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Vocal Cord Paralysis

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UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS

Visitors Welcome

November 8 - 13, 1948

No. 221

Monday, November 8

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; M-109, U. H.

8:00 - Fracture Rounds; A. A. Zierold and Staff; Ward A, Minneapolis General Hospital.

10:00 - 12:00 Neurology Ward Rounds; A. B. Baker and Staff; Station 50, U. H.

11:00 - 11:50 Roentgenology-Medicine Conference; Staff, Veterans' Hospital.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Eustis Amphitheater, U. H.

11:00 - 11:50 Physical Medicine Seminar; E-101, U. H.

12:00 - 1:00 Physiology Seminar; M. B. Visscher; 214 M. H.

12:15 - 1:20 Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

12:30 - 1:20 Pathology Seminar; 104 I. A.

12:30 - 1:50 Surgery Grand Rounds; A. A. Zierold, Clarence Dennis and Staff; Minneapolis General Hospital.

1:00 - 2:00 Kellogg Lecture; The Vitamins; Russell M. Wilder, Mayo Clinic; Eustis Amphitheater, U. H.

2:00 - 3:00 Kellogg Lecture; The Vitamins; Russell M. Wilder, Mayo Clinic; Powell Hall Amphitheater.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.

2:00 - 3:00 Surgery Problem Case Conference; C. Dennis and Staff; Small Class Room, General Hospital.

3:45 - Pediatric Seminar; Immunization Against Tuberculosis with B.C.G.; W. Anderson; 6th Floor, Child Psychiatry, U. H.

4:00 - 6:00 School of Public Health Seminar; 113 MeS.

5:00 - 6:00 Urology-Roentgenology Conference; D. Creevy and H. M. Stauffer and Staffs; M-109, U. H.

5:00 - 5:50 Clinical Medical Pathologic Conference; Todd Amphitheater, U. H.

8:00 - 9:00 Clinical Research Club; Eustis Amphitheater, U. H.

Tuesday, November 9

8:30 - 10:20 Surgery Seminar; Lyle Hay; Small Conference Room, Bldg. I, Veterans' Hospital.

9:00 - 9:50 Roentgenology Pediatrics Conference; L. G. Rigler, I. McQuarrie and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Surgical Pathological Conference; Lyle Hay and Robert Hebbel; Veterans' Hospital.

12:30 - 1:20 Pathology Conference; Autopsies; Pathology Staff; 102 I. A.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III, Veterans' Hospital.

2:00 - 3:00 Kellogg Lecture; Physiology of Starvation; Powell Hall Amphitheater.

3:15 - 4:20 Gynecology Chart Conference; J. L. McKelvey and Staff; Station 54, U. H.

3:30 - 4:20 Clinical Pathological Conference; Staff; Veterans' Hospital.

4:00 - 5:30 Surgery-Physiology Conference; O. H. Wangensteen and M. B. Visscher; Eustis Amphitheater, U. H.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

5:00 - 5:50 Urology Pathological Conference; C. D. Creevy and Staff; Todd Amphitheater, U. H.

5:00 - 6:00 X-ray Conference; Dr. Rigler and Staff; Powell Hall Amphitheater.

Wednesday, November 10

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-515, U. H.

8:30 - 10:00 Orthopedic-Roentgenologic Conference; Edward T. Evans; Room 1AW, Veterans' Hospital.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker and Joe R. Brown; Veterans' Hospital.

11:00 - 12:00 Pathology-Medicine-Surgery Conference; O. H. Wangensteen, C. J. Watson and Staff; Todd Amphitheater, U. H.

12:00 - 12:50 Radio Isotope Seminar; Current Literature; Richard Bridenbaugh; Rm. 216, Hospital Court, Temporary Bldg.

4:00 - 5:00 Infectious Disease Rounds; Powell Hall Amphitheater, University Hospitals.

Thursday, November 11 - HOLIDAY

Friday, November 12

8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U.H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:20 Medicine Grand Rounds; Staff; Veterans' Hospital.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

11:00 - 12:00 Surgery-Pediatric Conference; C. Dennis, A. V. Stoesser and Staffs; Minneapolis General Hospital.

