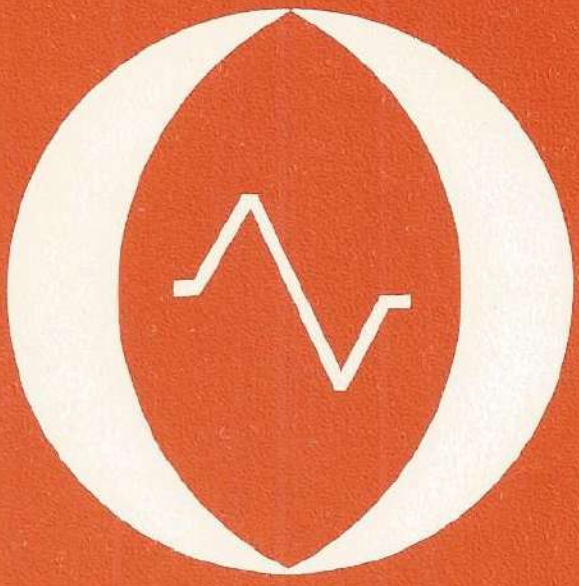
E.M.G. of the LARYNX



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with the collaboration of P. Knoops

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editions

E.M.G. OF THE LARYNX

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1987

From the same author

Précis de pathologie
et de thérapeutique
de la voix. 282 pp.
I.S.B.N. 2-7113-0162-1
Paris, 1980

La dysphonie de l'enfant. 190 pp.
I.S.B.N. 2-87077-269-6
Louvain-la-Neuve, 1984

Techniques de base d'évaluation
de la voix. 296 pp.
I.S.B.N. 2-87077-335-8
Louvain-la-Neuve, 1985

Foreword

The neuromuscular mechanism of laryngeal activities and its disorders are one of the most intriguing topics in otorhinolaryngology, neurology and speech pathology.The larynx is innervated by only two branches of the tenth cranial nerve.Its activities, however, are tremendous for many important functions including airway protection, respiration and phonation. Electromyography (EMG) is the most useful technique in studying the neuromuscular mechanism of laryngeal activities in living human beings because it shows directly the output of this complicated mechanism.

This book by Dr Dejonckere is a comprehensive monograph on laryngeal EMG, covering the basic anatomy and physiology, techniques and instrumentation for EMG, normal EMG patterns, pathological EMG patterns and their interpretations, laryngeal paralysis, and evoked EMG of the larynx.The book includes an extensive review of important literature for each topic and author's own valuable investigations.

I am convinced that this book is extremely useful for anybody who is interested in neurolaryngology and related areas.

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Contents

Chapter I

The laryngeal neuromuscular system. Anatomical and physiological backgrounds.

1.1. The laryngeal muscles

The muscles of the larynx are generally considered in two groups, extrinsic muscles on the one hand and intrinsic muscles on the other hand. Extrinsic muscles have at least one point of attachment to structures outside the larynx, while intrinsic muscles have all their insertions within the larynx.

1.1.1.EXTRINSIC LARYNGEAL MUSCLES

(Fig.I.1.)

Extrinsic laryngeal muscles may be considered as having a dual function :

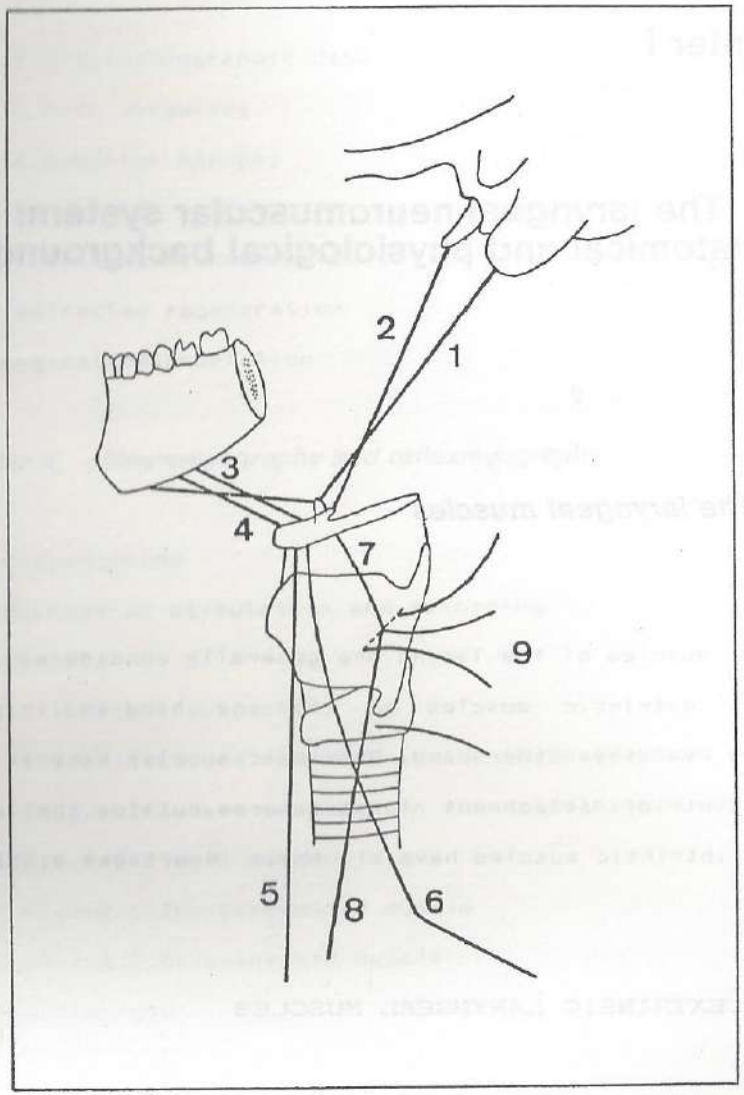


Fig. I.1. : Schematic representation of the extrinsic laryngeal muscles.

1 : Digastric muscle
2 : Stylohyoid muscle
3 : Mylohyoid muscle
3 : Geniohyoid muscle
5 : Sternohyoid muscle
6 : Omohyoid muscle
7 : Thyrohyoid muscle
8 : Sternothyroid muscle
9 : Inferior pharyngeal constrictor.

1. To support the larynx and to fix it in position

2. To elevate and depress the larynx.Anatomically speaking, the extrinsic muscles may be divided into two groups : Suprahyoid and infrahyoid muscles. Functionnally, they are usually divided into laryngeal elevators and laryngeal depressors.

There are four suprahyoid muscles, namely the digastric, the stylohyoid, the mylohyoid and the geniohyoid muscles.Functionally, they are all laryngeal elevators.

1.1.1.1.Suprahyoid muscles

1.1.1.1.1.Digastric muscle

The digastric muscle has ,as its name points out, two fleshy bellies.The anterior belly takes its origin from the inside surface of the lower border of the mandible near the symphysis.The fibers run downward and backward. The posterior belly, considerably longer than the anterior one, takes its origin from the mastoid process of the temporal bone.The fibers run downward and forward.The two bellies meet in an intermediate tendon, which perforates the stylohyoid muscle.This tendon is attached to the junction of the corpus and greater cornu of the hyoid bone by a fibrous loop, which is part of a more extensive suprahyoid

aponeurosis.

Function :

Contraction of the anterior belly draws the hyoid up and forward, while contraction of the posterior belly draws the hyoid upward and backward.Both parts of the muscle play a role in the early stages of swallowing : During the initial stage, the base of the tongue is elevated and pressed against the hard palate.The contraction of both bellies contributes to this mechanism by drawing directly upward the hyoid bone.During the second stage of deglutition, the posterior belly contracts, elevating the larynx and drawing it under the base of the tongue.This action helps to prevent food entering the larynx.

1.1.1.1.2.Stylohyoid muscle

The stylohyoid muscle is a long, slender muscle which takes its origin from the styloid process of the temporal bone.The fibers run downward and forward, just superficially with respect to the posterior belly of the digastric muscle.At its lower end, the muscle splits into two parts which pass on either side of the intermediate tendon of the digastric, to insert into the body of the hyoid bone at its junction with the greater cornu.

Function :

The stylohyoid muscle pulls the hyoid upward and backward.

1.1.1.1.3.Mylohyoid muscle

The mylohyoid muscle is a thin, single sheet of muscle fibers which form the floor of the mouth.The origin of the fibers is along the mylohyoid line, along the inner surface of the mandible from the mandibular symphysis to the last molar.The fibers run medially and downward and join at a tendinous midline raphe, which extends from the mandibular symphysis to the hyoid bone.Some of the posterior fibers insert directly into the corpus of the hyoid bone.

Function :

When the mandible is fixed, contraction of the mylohyoid muscle elevates the hyoid bone and the tongue, contributing to deglutition. Contraction of the mylohyoid muscle also assists in depressing the mandible, when the hyoid bone is held in a fixed position.

1.1.1.1.4.Geniohyoid muscle

Both right and left geniohyoid muscles ,rather cylindrical in shape, lie in direct contact with each other on both sides of the midline, just above the superior surface of the mylohyoid muscle.Their insertions are on the lower spine of the mandibular symphysis, and on the anterior surface of the body of the hyoid bone.The muscle fibers diverge slightly backward and downward.

Function:

The geniohyoid muscles pull the hyoid bone upward and forward when the mandible is fixed.They also depress and pull backward the mandible when the hyoid bone is fixed.

Furthermore, the hyoglossus and genioglossus muscles may also have the effect of elevating the larynx, although they have no direct attachment to laryngeal structures.

1.1.1.2.Infrahyoid muscles

Two paired extrinsic laryngeal muscles support the hyoid bone from below, while two others directly support the larynx.The inferior pharyngeal constrictor, although not usually classified as an infrahyoid muscle, must still be considered as an extrinsic muscle of the larynx.

1.1.1.2.1.Sternohyoid muscle

This muscle originates from the posterior surface of the sternal manubrium, from the medial end of the clavicle, and from adjacent ligamentous tissue, with insertion on the lower border of the corpus of the hyoid bone.Left and right muscles may come very near one another, and sometimes even lie in direct contact with each other.This point is to be emphasized in view of electrode placement for electromyography of the cricothyroid muscle.

Function:

The sternohyoid muscle pulls the hyoid bone downward.

1.1.1.2.2.Omohyoid muscle

The omohyoid muscle is a paired, two-bellied muscle.The inferior and posterior belly originates from the upper border of the scapula.Its fibers run forward almost horizontally to terminate at an intermediate tendon, which is held in position, above the sternum, by tendinous fibers running to the sternum and to the first rib.The superior and anterior belly originates from the intermediate tendon and runs almost vertically to insert along the lower border of

the greater cornu of the hyoid bone, laterally to the insertion of the sternohyoid muscle.

Function :

The omohyoid muscle depresses the hyoid bone.

1.1.1.2.3.Thyrohyoid muscle

It is a thin muscle located underneath the omohyoid and the sternohyoid muscles.It originates from the oblique line of the thyroid lamina, and inserts into the lower edge of the greater cornu of the hyoid bone.

Function :

When the thyrohyoid muscle contracts, the distance between the thyroid cartilage and the hyoid bone is reduced.

1.1.1.2.4.Sternothyroid muscle

The sternothyroid muscle is located just beneath the omohyoid and sternohyoid muscles, originating from the posterior face of the sternal manubrium and from the oblique line of the thyroid lamina.It has been noticed that some fibers may continue into the thyrohyoid muscle and even into

the inferior pharyngeal constrictor muscle.The anatomical localisation of this muscle is important to remember in order to prevent a faulty insertion of the electrode, when the cricothyroid muscle is to be explored electromyographically.

Function :

The main action of the sternothyroid muscle is to pull the thyroid cartilage downward.It may also contribute to enlarge the pharynx.

1.1.1.2.5.Inferior pharyngeal constrictor

The inferior constrictor originates from the oblique line of the thyroid lamina and from the lateral part of the cricoid arch between the erect and oblique parts of the cricothyroid muscle.Its fibers pass posteriorly through the lateral wall of the pharynx, turn medially to pass through the posterior wall, and meet the fibers coming from the opposite side at the median pharyngeal raphe.The raphe is not well defined, and some fibers may cross the midline.The lowest portion of this muscle, arising from the cricoid arch, is rather longitudinal and usually called the cricopharyngeus muscle.

Function :

The main function of this muscle seems to be to decrease the size of the pharynx.The pharyngeal raphe is free to move anteriorly, but there is no apparent way to stabilize the raphe in order to use the inferior pharyngeal constrictor to move the larynx.The cricopharyngeus plays the role of a sphincter at the entrance to the oesophagus.

1.1.2.INTRINSIC LARYNGEAL MUSCLES

(Fig.I.2. and I.3.)

A complex and intricate system of intrinsic muscles performs the various vital functions of the larynx : Opening the vocal folds for inspiration, closing the vocal folds for phonation, varying length, stiffness and configuration of the free edges of the vocal folds for voice adjustments, closing the larynx firmly for swallowing or during major physical efforts.

The larynx is a midline structure, but all of its muscles, except the transverse arytenoid muscle are paired both anatomically and functionally.As a matter of fact, it may be considered that, in a normal larynx, paired muscles always function bilaterally : one side never functions alone

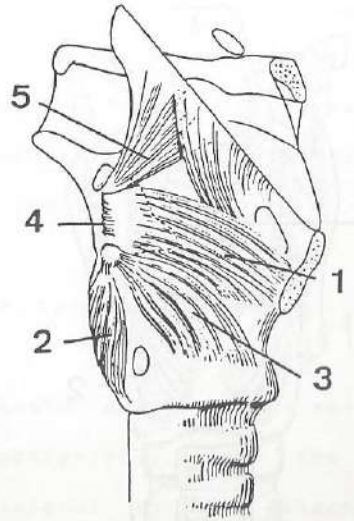


Fig. I.2 : Intrinsic laryngeal muscles.Lateral view.

1 : Thyroarytenoid muscle
2 : Posterior cricoarytenoid muscle
3 : Lateral cricoarytenoid
4 : Transverse arytenoid muscle
5 : Aryepiglottic muscle

(Piquet and Terracol, 1958)

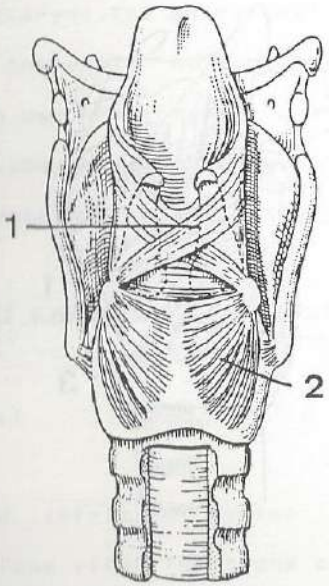


Fig. I.3 : Intrinsic laryngeal muscles.Posterior view.

1 : Oblique arytenoid muscle
2 : Posterior cricoarytenoid muscle

(Piquet and Terracol, 1958)

or independently from the other one.

The intrinsic laryngeal muscles may be classified as abductor, adductor, or tensor, according to their effects on the vocal folds. The main mass of the vibrating vocal fold is made up of the medial (internal) portion of the thyroarytenoid muscle (Fig.I.4.) , and is sometimes referred to as the vocalis muscle.

1.1.2.1.Thyroarytenoid muscle

The thyroarytenoid muscle is a relatively large muscle extending anteroposteriorly, from the deep face of the thyroid lamina,lateral to the attachment of the vocal ligament, to the arytenoid cartilage.Some fibers insert onto the inferior fossa of the arytenoid cartilage and the adjacent part of the vocal process.Other fibers insert onto the entire length of the dorsolateral ridge of the arytenoid cartilage.Because of its functional importance for phonation, this muscle has been the subject of a considerable amount of investigations.Schematically, and considering only the anatomical point of view, three points of discussion have arisen :

1.Whether there is an anatomical basis for considering the more medial (vocalis) and the more lateral part of the

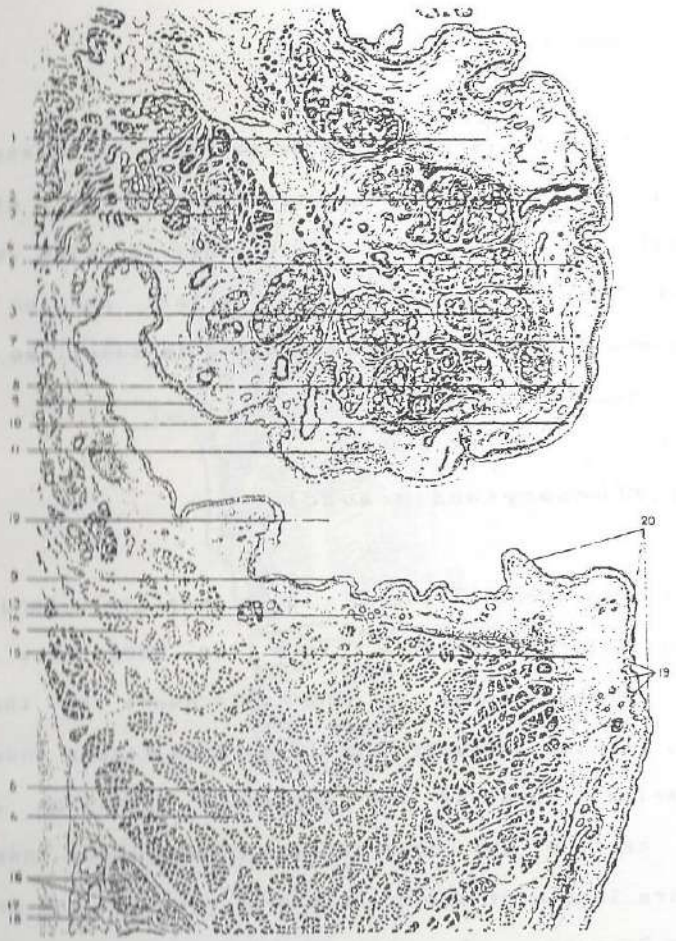


Fig. I.4 : Frontal section through vocal and vestibular folds.

1 : Submucosal tissue
2 : Glandular duct
3 : Mucosal glands
4 : Muscular fibers
5 : Blood vessels
6 : Thyroarytenoid muscle
7 : Lymph vessel
8 : Tunica propria mucosae
9 : Ciliated epithelium
10: Transition epithelium
11: Adenoid tissue
12: Laryngeal ventricle
13: Mucosal glands
14: Superficial vessels
15: Vocal fold
16: Nerve rami
17: Thyroid cartilage
18: Adipous tissue
19: Epithelial papilla
20: Squamous epithelium

thyroarytenoid as separate muscles.There is general agreement that no fascial planes separate the two parts of the muscle.Due to the absence of a well-defined epimysium separating the two portions, English and Blevins (1969) consider them as components of the thyroarytenoid muscle instead of separate muscles.There is some evidence that fibers from the two parts intermingle (Zenker, 1964) but probably only in the subglottic area (Dixon and Maue-Dixon, 1982).

2.Whether fibers of the internal (vocalis) portion of the thyroarytenoid muscle insert into or mingle with fibers of the vocal ligament.Some authors have described muscle fibers inserting into the vocal ligament (Goerttler, 1950; Behringer, 1955), but others were unable to confirm these findings (Sonesson, 1960; Rohen, 1968).English and Blevins (1969) report that the lower-most fibers of the musculus vocalis are embraced by connective tissue of the conus elasticus; superior and medial muscle fibers are closely related to the connective tissue of the vocal ligament and although some of them may insert tangentially on it, the majority of them are directed along its longitudinal axis to a point of insertion on the vocal process.

3.Whether the vocalis muscle is to be divided into two functionally distinct entities, i. e. a pars thyreovocalis

and a pars aryvocalis, or a portio thyreovocalis and a portio thyreomuscularis.Goerttler (1950), followed by others (Behringer; 1955; Seiter, 1955) claimed that the musculus vocalis was divided into a portio aryvocalis and a portio thyreomuscularis.Both parts are inserted into the vocal ligament, running obliquely with respect to the ligament : The portio thyreovocalis arises from the inner side of the thyroid cartilage, running in a postero-medial direction, and the portio aryvocalis arises from the muscular process of the arytenoid cartilage and runs in an antero-medial direction.The vocal ligament is regarded as an intermediate tendon within the vocalis muscle.From this point of view, the action of the vocalis muscle is to open the glottis.Wüstrow (1952) and van den Berg and Moll (1955) believe that the vocal muscle is divided into two portions arising from the thyroid cartilage and inserted respectively onto the vocal process (portio thyreovocalis), and onto the muscular process (portio thyreomuscularis) of the arytenoid cartilage.Wustrow expressed the view that the main function of the portio thyreovocalis is to effect closure of the glottis, while the portio thyreovocalis mainly serves to control the tension of the vocal fold.

At present, the most convincing data about the architecture of muscle fibers inside the thyroarytenoid muscle are those supplied by Rohen (1968) (Fig.I.5.), who

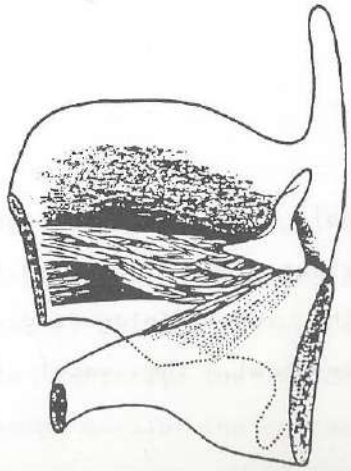


Fig. I.5.: Internal structure of the thyroarytenoid muscle, after Rohen (1968).Muscular fibers are grouped in bundles, which appear twisted and tightly breaded.Lateral view from inside the larynx.

also explained, on the basis of technical particularities, how misinterpretations of histological sections are possible.According to this author, the muscular fibers are grouped in bundles, which appear twisted and tightly breaded, without differenciation of structural sub-entities into the muscle.

Function :

The thyroarytenoid muscles, acting alone, would approximate the arytenoid cartilages and the thyroid angle, and thus shorten the vocal folds and adduct them.If the contraction is isometrical, the vocal fold stiffness is modified.

I.1.2.2.Posterior cricoarytenoid muscle

The posterior cricoarytenoid muscle is the only abductor muscle of the vocal folds.Its configuration is that of a large, fan-shaped paired muscle, located on the dorsal face of the cricoid lamina, and originating from a shallow depression on the posterior surface of this cricoid lamina.The fibers run upward and laterally, and converge upon the posterior face of the muscular process of the corresponding arytenoid cartilage.According to Zemlin et al. (1984), this muscle is to be considered as composed of two

separate bundles : One is superior-medial and consists of oblique fibers, and the other one - more important - is lateral and consists of vertically directed bipennate fibers.The two bundles have different insertions on the muscular process of the arytenoid cartilage (Fig.I.2. and I.3.)

