

Paparella: Volume I: Basic Sciences and Related Principles

Section 2: Physiology

Part 2: Head and Neck

Chapter 13: Physiology of the Larynx

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The primary function of the larynx is to act as a sphincter that prevents the entrance of anything but air into the lung. Comparative anatomic studies indicate that this is as true of the highly refined larynx of humans as it is of the primitive larynx of the lungfish. Even in humans, phonation remains a secondary, although highly refined, function of the larynx.

Comparative Anatomy and Physiology

The most primitive larynx is found in the lungfish (order Dipnoi), a species inhabiting rivers that periodically become dry. As a protection for the lungs against the entrance of water, a simple circular group of muscle fibers constitutes a sphincteric band at the upper end of the trachea. When the sphincter is closed, the lower respiratory tract is isolated from the upper, so that ingested water cannot invade the lungs (Negus, 1949).

In this primitive larynx, consisting only of constricting fibers, dilation is brought about merely by relaxation of the muscle. In higher forms of lungfish, a separate group of dilator fibers appears, passing laterally from each side of the sphincter muscle (Lemere, 1934). These are eventually represented in humans as the posterior cricoarytenoid muscle.

The sphincteric closure of the larynx ultimately came to serve other functions besides protection in higher animals. In humans, for example, phonation, cough, and straining are functions of the sphincteric mechanism. Certain respiratory and circulatory effects are also related functions.

In macrosmatic species, olfaction is a further function of the larynx. Some herbivorous animals, for example, defend themselves partly by their ability to sniff the air for the presence of enemies while grazing. In the deer, the free edge of the epiglottis lies on the nasopharyngeal surface of the soft palate, so that a continuous isolated passage for air exists through the nose to the lungs. As the deer breathes, food is swallowed along the lateral food channels formed by the epiglottis and aryepiglottic folds medially and by the soft palate above (Negus, 1937). The pyriform sinuses in humans (analogous to the lateral food channel) do not provide much protection for the larynx. Further, the descent of the human larynx from a position higher up under the base of the skull (where it is found in lower animals, in the human fetus, and in the newborn human for the first few months of life)(Sasaki et al, 1977) represents a regression in the efficiency of this structure as an olfactory, respiratory, and protective organ.

On the other hand, the low position of the human larynx results in a capacious pharynx, with an epiglottis free of contact with the soft palate, so that sounds can be emitted by both the mouth and the nose. The human larynx is not specialized for any one function, as we find in the apes (valvular action), the deer (olfaction), or the horse (respiration), but it is an extremely versatile and efficient organ.

Anatomy of the Intrinsic Laryngeal Muscles

The intrinsic laryngeal muscles comprise two groups, the adductors and abductors of vocal cords. The adductors include (1) the lateral cricoarytenoid muscle, which turns the vocal process inward; (2) the internal and external thyroarytenoid muscles, which form the bulk of the vocal cord and which serve as internal tensors of the cord; and (3) the interarytenoid muscle, which extends between the two arytenoids and which pulls the arytenoids together, closing the posterior glottic chink (Fig. 1).

The abductor is a single muscle on each side of the larynx, termed the posterior cricoarytenoid. By pulling dorsally on the muscular process of the arytenoid, it rotates the vocal process outward and abducts the vocal cord.

The internal portion of the thyroarytenoid muscle, known also as the musculus vocalis, modifies the margins of the glottis by virtue of the innumerable attachments of its small fibers to one another and to the conus elasticus. It appears well suited to provide gradations of internal tension in very small steps and within single parts of the vocal folds (Zenker, 1964). These functions of fine, internal, isometric vocal cord tension supplement the relatively crude isotonic tension provided by the externally located cricothyroid muscle (Luchsinger and Arnold, 1965).

