly, an unsuccessful diathermy is followed by scarring which makes secondary surgery more difficult. Thirdly, to do a radical cure takes little more time than a Dohleman procedure, and if a patient is fit enough for the one, he is fit enough for the other.

One final point about the mythical

Presented as introduction to film on the one-stage operative treatment of pharyngeal diverticulum.

cricopharyngeal spasm. Some surgeons advise that the muscle should be divided submucosally at the conclusion of the operative cure, as is done in some cases of bulbar poliomyelitis. This is totally unnecessary and may even do harm, and I have never done it.

The technique of operative cure, therefore, consists of removal of the pouch and reconstituting the pharynx by gathering together the muscular bands. There is usually no need to drain the wound. The patient is tube-fed for six days.

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DISCUSSION

DR. CHARLES S. GIFFIN: This is particularly interesting to me as I just reviewed cases in our group in Fort Wayne over the past six years. We have encountered 31 cases of Zenker's diverticulum, and we found it necessary by virtue of some complication to group these into three types. That is not new.

I have a comment and two questions for Mr. MacBeth.

Some of our complications occurred in absolutely massive diverticula. For some reason we have had eleven of these great, "brute-size" Zenker's diverticula and in attempting to excise these in one stage we found our complication rate was about 15 per cent. By complications, I mean delayed wound healing, early fistula and two cases of mediastinitis. So we therefore turned to Dr. Lahey's technique which he so adequately described in 1926 in which he did the classical two-stage operation. We have been doing the two-stage operation in these great, "brute-size" Zenker's and there have been no complications.

I have two questions for Mr. MacBeth.

One, do you treat all Zenker-type diverticula in this manner?

Secondly, in these massive Zenker's (our "type 3") do you have complications, and if so, at what rate?

DR. DOUGLAS P. BRYCE: It's certainly a pleasure to hear English spoken this far south and west! I don't know whether Mr. MacBeth considers it English or not, but it's certainly different, isn't it?

Some years ago, I gave up using this type of technique because I had some recurrences and because nobody else in the area in which I was working was using the Dohlman technique. Since that time, I have been very happy to use the Dohlman procedure in about 20 cases with no serious problems, and we are quite happy with it.

I wanted also to comment about one case we saw recently because we do occasionally see recurrences of pharyngeal diverticulum after they have been excised. In this recent case, the excision failed to cure the symptoms so that the patient was unable to swallow. This was followed by cricopharyngeal myotomy. We did this about two months ago. The patient is very happy, swallowing

satisfactorily, and I merely suggest whether it is physiologically sound or not. A cricopharyngeal myotomy can be used and solves the problem of dysphagia at least in the cases we have used it in.

DR. RONALD G. MACBETH (closing): Mr. Chairman, I realize of course that I stuck my neck out in being so positive about a technique that I have found rather satisfactory.

I am not certain that I wrote down word for word what the various questions were, but perhaps I might answer them by saying my indications for operation on a Zenker's diverticulum are really expressible in one sentence: If the patient's symptoms are insupportable, and this means whatever size however small or however large, and he cannot get by without having something done, then I do, have done, and teach that one should do, a radical cure. As far as recurrences are concerned, one of course does see them. I would be foolish to pretend that we have not had any. In this last 20 years' series that I quoted, I do not think we had any.

Now, I think one of the reasons for not getting a recurrence is this business of sewing the sterno-mastoid with some stout linen thread to the prevertebral fascia. It splints the area and protects it when you once begin to let your patient swallow again. And, so we get back to the question of the Dohlman procedure.

Before that, perhaps the two-stage operation is something that I have never felt necessary to do. It is perfectly true that if you do a two-stage, you get the mediastinum (and so forth) shut off. Quite honestly, I have never thought it was worth acquiring experience with the two-stage procedure because the other worked so well.

The Dohlman procedure still leaves the patient with a pouch. Whatever you do, however successful, you are in avoiding mediastinitis after the diathermy, the patient still has a pouch. As everybody knows, the rate of malignancy in pharyngeal diverticulum may be as high as five per cent. Personally, in a long and fairly active life, I have seen only one; but one swallow does not make a summer or a pouch!

ELECTROMYOGRAPHIC AND VISUAL EVALUATION OF RECURRENT LARYNGEAL NERVE ANASTOMOSIS IN DOGS

HERBERT H. DEDO, M.D.

SAN FRANCISCO, CALIF.

In 1926, Blalock and Crowe,¹ using dogs, inserted the end of the severed recurrent laryngeal nerve into a slit in the opposite recurrent nerve. There was no motion in either vocal cord after surgery, but seven weeks later both cords were reported to be moving almost normally. In 1928, Lahey⁸ reported a successful direct anastomosis of the recurrent laryngeal nerve in a case of bilateral abductor paralysis following thyroidectomy.

Since that time there have been conflicting reports regarding the return of vocal cord function following repair of a disrupted recurrent nerve. Siribodhi et al.¹¹ transected the recurrent nerve in two dogs; one died but the other animal developed small abnormal motions of the vocal cord and electromyographic (EMG) patterns. They felt that this was suggestive of misdirection of the regenerated nerve fibers. Doyle et al.⁵ reported "nearly 100% success" in the return of vocal cord function after repair of the recurrent laryngeal nerve. They reported that all four dogs with a simple section and repair had motion of the vocal cord in 17 to 22 weeks. In four humans there was partial recovery after anastomosis of a divided recurrent nerve.

Boles and Fritzell² cut and then repaired recurrent laryngeal nerves in a series of dogs. Within six to nine months, EMG patterns were largely normal. Histologically, axonal regeneration through the anastomoses had taken place. In spite of this, there was poor functional

recovery. They believed that this might have been due to random reinnervation.

Gordon and McCabe⁶ studied section and repair of the recurrent laryngeal nerves in seven dogs. After 300 days, symmetrical or only slightly diminished adductor function had returned in all seven dogs, with or without rotation of the nerve stump. The axonal count, distal to the neurorrhaphy, varied from 47 to 95% of normal. Harvey et al.⁷ felt that U-9189, a compound which may assist protein and RNA synthesis in cells of the central nervous system, had accelerated the return of recurrent nerve function after anastomosis. The five untreated dogs showed some recovery of abnormal vocal cord movement in 103 days as compared with 71 days for the five treated dogs. Murakami and Kirchner¹⁰ reported successful vocal cord abduction, during inspiration following recurrent nerve anastomosis, occurred only when the nerves to the adductors were divided.

In a previous group of experiments in our laboratory,^{3,4} the effect of recurrent nerve crush, 1 to 2 cm below the cricoid cartilage, was studied. We found that normal vocal cord EMG patterns and motion returned after 30 to 60 days.

Because of the controversy regarding effectiveness of recurrent nerve anastomosis, an experimental evaluation was made, using dogs. The aims of the study were to see if the recurrent nerve will regenerate — anatomically, electroneurophysiologically and functionally.

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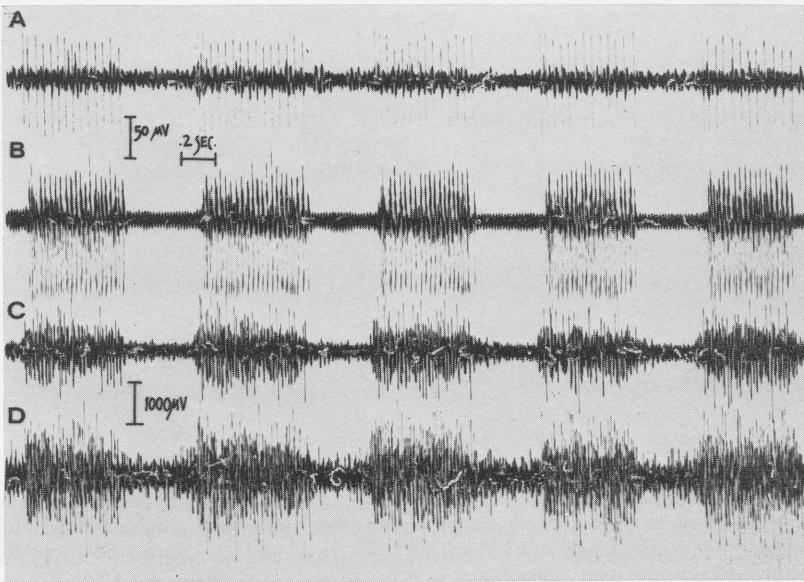


Fig. 1A. Small number of large single motor unit polyphasic potentials during phonations. Some nerve fibers have regenerated. A and B. Right and left thyroarytenoid. C and D. Right and left cricothyroid. B, C and D. Interference potentials (contraction) during each phonation.

METHOD

The dog was used because the gross and neural anatomy of the larynx is essentially identical to the human.⁹ In six dogs the larynx was exposed by a midline ventral incision. The right recurrent laryngeal nerve was divided with a scalpel, 2 cm below the inferior edge of the cricoid cartilage. Using the operating microscope the two cut nerve ends were joined by four monofilament 10-0 to 15-0 nylon sutures. They were carefully placed through the perineurium and equally spaced around the periphery of the nerve. Crush injury to the nerve fibers themselves was avoided by grasping only the perineurium with very delicate forceps during the time of suture. In two dogs the nerve branch to the posterior cricoarytenoid muscle was identified and ligated with a silk suture via the Woodman approach to the posterior cricoid area.

Under light anesthesia, bipolar concentric needle electrodes were used to record the intrinsic laryngeal neuromuscular function in the thyroarytenoid, interarytenoid, and posterior cricoarytenoid areas. This was done repeatedly for 12 to 18 months after the nerve was severed and repaired. The recording technique has been described in previous

papers.^{3,4} Laryngeal EMG recording techniques were supplemented by visual examination as well as by still photography and motion pictures.

RESULTS

Immediately after recurrent nerve section and repair, no electrical potentials were recorded from the paralyzed right vocal cord. Within a week, fibrillation potentials were present during inspiration and expiration. On some traces their frequency increased during phonation. Possibly this was due to cricothyroid contraction which stretched the irritable paralyzed fibers of the thyroarytenoid during phonation. These potentials had less than 100 μ V amplitude. By 35 to 105 days, 100 to 300 μ V polyphasic potentials had been recorded from the right thyroarytenoid during phonation. When they were replaced by 125 to 150 μ V interference potentials these still had lower frequency and lower average amplitude than normal (Fig. 1). With occasional exceptions in three dogs, they occurred during inspiration (Fig. 2). This return of phasic action po-

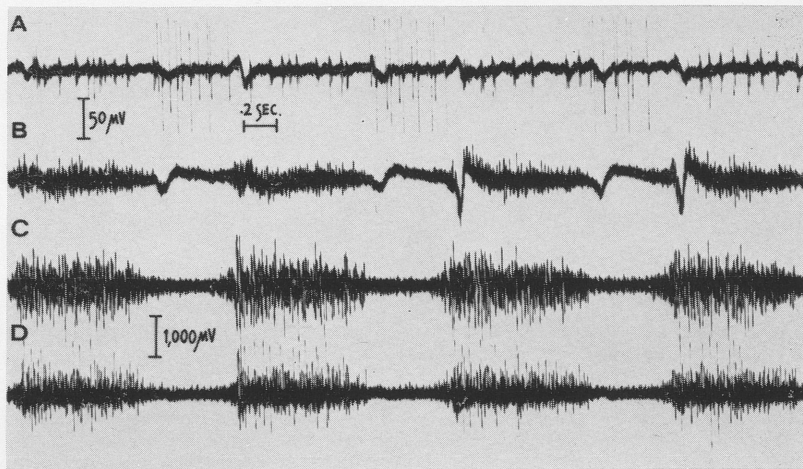


Fig. 2A. Small number of single motor unit potentials during each inspiration from misdirected posterior cricoarytenoid nerve fibers. A and B. Right and left thyroarytenoid. C and D. Right and left cricothyroid. B, C and D. Interference potentials (contraction) during each phonation.

tentials correlated satisfactorily with the return of motor unit potentials in 30 to 60 days after crush injury of the recurrent nerve in dogs.³ Their return established that recurrent nerve fibers had successfully regenerated to the thyroarytenoid muscle.

The 150 to 300 μ V interference potentials, recorded from the interarytenoid muscle, primarily during expiration, were not visibly affected by recurrent nerve section. This was true both before and after phasic potentials had returned to the thyroarytenoid. This confirms the bilateral recurrent nerve supply to the interarytenoid.

We are still not satisfied with EMG of the posterior cricoarytenoid muscle. EMG recordings from the posterior cricoarytenoid area, on the intact left side, showed 150 to 300 μ V interference potentials primarily during phonation when the anesthesia level was sufficiently light. In two dogs the appearance of fibrillation, polyphasic and reduced interference potentials occurred in the right posterior cricoarytenoid area approximately at the same time as in the right thyroarytenoid area. Most of the right posterior cricoarytenoid polyphasic and interference potentials occurred during

phonation. However, this does not confirm the misdirection hypothesis since most of the posterior cricoarytenoid area potentials on the intact side also occurred primarily during phonation.

Return of normal effective motion in the right vocal cord did not occur even after the interference potential patterns had reappeared. This was true whether the nerve branch to the posterior cricoarytenoid was ligated or not. Initially, as was found after crush injuries to the vocal cord,³ the vocal cord lengthened and partially adducted during expiration. This partial adduction occurred symmetrically in both vocal cords at the deeper levels of anesthesia, when bilateral cricothyroid but not thyroarytenoid contractions, as demonstrated by EMG were occurring during expiration. When the anesthesia level had lightened to the point where thyroarytenoid potentials were present during phonation in the normal left vocal cord, it would adduct to the midline while the right vocal cord continued to only partially adduct. During inspiration, especially forced inspiration, only the normal left vocal cord abducted from the resting position. This partial motion plus the passive mobility test showed that there was no fixation of the cricoarytenoid joint.

This confirms that the lengthening and apparent 30 to 40% adductive motion was the effect of cricothyroid contraction alone. It was not an indication of effective return of function of the right recurrent nerve, since it was present before and after the EMG records showed that the phasic action potentials had returned to the right thyroarytenoid. Since the cricothyroid is a significant adductor as well as tensor of the vocal cord of the dog, visual observation, without EMG and motion picture records, is an unsatisfactory technique for evaluation of the effectiveness of recurrent nerve anastomosis.

DISCUSSION

It is noteworthy that, in the dog, the cricothyroid muscle has enough adductive action so that the vocal cord has approximately 40 to 50% of the normal amount of adduction during phonation, even when there is EMG and clinical proof that the ipsilateral recurrent nerve is still paralyzed. This cricothyroid effect appears to be the basis for previous reports of successful partial return of adductive motion of a dog's paralyzed vocal cord after surgical repair of a divided recurrent nerve. The cricothyroid muscle was left intact in previous studies except for that of Boles and Fritzell,² and Harvey et al.⁷ Even in these studies, the cricothyroid may have been a factor since the superior laryngeal nerve can regenerate rapidly over the short distance involved and, in the meantime, the opposite intact cricothyroid can affect both vocal cords.

At the present time it is not clear why repair of the recurrent nerve does not achieve return of normal vocal cord motion when it does achieve nerve fiber regeneration, as proven by return of motor unit potentials in the thyroarytenoid. We used the same sutures and careful technique described by others who have claimed success with recurrent nerve anastomosis. Furthermore, the same technique has been successful in our hands in restoring function in the repair of the facial nerve, even when it was anastomosed to the hypoglossal nerve.

Although it may be the explanation, our data does not provide convincing support for the concept, as traditionally used, that misdirected abductor and adductor fibers have reached the wrong muscles. The two dogs with ligatures on the posterior cricoarytenoid nerve did not adduct any better than the others. Only occasionally were phasic right thyroarytenoid potentials recorded during inspiration. Posterior cricoarytenoid muscle recordings did not occur consistently enough during inspiration on the normal side to permit conclusions to be drawn from the repaired side. Instead, failure to achieve effective intrinsic laryngeal muscle contraction may be due as much to an inadequate number of effective motor units and mismatch of innervation ratio, fusion frequency, tetanus-twitch ratio, etc., as abnormal timing of impulses. Murakami and Kirchner¹⁰ found effective abduction during inspiration when regenerated adduction branches were ligated. However, we did not detect increase in adduction when the posterior cricoarytenoid branch was ligated.

CONCLUSIONS

Laryngeal EMG showed that some recurrent nerves successfully regenerated. In no dog has there been return of full normal adduction during phonation. Only the partial adduction from the cricothyroid effect has been noted. There has been no sign of abduction during expiration, despite the fact that these dogs have been examined repeatedly for 12 to 18 months following division and anastomosis. It would seem that anastomosis of the recurrent nerve after injury should not be performed until it is successful in experimental animals.

SUMMARY

The effectiveness of anastomosis of a divided recurrent nerve was studied in dogs with bipolar concentric needle EMG as well as moving and still pictures. Motor unit potentials returned during phonation to the thyroarytenoid and posterior cricoarytenoid after 35 to 105 days. This proves that nerve fibers regenerated past the anastomosis site and into the intrinsic laryngeal muscles. How-

ever, effective motion did not return to the affected vocal cord; it remained paralyzed in the paramedian position. Its

appearance of 40 to 50% of normal adduction was shown to be due to cricothyroid contraction effect.

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DISCUSSION

DR. DEGRAAF WOODMAN: May I congratulate Dr. Dedo on a beautiful piece of work.

The effort to succeed is certainly worthy and I am trying to compare the laboratory study with the clinical practical events that occur. That does not mean we should discourage or stop this type of study at all.

If you have a damaged nerve in the course of original surgery and if you *know* that it resulted from a light touch or a mild crush, you might wait and see if regeneration will take place; but, looking at it practically, if we delay several weeks to a few months hoping that it comes back (and we generally do delay), what are we then stepping into?

The surgeon is stepping into a scarred field. Dr. Dedo describes the difficulties even under laboratory conditions wherein the nerve ends are labeled. How many surgeons are trained and skilled in this particular field to be able to attempt anastomosis at the time of original damage?

I think Dr. Dedo has brought out the fact that practically, as yet, we have not solved anastomosis of the nerve at the time of injury. That may come in the future.

DR. JOSEPH ATKINS: I have enjoyed this paper very much because we have been inter-

ested in pharyngeal electromyography for a considerable period of time. We are examining most of our patients who have pharyngeal paralysis just to see what is going on.

Dr. Francis Bonner has been doing our pharyngeal electromyography. It is most interesting to see in these patients who have had injury how they will go from electrical silence to fibrillations and then polyphasic waves to sometimes complete recovery. Sometimes you see a patient whom you think has simply a posticus paralysis and you find there is a great deal more nerve damage in that larynx than you anticipated.

I think one of the things we could be doing is to take this from the laboratory and begin to study laryngeal pathology with the electromyograph far more frequently in the clinic than we have done in the past.

DR. JOHN A. KIRCHNER: I really enjoyed Dr. Dedo's paper and I think it is worth pointing out that one of the early significant contributions that Dr. Dedo has made to our study of reanastomosis of nerves and reinnervation of laryngeal muscles is that visual observation of the motion of the vocal cord after repair of a nerve is really an inadequate method of assessing success. You do get stretching of the cord and you get movement

of the arytenoid from the other normal recurrent nerve. Unless you do careful myography (the kind of careful myography that Dr. Dedo has done and has pioneered in) showing there are so many artifacts from surrounding muscle, one really must be very cautious about drawing conclusions.

I congratulate Dr. Dedo on this further evidence of his care in research.

DR. HERBERT H. DEDO (closing): Thank you for your comments.

I agree with Dr. Woodman that this research work corroborates what a lot of you have suspected clinically. I feel that anastomosis of the recurrent nerve after injury shouldn't be done in humans until it has been successful in experimental animals.

The fact that this technique has worked with other nerves but has not worked with the larynx is intriguing and I don't think we have the answer yet.

THE MANAGEMENT OF LONG TERM AIRWAY PROBLEMS IN INFANTS AND CHILDREN

BLAIR FEARON, M.D.

DAVID ELLIS, M.D.

TORONTO, CANADA

In the brief time allotted, this subject material cannot be presented in detail but an attempt will be made to select the more important conditions and features of long term airway problems in infants and children. No world shattering developments will be described, but because of many requests for information regarding the management of these problems in children, especially that of subglottic stenosis, we felt it worthwhile to record our experience in treating such patients at the Hospital for Sick Children over the past 20 years.

In defining the terminology "Long Term Airway Obstruction," we refer to any obstruction, congenital or acquired, of the major air passages that are sufficiently severe to require tracheotomy or repeated endoscopic procedures for more than an arbitrary period of six weeks. This includes most of the patients that have prolonged airway obstruction anywhere from the region of the mouth to the bifurcation of the trachea, but excluding patients with respiratory insufficiency due to central nervous system disease, congenital heart disease, pulmonary disease, etc. Only too frequently hospital personnel have a tendency to label children with tracheotomies for the relief of long term airway obstruction "chronic tracheotomies," ignoring the fact that the tracheotomy is necessary for the relief of the obstruction and not the cause of it.

A very common misunderstanding is that if a tracheotomy is performed on a

small patient, this patient automatically will need a tracheotomy for a long period of time. This is not so. We have shown previously and wish to emphasize that if the tracheal incision is made, rather than the classical incision through the second and third tracheal rings, well below the cricoid cartilage, extubation may be carried out as soon as the primary obstruction is relieved.⁹

It is most important to remember that the younger the patient the further away from the cricoid the tracheal incision must be made, even as low as the sixth or seventh tracheal rings. To many laryngologists this may seem to be dangerously low in the trachea; however, the trachea has 18 to 20 tracheal rings, and, therefore, there is sufficient length in the trachea in which to accommodate the appropriately sized tracheotomy tube. The size of the tracheotomy tube to be used may be judged on the basis of the size of the glottic aperture, and since the tracheal lumen on cross section is approximately twice the area of the glottic aperture, there is adequate space around the tube where it lies in the trachea. Table I illustrates the size of tracheotomy tubes and bronchoscopes used for younger age groups at the Hospital for Sick Children. It is important to remember that the tracheal length of the patient should be judged on age rather than on weight and size.¹⁰

CRANIOFACIAL ABNORMALITIES

Prolonged severe airway obstruction may occur from several types of cranio-

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TABLE I
SELECTION OF TRACHEOTOMY TUBES AND BRONCHOSCOPES

Age	Holinger Tracheotomy Tube	Bronchoscope Size	Age
Premature (< 4 lbs.)	No. 000 x 26 mm		
Premature (> 4 lbs.)	No. 00 x 26-33 mm	3 mm	< 5 lbs.
0-6 mos.	No. 0 x 33-40 mm	3.5 mm	0-6 mos.
6-12 mos.	No. 1 x 40-46 mm		6 mos.
12-18 mos.	No. 1 x 46 mm	4 mm	↓ 3 yrs.
18 mos.	No. 1 or 2 x 46-50 mm		
↓ 4-5 yrs.			3 yrs.
4-5 yrs.	No. 2 or 3 x 50-55 mm	5 mm	↓ 12 yrs.
↓ 10 yrs.			
10 yrs. +	No. 3-5 x 50-68 mm	6 mm	12 yrs. +

facial dysostoses such as the PierreRobin syndrome, Crouzon's syndrome, coronal synostosis, Franconi's syndrome, etc., in which under certain circumstances tracheotomy is advisable until the deformity causing the obstruction can be corrected, or the child's development may lessen the problems. In some instances adenoid and tonsil hypertrophy may precipitate respiratory obstruction. In these cases the course of treatment is obvious — remove the adenoids and/or tonsils.⁵

The PierreRobin syndrome, namely cleft palate, micrognathia and microglossia is the most common of these abnormalities and in the vast majority of infants good nursing care is all that is necessary. However, in our opinion, when the airway is severely compromised, tracheotomy is the treatment of choice. With a tracheotomy these patients are much more comfortable than those in which various methods of holding the tongue forward are used, and the nursing care is simpler. However, these patients may require a tracheotomy for upward to a year or even more before they are able to breathe through the natural airway adequately (Table II).

LARYNGOMALACIA (Tables II and III)

Seldom does laryngomalacia create a serious obstruction to the airway. None-

TABLE II
CONGENITAL ANOMALIES OF THE
LARYNX — 433 Cases, 1960-1970

1. Laryngomalacia	293
2. Subglottic Stenosis	84
3. Vocal Cord Paralysis	28
Bilateral	9
Unilateral	19
4. Vascular Anomalies	14
Hemangioma	13
Lymphangioma	1
5. Laryngeal Webs	9
Supraglottic	0
Glottic	3
Subglottic	6
6. Laryngoesophageal Cleft	2
7. Laryngeal Cysts	3
8. No Cases Found:	
— Glottic Atresia	
— Absences or Deformity of Cartilages	
— Duplication of Cords	
Total	433

TABLE III
LARYNGOMALACIA — 293 Cases

Sex: ¾ Male; ¼ Female
Onset of Symptoms: ¾ at Birth
Diagnosis: ¾ Under 6 Mo.
90% Direct Laryngoscopy
10% Clinical Grounds
S. S.*: 92% — Inspiratory Stridor
28% — Indrawing
23% — Dysphagia or Feeding Problems
20% — Recurrent URI
— Persistent Cough
— Recurrent Pneumonia
— Excess Mucus in Throat
— Cyanotic Spells
Associated Anomalies
28% — Innominate Artery Compressions
5% — Subglottic Stenosis
Treatment:
— Symptomatic
— 2 Operative Cases

*Signs and Symptoms

theless, in the extremely severe cases, tracheotomy may be mandatory for the preservation of life. In one such patient we were able to relieve the obstruction by suturing the epiglottis to the base of the tongue after first denuding the mucosa from the adjacent surfaces of the epiglottis and of the base of the tongue. Decannulization was accomplished a short time after the above procedure was carried out.

CONGENITAL LARYNGEAL WEBS AND STENOSIS (Table IV)

In our experience glottic webs are uncommon, or at least rarely recognized. Congenital subglottic stenosis is much more frequent and may vary from a thin weblike obstruction to a rather dense stenosis. The stenosis may not be apparent for some considerable time after birth, and often respiratory difficulty is precipitated by a lower respiratory infection. When these patients are healthy, they can breathe quite normally even though they may have a stridor which may be mistaken for laryngomalacia.

With the onset of a respiratory infection, the infant is unable to cough up secretions from the tracheobronchial tree and thus, acute respiratory distress occurs. We have often wondered whether some patients who have been tracheotomized for apparent laryngotracheitis have had, in fact, a preexisting subglottic stenosis. This might account for the difficulty in extubation of some of these cases under the erroneous notion that a tracheotomy on a small child must be necessarily a long term problem.³

When the subglottic stenosis is thin and weblike it may be managed by simple dilatation and without the need for a tracheotomy. Even one dilatation may suffice. Naturally, very close attention must be paid to these patients in the

TABLE IV
SUBGLOTTIC STENOSIS — 84 Cases

Sex: ¾ Male; ¼ Female
Onset of Symptoms: 65% Under 1 Mo.
Diagnosis: 70% Under 6 Mo.
98% Laryngoscopy
20% Incidental Findings
S. S.*: 40% — Inspiratory Stridor
40% — Respiratory Distress
25% — Initiated by Attack of URI
20% — Indrawing
— Recurrent Croup or Pneumonia
— Cyanotic Spells
— Dysphagia or Feeding Problems
— Persistent Cough
Associated Anomalies
20% — Innominate Artery Compressions
20% — Laryngomalacia
Treatment:
20% — Symptomatic
80% — Tracheotomy — ¾ for Resp. Distress
— Repeated Dilatations
Duration of Tracheotomy
70% 2 Years or Less
Complications:
2 Deaths

*Signs and Symptoms

immediate postoperative period, especially when a general anesthetic has been used in which atropine was a part of the anesthetic technique.

The majority of patients with severe obstructive subglottic stenosis required tracheotomy for a long period of time. In a ten year period, 25 of 84 patients required a tracheotomy for upward to a year, 34 required tracheotomy from one to two years of age, and the remaining 25 required a tracheotomy for more than two years. All patients except one were decannulated by the age of six years. Undoubtedly, a certain number of individuals have a relative stenosis and do not have a serious degree of respiratory embarrassment. It is our firm belief that whenever a stridor is being investigated one must do a thorough endoscopic examination, not only of the larynx, but of the tracheobronchial tree as well. Too often it has been taken for granted that a stridor is due to laryngomalacia on direct laryngoscopy, but later when an emergency situation has arisen, it was found that laryngomalacia was an incidental finding and a more serious lesion such as subglottic stenosis, vascular anomalies affecting the tracheobronchial tree, etc., was the real offender.

Acquired subglottic stenosis is secondary to trauma, and the more common trauma in the patients that we have treated has been that of prolonged endotracheal intubation. For many years laryngologists have been aware of the potential danger of endotracheal tubes when used for a long period of time. However, in 1965, McDonald and Stocks,¹³ and others^{1,2} advocated the use of prolonged endotracheal intubation for the relief of various airway problems, resulting in an increase in the incidence of subglottic stenosis despite assurances to the contrary.

In 1966 we reported a series of complications that were mainly due to prolonged endotracheal intubation in a 12-month period at the Hospital for Sick Children.⁶ In 29 of 72 patients that survived, one had a glottic web and five had severe subglottic stenosis necessitat-

ing tracheotomy. As a comparison, in 504 patients who were tracheotomized for acute supraglottitis and laryngotracheitis, of the 497 that survived the disease, not one could be considered a long term airway problem.³

It is true that "high" tracheotomies result in long term airway problems unless corrective measures are taken. In five patients with high tracheotomies referred from other Centers, a second tracheotomy was carried out lower in the trachea and four extubated uneventfully.

In the past four years in our area there has been a decided decrease in the cases of subglottic stenosis where repeated intubation or prolonged intubation has been the modality of treatment. This has been due, certainly in part, to the fact that the intubation technique has not been carried on for as long periods of time as before, but there must be other factors in this reduction of complications, and these factors may be a different type of material for the composition of the endotracheal tube, the method of sterilization of these tubes, the size of endotracheal tube being used, as well as the reduction in the length of time the tubes are used in most of the patients. We believe that intubation may be carried out without serious consequences for at least 48 hours and perhaps even longer.

TREATMENT OF SUBGLOTTIC STENOSIS

So far there is no satisfactory operative procedure to correct the subglottic stenosis in the small child and, indeed, the problem is difficult to correct in the adult. The key factor preventing satisfactory treatment by dilatation is the limitation imposed by the cartilaginous ring of the cricoid. We believe that if the lumen of the cricoid cartilage could be enlarged to the size equivalent to that of a 10 or 12-year-old, then the difficulty might be solved. In one patient with a congenital subglottic stenosis, and after several years of conventional treatment, the cricoid was split vertically in the midline and the edges held apart by a piece of rib cartilage. The inner surface was lined with a graft of mucosa

taken from inside the mouth, and a relatively good result was obtained.