Function :

The main force of this muscle would be along a line that bissects the longitudinal axis of the muscle and extends from the inferomedial cricoid lamina to the muscular process of the arytenoid cartilage.The force would be at a right angle to the long axis of the cricoarytenoid facet on the cricoid cartilage.In fact, the posterior cricoarytenoid muscle is ideally situated to tip the arytenoid cartilage dorsolaterally, abducting the vocal fold.

I.1.2.3.Lateral cricoarytenoid muscle

This glottal adductor is also a fan-shaped muscle, located deeply with respect to the thyroid cartilage in the anterolateral wall of the larynx.The medial surface of this muscle lies in direct contact with the conus elasticus.The muscle fibers take their origin from the upper edge of the arch of the cricoid cartilage.They run upward and backward

and rapidly converge before inserting into the anterior surface of the muscular process of the corresponding arytenoid cartilage (Fig.I:2.).

Function :

When contracting, this muscle tends to rotate the arytenoid cartilage , pulling the muscular process forward and the vocal process medially.It also pulls the whole arytenoid cartilage slightly forward.The primary function is thus adduction of the vocal folds, but the lateral cricoarytenoid muscle may also shape up the glottis for production of a whisper, with an opening of the cartilaginous portion of the glottal chink.This last characteristic feature also evokes a dyskinetic glottal configuration, as seen in some cases of functional voice pathology.

1.1.2.4.Oblique arytenoid muscle

This small muscle consists of two fasciculi, each of which originates from the medial half of the muscular process of one arytenoid cartilage and inserts into the apex of the opposite cartilage.When viewed from behind, the fasciculi appear to cross each other.Some muscle fibers continue around the apex of the arytenoids laterally and

angle upward and forward to insert into the lateral bords of the epiglottis.These fibers then constitute a part of the aryepiglottic muscle.A few fibers also seem to insert into the quadrangular membrane (Fig.I.3.).

Function :

The oblique arytenoid muscles approximate the arytenoid cartilages and act as adductors of the vocal folds.

1.1.2.5.Transverse arytenoid muscle

It is, among the intrinsic laryngeal muscles, the only impair one.Its fibers originate from the lateral margin and posterior surface of one arytenoid cartilage, run horizontally , and insert into the lateral margin and posterior face of the opposite arytenoid cartilage (Fig. I.2.)

Function :

Contraction of this muscle approximates the arytenoid cartilages, especially their median angles.However, the transverse arytenoid muscle would appear to operate most efficiently in holding the arytenoid cartilages in an adducted position once adduction has been achieved.

1.1.2.6.Aryepiglottic muscle

A few muscle fibers extend from the apex of the arytenoid cartilage to the epiglottis.Some or even all of these fibers may be extensions of the oblique arytenoid muscle (Fig.I.5.)

Function:

These fibers move the epiglottis backward and downward, shorten the aryepiglottic folds and slightly move the arytenoid cartilages toward adduction.

1.1.2.7.Thyroepiglottic muscle

A few muscle fibers extend from the deep face of the thyroid lamina, superior to the fibers of the thyroarytenoid,into the aryepiglottic fold.

Function:

These fibers pull forward on the apex of the arytenoid cartilage.

1.1.2.8.Musculoepiglottic muscle

This muscle has some fibers lying on the lateral face of the thyroarytenoid muscle, originating from the anterolateral arch of the cricoid cartilage, and inserting into the thyroid cartilage as two distinct parts.The more posterior belly, called the oblique portion, runs upward and backward to insert into the anterior margin of the inferior cornu of the thyroid cartilage.The more anterior belly, called the erect portion, runs nearly vertically upward and inserts along the inner aspect of the thyroid lamina.

Function:

When anterior fibers contract, the distance between the thyroid laminae and the cricoid cartilage is reduced.Thus, the distance between the thyroid cartilage, at the angle, and the vocal processes of the arytenoid cartilages is increased by a proportional amount.This action increases the length of the vocal folds.Contraction of the oblique portion pulls the thyroid cartilage forward on the cricothyroid joint, and this action further increases the distance between the arytenoid and thyroid cartilages.In addition, if the arytenoid cartilages are in the abducted position, contraction of the cricothyroid muscles results in adduction of the arytenoids.

1.1.2.9.Cricothyroid muscle

The cricothyroid muscle is a fan-shaped muscle broader above than below, originating from the anterolateral arch of the cricoid cartilage, and inserting into the thyroid cartilage as two distinct parts.The more posterior belly, called the oblique portion, runs upward and backward to insert into the anterior margin of the inferior cornu of the thyroid cartilage.The more anterior belly, called the erect portion, runs nearly vertically upward and inserts along the inner aspect of the lower margin of the thyroid lamina.Both in infants and adults, the weight of the cricothyroid muscle exceeds that of any other single intrinsic laryngeal muscle (Kahane and Kahn, 1984) (Fig. I.6.).

Function:

When anterior fibres contract, the distance between the thyroid laminae and the cricoid arch decreases. Thus, the distance between the thyroid cartilage, at the angle, and the vocal processes of the arytenoid cartilages is increased by a proportional amount.This action increases the length of the vocal folds.Contraction of the oblique portion pulls the thyroid cartilage forward on the cricothyroid joint, and this action further increases the distance between the arytenoid and thyroid cartilages.In addition, if the arytenoid cartilages are in the abducted position,

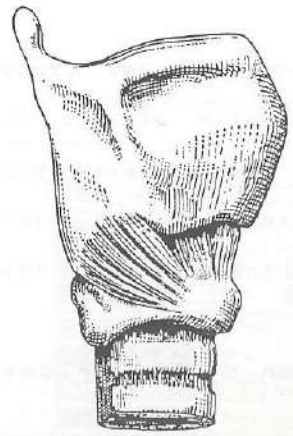


Fig. I.6. : Cricothyroid muscle.Lateral view.Oblique and erect portions.
(Piquet and Terracol, 1958)

contraction of the cricothyroid muscles results in adduction of the arytenoids.

1.1.3.FUNCTIONAL ASPECTS

Using excised canine larynges because of their similarity to human larynges, Koike et al. (1976, 1978) stimulated electrically each muscle with bipolar electrodes and observed both macroscopic and histological changes in position and shape of the vocal fold (Fig. I.7.).

1.1.3.1.Stimulation of the cricothyroid muscle

The effect is that the vocal fold is stretched, elongated and slightly adducted to the paramedian position.The edge of the fold on the stimulated side becomes thinner than on the non-stimulated side.When the crico-thyroid muscle is stimulated on one side, the vocal folds on both sides are elongated, but the degree of elongation is greater on the stimulated side than on the non-stimulated side.The upper level of the vocal fold is lowered. Histologically, there is a decrease, not only in the thickness of the entire cord, but also in the area of the lamina propria.Shipp et al. (1970) stimulated the cricothyroid muscle in vivo while the subject sustained phonation in the lower part of his frequency range, and they observed a rise in frequency.

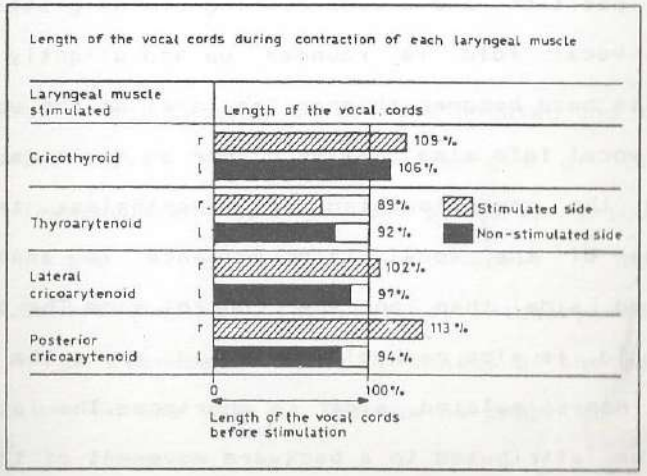


Fig. I.7.: Changes in length of the vocal cords upon stimulation of individual laryngeal muscles (Koike et al., 1976).

1.1.3.2.Stimulation of the posterior cricoarytenoid muscle

The effect is an abduction of the vocal fold to the lateral position, and a wide opening of the glottis.The edge of the vocal fold is rounded up and slightly thickened, while its base becomes thinner.The level of the upper margin of the vocal fold also appears higher on the stimulated side than on the non-stimulated one. Nevertheless, the width of the base of the vocal fold eminence is smaller on the stimulated side than on the control side.The ipsilateral vocal fold is also markedly elongated, while the vocal fold of the non-stimulated side is shortened.The latter effect should be attributed to a backward movement of the anterior commissure together with the thyroid cartilage.In spite of the fact that the edge of the vocal fold on the stimulated side becomes thicker, the area of the lamina propria decreases when the posterior cricoarytenoid contracts.

1.1.3.3.Stimulation of the thyroarytenoid muscle

The resulting effect is a shortening and thickening (bulging) of the vocal fold , as well as an adduction.Stimulation of the thyroarytenoid muscle on one side causes shortening of both vocal folds, but especially

on the stimulated side.The upper level of the stimulated vocal fold is raised upwards.The cross-sectional area of the lamina propria is increased.

1.1.3.4.Stimulation of the lateral cricoarytenoid muscle

The vocal fold becomes slightly elongated and thinner at its edge.The level of the upper margin of the fold is slightly lowered.Even as for the posterior cricoarytenoid muscle, the contralateral vocal fold is shortened.Contraction of the lateral cricoarytenoid muscle results in a decrease in the thickness of the edge of the vocal fold and in the area of the lamina propria.

1.1.3.5.Stimulation of the arytenoid muscle

The stimulation of this muscle causes an adduction of the arytenoid region, but without any significant effect on the shape of the membranous portion of the vocal fold.

1.2. The laryngeal nerves

(Piquet and Terracol, 1958; Zemlin, 1968; Truex and Carpenter, 1969; Dickson and Maue-Dickson, 1982; Graney, 1986)

1.2.1.CENTRAL NERVOUS SYSTEM

Cell bodies of the vagus nerve, which supply the larynx are found deep within the reticular formation of the medulla oblongata in the nucleus ambiguus.The nucleus ambiguus is a column of cells in the reticular formation about halfway between the spiral trigeminal nucleus and the inferior olive.It extends from about the caudal border of the lemniscal decussation to the level of the striae medullares.The nucleus is composed ot typical multipolar lower motor neurons whose axons innervate the muscles of the larynx and the pharynx.Experimental evidence in monkeys indicate that the upper third of the nucleus ambiguus is responsible for the pharynx and oesophagus.More caudally is a center for cricothyroid muscle function, and caudal to that is a center for abductor laryngeal muscle function.The most caudal region is responsible for adductor muscle function (Furstenberg and Magielski, 1955; Aronson, 1980; Lang, 1981).

Data about central nervous system control over the tenth nerve lack precision, although corticobulbar tracts probably form synapses with vagus nerve nuclei in the same way as they do with other cranial nerve nuclei.However, as pointed out by Aronson (1980), it is also likely that vagus nerve nuclei receive a disproportionately larger share of subcortical innervation to facilitate reflex laryngeal function, owing to its important life-sustaining responsability.The tenth nerve nuclei are under the influence of both pyramidal and extrapyramidal systems.The pyramidal system (the corticobulbar tracts) exerts voluntary control over the larynx.Corticobulbar tracts originate from the precentral gyri of both cerebral hemispheres, they descend as the corona radiata, they become condensed in the internal capsule, and they pass through the cerebral peduncles.At the upper border of the medulla, most of the fibers decussate to the opposite side of the brainstem, where they form synapses with vagal nuclei in the nucleus ambiguus.The remainder form synapses with vagal nuclei on the ipsilateral side.The nucleus ambiguus also receives impulses from the pharyngeal and laryngeal muscles for tonic control, and from secondary vagal, glossopharyngeal and trigeminal fibers.Fibers in these nerves convey impulses from the oral, the pharyngeal, and the whole respiratory mucosa ; they mediate various reflexes, such as coughing,

vomiting, and pharyngeal and laryngeal reflexes.Afferent vagal fibers enter the medulla in association with emerging efferent fibers (Truex and Carpenter, 1969).

1.2.2.PERIPHERAL NERVOUS SYSTEM

(Fig. I.8., I.9 and I.10)

1.2.2.1.Vagus nerve

The superficial origin of the vagus nerve consists of a number of small rootlets, which emerge between the olive and inferior peduncle, just underneath the roots of the glossopharyngeal nerve.The nerve presents two enlargements : the jugular (superior) and nodose (inferior) ganglia, at the level of the jugular foramen, by way of which it leaves the cranium.These ganglia contain cells for the sensory portion of the nerve.From the ganglia, some branches join several of the other cranial nerves, and others supply sensory fibers (dura mater; external ear).

In the neck, the vagus nerve gives off several branches, some of which directly control the speech mechanism.In addition, it receives fibers from other cranial nerves : so, motor fibers arising from the eleventh (accessory) nerve enter the vagus nerve and emerge as the inferior laryngeal

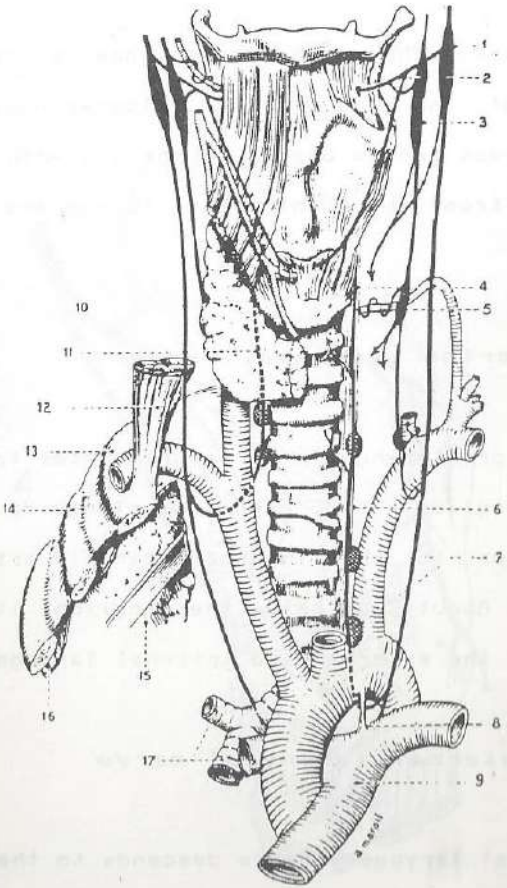


Fig. I.8.: Laryngeal nerves.

1.Vagus nerve
2.Superior laryngeal nerve
3.Superior cervical ganglion
4.Inferior laryngeal nerve
5.Inferior thyroid artery
6.Oesophagus
7.Lymphatic ganglions
8.Ligamentum arteriosum
9.Pulmonary artery
10.Thyroid gland
11.Inferior laryngeal nerve
12.Scalenus anterior muscle
13.First rib
14.Second rib
15.Lung
16.Pleura
17.Bronchi

(Piquet and Terracol, 1958)

(recurrent) nerve.Thus, there is some evidence that the innervation of the laryngeal and velopharyngeal musculature actually derives from a branch of the eleventh cranial nerve rather than from the tenth one (Dixon and Maue - Dixon, 1982).

1.2.2.2.Superior laryngeal nerve

The superior laryngeal nerve originates from the nodose (inferior) ganglion, and descends alongside the pharynx first posterior to the internal carotid artery, and then medial to it. About 2 cm below the ganglion, it divides into two branches : the external and internal laryngeal nerves.

1.2.2.2.1.Internal laryngeal nerve

The internal laryngeal nerve descends to the level of the thyroid membrane, pierces and enters it, and further divides into two additional branches.They convey afferent fibers from the mucous membrane of the larynx above the vocal folds, and also from muscle spindles and other stretch receptors in the larynx.Some of the terminal branches anastomose with fibers of the inferior laryngeal nerve.

1.2.2.2.2.External laryngeal nerve

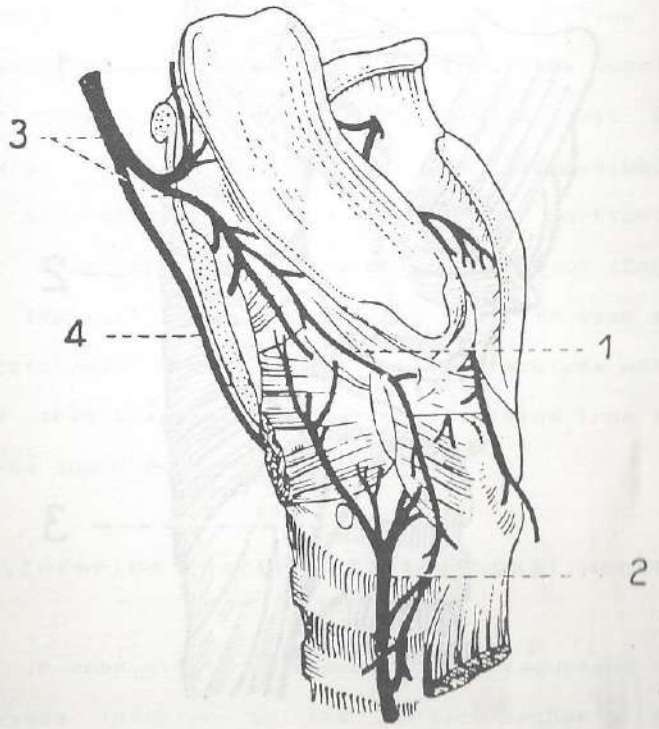


Fig.I.9.: Laryngeal nerves.

1.Galen's anastomosis
2.Inferior laryngeal nerve
3.Superior laryngeal nerve
4.External laryngeal nerve and internal laryngeal nerve
(Piquet and Terracol, 1958)

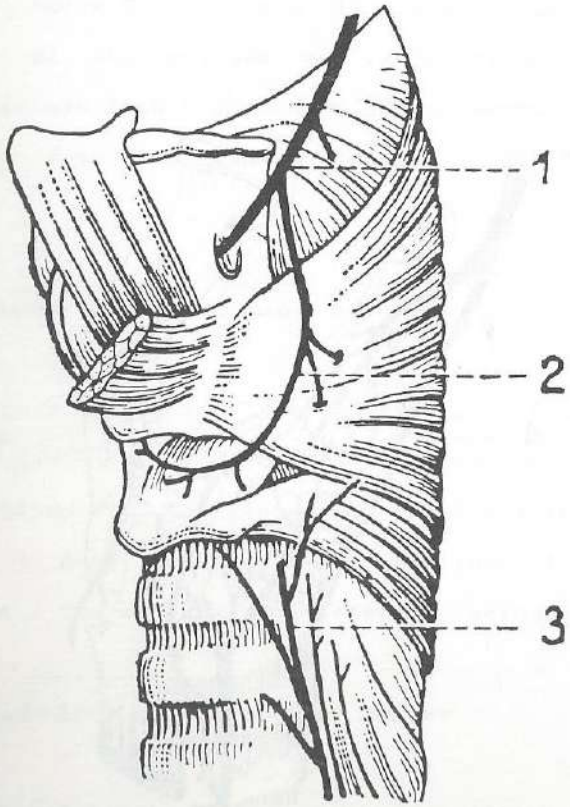


Fig.I.10.: Laryngeal nerves.

1.Superior laryngeal nerve 2.External laryngeal nerve
3.Inferior laryngeal nerve

(Piquet and Terracol, 1958)

The external laryngeal nerve descends in close relation to the oblique line on the posterolateral face of the thyroid catilage.It turns obliquely forward, near the cricothyroid muscle , so that it is in line with the longitudinal axis of the oblique head of the muscle.Two or three branches are formed, which divide just before or immediately after penetrating the epimysium.Anterior branches are directed toward the erect portion, and the posterior branches supply the oblique portion (English and Blevins, 1969).It must be noticed that, in some subjects, the cricothyroid muscle additionally receives motor nerve filaments that traverse the pharyngeal plexus from the vagus nerve (Wyke and Kirchner, 1976).

1.2.2.3.Inferior (recurrent) laryngeal nerve

Early in embryologic development, the recurrent laryngeal nerve passes inferior to the aortic arches of the heart before entering the larynx.During the process of growth, the larynx and aortic arches become increasingly separated, and the nerve thus descends to loop under the adult state of those arches (the subclavian artery on the right and the aortic arch on the left) from front to back, before rising to the larynx.

The right nerve ascends alongside the trachea behind the

common carotid artery, and in or near the groove between the trachea and the oesophagus.It enters the larynx just behind the articulation between the inferior horn of the thyroid cartilage and the cricoid cartilage.The nerve generally divides into two or more branches before entering the larynx (Aronson, 1980).

The left inferior laryngeal nerve also ascends laterally with respect to the trachea, and enters the larynx as on the right side.

During their course along the trachea, both inferior laryngeal nerves give sensory branches to the trachea.

Many authors described extralaryngeal divisions of the inferior laryngeal nerve in two or more branches.Nemiroff and Katz (1982) report extralaryngeal divisions in up to 40 % of cases, the majority of them occurring between 1.8 and 2.3 cm below the cricoid cartilage. It has been suggested that this variability may explain the different vocal fold positions observed in post-surgical paralysis (Claes and Jaco, 1986).Another variability in the course of the inferior laryngeal nerve and its terminal branches in relation to the inferior thyroid artery was pointed out by Reed (1943).A recent review has been made by Van Den Eeckhaut (1983).

The left-right difference makes the right recurrent nerve about 11.5 cm shorter compared to the left one, in subjects 155 to 173 cm in height (Peytz et al., 1965).This is in relation with a difference in embryologic development between left and right branchial arteries : Anatomic variants such as a non recurrent right inferior laryngeal nerve coincide with vascular anomalies of the neck (Claes and Jaco, 1986).

The inferior laryngeal nerve is primarily a motor nerve, and it supplies all the intrinsic muscles of the larynx except the cricothyroid.Inside the larynx, most of the posterior branches are directed to the body of the posterior cricoarytenoid muscle, but one of them continues upward to supply the oblique and transverse arytenoid muscles.The main inferior laryngeal nerve continues upward behind the cricothyroid articulation; it gives off two or three separate branches to the lateral cricoarytenoid muscle and then continues to form multiple branches supplying the thyroarytenoid muscle.The thyroarytenoid, the lateral cricoarytenoid and the posterior cricoarytenoid muscles are innervated by the homolateral inferior laryngeal nerve, and the arytenoid muscles (oblique and transverse) are innervated by bilateral ones (Tomita, 1967 ; Wyke and Kirchner, 1976).Before entering into the larynx, the

inferior laryngeal nerve also gives off a posterior branch which anastomoses with filaments of the internal branch of he superior laryngeal nerve (Galen's anastomosis).