Disturbances of innervation are discussed under the section on paralysis. However, the observer should be cautioned about interpreting movements of the vocal cord after complete section of the recurrent laryngeal nerve of the same side. Such movements may be the result of the adducting and stretching action of the intact cricothyroid muscle; the action of the bilaterally innervated interarytenoid muscle; the passive motion elicited by the passage of air, especially on inspiration; the effect of the descent of the trachea on inspiration (Fing et al, 1956; Fink and Demarest, 1978); or the activity of the external laryngeal muscles (Sonninen, 1956; Murakami and Kirchner, 1973).

Innervation of the Larynx

Recurrent Laryngeal Nerve. Each inferior or recurrent laryngeal nerve supplies motor innervation to all the intrinsic laryngeal muscles of the same side and to the interarytenoid muscle of both sides. In addition, it carries sensory fibers from the infraglottic mucosa and the trachea (Sampson and Eyzaguirre, 1964; Suzuki and Kirchner, 1969). Another group of extravagal afferent fibers traveling in the recurrent laryngeal nerve trunk originates in the cardiopulmonary structures (to be discussed), reaching the central nervous system by way of the ramus communicans, which joins the superior laryngeal nerve and which is present in most human subjects (Andrew, 1954; Suzuki and Kirchner, 1967; Bowden and Scheuer, 1961).

In the human recurrent laryngeal nerve, there is no separation outside the larynx of nerve trunks into those supplying adductor and those supplying abductor muscles. A partial injury to the cervical portion of the recurrent laryngeal nerve trunk, for this reason, cannot selectively injure nerves destined for the abductor or adductor muscles (Sunderland and Swaney, 1952).

Electrical stimulation of the recurrent laryngeal nerve of the dog produces abduction of the vocal cord at stimulus frequencies below 30 per second. If the stimulus is delivered faster than 30 per second, the vocal cord gradually moves to an adducted position, the degree of adduction depending on the frequency of stimulation. At 80 stimuli per second, the cord is markedly adducted. This phenomenon is not related to intensity of stimulation (ie voltage), but to frequency. It demonstrates that tension of the posterior cricoarytenoid muscle becomes maximum at about 30 cycles per second, whereas higher frequencies are needed to produce maximum tension in the adductors (Nakamura, 1964).

Superior Laryngeal Nerve. The superior laryngeal nerve divides extralaryngeally into an internal and an external division. The inner branch is purely afferent, supplying sensation to those portions of the larynx above the glottis. The external branch is the motor nerve to the cricothyroid muscle, the extrinsic vocal cord tensor. The external branch also supplies sensation to the mucous membrane lining the infraglottic larynx at the level of the cricothyroid membrane (Andrew and Oliver, 1951; Lemere, 1932). In addition, it carries afferent impulses arising in mechanoreceptors in the fibrous capsule of the cricothyroid joint (Suzuki and Kirchner, 1968).

Action of the Larynx During Coughing

The cough reflex is absent or very weak in the newborn human infant. This is probably related to the long fetal sojourn within the uterus, during which the larynx must remain open in order that amniotic fluid may enter the trachea. The long continued depression of cough in premature infants may be the cause of the frequent respiratory difficulties to which they are subject (Miller et al, 1952).

Closure of the false vocal cords is a vital part of the cough mechanism, since approximation of the true vocal cords alone cannot prevent egress of air from the lungs. When the increasing subglottic air pressure has reached a certain point, the sphincter mechanism suddenly relaxes, and the air under accumulated pressure from below escapes in a somewhat explosive fashion. The sudden egress of this large quantity of air under great pressure also forces out the mucus or other matter that initiated the cough effort.

Laryngeal movements during coughing were studied by von Leden and Ishiki (1965), who recorded the action on high-speed motion picture film at 3000 to 5000 frames per second. These authors called attention to the Bernoulli effect, which causes many of the movements of the supraglottic mucosa during the explosive current of air.