11:30 - 12:50 University of Minnesota Hospitals General Staff Meeting; Evaluation of Laboratory Tests in the Differential Diagnosis of Jaundice; C. J. Watson, F. W. Hoffbauer, Eugene D. Rimes and John K. Meinert; Powell Hall Amphitheater.

12:00 - 1:00 Surgery Clinical Pathological Conference; Clarence Dennis and Staff; Minneapolis General Hospital; Small Classroom.

1:00 - 1:50 Dermatology and Syphilology; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

2:00 - 3:00 Kellogg Lecture; Radiation Therapy of Metabolic Disorders; K. W. Stenstrom; Eustis Amphitheater, U. H.

Saturday, November 13

7:45 - 8:50 Orthopedics Conference; Wallace H. Cole and Staff; Station 21, U. H.

8:00 - 9:00 Pediatric Psychiatric Rounds; Reynold Jensen; 6th Floor, West Wing, U. H.

8:00 - 9:00 Surgery Literature Conference; Clarence Dennis and Staff; Minneapolis General Hospital, Small Classroom.

9:00 - 9:50 Surgery-Roentgenology Conference; O. H. Wangensteen, L. G. Rigler, H. M. Stauffer, and Staff; Todd Amphitheater, U. H.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; E-101, U. H.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater, U. H.

9:00 - 12:00 Neurology Conference; VA Hospital Annex, Fort Snelling.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

11:00 - 11:50 Urology Seminar; Perineal Prostatectomy. Richard Rodgers; E-101, U. H.

11:00 - 12:00 Anatomy Seminar; The Arrangement of the Cells of the Nucleus Ruber in the Cat; Ronald A. Dolan; 226 I. A.

II. VOCAL CORD PARALYSIS

Harold S. Ulvestad
L. R. Boies

Introduction

Vocal cord paralysis is both a symptom and a disease. As a symptom it is often a reflection of trouble elsewhere in the body. As a disease its most common manifestation is hoarseness. Hoarseness has been defined as a rough, harsh, quality of voice which is lower in pitch than is normal for an individual. (Jackson)

Incidence

We do not know how often vocal cord paralysis occurs in the general population. However, it is seen frequently in our Ear, Nose and Throat Clinic. Many reports are available on the various causes of vocal cord paralysis and the relative frequency with which each type occurs.

Etiology

A large number and variety of lesions can be responsible. The long and exposed courses of the recurrent laryngeal nerves, in the neck and within the mediastinum, render them more liable to injury from pressure, stretching, trauma and inflammation. Jackson has observed that paralysis would not have occurred in half of his cases if the inferior laryngeal nerve had passed directly to the larynx from the vagus nerve. The left cord is paralyzed about twice as often as the right, because of the more frequent involvement of the left recurrent nerve by mediastinal lesions. (New-Childrey). The more common causes of vocal cord paralysis are listed. Three only produce left sided and two right sided paralysis. (Boies). The rest may affect either side.

On the left:

1. Aneurysm of the arch of the aorta
2. Enlargement of the left auricle
3. Pericarditis

On the right:

1. Pleural thickening at the apex of the right lung
2. Aneurysm of the innominate artery

On either side:

1. Enlarged cervical or bronchial glands
2. Goiter
3. Diseases of the mediastinal tissue or glands: carcinoma, sarcoma, tuberculosis, syphilis, etc.
4. Direct traumatic injury in surgery of the neck
5. Intracranial growth at the base of the brain, or a growth in the neck involving the vagus at its exit from the skull
6. Cervical phlegmon
7. Aneurysm of the carotid artery
8. Large pleural effusions
9. Bulbar lesions
10. Peripheral neuritis

Three series, reported by Work, Suehs, and New and Childrey, and totaling 744 cases of vocal cord paralysis have been combined. Their findings are as follows:

1. 10% of the causes are central in origin. Most of these are lesions of the bulb, including tabes, bulbar palsy, amyotrophic lateral sclerosis, polioencephalitis, thromboses of the posterior inferior cerebellar artery.