The inferior laryngeal nerve also contains sensory fibers that supply the respiratory mucosae below the level of the vocal folds, and carries afferent impulses from stretch receptors.

Murtagh and Campbell (1952) observed that adductor fibers of the inferior laryngeal nerve continue to propagate action potentials despite a chemical or thermal blockade sufficient to paralyze abductor function.Furthermore, the chronaxy of adductor fibers, one of the most rapid thus far identified (0.1 ms) is tenfold less than that found in fibers subserving abduction.

The problem of a possible dual innervation of some laryngeal muscles by both the superior and the inferior laryngeal nerves has been discussed by some authors (Arnold, 1961).Exner's nervus laryngeus medius , a separate branch of the ramus pharyngeus nervi vagi , and innervating the cricothyroid muscle , seems to exist in rabbits and dogs, but not in humans.

Table I.1. summarizes the data about innervation of

Innervation of the intrinsic laryngeal muscles	
Muscle	*Innervation*
Cricothyroid	External branch of the superior laryngeal nerve. Sometimes (additionally nerve filaments that traverse the pharyngeal plexus from the vagus nerve.
Vocal	Inferior laryngeal nerve (recurrent nerve).
Oblique and Transverse arytenoid	Both inferior laryngeal nerves.
Lateral cricoarytenoid	Inferior laryngeal nerve.
Posterior cricoarytenoid	Inferior laryngeal nerve.

Table I.1.: Innervation of the intrinsic laryngeal muscles, after Wyke and Kirchner (1976).

intrinsic laryngeal muscles.

1.2.2.4.Autonomic nerve supply of the larynx

Parasympathic fibers originate in the dorsal motor nuclei of the vagus nerve, synapse in the inferior ganglion, and accompany the motor nerves to the larynx.Sympathetic nerve fibers enter the motor nerves through connections with the superior cervical ganglion.

1.2.2.5.Nerve supply of the extrinsic laryngeal musculature

The extrinsic musculature of the larynx may also be implicated in lower motor neuron pathology.Among suprahyoid muscles, the anterior belly of the digastric is innervated by the mylohyoid branch of the inferior alveolar nerve: the posterior belly by the seventh nerve, as the stylohyoid.The mylohyoid is innervated by the mylohyoid branch of the inferior alveolar nerve, and the geniohyoid by the first cervical spinal nerve (C1) via the hypoglossal nerve, as is the thyrohyoid.The remaining infrahyoid muscles - the sternohyoid, sternothyroid and omohyoid - are all innervated by the ansa cervicalis.

1.2.2.6.Segmental innervation of the larynx

The laryngeal muscles may be considered as developing from the two pharyngeal sphincters : the external and the internal.The external sphincter has again two derivatives : a dorsal one and a ventral one.The dorsal one becomes the thyro- and cricopharyngeus.The ventral one is the cricothyroid muscle.Originating from the fourth branchial arch, this muscle is innervated by the fourth branchial nerve, which is the superior laryngeal nerve, via its external motor branch.Similarly, the internal sensory branch of this nerve transmits afferent sensations from the upper larynx, which has the same branchial origin.

Derivatives of the internal sphincter are the intrinsic laryngeal muscles.Being derived from the sixth branchial arch, all internal laryngeal muscles are motorically supplied by the sixth branchial nerve.Their proprioceptive sensation is likewise transmitted through sensory fibres of the inferior laryngeal nerve, as are afferent sensations from the infraglottic larynx (Arnold, 1961).

1.3. Laryngeal behaviours

(Fig. I.11.)

1.3.1.Respiration

Respiration is temporally the dominant function of the larynx (Fink, 1975).Although already mentioned by Manoel Garcia (1855), respiratory movements of the larynx were only recently investigated in depth.Stanescu et al. (1972) and Clément et al. (1973) showed that lung volume and airflow rate greatly affect the size of the glottis.The most obvious respiratory movements of the larynx consist of widening of the glottic chink (abduction of vocal folds) during inspiration, and of narrowing during expiration (adduction of vocal folds).This movement is almost imperceptible during quiet respiration, but, as the depth of the respiratory movement increases, the inspiratory excursion becomes easily recognizable (Fink, 1975).

Furthermore, gross movements of the entire laryngeal framework take place during respiration : downward with inspiration, upward with expiration.Even as the glottic movements, the cranio-caudal displacement vary with the amplitude of ventilation (Fink, 1975).

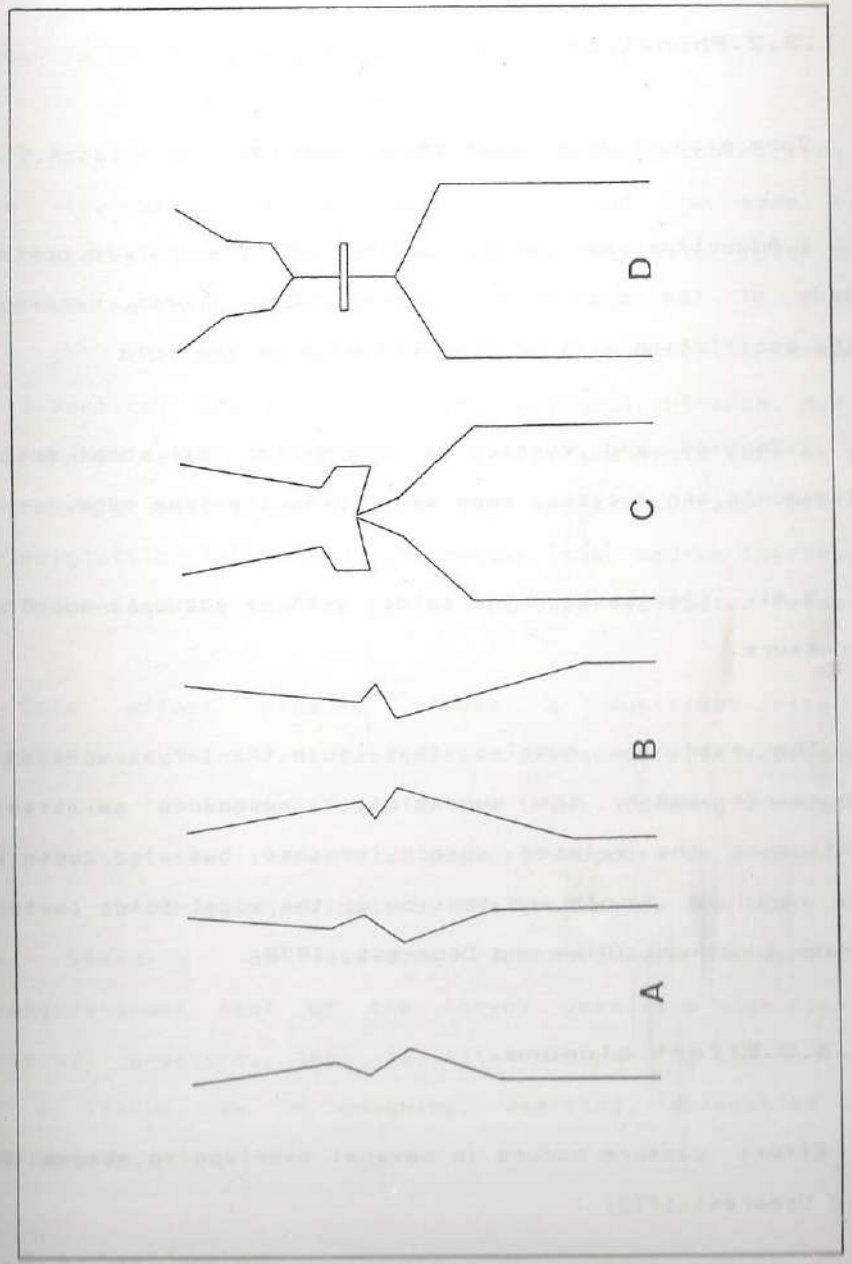


Fig.I.11.:Schematic frontal sections of the larynx

A.Inspiration B.Expiration
C.Phonation E.Effort closure

1.3.2.Phonation

Tone production schematically requires:

1.Adduction and stabilization of the mobile posterior ends of the vocal folds (arytenoid cartilages), determining the oscillation axis of the free edge of the fold

2.Tension and eventually elongation of vocal muscles, ligaments and mucosae, even as shaping the free edge.

3.Air flow between the folds, with an adequate subglottic pressure.

The extrinsic muscles that move the larynx upwards and downwards modify the supraglottic resonance cavities and influence the related speech formants, but also contribute to regulate length and tension of the vocal folds (external frame function) (Fink and Demarest, 1978).

1.3.3.Effort closure

Effort closure occurs in several overlapping stages (Fink and Demarest,1978) :

1.Adduction of the arytenoid cartilages, bringing the

vocal folds in apposition on the median line

2.Apposition of the vestibular folds by contraction of the thyroarytenoid muscles, which at the same time obliterates the sinus by drawing the vestibular folds down against the vocal folds.

3.Vertical shortening of the laryngeal intrance, due to an approximation of thyroid cartilage and hyoid bone by the thyrohyoid muscle.This folds the medial surface of the aryepiglottic folds and compresses the median thyrohyoid fold down against the top of the adducted vestibular folds.

This effort closure allows a sustained rise in intrathoracic and intraabdominal pressure, with subsequent stiffening of the framework of the trunk, forming a base for efforts by the limbs.This postural support is used during lifting or dragging efforts by the arms while the individual is standing or striding orthograde.Furthermore, a pressure-proof seal of the larynx permits a high rise in internal pressure, that facilitates expelling the contents of a viscus, as in coughing, vomiting, defecation and childbirth.

1.3.4.Swallowing

The main events in swallowing are similar to those of effort closure, but two additional mechanisms intervene , in order to prevent a puddle of bolus from collecting in the pit at the entrance of the closed larynx, and thus fall into the airway when the larynx reopens :

1.Downfolding of the epiglottis over the entrance

2.Hyperelevation of the closed larynx against the epiglottis

(Fink and Demarest, 1978).

Chapter II

Laryngeal motor units

2.1. Intrinsic laryngeal muscles

2.1.1.Size of intrinsic laryngeal muscles

Intrinsic laryngeal muscles are small. Their sizes have been measured by K. Faaborg-Andersen (1957) : Table II.1. summarizes the average lengths, breadths and thicknesses, and also gives volumes and weights,as calculated from the measured data.Specific gravity is taken as 1.06.

Weight measurements of infant (9 subjects) and adult (12 subjects) intrinsic laryngeal muscles have also been performed by Bowden and Scheure (1960), and by Kahane and Kahn (1984) .Owing to their small size and to the difficulty in determining the boundaries between them, the thyroarytenoid and lateral cricoarytenoid muscles were removed as a unit in infants.Comparison is made with adults after adding the weights of both muscles in the latter. Table II.2. relates mean values and standard deviations.Table II.3. expresses individual muscle weights

MUSCLE	Length (mm)	Breadth (mm)	Thickness(mm)	Cross-sectional area (mm2)	Volume (mm3)	Calculated weigth(mg)
Crico-thyroid	15	10	6	60	900	954
Vocal	15	4	5	20	300	318
Transverse arytenoid	15	7	5	35	525	556
Posterior cricoary-	12	10	5	50	600	636

Mean values of 4 excised human larynges : 2 men, weigth 68 and 80 kg, heigth 172 and 180 cm; 2 females, weigth 52 and 63 kg, heigth 160 and 165 cm.

Table II.1.: Average lengths, breadths, thicknesses, cross-sectional areas, volumes and calculated weights of intrinsic laryngeal muscles, after Faaborg-Andersen (1957).

MUSCLE	WEIGHT			
	Infant male	Infant female	Mean infants	Adults
CTh	70.4 (23.2)	77.5 (14.0)	73.6 (19.5)	692.2 (247.5)
LCA/TA	69.6 (20.7)	83 (38.0)	75.5 (29.5)	873.6 (364.3)
IA	27.4 (7.3)	31.1 (9.1)	29.1 (8.1)	304.1 (120.0)
PCA	44.2 (11.3)	46.7 (11.5)	45.3 (11.2)	408.2 (132.7)

Table II.2.: Mean weight (mg) and standard deviation of infant and adult intrinsic laryngeal muscles.Adult means were calculated from data by Bowden and Scheure (1960).
CTh : Cricothyroid muscle
LCA/TA : Sum of weights of the lateral cricoarytenoid and thyroarytenoid muscles.
IA : Interarytenoid muscle
PCA : Posterior cricoarytenoid muscle.
(After Kahane and Kahn, 1984)

GROUP	MUSCLE			
	(% of total weight of laryngeal muscles)			
	CTh	LCA/TA	PCA	IA
Infant	33	34	20	13
Adult	31	38	18	13

Table II.3.: Individual muscle weights expressed as a percentage of the total weight of intrinsic laryngeal muscles within each group, respectively.Same abbreviations as in Table II.2. (After Kahane and Kahn, 1984)

muscles within each group, respectively.

Zemlin et al. (1984), which specifically investigated the posterior cricoarytenoid muscle report a mean weight of 671.5 mg, with a range of 449.6 - 843.6, and a standard deviation of 138.3 mg.

2.1.2.Motor unit and innervation ratio

The functional unit of the neuromuscular system is the motor unit (Liddel and Sherrington, 1925).In the case of the larynx, this motor unit consists of the alpha motor neuron in the brainstem and all of the muscle fibers innervated by the axon of that motor neuron.Changes in tension of striated muscles are due to increases or decreases in the number and frequency of active motor units firing therein.The number of muscle fibers in a single motor unit varies widely for different skeletal muscles.As a general rule, a large muscle which is involved in relatively gross movements (e. g. the gastrocnemius) may include several hundreds of muscle fibers in a motor unit, whereas a muscle concerned with precise movements may have a small number of muscle fibers per motor unit.

The innervation ratio expresses the number of muscle fibers per motor nerve fiber : This may be computed by dividing the total number of muscle fibers in a muscle by

the total number of motor nerve fibers. In general, it is difficult to determine the exact number of motor nerve fibers in the nerve trunk since sensory (afferent) and small motor nerve fibers supplying the intrafusal muscle fibers are also present.Some authors assume that 40 % of the large sized nerve fibers are afferent (Goodgold and Eberstein, 1972).Each laryngeal nerve contains a proportion of myelinated afferent fibers, difficult to determine in humans, from a variety of sources, as well as many more that are unmyelinated (Wyke and Kirchner, 1976).

2.1.3.Laryngeal nerve fibers

In human nerves, alpha motor fibers are generally large myelinated axons the diameter of which measures up to 25 um.Laryngeal motor nerve fibers vary widely in diameter, but most of them appear somewhat smaller than those innervating other striated muscle (Wyke and Kirchner, 1976).Fig. II.1. shows the distribution of nerve fiber diameters in the inferior laryngeal nerve of a 65 year old man, two centimeters before the nerve's penetration into the muscle (1097 fibers) (Faaborg-Andersen, 1957).As a rule, small motor units are innervated by nerve fibers of smaller diameter than large motor units.

It is also important to notice that laryngeal motor fibers decrease in diameter by 4 - 6 µm as they traverse the

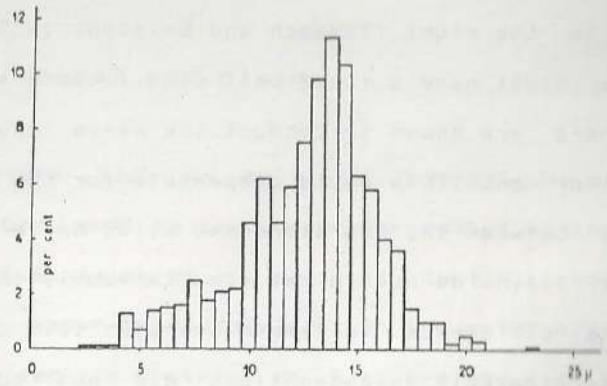


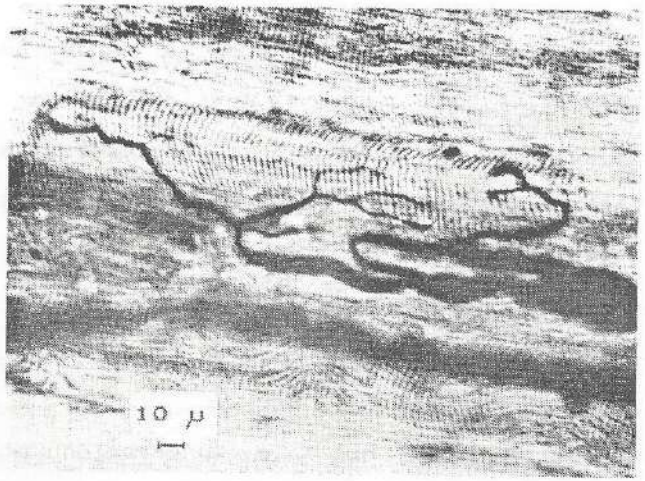
Fig.II.1.: Distribution of nerve fiber diameters in the inferior laryngeal nerve of a 65 year old man. (Faaborg-Andersen, 1957)

terminal muscular branches of the laryngeal nerves. This is confirmed by physiological measurements showing a slower conduction in the distal portion of the nerve compared to the proximal portion (Atkins, 1973).Some authors reported larger fiber diameters in the left inferior laryngeal nerve compared to the right (Tomasch and Britton, 1955; Krmpotic, 1957).This might have a functional significance since larger nerve fibers are known to conduct the nerve impulses faster than smaller ones.This would compensate for the differences in length between the two sides and allow nerve impulses to reach the larynx on both sides simultaneously (According to Peytz, the difference in length averages 11.5 cm).However, electrophysiological investigations did not support this concept that there is a greater velocity in the left inferior laryngeal nerve compared to the right one : Experiments suggest that the conduction velocity is the same in both nerves.These large fibers are mainly present in the vagus nerve, and the difference in diameter is practically imperceptible in the inferior laryngeal nerve near the larynx.This suggests that the larger fibers may not carry motor impulses to the intrinsic laryngeal muscles .Furthermore, if the average motor nerve conduction velocity is 60 - 70 m / sec, the delay in conduction between the two sides could not exceed the maximum difference in distance between the sides 18 cm divided by the conduction velocity 60 mm / msec.Now, the onset of electrical activation of the thyroarytenoid muscle appears about 350

msec before sound production.Thus it is obvious that the hypothetic delay is not a critical factor in voice production at all.Therefore, it is unnecessary to postulate a compensatory mechanism , and no such mechanism seems to exist (Atkins, 1973).

2.1.4.Laryngeal end plates

A possible dual or multiple innervation of the same muscle fiber must be considered for computing the size of a motor unit.Staining of end plates by Koelle's cholinesterase stain has shown that end plates in the cricothyroid muscles are localized on a strip, which covers about 15 per cent of the length of the muscle fibers (Faaborg-Andersen, 1957).In view of this finding, a dual innervation of any considerable number of muscle fibers in the laryngeal muscles seems hardly likely.Piquet and Barets (1960) find that motor nerve fibers of the vocal muscle very rarely divide in their terminal ending, in the vicinity of the motor plates, and this suggests that laryngeal motor units should be very small.Rudolph (1962) has observed muscular fibers of the vocal muscles presenting up to five separate neuromuscular junctions (Fig. II.2.and II.3.). The proportion of such (tonic ?) fibers was estimated about 20 %.Similarly, 2/3 to 3/4 of the muscle fibers of some extraocular muscles in humans have two or more motor end - plates (Kupfer, 1960). Rossi and Cortesina (1965) find that the proportion of



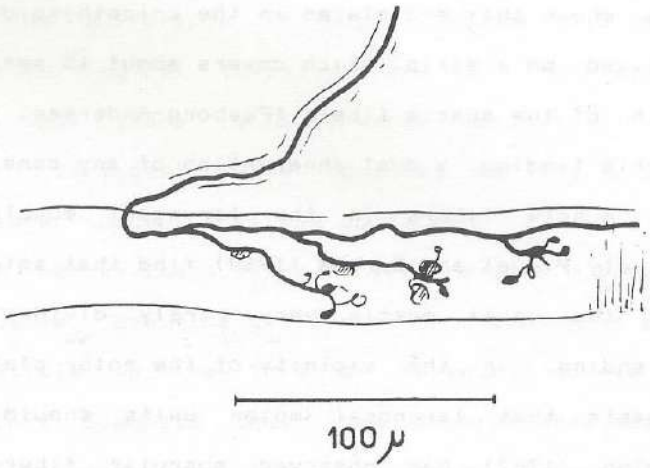


Fig.II.2.: Triple motor end plate in a human thyroarytenoid muscle (Photograph and drawing by Rudolph, 1959).

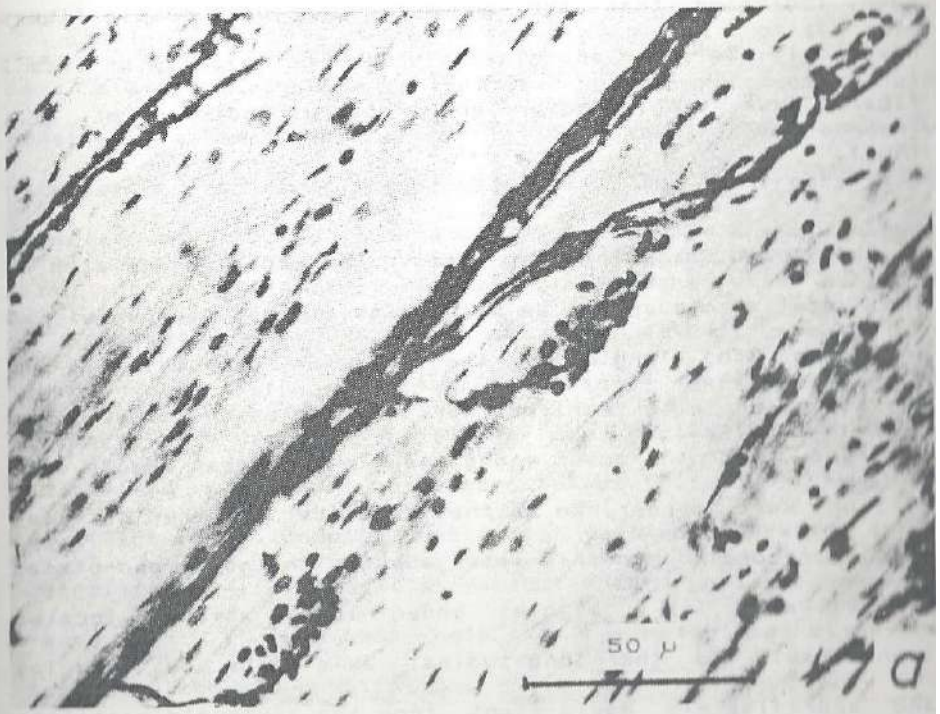


Fig.II.3.: Triple motor end plate in a human thyroarytenoid muscle (Rudolph, 1962).

muscular fibers with a multiple innervation reached 5 per cent in the posterior cricoarytenoid muscle and 70 to 80 per cent in the other intrinsic laryngeal muscles.It has also been indicated that the majority of type 1 muscle fibers have multiple motor end-plates, while the majority of type 2 fibers have single motor end-plates (Bendiksen et al., 1981).