Although the results from repeated dilatation of the subglottic stenosis are not striking, nonetheless we believe that gentle dilatation at regular intervals does help improve the airway as well as the development of the larynx and speech. The size of the tracheotomy tube used is the smallest that the patient can tolerate in providing an adequate pulmonary ventilation. In this way the larynx is used at its fullest degree. There is no way of knowing exactly how often dilatations need to be carried out on any one patient but our routine is to do our dilatations every four to six weeks in most patients. Jackson laryngeal dilators are used as well as different sized bronchoscopes. Overdilatation must be avoided to avoid necessary trauma that will only increase the fibrous tissue formation.

In most patients dilatations and bronchoscopies are carried out under general anesthesia. We have found that in these patients especially, diethyl ether is by far the most satisfactory anesthetic agent during the operative procedure and in the postoperative recovery period. When diethyl ether is used, the patients are up and about a few hours after the operation. With other anesthetic agents the same patients will need croupette therapy for days. Naturally this should only be done where there is no danger of igniting the highly flammable oxygen ether combination.

In acquired subglottic stenosis, obviously the age group differs from the congenital type. In the patients that have been under our care or are still being treated, the ages at the time of tracheotomy have ranged from three months to three years. One to three years of tracheotomy care has been necessary in 18 to 23 patients; five still have a tracheotomy.

CONGENITAL LOWER TRACHEAL STENOSIS

This is an uncommon condition that is most difficult to treat, and in five cases we have attended the stenosis has been characteristically two to three rings

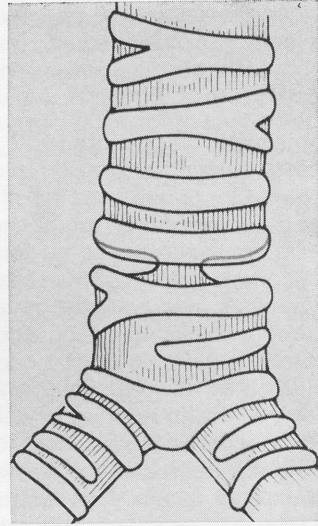


Fig. 1. Congenital lower tracheal stenosis.

above the carina (Fig. 1). These patients present first as respiratory emergencies and if the stenosis is not recognized promptly death may occur abruptly. Because of the proximity of the stenosis to the carina, a surgical resection with end-to-end anastomosis has proved to be technically impossible. Generally speaking, tracheotomy should be avoided in these patients because of the difficulty in placement of the tracheotomy tube. One in five patients was treated successfully by repeated dilatations with the bronchoscope; two patients died suddenly with acute respiratory distress while awaiting decision for definitive surgery; another patient was being managed by repeated dilatations but the problem was complicated by cystic fibrosis and the child died of overwhelming pulmonary infection; another was corrected surgically by Dr. James Simpson, Thoracic Surgeon at the Hospital for Sick Children, by incising through the trachea at the stenosis and through the stenosis itself, and the lumen widened by the use of a tantalum mesh patch lining the skin. This child did well in respect of the stenosis but about two years later died of a fractured skull. The postmortem specimen showed an excellent result from the above pro-

cedure. This operation would seem to be the ideal method of management for tracheal stenosis if dilatations cannot maintain an adequate airway. We would stress that if the surgery is to be done it should be done as soon as the diagnosis has been confirmed.

MISCELLANEOUS LONG TERM AIRWAY PROBLEMS (Tables V and VI)

Congenital bilateral abductor paralysis is rare and the etiology uncertain. It is important to do a tracheotomy when it is evident that the abductor muscles are not going to function, in our opinion, within two weeks of birth.

A high pitched stridorous breathing on occasion complicates certain patients that have hydrocephalus. This would seem to be an abductor spasm although the etiology of this is uncertain. It does

TABLE V
VOCAL CORD PARALYSIS — 28 Cases

Sex: ⅔ Male; ⅓ Female
Onset of Symptoms: 75% at Birth
Diagnosis: 75% Under 6 Mo. 90% Direct Laryngoscopy
S. S.*: 60% — Inspiratory Stridor 25% — Cyanotic Spells 25% — Dysphagia and Feeding Problems 20% — Respiratory Distress — Indrawing — Hoarse, Weak or Absent Cry — Initiated by Attack of URI
Associated Anomalies 20% — Laryngomalacia 20% — CNS
Exam: 70% Unilateral R-20%; L-80% 30% Bilateral
Etiology (50%) 5 — "Neurogenic" 4 — Meningocele or Hydrocephalus 2 — Birth Trauma 3 — Cerebral Agenesis, Spastic Origin, CVS
Treatment: 70% — Symptomatic 30% — Tracheotomy

*Signs and Symptoms

TABLE VI
HEMANGIOMA — 13 Cases

Sex: 7 Males; 6 Females
Onset of Symptoms: All Under 3 Mo.
Diagnosis: 12 Under 3 Mo. 12 Direct Laryngoscopy 1 Clinically
S. S.*: 6 — Inspiratory Stridor 4 — Respiratory Distress — Hoarse Cry — Persistent Cough — Recurrent Croup
Associated Anomalies: (7) 5 — Hemangioma in Other Areas
Exam: 8 — Subglottic 1 — Glottic 3 — Multiple
Path: 7 — Capillary 3 — Negative Biopsy 3 — No Biopsy
Treatment: 3 — Symptomatic 1 — Tracheotomy Alone 8 — Tracheotomy and Radiation 1 — Radiation Alone

*Signs and Symptoms

appear in patients who have had a shunting procedure, and would suggest that the shunt is not functioning. Tracheotomy should be done although a trial of intubation for a few days would be reasonable before tracheotomy is carried out.

In nine of the 15 patients with subglottic hemangioma, tracheotomy was necessary. It is of prime importance to make the diagnosis early and treatment by irradiation carried out as soon as feasible. Ferguson and Flake¹⁰ first pointed out the relatively large number of cases with subglottic hemangioma that were diagnosed only at postmortem. We always attempt to get a biopsy for definitive diagnosis, but Ferguson and others have concluded that biopsy is not necessary.

Vascular compression of the trachea is a more common problem than is generally realized as described some years ago.^{7,12} In this instance we feel tracheot-

omy is contraindicated in those who have severe respiratory distress, and that a nasotracheal intubation be performed first until surgical correction may be instituted.

Primary tracheomalacia has been rare in our experience, but secondary tracheomalacia to mediastinal tumors, double aortic arch, etc., is not unusual. When the double aortic arch has been corrected by cardiovascular surgery there often is a severe lower tracheal and bronchial malacia from pressure necrosis on the tracheal and bronchial cartilages. This presents an especially difficult problem and even tracheotomy may fail to save the patient.⁷

MANAGEMENT OF PATIENTS WITH TRACHEOTOMY

Good tracheotomy nursing care is paramount and the patient should be in a unit for tracheotomy patients only, and one which is staffed by nurses who are experienced in the care of the tracheotomized patient. Since long periods of hospitalization are necessary, our program is to encourage the parents to spend as much time with the child as they possibly can and help with the nursing and tracheotomy care. They are taught the technique of catheter aspiration and when they have gained sufficient confidence they are allowed to change the tracheotomy tube under supervision. As soon as it is deemed safe for the child to be taken out of the hospital, the initial step is for the parents to take the child home for a day or so at a time, and, depending on their success, eventually have the patient in the hospital only for active treatment.¹²

Tracheotomy tubes should be changed regularly to lessen complications such as erosion of the tracheal mucosa, crusted exudate beyond the tracheotomy, stenosis at the tip of the tracheotomy tube, and granulations forming in the lower trachea and bronchial tree. Changing the tube biweekly seems to serve in preventing the above complications. One patient who seemed to have a very unusual tissue reaction to even the slightest trauma developed granulomatous

lesions even in the bronchi. Routine catheter aspiration in this child was avoided and a solution of Decadron® with Xylocaine® dripped into the tracheotomy tube daily, in some inexplicable manner, eliminated the granulation tissue. This is the one patient in our series who had a tracheal stenosis at the site of the tracheotomy, and, as well, steroid was injected into this area at the time of dilatation.

A problem that may be encountered in children with relatively short necks and high shoulders is that this combination tends to cause an upward pull on the tracheotomy tape. This in turn creates an upward pull on the tracheotomy tube thus causing an indentation of the cartilage immediately superior to it. The tracheal airway around the tracheal lumen is then compromised. This problem may be avoided, as Holinger and Johnston described¹¹ by attaching a second tape to the tube, bringing the tape under the arms and tying it at the

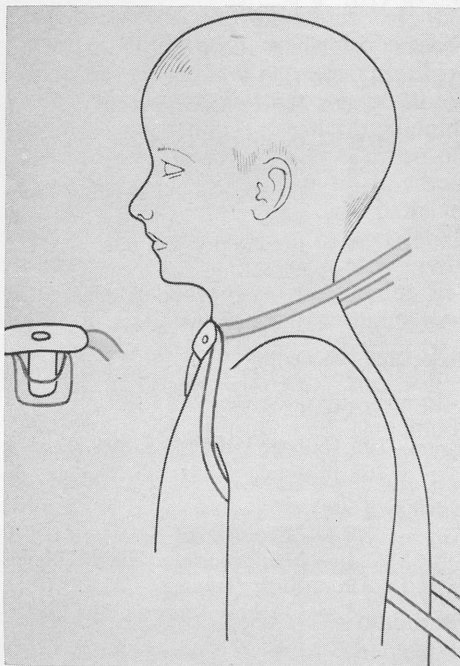


Fig. 2. Effect of the upward pull on tracheotomy tube by a single tape and method of attaching a second tape to tracheotomy tube to relieve problem.

back (Fig. 2). This maneuver counteracts the upward pull of the first tape, relieving the pressure on the tracheal cartilage by the tube. The redundant cartilage, if necessary, may be sutured to the superior margin of the tracheotomy stoma.

EXTUBATION TECHNIQUE IN LONG TERM AIRWAY PROBLEMS

In the long term airway problems of tracheotomy, eventually the time comes when the patients can breathe adequately through their larynx. As mentioned earlier, the size of the tracheotomy tube is gradually reduced to the smallest, namely, a 000 Holinger tracheotomy tube. This tube is blocked periodically for varying periods of time depending on the degree of airway obstruction. It is not unusual for the patient seemingly to be breathing satisfactorily through the larynx but an elevation in temperature develops. This is often the first indication that secretions are filling the tracheobronchial tree. If such is the case then the tracheotomy tube should be unplugged. On the other hand, if the tube can be plugged continuously for several days without any sign of obstruction or retention of bronchial secretions the tracheotomy tube may be removed but replaced with a specially made plastic stent (Fig. 3). Lucite was used originally for the composition of

the stent but we have found that silicone is more satisfactory for this purpose. At the same time the tracheotomy set is kept at the child's bedside. The child may be sent home with the stent in place and with the tracheotomy tube set, assuming that the parents are capable of introducing the tracheotomy tube if an emergency should arise.

If there is any question of adenoid and tonsil disease (and most of the above patients do have in our area) it is judicious to remove the adenoids and tonsils before allowing the tracheotomy to close.

After several years of experience we have found that the stent must be left in for three months at the very least, and longer if there is any reason for doubt that the child may not be able to cope with respiratory infections without a tracheotomy. If this seems to be an unduly cautious routine, I should say that on more than one occasion we and the parents have been thankful that the tracheotomy opening was kept patent with the plastic stent. The diameter of this stent is approximately that of a 000 tracheotomy tube. Incidentally, the stent causes virtually no reaction, and the patient can carry on normally and even go swimming without any hazard.

SUMMARY AND CONCLUSIONS

1. In this presentation the authors have attempted to describe the more common conditions in which there is a long term airway obstruction; the most common cause of long term obstruction is that of subglottic stenosis, either congenital or acquired.

2. The likely causes of acquired long term airway problems as well as incidence of congenital anomalies of the larynx have been listed.

3. Whereas earlier in practice subglottic stenosis had been predominantly congenital in origin, in the past five years acquired stenosis, due to long term intubation, has been much more common.

4. The authors' treatment of subglottic stenosis is explained and the manage-

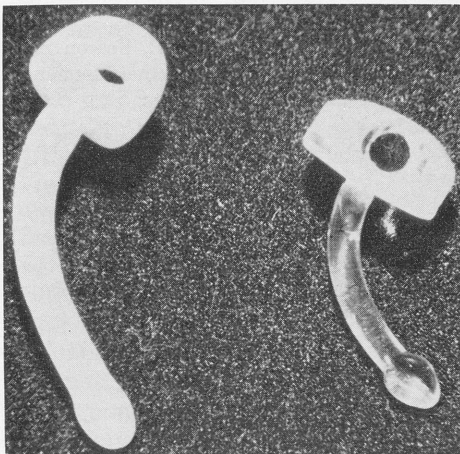


Fig. 3. Plastic stents to maintain tracheal stoma.

ment of a long term tracheotomy problem described.

5. The method of decannulization of the long term airway problems with tracheotomies is entirely different from the patient tracheotomized for short periods of time. An adequate trial with the patient breathing around the blocked tracheotomy tube is carried out for several days or even weeks. Then the tracheoto-

my tube is replaced by a specially made plastic stent to maintain the tracheotomy stoma for an absolute minimal period of three months. Until a satisfactory surgical procedure to correct the problem of subglottic stenosis in children can be devised, it would appear that periodic gentle dilatations of the stenosis is the only practical way of handling the problem.

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LOOKING AT THE LARYNX DURING RUNNING SPEECH

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In the course of research on speech perception, and in later work on articulatory dynamics, we were led to suppose that there is a close relationship between voicing distinctions, a prominent phonetic feature of the world's languages, and the relative timing of laryngeal and supralaryngeal articulatory events.^{4,5} Thus, certain classes of sounds in English may be distinguished by whether or not the arytenoid cartilages move apart during the production of these sounds, and when (relative to other events) they resume a phonatory position. The voiceless fricatives of the words *see*, *she* and *fee* regularly show separation of the arytenoids, while the voiced stops of *bay*, *day* and *gay* do not; but some consonant classes show a degree of variability in this respect, in particular, the voiceless stops of *pea*, *tea* and *key* when unaspirated, as they are before unstressed vowels. The initial data were from perceptual tests and measurements of sound spectrograms; further evidence has come from observations, indirect and direct, of the laryngeal gestures.

Various techniques have been developed during the past ten years or so for observing indirectly the changes in glottal aperture during running speech. These include transillumination,¹⁰ measurement of electrical impedance across the glottis,² ultrasonic monitoring,^{1,7} and x-ray motion pictures.³ The transillumination technique was used in earlier work at Haskins Laboratories,⁶ yielding valuable information on laryngeal maneuvers during speech. The data were ambiguous, however, at some points and raised questions that could best be solved

by direct visual examination of the glottis during ongoing speech. We have used a flexible fiberoptic bundle, employing a technique developed at the University of Tokyo,⁸ to examine the glottis visually and photographically.⁹

Preliminary experiments only have been made in collecting transillumination and pictorial data at the same time but it is clear that transillumination will be a useful supplement to visual observation. The data from an early experiment are shown in Figure 1 for the sentence, "It's a police whistle." Sample frames from the pictorial record obtained with a fiberoptic endoscope are shown for the open glottis (extreme left) and for three selected sounds that are identified by the phonetic transcription at the bottom of the figure. Closure of the glottis is indicated by the trace at its lowest level; partial opening, allowing the passage of some light, is shown by the small upward peaks of the transillumination trace.

The arrangements for direct visual observation employ a fiberoptic endoscope that contains two groups of glass fibers, one to carry light to the glottis and the other to bring out the image. The composite fiber bundle and plastic sheath has an outside diameter of about 5 mm. It is inserted through the nose and positioned in the hypopharynx at the level of the tip of the epiglottis, following surface anesthesia (Xylocaine®) of the nasal cavity and epipharynx. Illumination is supplied to the fiberscope (and then to the glottis) from a 500 watt slide projector that had been modi-

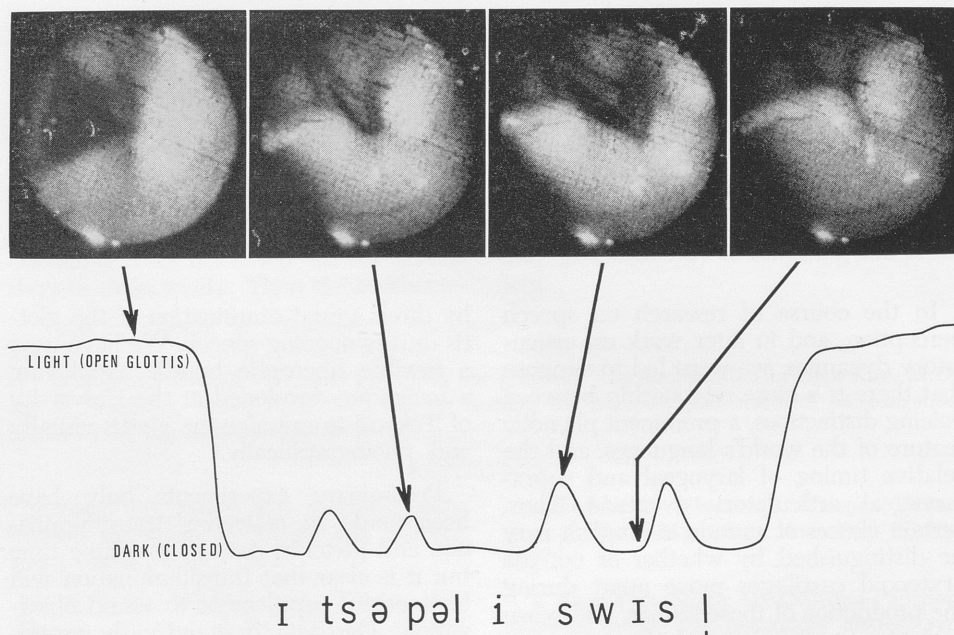


Fig. 1. Four views of the glottis through a fiberoptic endoscope and the transillumination trace for the spoken sentence, "It's a police whistle."

fied by substituting an Abbe substage condenser for the normal projection lens. The lens barrel of the slide projector served also to hold a metal block that positioned the fiber bundle so that it would collect maximum light from the condensing lens. The projection lamp draws its power from a regulated direct current supply, since this is essential for transillumination experiments and has the added advantage that it avoids slow changes in apparent illumination when motion pictures are taken at 60 frames per second. The eye piece of the fiberoptic was replaced by a metal sleeve that attaches directly to the lens mount of a Bolex 16 mm camera, equipped with a reflex view finder. The camera is driven at 24 or 60 frames per second by a synchronous motor. Speech signals are recorded on one track of a stereo tape recorder, and synchronizing signals on the other track. This arrangement allows an accurately synchronized sound track with movies made at 24 frames per second; it also permits exact identification of corresponding points in the film and in spectrograms of the spoken

sentences. The sound films were made on Eastman High Speed Ektachrome; the black and white films were taken at 60 frames per second on Eastman 4X (negative) film.

Three American English talkers were used as subjects, reading short sentences that contained both voiced and voiceless consonants. Constant monitoring of the picture through the reflex view finder was essential, as was the ability to deflect the tip of the endoscope to follow the changing positions of the larynx (relative to the endoscope). Figure 2 shows selected information from one of the sentences that was recorded by these procedures. The sentence was "Rub Billy's head with this towel." Each of seven frames from the motion picture is correlated with the moment of its occurrence in the sound spectrogram that is shown across the lower part of the figure. A narrow-band trace of the lower harmonics is displayed just above the wide-band spectrogram as an alternate way to show voicing during the speech. At the bottom of the figure is a phonetic transcription of the utterance, positioned

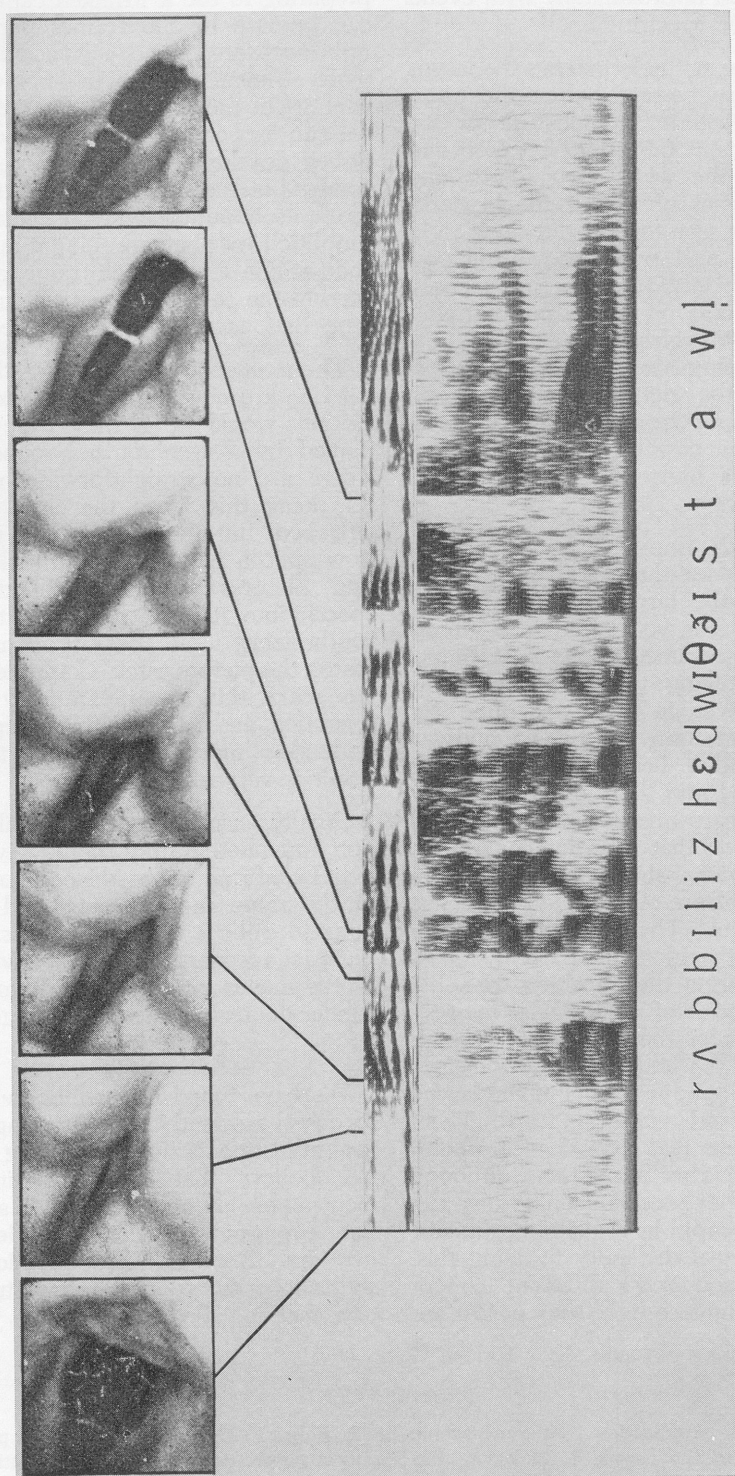


Fig. 2. Views of the glottis and corresponding moments along a sound spectrogram of the spoken sentence, "Rub Billy's head with this towel."

to correspond approximately with events shown in the spectrogram.

The frame at the left shows the larynx in respiratory posture before the utterance. The anterior commissure of the vocal folds is at the upper left corner of the frame; the wide open glottis and vocal processes of the arytenoid cartilages can be seen toward the lower right corner. The next frame shows the posture immediately prior to voice onset. The larynx is in phonatory position with the arytenoids closed, and there is a narrow opening along the membranous portion of the glottis. The posture, in the third frame from the left, is almost the same, but now the edges of the vocal folds are blurred, indicating vibratory motion.

The fourth frame is for the labial closure midway through "Rub Billy's." Here, also, the larynx is in phonatory position and the vocal folds are vibrating. The appearance in the next frame, for the following vowel is almost the same. However, in the sixth frame, for the transition from [z] to [h] midway through "Billy's head," the arytenoids are separated and the glottis is partially open. The sharp image of the vocal fold edges indicates that vibration has ceased. The final frame shows the glottis just before the release of the initial voiceless stop of "towel." The arytenoids are separated and the glottis is partially open, as it was during the transition from [z] to [h]. In terms of our original interest, a comparison of the fourth and seventh frames (for [b] and [t]) shows clearly the contrasting adjustments of the larynx for voiced and voiceless stops. There would be some real advantage in photographing at higher frame rates, although 120 frames per second should fully delineate even rapid laryngeal adjustments. There is no real difficulty in doing this, although it requires a different camera and a more intense light source. We are

preparing to use a Milliken camera that can operate to 500 frames per second (pin registered) and a Xenon arc with more efficient optics. An important advantage of more light is, of course, that we can expect to get better color, since forced development will not be needed. Analysis of the film and correlation with the speech sounds will be considerably simplified by photographing a numerical code at one side of each frame, and registering the same code on the magnetic tape that carries the speech recording.

The slow motion pictures of the present film are not nearly so easy to follow as they would be if they were accompanied by a synchronized sound track. There are no straightforward methods for doing this when the time scale is stretched, but it is possible to generate slow speech in step with the slow motion pictures by first analyzing the speech into its spectrum, and then re-synthesizing it at the rate required to match the picture track. Computer methods make this a comparatively simple operation, and we know from experience with slow motion x-ray films that the result is very effective.

Finally, simultaneous transillumination and photography should give additional confirmatory evidence about some of the more rapid events, and should help to fill in occasional gaps in the pictorial record, for example, when the glottis moves out of the field of view. Again, the techniques are manageable.

SUMMARY

We have found it possible and useful to observe directly the changing laryngeal adjustments during ongoing speech. We believe that substantial improvements in technique can be made and that the experimental methods described here are likely to yield significant insights into the coordinated gesture system that is called speech.

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SUBTLE LARYNGEAL ADJUSTMENTS

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Gross features of the larynx, such as color, size and symmetry of the several structures, adduction and abduction of the vocal folds, and the presence of pathology are scanned easily with simple mirror techniques. The usual laryngeal examination for medical purposes necessarily searches for evidence of disease or source of discomfort. However, the experienced examiner also observes the laryngeal configurations that are present during phonation and associates them more or less directly with the sounds produced. These correlations are noted particularly when the primary complaint of the patient is hoarseness, and vocal abuse is suspected as the basic cause. During phonation, the glottal and supraglottal areas are seen to vary from relatively tight constriction, during which the vocal folds are obscured from view, to an open larynx with vibration of the folds without closure.

Despite the substantial quantity of information obtained by standard indirect observation of the larynx, it is apparent that much occurs that is not detectable by this procedure. Rapid and delicate changes in arytenoid position, modification of glottal contour, tightness of glottal closure and detail of vocal fold vibration are not revealed to the unaided eye. These subtle adjustments are essentially time related and consequently can be made visible only through some special procedure such as ultra slow motion photography. This photographic technique is too complex in its present stage of development for routine clinical work. However, it can contribute importantly to the management of certain laryngeal problems. This philosophy is the primary motivation for much of our research program.

The subtle laryngeal adjustments on

which this paper focuses occur during the production of normal voice. They include glottal configurations and the motions of the vocal folds during phonation.

Research has demonstrated that there are two basic locations or positions of rest from which the vocal folds begin to vibrate. They may stand apart so that the free edges do not touch each other to any considerable degree as they sweep toward and away from each other, or they may be in contact along their glottal margins, thereby providing for glottal closure within each vibratory cycle. There is a scale of variation in the amount of contact between the vocal folds during vibration in relation to these two types of basic adjustment. At one extreme the glottis may never be completely closed; at the other, it may never be completely open, and between, there appear to be all degrees of incomplete closure. Furthermore, in most larynges, the changes from one extreme to the other with intermediate variations, can and often do occur within extremely brief periods of time.

When the rest positions are separated, each vocal fold is capable of oscillating independently. Both usually vibrate with the same frequencies, amplitudes and phase relationships, because their structures are similar and a common air stream activates them. However, if there is greater mass or elasticity in one vocal fold than the other, differences in Hertz, amplitude, phase, and contour may occur. The tendency toward independent movement is lessened when the folds contact each other during vibration.

The force of contact between the vocal folds during vibration varies from a delicate touch to strong impact. Since

vigorous impact occurs in loud voice and certain vocal qualities, and since these forms of voice production may be related to laryngeal trauma, this factor of impact acquires particular significance in this discussion.

(A short motion picture film was then shown assembled from research films, illustrating some of the structural relationships to which reference had been made and describing a few of the subtle adjustments which occur at the glottis.)

PAPILLARY LESIONS OF THE LARYNX IN ADULTS

A PATHOLOGIC STUDY

STANTON A. FRIEDBERG, M.D.

ROBERT STAGMAN, M.D.

GEORGE M. HASS, M.D.

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Papillary lesions of the larynx in adults may present laryngologists with several problems in diagnosis and management. In a previous report concerning 24 patients with laryngeal malignancies simulating benign growths, seven patients with innocent-appearing papillary lesions were found to have carcinoma.⁵ In five patients the lesions were small and localized; in the other two diffuse papillomatous masses were seen.

Another challenge has been the inability to differentiate lesions destined to remain benign from those, which although initially apparently benign, will undergo malignant transformation at some future time. This latter group must also include lesions which may be malignant initially, but from which random biopsies may not reveal the true nature of the lesion and consequently may be misinterpreted as benign.

A problem familiar to all laryngologists is that of the keratotic lesion, frequently papillomatous, which may undergo frank malignant transformation after being observed and treated with suspicion and some apprehension as a benign growth for varying periods of time.

The critical importance of this differentiation lies in the ability, through early detection, to initiate definitive therapy for the incipient malignancy at a time when such therapy can be expected to yield the highest possible cure rate. Although certain factors such as age and sex, socioeconomic status, personal hab-

its, duration of symptoms, and the gross clinical appearance of the lesion may suggest either benignity or malignancy, definitive information must ultimately depend on the microscopic examination and interpretation of tissue obtained from the lesion.

The purpose of this study is to determine those microscopic features that may indicate incipient malignancy in apparently benign papillary laryngeal lesions occurring in adults.