2. 90% are due to peripheral lesions. Of these, 24% followed thyroid surgery. One sixth of the post thyroidectomy group involved both cords, making thyroidectomy the most common cause of bilateral midline paralysis. Injury to one or both of the recurrent nerves may be the result of actual cutting of the nerve, or of postoperative hemorrhage, swelling or scar tissue formation. Spontaneous recovery, usually within six months, may be expected for 25% of these cases. Jackson believes an overlooked preoperative unilateral paralysis to be present in most cases considered to be postoperative, emphasizing the importance of examining the larynx prior to surgery.

3. Tumors comprise the largest group

among the causes of vocal cord paralysis. New reported 6% in the nasopharynx, all unilateral and all producing the jugular foramen syndrome. The tumor was located in the hypopharynx and esophagus in 11%. Malignant disease of the esophagus is reported to be the most common cause of paralysis of the right cord.

Types

There are five main types of vocal cord paralysis. These are, in the order of frequency of occurrence:

1. Unilateral midline paralysis
2. Unilateral incomplete paralysis
3. Bilateral midline paralysis
4. Bilateral incomplete paralysis
5. Complete paralysis

The larynx is an extremely complex mechanism. A detailed knowledge of its anatomy is necessary for a proper understanding of its normal function and for a diagnosis of the type of paralysis that may be present. For practical purposes the cartilaginous framework, the intrinsic muscles and the innervation of the larynx will be reviewed briefly.

The larynx is a box-like structure with a framework made up of three paired and three unpaired cartilages. The most important, functionally, are the thyroid cartilages, the cricoid, shaped like a signet ring, and the right and left arytenoid cartilages. Each inferior cornu of the thyroid articulates with the cricoid on its postero-lateral surface. This cricothyroid joint is a true joint which permits a rocking action of the two cartilages, with respect to each other. The arytenoids are mounted on the "signet" or posterior lamina of the cricoid. The crico-arytenoid joint is a true joint which permits a lateral gliding movement of the arytenoid cartilage away from the midline and a rotatory movement around a vertical axis. Two processes on each arytenoid are important. The anteriorly directed vocal process forms the posterior attachment for the vocal cord. The muscular process, directed laterally, forms the attachment for the adductor and abductor muscles of the larynx. All of the intrinsic muscles of the larynx, except the cricothyroid, are attached to the arytenoid. The remaining cartilages are the unpaired cartilage of the epiglottis, attached to the inner surface of the thyroid angle; the right and left corniculates mounted on the arytenoids, and the right and left cuneiforms extending laterally in the ary-epiglottic folds.

The intrinsic muscles of the larynx may be divided into three functional groups: abductors, adductors and tensors. These muscles act through either the crico-thyroid or the crico-arytenoid joints.

The Abductors are the paired posterior crico-arytenoid muscles. They arise from the posterior surface of the cricoid lamina and insert on the posterior surfaces of the muscular processes of the arytenoid cartilages. By rotating the vocal processes laterally they separate the cords. The lateral fibers, along with some from the lateral crico-arytenoid muscles slide the arytenoids away from the midline.

The Adductors are the paired lateral crico-arytenoid and the unpaired transverse arytenoid muscles. The lateral crico-arytenoid muscles arise from the posterior halves of the superior borders of the cricoid arch and insert in the anterolateral surface of each muscular process. Their action rotates the vocal processes medially and approximates the cords. The transverse arytenoid muscle inserts on the medial surface of each arytenoid cartilage and pulls them together in the midline. A superficial oblique segment of this muscle runs on up in the ary-epiglottic fold to the epiglottis. It has a sphincteric action, tending to close the laryngeal entrance.

The Tensors are the paired cricothyroid and thyro-arytenoid muscles. The cricothyroid muscle arises from the antero-lateral aspect of the cricoid arch and inserts into the postero-inferior border of the thyroid lamina and in-

to the inferior cornu. Their action elevates the cricoid arch. The result is a tipping of the cricoid lamina posteriorly and a putting of the vocal cord on stretch. The thyro-arytenoid muscle arises from the inner surface of the thyroid angle and inserts in the vocal process of the arytenoid. Its action is in a direction opposite to that of the crico-thyroid muscle, and, if unopposed, would relax or shorten the vocal cords. Some of the more lateral fibers of the thyroarytenoid muscle insert into the muscular process and thus act as adductors.