Rossi and Cortesina also observe that motor end-plates of the vocal muscle appear as being disseminated over about two thirds of the length of the muscular body.This is in contradiction with earlier work of Sonesson (1960), who reported that the motor end-plates in the vocal muscle were of the same appearance as that described in other striated muscles.According to this later author, the motor end-plates are situated in a single, broad zone, which is located transversely to the longitudinal axis of he vocal muscle, and approximately equidistant from the arytenoid cartilage and the anterior part of the thyroid cartilage.The motor end-plates of the lateral cricoarytenoid muscle could also be identified in a zone situated posterior and lateral to that of the vocal muscle.

More recent work by Rosen et al. (1983) in cats and humans shows that the end-plates are diffusely located throughout the thyroarytenoid muscle rather than limited to a band or any clustered configuration (histochemical

localization of neuromuscular junction acetylcholinesterase).

Gambino et al. (1985) demonstrated that that the motor end plate distribution in the human posterior cricoarytenoid muscle is somewhat more localized than in the randomly scattered pattern observed in the thyroarytenoid muscle.Rossi and Cortesina (1965) find only a small number (5%) of posterior cricoarytenoid fibers with multiple end-plates, while Yashihara et al. (1984) describe no evidence of such multiple end-plates.A somewhat scattered pattern of motor end-plates may partially reflect a scattering of motor unit territories that would permit an architecturally complex system for tension generation and a corresponding functionally varied motor unit sequence of recruitment : this could help the fine motor control needed for phonation and respiration.

De Vito et al. (1985) investigated the cricothyroid muscle : its end plates consistently appear to be located in the medial two thirds of the muscle in the anterior to posterior direction, in contrast to the arrangement typically seen in most other human skeletal muscles where they are found in a narrow band at the midpoint of the muscle.The authors also observed that the motor end-plates appear to be in groups within the muscle, with different groups being associated with different muscle fiber

directions.Rossi and Cortesina (1965) estimated that approximately 50 % of the muscle fibers contain two or three motor end-plates per muscle fiber.A substantial number of fibers have end-plates separated by distances greater than 50 µm.

From another point of view, it must be remembered that a motor fiber may, in theory, divide anywhere along the nerve trunk (Piquet and Barets, 1960).

2.1.5.Laryngeal muscle spindles

Further, some of the motor fibers of the laryngeal nerves supply the intrafusal muscle fibers in the muscle spindles (Faaborg-Andersen, 1957) : It is now well established that each of the intrinsic laryngeal muscles (in the cat and man) contains a few small muscle spindles (even as spiral endings) (Wyke and Kirchner, 1976), although they are more numerous in man than in other species (Fig.II.4.and II.5). The fiber diameter spectra of the inferior laryngeal nerve and the external laryngeal nerve embrace fiber sizes of the same order as those of the fusimotor nerve fibers (1 - 6 um) found in muscle nerves in other parts of the body (Wyke and Kirchner, 1976).

For the sake of completeness, it must be noticed that the laryngeal nerves also contain small diameter vasomotor and

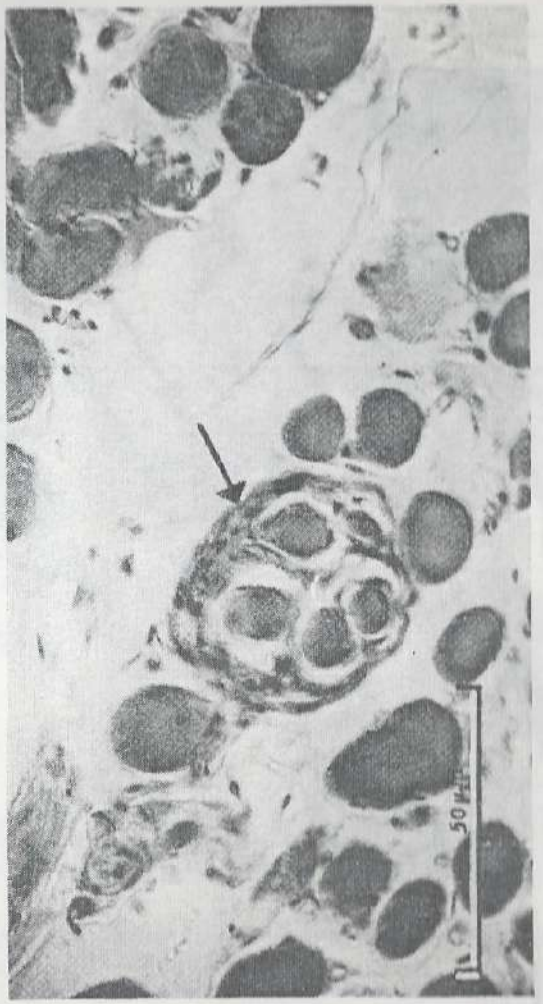


Fig.II.4.: Neuromuscular spindle in a thyroarytenoid muscle (Baken, 1971).

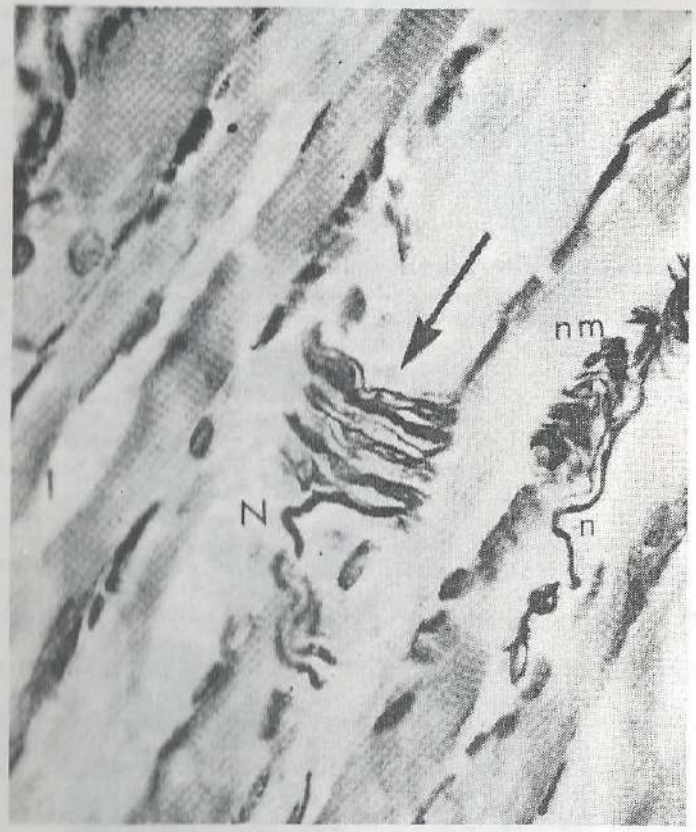


Fig.II.5.: Spiral ending (arrow) in a cricothyroid muscle.The afferent myelinated fiber is indicated N.A neuromuscular junction (nm) is also shown, with its terminal motor fiber (Baken, 1971).

secretomotor fibers of sympathetic (superior and middle cervical ganglion) and parasympathetic (dorsal nucleus of vagus) origin (Mitchell, 1954).

2.1.6.Size of laryngeal motor units

All these considerations contribute to explain some discrepancies between the sizes of the motor units of the intrinsic laryngeal muscles, as different authors hypothetized.Data available for humans are summarized in table II.4. The sizes and functional characteristics are probably not uniform throughout the laryngeal musculature, and even within individual muscles (Wyke and Kirchner, 1976).

2.1.7.Contraction characteristics of laryngeal muscles

As assumed by Wyke and Kirchner (1976), the intrinsic muscles with the fastest contraction times, such as the vocal and lateral cricoarytenoid (contraction times 12 - 18 msec in the cat) probably consist mainly of small motor units, with less than 20 fibers per unit, whereas muscles with slower contaction times, such as the cricothyroid and posterior cricoarytenoid muscles (contraction times 28 - 48 msec in the monkey and 30 - 50 ms in the dog and cat) probably consist of a higher proportion of larger motor

Author	Year	Nb. of muscle fibres/motor unit	Muscle
Faaborg-Andersen	1957	166	Cricothyroid
Faaborg-Andersen	1957	247	Transverse arytenoid
Faaborg-Andersen	1957	116	Posterior cricoarytenoid
Piquet and Barets	1960	1-3	Vocal
English and	1969	24,3*/32,1**	Cricothyroid

*If 66,6 % of the nerve fibres are considered as motor fibres.

**If 50 % of the nerve fibres are considered as motor fibres.

Table II.4.: Intrinsic laryngeal muscles : Number of muscle fibers in a motor unit, after different authors.

units containing perhaps up to 200 - 250 diffusely distributed fibers per unit.However, it is probable that the latter muscles also contain small, fast contracting motor units.

There are differences in contraction characteristics of individual intrinsic laryngeal muscles in response to stimulation of their motor fibers : maximal motor unit activity is obtained in the posterior cricoarytenoid muscle at relatively low frequencies (20 - 40 Hz) of stimulation of motor fibers in the inferior laryngeal nerve, whereas stimulus frequencies of 30 - 70 Hz are necessary to generate maximal activity in the vocal fold adductors.At a constant stimulus frequency, variations in stimulus voltage have no marked differential effect (Nakamura, 1964; Takenouchi et al., 1968; Kotby and Haugen, 1970).Furthermore, in response to protracted stimulation of their motor nerves, the fatiguability of the intrinsic laryngeal muscles is much less than that of limb muscles in the same species (cat) (Edström et al., 1973).

Kersing and Jennekens (1977) show that in vocal fold musculature type II fibers are found in a relatively larger proportion than in skeletal muscle.In vocal fold musculature, there probably also exists a larger variability in fiber diameter than in skeletal muscle. Type I fibers are slow in contraction time, fatigue - resistant, and have an

oxydative metabolism ,while type II fibers have faster contraction times. Type IIa fibers are adapted to an aerobic metabolism, but conserve a glycolytic activity.In type IIb fibers energy is provided by glycogenolysis.The muscle fiber composition of the posterior cricoarytenoid muscle consists of one-half type I, while most of the remaining fibers are type IIa.This mixed fiber composition allows continuous sustained activity (type I), while the type IIa fibers are capable of abducting the vocal folds via the glycolytic pathway in situations of hypoxemia, when vocal fold abduction is imperative (Hast, 1967 ; Crumley, 1982).

2.2. Extrinsic laryngeal muscles

Few data are available concerning motor units of the extrinsic laryngeal muscles : Hast (1968) studies canine contraction characteristics of the sternohyoid and thyrohyoid muscles, both of which have larger contraction times (50 - 52 ms) than those of the intrinsic laryngeal muscles.

Extrinsic laryngeal muscles (in contrast to the intrinsic muscles) are abundantly provided with muscle spindles (Wyke and Kirchner, 1976).

Chapter III

Basic principles of neuromuscular physiology applied to laryngeal electromyography

3.1. The electric signal

Electromyography is the detection and recording of electrical activity from a portion of a muscle ; The source of this electrical activity must be considered as related to the structure and the function of the muscle fiber membrane ; specifically its ability to store electrical charges and release bursts of electrical energy when properly stimulated.

The resting potential of a muscle fiber is due to

1. The fact that at rest the cell membrane is selectively more permeable to potassium ions than to sodium ions

2. The fact that an active transport mechanism maintains the internal Na+ concentration at a low level, and

3. The fact that the membrane is impermeable to intracellular organic anions.

The resting potential is the potential difference between the inside and outside of the cell : the outside is positively charged and the inside negatively. This can be measured directly by means of an intracellular micropipette electrode and is about - 90 mV.

When the transmembrane potential is reduced below a threshold level, changes occur in membrane permeability, which are specific to excitable cells.Once the membrane has been depolarized above the threshold, a rapid rise and fall of potential occurs, which is called action potential. The generation of the action potential is, in this condition, an automatic phenomenon, no longer controlled by the depolarizing stimulus.In response to a depolarization above threshold, the membrane of the muscle fiber becomes highly permeable to Na+ which, consequently flows inward.The influx of Na+ further reduces the internal negativity which, in turn, permits more Na+ to flow inward. In this way, a self-regenerating chain is established between depolarization and sodium permeability.The result is a complete reversal of the elecrical polarity of the membrane, that is, from positive to negative on the outside of the cell membrane and from negative to positive on the inside.Then, the influx of Na+ decreases back to its resting

level as abruptly as it had begun, usually after about 1 msec.This decrease and the accompanying increase in K+ permeability bring the membrane rapidly to its original polarized state.Finally, permeability to K+ returns to its resting level.

The entire sequence of these bioelectrical phenomena at the site of stimulation, including depolarization of the membrane and its recovery, occurs within a few milliseconds.This action potential, once initiated, propagates along the whole muscle fiber, away from the stimulus site, without decreasing in amplitude.In fact, as a consequence of the potential difference between the depolarized region and adjacent inactive regions, current will flow from the depolarized region to inactive regions, through both the intracellular and the extracellular fluids, forming a complete local circuit.This current acts to depolarize adjacent inactive regions of the membrane to the threshold, and in this way action potentials are generated in regions unaffected by the initial stimulus.The action potential thus self propagates along the entire muscle fiber (Hodgkin and Horowicz, 1959; Ruch and Patton, 1965; Goodgold and Eberstein, 1972).

When a voluntary contraction occurs in a normal muscle, all the muscle fibers innervated by a single lower motor neuron act together. The small action potentials of

individual muscle fibers are summed up and produce a larger action potential, which is called a single motor unit potential (Hirano, 1981).

3.2. Gradation of the muscular contraction

The weakest possible tension that could be developed by a muscle would be produced by a single action potential propagating along one axon and activating all the concerned muscle fibers, in other words, the tension developed by one motor unit in response to a single stimulus. If more strength is required, more motor units may be activated owing to the recruitment mechanism, which will allow considerable variations in the mechanical output.But tension developed by a muscle can also be graded by varying the frequency of discharge of each active motor unit.

At a higher frequency, the motor unit twitch tension summates to produce a tetanic contraction and an increase in force.As a general rule, and within limits, the higher the firing frequency, the larger the developed tension.In somatic muscles, recruitment is the predominant factor for strong contractions while for rather weak efforts, the tension is graded by varying the firing rate.For example, the frequency range for the abductor pollicis brevis has

second and 7 - 24 spikes / second for the biceps (Bigland and Lippold, 1954; Clamann, 1970).In the case of vocalis muscle, the signification of recruitment of new motor units and pulsating at a higher frequency is analyzed in Chapter VI.

Under normal conditions, each motor unit is activated at random intervals and different motor units are activated asynchronously.In extremely strong contractions, or in fatigue, different motor units may pulse synchronously (Hirano, 1981).

As a general rule, strength of contraction under many conditions is directly proportional to the mean amplitude of the integrated action potentials (Lippold, 1952).The mean amplitude gives an indication of the number of muscle fibers activated per unit time (Buchthal, 1959).

The force developed by an excited muscle fiber also depends on muscle length at the time of activation : The maximum force is developed when the fiber is at its mean resting length.At shorter lengths, less force is developed.At the resting length, the maximal force depends on the cross-sectional area - that is to say, on the number of muscle fibers available for activation (Fink, 1975).

3.3. Recorded extracellular potentials

Local current flows generated by action potentials will spread throughout the whole muscle, according to the volume-conducting properties of the electrolytic medium surrounding the fibers.

When a recording electrode is located near the fiber surface at a short distance from the point of stimulation, and a second "reference" electrode is at a point distant from the first, as depolarization at the excited region proceeds, the outward flow of current through adjacent regions of the membrane makes the active electrode more positive with respect to the reference electrode.The region where the active electrode is located acts as a source of current.Thus, the first result of the impulse approaching the electrode is a positive deflection.A short time later, the region of the active electrode will reverse its polarity ! now current flows from the adjacent regions into this area.The active electrode will thus become negative with respect to the reference , electrode, and a negative deflection is observed.This positive-to-negative deflection is very steep in slope and corresponds to the sharp rise of the intracellular action potential.It is followed by an absolute refractory period of about 1-2 msec and by a

the active electrode repolarizes, it again acts as a source of current.The recorded potential progresses again from negative to positive.As the impulse moves further along the fiber, the current flow ceases to influence the active electrode, and the observed potential slowly returns to the original base line.The complete potential change is thus triphasic with a steep linear positive-negative deflection (Goodgold and Eberstein, 1972; Hinzelin, 1979).

The shape of the recorded potential may vary to a large extent with the position of the electrode with respect to the active fiber. Furthermore, as a general rule, the amplitude of the extracellular potential is much less than that of the intracellular potential.

The motor unit potential recorded in the electrolytic medium surrounding the fibers is the sum of the electrical activity of several muscle fibers (except the case of motor units with only one muscle fiber), which may be geometrically scattered.Thus, for a given position of the active electrode, each muscle fiber differently contibutes - both in shape and in amplitude - to the global action potential.More distant fibers produce small and low-frequency contributions, while fibers located close to the electrode contribute with extended and high-frequency signals.A practical consequence is that the peak - to - peak

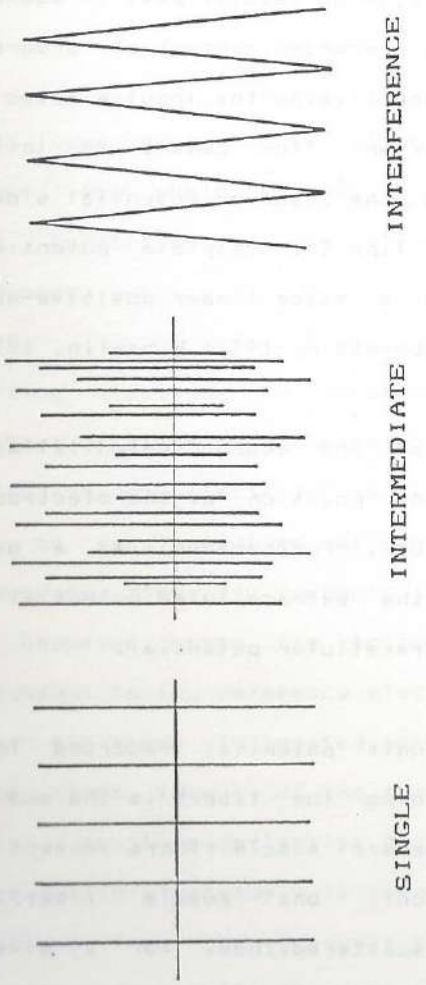


Fig.III.1.: Gradation of the muscular contraction, as recorded with intramuscular electrodes (schematically). Left : single motor unit potential pattern. Middle : More motor units are activated, with also a higher firing frequency of each active unit (intermediate pattern). Right : With maximal effort, individual potentials cannot be distinguished anymore, and the baseline is invisible (interference pattern).

potential will be determined by a few muscle fibers in the immediate vicinity of the active electrode.The voltage range of the electrical activity in the laryngeal muscles is approximately 0.003 - 2.5 mV.

The gradation of muscular contraction in normal conditions corresponds to the schematic electromyographical patterns of Fig.III.1.

Conventionnally, in electromyography, positive deflections are downwards.

3.4. Microphonic effect

(van den Berg and Spoor, 1957; Spoor and van Dishoeck, 1958; Greiner et al., 1958; Faaborg-Andersen, 1965; Dejonckere, 1980)

Besides the normal aperiodic action potentials, it is also possible to record, during phonation, a so-called microphonic effect in the vocal muscles (Fig. III.2.).This microphonic electrical activity has some specific features :

1. It can be obtained only with rigid electrodes : We never observed it with hooked wires.

subject, either normal or pathologic, who is able to generate laryngeal vibrations.

3. In a normal subject, the microphonic effect is easiest to obtain when the top of the needle-electrode is located very near to the free edge, in the region of Reinke's space, or when the electrode is in contact with either the lower edge of the thyroid catilage, or the upper edge of the cricoid cartilage.

4. Pathological subjects in which this effect is most evident are patients with chronic polypoid laryngitis (Reinke's oedema), and patients with one vocal fold completely denervated in median or paramedian position.

5. The microphonic effect arises as soon as the vocal folds begin to vibrate, and ceases when vibration ends.

6. There is a perfect synchronism between the microphonic effect and the sound oscillogram of the uttered voice.

7. The microphonic activity may have either a sinusoidal pattern, or a more complex shape.

8. The microphonic effect can be superimposed on normal or pathological asynchronous muscle action potentials, in a

superimposed on fibrillation potentials and slow denervation potentials, without voluntary muscle activation during phonation (Fig. III.2. and III.3).

9. The microphonic activity can sometimes be recorded alone, without any other electrical activity.

10. The amplitude of the microphonic effect is variable, but it may reach 0.5 mV (Dejonckere, 1980) and even more.

The microphonic effect is passive in nature : it is caused by passive simultaneous deformation of all muscle fibers and their membranes as a result of the vibration of the vocal folds.This microphonic effect is similar to the well-known Wever and Bray effect of the cochlea and the microphonic effect of the semicircular canals (van den Berg and Spoor, 1957).

However it does not seem possible to obtain a microphonic effect in excised larynges oscillated artificially. Living and polarized membranes are probably indispensable.

The microphonic effect is thus to be considered as an artifact, from an electromyographic point of view, but it may be used as a reference-signal for physiological investigation (Dejonckere, 1980).It may also contribute to

Fig.III.2.: Microphonic effect, with a sinusoidal pattern, in a case of denervation.