HISTORICAL ASPECTS

Over 30 years ago, New and Erich's report¹¹ of 722 benign laryngeal lesions indicated that epithelial hyperplasia, or "keratosis," was a potentially malignant lesion. Features which they felt pointed to an early malignant papillary lesion included a solitary lesion, inflammatory reaction around the tumor base and ulceration. They advised therapy of papillary lesions with electrocoagulation. Clerf³ discussed eight patients with laryngeal keratosis of whom four developed epidermoid carcinoma. He wrote that it was impossible to predict the course of the lesion from the initial pathology. Gordon⁷ believed that keratosis of the larynx was "predominantly precancerous," and recommended early complete excision by thyrotomy and cordectomy if there was any question as to the clinical or histologic appearance.

Niskanen¹² felt that keratosis of the larynx could be reversible following removal of any of several precipitating

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causes among which he mentioned vocal abuse, alcohol, tobacco, and chronic respiratory infection. Two of his 12 patients developed carcinoma, and he stressed the difficulty of defining the premalignant lesion on the basis of cellular morphology. Grossman and Mathews⁸ reported carcinoma developing from a benign keratosis after four years, and they advocated stripping of involved mucous membrane for keratosis.

Putney and O'Keefe¹⁵ reported 27 carcinomas which developed in 125 observed cases of benign keratosis. They believed that gross papillary projections were suggestive of early malignancy while the earliest histologic change was felt to be intraepithelial carcinoma *in situ*. They recommended radiation for extensive recurrent benign lesions. Negus et al.¹⁰ advocated cordectomy for any keratosis not responding to local stripping.

Bjork and Teir¹ compared benign and malignant papillomata histologically and stressed the importance of intraepithelial anaplasia as an indicator of malignancy. Other important criteria seen only in malignant lesions were loss of intracellular bridges, loss of polarity, and pathologic mitoses. Bjork and Weber² previously noted that limitation of cord mobility on clinical examination was an important sign of malignancy.

Priest¹⁴ stated that any keratosis occurring over ten years would inevitably progress to invasion. He questioned the efficacy of radiation therapy. Cracovaner⁴ believed that atypical cells progressing to carcinoma *in situ* were the earliest indicator of malignancy and that malignant lesions were more likely to show chronic inflammation and distortion of deep layers of the epithelium. He recommended laryngectomy despite benign biopsies for extensive lesions.

McGavran et al.⁹ emphasized that no etiologic relationship had been established between laryngeal keratosis and carcinoma. They stressed conservative therapy, relying upon follow-up laryngoscopy to identify developing carcinoma. Only three of 84 patients with

benign keratosis developed epidermoid carcinoma. Gabriel and Jones⁶ studied 30 instances of benign laryngeal keratosis. Six were treated with radiation, of which one developed carcinoma. They stressed the regression of keratosis, despite atypical histology, under medical therapy.

Norris and Peale¹³ studied 116 patients with benign keratosis of whom 12 developed carcinoma. They concluded that review of histology gave no clue as to which patients would develop malignancy, but that the necessity of multiple removals increased the probability of malignant degeneration. They recommended treatment by laryngoscopic removal until malignancy was proven.

It is apparent from the above review that a wide diversity of opinion exists with respect to the management of benign papillary and keratotic laryngeal lesions.

METHOD

This study is based upon retrospective microscopic examination of tissues from papillary laryngeal lesions in a group of patients who eventually developed epidermoid carcinoma requiring laryngectomy, despite at least one preceding biopsy which was interpreted as benign. The pathologic findings in this group were considered and interpreted relative to generally accepted pathologic criteria for benignity in squamous papillary lesions. The criteria are as follows: a) preservation of polarity — plump basal cells oriented perpendicular to the basement membrane progressing to gradual flattening and elongation towards the surface; b) uniformity of nuclear size and shape within each stratum of the tissue; c) infrequent mitoses which are morphologically normal; d) cellular uniformity within the basal layer without proliferation or nuclear hyperchromicity; and e) absence of intraepithelial anaplasia.

In helping to arrive at these criteria, we reviewed pathologic material from our population of patients with benign papillary laryngeal lesions.

In this survey, the group in which malignant degeneration of a papillary laryngeal lesion occurred consisted of five pa-

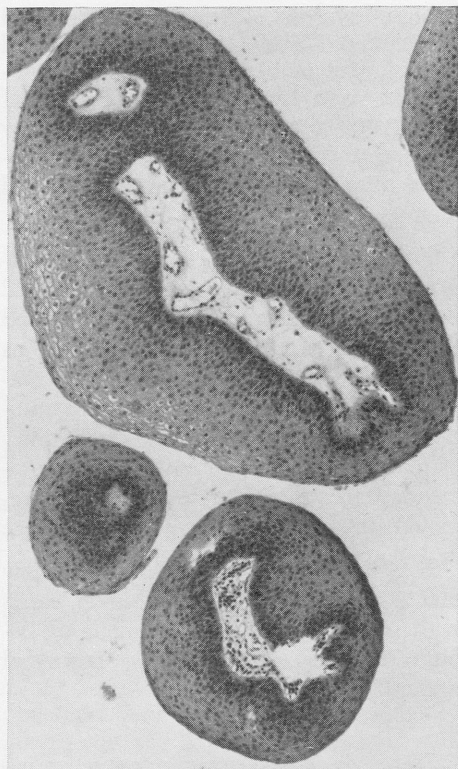


Fig. 1. Case 1. Recurrent benign laryngeal papilloma in patient age 91, 16 years after onset. Note orderly basal layer, uniform size of cells, uniform cell polarity and rare mitoses.

tients. We are reporting an additional patient whose laryngeal biopsies showed strong indication of incipient malignancy, but in whom a diagnosis of epidermoid carcinoma cannot, as yet, be made. The group consists of five males and one female with an age range from 45 to 79 years.

Eight additional adult patients (6 males and 2 females) with recurrent unmistakably benign papillomas have also been studied and followed during the present study.

The following case reports describe laryngeal pathology from five patients with malignant papillary laryngeal lesions and one patient with what we believe to be an incipient carcinoma.

REPORT OF CASES

Case 1. This 91-year-old male was first seen at age 75 with hoarseness of several months' duration. Initial examination revealed a papillary lesion involving the entire endolarynx. He was treated by laryngoscopic removal of the tumor, and over the following 16 years until the present has required laryngoscopic removal of this papillary laryngeal lesion on 30 occasions for hoarseness and obstruction. The pathologic report has continued to be "papilloma without atypia." The patient has remained well without indication of malignancy on repeated histologic study.

Pathology. A representative section from his most recent laryngeal biopsy (Fig. 1) serves as a baseline for comparison with the following cases. The basal layer is orderly and regular. Polarity is maintained. Nuclear size and shape is uniform. Mitoses are rare and, when seen, are morphologically normal. There is no evidence of intraepithelial anaplasia.

Case 2. This 53-year-old male presented with a five month history of hoarseness. Indirect laryngoscopy revealed a papillary exophytic lesion involving the right true cord, ventricle, and false cord, left true cord, and posterior commissure (Fig. 2). Over the following nine months he underwent direct laryngoscopy on six occasions and thyrotomy on two occasions for a rapidly proliferating lesion which, on pathologic examination, was repeatedly called "hyperkeratosis" and "squamous papilloma." The gross appearance of the lesion demanded repeated biopsies, and only after nine months was a diagnosis made of epidermoid carcinoma.

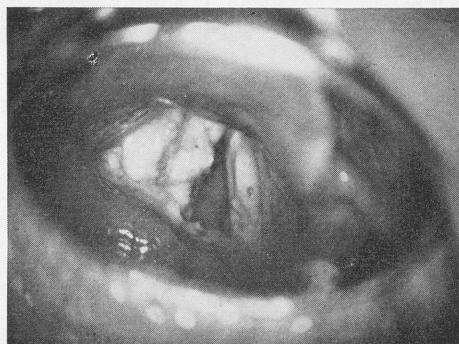


Fig. 2. Case 2. Mirror view of ulcerating papillary exophytic lesion involving right ventricular band, right true cord, right ventricle and posterior commissure.

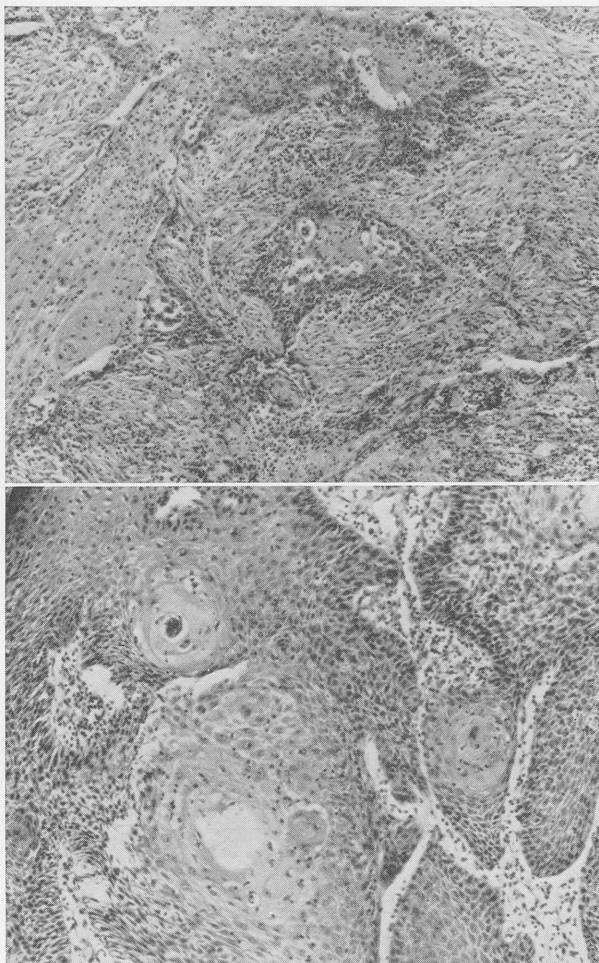


Fig. 3. Case 2. Biopsy specimen removed eight months prior to laryngectomy. This epithelial tissue is characterized by loss of cell polarity, nuclear variation, abnormal cells, deep intraepithelial pearls, all indicative of marked intraepithelial anaplasia.

Fig. 4. Case 2. Tissue obtained via thyrotomy eight months prior to laryngectomy. Changes in the basal cell layer, hyperchromatism, nuclear atypia and pearl formation are evident and are limited to the surface epithelium.

Laryngectomy and neck dissection was performed. Pathologic examination confirmed metastasis to one cervical node. Eighteen months postoperatively he was free of tumor, as confirmed by autopsy examination following death from alcoholism and pneumonia.

Pathology. Examination of biopsy material taken from this lesion eight months prior to laryngectomy (Fig. 3) revealed loss of polarity and marked intraepithelial anaplasia characterized by nuclear variation, abnormal

cells, and formation of deep intraepithelial pearls. Tissue obtained at thyrotomy, again eight months prior to laryngectomy (Fig. 4), revealed striking changes in the basal layer with intense proliferation, hyperchromatism, and nuclear atypia.

The laryngectomy specimen demonstrated a pattern of dramatic epithelial change from well differentiated, keratinized tissue at the surface to anaplastic, invasive neoplasm beneath. This pattern is illustrated in the following case.

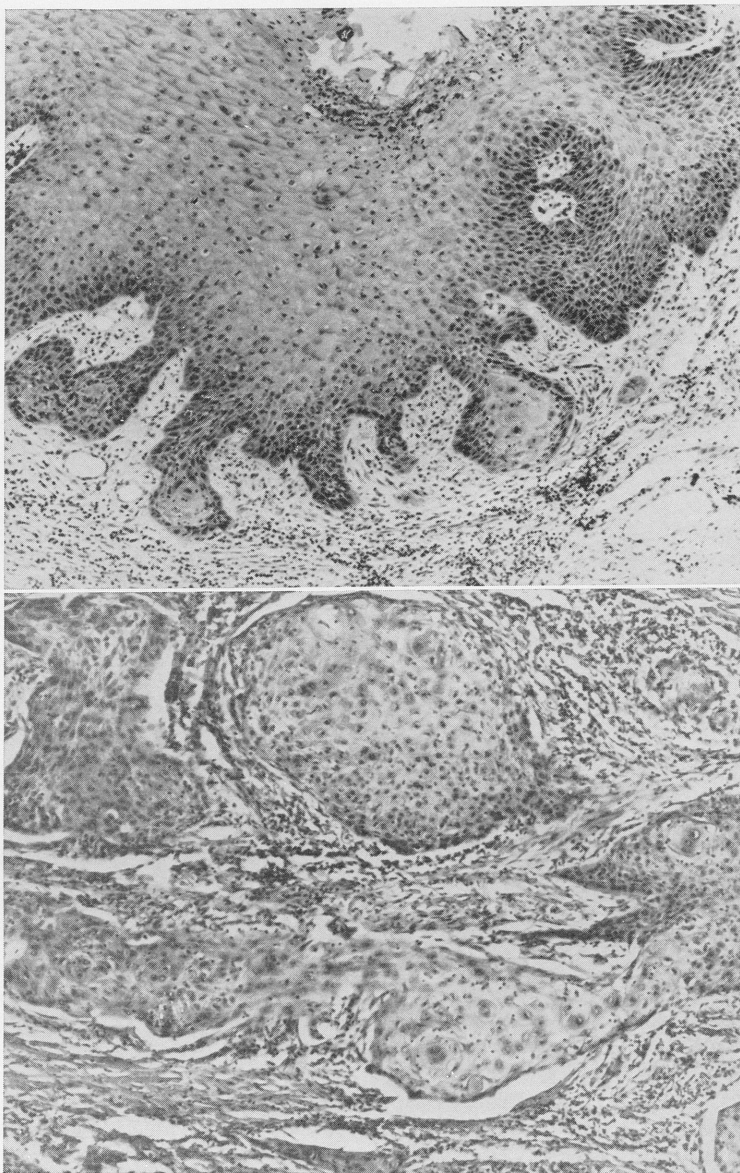


Fig. 5. Case 3. Biopsy specimen 16 months prior to laryngectomy. Deep pearl formation is evident in the disorganized depths of several epithelial pegs.

Fig. 6. Case 3. Specimen from larynx, following its removal, illustrates the contrast between the benign hyperkeratotic surface epithelium and the invasive carcinoma below.

Case 3. This 79-year-old male presented with increasing airway obstruction and hoarseness of several months' duration. He subsequently underwent direct laryngoscopy on ten occasions over a 16 month period for removal of a rapidly recurring papillary laryn-

geal mass diagnosed pathologically as "hyperkeratotic papilloma." After 16 months a diagnosis of epidermoid carcinoma was made and laryngectomy performed. He has remained free of disease for two years.

Pathology. Sixteen months prior to laryn-

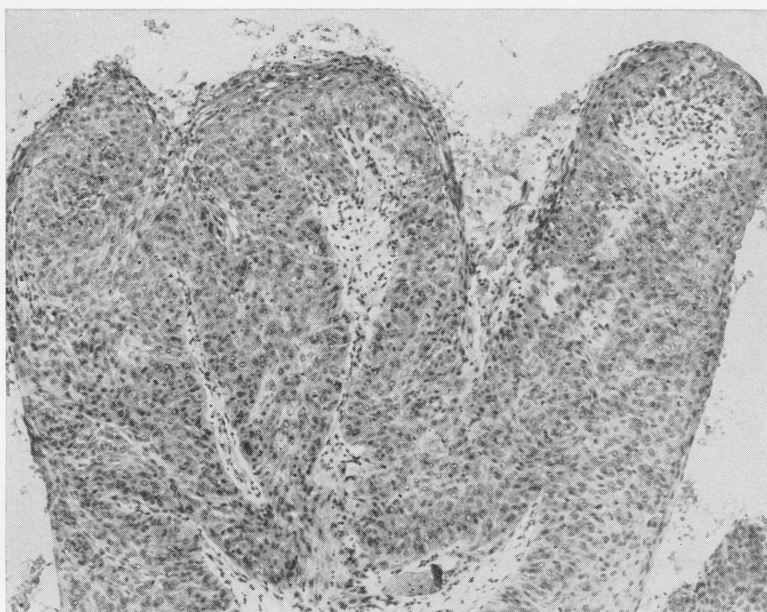


Fig. 7. Case 4. Biopsy specimen obtained six months prior to laryngectomy showing papillary projections characterized by intraepithelial anaplasia with loss of polarity, nuclear variation, abundant mitoses and basal layer disorganization.

gectomy, biopsy of the laryngeal lesion (Fig. 5) showed conspicuous deep pearl formation in the disorganized depths of several epithelial pegs. The laryngectomy specimen (Fig. 6) strikingly demonstrates the contrast between the benign hyperkeratotic surface epithelium and the invasive carcinoma at the base.

Case 4. This 44-year-old female required tracheotomy for an extensive papillary lesion of the larynx diagnosed as "squamous papilloma." Laryngoscopic removal was required on three occasions over the next year. There was no recurrence until nine years later, following which she required nine laryngoscopies over the next 15 months for a rapidly recurring papillary lesion which was repeatedly called "squamous papilloma" on pathologic examination. Eventually a diagnosis of invasive epidermoid carcinoma was made, 11 years after onset of the lesion, and laryngectomy performed. Two years postoperatively she was free of disease.

Pathology. Eighteen months prior to laryngectomy, deep intraepithelial pearls were seen in biopsy material. Six months prior to laryngectomy (Fig. 7) there was marked intraepithelial anaplasia with loss of polarity,

marked nuclear variation, frequent mitoses, and basal layer disorganization.

Case 5. This 45-year-old male presented with hoarseness of eight months' duration. Mirror examination of the larynx revealed a papillary lesion involving the right true cord, aryepiglottic fold, and laryngeal surface of the epiglottis. Biopsy was read as "squamous papilloma and leukoplakia." Rapid recurrence prompted repeat laryngoscopy three months later, and a diagnosis of epidermoid carcinoma was made. Total laryngectomy was performed, and two years postoperatively he was free of disease.

Pathology. Three months prior to laryngectomy, biopsy (Fig. 8) showed prominent intraepithelial anaplasia with loss of polarity, basal layer disorganization and hyperchromatism, marked nuclear variation, and deep pearl formation.

Case 6. This 45-year-old male presented in October, 1966, with hoarseness of many years' duration. Laryngoscopy revealed a papillary exophytic lesion involving the left true cord, anterior and posterior commissure with subglottic extension posteriorly. Biopsy was read as "papilloma with marked atypical epithelial hyperplasia." One month later tra-

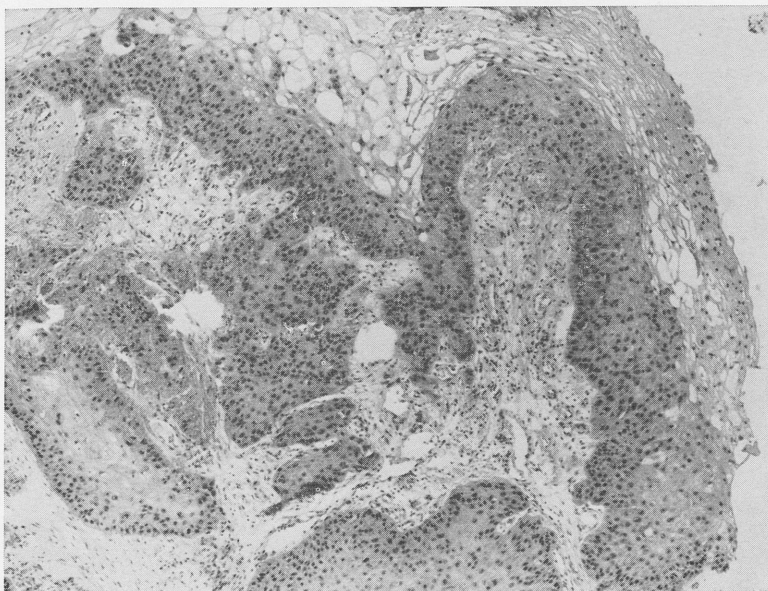


Fig. 8. Case 5. Biopsy specimen obtained three months prior to laryngectomy. There is hyperkeratosis of the surface epithelium, prominent intraepithelial anaplasia with loss of polarity, basal layer disorganization and hyperchromatism, considerable nuclear variation and deep pearl formation.

cheotomy was required for dyspnea due to the obstructing mass. Over the next 18 months laryngoscopy was performed six times for removal of large masses of papillomatous tissue. Pathologic interpretation continued to be "squamous papilloma."

In April, 1968, dysphagia developed, and esophagoscopy and barium swallow revealed extraluminal compression of the cervical esophagus anteriorly. Exploratory pharyngotomy in May, 1968, confirmed a mass in the tracheoesophageal party wall which was read as epidermoid carcinoma on frozen section. Total laryngopharyngectomy was performed. A protracted period of reconstruction followed. Eighteen months postoperatively recurrence of carcinoma appeared in the post-surgical scar which was felt to be inoperable, and palliative radiation therapy was begun.

Pathology. Review of the initial biopsy material taken 19 months before laryngectomy was performed (Fig. 9) demonstrated marked anaplasia. Deep intraepithelial pearls were present. There was loss of polarity, frequent mitotic figures, basal layer disorganization, and prominent nuclear variation with abnormal cell forms and several tumor giant cells.

The last case is presently included in our

series of benign papillary lesions, but is the only member of this group to show the pathologic changes which we feel are indicative of incipient malignant degeneration. Our pathologic indicators suggest that this patient is a prime candidate for overt malignancy if, in fact, it is not already present.

Case 7. This 68-year-old male had a history of laryngeal papilloma beginning at the age of four years. However, he was relatively asymptomatic until age 60 when rapid growth of a diffuse papillary laryngeal lesion became apparent. Since age 64, he has required a tracheotomy because of airway obstruction. Laryngoscopy with removal of large masses of papillary tissue has been required on four occasions within the last two years. Pathologic interpretation has on all occasions been "papilloma with dysplasia." The patient remains generally well, but is hoarse and continues to require his tracheotomy.

Pathology. A biopsy obtained from the papillary laryngeal lesion in this patient almost 36 months ago (Fig. 10) demonstrated an area of marked intraepithelial anaplasia compatible with carcinoma in situ. There was complete disorganization with loss of polarity,

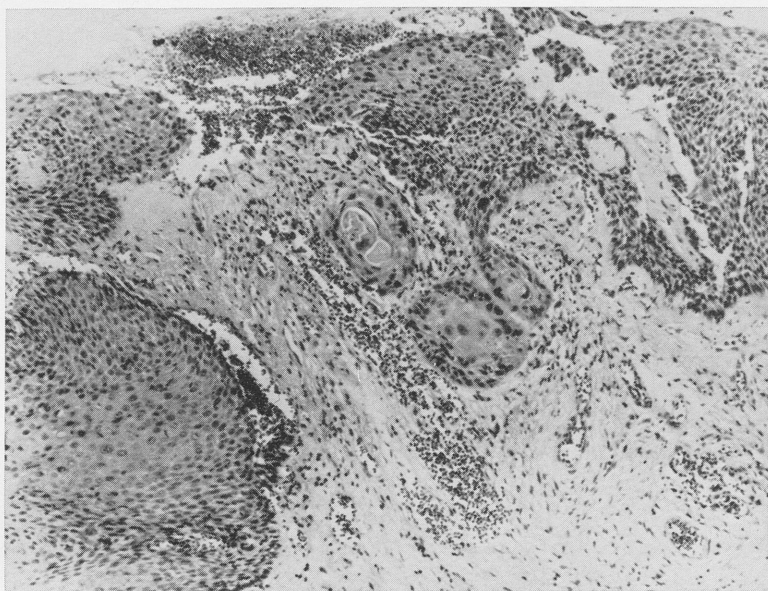


Fig. 9. Case 6. Biopsy specimen from an extensive papillary exophytic lesion obtained 19 months prior to laryngectomy. Marked intraepithelial cellular disorganization, deep intraepithelial pearls, abnormal cell forms, nuclear variations and several giant tumor cells are evident.

marked nuclear variation with bizarre forms, and frequent abnormal mitoses.

DISCUSSION

From detailed retrospective microscopic examination of laryngeal biopsies in adults with papillary lesions of the larynx, it is our impression that those patients destined to develop epidermoid carcinoma can be identified at an early date. The points which we feel are of importance to facilitate the earliest possible detection of an incipient malignancy in these lesions are described below.

Adequate biopsies of the lesion are mandatory. Since the ability of an invasive carcinoma to hide beneath a hyperkeratotic, histologically benign surface epithelium has been clearly demonstrated, the depths of the lesion must be sampled. If laryngeal biopsy forceps are employed, several biopsies should be taken at progressively deeper levels within one area of the lesion. If any question arises as to the adequacy of biopsies, or there is concern over the clinical appearance or course of the le-

sion, thyrotomy with open biopsy or cordectomy should be employed at an early date. To withhold open biopsy from the patient with the idea that it is too "radical" may prove disastrous, as seen in Case 6.

Once tissue is obtained, the pathologist must be properly oriented to the specimen and order sections to be made from surface to base, thus avoiding tangential surface cuts where malignancy is unlikely to be detected, even though present in the specimen. Figure 6 illustrates that cuts parallel to the surface will not detect the malignancy until the edge of the tissue block is reached, whereas cuts from surface to base will readily reveal the invasive lesion.

Microscopically, the incipient malignancy heralds its appearance with intraepithelial anaplasia characterized by loss of polarity, basal layer disorganization with proliferation and hyperchromicity, nuclear variation with cellular dysplasia, increase in mitoses with abnormal forms, and epithelial swirling with deep pearl formation. Early diagnosis

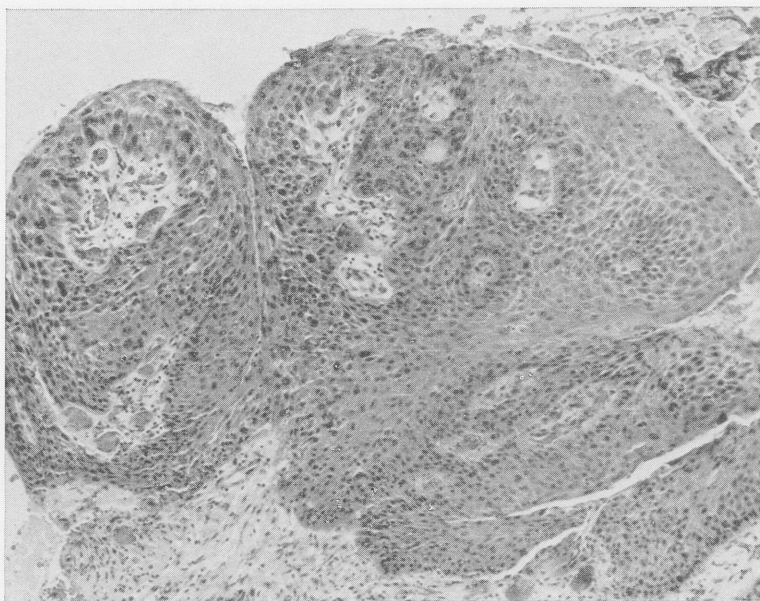


Fig. 10. Case 7. Biopsy specimen obtained 36 months previously in a patient who continues to display papillomatous regrowths without frank invasive carcinoma. This represents marked intraepithelial anaplasia of a high degree. Note marked nuclear variations with bizarre forms, loss of cellular polarity and prominent mitotic figures.

demands meticulous scrutiny of all biopsy material. Our review has revealed markedly dissimilar pathologic material appearing in different tissue fragments taken at the same time. Detection of any of the previously mentioned pathologic indicators of early malignancy demands vigorous perusal of truly representative biopsy material.

When malignancy develops in these lesions, both their well-differentiated character (with only late metastatic potential) and their tendency to be limited to the endolarynx should yield high cure rates with definitive surgery. However, the neglected lesion allowed to infiltrate locally under the mistaken impression of benignity creates a formidable therapeutic problem with dismal hope of long term survival.

Continued pursuit of accurate clinicopathologic correlation in these treacher-

ous lesions may eventually allow true prophylactic partial or total laryngectomy to be undertaken before advanced local invasion can occur. The diagnosis and management of the adult patient with a papillary laryngeal lesion may severely tax the abilities of both the laryngologist and pathologist.

SUMMARY

The history of the management of papillary lesions of the larynx in adults is briefly reviewed. A study based on retrospective microscopic examination of laryngeal biopsies in patients with papillary lesions who subsequently developed epidermoid carcinoma has been undertaken. Pathologic features felt to be of importance in detecting incipient malignancy in papillary lesions are illustrated. Points of importance in the early diagnosis of malignancy in these lesions are outlined.

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DISCUSSION

JOHNSON PUTNEY, M.D.: I was privileged to study Drs. Friedberg's and Stagman's paper and their presentation of this puzzling condition has been well thought out.

As they have pointed out carcinoma may exist deep within masses of papilloma, and when multiple areas are involved, it is impossible to remove representative tissue from each separate lesion. The possibility of undetected carcinoma or progression to malignancy requires multiple and frequent biopsies.

The interpretation of cellular pathology has been a great help, and when histologic activity is present, malignancy seems destined to occur sooner or later. It is questionable whether an aggressive approach is desirable, for if the patients can be cured by laryngectomy after malignancy develops, this is probably the correct course as illustrated by these 7 cases with one death from cancer. In my experience this type of lesion has been too extensive to be treated by any method other than laryngectomy, so that even if malignancy could have been predicted, laryngectomy would have been necessary, and conservation surgery was not possible. Perhaps prophylactic surgery in lesions exhibiting cellular atypia and dysplasia may eventually be used, although removal of a larynx, unless malignancy can be demonstrated, seems unjustified.

I have managed these lesions much in the manner outlined with repeated biopsies, but have been disturbed by the results in two cases. I waited for the development of malignancy, being almost certain in my own mind that this would occur, and after laryngectomy they died from local recurrence around the tracheal stoma with extension into the mediastinum.

At one time irradiation seemed to offer some help, but this has not been borne out in these well-differentiated squamous lesions and has been discontinued.

The authors' handling of this difficult and still controversial subject points up the importance of the clinical evaluation of the lesion and the judgment of the laryngologist.

DR. STANTON A. FRIEDBERG: I would like to mention that there is an annual residents' competition in the City of Chicago for the best essay and the man you have heard this morning, Dr. Stagman, was a co-winner of the Norval Pierce Award last year.

Dr. Stagman mentioned that this was a retrospective study and it is indeed true that when one goes back over all of the material and adds additional sections, some things come to light which were not initially apparent.

Secondly, it's hard to understand or explain the biology of some of these tumors. Two of our patients carried papillomas around for sixty years and others developed neoplastic changes within a relatively short period of time.

Our dilemma in the care and management of these lesions is that if you wish to bite deeply enough to be sure you're uncovering the pathology in the depth of the lesions, you worry that the voice will be altered by this procedure.