An aberration of the protective cough reflex is that elicited by touch stimulation of the tympanic membrane or adjacent parts of the external auditory canal. The afferent arc is via the auricular branches of the vagus and the glossopharyngeal nerves, producing the sensation of irritation or tickling in the larynx. During the sensory phase of the reflex, laryngeal

phonatory performance is modified, suggesting a comparison of coughing and voluntary phonation and a possible contribution of sensation to voluntary phonation (Geoffrey et al, 1984).

Action of the Larynx During Swallowing

The usual mechanisms that protect the laryngeal inlet during swallowing include (1) reflex inhibition of respiration; (2) closure of the glottic sphincter; (3) elevation and anterior displacement of the larynx, bringing its inlet under the protection of the base of the tongue; and (4) clearing of ingested material from the pharynx before inspiration is resumed.

In humans, unlike deer, respiration ceases during deglutition. This is a reflex act resulting from stimuli arising in the pharynx as food enters, the stimuli being conducted centrally through cranial nerves IX and X. The reflex is involuntary, occurs in decerebrate animals, and is triggered by the receptor end organs that exist in great abundance in the mucous membrane of the pharynx and larynx (Ranson, 1921; Pressman and Kelemen, 1955; Doty and Bosma, 1956; Yamashita and Urabe, 1960). The most densely innervated regions of the laryngeal mucosa are those on the laryngeal aspect of the epiglottis, in the aryepiglottic folds, the ventricular bands, and the interarytenoid area (Koizumi, 1953; König and von Leden, 1961; van Michel, 1963).

Closure of the glottic sphincter is a reflex act, initiated by stimuli carried centrally in the internal branch of the superior laryngeal nerve. Electrical stimulation of the central cut end of the superior laryngeal nerve produces swallowing movements, closure of the glottic sphincter, and inhibition of respiration (Aldaya, 1936; Murtagh, 1945; Ogura and Lam, 1953; Doty and Bosma, 1956). Closure begins by approximation of the true vocal cords. Next, the false cords close against one another and against the base of the epiglottis. The posterior commissure is sealed off by an inward rotation and approximation of the arytenoid cartilages. When the false cords have been brought into apposition, a squeezing effect results, occurring according to Pressman and Kelemen (1955) as a result of intrinsic muscular activity within the mass of the false cords themselves. Passive forces may also contribute to the sphincteric closure of the supraglottic structures. Elevation of the larynx and increased intrapharyngeal pressure during swallowing, by compressing the pre-epiglottic body between the thyroid cartilage and hyoid bone, push the base of the epiglottis posteriorly against the elevated ventricular bands and help to complete closure of the laryngeal inlet (Fink, 1956). The forward pull of the thyroepiglottic ligament may also help to approximate the vocal folds (Josephson, 1927).

In any event, normal sphincteric action produces an uninterrupted tissue surface at the laryngeal inlet. If this barrier is rendered incomplete because of loss of a part, as by malignant or other destructive disease, by surgical removal of a portion, or by paralysis that prevents close approximation of the surfaces, the sphincter becomes incompetent and aspiration may occur.

The precise coordination of laryngeal respiratory and protective functions may be altered by tracheotomy. Sasaki and associates (1977) demonstrated latency shifts, increased threshold, and weakening of the reflex protective closure of the larynx in long-term tracheotomized dogs.

Individual variations in the anatomy of the larynx may explain why some individuals compensate better than others for loss of portions of the laryngeal sphincter mechanism. For example, removal of one vocal cord and arytenoid cartilage for glottic cancer usually results in no disability of deglutition. An occasional individual, however, continues to aspirate a part of ingested liquids, particularly when large mouthfuls are taken. This may be the result of several defects that occur in varying degrees as a result of surgical changes in the laryngeal anatomy. In the Som-type hemilaryngectomy, for example, in addition to removal of the arytenoid cartilage, the aryepiglottic fold is carefully thinned out so as to allow it to be brought down as a lining for the larynx over the area of resection. This maneuver produces two changes in the laryngeal anatomy that may cause aspiration. First, the cartilages of Wrisberg are removed in the thinning process so that their normal function of helping to fill the posterior gap is lost (Negus, 1937). Second, the downward mobilization of the mucous membrane remaining after removal of the arytenoid cartilage results in a flattening out of the pyriform sinus, since much of its medial wall has been brought into the laryngeal inlet. In this event, the sinus no longer lies below the level of the glottis but above it, particularly in an individual whose pyriform sinus was relatively shallow to start with. Ingested fluids may thus be directed straight toward the glottis.