Innervation. The superior laryngeal branch of the vagus nerve supplies sensory innervation to the larynx through its internal branch. In addition, it probably gives some motor supply to the transverse arytenoid muscle. The external branch supplies the cricothyroid muscle. All the other intrinsic muscles of the larynx are innervated by the recurrent branch of the vagus nerve. The motor nerves of the larynx arise in the paired nuclei ambigui of the medulla. Each nucleus ambiguus receives corticobulbar fibers from both the right and the left motor cortex. For this reason, a lesion above the nucleus ambiguus does not produce a unilateral paralysis of the larynx. If it is large enough to involve the corticobulbar fibers of both sides it produces a bilateral laryngeal paralysis of spastic type. Such a paralysis is rare. A nuclear or infra nuclear lesion produces a flaccid type of paralysis which may be unilateral or bilateral. (Furstenberg). Lesions in the region of the jugular foramen or just distal to it may also involve the glossopharyngeal, spinal accessory and hypoglossal nerves and produce various combinations of cranial nerve palsies.

In a discussion of vocal cord paralysis mention should be made of the Semon Law. Semon believed that a larynx which has become completely paralyzed, including a loss of tonus, as the result of an organic lesion, arrives at such a stage through a definite sequence of changes. First the abductor muscles are paralyzed, next the "tensors", then the adductors and finally the tonus. This sequence does occur frequently but there are enough exceptions so that Jackson prefers to refer to it as Semon's rule rather than law. The reason for this decreased resistance of the abductors is not clear. One theory is that the opening muscles of the larynx are much younger in evolutionary progress than the closing muscles or adductors.

Mention should also be made of the term "cadaveric position". This refers to the position which the vocal cords are believed to assume after death. This position is between that of abduction and of adduction. The term signifies a complete paralysis and as such it means that all of the intrinsic muscles of the larynx are involved and all tonus is lost. Not infrequently the position of the vocal cords has been described as "cadaveric" when the paralysis is either a unilateral incomplete or a bilateral incomplete type. Because of the confusion that has resulted through its incorrect use Jackson believes that the term "cadaveric position" should be dropped.

Unilateral Midline Paralysis affects only the posterior crico-arytenoid muscle on one side. The involved cord is held firm and in the midline by the action of the crico-thyroid, the lateral crico-arytenoid and the thyro-arytenoid muscles during inspiration and phonation. On inspiration the normal cord swings laterally and, at the same time, the arytenoid prominence on the paralyzed side tips forward. As a result the arytenoid appears larger than normal and may be mistaken for a tumor. When the normal cord swings medially on phonation the tipped arytenoid is jostled back into position again by the impact of the active arytenoid. This is an important diagnostic sign. In addition the glottic slit is slightly askew on phonation. The cause may lie anywhere along the course of the laryngeal neurones, from the nucleus ambiguus on down the neck into the mediastinum and back up again to the larynx. The lesion usually involves the recurrent nerve, and more frequently the left recurrent. Except for a transient hoarse-

ness there are no symptoms.

Unilateral Incomplete Paralysis involves all of the muscles of one side except the transverse arytenoid. The latter muscle is bilaterally innervated and therefore is not affected. The paralyzed cord remains stationary midway between phonation and inspiration. Its edge is concave and may be displaced upward by the expiratory blast. On inspiration the glottic silhouette is paraboloid. On phonation the normal cord crosses the midline to meet the disabled cord, causing a jostling of the sound arytenoid. The voice is husky at first but improves with the compensatory adjustments that are made by the uninvolved muscles. The lesion is usually in the vagus nerve above the origin of the superior as well as the inferior laryngeal nerves.

Bilateral Midline Paralysis involves both posterior crico-arytenoid muscles, resulting in a loss of abduction. The cords remain in the midline and the larynx is paralyzed shut. (Jackson) There is usually a marked obstruction to inspiration, producing an inspiratory stridor. Occasionally one cord is at a higher level than the other, leaving a chink between which is invisible on mirror examination but which provides an increased airway. Obstruction may be severe enough to necessitate a tracheotomy to prevent asphyxia. The voice is usually good because the cords are in approximation on phonation.