We also have to recognize the necessity for orienting the pathologist to the specimens you remove and for the need for cutting the entire block because one sees changes in the depths which may not be seen in the initial few sections.

Perhaps laryngeal microsurgery will help in our selection of biopsy sites and perhaps the electron microscope may add to the solution of this rather difficult problem.

DR. ROBERT STAGMAN (closing): I would like to thank the discussers for these points.

I think the early entry into the larynx by open biopsy for adequate biopsy material has been very helpful and a number of diag-

noses of invasive carcinoma have been made by wedge biopsies obtained at thyrotomy. This allows one to get a much better piece of tissue and the pathologist can be very readily oriented to the specimen. It is a great deal more difficult when the typical punch laryngeal biopsies are taken.

Certainly the point about a prophylactic laryngectomy is well made. A number of our patients coming to laryngectomy were already severely obstructed, requiring long term tracheotomy; were virtually aphonic with the exception of very hoarse and almost inaudible voices; had dysphagia secondary to ulceration of the posterior commissure and arytenoid and had frequent aspiration secondary to incomplete posterior glottic closure.

I think that with changes of this kind observed pathologically, plus the symptoms which I have just outlined, perhaps the recommendation of laryngectomy is really not to be considered radical at this stage.

Secondly, we have observed the late effects in one of our patients of waiting until a diagnosis was established and a total laryngectomy had to be performed. Following this there was recurrence after long delayed reconstructive procedures and this was an extremely disappointing outcome.

ELECTRON MICROSCOPIC IDENTIFICATION OF VIRUS-LIKE PARTICLES IN LARYNGEAL PAPILLOMA

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The emerging role of viruses in the etiology of human neoplastic diseases has reinforced interest in defining the elusive causative agent in laryngeal papilloma. A rational therapeutic approach might be anticipated should such an agent be identified. Most of the evidence found in the literature at this time points to a viral etiology. The purpose of this study was to examine the ultrastructure of papillomas of children and adults for viral particles, and to identify virus within the cells of the tumor by immunofluorescent techniques.

METHODS AND MATERIAL

Tissues from seven different papilloma specimens obtained from three children and one adult were examined in this study. Two children and the adult had specimens obtained on two separate occasions. All specimens were obtained by surgical excision at direct laryngoscopy.

Five serum samples were also examined in an attempt to find circulating antibodies against the tumorigenic virus. Four of the serum samples were from the above patients and one was from a patient who was considered cured of his disease, and from whom no tissue was obtained for study. This patient had been treated surgically on repeated occasions with complete regression of his disease over the past three and one-half years. This patient was included in the study since it was felt there was an excellent chance of finding circulating antibody in his serum.

Tissue specimens were examined by light and electron microscopy and by immunofluorescent techniques. Light microscopy was performed after standard paraffin block sectioning technique and staining with hematoxylin and eosin.

Electron microscopy was performed after overnight fixation in 3% distilled glutaraldehyde buffered to pH 7.4 with phosphate or cacodylate buffer. Postfixation was for 90 minutes in osmium tetroxide, dehydration in ethanol and embedding in araldite.

In the immunofluorescent study, frozen sections of tissues were examined by both single and double plating techniques. Single plating (direct) technique was done with fluorescein-tagged antihuman globulin obtained from rabbits. This was also used for the over-stain in the double plating technique. This serum contained antibodies against human IgG, IgA, and IgM. Portions of normal human pharyngeal mucosa were used as negative controls.

The double plating technique (indirect) was done in three ways, with different sera used in the intermediate step: 1) Patient's autologous serum; 2) Homologous and autologous serum pooled from the five samples; and 3) Antihuman globulin serum (Coomb's) which was not fluorescent tagged. The third method was done to induce blocking of fluorescent staining, and help confirm specificity.

Staining was done in the following manner: frozen sections on clean glass slides were washed twice for five minutes each in phosphate buffered pH 7.2 saline at room temperature, then immersed in cold (4°C) acetone for 30 seconds, then washed twice more with buffered saline. In the single plating technique, slides were incubated with fluorescent tagged antiserum for 30 minutes at 37°C in a moist chamber and washed twice in buffered saline. The slides were dried and cover slips were applied using glycerol mounting medium. They were examined with a fluorescent microscope equipped with a dark field stage. In the double plating technique,

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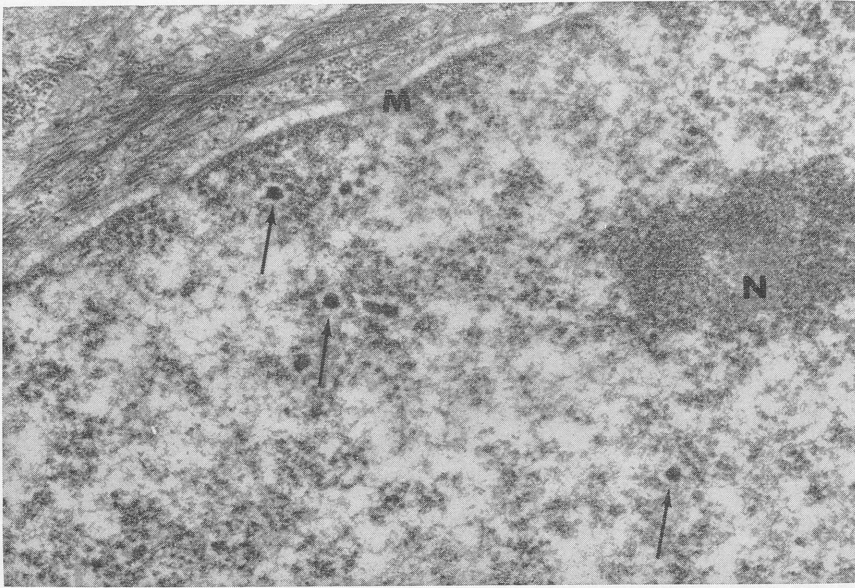


Fig. 1. Cell from superficial layer of epithelium showing viral-like particles (arrows). Size approximately $40\text{ m}\mu$ scattered within nucleus. M: nuclear membrane; N: nucleolus. Numerous tonofibrils in cytoplasm. X50,000.

slides were incubated with the serum being tested (corresponding to one of the three aforementioned methods) in a moist chamber for 30 minutes at 37°C and washed twice. They were overstained with fluorescent tagged antiserum for 30 minutes at 37°C in a moist chamber, washed twice and examined with the fluorescent microscope.

RESULTS

By light microscopy, all the lesions exhibited the usual features of typical squamous papilloma. By electron microscopy, the lesions showed a fairly uniform pattern. The epithelial cells showed many small granules present in the cytoplasm which appeared to be glycogen. Rough endoplasmic reticulum was present, though not prominent. Mitochondria were present but not plentiful. The cytoplasm of all the epithelial cells contained numerous tonofibrils. Each cell had numerous interdigitations of the cytoplasmic membrane with desmosomes at each interdigitation with the adjacent cell. Tonofibrils ran into the cytoplasm from each desmosome. The cells were packed closely together in the basal layers, becoming more widely separated in the

more superficial layers. In the superficial layers, the intercellular spaces widened, with elongation of the interdigitations corresponding to the intercellular bridges characteristic of squamous epithelium seen by light microscopy. The nuclei were rounded to oval and generally the nuclear membranes were quite regular. The nucleoplasm were regular and evenly distributed, and many nuclei contained nucleoli. The overall appearance was typical of the structure described in previous ultrastructural studies of papillomatosis lesions.^{4,8,9,14,15}

Exhaustive scrutiny failed to reveal particles with definite discernible virus-like structure, or crystalline lattice-work assemblies characteristic of virus. However, many nuclei showed dense bodies surrounded by clear haloes scattered singly throughout the nucleoplasm (Fig. 1). These were of fairly uniform size, measuring around 40 millimicrons, with a range of 38 to 45 millimicrons. Also seen in the nucleus of one cell was an aggregation of dense particles which were poorly defined but appeared similar in size and configuration to the afore-

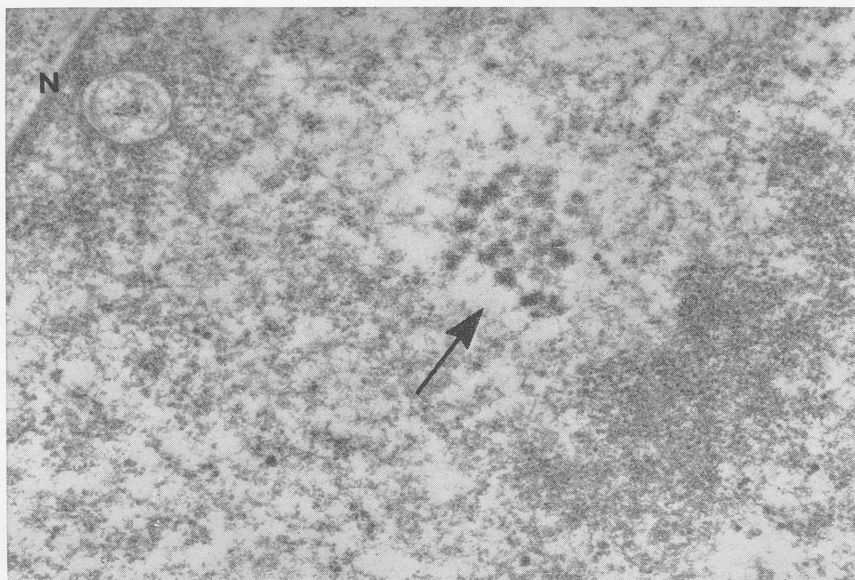


Fig. 2. Aggregation of dense bodies (arrow) in nucleus of cell from superficial epithelial layer. Not as clearly defined as particles in Fig. 1 but with same size range. N: nuclear membrane. X35,000.

mentioned single dense bodies (Fig. 2). These dense bodies were found in the nuclei of all specimens examined.

In the immunofluorescent staining technique, a lesion was considered positive when there was definite intranuclear fluorescence. Cytoplasmic fluorescence was considered negative. With the single plating technique, three specimens showed definite intranuclear fluorescence (Table I). Of the three, two were from children and one from an adult. With the double plating technique the following results were obtained: 1) Autologous

serum: three of five specimens positive; two from children and one from the adult; 2) Homologous and autologous serum: five of seven specimens positive; and 3) Blocking serum: only one lesion continued to show fluorescence.

Fluorescence was observed only in random nuclei in the more superficial layers (Fig. 3), with the great majority of nuclei being entirely negative. Only minimal cytoplasmic fluorescence was noted in one specimen. Fluorescence was not demonstrable in any of the deeper layers.

TABLE I
RESULTS OF IMMUNOFLOUORESCENT TECHNIQUES. SPECIMENS C AND E
FROM ADULT

	SPECIMEN							Control
	A	B	C	D	E	F	G	
Single plating	—	—	+	+	—	—	+	—
Double plating (autologous serum)	—	+	N.O	+	+	—	N.O	—
Double plating homologous and autologous pooled serum	—	+	+	+	+	—	+	—
Double plating (blocking technique)	—	—	—	—	—	—	+	—

N.O: not observed

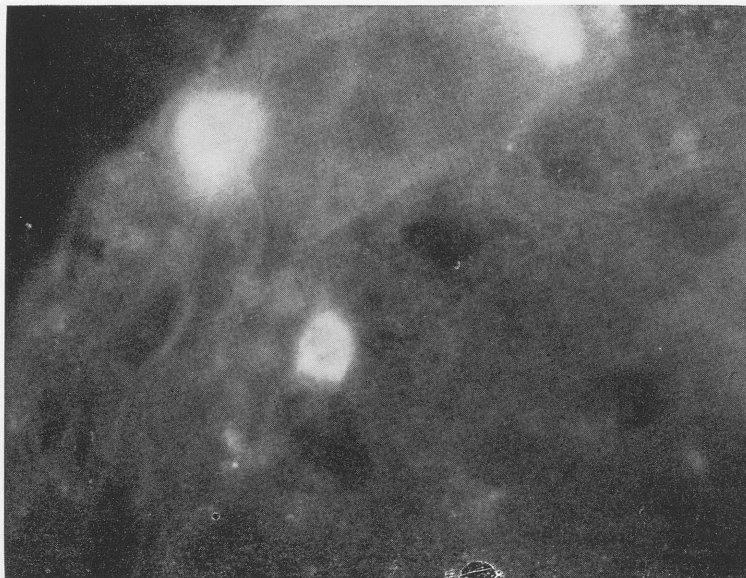


Fig. 3. Immunofluorescent stained section (single plating) from superficial layers of lesion with individual nuclei showing fluorescence.

DISCUSSION AND CONCLUSIONS

Virus-like particles have been reported in electron microscopic studies of human intranasal⁹ and oral^{6,7} papillomas. Intranuclear viral-like particles have been described in laryngeal papilloma by one investigator.⁴ The morphology of these particles is consistent with the characteristics of the papova group of viruses as defined by Melnick.¹⁰ These characteristics include a DNA virus of primarily intranuclear location; a spherical rather than helical configuration; and a size range of approximately 40 to 50 millimicrons. These viruses are always multiple within the nucleus, usually in close association with the nucleolus.¹³ Previous reports suggesting viral-like particles in the cytoplasm of the lesion² probably do not represent viruses associated with this disease.

Particles identified in this study are similar in size and configuration and ultrastructural appearance to those papilloma viruses previously reported. The particles are also similar in appearance to members of the papova virus group which have been definitely identified as the causative agent in papillomas of

rabbits,¹³ horses,⁸ dogs,^{3,15} and in human cutaneous¹ and genital⁵ warts. Why then has the laryngeal papilloma virus been so difficult to identify? Shope,¹² in his work with the papilloma virus in rabbits, offers a plausible explanation. The virus exists in two states within the nucleus of the infected cell. First, it exists as the complete or mature virus capable of stimulating antibody production; is identifiable by electron microscopy; and can be made visible within the cells of the tumor by treatment with immunofluorescent techniques. Second, it exists as the incomplete or masked virus which lacking its protein capsule is not antigenic; is not identifiable by electron microscopy; and cannot be detected by immunofluorescent methods. Current theory holds that the papilloma virus, a DNA virus, enters the tissue cells, loses its protein capsule and nullifies the genetic instructions, replacing them with its own which results in neoplastic replication. At some point after cell division ceases, the DNA regains its protein capsule and becomes a complete infective viral particle. It is apparent from these studies that the laryngeal papilloma virus exists primarily in this masked form.

The demonstration of intranuclear fluorescence in this study is further evidence that a viral antigen is present in the epithelium of laryngeal papillomas. The fact that the application of patient serum to the tissues intensifies the reaction is also evidence of circulating antibodies to the viral antigen. Specificity is suggested by the blocking technique. The failure of blocking of staining in one specimen does not negate this. The blocking technique is subject to certain difficulties as outlined by Nairn.¹¹ The antigen-antibody complex may not be entirely stable and the blocking (un-tagged) antibody may be partly replaced by the fluorescent tagged antibody during overstaining.

The random staining of nuclei in the epithelium of the papilloma would tend to negate the possibility of a nonspecific antinuclear antibody (ANA). If ANA were present, one would expect most of the nuclei to show fluorescence, if not all. The random staining of nuclei would also help account for the difficulty of finding intranuclear viral particles with electron microscopy. The difficulty of sampling is obvious when only one in 100 or more nuclei contains enough viral antigen to be detectable.

The results of these laboratory studies add supportive, but as yet not conclusive evidence of the role of a virus in laryngeal papillomas. In order to be conclusive, the virus must be isolated in the laboratory in tissue culture. It must be shown to be a filterable agent, and then transplanted into an animal model or into a human volunteer and be shown to cause the same type lesions as in the original host. At this time, six laryngeal papillomas are being studied in tissue culture. No agent has as yet been identified and, therefore, these are not included in this report.

The small amount of virus concentration as evidenced in these studies probably accounts for the variable response

to vaccine therapy in this disease. If the tissue used for vaccine production has an inadequate viral antigen content, then an effective neutralizing antibody response could not be anticipated. The location of the infective virus particles in the superficial cells of the tumor would explain the ease of seeding following surgical manipulation. Cross antigenicity of the papilloma virus does not necessarily occur, and this might account for the many failures associated with the use of bovine vaccine. The tumor regressions previously reported by the authors in surgical treatment of this disease in children may be attributed to the stimulation of immune response.² Frequent careful surgical endoscopic removal of the papilloma lesions with the aid of the operating microscope not only reduced the tumor population, but also allowed circulating neutralizing antibodies to be more effective on the residual virus population. Each papilloma lesion has the potential to perpetuate the entire disease process. The surgical avulsion of the tumor would result in the release of virus particles into the systemic circulation and, therefore, stimulate further antibody production. It is apparent that the final solution to this disease has not been achieved. However, until such time as an effective vaccine or antiviral agent can be developed, we feel that the treatment of choice is careful surgical excision of the papilloma lesions, frequently repeated.

SUMMARY

Seven laryngeal papilloma lesions from four patients have been studied by electron microscopy and immunofluorescence techniques. Intranuclear viral-like particles have been identified by electron microscopy. Immunofluorescence techniques suggest the presence of intranuclear viral antigens. Conclusions have been drawn as to the nature of the virus and a therapeutic approach proposed.

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DISCUSSION

DR. BLAIR W. FEARON: I should like to compliment the authors on their very fine presentation and I have nothing to criticize in their work. In fact, I was most fascinated.

I should only like to say with regard to papilloma of the larynx that tracheotomy should be avoided at all costs. I have not done tracheotomies in any of our patients, although we've had many worries from not. The only ones that have gone on to very severe seeding are those that have had tracheotomies or traumatic bronchoscopies.

Another interesting case we had was a nine year old boy who had been a heavy smoker for two years who had a massive recurrence in four weeks. He was not convinced that he had to stop smoking. The next four weeks when we examined his larynx there was no recurrence and he had not been smoking. A year later he had a small recurrence and it was evident he had been sneaking some cigarettes. This is the first time that I have heard of smoking probably related to papilloma of the larynx and throat.

DR. FRANCIS L. WELLS: I would like to ask the essayists a question. It's believed that a vaccine against any viral agent merely produces a receptor on the cell surface to prevent the viral agent from getting into the cell.

Have they tried the only agent that has been known to be effective against any viral agent, Interferon? It is believed by Isaacs who discovered this, I think in 1957, that you can take influenza vaccine, not sterilized in formalin but by heat or ultraviolet light, and this will produce at will the Interferon itself.

Have they tried doing anything on this principle?

DR. WILLIAM F. BOYLE (closing): I do not think we have the answer to your last point, Dr. Wells.

I would like to thank the society on behalf of the authors for the privilege of being allowed to present this paper here today.

ADENOCARCINOMA OF ETHMOIDS IN FURNITURE WORKERS

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OXFORD, ENGLAND

Adenocarcinoma of the ethmoids is a rare disease. In the United Kingdom the crude annual incidence in males aged 15 to 64 years is .0006 per 1,000 of the population, or just over one-half per million. In the male workers of the furniture industry in High Wycombe and the contiguous area it is 0.7 per 1,000, or 700 per million.

We noted a possible correlation between the disease in 1964 and the occupation⁹ and a field study by Acheson showed that the correlation was statistically significant.¹ This has been documented by Acheson et al. in a series of papers¹⁻³ and the condition has been recognized by Government as a prescribed disease under the Industrial Diseases Acts. This means that sufferers and their families may seek increased benefits under Workmen's Compensation arrangements.

Since that time papers have come from Debois,⁴ of Belgium, and Gignoux and Bernard,⁵ of France, noting a similar incidence of adenocarcinoma in the furniture industry of their areas.

AREA UNDER SCRUTINY

High Wycombe is situated in a fold of the Chiltern Hills on the ancient main route between London and Oxford, being rather nearer to Oxford. These hills form a natural watershed between the plain around London and that around Aylesbury. They have been heavily wooded since time immemorial, and beech trees have predominated. A simple domestic furniture trade has been carried on for more than 200 years, so that when Sheraton and Hepplewhite

were making fine furniture for the gentry, Wycombe was making Windsor chairs and tables for the farmers, the cottagers and the servants' hall. Now 20% of all English furniture is made there, and 80% of English wooden chairs. There are about 120 factories in the area, and something like 4,000 people are employed. Since these men are specialists, only rarely do they change their type of job, and they tend to stay in the industry for most of their working lives.

WOODS AND DUST

Beech wood still has an important place, but most of it now comes from France and Belgium. Additionally, a number of other hard woods are imported from places as far afield as Africa and South America. Mahogany and afrormosia are particular examples.

Originally the work was done by hand in simple sheds, now the processes are highly mechanized and take place in large enclosed workshops. The machines used produce a great deal of very fine dust. The mechanization took place between 1920 and 1939, but extraction by exhaust ventilation was introduced only after World War II.

We have found that the adenocarcinoma occurs only among those men who are working, or have at some time worked, in conditions exposing them to this fine hard-wood dust. Workers in the sawmill jobs who are exposed to coarse dusts are not affected, nor are those who have worked exclusively in the upholstering and varnishing departments.

We have found with interest that, as is the case with bronchial carcinoma and

smoking, there may be a latent period between exposure to the irritant and incidence of the disease. We have a number of cases who have not, in fact, been exposed to the dust for years, but who were so exposed in the past. Perhaps the maximal incidence of the disease is with us at present, and perhaps it corresponds with the period of minimally efficient ventilation in the 1930's and 1940's. We may see a fall in the coming years, but we wish we could be satisfied that the dust is, in fact, controlled.

SITE OF THE DISEASE

It originates almost invariably in the anterior ethmoidal area and spreads only secondarily to the maxillary sinus. This is in sharp contrast with the more common squamous cancer, which predominantly originates in the maxillary.

SURVEY

Because so little was known about this disease of the ethmoids, one of us, (RGM), made a survey of the noses of the workers in the industry in the actual factories. This survey was financed partly by the local Society of Furniture Manufacturers, and partly by the trade's national body, with the enthusiastic cooperation of the Trades Unions. The objects of the exercise were: 1) to observe the appearances of the noses under working conditions; 2) to make an early diagnosis of frank malignancies; 3) to attempt to recognize premalignant states; and 4) to assess (if possible) the effect of extraneous factors such as smoking and snuff-taking.

From mid-June, 1969, to mid-June, 1970, a nasal examination was performed in 3,100 workers. An attempt was made to see all the workers in wood, but he opened the clinic to workers in other departments if they wished to come. Since July, 1970, he has been engaged in a follow-up survey but has restricted this to wood-workers, to those over 35 years of age, and to men who commenced work in the industry 15 years ago or more. An attempt is being made to trace and see those who have

left the industry by reason of retirement or change of job.⁶

RESULTS OF SURVEY

The noses of workers in the office, the maintenance department, and upholstery, were, on the whole, within limits of normality. The noses of a few old hands in upholstery showed a fine white dust, presumably of cotton or plastic fiber. The wood-workers very commonly showed a deposit of dust just where one would expect this in relation with Proetz's air-current maps, i.e., on the septum, and particularly at the anterior ends of the middle turbinates. Although biopsies of a small number of middle turbinates had shown an increase squamous metaplasia in this region, we thought that cytologic smears taken *in situ* might be interesting. These will be discussed later.

Only one frank malignancy was found in the first year's survey. There were, however, three other patients who were employed in factories not yet visited. These had reported to their family doctors and had been referred to a hospital in the usual way. The mere fact that the survey was proceeding, however, had obviously alerted both patients and doctors. The single case found presented no difficulty in clinical recognition, and was quickly confirmed by x-ray and biopsy.

The attempt to recognize a premalignant condition has been disappointing. We can say two things: 1) the workers exposed long-term to the dusts show an increased squamous metaplasia in the smears; and 2) although simple polyposis is common in these people, it does not seem to lead to malignant change.

Smoking does not appear to be an adjuvant factor. Our malignant cases smoked no more and no less than the population at large.

Snuff-taking is common among these people, because they are not allowed to smoke in the factories. One is up against the question of definition of what constitutes habitual snuff-taking. Some evidence was given in our earlier papers

TABLE I
MID-TURBINATE SMEARS: PILOT INVESTIGATION

	<i>Total</i>	<i>Normal</i>	<i>Squamous Metaplasia</i>	<i>Adeno- Carcinoma</i>	<i>Unsat- isfactory</i>
Workers	55	9	35	1	10
Controls	28	20	5	0	3
	83				

TABLE II
MID-TURBINATE SMEARS: SECOND SERIES

	<i>Total</i>	<i>Normal</i>	<i>Squamous Metaplasia</i>	<i>Adeno- Carcinoma</i>	<i>Unsat- isfactory</i>
Path. Group	4	2	2 (1)*	0	0
Dust Group	15	3	11 (3)	0	1
Snuff Group	22	5	16 (5)	0	1
Repeat Group	7	1	6 (3)	0	0
	48				

*Figures in parentheses indicate marked metaplasia

that snuffing was more common in the cases of adenocarcinoma than in the average run of workers. A series of smears has been taken from people who admit to snuffing at least once a week.

CYTOLOGIC SMEARS*

The earlier smears were transferred directly to slides and fixed at once, but some of these were unsatisfactory because of cellular damage. We now use an improved technique whereby the smear is taken on an ordinary throat swab, and the cotton end is transferred to a pot of fixative, where it is shaken. The fixative contains wax in solution, so that after centrifuging and drying on a slide, the cells are fixed in the wax. It is then stained.

We have taken sets of smears from: 1) noses showing some abnormality near the anterior end of the middle turbinates (the "pathological" group); 2) dust-laden noses; 3) established snufflers; 4)

re-smear series, i.e., from patients smeared 12 months previously; and 5) a control series from the noses of patients without nasal symptoms who were seen for other reasons in the ENT Department, usually ear cases.

In general, we found a minimal squamous metaplasia in the control series, although the patients were approximately in the same age group.

The "pathological group" showed considerable metaplasia but also usually many pus cells.

The "dust group" showed a great deal of metaplasia and occasional glandular elements.

The "snuff group" showed much the same metaplasia as in the non-snuff group.

The "re-smear group" showed no significant change from one year previously. Tables I and II epitomize the findings.

*We are indebted to Dr. Arthur Spriggs of Oxford for directing this side of the investigation, and for examining and reporting on the smears.

DISCUSSION

Our work with these people has shown that the common factor in all of them has been exposure to the fine dust of hard woods at some time. The men themselves blame the "foreign woods," because often these are aromatic and irritating. Since these woods are of comparatively recent introduction we are forced to conclude that the native beeches, elms and oaks, are more likely to be implicated. At present we have no evidence about what may be the carcinogen, nor in which wood it comes.

Snuff may be an adjuvant factor. It is, of course, largely powdered tobacco, but the interesting thing is that unburnt tobacco does not contain the coal tar derivative 3-4 Benzpyrene thought to be the villain in cigarette smoking. Interesting work has come from South Africa where the Bantu use a snuff made up of the ground and calcined root of aloes, tobacco and clay. This contains the 3-4 Benzpyrene and a high proportion of the snuffers develop squamous cancer of their sinuses.^{7,8}

We have already said that the disease we are studying originates in the anterior ethmoidal area and that these workers show squamous metaplasia of their mucosa. Why then is the disease an adenocarcinoma? As yet we can only guess and our thoughts are as follows: 1) the mucosa of the middle turbinate is richly supplied with mucous glands (whereas the antral mucosa is not). 2) The squamous metaplasia in the area permits the deposition of the fine dust. This is not carried away by ciliary action and possibly may get into the mucous glands in solid form or in solution. 3) Adenocarcinoma is a slowly-growing indolent condition which, we might guess, is a result of a long continued minor irritant.

OTHER INVESTIGATIONS UNDER CONSIDERATION

Attempts are being made to culture the tissue from the middle turbinate, with a view to subjecting the cultures

to the dusts. There are technical difficulties here.

Experimental animals may be subjected to an atmosphere of hard-wood dusts.

The biochemistry of the woods will be looked at. It is quite unknown at present.

We have recommended a survey of the efficiency of the extraction systems in use in the factories.

TREATMENT

This has been dealt with fully elsewhere. Two points, however, should be stressed — full radiotherapy and ruthless surgery are both needed. If there is any question about sacrificing the eye, there should be no doubt; it must be removed.

SUMMARY

1. Adenocarcinoma of the ethmoid has been established as an industrial hazard in the wood-workers in the furniture trade of the United Kingdom.

2. The workers are specialists and remain in their jobs for years.

3. Prerequisites seem to be hard woods and fine particles.

4. A survey of the noses of the workers in the factories has been instituted. One case has been picked up in the survey; no precancerous change has yet been identified.

5. Cytologic smears from the anterior end of the middle turbinate area (i.e., that of maximal deposition of dust), have shown a high proportion of squamous metaplasia.

6. Smoking almost certainly has no adjuvant effect. Snuffing is indeterminate.

7. A theory is advanced to account for the fact that the disease is an adenocarcinoma, whereas the changes in the smears show squamous metaplasia.

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DISCUSSION

DR. RONALD G. MACBETH (closing): A query which came to me just now was, "why don't they wear masks?"

The answer is, "Well, it's human nature." The masks are there in nearly all factories, but they just do not bother to wear them. Nor, do I really think these masks are much good. They are paper things. Finally, to some degree it is rather like protecting your ears

against noise in a factory. If you do that, you cannot hear what the foreman says. Now, if the foreman is wearing a mask and you are wearing a mask, you cannot always see and hear what he is saying. The noise in these factories is something else again and unless you can see the man's lips you will not be able to hear what he says. So, the masks are there — the "blighters" won't wear them!

TREATMENT OF ACUTE BLUNT LARYNGEAL INJURIES

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WILLIAM K. MILES, M.D.

ANN ARBOR, MICH.

The acute, closed, blunt or crushing type of laryngeal injury presents special problems in diagnosis and treatment. How does one ascertain the extent of damage? Which cases should be treated by open operation? Is closed reduction and internal splinting preferable? Would steroids be of value? These important questions are similar to the problems in blunt injuries of the chest or abdomen where the intact skin may hide severe, life-threatening injury or almost none at all.

At the University of Michigan, we have wrestled with this problem as have many other laryngologists. In fact, in recent years, the problem has become more important than ever, since, in our experience, the frequency of this injury is increasing.

As noted by Chadwick³ in 1960, case reports of acutely treated blunt laryngeal injuries have been slow to appear. Since his report of 18 cases, there have been excellent case reports⁶⁻⁸ of treatment of acute laryngeal injuries, but these include both open and closed types. Only two recent articles besides Chadwick's specifically refer to closed injury,^{2,11} but between them, they report only four cases. We have, therefore, decided to recover and analyze as many of our cases as we can. In addition, we undertook a research project to study aspects of the diagnosis and treatment of these injuries in the dog.