After supraglottic laryngectomy, the base of the tongue affords the chief protection to the laryngeal inlet during swallowing. After removal of the epiglottis and both false vocal cords, the true vocal cords take over the entire function of the laryngeal sphincter mechanism. The cords must meet tightly in the midline, and the posterior commissure must be closed by the arytenoids or, if their resection is required, by a suitable substitute.

The absence of the epiglottis produces only a minimal disability in these patients. Since the valleculae no longer exist after epiglottidectomy, small residues of liquid after deglutition remain on the superior surface of the true cords. When the cords separate at the end of the swallow, the residual fluid must be cleared by a cough before inspiration is resumed or the fluid will drop into the trachea. It is primarily for this reason that normal pulmonary function is important in rehabilitating deglutition in patients subjected to supraglottic laryngectomy.

Anatomic structures that can be resected without destroying the laryngeal protective mechanism include the epiglottis, both aryepiglottic folds, the false cords, the pre-epiglottic space, and the upper third of the thyroid cartilage (Staple and Ogura, 1966; Conley and Seaman, 1963). Loss of these structures still allows operation of the "squirt mechanism" produced by pressure of the tongue against the soft palate and faucial arches. If, on the other hand, the base of the tongue is resected so as to prevent contact with the soft palate, residual fluid can remain in the posterior part of the mouth and pharynx after swallowing and can flood the laryngeal inlet with the next inspiration (Sheed et al, 1960, 1961; Kirchner, 1967).

Similar flooding of the larynx and trachea may result from paralysis of the pharynx for several reasons. First, in the absence of a normal stripping wave by the constrictor muscles, liquids may accumulate in the atonic pyriform sinus until they spill over the laryngeal inlet. Second, the lesion high in the vagus or in the medulla that causes the paralysis also causes a paralysis of the vocal cord in the intermediate position. The open glottis thus allows intrusion by fluids and saliva.

Paralysis of the cervical portion of the recurrent laryngeal nerve usually produces no aspiration, partly because the true vocal cord lies immobile in the paramedian position where the opposite cord can meet it. Paralysis of the recurrent laryngeal nerve in the thorax due to cancer of the esophagus or lung may result in a nearly intermediate position of the cord with an incompetent glottis. In such a case, the voice is weak and breathy and, more important, ingested liquids may pass into the trachea. The reason for this difference in behavior of the paralyzed vocal cord may depend on the simultaneous invasion of the intrathoracic vagus and the recurrent nerve by the lesion producing the paralysis (Fukuda and Kirchner, 1972).

Action of the Larynx During Respiration

The glottis opens a fraction of a second before air is drawn in by descent of the diaphragm (Green and Neil, 1955). This opening is brought about by contraction of the posterior cricoarytenoid muscles. The recurrent nerve supply to these muscles provides a rhythmic burst of motor activity that begins just before the motor activity in the phrenic nerve (Bianconi and Raschi, 1964). It is driven by the respiratory center and, like the activity in the phrenic nerve, is accentuated by hypercapnia and ventilatory obstruction and is depressed by increases in arterial oxygenation and by hyperventilation. These rhythmic inspiratory bursts in the recurrent laryngeal nerve persist, at least for a time, after respiratory movements have been arrested by succinylcholine paralysis (Suzuki and Kirchner, 1969). They can be abolished by tracheotomy, presumably as the result of decreased ventilatory resistance (Sasaki et al, 1973; Kirchner and Sasaki, 1973).