Midline paralysis may be simulated by a fixation of the crico-arytenoid joints in a midline position. The differentiation may be made through the direct laryngoscope by performing the passive mobility test--pushing on the arytenoids with a blunt probe. Treatment is aimed at creating an adequate airway and still preserving a useful voice. This will be discussed later.

Bilateral Incomplete Paralysis involves all of the intrinsic muscles except the transverse arytenoid. The vocal cords lie motionless in a position halfway between phonation and inspiration. The cord edges are symmetrically concave. The glottic chink is ellipsoidal in outline and changes little or not at all during phonation, inspiration or expiration. The voice is husky and air waste is marked. The patient has a wheezy cough and has difficulty in expelling secretions from the tracheobronchial tree. Casual observation might lead one to suspect asthma. Goiter surgery and syphilis are the most common causes of this type of paralysis. Not infrequently the cords gradually shift to a midline position, respiration becomes obstructed and a tracheotomy has to be performed. Occasionally a spontaneous cure takes place.

Complete Paralysis of the Larynx involves all of the intrinsic muscles. In addition, all tonus is lost. The glottic silhouette is ellipsoidal anteriorly and triangular posteriorly between the arytenoids. All movement is lost and the whole appearance has been described by Jackson as "wooden". The causative lesion usually but not always lies above the branching off of the superior laryngeal nerve from the vagus. It involves the nerve supply to both sides of the larynx. There is no local treatment and no improvement can be expected. Pulmonary complications are the rule because of the patient's inability to get rid of tracheobronchial accumulations.

Besides the five just described there are three additional types of vocal cord paralysis. They are rarely seen as distinct clinical entities but frequently occur in association with the others. They are: adductor paralysis, thyroarytenoid paralysis and crico-thyroid paralysis.

Adductor paralysis occurs in two forms. If the transverse arytenoid is not affected the glottis closes posteriorly on phonation. An aphonic voice and air waste is present. If only the transverse arytenoid muscle is paralyzed phonation produces a triangular glottic chink posteriorly and the voice remains good. As a sole condition adductor paralysis requires a supranuclear lesion which in-

volves the fibers of both sides.

Thyro-arytenoid paralysis, if it is unilateral, produces a moderate bowing of the involved cord on phonation and a low pitched hoarse voice. If bilateral, the arytenoids approximate on phonation but a glottic chink is present anteriorly. Hoarseness and a low voice is more marked than in the unilateral condition.

Crico-thyroid paralysis may affect one or both cords. Abduction and adduction is normal but the involved cord margins are wavy or wrinkled because of the unopposed pull of the thyro-arytenoid muscle. The voice is weak and rough, and the respiration wheezy, especially if the paralysis is bilateral.

Abductor paralysis in the newborn is not uncommon but it is often overlooked. Practically all patients who survive recover perfect motility and laryngeal function, indicating that the cause was either a bulbar circulatory disturbance or pressure on nerve trunks. Jackson noted that forceps were used in the delivery in nearly all of his cases. Intense dyspnea, cyanoses and stridor are present if the paralysis is bilateral. If unilateral, stridor is less severe and the cry is hoarse. Diagnosis requires direct laryngoscopy. Treatment is aimed at preventing asphyxia until normal cord function returns. Gentle insufflation of an oxygen-carbon dioxide mixture through a bronchoscope or catheter may be necessary. Occasionally tracheotomy is required. (Jackson)

Diagnoses. Work has listed the symptoms of vocal cord paralysis in order of their frequency of occurrence as follows:

No symptoms	30%
Hoarseness	43%
Weakness, tiredness of voice	10%
Dyspnea	8%
Strangling on foods	6%
Laryngeal stridor	1%

In the great majority of cases a diagnosis of vocal cord paralysis can be made simply by an examination with the laryngeal mirror. In a small per cent direct laryngoscopy will be necessary to adequately visualize the glottis as well as to rule out by palpation a crico-arytenoid joint fixation. Once a paralysis has been found, the cause should be determined. To do this may require a very complete history and general physical examination, neurologic examination, serology tests, roentgen-ray examination of the head, neck and chest, including an esophogram. It should include an inspection of the nasopharynx, palpation of the laryngeal cartilages, a search for tenderness or a lesion along the course of the four laryngeal nerves. Bronchoscopy or esophagoscopy may reveal the presence of a tumor involving one or both of the recurrent nerves. In spite of all this, the examiner will fail to find the causative lesion in one case out of every three!