*A table listing details of the patients summarized in this article is available by writing to Nels R. Olson, M.D., University of Michigan Medical Center, Dept. of Otorhinolaryngology, Ann Arbor, Michigan 48104.

REPORT OF CASES

We have reviewed 25 cases which meet our criteria of blunt nonpenetrating injury involving the larynx and hyoid which were seen and treated early by laryngologists. All of these patients, except one, were seen and evaluated by one or both of the authors.* Treatment, however, was determined by the physician in charge when the patient came to the hospital and was highly individualized. The patients ranged from 6 to 67 years of age; there were 11 females and 15 males.

ANALYSIS OF CASES

Etiology. Automobile and truck accidents were responsible for 23 of the injuries. The other three resulted from fisticuffs, an industrial accident and a bicycle-car accident. The most common specific cause was striking the neck against the protruding edge of a hooded dashboard which occurred in 12 patients and which is the subject of a separate report.¹⁴ Only two were known to occur from striking the steering wheel.

Associated Injuries. The most common significant associated injury was facial bone fracture, occurring in seven patients. There were five patients with extremity fractures and five with cervical spine fractures. Four had closed head injuries, three rib fractures and two pneumothoraces (1 bilateral).

Diagnosis. The mainstay of diagnosis

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has been *indirect laryngoscopy*. Twelve of the patients who were treated conservatively had only this type of examination in addition to soft tissue x-rays of the neck or laminography in the A-P plane. Two patients treated conservatively had direct laryngoscopies, one of them, a six-year-old child who had undergone tracheotomy.

Of the 12 patients treated by operation, the decision was basically made on the basis of preoperative x-rays and indirect laryngoscopy in six, although direct laryngoscopy was usually carried out at the time of operative treatment. In four, direct laryngoscopy led to operative treatment and in one, the major diagnosis was made when emergent laryngotomy was performed. The twelfth patient was first seen after application of tongs for cervical traction and the larynx could not be examined, so open operation was undertaken primarily for diagnosis and treatment on the basis of initial symptoms and clinical findings other than laryngoscopy.

Careful review of the signs and symptoms in these patients did not show a high correlation between certain findings and the severity of injury or ultimate results, except that if the larynx was crushed so badly that an early urgent or emergent tracheotomy was needed the injury was usually more severe. This point has been made by other observers.^{2,6,10,11} Thus, the need for an early tracheotomy becomes important diagnostically. The other findings judged most significant were 1) bare cartilage in the lumen on direct or indirect laryngoscopy or at open operation, and 2) cricoid fracture.

The presence of cervical spine fracture made diagnosis difficult in one patient whose larynx could not be visualized. In four others, indirect laryngoscopy could be done and we feel the correct diagnosis was made in these patients.

Conservative Treatment. In patients who did not have open or closed reductions, the usual measures including bed-rest, voice rest, humidified air or oxygen,

and prophylactic antibiotics were usually used. Certain authors have promoted the use of steroids.^{6,10,11} Five of our closed injuries were given a course of steroids beginning soon after their injuries and they had very good results, but more experience will be required to further evaluate this approach. We plan to continue to use them.

Operative Treatment. Of the 26 patients presented, 12 had operative treatment. The forms of operative treatment were open reduction, suture of mucosa, suture of cartilage and splinting (3 cases); open reduction, suture of cartilage and splinting (1 case); open reduction without splinting (2 cases); open splinting without reduction or suture (4 cases); closed reduction and splinting (2 cases).

Splints used were of Portex, solid silicone, silicone sponge, Tygon, sponge covered by a finger cot, and Teflon®. Splint duration was generally long, averaging 14 weeks but varying from 2 to 27 weeks.

There are many fears connected with surgical therapy, especially the fear that opening the larynx may actually worsen the patient by disrupting normal mucosa and adding additional scars and cartilaginous disruption. This fear has been answered with the concept of closed reduction and splinting, a method we have used. This method does not allow for repair of the mucosa, an objection which has not been specifically answered in the literature.

One of the serious problems of therapy is timing. Many patients cannot be operated immediately because of other injuries. We have specifically listed the interval from injury to treatment so that comparison with results can be made. Our operated patients were all operated by the twelfth day except three who were operated at 24, 30 and 34 days. The latter were left in this study so that results in moderately delayed cases could be evaluated.

RESULTS

The main criterion of success was a

permanently satisfactory airway after the first or initial treatment effort, whether conservative or surgical.

Of the patients evaluated and treated by the twelfth day, all who did not require early tracheotomy were successes. Two patients in this group receiving early tracheotomies were failures. These both had operative treatment, one on day 10 (Case 4, open reduction, suture mucosa, suture cartilage and silicone sponge splint), the other on day 12 (Case 25, closed reduction, Teflon splint). Unfortunately, these patients still wear trach tubes 38 and 46 months after injury in spite of continued efforts at repair. Case 4 had a severe purulent infection postoperatively with the splint in place.

Of the three patients treated after 12 days, two were failures. Both received operative treatment (open placement of Portex splints), one at 24 and one at 34 days. This latter patient did not require tracheotomy initially but had to have one 20 days after injury. Both of these patients have been decannulated after secondary treatment.

Voice and airway results (except for those still needing tracheotomy) were graded on a plus system. There appeared to be no good correlation between voice and airway results and severity of injury or treatment.

DISCUSSION

Diagnosis. In the study of treatment of acute blunt laryngeal injuries, we are basically facing two problems; optimizing diagnosis and optimizing wound healing. The first, most important, and most difficult, is establishing a correct diagnosis. In order to clearly establish criteria for diagnosis, we would have to present a control series of cases who were left untreated and determine the results, an unacceptable procedure. In studying the reports of others and our own cases, certain diagnostic features appear to be important. The first is the need for early tracheotomy. The importance of this diagnostically has been stressed by McCoy and McAfee,¹⁰ Fitz-

Hugh et al.,⁶ Brandenberg,² Ogura and Powers,¹³ and others. We feel it should be stressed that if a patient has an emergent, life-saving tracheotomy, cricothyrotomy or laryngotomy after a blunt injury, the indication is usually present for open exploration and repair.

A second diagnosis of importance is cricoid fracture. Although only two of our patients had proven cricoid fractures, both required immediate tracheotomy, and one of them is in the failure group and still has not been decannulated.

A third important observation is the presence of bare cartilage in the lumen of the larynx. This indicates severe disruption and we feel is an important reason to strongly consider open treatment. We had six patients who showed bare cartilage on direct or indirect laryngoscopy. Of these, all had operative treatment, three were initial failures and two of them still wear tracheotomy tubes.

Other diagnostic findings such as edema, hematoma, mucosal laceration or bleeding, pharyngeal tears, hyoid fracture, arytenoid dislocation, palpable thyroid fracture or flattening, neck contusion, subcutaneous emphysema, hoarseness or aphasia, coughing and odynophagia or dysphagia did not correlate as well with severity and prognosis. Associated injuries, especially fracture of the cervical spine, also failed to correlate well with prognosis in this group.

Wound Healing. The second basic problem in management of blunt laryngeal injuries is facilitation of rapid, efficient, unimpeded wound healing, a goal of all who perform surgical treatment. In the crushed larynx, there are two basic requirements for success. The first is an intact, reasonably properly shaped skeletal framework to hold the air passage open, and the second is complete epithelial lining of adequate size and of reasonably normal shape. It may be argued that neuromuscular function and sensory innervation are also of great importance, but we have found, as have others, that the patients usually get along and are satisfied if their tracheotomy can be removed even if the voice

is hoarse or breathy. (All of our patients, whether decannulated or not, were able to eat without significant aspiration.) This is not to deny the importance of voice function in the results and we feel that when we have learned to regularly, dependably, and correctly diagnose and treat these injuries with early restoration of the airway, we can then move on to the detailed problem of restoring a good voice as part of the initial treatment as well.

As regards the problem of skeletal restoration, there can be no doubt that an early open operation with direct suture of cartilage into normal position is theoretically the best approach. We feel most laryngologists would agree if they felt they could be accurate in diagnosis. We have offered, we feel, important points to help make this crucial decision. In cases of doubt, there can be little lost by careful exploration in this day of well controlled surgery and anesthesia. It would be better to have several "negative explorations" than to commit one patient to multiple procedures and prolonged disability. The method of "ironing out" laryngeal fractures appears to us inadequate when the consequences of failure are so severe.

Splinting of the lumen to allow fibrous union between cartilage fragments and to hold the shape of the lumen is felt to be necessary by most people and we would agree. It appears reasonable to accept the proposition that firm splints may damage epithelium and should perhaps be avoided as noted by Harris et al.^{7,8}

The restoration of an epithelial lining in acute blunt injuries is a problem not extensively discussed in the literature. Except for Harris et al.,^{7,8} authors generally minimize grafting and prefer reepithelialization by mucosal suture or mucosal flaps, or even by allowing small areas to reepithelialize over a splint or mold in the lumen. Wound healing principles dictate certain disadvantages of the latter method. One is that migration of epithelial cells is adversely affected by trauma (in this case, from a splint)

since this leads to loss of cells, and a second is that a granulating wound continues to granulate, forming collagen which must later contract, until covered by an epithelium.⁹

These basic principles were understood by Arbuckle¹ and Figi⁴ who advocated repairing *chronic* stenosis by excising scar and grafting denuded areas with skin. Figi made the point that if there was a relatively firm skeleton, the tendency of the skin graft to contract would be less since the cartilages themselves acted like an external splint. They did not use internal splints. Surgical repair of chronic stenosis is somewhat like repairing acute injuries since, with excision of the scar, a new acute surgical wound has been created and the same principles of epithelial healing will apply in either case.

Negus,¹² in 1938, expanded on these principles and pointed out the importance of an intact skeleton. Figi⁵ did the same in 1947 and stated "A fundamental on which the success of many plastic procedures depends is the necessity of furnishing a lining membrane in repair or restoration of a body cavity." For repair of chronic stenosis, he advocated applying skin grafts over sponge which conforms to the lumen. His sponges were left in for ten days, followed by splinting, especially if there was lack of rigidity of the cartilages. If he used splints after grafting, they were of sponge covered by a finger cot in order to avoid pressure necrosis. He summarized that splinting after removal of the sponge in ten days depended on the cartilages. If they were good, no splinting was probably required.

In more recent literature, Harris and Ainsworth,⁷ and Ogura and Powers¹³ have promoted these principles for treatment of acute injuries. Ogura reported three acute cases with excellent results and mentions the use of mucosa grafts taken from the epiglottis but does not specify that it was used in his cases. Harris relied on open operation to reduce fractures and repair lacerated membranes in all severe acute cases. When

mucosal closure was tenuous or inadequate, he applied a skin graft. Splints were never left in beyond 24 days. He had four patients in whom initial treatment failed (3 of whom were later salvaged) as did we, but his successful patients obviously had faster, more efficient healing and less morbidity than did ours, who generally had longer splinting times, using firm splints. Harris makes the important observation that if a splinted larynx becomes infected, including cellulitis of surrounding soft tissues, the results are bad. We have had the same experience. If splints are used after mucosal suture or skin grafting, and if the skeleton is firmly healed, the rationale appears to us to be to prevent or minimize graft or suture line contracture.

Grafting of the larynx with mucosa, as mentioned by Ogura, appears theoretically preferable to skin. Little is known about mucosa grafts in the larynx from either the experimental or clinical standpoint. We feel that this method should be studied further.

Splinting, we feel, may serve two purposes. The first is to provide support for loose cartilage fragments or to support a lumen when cartilage fragments are missing. This indication should not require prolonged splinting unless, perhaps, there was a large skeletal defect. The second rationale for splinting would logically be to prevent contracture of suture lines or grafts placed within the lumen, especially if the skeletal restoration is tenuous or skeletal parts are missing. This indication would require longer splint retention than the first since collagen shrinkage is slow. However, the comments of Figi⁵ are recalled, implying that when the skeleton is firm, it may

serve as an external splint to prevent this contracture.

The use of steroids in these injuries is now being advocated. Their possible effects may be many, including reducing hematomas and edema in the early post-injury period, preventing granulation until definitive repair can be done, reducing fibrosis in the healing phase and minimizing neuromuscular dysfunction. No clinical studies prove their efficacy but further trials seem indicated keeping in mind their potential adverse effects systemically and locally.

SUMMARY

The most important *diagnostic* features of acute blunt laryngeal trauma that we have been able to define from our experience and that of others are 1) need for urgent or emergent tracheotomy; 2) bare cartilage visible in the lumen, and 3) cricoid fracture. If any of these are present, one should strongly consider open surgical treatment. Patients with none of these findings acutely may usually be treated conservatively.

Treatment of acute blunt laryngeal injuries is often nonsurgical or supportive. Without evidence of their efficacy, we have added steroids to the conservative program. Severe injuries needing reduction should be treated by open exposure of the cartilages for exact reduction and suture and, if necessary, direct closure of mucosa through a laryngotomy. If mucosa is absent or closure seems tenuous, a graft of skin or mucosa as part of the primary treatment should be considered. Splinting is still recommended in certain situations and should be done using methods involving the least possible interference with epithelial healing, and for as short a period as possible.

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ACUTE TREATMENT OF EXPERIMENTAL LARYNGEAL FRACTURES

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Automobile accident injuries are the outstanding cause of severe laryngeal fractures.^{3,4,6,7,11} The dashboard and steering wheel inflict blunt trauma to the larynx, producing acute airway obstruction with hemoptysis, subcutaneous emphysema, and voice impairment, although there are no data available regarding the incidence of laryngeal fractures in fatal automobile accidents. The signs and symptoms have been clearly elucidated in other articles,^{2,3,8,10} and are not the subject of this discussion; yet this injury may still go undiagnosed, particularly in the semicomatose patient, because external laceration is unusual, and such semicomatose patients often have other more obvious reasons for hemoptysis and airway distress. They have usually sustained greater laryngeal trauma than the more alert patient, and, therefore, represent the major group of severely comminuted laryngeal fractures, which unfortunately have the poorest prognosis even with treatment. Furthermore, this is just the patient needing earliest possible treatment that usually gets a tracheostomy to maintain his airway, and no treatment to his undiagnosed laryngeal fracture. It goes undiagnosed for two to three weeks while other injuries are being treated, and then inability to extubate the patient occurs, calling attention to the laryngeal injury as a late diagnosis or laryngeal stenosis.¹

When early diagnosis and treatment are established, treatment of laryngeal contusion or laceration is usually adequate.^{2-4,6} However, the severe, comminuted fractures of the thyroid and cricoid cartilages remain the greatest challenge for acute therapy. Objectives
*Remington Stunner®

of treatment in these severely fractured larynges are primarily to obtain an "acceptable airway" and secondarily to preserve "vocal function." Only Fitz-Hugh et al.,^{3,4} Harris et al.,^{5,6} and Chadwick² report significant numbers of laryngeal fractures treated acutely, and even with their experience, numbers of really severe, comminuted fractures are small. Furthermore, there is nothing in the literature regarding animal experimentation to refine our methods of treating the comminuted larynx.

This report is of a pilot study to develop research methods in treatment of the severely comminuted larynx. Laryngeal contusions and lacerations are not the subject of this study, nor are mild fractures. In acute treatment of laryngeal fractures, three variables widely disputed in the literature are *duration* of splinting (short term vs. long term), *open* — operative vs. *closed* — laryngoscopic reduction, and whether there might be any efficacy of *steroids* in management. Trends regarding these variables are explored in this study.

METHODS AND MATERIAL

Permanent, tubeless tracheostomies designed by the author (WKM)⁹ were performed on 47 dogs (Fig. 1). Twenty-eight dogs with severe laryngeal fractures survived injury, treatment, and a four month follow-up period. Fractures were produced under general anesthesia with a high velocity, blunt impact, powered by a 22 caliber blank cartridge mechanism*. Impact was centered over the thyroid and cricoid cartilages and designed to produce severe fracture in the glottic and subglottic regions. All 28 animals received antibiotics. There were four major groups of animals (Table I); the first consisted of four



Fig. 1. Open reduction postoperative appearance with stay sutures from the intralaryngeal splint tied over buttons above the tracheal stoma.

fracture controls, two of which received steroids and none were splinted. Group II consisted of three week splint duration and was divided into groups of four dogs treated with open reduction, and four dogs treated with closed reduction. Group III was the same as Group II, except all were given steroids from the time of fracture until after splint removal. Group IV was the same as Group II, except splint duration was nine weeks.

All of the animals were carefully selected adult mongrels which had been quarantined and approved by the veterinary department for chronic experimental study. Calcification on microscopic sections of the canine larynx was seen to be of similar degree to that of the human larynx, and this was also studied by x-ray of canine larynges. The structural nature of the canine larynx is very similar to

*Dow-Corning No. 382 Silastic®

that of humans, although the canine laryngeal cartilages have somewhat greater flexibility.

Since edema and hematoma formation are rapid,^{2,3} a three to five day period was allowed postfracture for tissue reaction to subside before endoscopy and treatment. This waiting period, which Fitz-Hugh et al. proposed,^{3,4} parallels the management of facial fractures in which edema also is rather rapid and interferes with accurate surgery. Furthermore, we felt the endoscopic and operative evaluation would be enhanced by allowing edema to subside, and that installation of a splint onto already swollen, recently traumatized mucosa might be detrimental. Furthermore, this time interval parallels in reality the time gap often seen before treatment in the critically injured patient with a comminuted larynx among other multiple injuries.

Antibiotics used in the study included routine use of 1.2 million units of all purpose Bicillin®, given at the time of tracheostomy and injury, as well as at the time of treatment, and weekly during the maintenance of a laryngeal splint. Further specific antibiotic was used as needed for specific organisms cultured if gross infection occurred. While awaiting antibiotic sensitivity report, Chloromycetin® was used in these grossly infected cases.

Prednisone orally or prednisolone intramuscularly, in an average dose of 1.5 mg per kg per day, was administered to the steroid treated cases from the time of injury through the first two weeks after injury, then slowly tapered to .35 mg per kg per day by the second week after splinting. This dose was then maintained through one to three weeks following splint removal, with a rapid taper in the final week.

Splints were made from medical grade silicone* in varying sizes. Canine cadaver larynx imprints of varying sizes were used to make stone molds into which the silicone could be formed with a No. 1 nylon suture implanted in the splint. The suture formed a loop extending from the top and bottom of the splint anteriorly. Some splints were made extra long to accommodate upper tracheal injury. The splints were smoothed with an abrasive dental wheel. In all cases they were held in position by heavy nylon sutures passed from the loops on the splint, and out through

TABLE I

			Fracture Severity*	Airway Result**
Group I Fracture Controls (No splinting)	A. W/steroids	1	MS	—
		2	MS	—
	B. W/O steroids	1	MS	—
		2	M	+
Group II 3 week splint W/O Steroids	A. Open red.	1	MS	+
		2	VS	—
		3	VS	+
		4	VS	+
	B. Closed red.	1	M	+
		2	MS	—
		3	VS	—
		4	MS	—
Group III 3 week splint W/Steroid	A. Open red.	1	MS	+
		2	MS	+
		3	MS	+
		4	MS	+
	B. Closed red.	1	MS	+
		2	MS	+
		3	M	+
		4	M	+
Group IV 9 week splint W/O Steroids	A. Open red.	1	MS	+
		2	MS	+
		3	MS	+
		4	VS	—
	B. Closed red.	1	M	+
		2	MS	+
		3	MS	+
		4	MS	—

*M: Mild; MS: Mod. Severe; VS: Very Severe

**Acceptable = +; Unacceptable = —

the petiolus region of the epiglottis and trachea. The stay sutures were then tied over buttons on the midline anterior neck skin (Fig. 1), thus holding the splint well into the anterior commissure and insuring that it would not ride up or down significantly.

An epithelial lining graft, as advocated by Harris et al.^{5,6} and condoned in principle by the authors, was not used in this study, thus avoiding another variable, except in one animal with an extensive tracheal tear and avulsed mucosa. A buccal mucosa graft was used in that instance and a perfect result was obtained with the graft being indistinguishable from adjacent mucosa.

Open reduction was performed through a horizontal skin incision over the larynx (Fig. 1). The strap muscles were then split in the

midline and a thyrotomy performed as necessary for suturing of mucosal lacerations with fine catgut before installing a splint. Approximation of all cartilages with nonabsorbable suture (Tevdek 4-0) was employed. The closed reductions consisted simply of passing the stay sutures through the anterior neck skin with a long curved needle, then attaching the sutures to the splint extraorally before laryngoscopic introduction of the splint into the larynx. Splint removal was performed laryngoscopically in all cases treated.

Color photography was performed laryngoscopically on all cases at the time of injury, treatment, and splint removal, as well as at the time of sacrifice. Data was gathered at each laryngoscopy without knowledge of type of prior treatment. All animals except four

had a follow-up for four months. Those four animals expired in the interim period and were included in the analysis of results because all four of them had "poor" results which were, therefore, not expected to improve with more follow-up time.

Larynges were removed from all animals at autopsy and examined, then prepared for microscopic sectioning. Review of microscopic data was undertaken independently by the authors in a random fashion without knowledge of type of treatment. Correlation of microscopic data gathered in this manner was extremely close to data gathered by gross endoscopic evaluation; thus, the results in the following section have been derived from collated "gross" endoscopic data and "microscopic" postmortem data.

SEVERITY OF INJURY

There was some variability in degree of fracture, as would be expected; however, all but five of the fractures were of the severe, comminuted type. These five cases were frank laryngeal cartilage fractures, but of mild degree, and were, therefore, excluded in tabulation of results, as the study is confined to only the severe laryngeal comminution (Table I). Definition of terms regarding severity of fracture is as follows:

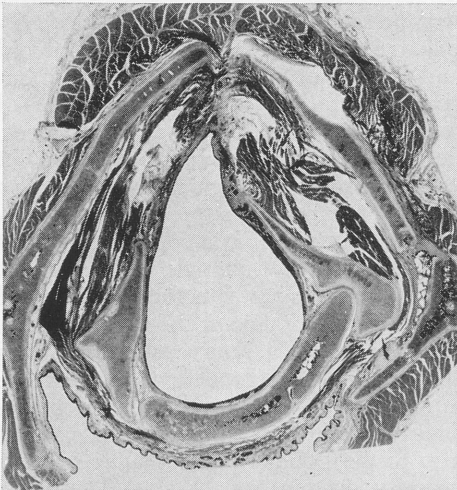


Fig. 2. An example of an experimental mild fracture, with a good result four months posttreatment with closed reduction and nine week splinting without steroids.



Fig. 3. Five days postfracture endoscopy showing subglottic cricoid fragments occluding the lumen, along with gross disorganization and tearing of the right arytenoid and vocal cord. This is a moderately severe fracture which attained an acceptable result by closed reduction for three weeks splint plus steroids.

"MILD" FRACTURE (Fig. 2).

A. More than just a contusion; mild distortion of airway clinically, with or without mucosal tears but with no fragments in the lumen nor exposed cartilage (yet all had fracture lines on postmortem microscopic examination).

B. May have involvement of more than one region.

"MODERATELY SEVERE" FRACTURES (Figs. 3, 4, 6, 8, 9 and 10).

A. Mucosal tears, with or without cartilage fragments in the lumen, plus mild to moderate degree of lumen compromise.

B. Severe involvement of one region of the larynx (such as subglottic) or mild to moderate involvement of more

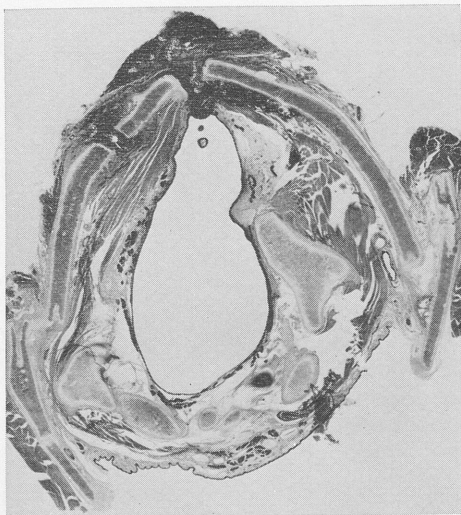


Fig. 4. An example of a moderately severe fracture with arytenoid fracture-dislocation and bilateral posterior alar fracture-dislocations, yet an acceptable (good) result was obtained as seen here four months after open reduction and three weeks splinting plus steroids.

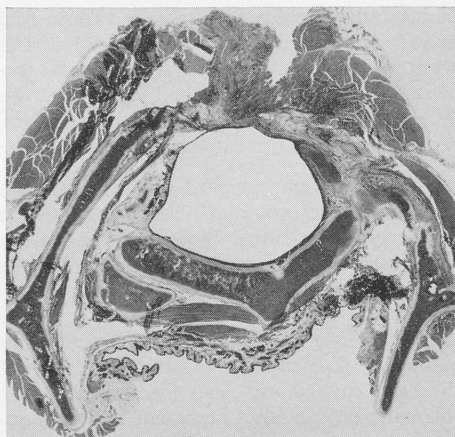


Fig. 5. An example of a very severe fracture with resorption of multiple right alar fragments. This fair airway seen four months after open reduction and three weeks splinting without steroids, is a result at the low end of the acceptable spectrum.

than one region of the larynx (such as subglottic plus glottic).

"VERY SEVERE" FRACTURES (Fig. 5).

A. Gross disorganization with cartilage fragments in the subglottic lumen, which is severely compromised, plus marked decrease in anterior-posterior diameter of the glottis, and the cricothyroid membrane is dehiscant; also, often with distortion of other structures such as vocal cords, arytenoids, or epiglottis.

B. Severe involvement of more than one region of the larynx.

All cases, including the "mild" ones, had glottic plus subglottic injuries involving microscopically proven fractures of the thyroid and cricoid cartilages plus arytenoid injury of some degree. Of the 23 "moderately severe" and "very severe" fractures, all cases had greater than two cricoid fracture lines and there were only eight cases with just three cricoid fracture lines. The greatest number of cricoid fracture lines was six and this was present in four of the 23 cases. It should

also be noted that even in the "mild" fractures there were at least two cricoid fracture lines. All cases in the series had at least one thyroid fracture line. Of the 23 "moderately severe" to "severe" cases, 15 of the 23 had greater than two thyroid fracture lines. The greatest number of thyroid fracture lines was five, and this was present in five of the 23 cases. Displacement of fracture lines of the cricoid and thyroid cartilages was taken into account in grading severity of injury. All cases in the series, even the "mild" ones, demonstrated microscopic evidence of some degree of arytenoid injury. X-rays were not carried out to delineate presence of cervical spine fractures, but there were a few animals with transient loss of limb motor function and several cases became apneic at the time of injury under anesthesia, but responded to a short period of controlled ventilation. The apneic occurrence may be related to the Innovar® and Pentothal® anesthesia, but also to peripontine hemorrhages seen on autopsy examination of animals studied during the development of fracture methods.

CRITERIA FOR STUDY

Among the 23 remaining cases, after

excluding the five mild fractures, there remained some variability in fracture degree which was taken into account in evaluating the efficacy of treatment. These 23 cases were, however, all in comminuted larynges. Emphasis in final evaluation of treatment was placed on the primary goal of obtaining an "acceptable airway." Evaluation of phonation was not possible due to the large permanent tracheal stoma in these animals, although a few of them were able to phonate in spite of it. Examples of the range of "acceptable airways" are shown in Figures 4, 5, 9, 10 and 11 as "fair" to "good" lumen area results, and do not necessarily imply perfect cartilaginous fragment reduction. "Unacceptable" air-

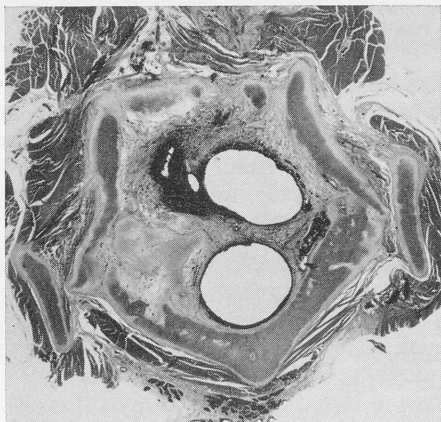


Fig. 6. An example of a moderately severe fracture with an unacceptable (poor) result due to cricothyroid membrane fibrosis, as seen four months after open reduction and nine weeks splinting without steroids.

ways were frankly stenotic (Fig. 7), or those classed "poor" (Figs. 6 and 8).

RESULTS

Control Animals. Three of the four unsplinted control animals had severe injuries and all three developed severe laryngeal stenosis (Fig. 7), in spite of two of them being given steroids. The fourth control animal had a good airway but was excluded because his fracture was only of mild degree.

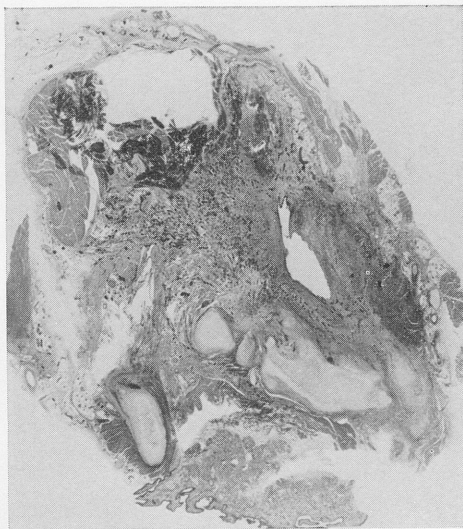


Fig. 7. Control larynx with a moderately severe fracture treated only with steroids acquired cicatricial subglottic stenosis, two and one-half months after fracture.

Steroid Treatment. Steroid therapy in Group II versus Group III (both with only three week splinting duration), clearly favored the steroid treated group, although numbers were small. Only three of seven cases not given steroids developed acceptable airways, whereas six of six given steroids were acceptable. Furthermore, careful examination of results by degree of injury and open vs. closed reduction, showed no skewing of the two groups with regard to these two other variables. Data gathered by gross observations at long term follow-up correlated perfectly with postmortem microscopic data for these two groups. Gross observations at the time of open reduction operations showed markedly less granulation tissue in steroid treated cases, but this was not conclusively delineated by the amount of submucosal and fracture line scarring seen on long term follow-up postmortem microscopic sections.