Chemosensory corpuscles have been identified in the supraglottic mucosa of primates (Ide and Munger, 1980). Their stimulation, such as during hypercapnia, has been shown to decrease laryngeal resistance during both inspiration and expiration (McCaffrey and Kern, 1980).

Inspiratory widening of the larynx is not confined to the glottis, nor is it dependent entirely on muscular activity. Fink and co-workers (1956) showed that the inspiratory descent of the larynx away from the hyoid bone causes the true and false vocal folds to become stretched, the arytenoid cartilages to slide laterally, and the glottis to open (Fink, 1975; Fink and Demarest, 1978).

Passive widening of the larynx is further enhanced by the phasic inspiratory activity of the external laryngeal muscles (Murakami and Kirchner, 1973).

As a result of variations in the size of the glottic aperture during respiration, the larynx may be an important contributor to adjustments in the intrinsic airway resistance during respiration. Abduction of the vocal cords produces glottic dilation and reduction of resistance during inspiration. Adduction with constriction of the glottis produces increased expiratory pressures, which influence the depth and rate of respiration (Bartlett et al, 1973). These reflex changes are triggered by pressure receptors in the lung and in the subglottic trachea (Sant'Ambrogio et al, 1983; Mathew et al, 1984), and may assist air mixing within the lungs (Otis et al, 1956). After vagal de-afferentation, neither inflation nor deflation affects the level of spontaneous respiratory activity in the posterior cricoarytenoid (Fukuda et al, 1973). Rattenborh (1961) concluded from his studies that adjustments in the glottic aperture compensate for changes in total airway resistance arising in the nose and bronchi.

O'Neil (1959), in this regard, reported a lowered maximum breathing capacity and an impaired intrapulmonary mixing in laryngectomized subjects.

Prenatal development of the fetal lung has been shown to depend partly on intrapulmonary pressure that exceeds that of the surrounding amniotic fluid (Adams et al, 1967; Alcorn et al, 1977; Liggins and Kitterman, 1981). The work of Harding and associates (1984, 1987) indicates that the site of resistance between the trachea and amniotic fluid is at the larynx, that the resistance is closely related to tonic activity in the thyroarytenoid muscles, and that it can be abolished by the muscle relaxant Flaxedil.

Cardiovascular Reflexes from the Larynx

Arrhythmia, bradycardia, and occasionally cardiac arrest may result from stimulating the larynx, particularly in infants (a fact well known to the bronchoscopist). The mechanism appears to be related to stimulation of nerve fibers that arise in aortic baroreceptors and, in some individuals, travel to the central nervous system by way of the recurrent laryngeal nerve, ramus communicans, and superior laryngeal nerve. These nerve fibers, when stimulated within the larynx, slow the heart rate. They pass through the larynx in the deep tissues near the thyroid ala and, thus, are not influenced by topical anesthetics. They are most effectively stimulated when the larynx is dilated, as with a bronchoscope or a tight endotracheal tube. The reflex cardiac effects can be controlled by atropine (Suzuki and Kirchner, 1967) and are enhanced by morphine (Reid and Brace, 1940). A study by Jacobs and associates (1976) suggests that at least one other pathway, in addition to the laryngeal aortic baroreceptor fibers, may be active in the production of cardiac effects associated with intubation.

Burstein and associates (1950) reported electrocardiographic disturbances in 68 per cent of patients anesthetized with the common agents. With intravenous procaine, the incidence fell to 24 per cent. Factors that enhanced these disturbances include light anesthesia, prolonged laryngoscopy, repeated attempts at intubation, respiratory obstruction, or tracheal irritation. Hypoxia and hypercapnia are also thought to contribute to reflex cardiac disturbances (Converse et al, 1952; Denson and Joseph, 1954). These disturbances during intubation are generally transient and are unaccompanied by decreased cardiac output, as reflected by the blood pressure.