Therapy. There is no local treatment of any value for any of the types of paralysis described, with the exception of bilateral midline or abductor paralysis. For the others, therapy, if indicated, is directed against the causative agent. Satisfactory surgical procedures have been developed, however, for the treatment of bilateral midline paralysis. Many of them are modifications of the original King operation. Essentially they consist of the removal or the fixation of one arytenoid cartilage against the lateral wall of the larynx. This is accomplished through an external approach. An adequate airway may thus be provided between the posterior portion of the vocal cords, leaving the anterior portion for phonation. By this method a useful voice is preserved.

The Woodman modification has been used at the University Hospitals with good results. In each case, if the patient has not already been tracheotomized, a tracheotomy is performed either before or immediately after the laryngeal surgery. A skin incision is then made anterior to the sternocleidomastoid muscle from the level of the upper edge of the thyroid cartilage to the level of the cricoid cartilage. The sternocleidomastoid muscle is retracted and the posterior edge of the thyroid lamina is exposed. The attachment of the inferior cornu to the

cricoid cartilage is an important landmark. A vertical incision is made through the perichondrium of the thyroid lamina along its posterior border and the inferior constrictor muscle separated, both posteriorly and from its medial attachments on the inner surface of the cartilage. The crico-thyroid joint is disarticulated. An incision is then made through the perichondrium on the lateral wall of the cricoid cartilage and extended in the direction of the arytenoid cartilage. The latter is dissected subperichondrially and the crico-arytenoid joint disarticulated. A traction ligature is placed around the arytenoid cartilage, and, by rotating it laterally the vocal process is exposed. A chromic catgut suture is passed around the vocal process submucosally and through some of the fibers of the thyro-arytenoideus muscle near its attachment to the process. All of the arytenoid cartilage but the vocal process is removed, the vocal cord drawn laterally and tied to the inferior cornu of the thyroid cartilage. This, in turn, is anchored to the edge of the sternocleidomastoid muscle. An inspection of the larynx is then made with the direct laryngoscope to determine the position of the vocal cord. The wound is closed, leaving a Penrose drain in the lower end. A glottic chink 3 or 4 mm. wide is adequate for an airway and still leaves a good voice. Increasing the space beyond 6 mm. decreases the quality of the voice.

This operation has been performed in the University Hospitals on 11 cases in the past four years with good results. In ten of these cases a unilateral operation was adequate. One case required bilateral operation in order to provide an adequate airway.

The case histories are briefly summarized as follows:

Mrs. . Age 38

This patient gave a history of previous thyroid surgery on two occasions, the last one about a year previous to this operation. The resulting bilateral midline paralysis necessitated a tracheotomy. A right arytenoidectomy was performed on March 10, 1944, after the technique of Orton (removal of posterior third of thyroid lamina).

Mrs. . Age 48

A thyroidectomy in 1925 was followed by a complete loss of voice for 3 months. Her voice gradually improved but she complained of increasing difficulty in breathing. Admission examination on March 6, 1945 revealed a bilateral midline paralysis. A tracheotomy was performed on March 7, 1945. This was followed in ten days by a right arytenoidectomy, after the method of Orton.

Mrs. Age 59

This patient developed a hoarse, wheezing voice following thyroidectomy in 1942. Admission examination revealed a bilateral midline paralysis with the left cord at a slightly higher level than the right. A tracheotomy was performed on July 20, 1945 and was followed by a right arytenoidectomy on August 3, 1948.

Mrs. Age 74

This patient was operated on for goiter on two occasions about 20 years prior to admission. Difficulty in breathing followed the second procedure. In January, 1945, she developed a laryngitis which increased her respiratory difficulty. Examination on admission on August 5, 1945 revealed a bilateral midline paralysis and a moderate stridor on exertion. A tracheotomy was performed on August 6, 1945 and was followed in ten days by a lateral fixation of the right vocal cord.

Mrs. Age 58

Thyroid surgery in February, 1945 was followed by difficulty in breathing. Examination on October 4, 1945 revealed a bilateral abductor paralysis with a small chink through the midline. A tracheotomy was performed on the following day. Ten days later a lateral fixation of the right arytenoid cartilage was accomplished.