Open Vs. Closed Reduction. Acceptable airways were achieved in five of six cases of open reduction vs. two of five cases of closed reduction. These results are on three week splinting durations and include only the very severe in-

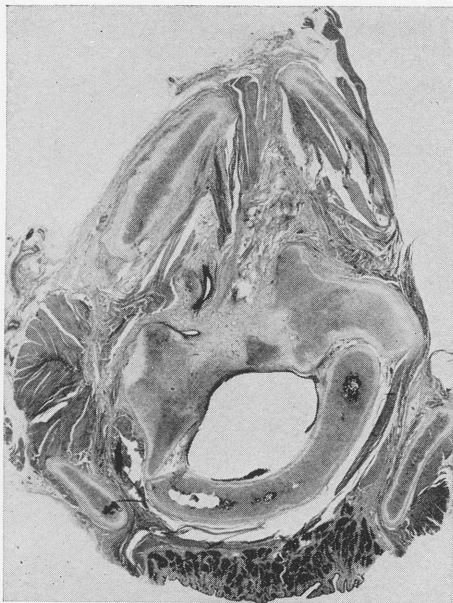


Fig. 8. Moderately severe fracture seen four months after closed reduction and nine weeks splinting without steroids, shows dislocated arytenoids which are fused producing an unacceptable (poor) result.

juries. Inclusion of data from nine weeks splinting improves the closed reduction results only slightly, and the open reduction results not at all. The results with closed reduction, however, might be better if more had been treated with steroids. If moderately severe and mild cases are included, the open reduction results remain unchanged (10 of 12 acceptable), whereas closed reduction results improve (6 of 10 acceptable); yet open reduction would still remain slightly better as a trend.

Duration of Splinting. With regard to duration of splinting, comparing the groups splinted nine weeks vs. those splinted three weeks, all without steroids, slightly favors longer term splinting. Slight skewing of these results, however, may have occurred in that the cases splinted three weeks had slightly greater severity of injury. Acceptable airways were obtained in three of seven cases splinted three weeks and in five of seven splinted nine weeks. The num-

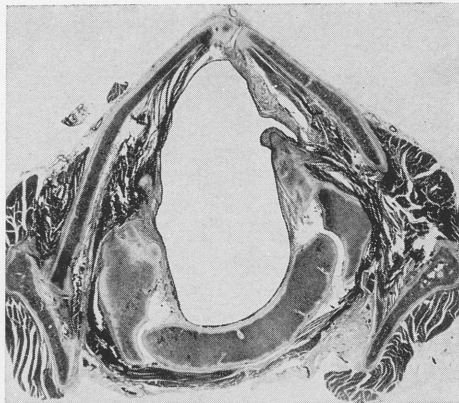


Fig. 9. This moderately severe fracture attained an acceptable (good) result demonstrating arytenoid resorption without ankylosis four months after closed reduction, with three weeks splinting and steroids.

bers of closed and open reductions were comparable in the two groups. Thus, perhaps no significant trend can be ascertained in these small groups with skewing of degree of injury to slightly favor long term splinting.

Other Observations. With regard to waiting three to five days postinjury for final endoscopic evaluation before treatment, 26 cases with complete data from endoscopy at time of fracture, as well as three to five days later, showed that over half of them were misdiagnosed at the initial fracture period endoscopy. At endoscopy performed three to five days after injury (Fig. 3), seven cases were found to be more severe than previously recognized, and eight cases found to be less severe than previously determined. Eleven cases had no change in diagnosis. Furthermore, findings at open reduction were relatively consistent with the endoscopy performed just prior to open reduction.

As Chadwick² and Fitz-Hugh et al.³ have stated, *hematoma formation* and *edema* in the loose tissue of the larynx and hypopharynx occur rapidly after fracture. This was confirmed in our early postfracture endoscopy and is probably maximal by six hours postinjury.

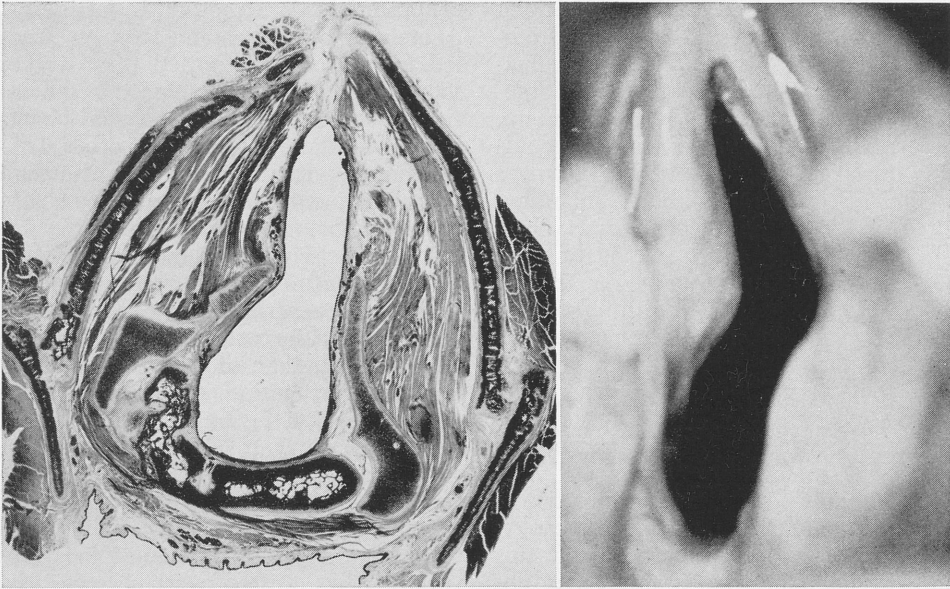


Fig. 10. This moderately severe fracture attained an acceptable (good-fair) result as seen four months after open reduction with three weeks splinting and steroids, despite having scratched his operative wound open, producing a severe neck infection. Note the left arytenoid fracture dislocation and ankylosis with resorption, and also the bilateral posterior thyroid alar margin fracture dislocation.

Fig. 11. This is the same larynx as Fig. 10 at four months follow-up, demonstrating an acceptable (good-fair) result in spite of residual arytenoid obstruction.

With regard to *infection*, 20 of the 28 cases had purulent secretion from the tracheal stoma and six cases had soft tissue neck infection. Stomal purulence was treated with appropriate antibiotics and usually subsided after splint removal. All of the cases given steroids had infection except one of the control animals on steroids. However, only two of the six cases of soft tissue neck infection were on steroids. Only two cases had wound dehiscence, which in both instances was from scratching the wound open, and only one of those animals was on steroids (Fig. 10). Interestingly enough, infection did not skew airway lumen results. Three-ninths of the unacceptable results were not infected and 14 of the 19 acceptable results were infected. Only two of the six with neck soft tissue infection had unacceptable results. On microscopic postmortem examination, five of the six animals with neck infections had heavy fibrosis in the submucosal and fracture line sites. The one animal

without heavy fibrosis had received steroids (Fig. 10). All cases in the series had some degree of fibrosis on microscopic examination. There were eight uninfected animals in the whole series, and two of them had heavy fibrosis as did eight of the remaining 20 animals without infection. Thus, no significant difference in amount of microscopic fibrosis could be found with relation to presence or absence of tracheal/stoma purulence, except in those cases with soft tissue neck infection.

Arytenoid injury, unless with gross dislocation (Figs. 10 and 11), was not well appreciated endoscopically except for lacerating fractures through the vocal process (Fig. 3). However, on postmortem microscopic examination such fractures at the vocal process were seen in a majority of cases (Figs. 4, 9 and 10), as well as frequent occurrence of cricoarytenoid joint ankylosis (Figs. 2 and 10), which might be assumed secondary

to some degree of disruption of the joint by trauma. These results did indeed correlate with the gross endoscopic evaluations of cord immobility prior to sacrifice at the end of long term follow-up. Resorption of all or part of arytenoids was observed in many of the larynges at microscopic evaluation, including three of the four control animals (Fig. 7). Resorption was also present to some degree in a vast majority of the treated cases (Figs. 6, 8 and 9), with no greater proportion in the group with long term splinting than in the comparable group with short term splinting. Furthermore, the degree of arytenoid resorption was not clearly correlated to the presence or absence of infection. Further work with regard to splint pressure and infection is being evaluated. Regardless of etiology, when arytenoid resorption occurred, this "auto-arytenoidectomy" may have enhanced laryngeal airway results in some cases (Fig. 9). Microscopically, there was suggestion of arytenoid injury even in the mild cases of fracture (Fig. 2), all of which developed acceptable airways. Status of the *recurrent laryngeal nerves* was not adequately studied, as special stains have not yet been performed.

Other factors affecting the lumen were observed. In the entire series of 28 animals, fracture of the thyroid ala posteriorly (Figs. 2, 4, 5, 6, 9 and 10) was present bilaterally in 12 cases and unilaterally in nine cases. All 28 cases had some degree of midline thyroid cartilage fracture also. However, endoscopic findings of anterior-posterior glottic diameter narrowing and site of arytenoid injury did not correlate as well as would be expected with either midline or posterior alar fracture sites. Perhaps decreased glottic diameter viewed endoscopically before treatment was related to soft tissue changes. Nevertheless, in some microscopic sections, a bilateral dislocated fracture of the alae posteriorly could be envisioned allowing posterior subluxation of the thyroid cartilage over the cricoid cartilage, to narrow the anterior-posterior glottic diameter and produce a tortuous sagittal airway. Certainly the frequency

of posterior alar fracture is intriguing. Acutely fractured canine larynges studied in previous unreported pilot studies showed fracture dislocations of the inferior cornua and cricothyroid joints present in almost all cases. Another striking factor with regard to lumen result from treatment, was the amount of *submucosal fibrosis* particularly at the cricoid and cricothyroid membrane level (Figs. 6 and 7). This phenomenon of soft tissue scarring several times appeared more important in affecting the airway result than did the lack of careful reapproximation of cartilage fragments (Figs. 4, 5, 9 and 10).

DISCUSSION

This study has shown feasibility of adapting dogs to the study of laryngeal fracture therapy. The multiplicity of variables has created overlap among the treated groups, which were admittedly small in this pilot study, and exhibited variable degrees of injury, precluding any statistically significant conclusions. Nevertheless, judgments of results were made in light of degree of injury and certain gross observations and trends were observed. Extrapolation of these trends to humans might be possible in certain regards, especially in light of our human clinical experiences.

We must concur with Fitz-Hugh et al.³ that optimum laryngologic assessment is not attainable in the severely fractured larynx immediately after injury, and that better assessment is attained after allowing edema and hematoma to subside for three to five days. We can also confirm that intralaryngeal hematoma and edema are extensive within the first hour after injury, and probably maximum by six hours after injury. Thus, if airway compromise in laryngeal injuries has not occurred by that time, it will likely not occur unless the patient is coughing or speaking into a laryngeal laceration enough to produce progressive subcutaneous emphysema. Likewise, if tracheostomy is not needed, it is probably not a laryngeal fracture of the severely comminuted type. The very severe fractures have cartilage frag-

ments in the subglottic lumen at endoscopy (Fig. 3).

The effect of steroids in preventing granulation tissue formation during this three to five day period before final evaluation and treatment, was grossly impressive in the open reduction cases receiving steroids, as opposed to those that did not receive steroids. In the latter cases, there was extensive heap-up, friable granulation tissue which bled profusely and was impossible to suture through satisfactorily. Bennett¹ has observed this phenomenon also in humans. Thus, steroid administration would seem especially efficacious in facilitating open reduction surgery done over 24 hours after injury. Thus, if one is going to wait three to five days postinjury to do definitive endoscopy and for repair, steroids are indicated to inhibit heavy granulation in the wound. Experimentally, steroids started after 24 to 48 hours do not inhibit fibroblastic proliferation significantly. Antibiotics also seem advisable in this type of management of an open fracture.

The only clearly delineated trend in this pilot study was the correlation of acceptable results in all of the steroid treated animals as opposed to less than half of the comparable group not given steroids. No adverse effects regarding wound healing were observed with steroids, and it would seem advantageous to continue for at least two to three weeks postsplint removal, as such continuation correlated with less fibrosis submucosally and at the fracture lines in a few cases treated in that manner, as opposed to the steroids being stopped at the time of splint removal.

No clear-cut conclusions can be drawn regarding open reduction vs. closed reduction, although results with open reduction were slightly more favorable. With the severe morbidity of chronic laryngeal stenosis, open exploration would, therefore, be justifiable as a method of evaluating the thyroid and cricoid cartilages whenever there is doubt regarding the degree of injury. The data did not indicate how severe a

fracture has to be to cause laryngeal stenosis, but all of the mild fractures did obtain acceptable airways with treatment, and one of them was an untreated control. Therefore, in the mild injury, if it can accurately be ascertained, perhaps less aggressive therapy is appropriate. A decision whether or not to splint such mild fractures should probably be based upon presence of airway lumen distortion other than by edema or hematoma, and closed laryngoscopic introduction of splints in such cases is probably adequate. However, when there is any doubt, open reduction is preferable. Certainly fractures with cartilage fragments in the lumen or gross evidence of lumen obstruction, should have open reduction with suture of lacerations and reapproximation of cartilage fragments with nonabsorbable suture. The type of splint to be used remains somewhat debatable but should be relatively inert and soft.

We have not studied lining the larynx with an epithelial graft, but agree with Harris that this would seem very important in diminishing submucosal scarring from open granulating areas. Certainly we did see evidence that most posttreatment failures are secondary to cicatricial scarring rather than lack of careful cartilaginous reapproximation, which does not appear to be quite as significant in obtaining an acceptable airway. We might prefer a mucosal graft rather than split thickness skin on theoretical and limited clinical grounds for the epithelial graft.

In this study no significant trends can be ascertained with regard to duration of splinting in these small groups of animals with skewing of degree of injury slightly favoring long term (nine weeks) splinting. Harris et al's.^{5,6} good results in humans have been with short term (two to three weeks) splinting, but combined with the use of an epithelial lining graft, and we favor this concept. However, where there is loss of cartilage fragments resulting in an unsupported wall, slightly longer term splinting may actually be preferable. The purpose of splinting appears to be to maintain car-

tilage fragments in a favorable position while early fibrous unions are started in treatment of acute fractures.

Arytenoid injury of minor degree is difficult to ascertain endoscopically or to differentiate from recurrent laryngeal nerve trauma paralysis. Patterns of arytenoid injury and cartilage resorption, as well as other factors affecting the lumen such as inferior thyroid cornu fracture dislocation and subluxation of the thyroid cartilage onto the cricoid cartilage were discussed in the results. We

would concur with other authors that arytenoidectomy should not be performed initially, as vocal cord function from paralysis can return up to a year following injury. Furthermore, some resorption of obstructing arytenoid cartilage may occur during healing, as seen in our studies, thus improving the lumen by an "autoarytenoidectomy."

It is hopeful that this work will facilitate further animal experimentation and improve understanding of the treatment of these potentially severely debilitating injuries.

UNIVERSITY OF MICHIGAN, ANN ARBOR, MICHIGAN 48104 (NELS R. OLSON, M.D.)

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Request for reprints should be sent to the Dept. of Otolaryngology, University Hospital, Ann Arbor, Michigan.

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DISCUSSION

DR. SYLVESTER C. MISSAL: At our hospital we have seen two very severe cases of laryngeal injury which resulted in unilateral cord paralysis.

We elected to leave these cases alone rather than try any transposition operation because they seemed to do very well.

I should like to ask the authors if, in their clinical series, they saw any vocal cord paralysis, and, if so, how they handled it.

DR. THOMAS C. GALLOWAY: This is a very scholarly and finely worked out study. However, laryngeal injuries may be very simple in these cases. For instance, one case had a complete collar incision, a horizontal split of the cricoid and the trachea was pulled out of the chest. Simply by finding an intact half of the cricoid and suturing the lower half of that and doing a tracheotomy, this patient did very well, but he had a complication that I did not hear mentioned and that was the separation of the recurrent laryngeal nerve.

I wonder what the authors found out about that.

In a study of a hundred cases of neck injuries, including cut throats, we found that most of these did very well with a tracheotomy, but if the hypopharynx were involved, the infection was something to be very gravely concerned about, at least in the days before antibiotics.

This is a fine study and should save a lot of lives.

DR. CLAUDE LEE PENNINGTON, JR.: I would like to congratulate the two authors on their paper.

There was one exception in severe pathology that I have come in contact with over the past ten years in treating these injuries and this is the rupture of the pre-epiglottic ligament into the anterior glottic lumen. It is a supraglottic injury and it occurs in blunt trauma. It is probably the most common, and most commonly overlooked, injury that occurs, at least in my own experience.

Montgomery was the first to call attention to this pathology and pointed out that an Omega resection of the base of the epiglottis would eliminate stenosis. I add to that the excision of all the adipose tissue that is carried with the dislocated base of the epiglottis from the pre-epiglottic space. This space, if you will go back and review your anatomy books, is compartmentalized and when this severe injury occurs, it explodes right into the glottis and then the fat tissue turns into granulation tissue and along with the other denudation of epithelium in the anterior glottis, you get a marked amount of adhesion.

In two of the slides that were shown in the research paper, I believe I could detect a marked posterior displacement of the base of the epiglottis.

DR. HERBERT H. HARRIS: I think these are two of the most outstanding papers that I have listened to in years.

First of all, I may say it confirms my earlier suggestions and recommendations, so of course that makes it a good paper! But, I do want to congratulate the authors and I do not think that I should make any comments because I agree with them all down the line.

There is one thing that I might say. It is gratifying to know that some of their delayed reductions came out well. I have never had enough of that experience to know whether they would or not.

There is one question that I would like to ask. I have not had the opportunity to have used a splitthickness graft from the inner side of the cheek. It is my understanding that you can now get a disposable dermatome. How big a graft you can get, I'm not sure, but I wonder if this would not be superior to the

use of split-thickness, keratinizing skin which they have suggested.

Have they had any experience or has anyone had any experience?

DR. W. FRANKLIN KEIM: I think Dr. Miles and Dr. Olson are to be congratulated on a very beautifully presented pair of papers.

One of the problems that we have in the patients with severe blunt injury which is often in the multiple injury category, has been to persuade the members of the other disciplines, such as neurosurgery and orthopedics, that there is a problem in the larynx, or in the trachea.

They are so interested in the intracranial problem or the cervical spine problem with a patient on a Striker frame or in tongs that they are very reluctant to let us go ahead and do something to the larynx. On a couple of occasions, we have practically had "to hit them over the head" before they would let us go ahead with exploration.

I think one of the authors stated that if no tracheostomy was necessary within the first twenty-four hours, you could be sure that there was no gross displacement of the laryngeal cartilages. I would take exception to that. We have seen patients who have had rapidly developing obstruction, due to hemorrhage from one of the cricothyroid vessels who have required tracheotomy and who, on exploration, proved to have no actual laryngeal displacement or displacement of the laryngeal cartilages.

I should like to ask the authors whether when they are doing a tracheostomy it would not be a good idea just to take a look at these structures. It certainly seems to be a better idea to explore these patients if there is any question in your mind as to whether there is any gross displacement.

DR. NELS R. OLSON (closing): Thank you very much for your interest.

About vocal cord paralysis, we think it's hard to diagnose and do hope to follow up with more special histology on the specimens to see whether the nerve was injured.

We were able to show that cricoarytenoid ankylosis does occur in the animal.

The question of open injuries to the larynx, the stab wound in the neck and so forth, was not covered in our discussion, and we agree that they generally have excellent results because they require operative treatment in any case.

I appreciate Dr. Harris's comments. The more one looks at this, the more his rationale is obvious and worthwhile.

The use of mucosa grafts from the buccal area is possible. I tried to find out more

about mucosa grafts and went to Mustarde's book on ophthalmic plastic surgery, which is one of only two texts which discuss this technique in any detail.

Mucous membrane grafting is usually full thickness grafting, and if the mucous membrane is selected from some areas of the upper air and throat passages, there is very little submucosa and these grafts shrink as much as to one-third their original size.

Mustarde does reconstruct lower eyelids by taking nasal septal mucous membrane which has a good submucosa and even incorporating a piece of nasal septal cartilage into the graft, making a composite graft. He claims that he can take a piece of mucous membrane from the septum, measuring two by four centimeters, very easily without a significant effect on the nose, and that this graft is less likely to shrink as the buccal grafts do.

We did graft one of our dog larynges with a piece of mucosa taken from the gingivallabial sulcus and the graft took beautifully.

In the matter of questionable cases when no early tracheotomy is required, we feel ex-

ploration should still be carried out if there is evidence of cricoid fracture or if there is bare cartilage in the lumen.

I will let Dr. Miles answer Dr. Pennington about the pathology in the region of the base of the epiglottis.

We appreciate this opportunity to come here very much.

DR. WILLIAM K. MILES (closing): I also appreciate the opportunity and the kindness given to us.

I agree with Dr. Pennington about that observation. That went through my mind several times on reviewing these slides and we looked at that rather closely, although the injury in the supraglottic area was not as common as subglottically. The decision we finally came to on that was the concept of subluxation of the thyroid cartilage back over the cricoid giving a retro-displacement of the thyroid cartilage. To some extent this may be giving the same appearance.

However, I think that your point is well taken and perhaps our interpretation of that was not just fully correct.

Memorials

C. STEWART NASH, M.D.

(1893-1971)

It is easy to write that C. Stewart Nash, who died early in May, 1971, was an unusual physician. It is difficult, indeed, to put into words a proper and intelligent appraisal of this multi-faceted physician.

It should be noted at the outset that he was so popular and so well-liked that none of his colleagues called him by his proper name. He was just plain "Stew" to all. This was perhaps because he loved people and enjoyed meeting and talking to them. He certainly had more friends than most of us.

Stew chose Michigan as his Alma Mater. It was here that he elected otolaryngology to be his professional milieu. This brought him under the tutelage of Bishop Canfield, one of the great teachers of otolaryngology of another day. This set the stage for a notable career in his chosen field.

Stew exercised a high degree of self-discipline. Early in life, he adopted for himself two daily tasks. One was to do something which he never had done before and the other was to force himself to do something he did not like to do. It was probably because of this that Stew possessed an unusually well-organized mind which in turn permitted him to be a good teacher and an excellent administrator. While it might be in order to detail his many accomplishments as a successful practitioner of otolaryngology, it was as a teacher and an administrator that he was known best.

Pages might be written to detail his many honors and accomplishments, but only a few will be mentioned so that space will remain to write of his two main interests. Stew was a member of all the major otolaryngologic societies for which he was eligible. He served for a quarter of a century as an examiner of the American Board of Otolaryngology. He held many important posts in his own state, New York, and in his own city, Rochester.

Early in his career, Stew became involved in the question of hearing loss due to exposure to noise and, in particular, hearing loss due to industrial noise. His large clinical experience qualified him to be a leading figure in the national attempt, begun approximately 25 years ago, to ameliorate some of the problems inherent in this situation. His contributions were many and were highly significant.

However, it is as secretary of the Triological Society that Stew Nash is remembered best. Stew "ran" this Society for more than 25 years. His efforts to improve the organization were so significant that he was elevated to its presidency, but he continued as secretary of the Society during the year he was its chief presiding officer, something which never before or since has happened. He continued as secretary of the Triological until he reached the official retirement age. Up to the time of his passing, many years later, he never adjusted to this mandated change.

Stew became secretary when the Triological was a smallish group with little national purpose. Gradually his influence molded the Triological into a well-organized, cohesive company almost double the size it was when Stew took office. While his command of the ship was almost ironclad, his jovial spirit and ready wit softened the effect of his many "musts." No one who was privileged to sit in an annual meeting of the officers and directors of the Triological or who was a member of a nominating committee of the Society will ever forget his quiet but firm dominance in those sessions. Some youngsters in the last few years of his secretaryship objected

to his authoritarianism but those who knew him best, those who had watched over the years what his efforts had accomplished, supported him to the last. In these later years, he became known as "Mr. Triological," a sobriquet never given to any other person.

Mrs. Nash and Stew were blest with only one child. Their boy, now a grown man in his mid-forties, was born blind. Stew read aloud to this child every day that Stew was at home, although Donald, the son, became expert at reading Braille early in his life. This was a task which Stew relished and to it he contributed many hours of careful selection. Who knows whether his greatest contribution in life was as a physician or as a husband and father?

Stew Nash was a remarkable man.

G. D. H.

THEODORE EDWIN WALSH, M.D.

(1900-1971)

American Otolaryngology lost a stalwart in the death of Theodore Walsh in St. Louis on April 29, 1971.

Theo was born in Calcutta where his father was in the Indian Civil Service. He obtained a A.B. degree in 1921 from Cambridge University and his medical degree in 1925 from St. Thomas Hospital Medical School, London, England. After an internship in the Royal Hampshire County Hospital, Theo was appointed as a Medical Officer to the Sarawak Government in Borneo. In this role his medical activities were general and included a considerable experience in general surgery.

After nearly two and a half years in Borneo a return to England was planned by way of San Francisco to spend several months observing surgery in the United States. At the University of Chicago he met Dr. Douglas Phemister, Professor of Surgery, who persuaded Theo to accept an appointment as a resident in Otolaryngology, which was a sub-specialty of the Surgical Department under Dr. Phemister. About a month later Molly McNamara entered the picture. She was an attractive nurse who had trained at the University of Minnesota and on graduation accepted an appointment to the nursing staff at Billings Hospital of the University of Chicago. They were married while Theo was a resident. That cemented a plan for Theo to stay in the United States, and upon completion of his residency he was appointed to the faculty in Otolaryngology at Chicago.

The Otolaryngological world soon became acquainted with Theo through his research interest in immunization of the upper respiratory tract, in collaboration with Professor Paul R. Cannon of the Department of Pathology, and his publications on this, and on other subjects co-authored with Dr. John R. Lindsay.

In 1940 Dr. Walsh was lured to St. Louis as Head of the Department of Otolaryngology of Washington University and as a consultant to Barnes Hospital. His tenure there for almost two decades was to witness the development of the Department as one unsurpassed in any other institution in the matter of graduate training, research, etc.

In 1942 Dr. Walsh became Editor of the Laryngoscope, a position he held until his death and to which he brought editorial skill of a high order. He was also Editor of the Transactions of the Triological Society, an unenviable task which he accomplished superbly from 1946 to 1967.

Dr. Walsh was an outstanding member of numerous societies, among them The American Otological Society, The American Laryngological Association, The Triological Society, The Royal Society of Medicine, The American Academy of Ophthalmology and Otolaryngology and the American College of Surgeons. He was honored with the presidency of both the Otological and Triological Societies. He served on important training grant and research study sections in the National Institutes of Health.

Dr. Joseph Ogura, who had his residency training under him and succeeded him as Head of the Department of Otolaryngology at Washington University, recently stated:

"Dr. Walsh considered honesty and integrity to be the most important prin-



THEODORE EDWIN WALSH

ciples that he could teach his residents. By his own professional and personal life he set an unobtainable standard."

In his later years Dr. Walsh limited his clinical work and research largely to Otology, though he maintained a broad interest in the whole of Otolaryngology. His publications and participation in otological programs earned him a reputation as a scholar and independent thinker.

Theo had broad interests in addition to his medical activities. These included music and art of all types, and painting. He was an expert golfer.

Molly McNamara Walsh shared in these interests and was his constant companion in their leisure hours. She survives him with their son Peter and his wife and three grandchildren. Peter is a doctor of Medicine and is currently an NIH Fellow at Oxford in England doing research in hematology.

L. R. B.

EXECUTIVE SESSIONS
GENERAL SYMPOSIUM
SUBMITTING MATERIAL MAY 23, 1971
1971 IN THE SYMPOSIUM

The Executive Session of the Twenty-Second Annual Meeting of the American Psychological Association was held on May 23, 1971, at the Sheraton Hotel in Washington, D.C. The session was presided over by Dr. William D. Woodworth, President of the Association. The session was held in the morning and was attended by approximately 100 members of the Association. The session was held in the morning and was attended by approximately 100 members of the Association.

Executive Sessions

The Executive Session of the Twenty-Second Annual Meeting of the American Psychological Association was held on May 23, 1971, at the Sheraton Hotel in Washington, D.C. The session was presided over by Dr. William D. Woodworth, President of the Association. The session was held in the morning and was attended by approximately 100 members of the Association. The session was held in the morning and was attended by approximately 100 members of the Association.

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Honorary Fellow: Dr. Frederick T. Lill
Executive Fellow: Dr. A. J. Farrow
Dr. Eleanor D. Woodworth

Contributing Fellow: Dr. James C. Whitely
Dr. Andrew A. Kagan

Since the Annual Meeting we have received word of the death of C. Stewart Nash on May 1, 1971, and Theodore Walsh on April 28, 1971. Dr. Nash was elected an Active Fellow in 1967 and an Executive Fellow in 1970. Dr. Walsh was elected an Active Fellow in 1971.

The Distinguished Medal was presented to Dr. Francis E. Lanyon, New Orleans, Louisiana, at the Annual Meeting.

EXECUTIVE SESSIONS

SUNDAY MORNING, MAY 23, 1971

The Executive Session of the Ninety-Second Annual Meeting of the American Laryngological Association convened at eight-fifteen o'clock, Sunday morning, May 23, 1971, in Continental Parlors VI and VII of the San Francisco Hilton Hotel, San Francisco, California, Dr. DeGraaf Woodman, President of the Association, presiding.

PRESIDENT WOODMAN: It is my pleasure to start the Ninety-Second Annual Meeting and we begin with the election of emeritus, corresponding and active fellows.

Will our Secretary please announce them?

DR. FRANK D. LATHROP (Secretary of the Association): Mr. President, all candidates have been duly elected.

For Active Fellowship:

Dr. S. R. Cohen

Dr. R. Williams

For Emeritus Fellowship:

Dr. V. K. Hart

Dr. P. M. Moore

Dr. J. A. Murtagh

For Corresponding Fellowship:

Dr. D. F. N. Harrison

Dr. A. Rethi

Dr. R. G. MacBeth

All have been elected to membership.

ANNUAL BUSINESS MEETING

MONDAY MORNING, MAY 24, 1971

The Annual Business Meeting of the Ninety-Second Annual Meeting of the American Laryngological Association convened at eight-five o'clock, Monday morning, May 24, 1971, in Continental Parlors VI and VII of the San Francisco Hilton Hotel, San Francisco, California, President DeGraaf Woodman, presiding.

REPORT OF THE SECRETARY

The Ninety-First Annual Meeting of the American Laryngological Association was held on April 24th and 25th, 1970, in the Hollywood Beach Hotel, Hollywood, Florida. President Alden H. Miller alternated with Vice-Presidents Joseph P. Atkins and Stanton A. Friedberg in presiding over various portions of the program. The meeting attendance was excellent, 80 Fellows and 235 Guests registering.

The program arranged by President Miller was excellent. Fourteen scientific papers were presented with the majority oriented toward laryngeal disease. The papers, which were presented well, were received with enthusiasm in spite of intermittent malfunction of the projection equipment.