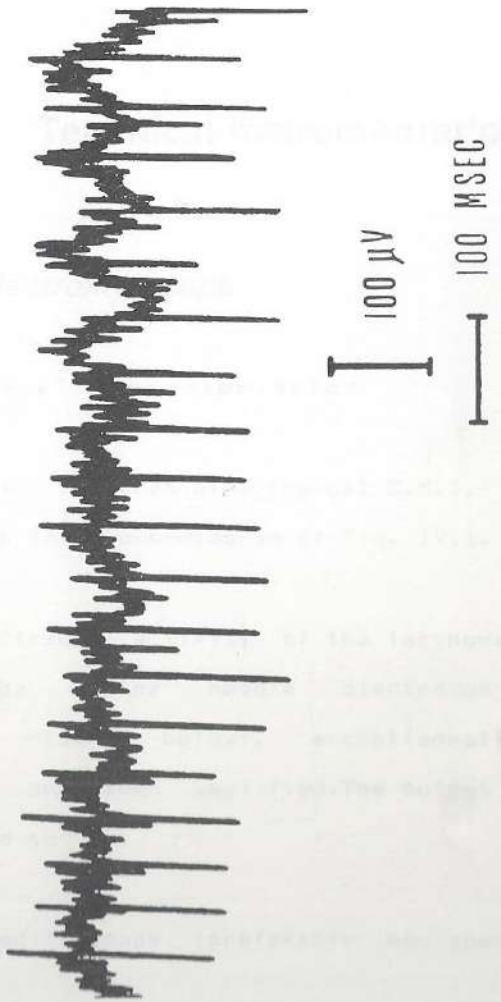


Fig.III.3.: Microphonic effect, with a superimposed single motor unit activity.Both electrical activities are obviously asynchronous.

denervation tracing and without concomitant voluntary action potentials.

Chapter IV

Technical instrumentation

4.1. The electromyograph

4.1.1.General characteristics

The basic features of a typical E.M.G.- installation are presented in the block-diagram of Fig. IV.1.

The electrical activity of the laryngeal muscles can be recorded by either needle electrodes or hooked-wire electrodes (see below), exceptionnally by surface electrodes, and then amplified.The output of the amplifier is connected to

- an oscilloscope (preferably equipped with a storage facility)

- a system of photographic recording

- an audio-amplifier with loudspeaker, to allow acoustic monitoring of potentials, and possibly a tape recorder.

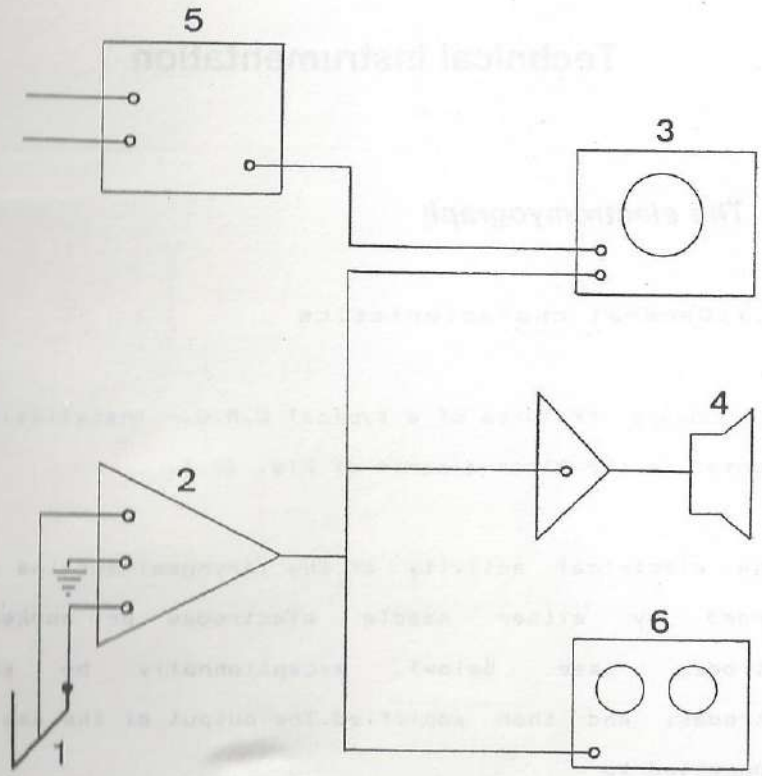


Fig.IV.1.: Basic features of a typical E.M.G.-installation.
1.Electrode 2.Amplifier
3.Oscilloscope with camera 4.Loudspeaker
5.Stimulator 6.Tape-recorder

- an integrator circuit.

An electrical stimulator is synchronized with the recording system.

The amplifier must satisfy rigorous requirements :

1. High frequency uniform voltage gain over the whole frequency range

2. Frequency range of 2 - 20.000 Hz

3. Differential input

4. High input impedance and low input capacitance

5. High common mode rejection

6. Low inherent noise.

4.1.2.Input impedance

It is the ratio of voltage applied at the input over current drawn by the amplifier.This input impedance depends upon the resistance of the amplifier input to the flow of current and is measured in Ohms.For most applications, the

impedance of the electrode.The input impedance of the E.M.G.-amplifier needs to be larger than 2 megohms to ensure distortion-free recording.

4.1.3.Differential input

A differential amplifier is characterized by its rejection of voltages which are identical, both in amplitude and time, at its two input terminals and the amplifications of the voltage difference between the two input terminals.An E.M.G.-electrode which is inserted into a muscle will pick up action potentials, but also mains - hum : The latter, if passed through an ordinary single-ended amplifier, would be amplified along with the muscle potentials.When a differential amplifier is used, in-phase signals arising from power lines or from sources outside the electrode field, which would normally interfere with the muscle potentials, will be rejected by the amplifier.

4.1.4.Frequency response

The frequency response is the speed with which a given instrument can respond without distortion to voltage changes applied at its input.For example, in electromyography, variations of 100 uV may occur in less than 1 msec.A common method for testing the frequency response of an amplifier is to compare the amplitude of the output with that of the

input for sine waves of varying frequency.For electromyography of intrinsic laryngeal muscles, a frequency-response from 2 Hz to 20 000 Hz is necessary, but it is sometimes advisable to reduce the frequency-response in order to increase the signal-to-noise ratio.All the recording equipment which is connected to the amplifier must satisfy the same requirements.

4.1.5.The EMG 200 Electromyograph from F.J. Electronics (Denmark)

The EMG 200 Electromyograph is a three channel electromyograph with built-in high-pass and low-pass filters, and a linear rectifier followed by an integration circuit with smoothing filters.It is specially designed for research and diagnosis of speech dysfunctions (Fig.IV.2).

The electrodes (either needle, hooked-wire or surface) are connected to a probe which, owing to its small physical dimensions, may be fixed on the skin near the point of measurement.The stainless-steel base of the probe serves as a grounding electrode.The probe contains a dual FET input operational amplifier which serves as two independent voltage followers with high input impedance and very low output impedance.

The electromyograph cabinet contains an amplifier with

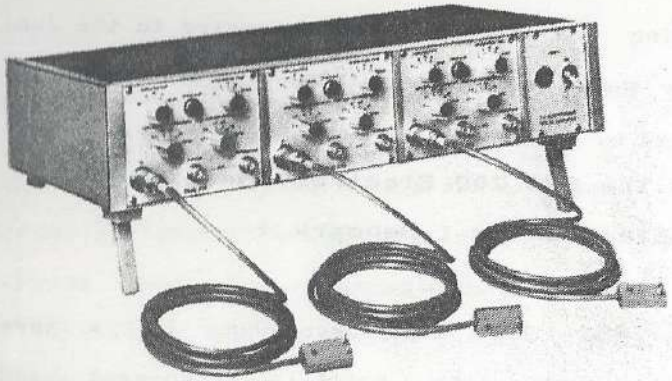


Fig.IV.2.: The EMG 200 Three-Channel Electromyograph from F.J.-Electronics (Denmark), specially designed for research and diagnosis of speech dysfunctions.

gain ajustable from 40 to 80 dB in 10 steps.An overload indicator indicates excessive input level and amplification.The frequency response of the amplifier system is limited to the range 2 Hz - 20 kHz in order to reduce the nois level.Furthermore, each E.M.G. channel has built-in low-pass and high-pass filters with a roll-off of 18 dB / octave.The cut-off frequencies are selected with special reference to speech analysis. The output is available both as amplified spikes (unintegrated and unsmoothed) and as an integrated signal with switch-selectable smoothing times.

Specifications

Probe amplifier : dual operational amplifier with FET inputs

Amplification : unity gain

Input impedance : 20 megohms

Output impedance : Less than 1 ohm

Frequency response : DC to 1 MHz

Main frame amplifier : 3 E.M.G. channels and +/- 12-volt power source

Input resistance : 2 200 ohms

Amplification : 40 - 50 - 60 - 70 - 80 dB switchable

Frequency response : 2 Hz - 20 000 Hz (-3 dB)

Low-pass filter : 18 dB / octave roll-off at 2.2 - 3.3 - 4.7 - 6.8 - 10 - 20 kHz

Rectifier : linear from 10 millivolt to 4 volt output

Integration : Active 18 dB / octave smoothing filter 2 - 5 - 10 - 20 - 50 ms.

Outputs : spikes and integrated

Output impedance : 75 ohms

Output level : Max. 4 volts peak-to-peak

Output current : Max. 20 mA

4.1.6.The Medelec MS6 Electromyograph

The MS6 - system includes a mainframe, together with plug-in modules suitable for various applications.The complete assembly is housed in a bench cabinet, and mounted on a trolley which also provides a loudspeaker and preamplifier mounting arm.This arrangement allows connections to patient electrodes to be kept very short, thus minimizing interference pick-up.The fiber optic cathode ray tube provides single frame and continuous recording on daylight developing or darkroom processed papers.The ultraviolet sensitive paper is held in close contact with the front-plate.Permanent records may be produced without any processing delay.Alternatively, by using full chemical processing, an enhanced contrast is achieved with archival permanence.A staircase time calibration waveform is generated in the mainframe.A built-in audio-amplifier enables displayed signals to be aurally monitored using the external loudspeaker (Fig. IV.3.).

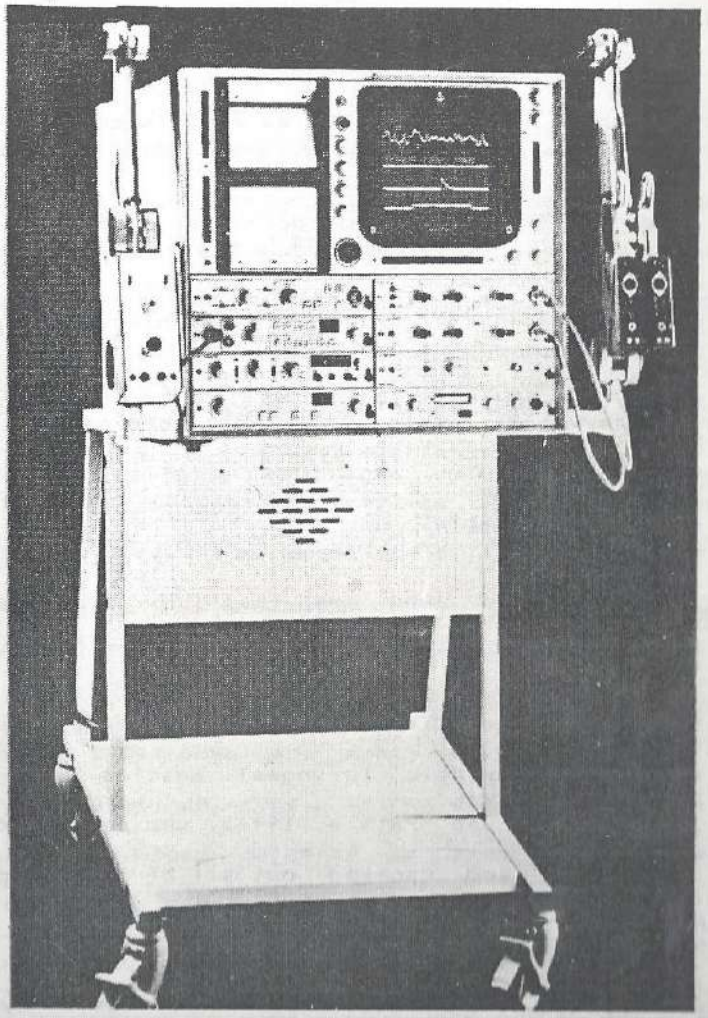


Fig.IV.3.: The Medelec MS6 Electromyograph : A classic all-purpose electromyograph with plug-in modules.

Specifications

Monitor display : 0.05 to 500 ms / division.Area 14 x 11.4 cm.
Paper speed : (for continuous or raster recording) : 0.5 to 100cm / s.Display area : 10 x 8 cm.
A.C. Coupled biological amplifier.Bandwidth selection over a wide range.
Isolated nerve stimulator : single or paired constant voltage pulses.Stimulus amplitude, duration, repetition and delay are all adjustable.

4.2. Electrodes

Historically, the first attempt to record motor unit potentials of intrinsic laryngeal muscles was made by Weddel, Feinstein and Pattle (1944), who used a concentric needle electrode and carried out all of the manipulations through a (direct) laryngoscope.

Several types of electrodes may be used for electromyographic investigation of the laryngeal muscles.

4.2.1.Surface electrodes

These electrodes pick up electric activity from a wide area of the skin.In this way,the signal represents the

global activity of all muscles in that area.

Such electrodes are very easy to apply,and cause no discomfort to the patient.The main disadvantage, particularly in the field of the small laryngeal muscles, is that on does not know exactly what activity is picked up.The signal is in fact the result of an integration over time and space of several muscles contracting beneath the skin.It is also impossible to observe single motor unit activity, and high frequency components are lost (Goodgold and Eberstein, 1972).Furthermore, the amplitude of the signal from surface electrodes is of lower level than that from other types of electrodes : The maximum is approximately 500 µV.

Surface electrodes are most conveniently made of small silver or platinum cups, with a diameter of 4 - 5 mm, and with a thin lead soldered to each cup.The leads are connected to the pre-amplifier.

After cleaning the surface electrodes by sanding or by an electrolytic treatment, the skin of the neck must be cleansed by a 50 % mixture of alcohol and ether.

Both electrodes must be smeared with EMG - jelly, and fixed onto the skin with tape.Theoretically, both electrodes are to be located on the cutaneous projection area of a

attached to the patient's wrist.

Practically, selective recording of the electrical activity of an individual - even extrinsic - laryngeal muscle does not seem possible, because several muscles of different sizes are overlapping and are all covered by the platysma.Conclusions about the activity of the crico-thyroid muscle during speech and singing,when recorded with skin electrodes (Lehmann, 1981), should be considered with some suspicion.

Nevertheless, skin electrodes may be very useful, and even specifically indicated, for recording of "general laryngeal area" muscle tension, in order to develop, for example, a biofeedback control (Stemple et al., 1980).In the latter case, one electrode is applied onto the region of the left thyroid lamina, the second one is attached to the right earlobe, and a ground electrode is fitted under the chin.

In order to investigate voice dysfunction in singers, we commonly use two surface electrodes (32 x 24 mm) applied onto the skin, at the level of both thyroid laminae.A grounding electrode is connected to the wrist.By this way,it is possible to have an indication about the dynamics of extrinsic laryngeal muscles as a function of pitch and loudness.

4.2.2.Needle electrodes

Three types of needle electrodes are to be considered (Fig. IV.4) :

4.2.2.1.Monopolar

A solid needle electrode, isolated except at the tip, is used in connexion with a surface reference electrode.Such electrodes are rarely used for recording electric activity of a muscle, but they may be suitable for stimulation of muscles or nerves.

4.2.2.2.Single core concentric

A cannula serves as a reference electrode and the bare tip of an isolated inner core in the cannula serves as the active electrode.The central wire is generally made of platinum and has a 0.1 mm diameter (Bronk's needle).The outside diameter of the electrode vary from 0.3 to about 1 mm.For laryngeal use, the optimum outer diameter is 0.55 or 0.6 mm.Smaller sizes are not rigid enough to perforate adequately the crico-thyroid or thyro-hyoid membrane in some older patients, while larger diameters become more painful.An electrode-length of 50 mm is necessary to reach the transverse and oblique arytenoid muscles.

Needle electrodes

1. Monopolar : These solid stainless steel needles have conically sharpened points and are insulated leaving only the tip of the needle as the electrode pick-up area.

2.Single core concentric : The platinum wire core is located within the stainless steel cannula by a dimethacrylate ester infill.Outer diameters vary from 0.3 to 0.65 mm, with lengths from 25 to 75 mm.

3.Bipolar (double core concentric) : This electrode has insulated Nickel/Chrome alloy cores located within the cannula of a stainless steel hypodermic needle by an epoxy resin infill.Core spacing is 0.014 or 0.20 mm.

Fig.IV.4.

The impedance of such electrodes ranges between 50 000 and 200 000 ohms at a measuring frequency of 150 Hz.Both the inside wire and the outside cannula are connected to the differential amplifier input terminals.

Potentials are measured between the tip of the platinum wire and the surrounding steel shaft.The tip angle of the cannula is 15 - 20 °, so that the recording area of the platinum wire is 0.03 mm2.

Standard concentric needle electrodes registrate activity of 10-20 muscle fibers (Mercelis, 1986).

The grounding electrode is generally a large plate electrode affixed to the skin.

The concentric needle electrode is the most suitable for recording single motor unit action potentials.A disadvantage of rigid electrodes is that they may interfere with natural phonatory or articulatory movements and also that they may move inside the muscle.

4.2.2.3.Bipolar concentric electrodes

Two isolated wires are cemented into the needle cannula.The two bare ends of the wires serve, one as an

active electrode, and the other as a reference electrode.The cannula is grounded.The use of bipolar electrodes is limited to detecting potentials from a smaller volume than when using concentric electrode.The recording area for each platinum wire is 0.03 mm3, and the distance between the midpoints of the two leading-off areas is 0.2 mm.

4.2.3.Wire electrodes

Wire electrodes have several advantages over needle electrodes due to their flexibility and lightness.Most kinesiologic studies use intramuscular wire electrodes (Nichols, 1970).They were extensively described by Basmajian and Stecko (1962), and by Hirano and Ohala (1969) for specific laryngeal use.Two thin isolated wires (35 - 50 u teflon isolated wires) with bare ends are threaded through a thin hypodermic cannula (e. g. N° 27).The free ends of the wires are hooked over the end of the cannula, and cut with a razor blade approximately 0.5 and 1 mm away from the tip of the cannula (Fig. IV.5.). At the other ends of the wires the insulation is scraped off for connection to the differential amplifier.A ground lead is placed on the ear lobe of the subject.The hooked wire electrodes are inserted into the muscle with the hypodermic needle, which is later removed, leaving the wires in place.

Hirano and Ohala (1969) used a 40 gauge copper wire,

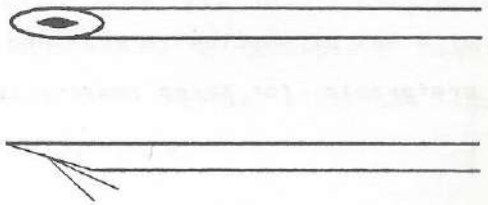


Fig.IV.5.: Hooked wire electrode : Two thin teflon isolated wires with bare ends are threaded through a thin hypodermic cannula.The free ends are hooked over the end of the needle, and cut 1 mm away from the tip.After insertion into the muscle, the hypodermic needle is removed, leaving the wires in place.

0.087 mm in diameter.Hirose (1971) adopted a platinum-iridium alloy wire with polyester coating, the diameter of which is approximately 0.05 mm.

4.2.4.Specific indications

As a general rule, when a kinesiological pattern of a muscle or that of a set of muscles is examined, hooked wire electrodes are preferable, for three reasons (Hirano, 1981)

1.They cause a minimum discomfort to the subject, and allow phonation and articulation in nearly physiological conditions

2.They stay fairly well in place regardless of movements of the investigated muscle and of the whole larynx

3.They allow considerable localization of the area from which electrical activity is recorded.

Contrarily, when vocal fold paralysis or paresis is investigated, i. e. disorders of the motor unit, a needle electrode should be used.The leading-off surface of the platinum core is about 0.03 to 0.07 mm2 : This corresponds to an area of approximately 2 mm diameter (Notermans et al.

In order to avoid interfering signals from adjacent muscles, Dedo and Hall (1969) and Dedo (1970) prefered a bipolar needle electrode.This electrode detects potentials from a smaller volume than the concentric electrode.

Action potentials may differ considerably in shape when recorded either with a concentric needle electrode or a bipolar concentric needle electrode (Notermans et al., 1981).

An important feature is that, in motor neuron disease, it is necessary to move the tip of the needle electrode inside the muscle during the investigation, because different motor unit patterns can be found in a given muscle.Especially when a spontaneous pathological restig activity is looked for, recordings must be made from different points within the muscle.

4.2.5.Stimulation electrodes

In our experience, needle electrodes are much more suitable for laryngeal nerves than surface electrodes (two metal rods encased in a plastic handle), although the latter are commonly used by Thumfart (1986).

needle (diameter 0.4 mm: length 50 mm), insulated except for a 3 mm segment at the tip.A similar reference electrode is placed subcutaneously in the supraclavicular fossa.

Chapter V

Electrode positioning

5.1. Methodological aspects

Electromyography of laryngeal muscles may be performed in six different ways :

5.1.1.Direct approach through a surgically preformed pharyngostoma or laryngofissure :

This method is rather exceptional, but has historical interest (Portmann et al., 1955 ; Spoor and van Dishoeck, 1958). Greiner et al. (1960) compared recordings obtained by this direct approach and by the trancutaneous way, and they found that they were similar and concluded that the "blind" way was valuable.

5.1.2.Per viae naturales, through direct laryngoscopy :

Early work, by Weddel et al. (1944) was performed by this way.However, Shipp et al.(1970) still used this method, considering that direct laryngoscopy affords greater maneuverability and illumination of the structures than

indirect laryngoscopy.Both a topical application of cocaine solution and a neuroleptanalgesia are required.General anesthesia is not indicated, because the cooperation of the patient is necessary to induce voluntary activity.Either needle or hooked wire electrodes are appropriate.

5.1.3.Per viae naturales, through indirect laryngoscopy :

Faaborg-Andersen (1957) in his fundamental study on laryngeal electromyography managed to reach all the internal laryngeal muscles by needle electrodes passed through the mouth and secured in place under mirror control.The cricothyroid was approached through the skin.Several authors described equipment specifically designed for this purpose, as L-shaped probes (Hirose,1971), forceps (Guerrier and Bassères,1965) and applicators (Thumfart, 1981).The zoom laryngoscope enables long term supervision of the larynx with various viewpoints and magnifications: It constitutes an excellent instrument for the transoral application of electrodes (needle or hooked wire), with a technique similar to that used for taking specimens out of the larynx for histological investigation.Insertion of electrodes into the vocal muscle is quite easy after slight displacement of the false cord.The rear side of the arytenoid cartilage is used for orientation while inserting the electrode into the posterior cricoarytenoid muscle at the rear of the cricoid cartilage (Thumfart, 1981; 1983; 1986).