Mr. Age 51

Thyroid surgery on two occasions resulted in a left midline paralysis following the first and a right midline paralysis following the second procedure. During the course of the second operation the patient suddenly became obstructed as a result of injury to the right recurrent nerve. An immediate tracheotomy was necessary. On January 9, 1946 a left arytenoidectomy was performed, after the technique of Orton.

Mis Age 45

Difficulty in breathing as a result of a thyroidectomy had been present for several years prior to admission. A bilateral midline paralysis was noted. A Woodman type right arytenoidectomy was performed on July 9, 1947.

Mrs. Age 61

The patient complained of hoarseness after thyroid surgery. Six months later she began to have difficulty in breathing. A diagnosis of asthma was first made. Examination on December 15, 1947 revealed an inspiratory stridor and a bilateral midline paralysis. Her voice was good. The following day an emergency tracheotomy became necessary. On December 23 a right arytenoidectomy was performed, after the technique of Woodman.

Mr. Age 71

This patient complained of difficulty in breathing ever since he sustained a head injury in 1928. He continued to work in the iron mines until shortly before admission. At that time a head cold was followed by marked laryngeal obstruction which necessitated an emergency tracheotomy by the referring surgeon. A bilateral midline paralysis was noted on admission April 20, 1948. On the following day a Woodman type right arytenoidectomy was performed.

Mrs. Age 38

Gradually increasing respiratory obstruction followed thyroid surgery one year previously. Admission examination on May 25, 1948 revealed a bilateral midline paralysis. On May 26 a Woodman type procedure was done on the right side. She continued to have some stridor after decannulation, especially at night. The patient was a stocky obese woman with a small larynx. A second operation was performed on the left side on July 21, 1948 and the patient was decannulated a month later. She has an adequate airway and a fair voice.

Summary

1. Vocal cord paralysis is relatively common.
2. It may reflect serious disease elsewhere in the body.
3. Carcinoma along the course of the nerve supply to the larynx is the most common cause of a one-sided paralysis.
4. Goiter surgery is the most common cause of a bilateral paralysis.
5. No causative lesion can be found in approximately one-third of the cases.
6. Bilateral midline paralysis is characterized by a good voice and severe respiratory obstruction.
7. An arytenoidectomy, or lateral fixation of an arytenoid cartilage, creates an adequate airway and preserves a good voice.

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Archives of Otolaryngology, 34:267, August, '41.

III. MEDICAL SCHOOL NEWS

Progress of Minnesota Medical Foundation

The alumni of the Minnesota Medical School may feel proud of their achievements to date in the Minnesota Medical Foundation. At the annual meeting held on Friday, October 22, at the Campus Club, it was revealed that receipts since the Foundation was organized in October 1939, totaled $91,660. Membership dues make up almost $37,000 of this total. Grants from individuals, voluntary organizations, scientific societies, and industry total almost $50,000. Future issues of the Bulletin will tell the story of some of these grants. Disbursements of the Foundation since its organization in 1939, total $80,840. The total assets of the Foundation at present amount to $37,016.

A drive for new members, which was conducted in October, has already resulted in the enrollment of 80 new members. Members now receive the "Bulletin of the University of Minnesota Hospitals and Minnesota Medical Foundation," in addition to their participation in the affairs of the Foundation.

Trustees elected at the recent meeting of the Foundation included the following:

For four years (1952):
Dr. Russell J. Moe, 205 W. 2nd St., Duluth, Minn.
Dr. William A. Hanson, 1005 Med. Arts Bldg., Minneapolis, Minn.
Dr. Charles F. Code, Mayo Foundation, Rochester, Minn.

For three years (1951):
Dr. Lloyd H. Rutledge, Detroit Lakes, Minn.
Dr. Edwin J. Simons, Swanville, Minn.
Dr. William W. Will, Bertha, Minn.