Dr. Francis E. LeJeune was the Honored Guest. His presentation entitled "When Color Came to Laryngeal Movies" was educational as well as being historically interesting. He was presented with a suitably engrossed certificate as the Honored Guest.

The President's Reception and Dinner was held on Friday Evening, April 24th, in the Pageant Room of the Hollywood Beach Hotel. The menu chosen by President and Mrs. Miller was superb. Music for entertainment was furnished by a calypso group which were quite unique.

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

Honorary Fellow:	Dr. Frederick T. Hill
Emeritus Fellows:	Dr. A. C. Furstenberg
	Dr. Fletcher D. Woodward
Corresponding Fellows:	Sir Terence Cawthorne
	Dr. Andrew A. Eggston

Since the Annual Meeting we have received word of the death of C. Stewart Nash on May 1, 1971, and Theodore Walsh on April 29, 1971. Dr. Nash was elected to Active Fellowship in 1937 and to Emeritus Fellowship in 1970. Dr. Walsh was elected to Active Fellowship in 1954.

The DeRoaldes Medal was presented to Dr. Francis E. LeJeune, New Orleans, Louisiana, at the Annual Dinner.

The Newcomb Award was presented to Dr. Louis H. Clerf, St. Petersburg, Florida, at the Annual Dinner.

The following were elected to Fellowship:

Active:

Dr. John J. Ballenger, Winnetka, Ill.
Dr. John P. Frazer, Rochester, N. Y.
Dr. Gabriel F. Tucker, Jr., Philadelphia, Pa.

Emeritus:

Dr. Robert L. Goodale, Ipswich, Mass.
Dr. Gordon D. Hoople, Syracuse, N. Y.
Dr. P. E. Ireland, Bowmanville, Ontario, Canada
Dr. Francis E. LeJeune, New Orleans, La.
Dr. William J. McNally, Montreal, Canada
Dr. C. Stewart Nash, Rochester, N. Y.

Honorary:

Dr. H. L. Williams, Jr., West St. Paul, Minn.

Corresponding:

Sir George C. Halliday, M.D., Sydney, Australia
Jack Hutcheon, M.D., Brisbane, Australia

The following officers were elected for the coming year:

President: Dr. Degraaf Woodman
First Vice-President: Dr. Robert B. Lewy
Second Vice-President: Dr. Oliver W. Suehs
Secretary: Dr. Frank D. Lathrop
Treasurer: Dr. Charles M. Norris
Librarian, Historian and Editor: Dr. F. Johnson Putney
First Councilor: Dr. Francis A. Sooy
Second Councilor: Dr. Lawrence R. Boies
Third Councilor: Dr. Francis W. Davison
Fourth Councilor: Dr. Alden H. Miller

During the Annual Business Meeting, the President brought to the attention of the Fellowship, the opinion of the Council that at future meetings a registration fee should be established for both Fellows and Guests in order to help defray the increasing costs of the Annual Meeting. During the discussion which followed, it was established that the principal item of concern was the increasing cost of the President's Reception and Annual Dinner. In view of this, the Fellowship decided that at future meetings an appropriate charge should be made for those attending the President's Reception and Annual Dinner.

The next Annual Meeting will be held in the San Francisco Hilton Hotel, San Francisco, California, on the mornings of May 23rd and 24th, 1971.

The dates and locations for future meetings are as follows: 1972, April 28-29, The Breakers Hotel, Palm Beach, Florida; 1973, April 1-2, Chase-Park Plaza, St. Louis, Missouri.

The current Fellowship of the Association is as follows:

Active	90
Emeritus	29
Corresponding	10
Honorary	10
	<hr/>
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Respectfully submitted,

Frank D. Lathrop, M.D., Secretary

(This report was duly accepted by the Fellows.)

REPORT OF THE TREASURER

Exhibit A

AMERICAN LARYNGOLOGICAL ASSOCIATION
GENERAL FUND
OPERATING STATEMENT AND FUND BALANCE
FISCAL YEAR ENDED MARCH 31, 1971

Income

Dues (88 @ \$75.; 3 new members @ \$115.)	\$6,945.00	
Sale of Transactions	1,040.00	
Sale of Group Photographs	40.25	
Interest on certificates of deposit\$ 437.50		
Interest on savings accounts	273.68	
Interest on U. S. Treasury Notes	284.80	995.98
		<u>9,021.23</u>

Expenses

Annual Meeting, 1970

Reception and Banquet	\$1,498.95	
Reporting	393.69	
Projection	343.20	
Guest of Honor	356.08	
Programs and Miscellaneous	662.12	
Total Annual Meeting Expense		\$3,254.04
Council Meeting Expense		2,396.34
Transactions and Mailing		<u>3,221.12</u>

General Expense

Stationery, Printing, Postage	\$ 790.21	
Clerical Hire	450.00	
Auditing	100.00	
Treasurer's Bond	55.00	
Dues International Federation of Otolological Societies	100.00	
Contribution American Council Otolaryngology	200.00	
Contribution National Society Medical Research ..	100.00	
Contribution Terence Cawthorne Memorial	100.00	
Presidents Keys	31.80	
Bank Charges and Foreign Exchange	6.89	
Total General Expense		<u>1,933.90</u>
Total Expenses		<u>\$10,805.40</u>
Net Income (Loss) for the year		(1,784.17)
Fund Balance March 31, 1970		<u>7,178.09</u>
Fund Balance March 31, 1971		<u>5,393.92</u>

Exhibit B

AMERICAN LARYNGOLOGICAL ASSOCIATION
TRUST FUND
OPERATING STATEMENT AND FUND BALANCE
FISCAL YEAR ENDED MARCH 31, 1971

Income

Interest on U. S. Series H Bonds	\$ 311.33	
Dividends on Common Stocks	3,304.20	
Sale of Rights (A. T. and T.)	31.61	\$ 3,647.14
Donation to Memorial Fund (Dr. Woodman)		500.00
Bequests to Memorial Fund		
Lila P. Kennedy (Dr. George Fetterolf)		1,000.00
Dr. August C. Beck (Final Distribution)	1,662.80	\$ 6,809.94

Expense

DeRoaldes Medal	202.40
Net Income for Fiscal Year	6,607.54
Fund Balance March 31, 1970	65,142.68
Fund Balance March 31, 1971	71,750.22

TRUST FUND ASSETS

Cash	\$20,144.70
Accounts Receivable (E. F. Hutton and Co.)	46.41
Investments (Exhibit D)	
U. S. Bond Series H at cost, par and maturity values	\$ 6,500.00
Common Stocks Purchased (at cost)	\$5,672.38
Donated (Market Value at Donation)	2,362.00
Mutual Fund (at cost)	37,024.73
Total Trust Fund Assets	71,750.22

CASH SUMMARY

Cash in Commercial Account (Girard Bank, Philadelphia)	815.29
Cash in Savings Account (Girard Bank, Philadelphia) ..	24,723.33
	25,538.62
Allocation:	
General Fund (Exhibit A)	\$ 5,393.92
Trust Fund (above)	20,144.70
	25,538.62

Exhibit C

AMERICAN LARYNGOLOGICAL ASSOCIATION
INDIVIDUAL TRUST FUNDS
MARCH 31, 1971

	<i>Principal and Retained Earnings 3/31/70</i>	<i>Earnings*</i>	<i>April 1, 1970 to March 31, 1971 Donations- Bequests</i>	<i>Awards</i>	<i>Principal and Retained Earnings 3/31/71</i>
Casselberry	\$ 9,116.76	\$ 510.42			\$ 9,627.18
Johnston	8,631.87	483.28			9,115.15
DeRoaldes	5,939.86	332.55		(\$202.40)	6,070.01
Newcomb Trust	3,388.89	189.72			3,578.61
Newcomb Prize	790.99	44.28			835.27
Research	26,965.51	1,509.73			28,475.24
Memorial	10,308.80	577.16	\$3,162.80		14,048.76
	\$65,142.68	\$3,647.14	\$3,162.80	(\$202.40)	\$71,750.22

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

Exhibit D

AMERICAN LARYNGOLOGICAL ASSOCIATION
TRUST FUND INVESTMENTS
MARCH 31, 1971

U. S. Government Bonds:

Six \$1,000.00 Series H Bonds (at cost)	\$6,000.00	
One \$500.00 Series H Bond (at cost)	500.00	
Total U. S. Government Bonds		\$ 6,500.00

Common Stocks:

	<i>Number of Shares</i>	<i>Cost</i>	<i>Market Value at Date of Donation</i>	
American Telephone and Telegraph Co.	70	\$4,845.02		
International Business Machine Corp.	1		\$ 517.00	
International Business Machine Corp. (1)	7	803.98		
McDonnell Douglas Corp. (2)	112		1,170.00	
McDonnell Douglas Corp. (3)	14	23.38		
McIntyre-Porcupine Mines, Ltd.	15		675.00	
		<u>\$5,672.38</u>	<u>\$2,362.00</u>	8,034.38

Mutual Fund—Massachusetts Investors

Trust (at cost)	37,024.73
Total Trust Fund Investments	\$51,559.11

(1) Includes 5¼ shares received in stock dividends.

(2) Includes 86 shares received in stock dividends.

(3) Includes 13.44 shares received in stock dividends, and exercised right to purchase .56 share.

Charles M. Norris, M.D., Treasurer

(This report was duly accepted by the Fellows.)

FINANCE COMMITTEE REPORT

March 31, 1971

Assets Consist of:

\$ 6,500.00	United States Series H Bonds @ par @ maturity
24,723.33	Cash in savings account
815.29	Cash in commercial account
46.41	Accounts receivable (Credit with E. F. Hutton)
\$32,085.03	

Plus Stock Portfolio of:

<i>Cost</i>	<i>Market Value 3/31/71</i>	<i>Shares</i>		<i>Share Value 3/31/71</i>	<i>Range High</i>	<i>1970-1971 Low</i>
\$ 4,845.02	\$ 3,412.50	70	Amer. Tel. and Tel.	48%	53%	40%
1,320.98	2,860.00	8	Int. Bus. Machines	357½	387	218%
37,024.73*	30,133.95	2071	Mass. Invest. Trust	14.55 bid (av. cost 17.88)		
1,193.38	4,410.00	126	McDonnell-Douglas	35	38%	13
675.00	2,040.00	15	McIntyre-Porcupine	136	179%	128
			D. J. Industrials	904.37	916.83	631.16
\$45,059.11 — \$42,856.45 = \$2,202.66 paper loss (paper loss on 3/31/70 was \$3,440.17).						

*Including initial sales charge and dividends reinvested by purchase additional shares. Dividends on Mass. Inv. Trust for period 3/31/70 to 3/31/71 were \$2,347.92.

AMERICAN LARYNGOLOGICAL ASSOCIATION
YEAR END BALANCES

	<i>General Fund</i>	<i>Trust Fund</i>
1959	\$ 3,257.75	
1960	4,699.66	\$43,760.77
1961	6,022.61	45,506.03
1962	7,717.68	48,904.10
1963	9,524.84	51,051.56
1964	12,909.40	52,594.12
1965	10,543.63	55,047.98
1966	9,226.61	56,586.92
1967	11,200.04	53,798.11
1968	11,191.65	56,310.08
1969	9,567.10	59,420.00
1970	7,178.09	65,142.68
1971	5,393.92	71,750.22

ACCOUNTANT'S REPORT

To the Members of the American Laryngological Association
Gentlemen:

We have examined the statements of cash receipts and disbursements of the AMERICAN LARYNGOLOGICAL ASSOCIATION, including Trust Funds, for the fiscal year ended March 31, 1971. Our examination was made in accordance with generally accepted auditing standards, and accordingly included such tests of the accounting records and such other auditing procedures as we considered necessary in the circumstances.

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

In our opinion, the accompanying statements of cash receipts and disbursements present fairly the financial position of the American Laryngological Association and its Trust Funds at March 31, 1971, on a cash basis, and the results of its operations for the year then ended, in conformity with generally accepted accounting principles applied on a basis consistent with that of the preceding year.

Respectfully submitted,

NEMORE, GRAHAM & COMPANY
Certified Public Accountants

May 6, 1971

REPORT OF THE LIBRARIAN, HISTORIAN, AND EDITOR

The Ninety-First Meeting of the American Laryngological Association was held on April 24 and 25, 1970, at the Hollywood Beach Hotel, Hollywood, Florida. There were 80 Fellows and 235 Guests registered. The meeting was ably reported by the Martin C. Johnson Reporting Service, and the papers and discussions with two exceptions were sent to the Annals Publishing Company on June 29, 1970. The procrastination of two authors and the fact that the completed printed matter fell off the transporting truck en route to the bindery delayed publication until December when the Transactions were mailed to the membership and the papers appeared in the Annals.

Two hundred twenty-five volumes of the 1970 Transactions were ordered. One hundred seven books were sent to the active and honorary members, The Library of Congress, the National Library of Medicine for the Index Medicus and the families of the deceased members.

There were 43 standing orders and 33 additional single orders. The price of the 1970 Transactions was \$3,115.02, an increase of \$464.09 over 1969. The cost of printing and binding rose sharply during the past year in spite of the fact that the format of the Transactions was changed to conform with that of the Annals, thereby reducing additional type setting expenditures. By direction of the Council the cost of the Transactions was increased to \$12.00 per volume. The present page size is 10 x 6 $\frac{3}{4}$ " with the same hard binding. An error in listing the officers for 1968-1969 in Volume 90 (1969) was amended by mailing to the members an adhesive page to place over the incorrect sheet.

The Audio-Digest foundation recorded the 1970 meeting, and a complete tape of the recording was sent to the Editor.

Respectfully submitted,

F. Johnson Putney, M.D.
Librarian, Historian and Editor

(This report was duly accepted by the Fellows.)

REPORT OF THE NOMINATING COMMITTEE

DR. VICTOR R. ALFARO (Washington, D. C.): President Woodman, and Fellow Members.

The Nominating Committee met yesterday. It presents the following slate of officers and members of the Council:

F. Johnson Putney;
James A. Harrill;
James D. Baxter;
Frank D. Lathrop;
Charles M. Norris;
Charles F. Ferguson;
Lawrence R. Boies;
Francis W. Davison;
Alden H. Miller;
DeGraaf Woodman.

PRESIDENT WOODMAN: Thank you, Victor.

(The report was duly accepted by the Fellows.)

REPORT BY THE REPRESENTATIVE FROM THE
AMERICAN BOARD OF OTOLARYNGOLOGY

Since the last Annual Meeting of the American Laryngological Association, the American Board of Otolaryngology has held two meetings. The first of these was held in Toronto, Canada, in May, 1970, where 91 candidates were examined. Of these, 8 were conditioned, 6 failed and 77 passed the examination.

The second examination was held in Chicago, Illinois, in September, 1970, just prior to the meeting of the American Academy of Ophthalmology and Otolaryngology. The format of this examination differed from those of previous years in that it was a blend of old and newer methods of examining candidates. The innovations introduced on this occasion were the result of recommendations made by the Examination Committee in its continuing search for better methods of assessing a candidate.

One of the changes in this examination was a 2½ hour period during which the candidate answered a series of multiple choice questions. In addition, the number of oral examinations taken by the candidate was reduced and their character changed to that of a structured oral so that the breadth of knowledge in the specialty of otolaryngology, and to some extent, the candidate's application of this knowledge might be better evaluated. The clinical examinations to which a candidate was exposed were reduced in number, also. However, the reduction was off-set by the addition of a patient simulation examination designed to give the examiner information relative to a candidate's ability to diagnose, treat and handle an emergency situation and also to discuss the management of a given situation with both the patient and his family.

One hundred candidates took this examination. Seventeen percent either conditioned or failed the examination while 83 percent passed. At completion of the examination the candidate received an individual analysis of his performance on the examination. This was another innovation which should prove valuable to the candidate in evaluating his weaknesses and strength in otolaryngology.

Further changes in the format of the examination will be in effect for the examination to be held at the Palmer House, Chicago, Ill., October 30 to November 4, 1971. It is believed that with these additional changes in the examination, only one examination will be necessary each year to accomodate the applicants for that year.

Respectfully submitted,

Frank D. Lathrop, M.D., Representative

(This report was duly accepted by the Fellows.)

REPORT FROM THE BOARD OF GOVERNORS
AMERICAN COLLEGE OF SURGEONS

The Board of Governors of the American College of Surgeons met on Sunday, October 11th, 1970, at the Conrad Hilton Hotel in Chicago. 153 Governors were present.

Dr. Joel W. Baker, President, reviewed activities of the College, citing its concern for the burgeoning malpractice problem, the delivery of medical care with survival of quality and the planned surgical education self-assessment program.

Dr. William Longmire, Chairman of the Board of Regents, put the membership of the College at about 32,000, of whom 29,000 are in the United States and Canada. Of the 1,500 new candidates to be inducted in 1970, 591 are in general surgery, 960 in the surgical specialties. The increasing number in the latter category underscores a trend evident for the past several years. He also called attention to improved communications provided by a pamphlet "Patients, Surgeons and Hospitals," and by the revised Bulletin, issued monthly, both distributed to the entire membership.

Dr. C. Rollins Hanlon, Director of the College, reviewed the changes in the Board of Governors to make it less unwieldy. New societies in the future will be represented by one Governor. Those societies now having three Governors will in the future have two, those with two will continue to have two.

A report by Mr. Robert Happ, Comptroller, cited a small surplus of funds until 1972, when in spite of the recent dues increase, new sources of income will be needed.

Dr. Eliot Snow, Chairman of the Committee on Malpractice and Professional Liability, presented the final report of his group with short and long term recommendations to ameliorate the malpractice situation. These involved co-operation with other societies and groups such as the American Hospital Association and the American Ins. Association, to avoid reduplication of effort, encouragement of state societies to get involved in remedial legislation and inception of efforts to get malpractice claims out of courts and into properly constituted arbitration bodies.

Discussion of the matter of physicians on hospital governing bodies culminated in endorsement by the Governors of a resolution of the Board of Regents, reaffirming its stand that medical Staff members serve on governing boards and urging the Joint Commission on Accreditation of Hospitals to support and implement this policy. The Governors further urged the Regents to recommend to the Joint Commission on Accreditation of Hospitals that such representation of physicians on Boards of Trustees be of more than the token variety.

During the afternoon the Governors broke up into eight discussion groups. Your representative participated in that on "How Can the College Best Help Its Fellows to Deal with Current and Coming Changes in Medical Practice?" Infor-

mation from Washington and Canada was made available as a point of departure. A recommendation was made to the Regents for a committee to continue study of this rapidly changing subject.

On February 6, 1971, a special meeting of the Board of Regents, Board of Governors, and Chapter officers was called at Murphy Memorial Auditorium in Chicago to discuss the general conclusions of the Curlie House Conference, jointly sponsored by the American College of Surgeons and the American Surgical Association. I was unable to attend the meeting, but have read the reports of the ten discussion groups who reviewed many of the socio-economic problems impinging on the practice of surgery. Since this is the first concerted effort of two prestigious surgical societies to study, dissect and evaluate the needs of surgery and of our society today, I urge the members of the Association to read the summaries of these workshops as published in the April Bulletin of the American College of Surgeons. You will not find the answers, but you will be alerted to the problems that confront us and the efforts being made to analyze and solve them.

Respectfully submitted,

W. Franklin Keim, M.D.

(This report was duly accepted by the Fellows.)

REPORT OF THE LARYNGOLOGICAL RESEARCH FUND COMMITTEE

DR. ANDERSON C. HILDING: The Research Fund Committee met yesterday. Present were Drs. Ogura, Alden Miller, Daniel Baker and Hilding.

The amount of our research fund is small compared as these things go. In the principal is \$16,000 (approximately). There is approximately \$12,000 of unused income and the annual income is about \$1,500 a year.

The committee recommends that \$10,000 of the unused funds be added to the principal making a total of \$26,000 and this be kept as principal.

The Committee accepted the offer of the Council to add to the \$1,500 a year annual income approximately \$500 from the Memorial Fund.

It is recommended that a grant not be made every year, but that two or three years be allowed to accumulate, so that the sum would be \$5,000 or \$6,000 and then granted to a promising project.

It is further recommended that the money be granted as a supplementary fund for some ongoing project, or toward a pilot study, that the money be used for supplies, animals, publication cost and so on, but that no money be used for salaries of investigators.

It is recommended that any application must specifically describe the nature and purpose of the project and that a report be made at the end of the study as to the results.

Applications for funds:

At present there are no applications on hand. There have been two inquiries.

It is recommended further that the amount available toward a project be published in the Transactions.

Respectfully submitted,

Anderson C. Hilding, M.D., Chairman

PRESIDENT WOODMAN: We will now have an announcement by Dr. John Bordley, Executive Director of the American Council of Otolaryngology.

DR. JOHN BORDLEY: The American Council of Otolaryngology set up a Committee on Malpractice which is now the Committee on Professional Liability, and through the very active work of the committee, they have been able to hold a recent meeting in Chicago in which 36 organizations contributed, including the AMA and the College, the Hospital Association, and really all of the important associations connected with medicine.

They have set up seven committees. They have recommended the formation of a national foundation to study professional liability. They are holding their first organizational meeting for this foundation in July.

They are expecting to raise a working budget of about \$150,000 and the government, through HEW, is very interested in this and is sending people not only to advise but to participate in the formation of such an organization.

They have also enlisted the interest of the judiciary and of the practicing lawyers.

I thought that this was of interest. This has moved very rapidly, but it seems to be very firmly organized and it looks as if we are going to have now an instrument to study the problems of malpractice, not just try to reduce the cost of malpractice insurance.

PRESIDENT WOODMAN: Thank you very much, John. That gives some hope that young men starting out today will be able to afford to practice and the old men who are fading out, will not fade out too fast. I heard the other day that a young orthopedic surgeon starting in California has to carry and pay practically \$10,000 premium a year. Soon we will have no doctors at that rate unless we get some legislation. Of course, our main thing is the contingent basis on which legal cases are taken.

EXECUTIVE SESSION

The Executive Session of the Ninety-Second Annual Meeting of the American Laryngological Association reconvened at twelve o'clock, Monday, May 24, 1971, President DeGraaf Woodman presiding.

PRESIDENT WOODMAN: Our Secretary will now read the proposed amendment passed by the Council.

SECRETARY LATHROP: You have all received notification as required by the Constitution concerning a proposed amendment change. That was given to you in a post-Council letter of February 8th, 1971, so we are well within the time limit required.

The Council at its second Council meeting of April 23, 1970, held at The Hollywood Beach Hotel, Hollywood, Florida, voted to submit to the fellowship at the annual meeting the proposed change in Constitution and By-Laws of the American Laryngological Association, Incorporated, whereby Article V, Section 1, relating to property rights be changed so as to minimize the possibility of losing the tax-free status of the Association.

In addition, the Council proposed also that Section 2 of Article V relating to rights and privileges be transferred as a new Section "E" to Article IV. Article V, Section 1, now reads: "All rights, title and interest in and to any and all property of the Association shall be vested in the Active Fellows and no others."

It is proposed that Article V be changed to read:

"Any property interest the Association may have shall belong solely to the Association as a corporate entity and none of its members shall have individual interest or share therein. Upon dissolution, liquidation or termination of the Association, the Council shall distribute all assets of the Association remaining after all liabilities have been satisfied to such scientific or educational institutions or organizations as the Council shall deem most capable of fulfilling the purposes of the Association. If a majority of the Council shall be unable to reach a decision as to an appropriate distribution of the assets, the Council shall file a petition of distribution under order of the court pursuant to Section 56 of the New York Membership Corporation Law." I would suggest we take up this particular amendment before going on to the next.

PRESIDENT WOODMAN: You've heard the amendment. May I have a motion?

DR. ALFARO: I move it be adopted.

DR. MILLER: Second.

PRESIDENT WOODMAN: All those in favor say "aye"; so carried.

SECRETARY LATHROP: The next amendment is Section 2 of Article V relating to rights and privileges being transferred as a new Section "E" to Article IV.

Section 2, Article V now reads:

"Only active fellows shall have the right to vote and hold office. Any fellow regardless of classification may be appointed by the President, subject to the approval of the Council, to serve on the committee or be assigned to a task for which he is eminently qualified."

It is proposed that this section be transferred intact as a new Section "E" to Article IV which pertains to qualifications for membership.

PRESIDENT WOODMAN: You've all heard this proposal. May I have a suggestion from somebody?

DR. SUEHS: I move it be adopted.

PRESIDENT WOODMAN: Second?

DR. MILLER: Second.

PRESIDENT WOODMAN: All those in favor say "aye"; so carried.

PRESIDENT WOODMAN: We are now ready for our election of officers and Board of Directors.

SECRETARY LATHROP: Those nominated are: Drs. Putney, Harrill, Baxter, Lathrop, Norris, Ferguson, Boies, Davison, Miller and Woodman.

DR. DALY: I move that nominations be closed.

PRESIDENT WOODMAN: Second?

DR. BAKER: Second.

PRESIDENT WOODMAN: You've all heard the motion that nominations be closed. All those in favor say, "aye;" so carried. The Board of Directors will meet in the corner of the room for a few minutes.

(There followed a brief recess while the new Board of Directors met.)

SECRETARY LATHROP: The Board of Directors held a brief organizational meeting and have come up with the following slate:

For President: Dr. F. Johnson Putney;
For First Vice-President: Dr. James A. Harrill;
For Second Vice-President: Dr. James D. Baxter;
For Secretary: Dr. Frank D. Lathrop;
For Treasurer: Dr. Charles M. Norris;
For Editor, Librarian, Historian: Dr. Charles F. Ferguson;
For First Councilor: Dr. Lawrence R. Boies;
For Second Councilor: Dr. Francis W. Davison;
For Third Councilor: Dr. Alden H. Miller;
For Fourth Councilor: Dr. DeGraaf Woodman.

PRESIDENT WOODMAN: I think you have heard the slate. Is everybody satisfied with it? No objections? Will somebody make that motion please? (The motion was made and seconded from the floor.) All right, so moved. Now, we have the duty of congratulating the new slate and particularly our new President. If Johnson will step up, I will hand him the gavel.

(Whereupon Dr. Putney then came forward to the podium to accept the gavel from President Woodman.)

Johnson, you've been around long enough and seen enough to know that you are going to enjoy a wonderful year. I hope you will continue to have the cooperation the past few Presidents have had. It's going to be a great one for us.

PRESIDENT PUTNEY: Thank you, Dr. Woodman.

It has been a pleasure to have been associated with this society in the capacity of Editor and now to turn this duty over to an experienced and accomplished Bostonian. I hope he will have better success than I in securing the completed papers before the deadline. This is the first year in my tenure that all papers have been turned in on time, so I hope it will continue.

I think we are also indebted to Dr. Ferguson for taking on this job since he is in the midst of completing a textbook on pediatric otolaryngology. I am sincerely grateful and appreciate your confidence in selecting me as your President and can only say thank you. I realize that I follow a long line of distinguished presidents and will do my best to live up to this heritage.

I am looking forward to another excellent meeting next year in Florida. I welcome any suggestions that any of you might have. You will be hearing further about certain ideas in that connection and I would just like to throw out a preliminary thought for you to consider. Please do not think of it negatively until you have had an opportunity perhaps to mull it over. I will be in contact with you later. This is the advisability of holding our meeting with the AMA at some future date, similar to that which is being done this year by the Society of Head and Neck Surgery. We had in mind a possibility of 1973 and please think of this carefully. It is a variance as to what this Association has done in the past, but medicine is changing, and changing very rapidly and drastically. It might be something we should consider seriously as an individual participation with the AMA. We shall not lose our own identity. We shall have our own meeting, our own officers, our own banquet. It will, I think, give us in otolaryngology somewhat more representation and it will improve our image with the general medical public.

Now, as my first duty, it is a pleasure to have Dr. Woodman come to the podium please.

(Whereupon Dr. Woodman then came to the podium as requested.)

I have here a key engraved to Dr. Woodman which is given by our Association as an expression of our esteem for his association with us and for his presidency.

It seems that each year the President gives a better program than the one previously and Dr. Woodman's program, I think we would all agree, this year has been outstanding. I think I express that sentiment from all our members. It has been a very nice association and we hope it will continue further.

(Whereupon President Putney then presented the engraved key to Dr. Woodman.)

DR. WOODMAN: Thank you, Johnson, and thank you all. It has been a wonderful year for me.

PRESIDENT PUTNEY: Is there any other business to come before the society?

If not, we stand adjourned.

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

THE JAMES E. NEWCOMB AWARD

ROBERT E. PRIEST, M.D.*

MINNEAPOLIS, MINN.

In 1941, the James E. Newcomb Award was established by the American Laryngological Association.¹ Inquiry has revealed very little knowledge of Newcomb's life and achievements, even among senior members of the association. As a matter of personal curiosity the author looked into Newcomb's bibliography, read some of his papers and became slightly acquainted with his life. Upon discussing these matters with other members of the association, it became apparent that some general interest existed. Therefore, at the request of the Historian, this material regarding Newcomb has been summarized.

Newcomb's first medical paper appeared in 1884 when he was twenty-seven years of age. He published thirty-two papers through the years until 1912. A major achievement was his editorship of the American Edition of Grunwald's "Atlas of Diseases of the Mouth, Pharynx, and Nose." His President's Address to the American Laryngological Association was the opening paper in the Transactions of the Thirty-Fourth Annual Meeting; later in the same volume his obituary appeared.²

Newcomb's medical writing dealt with general medical subjects until he was thirty-four years old and had published five papers. From then on he treated only laryngological subjects. His bibliography and obituary³ are appended to these notes.

Newcomb graduated from Yale College in 1880 and in 1883 received the Doctor of Medicine degree from the College of Physicians and Surgeons, New York. After serving as house officer at the Roosevelt Hospital, New York, he began the practice of medicine in the City of New York. He became a specialist in diseases of the ear, nose and throat. He was consulting laryngologist to the Roosevelt Hospital, and Professor of Laryngology at the Cornell Medical School. For several years he was a lecturer in laryngology at Columbia College.³

Newcomb died in 1912 in the year following his presidency of the American Laryngological Association; he had been secretary for eleven years before his presidency and had been elected to membership in 1893. His presidential address reveals him as a kind, modest, far-seeing man. The paper is worth reading.

His widow, Mrs. Elizabeth W. Newcomb, lived to will \$2,500 to the association in 1941. Of this sum, \$2,000 was to be invested and the proceeds used to maintain the archives of the association. The balance was to be used to maintain an annual award.

The award was to be "given annually to a member of the American Laryngological Association for Scientific Attainment in Rhinology and Laryngology, or as a mark of esteem and appreciation for services to the association."

This brings us to the business at hand: the presentation of the 1971 James E. Newcomb Award. Newcomb himself, would undoubtedly be interested in the qualifications of this year's recipient.