5.1.4.Percutaneous approach, through the soft tissues overlying the larynx :

Hiroto et al. (1962, 1967) and Hirano and Ohala (1969) demonstrated how it is possible to reach each of the intrinsic laryngeal muscles by this method.The subject lies on his back with his head tilted back.The skin of the neck is disinfected with alcohol.Surface anesthesia is applied to the laryngeal mucosae.Xylocain may be injected via the cricothyroid membrane.Either hooked wire or needle electrodes can be used.Initially, these authors proposed , in order to investigate the vocal muscle, to insert the needle into the subglottic cavity through the cricothyroid space on the midline.The tip of the needle is then tilted upwards and sideways, and penetrates the mucosa of the lower surface of the vocal cord.In fact, it is better to pierce the skin at a point close to the midline and to pass submucosally through the submucous tissue into the muscle (Hirano, 1981) (Fig.V.1. and V.2.)

For the lateral cricoarytenoid muscle, the needle is inserted through the cricothyroid space ,slightly laterally , penetrating the cricothyroid membrane anterior to the inferior tuberculum of the thyroid cartilage (Fig. V.3.). Then the needle is pushed posteriorly, slightly laterally,

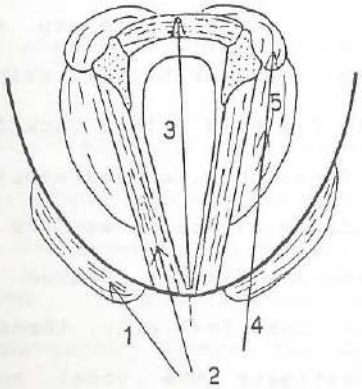


Fig.V.1.: Percutaneous insertion of the electrode into the intrinsic laryngeal muscles : Schematic superior view (after Hirano, 1976).For the posterior cricoarytenoid muscle, a curved needle is sometimes helpful.

1.Cricothyroid muscle
2.Thyroarytenoid muscle
3.Interarytenoid muscle
4.Lateral cricoarytenoid muscle
5.Posterior cricoarytenoid muscle

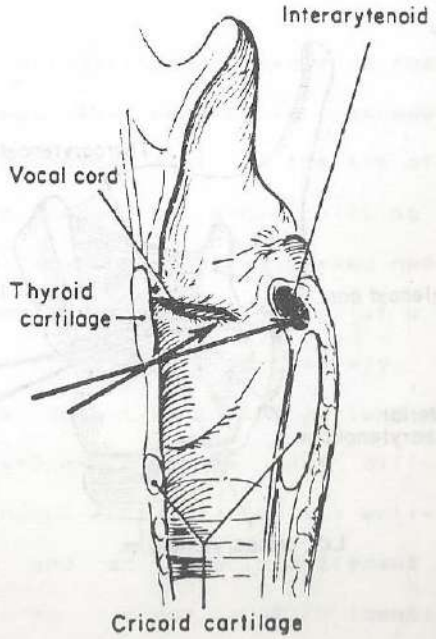


Fig.V.2.: Percutaneous insertion of the electrode into the thyroarytenoid and interarytenoid muscles, according to Hirano and Ohala (1969).For the vocal muscle, it is better to pierce the skin at a point close to the midline and to pass submucosally through the submucous tissue into the muscle (Hirano, 1981).Lateral view after sagittal section.

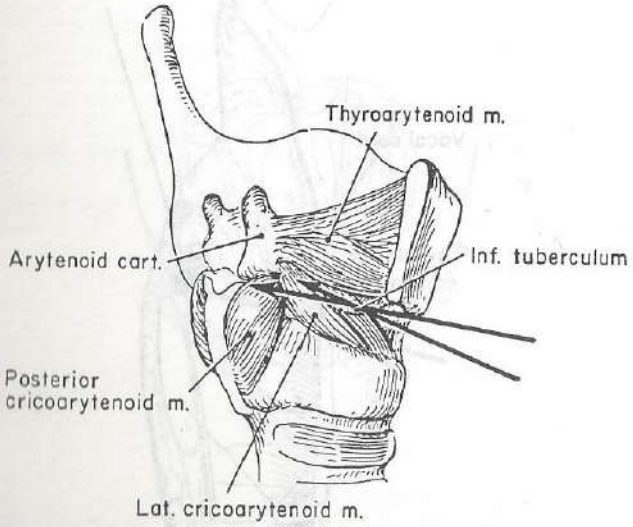


Fig.V.3.: Percutaneous insertion into the lateral cricoarytenoid muscle.Posterolateral view.Thyroid cartilage partially resected (Hirano and Ohala, 1969).

(Fig.V.4.). If the needle is directed medially or too far upwards, it may be placed in the external part of the thyroarytenoid muscle.

The posterior cricoarytenoid muscle is reached following the same path, but the needle is introduced 5 to 10 mm deeper.A slight downward tilt of the tip of the needle is often required in order to avoid hitting the arytenoid cartilage.In some cases, a slightly curved needle is helpful (Fig.V.5). The needle must be inserted at a right angle to the long axis of the muscle.Fortunately, the muscle is thickest near its insertion to the arytenoid cartilage.The posterior cricoarytenoid is the most difficult muscle to reach by percutaneous approach, but its activity pattern is so characteristic and so clearly different from all other muscles that no error of identification is possible.Nevertheless, in cases of denervation , an exact localization of the electrode is not possible.Furthermore, in some patients, anatomical configuration of the cartilages practically makes that this muscle cannot be reached by an percutaneous approach ; in such cases, the posterior cricoarytenoid will be reached by the transoral way.

For the arytenoid muscle, local anesthesia of the laryngeal mucosa is indispensable.The needle is inserted in the subglottic space through the cricothyroid space on the midline, and then pushed upwards and backwards.The anterior

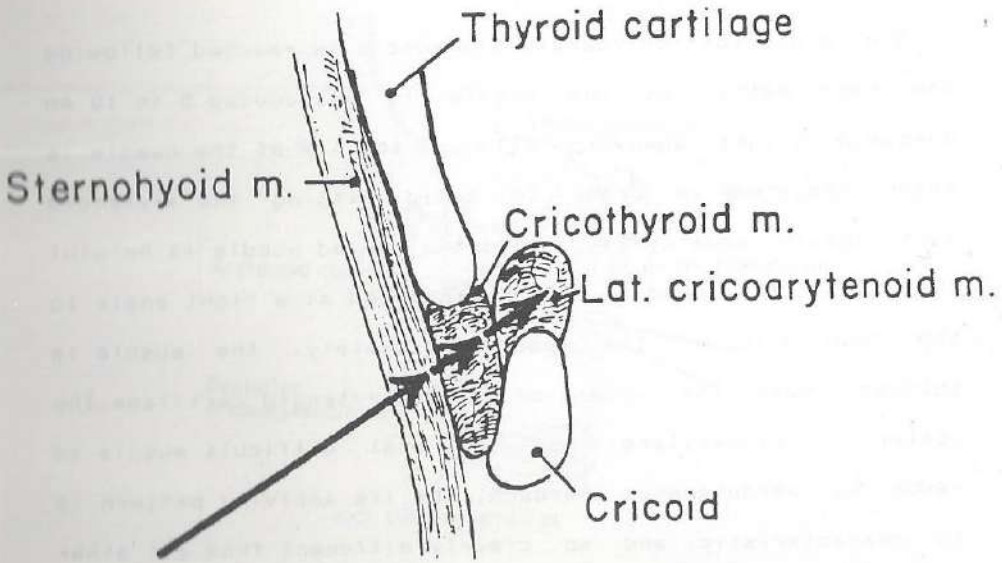


Fig.V.4.: Percutaneous insertion into the lateral cricoarytenoid muscle.Oblique cut along the needle in Fig.V.3.The needle-electrode can be placed in any of the sternohyoid, cricothyroid or lateral cricoarytenoid muscles depending on the depth of insertion (Hirano and Ohala, 1969).

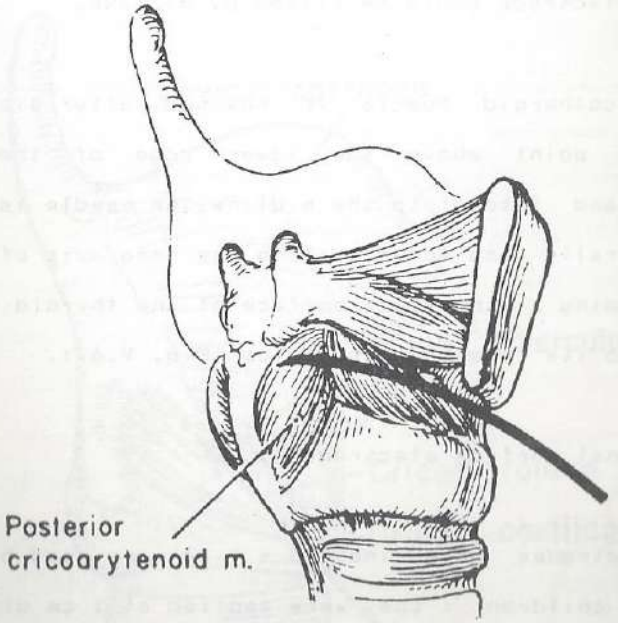


Fig.V.5.: Percutaneous insertion into the posterior cricoarytenoid muscle with a curved needle electrode (Hirano and Ohala, 1969).

wall of the inter arytenoid mucosa is pierced.The position of the needle can be monitored by indirect laryngoscopy (Fig. V.1. and V.2.). There is no neighboring muscle in which the electrode could be placed by mistake.

The cricothyroid muscle is reached after piercing the skin at a point above the lower edge of the cricoid cartilage and lateral to the midline.The needle is directed posterolaterally and upwards along the long axis of the pars obliqua aiming at the lower surface of the thyroid cartilage posterior to its inferior tuberculum (Fig. V.6.).

5.1.5.External surface electrodes :

Two electrodes measuring 0.5 x 1 cm were used by Lehmann (1981) in children : they were applied at 1 cm distance in the region of the cricothyroid space, on the right or left side.The author observed a minimal activity at a pitch corresponding to the usual speaking frequency, and an increase of this activity with rising pitch, possibly due to the cricothyroid muscle.When pitch was lowered, a even stronger activity was recorded.The latter was probably in relation with a contraction of extrinsic laryngeal musculature, tending to lower the thyroid cartilage.

Jacoby et al. (1984) recorded surface electromyograms

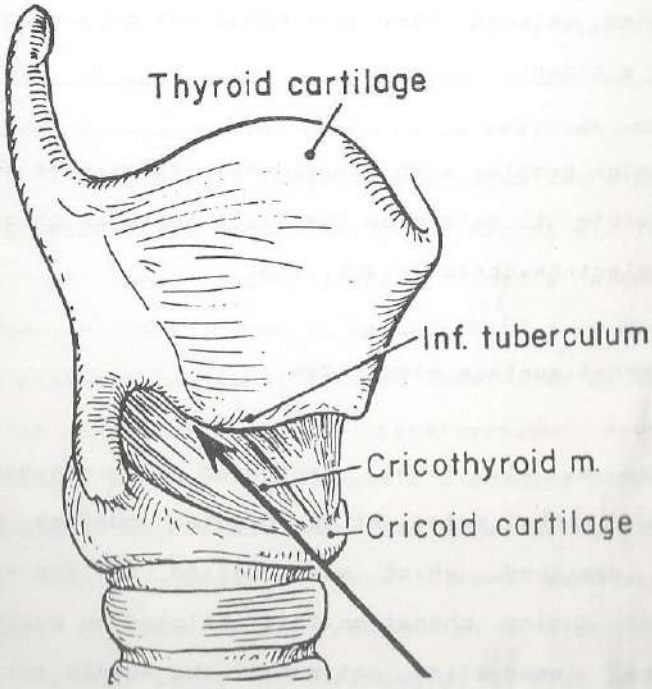


Fig.V.6.: Percutaneous insertion into the cricothyroid muscle (Hirano and Ohala, 1969).

laryngeal positioning, expiration and articulation activity during singing.The results indicate that surface electromyography is appropriate for the analysis of muscle groups involved in singing and that specific tendencies can be observed between the warming-up period and the fatigue period of a singer.

The major problem with surface skin electrodes is that it is impossible to determine the exact muscular origin of the recorded electromyographic activity.

5.1.6.Internal surface electrodes :

Lastovka et al. (1984) performed electromyography of vocal muscle by means of a bipolar surface electrode specially designed, which was applied on the vocal fold before and during phonation.This allowed an evaluation of the general innervation pattern of the muscle activity.The authors limited the use of this electrode to investigating of functional disorders of the larynx.

5.2. Clinical standard investigation

5.2.1.General principles

Contrary to kinesiological investigation, which is performed in a scope of physiological research or study of

functional disturbances of the larynx, clinical electromyography more specifically concerns laryngoscopic paralyses and pareses.For clinical purposes, the electromyographic investigation is commonly reduced to the exploration of both cricothyroid and thyroarytenoid muscles, in order to test both superior and inferior laryngeal nerves.Such an examination needs to be realized in a minimum time and with topical anesthesia on ambulatory patients.In our experience, and according to Greiner et al. (1960), the sampling of the thyroarytenoid muscle alone may be considered in most cases as representative of the activity of the intrinsic laryngeal muscles innervated by this nerve, except for isolated abductor paralysis.But even in the latter case, peripheral neurogenic signs are often found in thyroarytenoid muscles.Hirano (1976), reviewing 130 cases of complete and incomplete vocal cord paralysis, found significant differences in frequencies of electromyographic patterns neither among thyroarytenoid, lateral and posterior cricoarytenoid muscles, nor between the adductor and abductor muscles : This suggests that there should be no differences in vulnerability or recovery between muscles in the majority of the clinical cases. Furthermore, in cases of unilateral paralysis of the inferior laryngal nerve, the interarytenoid muscle always presented normal electromyographic findings.Berry et al. (1977) confirm that, for most laryngal abnormalities in which paralysis is present or suspected, it is probably adequate to examine

motor function within both the territories of innervation by percutaneous electromyography of cricothyroid and thyroarytenoid muscles, without sampling additional individual muscles.

In this scope, the technique of inserting the needle electrode percutaneously through the cricothyroid space (or exceptionnally through the thyrohyoid space) seems the most suitable (Dejonckere, 1976).

As a general rule, a very important point in our opinion is a systematic exploration of the whole muscle mass , by carefully moving the tip of the electrode, while looking at the oscilloscope, and listening to the sound produced in the loudspeaker by the electrical activity of the muscle.Fibrillation potentials, for example, are frequently picked up more easily, or even only, in some well individualized parts of the muscle.

Although the tip of the electrode remains submucosally, it is preferable to anesthetize the laryngeal mucosae, as well as the subglottic space, with a 4% xylocaine spray.This contributes to ralaxation of the laryngeal musculature.In patients with paresis or paralysis of the vocal fold(s), such an anesthesia does not significantly influence voice production. Exceptionnally, 0.5 mg atropine is given I.M. 15 minutes before the examination.

Fiberscopic visualisation is possible during the whole procedure.

Children are difficult to examine, except when they are tracheotomized or intubated.Even neonates may be investigated if intubated.

The patient is advised not to drink or eat for about half an hour after the examination.

5.2.2.Cricothyroid muscle

The arch of the cricoid cartilage is palpated, and the needle is inserted along the edge of the cartilage, slightly posterolaterally and upwards, aiming at the lower face of the thyroid cartilage.If the electrode is introduced beyond the inner face of the thyroid cartilage, it may be placed in the lateral cricoarytenoid muscle instead.On the other hand, if the insertion is not deep enough, the activity picked up may be that of the sternohyoid muscle (Hirano, 1981).It is also important that the tip of the needle be not in contact with the inferior edge of the thyroid catilage, because this may produce a microphonic effect at loud phonation.Correct electrode positioning is easily verified : In the normal muscle, transition from modal voice to falsetto elicits a considerable increase of the electromyographic activity.A

respiratory activity is always present in normal subjects (Dejonckere and Lebacq, 1983), but some motor units have an inspiratory activity, while others have an expiratory activity (Barillot and Bianchi, 1971).

5.2.3.Thyroarytenoid muscle

According to Nasser Kotby (1975),the vocalis muscle can be reached, during quiet respiration, by a needle passing through the cricothyroid space, parasagittally, at 6 mm from the midline, at an angle of about 40° upwards, backwards and cranially (Depth of the electrode tip : 18 mm). In females and children, we insert the needle closer to the midline (3 mm), and orient the needle more upwards (about 70°), and slightly laterally as soon as the cricothyroid membrane is traversed (Depth of the electrode tip : about 13 mm.When the electode is inserted laterally with respect to the midline, penetration of the tough part of the cricothyroid ligament is avoided, thus allowing easier maneuverability.

It is very important to perform all manipulations submucosally, and to avoid penetration of the sensitive mucosa of the subglottic space.

In the normal subject, the verification of correct electrode positioning is based on the following points :

- Electrical silence is practically impossible to obtain.

- There is a respiratory-linked rhythmical activity, which increases with forced respiration.

- A burst of motor unit potentials usually precedes (200-400 ms) the phonatory activity.

- High pitched sounds considerably enhance the electrical activity.

5.2.4.Morphological particularities of paralyzed larynges

Three characteristic morphologic features of unilaterally paralyzed larynges are of critical importance for electrode positioning :

1.Modification of the level (in frontal section) of the paralyzed vocal fold, with respect to the normal one (Fig.V.7)

2.A global rotation of the larynx in the neck

3.An "over-compensation" by the healthy vocal fold, overshooting the midline during phonation (Fig.V.8)

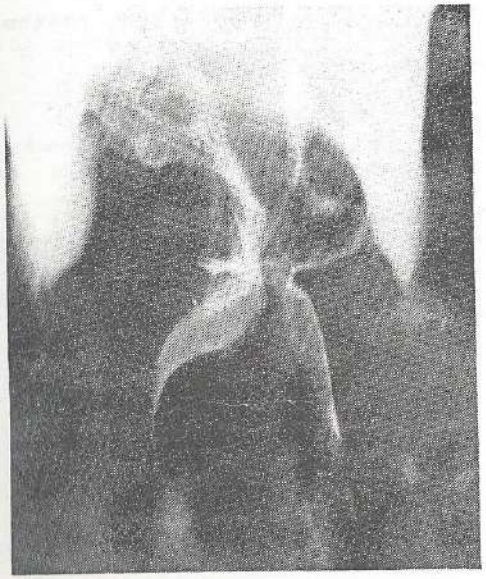


Fig.V.7.: Complete peripheral lesion of the left inferior laryngeal nerve, as demonstrated by laryngography (frontal view) during a phonation effort. Difference of level between both folds, and important atrophy of the paralyzed fold.

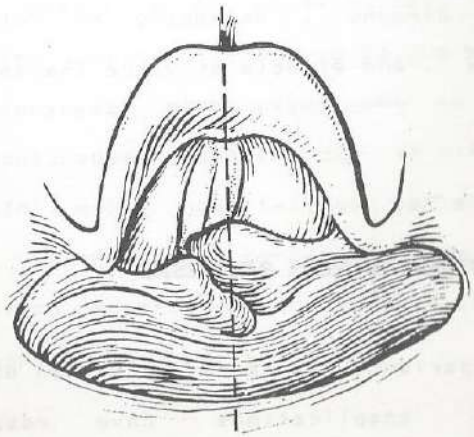


Fig.V.8.: Schematic illustration of compensation of a right vocal fold paralysis in intermediate position, as obtained by voice training. During phonation, the left fold overshoots the midline, in order to obtain an adequate glottic closure.

All these characteristics modify the classical transcutaneous technique, and require a careful clinical examination prior to electrodiagnostic procedures.

Muscular atrophy , asymmetry of intrinsic laryngeal muscle tonus , and effects of voice therapy are essentially responsible.

5.3. Undesirable effects and risks

In our experience (12 years: more than 650 examinations), no serious complications have resulted from an electromyography of laryngeal muscles.Most of the authors do not even mention any side effects or dangers.

A slight pain is usually experienced by the patient for 1 - 2 hours after the examination.

A limited local bleeding sometimes occurs in the vocal fold.A control laryngoscopy is always performed after the electromyography, and bleeding was observed in only about 1 % of the subjects.

Laryngeal spasm never occurred in our experience, but was related by Greiner et al. (1960).The authors recommended an

Oedema of the vocal cords after electromyography is probably systematic, but of minimal importance, and not perceived by the majority of subjects.Sometimes, patients with unilateral paralysis or paresis feel a temporary improvement in the quality of their voice.One must formally advise against laryngeal electromyography in out-patients presenting a spontaneous dyspnoea due to bilateral vocal fold paralysis.In such hospitalized patients, if the examination is imperative, corticosteroids should be administrated. Close supervision during several hours after the electromyography is indispensable.

Vagal reactions with cardio-circulatory repercussion are theoretically possible, but were never observed in our practice.

Vocal rest is to be recommended for some hours after the examination.

Chapter VI

E.M.G. of the normal larynx

6.1. Intrinsic laryngeal muscles

In the normal larynx, an electrical activity is generally present in the intrinsic laryngeal muscles during quiet respiration, in spite of efforts to achieve relaxation.Even when the subject holds his breath, this activity remains present.Its mean amplitude is up to 200 uV, and varies considerably from subject to subject, and even in a given subject.In the posterior cricoarytenoid muscle, the amplitude of this resting activity is more important : 200 - 500 uV (Faaborg-Andersen, 1957).

6.1.1.Thyroarytenoid muscle

6.1.1.1.Respiration

All authors agree that there exists a phasic activity related to the respiratory rhythm (Faaborg-Andersen, 1957, 1965: Kotby and Haugen, 1970: Dejonckere and Lebacq, 1984) (Fig. VI.1).Experiments in cats demonstrate that there are probably in all the intrinsic laryngeal muscles, inspiratory

Fig.VI.1.: Phasic respiratory activity in the thyroarytenoid muscle, in a normal subject.

and expiratory motor units (Barillot and Bianchi, 1971; Lucier et al., 1978).

Knutsson et al.(1969) found that in some motor units the discharge frequency is quite stable and independent of the respiratory phases.In other units, the frequency varies with respiration : some of such units discharge only during one of the respiratory phases, while others fire continuously, modulating their frequency with respiration.There are large fluctuations in the number of cyclically active units in different subjects.There are also interindividual variations with regards to the respiratory phase in which the units are active : In about one-third of the subjects the authors encountered only inspiratory units, in about one third only expiratory units, and in the remaining third both inspiratory and expiratory units in the same muscle.Faaborg-Andersen (1965) observed an increase in electrical activity during the inspiratory phase more frequently during forced respiration than during quiet respiration.