For two years (1950):
Dr. Wesley W. Spink, 221E, Univ.Hospital
Dr. Maurice B. Visscher, 318 Millard Hall
Dr. Owen H. Wangensteen, 201E University Hospital

For one year (1949):
Dr. George Earl, 1210 Lowry Med. Arts Bldg., St. Paul, Minn.
Dr. Erling S. Platou, 953 Med. Arts Bldg., Minneapolis, Minn.
Dr. Karl Anderson, N.W. Nat. Life Ins. Co., 430 Oak Grove, Minneapolis

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Dr. Hunter Visits Medical School

Dr. Warren C. Hunter, Professor of Pathology at the University of Oregon Medical School will be Dr. Wangensteen's guest at the surgery conference from 9 to 12 a.m., Saturday, Nov. 6. He will speak on the subject of "Diagnosis of Cancer by Smear Technics." All physicians are cordially invited.

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Dr. E. R. Hayes of the Department of Medicine and Dr. R. N. Holly of the Department of Obstetrics and Gynecology journeyed to Willmar, Minnesota, on November 1 to speak at the staff meeting of the Rice Memorial Hospital in that city. Dr. Hayes spoke on the subject of "Lymphoblastomas." Dr. Holly's subject was "Adenocarcinoma of the Endometrium."

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Correction

In last week's Bulletin, the name of Dr. E. L. Tuohy should have appeared beneath the first letter regarding Dr. Litzenberg. We regret this omission and wish to acknowledge Dr. Tuohy's contribution.

Alumni Notes

Dr. Russell Joseph Kotval (1946 M.D.) has located in Pipestone, Minnesota, where he will practice medicine at new offices over the Gamble Store there. Dr. Kotval and his wife, the former Maxine Helen Knutila (1942 G.N.) have been living at Fayetteville, Arkansas, where Dr. Kotval was with the Veterans Hospital. They are now residing at 214 - 3rd Ave. S.E., Pipestone.

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Dr. Louis C. Jensen, Jr., (1945 M.D.) is working on a fellowship in internal medicine at the Veterans' Hospital at Coral Gables, Florida. His wife, the former Joy Sunderman (1946 Md.T.), is an anesthetist at the Jackson Memorial Hospital in Miami. Dr. Jensen is the son of Louis C. Jensen (1914 Phm.B.) and Mrs. Jensen. They are residing at 301 SW 21st Road, Miami.

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Dr. Leonard T. Peterson (1932 M.D.) has been promoted from Clinical Instructor in Surgery to Assistant Clinical Professor of Orthopedic Surgery at George Washington University, Washington, D.C. Dr. and Mrs. Peterson (Gretchen Albrecht '29Ed.) reside at 3378 Stephenson Place, Washington, D.C.

Dr. Gardner S. Reynolds (1924 M.D.) died recently in St. Paul. He was fifty years of age. Dr. Reynolds was an x-ray and radiology specialist and was head of the x-ray department of St. Elizabeth's Hospital, Danville, Illinois, for many years. He resigned in April due to ill health. He began his medical practice at Sacred Heart Hospital, Eau Claire, Wisconsin, and in 1927 he joined the staff of Henry Ford Hospital in Detroit.

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Dr. Andrew B. Rivers died recently in Rochester, Minnesota. He was 54 years of age. Dr. Rivers had for years been interested and actively working in the field of gastroenterology. He was well known for his work at the Mayo Clinic. Dr. Rivers was a member of Sigma Xi and Phi Beta Pi fraternities, the American Medical Association, and the American Gastroenterological Association.

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William L. Hoffman (1904 M.D.), retired member of the Army Medical Corps, died in Washington in October. He was 69 years of age.

Kellogg Foundation Lectures

The following lectures will be given during the week of November 8. All medical students, interns, nurses, technicians, dietitians, and physicians are cordially invited to attend these lectures. A special invitation is extended to University Fellows.

Dr. Russell M. Wilder (Mayo Clinic)	The Vitamins	Monday, November 8, 1:00-2:00 p.m. Eustis Amph. 2:00-3:00 p.m. Powell Hall Amphitheater
Dr. Ancel Keys	Physiology of Starvation	Tuesday, Nov. 9, 2:00 p.m., Eustis Amphitheater, U. H.
Dr. K. W. Stenstrom	Radiation Therapy of Metabolic Disorders	Friday, November 12, 2:00-3:00 p.m., Eustis Amphitheater, U. H.