Phonation

Sound production originates in the larynx as a fundamental tone that is then modified by various resonating chambers above and below the larynx. The sound is ultimately converted into speech by actions of the pharynx, tongue, palate, lips, and related structures. The fundamental frequency of a tone is produced by vibrations of the vocal folds against one another as a result of the passage of an air stream from below. The vibrations are passive, for they occur in the paralyzed larynx (Froeschels, 1957; von Leden et al, 1960) or in the larynx of a cadaver upon application of a subglottic air stream (Müller, 1837). Similarly, they can be abruptly terminated by diverting the subglottic air stream through a tracheostomy cannula (Dunker and Schlosshauer, 1957).

This interpretation of vocal cord vibration is known as the myoelastic-aerodynamic theory, as contrasted with the "neurochronaxic" theory of Husson, which enjoyed acceptance in some quarters for a time after it was proposed in 1950. Husson's theory explained vocal cord vibration by rhythmic impulses in the recurrent laryngeal nerves corresponding, beat by beat, with the frequency of the sound produced (Husson, 1950). The theory is no longer accepted, having been shown to lack either physiologic (Weiss, 1959) or acoustic merit (van den Berg, 1958).

Our understanding of vocal cord movement during phonation is derived from the high-speed motion picture film made by the Bell Telephone Laboratories in 1940. This classic film of vocal cord movements was made at 4000 frames per second and has been analyzed by many observers (Farnsworth, 1940). Other methods of observing vocal cord movements during phonation include frontal tomography (Hollien and Curtis, 1960; Fink and Kirchner, 1958) and electronic stroboscopy (Smith, 1954).

During phonation, the vocal cord is adducted to near the midline by the cricothyroid muscle, which serves as a crude isotonic tensor of the cord. Finer isometric adjustments are then made by the thyroarytenoid muscle. Medial movement of the cord, bringing it into contact with the fellow cord, is brought about by three forces: (1) tension in the cord, as just described; (2) decrease in subglottic air pressure with each vibratory opening of the glottis; and (3) the sucking-in effect of the escaping air (Bernoulli effect)(van den Berg, 1958; Smith, 1954). The result of this rapidly repeating cycle of opening and closing at the glottis is the release of small puffs from the subglottic air column that form sound waves.

During ordinary phonation, the anterior two-thirds of the vocal cords form the vibrating portion, the vocal processes of the arytenoids being held firmly in apposition (Pressman, 1942; Sonninen, 1954). Fink analyzed the Bell Telephone film and concluded that the axis of vibration of human vocal folds is not in the midline but is paramedian and appears bowed and elliptical. One possible advantage of a paramedian axis might be a minimum velocity at the time of impact, thereby lessening the risk of trauma (Fink, 1962).

If the glottis is observed in the frontal plane, it will be seen that the area of vocal cord surface in contact with its partner varies according to pitch. At low pitches, the cross-sectional area of the vocal folds is large. As pitch is raised, the folds become thinner (Hollien and Curtis, 1960).

At low pitches, opening of the glottis begins from the inferior surface of contact, the opening proceeding upward between the two surfaces of the vocal folds. The lower portion is also the first to close. In other words, there is a phase difference in the motion between different vertical portions of the vocal folds. Smith compared the mechanism of vocal cord vibration to the passage of an air bubble between two soft, elastic pillows (Smith, 1954).

Pitch

The frequency of vocal cord vibrations depends on (1) the effective mass of the vibrating part of the vocal fold and (2) the effective tension in the vibrating part of the vocal fold. Other related forces include (1) damping of the vocal folds, (2) subglottic pressure, and (3) the glottic area, which, in turn, influences resistance and which determines the value of

the Bernoulli effect (van den Berg, 1958).