6.1.1.2.Phonation

In conditions of normal phonation, the electrical activity markedly increases (Fig.VI.2).As a general rule, this increase in activity begins and reaches its maximum before an audible tone is recorded by the microphone.This

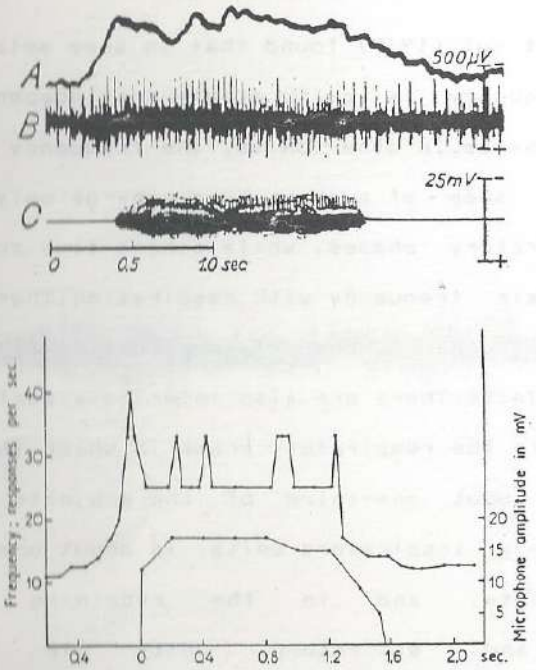


Fig.VI.2.:
Above : Thyroarytenoid muscle in a normal subject.Phonatory activity with normal E.M.G. action potential pattern (B), mean action potential amplitude (A), and microphone recording (C). Pitch : 357 Hz.Only a few motor units are recorded. The electrical activity increases before an audible tone is recorded by the microphone.
Below : Discharge frequency for a single motor unit from the record above (upper tracing) and amplitude of the microphone recording in mV (lower tracing) as a function of time is seconds (Faaborg-Andersen, 1957).

has been called the "pre-phonatory tuning" of the vocal fold.During voice emission, the electrical activity slightly decreases, but remains higher than in the absence of phonation.At the end of phonation, when audible tone ceases, the level of the electrical activity also decreases.Sometimes the increase in electrical activity disappears a little after the audible tone.The time interval between the start of the change in electrical activity and the onset of voice emission is 50 to 600 ms.This time interval is largest for vowel syllabes, and smaller for nasal, voiced consonant and voiceless consonant syllabes in decreasing order.

There is no significant difference in electrical activity for emission of different vowels.

In thyroarytenoid muscle the change in electrical activity begins earlier and is significantly larger in case of a "stopped" or "hard" attack than in a breathy attack.

In case of whispered voice, an increase in activity is recorded, but somewhat less than in case of ordinary phonation.The same effect is noticed with silent speech.This electrical activity in the thyroarytenoid muscle during silent reading is more important when the subject reads a text in a foreign language which he is not accustomed to read.

(Faaborg-Andersen, 1957, 1965; Greiner et al., 1960; Buchthal and Faaborg-Andersen, 1964; Hiroto et al., 1967; Shipp et al., 1970; Fink, 1975)

In the chest register, an increase in pitch is accompanied by increase in electrical activity in the thyro-arytenoid muscle (Faaborg-Andersen, 1957; 1965 ; Yanagihara and von Leden, 1966; Hirano et al., 1969; Gay et al., 1972).At a controlled constant psychophysiological sound level, the averaged electrical activity systematically increases in 15 normal subjects out of 15 for a pitch rise of a fifth above the usual speaking frequency (Fig.VI.3). The basic mechanism , for a minimal rise in pitch, seems to be a rise in the pulsing frequency of activated motor units, possibly associated with a recruitment by activation of initially silent proximal motor units (Dejonckere, 1981) (Fig.VI.4).

In contrast, the activity of the thyroarytenoid muscle decreases with the change from chest to falsetto voice (Faaborg-Andersen, 1957; 1965; Sawashima et al., 1969; Gay et al., 1972).Register is primarily regulated by the vocalis muscle (Hirano et al., 1970).

Silent singing of a well-defined interval reproduces the

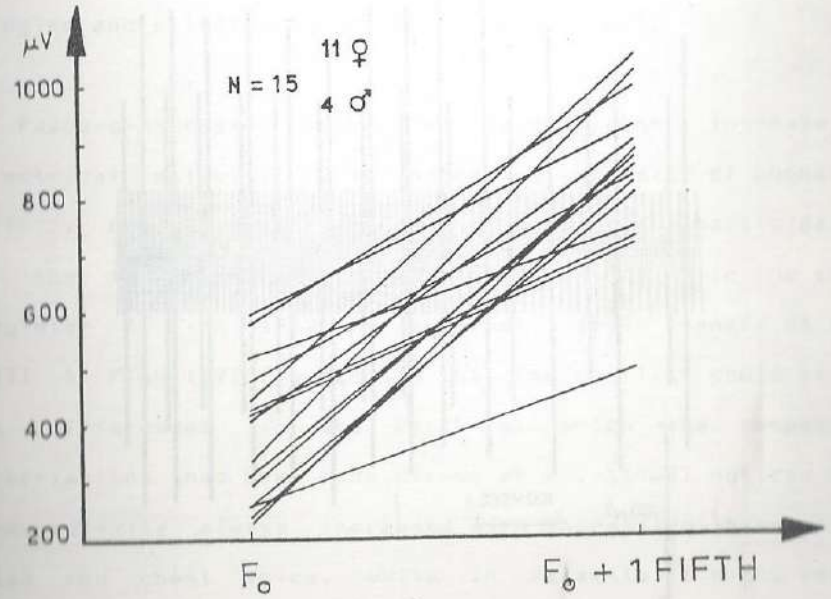


Fig.VI.3.: Averaged mean amplitude of the electrical activity in both thyroarytenoid muscles during phonation
1.On the usual mean speaking frequency (Fo)
2.One fifth higher than Fo.
Loudness is controlled at a constant 75 dB(A) at 20 cm from the lips.
15 normal subjects : 11 females, 4 males. (Dejonckere, 1981).

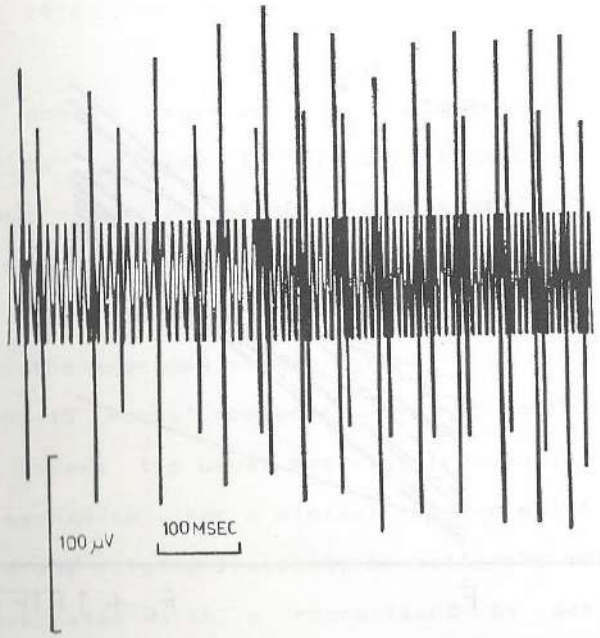


Fig.VI.4.: Schematic electromyographic recording of two motor unit potentials in a thyroarytenoid muscle during phonation, with a superimposed microphonic activity. The tip of the needle electrode is located near the edge of the vocal fold, and inserted superficially into the muscle. For a minimal rise in pitch, as shown by the microphonic effect, both activated motor units increase their pulsing frequency ; recruitment of other motor units may also occur (Dejonckere, 1981).

electrical activity is smaller, and the transitions are less clearcut. When either proprioception or auditive control are restricted in a subject singing normally, a pattern is observed with an amplitude intermediate between normal singing and silent singing (Dejonckere, 1982).

Faaborg-Andersen found no significant increase in electrical activity with increasing intensity of phonation (1957), but later, several authors observed a participation of the thyroarytenoid in regulating intensity in the chest register (Fink, 1962; Hirano et al., 1969; Vennard et al., 1971). Fink (1975) suggested that the conflict could be due to differences in the pitch at which the respective observations had been made. Hirano et al. (1969) noticed that the activity always increased with increasing intensity in head and chest voice, while in falsetto it might remain unchanged. Furthermore, intersubject variability is quite marked, as shown by Gay et al. (1972). At constant pitch (mean speaking frequency), 13 normal subjects out of 15 showed a slight increase in the averaged electrical activity (Fig.VI.5) ; The basic mechanism seems to be a recruitment of new motor units, although simultaneously the pulsing frequency of primarily active motor units simultaneously is accelerated (Dejonckere, 1981) (Fig.VI.6).

The vocalis muscle is the most important in regulating the style of phonation ; Its activity is highest for

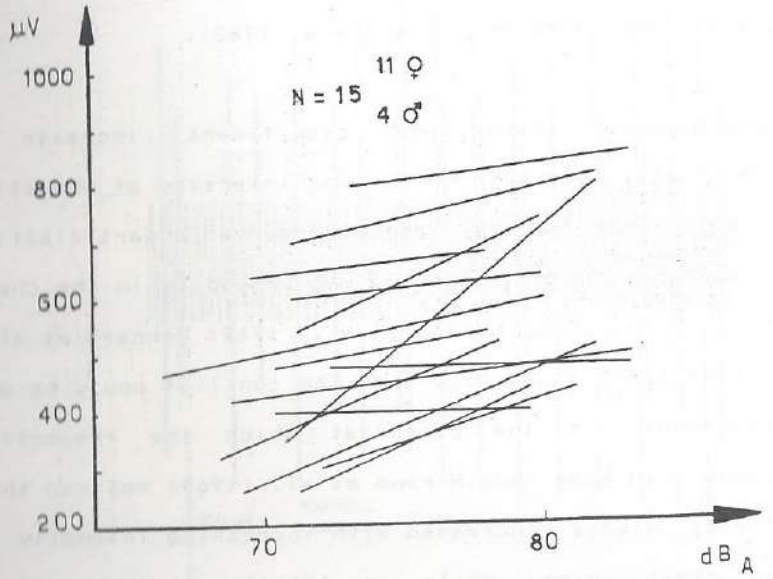


Fig.VI.5.: Averaged mean amplitude of the electrical activity in both thyroarytenoid muscles during phonation
1.At a loudness of about 70 dB(A)
2.About 10 dB(A) louder.
Intensity is measured at 20 cm from the lips.Pitch is maintained constant and corresponds to the usual speaking frequency (Fo) of the subject.13 normal subjects out of 15 show a slight increase in the electrical activity (Dejonckere, 1981).

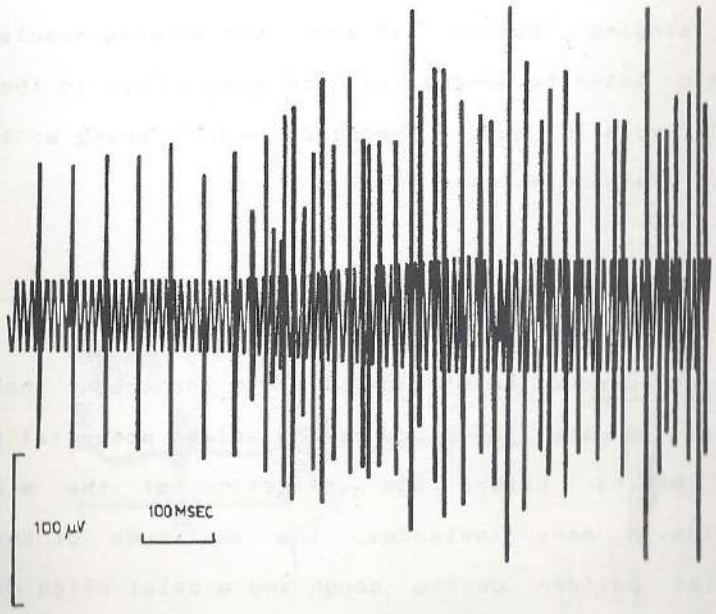


Fig.VI.6.: Schematic electromyographic recording of a single motor unit potential in a thyroarytenoid muscle during phonation, with a superimposed microphonic activity.The tip of the electrode is located near the edge of the vocal fold, and inserted superficially into the muscle.For a slight rise in loudness, as shown by the microphonic effect, there is a recruitment of new motor units, while usually the pulsing frequency of primarily active motor units is also accelerated (Dejonckere, 1981).

hypertense style, and smallest for hypotense style (Hirano et al., 1969).

In singing, during vibrato, the vocalis muscle shows a rhythmic activity.Generally, the energy peak in the vibrato of the vocalis muscle precedes the high phase of the pitch vibrato (Vennard et al., 1970).

6.1.1.3.Sphincteric action

During production of glottal click and cough, there is an increase in the amplitude of the action potential pattern, which begins before the deflection of the microphone recording.In many instances, the amplitude of the action potential pattern during cough and glottal click decreases somewhat when the audible tone begins as shown by the microphone recording, only to increase again and then to disappear about simultaneously with the disappearance of the audible tone.When the subject coughs, the interval between the onset of the increase in the action potential pattern's amplitude and the microphonic deflection is greater than with glottal click (Fig.VI.7).

During swallowing, there also appears a brief abrupt increase in muscle activity, without systematic difference with cough and glottal click (Faaborg-Andersen, 1957, 1965)

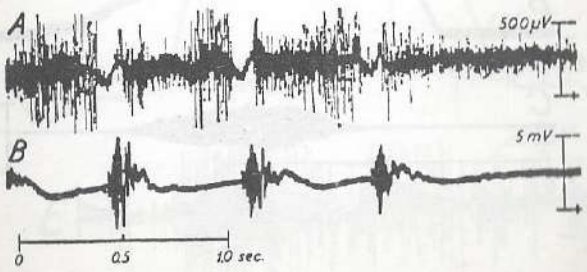


Fig.VI.7.: Thyroarytenoid muscle in a normal subject.Glottal click.Motor unit action potential pattern (A) and microphone recording (B).There is an increase in the amplitude of the action potential pattern before the sound is recorded (Faaborg-Andersen, 1957).

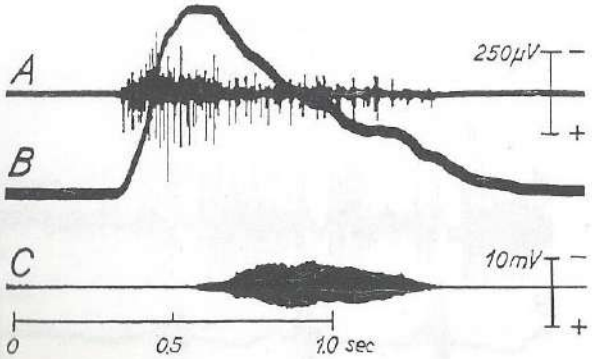


Fig.VI.8.: Thyroarytenoid muscle in a normal subject. Cough. Motor unit action potential pattern (A) , mean action potential amplitude (B), and microphone recording (C).Even as for phonation and production of a glottal click, the electrical activity precedes the sound emission (Faaborg-Andersen, 1957).

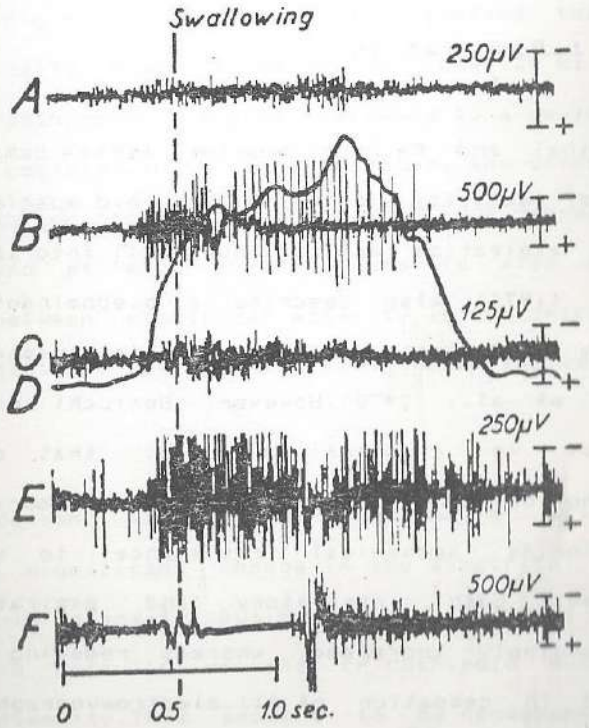


Fig.VI.9.: Swallow in a normal subject : Motor unit action potential in the

A and E : cricothyroid muscle
B : thyroarytenoid muscle
C : interarytenoid muscle
F : posterior cricoarytenoid muscle

Mean action potential amplitude in the thyroarytenoid muscle (D).

The onset of swallow is indicated by the vertical stippled line.

(Faaborg-Andersen, 1957)

6.1.2.Cricothyroid muscle

6.1.2.1.Respiration

Buchthal and Faaborg-Andersen (1964) demonstrate that a burst of activity in the cricothyroid muscle begins at the end of expiration and continues well into inspiration.Ganz et al. (1974) also describe a predominantly inspiratory activity . Dyspnoea stimulates this inspiratory activity (Suzuki et al., 1970).However, Horiuchi and Sasaki (1978) observed, in anesthetized dogs, that during eupneic breathing cricothyroid activity was primarily observed in expiration.As mechanical resistance to ventilation is increased, both inspiratory and expiratory activity correspondingly increased, whereas removing the resistance resulted in cessation of all electromyographic activity.In fact, a synergic activation of cricothyroid and posterior cricoarytenoid muscles allows maximal glottic dilatation (Konrad and Rattenborg, 1969; Horiuchi and Sasaki, 1978).Sutton et al. (1972) observed that the posture of the subject exerted a significant effect upon unit discharge.

6.1.2.2.Phonation

In the chest register, increases in fundamental frequency are accompanied by strong increases in electrical activity

of the cricothyroid muscle. (Faaborg-Andersen, 1957, 1965; Yanagihara and von Leden, 1966; Hirano et al. 1969; Gay et al, 1972) (Fig.VI.10).Arnold (1961) observed that this muscle practically began to be active above 150 Hz in male voice.The cricothyroid activity increased to a maximum with the falsetto register.In falsetto register, the cricothyroid exerts itself too greatly to change with each degree of the scale (Vennard et al., 1971). There is also a close correlation between electrical activity in the cricothyroid muscle and glottal resistance (Yanagihara and von Leden, 1966).

Faaborg-Andersen (1957) and Sawashima et al. (1968) mentioned no significant change in the electrical activity with changes in intensity, but Hirano et al. (1969) observed a decrease in activity of the cricothyroid muscle with increasing intensity.This appears to be necessary to keep the fundamental frequency constant, because the factors that increase the intensity can also increase the fundamental frequency.Sutton et al. (1972) found that, for a given pitch, no consistent changes in firing rate of motor units when the subject shifted from lowest to highest intensities.

The cricothyroid muscle is not essential to style regulation (hyper- or hypotense phonation) (Hirano et al., 1969).

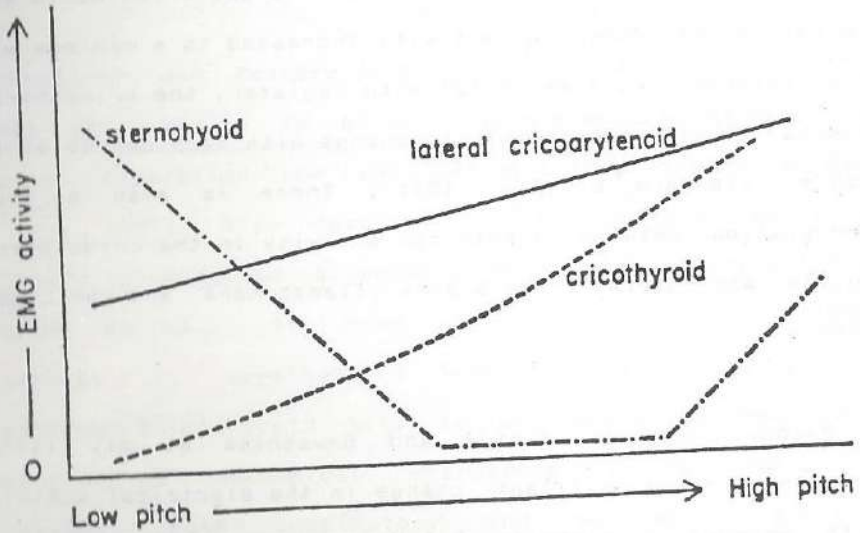


Fig.VI.10.: Schematic diagram of the pattern of activity of the sternohyoid, lateral cricoarytenoid and cricothyroid muscles as a function of pitch.These patterns are useful as a physiological basis of identification (Hirano and Ohala, 1969).

In whispered voice and silent speech, there is a rather slight activation of the cricothyroid muscle (Faaborg-Andersen, 1957, 1965; Zenker, 1960).

In singing, vibrato can be accompanied by a rhythmic activity of the muscle (Vennard et al., 1970).

6.1.2.3.Sphincteric activity

Modifications in action potential pattern amplitude are similar to those observed in the thyroarytenoid muscle (Faaborg-Andersen, 1957, 1965; Fink, 1975) (Fig.VI.9).

6.1.3.Lateral cricoarytenoid muscle

6.1.3.1.Respiration

No specific information seems available from the literature, but, in our own experience, the behaviour of this muscle is similar to that of the thyroarytenoid muscle.

6.1.3.2.Phonation

Hirano and Ohala (1969) as well as Gay et al. (1972) observed a slight increase of electrical activity with rising pitch (Fig.VI.10).

There is an active participation of the lateral cricoarytenoid muscle in regulating intensity in chest register, but with a nonsystematic pattern, and with a reduction in activity in falsetto (Hirano et al., 1969).The increase of activity with loudness is sharper at high pitch-chest (Gay et al., 1972).

This muscle also participates in regulating the hyper- or hypotense character of phonation (Hirano et al., 1969).

A hard attack is accompanied by a marked increase in the lateral cricoarytenoid activity during the prephonatory period.This seems to be related to the strong medial compression or constriction of the glottis prior to release (Hirose and Gay, 1973).

In singing, a rhythmic activity can be observed during vibrato.Generally, the energy peak in the vibrato precedes the high phase of the pitch vibrato (Vennard at al.,1970).

6.1.3.3.Sphincteric action

The lateral cricoarytenoid muscle is invariably active during a glottal stop (breath holding), and swallowing (Hirano and Ohala, 1969).