*Chairman, 1971 James E. Newcomb Award Committee.

The recipient was born in Philadelphia, December 19, 1908. He studied pre-medical subjects at the University of Pennsylvania and received his Doctor of Medicine degree from Jefferson Medical School in Philadelphia in 1933. In 1939, Columbia University in New York granted him a Medical Doctor of Science degree in Otolaryngology. In 1938, he married Geraldine Dieck; the couple have had five children: Geraldine, Daniel Clifton III, Judith, Marianne, and Elizabeth.

In 1964, after long service on the faculty of the Columbia College of Physicians and Surgeons, he became Professor of Otolaryngology and Chairman of the Department of Otolaryngology, as well as Director of the Otolaryngological Service of Presbyterian Hospital of New York City.

The James E. Newcomb Award committee of the American Laryngological Association, for reasons consistent with the purposes of the award, has selected Daniel Clifton Baker, Jr., to receive the 1971 Newcomb Award.

BIOGRAPHY OF JAMES EDWARD NEWCOMB, M.D.

Dr. James Edward Newcomb was born at New London, Connecticut, on August 27, 1857. He graduated from Yale College in 1880, and in 1883 received the degree of M.D. from the College of Physicians and Surgeons, New York. After serving as house-officer at the Roosevelt Hospital, New York, he began the practice of his profession in that city, when he became a specialist in diseases of the ear, nose, and throat.

He was consulting laryngologist to the Roosevelt Hospital and professor of laryngology at the Cornell Medical School. He was also for several years a lecturer in Laryngology at Columbia College. He was a member and former president of the American Laryngological Society, a member of the American Academy of Medicine, the New York Academy of Medicine, of the New York County Medical Society, of the West End Medical Society, and of the Hospital Graduates Club. He was a frequent contributor of special articles to medical journals and edited the American edition of Grunwald's "Atlas of Diseases of the Mouth, Pharynx, and Nose."

Dr. Newcomb died of arteriosclerosis on August 12, 1912, at Lake Kushaqua in the Adirondack Mountains.

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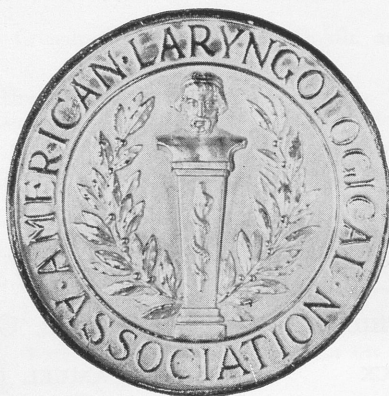
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ACKNOWLEDGMENT—Appreciation for bibliographic searching is expressed to Dr. Gerald Jurgens, Fellow in Otolaryngology, University of Minnesota Medical School.

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- *1936. Ballenger, Howard C., Winnetka, Ill.
- *1923. Barlow, Roy A., Nova Scotia, Canada.
- *1915. Barnes, Harry Aldrich, Kingston, Mass.
- *1928. Beck, Joseph C., Chicago, Illinois.
- *1921. Berry, Gordon, Worcester, Mass.
- *1941. Bowers, Wesley C., New York, N. Y.
- *1901. Brown, J. Price, Toronto, Canada.
- *1891. Bryan, Joseph H., Washington, D. C.
- *1913. Butler, Ralph, Philadelphia, Pa.
- *1930. Campbell, Edward H., Philadelphia, Pa.
- *1897. Clark, J. Payson, Boston, Mass.
- *1899. Cobb, Frederick C., Bradenton, Florida.
- *1939. Cody, Claude C., Jr., Houston, Texas.
- *1905. Coffin, Lewis A., New York, N. Y.
- *1893. Coolidge, Algernon, Boston, Mass.
- *1937. Crowe, Samuel J., Baltimore, Maryland.
- *1941. Cunning, Daniel S., New York, N. Y.
- *1913. Dabney, Virginia, Washington, D. C.
- *1882. De Blois, Thomas Amory, Boston, Mass.
- *1941. Dixon, Fred W., Shaker Heights, Ohio.
- *1892. Farlow, John W. Boston, Mass.
- *1930. Figi, Frederick A., Rochester, Minn.
- *1922. Forbes, Henry H., New York, N. Y.
- *1933. Foster, John H., Houston, Texas.

- *1905. Freer, Otto T., Chicago, Ill.
- *1932. Furstenberg, Albert C., Ann Arbor, Mich.
- *1940. Gatewood, E. Tribble, Richmond, Va.
- *1928. Gittins, Thomas R., Sioux City, Iowa.
- *1880. Gleitsmann, Joseph W., New York, N. Y.
- *1922. Goldsmith, Perry G., Toronto, Canada.
- *1898. Goodale, Joseph L., Ipswich, Mass.
- *1932. Graham, Harrington B., San Francisco, California.
- *1906. Greene, D. Crosby, Jr., Boston, Mass.
- *1917. Greene, Joseph B., Asheville, N. C.
- *1950. Hall, Colby, Encino, Calif.
- *1905. Halsted, Thomas H., Los Angeles, Calif.
- *1896. Hardie, Thomas Melville, Chicago, Ill.
- *1915. Hastings, Hill, Los Angeles, Calif.
- *1944. Havens, Fred Z., Rochester, Minn.
- *1888. Hinkel, Frank Whitehill, Buffalo, New York.
- *1895. Hopkins, Frederick E., Springfield, Mass.
- *1930. Houser, Karl M., Ardmore, Pa.
- *1927. Hubbard, Thomas, Toledo, Ohio.
- *1919. Hurd, Lee Maidment, Rowayton, Conn.
- *1920. Imperatori, Charles J., Essex, N. Y.
- *1904. Ingersoll, John Marvin, Miami, Fla.
- *1928. Jarvis, DeForest C., Barre, Vt.
- *1939. Johnston, Willam H., Santa Barbara, Calif.
- *1942. Kelly, Joseph D., New York, N. Y.
- *1918. Kenyon, Elmer L., Chicago, Ill.
- *1921. Kernan, John D., New York, N. Y.
- *1929. Kistner, Frank B., Portland, Oregon.
- *1885. Knight, Charles H., New York, N. Y.
- *1939. Large, Secord H., Cleveland, Ohio.
- *1894. Leland, George A., Boston, Mass.
- *1922. Lillie, Harold I., Rochester, Minn.
- *1943. Lincoln, William R., Cleveland, Ohio.
- *1928. Lyman, Harry Webster, St. Louis, Mo.
- *1886. MacCoy, Alexander W., Philadelphia, Pa.
- *1941. Martin, Robert C., San Francisco, Calif.
- *1896. Mayer, Emil, New York, N. Y.
- *1951. McCart, Howard W. D., Toronto, Canada.

- *1939. McCaskey, Carl H., Indianapolis, Indiana.
- *1943. McCullagh, Samuel, New York, N. Y.
- *1923. McKinney, Richmond, Memphis, Tenn.
- *1928. MacPherson, Duncan, New York, N. Y.
- *1939. Myers, John L., Kansas City, Mo.
- *1901. Myles, Robert C., New York, N. Y.
- *1937. Nash, C. Stewart, Rochester, N. Y.
- *1922. New, Gordon B., Rochester, Minn.
- *1923. Newhart, Horace, Minneapolis, Minn.
- *1903. Packard, Francis R., Philadelphia, Pa.
- *1878. Porter, William, Ocean Springs, Miss.
- *1942. Potts, John B., Omaha, Nebr.
- *1903. Renner, W. Scott, Buffalo, N. Y.
- *1897. Rhodes, John Edwin, Chicago, Ill.
- *1884. Rice, Clarence C., New York, N. Y.
- *1905. Richards, George L., South Yarmouth, Mass.
- *1878. Robinson, Beverly, New York, N. Y.
- *1921. Sauer, William E., St. Louis, Mo.
- *1923. Sewall, Edward C., Palo Alto, Calif.
- *1930. Seydell, Ernest M., Wichita, Kansas.
- *1907. Shambaugh, George E., Chicago, Ill.
- *1908. Smith, Harmon, New York, N. Y.
- *1923. Spencer, Frank R., Boulder, Colo.
- *1927. Tobey, Harold G., Boston, Mass.
- *1925. Tucker, Gabriel, Haverford, Pa.
- *1892. Wagner, Henry L., San Francisco, Calif.
- *1892. Watson, Arthur W., Philadelphia, Pa.
- *1948. Whalen, Edward J., Hartford, Conn.
- *1922. White, Francis W., New York, N. Y.
- *1939. Wilson, J. Gordon, Old Bennington, Vt.
- *1905. Wood, George B., Wynnewood, Pa.
- *1935. Woodward, Fletcher D., Charlottesville, Va.

Active Fellows

- *1880. Allen, Harrison, Philadelphia, Pa.
- *1917. Arrowsmith, Hubert, Brooklyn, N. Y.
- *1879. Asch, Morris J., New York, N. Y.
- *1942. Ashley, Rae E., San Francisco, Calif.

- *1923. Babbitt, James A., Philadelphia, Pa.
- *1906. Ballenger, William L., Chicago, Ill.
- *1880. Bean, C. E., St. Paul, Minn.
- *1949. Beck, August L., New Rochelle, N. Y.
- *1904. Berens, T. Passmore, New York, N. Y.
- *1924. Bigelow, Nolton, Providence, R. I.
- *1938. Blassingame, Charles D., Memphis, Tenn.
- *1893. Bliss, Arthur Ames, Philadelphia, Pa.
- *1951. Boyden, Guy L., Portland, Oregon.
- *1895. Boylan, J. E., Cincinnati, Ohio.
- *1932. Brown, John Mackenzie, Los Angeles, Calif.
- *1892. Brown, Moreau R., Chicago, Ill.
- *1933. Buckley, Robert E., New York, N. Y.
- *1915. Canfield, R. Bishop, Ann Arbor, Mich.
- *1934. Carmack, John Walter, Indianapolis, Ind.
- *1924. Carmody, Thomas E., Denver, Colo.
- *1889. Casselberry, William E., Chicago, Ill.
- *1883. Chamberlain, C. W., Hartford, Conn.
- *1917. Chamberlin, William B., Cleveland, Ohio.
- *1896. Chappell, W. F., New York, N. Y.
- *1882. Chapman, S. Hartwell, New Haven, Conn.
- *1902. Coakley, Cornelius G., New York, N. Y.
- *1913. Coffin, Rockwell C., Boston, Mass.
- *1918. Cox, Gerald H., New York, N. Y.
- *1880. Cushing, E. W., Boston, Mass.
- *1878. Cutter, Ephraim, West Falmouth, Mass.
- *1880. Daly, W. H., Pittsburgh, Pa.
- *1878. Davis, F. H., Chicago, Ill.
- *1941. Davis, Warren B., Philadelphia, Pa.
- 1926. Dennis, Frank Lownes, Colorado Springs, Colo.
- *1901. Dickerman, E. T., Chicago, Ill.
- *1878. Donaldson, Frank, Baltimore, Md.
- *1947. Engler, Clarence W., Cleveland, Ohio.
- *1954. Equen, Murdock S., Atlanta, Ga.
- *1935. Eves, Curtis C., Philadelphia, Pa.
- *1919. Faulkner, E. Ross, New York, N. Y.
- *1914. Fetterolf, George, Philadelphia, Pa.
- *1901. Freeman, Walter J., Philadelphia, Pa.
- *1917. Friedberg, Stanton A., Chicago, Ill.
- 1940. Fuchs, Valentine H., New Orleans, La.

- *1897. Frothingham, Richard, New York, N. Y.
- *1909. Getchell, Albert C., Worcester, Mass.
- *1907. Gibb, Joseph S., Philadelphia, Pa.
- *1940. Gill, William D., San Antonio, Texas.
- *1878. Glasgow, William Carr, St. Louis, Mo.
- *1913. Goldstein, Max A., St. Louis, Mo.
- *1905. Grayson, Charles P., Philadelphia, Pa.
- *1934. Grove, William E., Milwaukee, Wisconsin.
- *1933. Harkness, Gordon F., Davenport, Iowa.
- *1878. Hartman, J. H., Baltimore, Md.
- *1945. Hickey, Harold L., Denver, Colo.
- *1879. Holden, Edgar, Newark, N. J.
- *1907. Holmes, Christian R., Cincinnati, Ohio.
- *1882. Hooper, Franklin H., Boston, Mass.
- *1893. Hope, George B., New York, N. Y.
- *1938. Hourn, George E., St. Louis, Mo.
- *1939. Hunt, Westley Marshall, New York, N. Y.
- *1901. Hyatt, Frank, Washington, D. C.
- *1925. Iglauder, Samuel, Cincinnati, Ohio.
- *1878. Ingals, E. Fletcher, Chicago, Ill.
- *1882. Ives, Frank L., New York, N. Y.
- *1938. Jackson, Chevalier L., Philadelphia, Pa.
- *1880. Jarvis, William C., New York, N. Y.
- *1878. Johnson, Hosmer A., Chicago, Ill.
- *1879. Johnson, Woolsey, New York, N. Y.
- *1960. Johnston, Kenneth C., Chicago, Ill.
- *1944. Jones, Marvin F., New York, N. Y.
- *1879. Kealhofer, R. H., St. Louis, Mo.
- *1942. King, Edward D., North Hollywood, Calif.
- *1901. King, Gordon, New Orleans, La.
- *1885. Knight, Charles H., New York, N. Y.
- *1878. Knight, Frederick Irving, Boston, Mass.
- *1898. Kyle, D. Braden, Philadelphia, Pa.
- *1880. Langmaid, Samuel W., Boston, Mass.
- *1878. Lincoln, Rufus P., New York, N. Y.
- *1911. Lockard, Lorenzo B., Denver, Colo.
- *1913. Loeb, Hanau W., St. Louis, Mo.
- *1897. Logan, James E., Kansas City, Mo.
- *1935. Looper, Edward A., Baltimore, Md.

- *1888. Lowman, John H., Cleveland, Ohio.
- *1919. Lynah, Henry L., New York, N. Y.
- *1952. Lynch, Mercer G., New Orleans, La.
- *1915. Lynch, Robert Clyde, New Orleans, La.
- *1879. McBurney, Charles, New York, N. Y.
- *1927. McGinnis, Edwin, Chicago, Ill.
- 1936. McGregor, Gregor, Toronto, Canada.
- *1913. McKimmie, O. A., Washington, D. C.
- *1945. McLaurin, John G., Dallas, Texas.
- *1885. McSherry, H. Clinton, Baltimore, Md.
- *1914. Mackenty, John E., New York, N. Y.
- *1898. Makuen, G. Hudson, Philadelphia, Pa.
- *1881. Major, G. W., Montreal, Canada.
- *1948. Maxwell, James H., Ann Arbor, Mich.
- *1954. Meltzer, Philip E., Boston, Mass.
- *1881. Morgan, E. C., Washington, D. C.
- *1950. Morrison, Lewis F., San Francisco, Calif.
- *1940. Morrison, William W., New York, N. Y.
- *1886. Mulhall, J. C., St. Louis, Mo.
- *1925. Mullin, William V., Cleveland, Ohio.
- *1914. Munger, Carl E., Waterbury, Conn.
- *1892. Murray, T. Morris, Washington, D. C.
- *1881. Mynter, H., Buffalo, N. Y.
- *1893. Newcomb, James E., New York, N. Y.
- *1895. Nichols, J. E. H., New York, N. Y.
- *1927. Orton, Henry B., Newark, N. J.
- *1894. Park, William H., New York, N. Y.
- *1892. Porcher, W. Peyre, Charleston, S. C.
- *1927. Porter, Charles T., Boston, Mass.
- *1954. Pressman, Joel J., Los Angeles, Calif.
- *1908. Randall, B. Alexander, Philadelphia, Pa.
- *1882. Rankin, D. N., Allegheny, Pa.
- *1934. Richards, Lyman G., Wellesley Hills, Mass.
- *1902. Richardson, Charles W., Washington, D. C.
- *1930. Ridpath, Robert E., Philadelphia, Pa.
- *1879. Roe, John O., Rochester, N. Y.
- *1945. Robb, James M., Detroit, Michigan.
- *1953. Roberts, Sam E., Kansas City, Mo.
- *1881. Robertson, J. M., Detroit, Mich.

- *1913. Roy, Dunbar, Atlanta, Ga.
- *1878. Rumbold, T. F., St. Louis, Mo.
- *1879. Seiler, Carl, Philadelphia, Pa.
- *1928. Shea, John Joseph, Memphis, Tennessee.
- *1893. Shields, Charles M., Richmond, Pa.
- *1909. Shurly, Burt R., Detroit, Mich.
- *1878. Shurly, E. L., Detroit, Mich.
- *1892. Simpson, William Kelly, New York, N. Y.
- *1919. Skillern, Ross H., Philadelphia, Pa.
- *1909. Sluder, Greenfield, St. Louis, Mo.
- *1879. Smith, Andrew H., Geneva, N. Y.
- *1932. Smyth, Duncan Campbell, Boston, Mass.
- *1928. Sonnenschein, Robert, Chicago, Ill.
- 1924. Stein, Otto J., Chicago, Ill.
- 1934. Stevenson, Walter, Quincy, Ill.
- *1911. Staut, George C., Philadelphia, Pa.
- *1879. Tauber, Bernhard, Cincinnati, Ohio.
- *1924. Taylor, Hermon Marshall, Jacksonville, Fla.
- *1903. Theisen, Clement F., Albany, N. Y.
- *1899. Thorner, Max, Cincinnati, Ohio.
- *1892. Thrasher, Allen B., Cincinnati, Ohio.
- *1937. Tobey, George L., Jr., Boston, Mass.
- *1888. Van der Poel, S. O., New York, N. Y.
- *1938. Vail, Harris H., Cincinnati, Ohio.
- *1936. Voislowsky, Antonie P., New York, N. Y.
- *1954. Walsh, Theodore E., St. Louis, Mo.
- *1933. Wanamaker, Allison T., Seattle, Wash.
- *1896. Ward, Marshall R., Pittsburgh, Pa.
- *1879. Ward, Whitfield, New York, N. Y.
- *1886. Westbrook, Benjamin F., Brooklyn, N. Y.
- *1924. Wherry, William P., Omaha, Neb.
- *1924. White, Leon E., Boston, Mass.
- *1939. Williams, Horace J., Philadelphia, Pa.
- *1942. Wishart, D. E. Staunton, Toronto, Canada.
- *1922. Wishart, David J. G., Toronto, Canada.
- *1940. Wood, V. Visscher, St. Louis, Mo.
- *1896. Wollen, Green V., Indianapolis, Ind.
- *1953. Wilkerson, William W., Nashville, Tenn.

Roster of Present Fellows

LIST OF FELLOWS

Date Indicates Year Admitted to Active Fellowship

Attendance at meetings is shown by figures following each name.

Asterisk (*) indicates year paper was read.

Active Fellows

1962. ALEXANDER, FRED W., 10515 Carnegie Ave., Cleveland, Ohio 44106, 1962, '63*, '64, '66, '68, '69, '70, '71.
1958. ALFARO, VICTOR R., 916 - 19th St., N. W., Washington, D.C. 20006, 1958, '59, '60, '61, '62, '63, '64, '65, '66, '67, '69, '70, '71.
1969. ANDREWS, ALBERT H. JR., 122 S. Michigan Ave., Chicago, Ill., 60603, 1970, '71.
1962. ARNOLD, GODFREY E., 2500 N. State St., Jackson, Mississippi 39216, 1962, '63*, '64*, '66, '69, '70.
1958. ATKINS, JOSEPH P., 3400 Spruce St., Philadelphia, Pa. 19104, 1958, '59*, '60, '61, '62, '63, '64, '65, '67, '68, '69, '70, '71*.
1969. AUSBAND, JOHN R., Bowman Gray School of Medicine, Winston-Salem, N. C. 27103, 1970, '71.
1956. BAKER, DANIEL CLIFTON, JR., 180 Fort Washington Ave., New York, N. Y. 10032, 1956, '57, '58, '59*, '60, '61, '62*, '63, '64, '65, '66, '69*, '70, '71*.
1970. BALLENGER, JOHN J., 723 Elm St., Winnetka, Ill. 60093, 1971.
1963. BAXTER, JAMES D., 1509 Sherbrooke St. West, Montreal, P.Q., Canada, 1963, '65*, '66, '67, '68, '69, '70, '71.
1944. BOIES, LAWRENCE R., Box 396, University Hospital, Minneapolis, Minnesota 55455, 1944, '46, '47, '48*, '49, '50*, '51*, '53, '54, '55, '56, '57, '58, '59, '60, '61, '62, '63, '64, '65, '66, '67, '68*, '69, '71.
1955. BORDLEY, JOHN E., Johns Hopkins Hospital, Baltimore, Maryland 21205, 1955, '56, '57, '59, '60, '62, '63, '64, '65, '66, '67, '69, '70, '71.
1959. BREWER, DAVID W., 1100 E. Genesee St., Syracuse, N. Y. 13210, 1959, '60*, '61, '62*, '63*, '66, '70.

1955. BROWN, LESTER ALEXANDER, 490 Peachtree Street, N.E., Atlanta, Georgia 30308, 1955, '56, '57, '58, '59, '61, '63, '65, '68, '69, '70, '71.
1940. BROYLES, EDWIN N., 11 East Chase St., Baltimore, Maryland 21202, 1940, '41*, '42, '46, '48*, '49, '50, '51*, '52, '53, '54, '55, '56, '58, '59*, '60, '61*, '63, '64, '65, '67, '71.
1963. BRYCE, DOUGLAS P., O.P.D. Toronto General Hospital, Toronto, Canada, 1963*, '64, '66*, '67*, '68*, '69, '70, '71.
1968. CHANDLER, J. R., 1700 N.W. 10th Ave., Miami, Fla. 33136, 1968, '69*, '70, '71.
1967. COCKE, EDWIN W., JR., 899 Madison, Memphis, Tenn. 38102, 1967, '68, '69, '70.
1964. CODY, CLAUDE C., III, 1600 Memorial Professional Building, Houston, Texas 77002, 1964, '65, '67, '69, '71.
1971. COHEN, SEYMOUR R., 6221 Wilshire Blvd., Los Angeles, Calif. 90048, 1971.
1957. CONLEY, JOHN J., 139 East 36th Street, New York, N. Y. 10016, 1958*, '60*, '61, '62*, '63*, '64, '65, '70.
1957. CONVERSE, JOHN MARQUIS, 722 Park Avenue, New York, N. Y. 10021, 1960, '62*, '67.
1959. CRACOVANER, ARTHUR J., 103 E. 78th St., New York, N. Y. 10021, 1959, '60, '61*, '62, '63, '65, '66, '67, '68, '69.
1960. DALY, JOHN F., Faculty Practice Offices, 566 First Ave., New York, N. Y. 10016, 1960, '61*, '62, '63, '64, '65, '66, '67, '68, '69, '70, '71.
1951. DAVISON, FRANCIS W., Geisinger Medical Center, Danville, Pa. 17812, 1951, '52, '53*, '54, '55, '56, '57, '58, '59, '60, '61, '62, '63*, '64, '65, '66, '67*, '68, '69*, '70, '71.
1966. DEVINE, KENNETH D., Mayo Clinic, Rochester, Minn. 55901, 1966, 1948*, '49, '51, '53, '54, '56, '57, '59, '62, '64, '65*, '66, '67, '68.
1968. DEWEESE, DAVID D., 3181 S.W. Sam Jackson Park Rd., Portland, Ore. 97202, 1968, '69*.
1969. DICKINSON, JOHN T., 1501 Locust St., Pittsburgh, Pa. 15219, 1970, '71.
1952. ERICH JOHN B., Mayo Clinic, 102 Second Avenue, Rochester, Minn. 55901, 1953, '56*, '57, '65.

1968. FARRIOR, RICHARD T., 509 Bay St., Tampa, Fla. 33606, 1968, '69, '70*.
1964. FEARON, BLAIR, 308 Medical Arts Building, Toronto 5, Ontario, Canada, 1964, '66, '67, '68, '69, '70, '71*.
1963. FERGUSON, CHARLES F., 300 Longwood Ave., Boston, Mass. 02115, 1963, '64*, '66, '67*, '68, '69*, '70, '71.
1955. FITZ-HUGH, GLASSELL SLAUGHTER, 2003 Spottswood Road, Charlottesville, Virginia 22903, 1955, '57, '59, '60, '61, '62*, '64, '65, '66, '67*, '68, '69, '70, '71.
1970. FRAZER, JOHN P., 260 Crittenden Blvd., Rochester, N. Y. 14620, 1971.
1956. FRIEDBERG, STANTON A., 1725 West Harrison St., Chicago, Ill. 60612, 1956, '57*, '58, '59, '60, '61, '62, '63, '64, '65*, '67, '68, '69, '70, '71*.
1959. GOLDMAN, JOSEPH L., 1050 Park Ave., New York, N. Y. 10028, 1959, '60*, '61, '62, '63, '64, '65, '66, '67, '68, '69, '70, '71.
1965. HANCKEL, RICHARD W., 80 Barre St., Charleston, S. C. 29401, 1965, '66, '67, '69, '70, '71.
1957. HARRILL, JAMES A., Bowman Gray School of Medicine, Winston-Salem, N. C. 27103, 1957, '58, '59, '61, '62, '63, '64, '66, '67, '68, '69, '71.
1960. HARRIS, HERBERT, 907 Hermann Professional Building, Houston, Texas 77025, 1960, '61*, '62, '63, '64, '65*, '66, '67, '68, '69, '70, '71.
1959. HENRY, G. ARNOLD, 170 St. George St., Toronto, Canada, 1959, '61, '63, '66, '67, '69, '70.
1943. HILDING, ANDERSON C., Research Laboratory, St. Luke's Hospital, Duluth, Minnesota 55805, 1944*, '46, '47, '48*, '49, '50, '51, '53, '55, '56*, '57, '60, '61, '62*, '63, '64*, '65, '66, '67, '68, '69, '70, '71.
1955. HILGER, JEROME A., Suite 6A, 393 Dunlap St., St. Paul, Minnesota 55104, 1955, '56*, '57, '58, '59, '60, '62, '63, '64, '65, '66, '68, '69, '70, '71.
1945. HOLINGER, PAUL H., 700 North Michigan Ave., Chicago, Illinois 60611, 1946, '47, '49, '50*, '51*, '52, '53, '54*, '55, '56, '57, '58*, '59, '60, '61*, '62*, '63, '64, '65, '66*, '68, '69*.
1957. HOLMES, EDGAR M., 2000 Washington St., Newton, Mass. 02162, 1957, '59, '61, '63, '68, '70, '71.

1961. JONES, EDLEY H., 1301 Washington St., Vicksburg, Miss. 39180, 1961, '62, '63*, '65, '66, '67, '68, '69, '70, '71.
1964. KEIM, W. FRANKLIN, 75 Union St., Montclair, N. J. 07042, 1964, '66, '67, '68*, '69, '70, '71.
1965. KING, JAMES T., Baptist Professional Building, Atlanta, Ga. 30312, 1965, '66, '67, '69, '70, '71.
1963. KIRCHNER, JOHN A., 333 Cedar St., New Haven, Conn. 06510, 1963, '65*, '67*, '68, '69*, '70, '71*.
1965. KNIGHT, JOHN S., 305 West 43rd St., Kansas City, Mo. 64111, 1965, '66*, '67, '69, '70, '71.
1963. LATHROP, FRANK D., R.F.D. No. 1, Pittsford, Vt. 05763, 1963, '64*, '67*, '69, '70, '71.
1953. LEDERER, FRANCIS LOEFFLER, 1300 Lake Shore Drive, Apt. 32D, Chicago, Illinois 60610, 1953, '54, '55, '56, '57*, '58, '60, '61, '62, '63*, '64, '65, '66, '68*, '69, '70.
1966. LEWIS, JOHN S., 3 East 71st Street, New York, N. Y. 10021, 1966, '67, '68, '69*, '70, '71.
1961. LEWY, ROBERT B., 25 East Washington St., Chicago, Ill. 60602, 1961, '62*, '63, '65*, '67, '68, '69, '70, '71.
1966. McCABE, BRIAN F., Dept. of Otolaryngology, University Hospitals, Iowa City, Iowa 52240, 1966, '69.
1963. McGOVERN, FRANCIS H., 139 South Main St., Danville, Virginia 24541, 1963, '64, '66, '68, '71.
1951. McHENRY, LAWRENCE C., 301 N. W. 12th St., Oklahoma City, Okla. 73103, 1953*, '62, '63, '64, '65, '66, '68, '71.
1952. MILLER, ALDEN H., 1407 N. Vermont Ave., Los Angeles, Calif. 90027, 1952, '53*, '55, '57, '58*, '61, '62, '64, '66, '67*, '68, '69, '70, '71.
1965. MILLER, DANIEL, Deaconess Medical Building, 110 Francis St., Boston, Mass. 02215, 1965, '66, '67, '68, '69*, '70, '71.
1964. MONTGOMERY, WILLIAM W., 243 Charles St., Boston, Mass. 02114, 1964, '65*, '66*, '68*, '70, '71.
1958. MONTREUIL, FERNAND, Hospital Notre Dame, 1560 E. Sherbrooke, Montreal 24, Canada, 1958, '60, '64, '67, '69.

1957. MUNOZ-MACCORMICK, CARLOS EDUARDO, 71 Park Terrace, Santurce, Puerto Rico, 1957, '58, '59, '60, '61, '62, '63, '64, '65, '66, '67, '68, '69, '70, '71.
1956. NORRIS, CHARLES MORGAN, 3401 North Broad Street, Philadelphia, Pennsylvania 19140, 1956, '57, '58*, '59, '60, '61*, '62, '63, '64, '66, '67, '68*, '69, '70*, '71.
1961. OGURA, JOSEPH H., 640 So. Kingshighway, St. Louis, Mo. 63110 1961*, '62*, '63, '64*, '65, '66*, '67, '68*, '69, '70, '71*.
1958. O'KEEFE, JOHN J., 255 S. 17th St., Philadelphia, Pa. 19103, 1958, '59*, '60, '62, '64, '67, '71.
1961. O'NEIL, JAMES J., 612 Medical Arts Building, Omaha, Nebraska 68102, 1961, '62*, '64, '67, '69, '70, '71.
1961. PANG, LUP Q., 1374 Nuuanu Ave., Honolulu, Hawaii 96817, 1961, '62*, '64, '66, '67, '70, '71.
1961. PASTORE, PETER N., 5503 Riverside Drive, Richmond, Va. 23225, 1961, '62*, '64, '66, '67, '69, '71.
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