With rising vocal pitch, the vocal cord is lengthened by the action of the cricothyroid muscle (Pressman, 1942; Sonninen, 1954). Although this would tend to lower the pitch, its effect is counteracted by contraction of the thyroarytenoid muscle, which thins the vocal cord and increases its tension (Fing, 1962). Because of its large number of extraordinarily thin, short fibers that can pull in various directions, the thyroarytenoid muscle appears to be well designed to produce a wide range of tension in many small steps (Zenker, 1964).

The extrinsic laryngeal muscles may lengthen or shorten the vocal cords in certain head positions by changing the relation of the thyroid to the cricoid cartilage. Sonninen cites four observers who reported lowering of the voice and loss in range along with failure of the glottis to close completely at high tone ranges after section of various extrinsic laryngeal muscles at thyroidectomy. He also reported an experiment performed during an operation under local anesthesia in which the patient sang a sustained note. In this experiment, stimulation of the sternothyroid muscle lowered the note by a half-tone when the neck was extended. When the head was held in the same plane as the body, there was no change in pitch (Sonninen, 1954; Faaborg-Andersen and Sonninen, 1960).

The biomechanics of pitch control are well illustrated in the publication of Fink and Demarest (1978).

Proprioception in the Larynx

A challenging problem to students of laryngeal physiology is the source of control over the intrinsic laryngeal muscles during singing and speaking. Is the necessary degree of coordination between the central nervous system; the respiratory muscles; the extralaryngeal, pharyngeal, and oral musculature; and the intrinsic muscles of the larynx monitored entirely by the auditory apparatus? The flat, unmodulated voice of the completely deaf person attests to the importance of hearing in regulating vocal performance (Proctor, 1968). On the other hand, the ability of a trained singer to produce a precise pitch at the moment the sound is emitted indicates that a monitoring receptor system exists in the laryngeal structures that signals position, movement, and tension in the vocal cords and related structures. The system is therefore independent of any additional adjusting influence signaled by the hearing mechanism after the sound is emitted (Schultz-Coulon, 1978; Wyke and Kirchner, 1976). Furthermore, the performance of a singer can be impaired by the application of a local anesthetic to the pharynx and larynx (Proctor, 1965).

The case for a feedback system of control originating within the larynx is supported by:

1. The presence of receptor end-organs in the laryngeal mucosa (Koizumi, 1953; König and von Leden, 1961; van Michel, 1963), muscles (Rossi and Cortesina, 1965; Lucas Keene, 1961; Rudolph, 1961; Wyke, 1974; Ibrahim et al, 1980; Winckler, 1982; Abo El-Enene and Wyke, 1966), perichondrium (Jankovskaya, 1959; Gracheva, 1963), and joint capsules (Andrew, 1950; Kirchner and Wyke, 1964).

2. The recording of afferent nerve discharges in the superior and the recurrent laryngeal nerves in response to excitation of receptors by stimuli that occur during phonation. These include touch, vibration, pressure, passage of air across mucosal surface, changes in the length of the vocal cords, and stretching of the fibrous capsules of the cricothyroid joint (Sampson and Eyzaguirre, 1964; Suzuki and Kirchner, 1967; Martensson, 1964). The responses are consistently abolished by topical or local anesthesia applied to the joint capsule (Kirchner and Suzuki, 1968; Suzuki and Kirchner, 1968, 1969).

How this neural traffic originating in the laryngeal components might contribute to reflex modulation of vocal performance is still a matter of speculation (Tanabe et al, 1975; Adzaku and Wyke, 1982). It would be difficult, if not impossible, to design a properly controlled experiment in humans. Demonstrating changes in the resting position or respiratory activity of the laryngeal muscles during manipulations of subglottic or intrapulmonary air pressure in the experimental animal would still leave the question unanswered, given the complex nature of singing as compared with centrally-evoked phonation (Testerman, 1970).

In summary, the "mechanism of the larynx" continues to challenge the ingenuity of the investigator today as surely as it did at the time of Negus' original and monumental contribution to the subject more than 50 years ago (Negus, 1929).