6.1.4.1.Respiration

A phasic activity, related with phonation, exists.Especially during forced respiration, there is an inspiratory increase in amplitude of the action potential pattern (Faaborg-Andersen, 1957,1965). Ganz et al. (1974) also described a predominantly inspiratory activity during normal spontaneous respiration (in lying down position, during microlaryngoscopy).

6.1.4.2.Phonation

There is a burst of activity just prior to and continuing throughout sustained phonation (Shipp et al., 1970). The activity is greater with whispered voice than with ordinary phonation (Faaborg-Andersen, 1957, 1965).Hirose and Gay (1972) found , during the production of voiced and voiceless consonants a reciprocal pattern when compared to the posterior cricoarytenoid muscle.

This muscle also usually participates in regulating the style of phonation (Hirano et al., 1969).

The arytenoid muscle seems to be the only intrinsic muscle which does not show a vibrato (Vennard et al., 1970).

6.1.4.3.Sphincteric action

Modifications in action potential pattern amplitude are similar to those observed in thyroarytenoid muscles (Faaborg-Andersen, 1957,1965: Hirano and Ohala, 1969: Fink, 1975).

6.1.5.Posterior cricoarytenoid muscle

An excellent review of electromyographic litterature was made by Zemlin et al. (1984).

6.1.5.1.Respiration

In his fundamental work (1957), Faaborg-Andersen reported a resting phase activity during respiration of 200 - 500 mV, a value much higher than that of other intrinsic laryngeal muscles at rest.A high resting potential is also reported by Kotby and Haugen (1970), who suggested that the posterior cricoarytenoid muscle was responsible for stabilizing the cricoarytenoid joint.

Faaborg-Andersen found also (1957: 1965) that, during quiet respiration, the amplitude of the action potential pattern is mostly reduced during inspiration, while during forced inspiration, there is an increase in amplitude of the action potential pattern over and above that of the resting

activity.During expiration, there is no change in electrical activity as compared with rest. Kotby and Haugen (1970) also reported inspiratory activity during forced inspiration.Shipp et al. (1970) confirmed that posterior cricoarytenoid activity coincided with the inspiratory effort.

An increase in electrical activity during inspiration is observed in most of the single units of this muscle in cat (Orem et al., 1978: Lucier et al., 1978).

Hiroto et al. (1967), Fukuda et al. (1973), and Sasaki et al.(1980) reported an increase in electrical activity of the posterior cricoarytenoid muscle in case of airway obstruction, thus suggesting that the muscle plays an active role in inspiration when the glottis is dilated.However, a minimum critical level of ventilatory resistance is essential for continued inspiratory contractions of the muscle : Tracheostomy abolishes this activity in anesthetized dogs (Sasaki et al., 1980), but phasic abductor activity can be re-established in three to five minutes by gradually increasing ventilatory resistance gradually (Buckwalter and Sasaki, 1984) (Fig.VI.11). In fact, the inspiratory resistance is primarily controlled by phasic abduction produced by the posterior cricoarytenoid muscle (Suzuki and Kirchner, 1969).

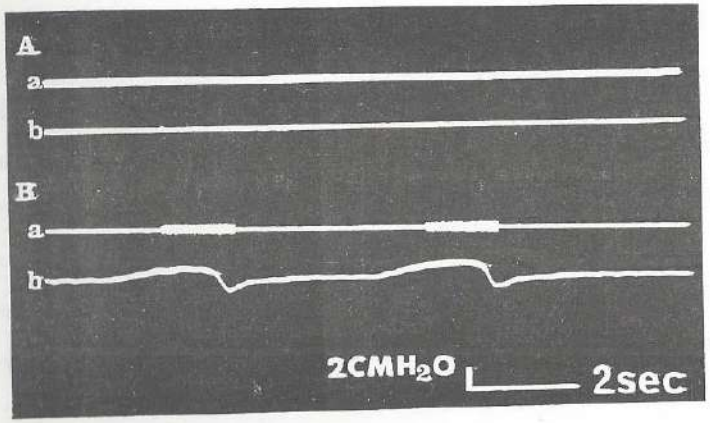


Fig.VI.11.: Laryngeal abductor activity in dog one week after tracheotomy. A.Tracheotomy open. B.tracheotomy partially occluded. a. Posterior cricoarytenoid electromyography. b. Intratracheal pressure.Abductor activity is absent with an open tracheotomy, but a slight posterior cricoarytenoid muscle activity can be elicited when the tracheotomy cannula is partially occluded.Three weeks later, closure of the cannula produces no return in posterior cricoarytenoid muscle activity (Buckwalter and Sasaki, 1984).

6.1.5.2.Phonation

As a general rule, and since Faaborg-Andersen (1957), all authors describe a decrease in posterior cricoarytenoid activity during phonation (Fig.VI.12).Hiroto et al. (1967) reported a sudden decrease in activity during phonation of vowels, nasals and voiced consonants, a decrease in activity at the end of phonation, and an increase again at the onset of phonation.Hirose and Gay (1972) and Hirose (1976) reported a participation of the posterior cricoarytenoid for voiceless consonants and suppression for voiced consonants, while Gay et al (1972) found a gradually decreasing activity for a breathy vocal attack and a sudden decrease before a simultaneous attack.

These latter authors also reported that the posterior cricoarytenoid muscle seems to regulate vocal fold tension since it is more active at the highest pitch levels; these results are inconsistent with those of Faaborg-Andersen (1957; 1965), who found a decrease in muscle activity with a simultaneous increase in fundamental frequency.

During silent reading, the activity of the posterior cricoarytenoid muscle is also inhibited (Faaborg-Andersen, 1965).

6.1.5.3.Sphincteric action

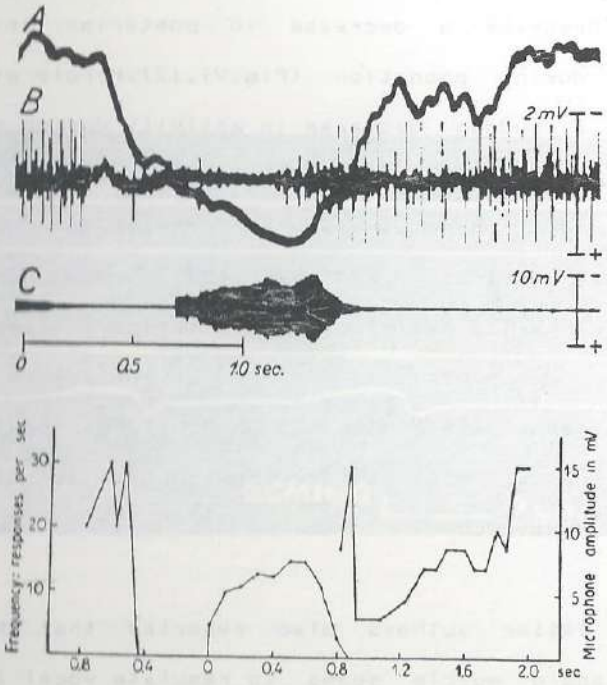


Fig.VI.12.: Posterior cricoarytenoid muscle in a normal subject.Phonation (pitch 333 Hz)
Above :
A : Mean amplitude
B : Action potential pattern
C : Microphone recording
Below : Discharge frequency for the single motor unit and amplitude of the microphone recording in mV (thin line) as a function of time in seconds (Faaborg-Andersen, 1957).

With cough, glottal click, and swallow, an inhibition appears in the activity of the posterior cricoarytenoid muscle (Faaborg-Andersen, 1965 ; Hirano and Ohala, 1969) (Fig.VI.9). Sasaki et al. (1973) also found an abrupt inhibition of action potentials just before sphincteric action.On the other hand, Kotby and Haugen (1970) reported a large electrical activity during sphincteric gestures and postulated that the posterior cricoarytenoid serves as a counterbalance for the forward action of the laryngeal adductors.

Zemlin et al. (1984) proposed an explanation for some inconsistent and paradoxical electromyographic results involving this muscle : The posterior cricoarytenoid muscle is to be considered not as one muscle mass, but as two separate bundles, one medial and one lateral, with a distinct tendon, each muscle part possibly performing a discrete and different function.The pattern of electromyographic activity should thus be dependent on the placement of the electrode.

6.2. Extrinsic laryngeal muscles

6.2.1.Respiration

Fink et al. (1956) found a faint activity in the

sternothyroid muscle, the sternohyoid muscle and the thyrohyoid muscle at rest, only slightly influenced by respiration.At forced respiration, the sternothyroid muscle showed pronounced activity during inspiration, while the thyrohyoid muscle shows pronounced electrical activity during expiration.

Omohyoid and sternohyoid muscles principally become active when partial airway obstruction and hypoxia are induced (Ellenbogen et al., 1981).

6.2.2.Phonation

Variations can be demonstrated in certain of the extrinsic laryngeal muscles as a function of the pronounced vowel : The hyoid bone and root of the tongue is pulled forward in closed vowels while it is pulled backward in open vowels (Faaborg-Andersen, 1965).

The mylohyoid muscle is active during silent speech and silent reading, especially in subjects with low reading ability (Faaborg-Andersen, 1965).

In the sternothyroid muscle, a moderate increase in activity was observed beginning approximately 0.5 seconds before phonation (Faaborg-Andersen, 1965).

As a general rule, in singers, extrinsic muscle activity tends to be directly pitch-correlated in the upper two octaves of the voice, and inversely pitch-correlated in the lower octave (Vennard et al., 1971). There is also an influence of voice quality, in relation with differences in laryngeal posture (Honda and Estill, 1986).

So, Hirano and Ohala (1969) found that the activity of the sternohyoid decreases with increasing pitch, at least in the chest register.For very high pitches, it increases again (Fig.VI.10).

However, Arnold (1961) found increasing activity in the sternothyroid muscle with rising pitch, the activity growing evenly through the full tone scale from low to high pitch.

Pahn et al. (1984) observed a relation between the activity of the sternothyroid muscle and vocal registers in singing : Activity increases when the larynx is in low position.

The sternohyoid muscle also assists in regulating the style of phonation (Hyper- or hypotense) (Hirano et al., 1969).

During vibrato in singing voice, Vennard et al. (1970) could observe in some cases a diadochokinesis between the

sternohyoid and the lateral cricoarytenoid muscles.Vibrato can appear in all the extrinsic laryngeal muscles (Vennard et al. , 1971).

6.2.3.Sphincteric closure

During coughing and straining efforts, the electrical activity increases (Fink, 1975).

6.3. Characteristics of laryngeal motor unit action potentials

6.3.1.Duration

The mean duration of single motor unit potentials from a defined muscle is a fundamental physiological characteristic of major clinical significance.

The motor unit action potential results from the summation of action potentials from several muscle fibers of this unit, the contribution from each fiber depending on its distance to the electrode.Potentials originating from fibers at a distance of more than 1 mm contribute to the low amplitude slow initial and terminal phases of the motor unit potential and add to its total duration.The dispersion of arrival times at the monopolar electrode depends on the spatial distribution of motor end plates for the different

fibers of the motor unit and the differences in conduction velocity.The different points of origin of the electrical impulses account for the spread of arrival times at the electrode, and thus for the total duration of the unit action potential.In most skeletal muscles, the majority of nerve endings are localized within a narrow band (Goodgold and Eberstein, 1972).

In general, motor unit potentials recorded with bipolar concentric needle electrodes are shorter in duration than those from monopolar concentric needle electrodes, because the distant potentials arise simultaneously at each pick-up electrode and cancel each other out (Goodgold and Eberstein, 1972).

Classically, motor unit potential duration increases with age (Goodgold and Eberstein, 1972).

Mean duration of motor unit action potentials in thyroarytenoid and cricothyroid muscle have been investigated in 50 normal subject, aged 5 to 77 years (21 males; 29 females).10 action potentials were recorded in each muscle with a concentric needle electrode.Material and methods are similar to those used in clinical routine (Medelec MS 6 Electromyograph).The duration is measured as the time interval between the first deflection from the baseline to the point at which the deflection finally

returns to the baseline.Results are shown in Fig.VI.13.

Statistical calculations demonstrate that:

1.The thyroarytenoid and cricothyroid muscles have significantly shorter mean durations of motor unit potentials than skeletal muscles.

2.There is no significant difference between right and left muscles.

3.Mean duration of action potentials increases as a function of age, and the regression line may be considered as effectively linear.The correlation-coefficient is significantly different from 0 ($p < 0.001$) for both muscles.Nevertheless, the slope of the regression line is less steep than that of facial muscles.

4.There is no significant difference between mean durations of action potentials of thyroarytenoid and cricothyroid muscles, although variances differ significantly ($p = 0.01$) : Variance is larger in the cricothyroid muscle.

Haglund (1973) found a mean value of 3.99 +/- 1.40 ms in the cricothyoid muscle (mean age 31).Knutsson et al. (1969) related for vocal muscles values of 3.43 +/- 1.03 ms (age

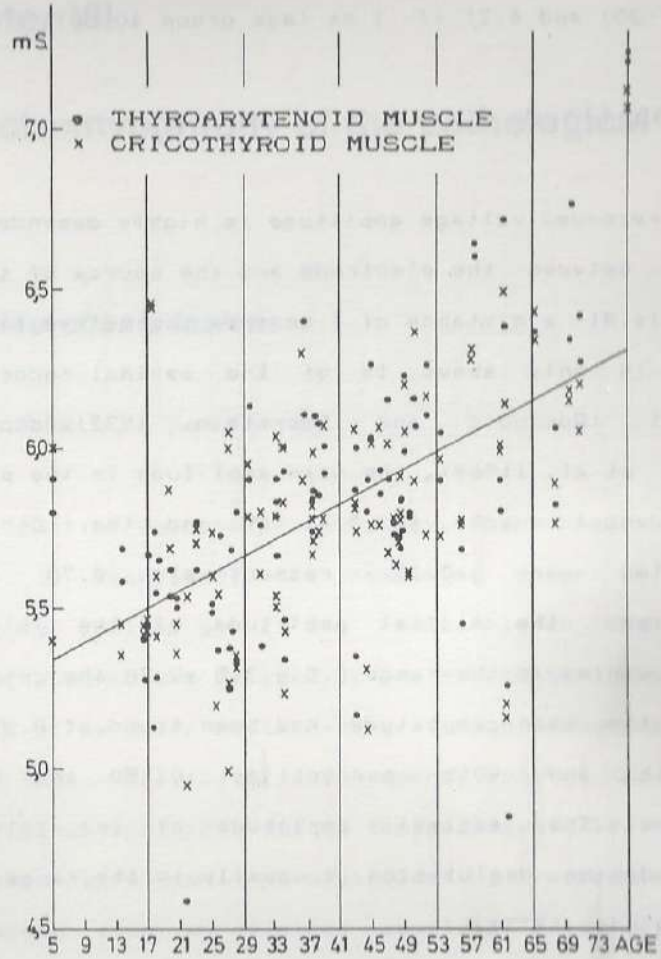


Fig.VI.13.: Mean duration of normal motor unit action potentials in thyroarytenoid and cricothyroid muscles of normal subjects, as a function of age.Each point represents the arithmetic mean of 10 motor unit action potentials sampled from one muscle.Each potential has been identified at least 10 times.

group 20-30) and 4.21 +/- 1 ms (age group 40-60).

6.3.2.Amplitude

The recorded voltage amplitude is highly dependent on the distance between the electrode and the source of the action potentials.At a distance of 1 mm from the active fibers, the voltage is only about 1% of the maximal recorded spike potential (Goodgold and Eberstein, 1972).According to Knutsson et al. (1969), the mean amplitude in the potentials of the vocal muscle is 0.41 mV and the 10th and 90th percentiles are 0.15 respectively 0.70 mV.During deglutition, the maximal amplitude of the interference pattern varies in the range 1.5 - 2.5 mV.In the cricothyroid muscle, the mean amplitude has been found of 0.394 mV and the 10th and 90th percentiles 0.150 and 0.800 mV respectively.The maximal amplitude of the interference pattern during deglutition is usually in the range of 1.2 - 2 mV (Haglund, 1973).

6.3.2.Shape

Polyphasic potentials may be observed in normal muscles, but they are rare in cricothyroid muscles (1 %) (Haglund, 1973).In vocal muscles, 5% are polyphasic (Knutsson et al., 1969).

Chapter VII

Electromyography of the pathological larynx

7.1. Neurogenic lesions

7.1.1.E.M.G.- patterns

In partial neurogenic pathology, when the number of motor units is decreased, the remaining motor units , when activated, pulse at firing rates higher than usual. In extreme cases, the pattern at maximal contraction is made by one or a few motor units firing at high frequency (Mercelis, 1986) (Fig. VII.1.).Spatial recruitment of new motor units has become impossible due to the neurogenic lesion, and only temporal recruitment persists.Such a reduced activity may correspond with a clinical complete palsy of the vocal fold.In partial lesions of laryngeal nerves , a high frequency pattern of one or two motor units may be observed only at the time of the pre-phonatory burst.

In case of degenerative paralysis, the most important immediate criterion of a complete lack of voluntary activity is the electrical silence in the electromyogram.About 10 days after onset of degeneration, pathological spontaneous

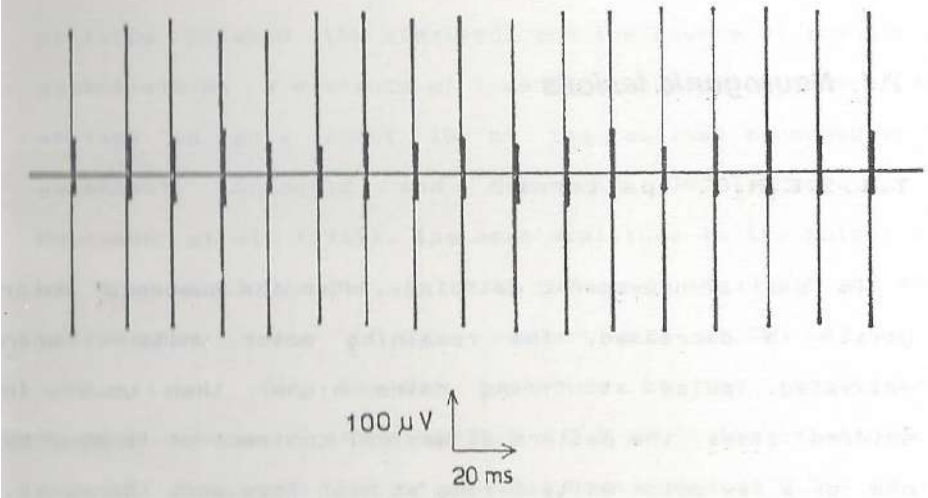


Fig.VII.1.: Single motor unit potential pulsing at high rate frequency (> 25 cycles per second) during strong contraction, indicating that several but not all motor units are lost (schematic).In a normally innervated muscle, strong contraction is always accompanied by recruitment of other motor units.

potentials begin to appear, such as fibrillation potentials (Fig. VII.2.) and positive sharp waves (Fig. VII.3.).Although still controversial, increasing evidence indicates that the lack of the neuromuscular transmitter, acetylcholine, is the cause of the spontaneous denervation potentials.Other mechanisms have been proposed to explain the occurrence of fibrillation potentials : spontaneous oscillations of the membrane potential of denervated muscle fibers trigger propagated spikes whenever the depolarization reaches treshold: functional changes in the sarcoplasmic reticulum, as shown by the increased calcium binding by the sarcoplasmic reticulum in denervated guinea pig muscle , as well as alterations in the muscle fiber membrane may be the cause of fibrillations in denervated muscle (Goodgold and Eberstein, 1972).It is most likely that fibrillation potentials originate from a single muscle fiber.They produce a particular , easily identified, sound in the loudspeaker, and pulse regularly at a frequency of 1-10 / second.Positive sharp waves are considered to originate from single muscle fibers and are probably detected near a damaged region of the fibers.The frequency of discharge is usually around 10 / second .Fibrillation potentials and positive sharp waves may also be observed in dystrophic muscle : They are not absolutely pathognomonic of peripheral nerve lesion (Goodgold and Eberstein, 1972).

If the lesion is partial, these denervation potentials

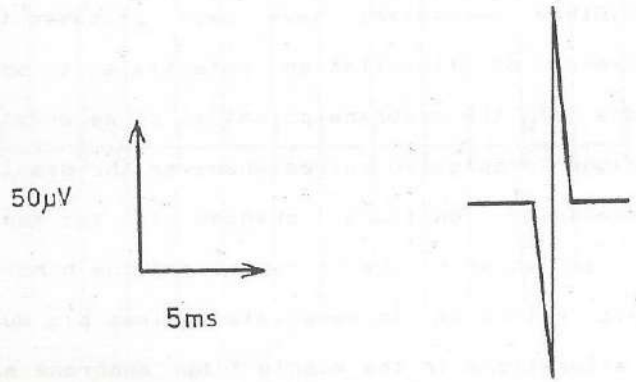


Fig.VII.2.: Fibrillation potential, arising from an individual muscle fibre (schematic) : It is of short duration, variable but rather small amplitude, and bi- or triphasic shape with initial positive phase (out of the endplate zone).

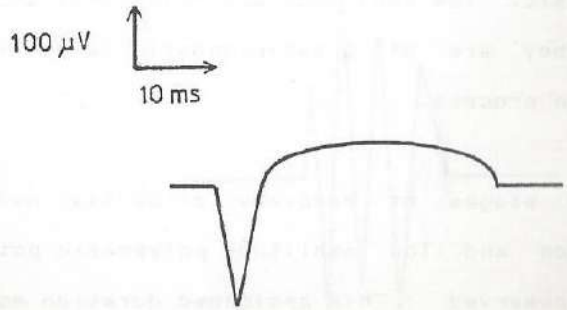


Fig.VII.3.: Positive sharp wave (schematic), with a sharp positive onset and a slow return to the baseline.

are recorded simultaneously with single motor unit potentials, or reduced voluntary activity, when the subject tries to produce a maximal contraction.

After several weeks, especially in cases of initially complete denervation, nascent motor units may appear : They are polyphasic, low amplitude and relatively short duration potentials.They are of great pronostic value, indicating a reinnervation process.

In early stages of recovery of partial nerve lesions, long duration and low amplitude polyphasic potentials are frequently observed : This prolonged duration most probably depends on increased temporal dispersion of the neuromuscular transmission, and the consequent spreading in time of arrival at the recording electrode of the action potentials propagated in different muscle fibers.Another factor of significance appears to be a low conduction velocity in sprouting collaterals and in newly reinnervated muscle cells (Buchthal and Rosenfalck, 1958).

In later stages of recovery (5 - 12 months after onset of degeneration), spontaneous denervation potentials progressively disappear, and polyphasic potentials become larger sized and shorter than initially, but still longer than normal potentials (Fig.VII.4.). Giant motor units are created (Goodgold and Eberstein, 1972; Thumfart, 1986).A

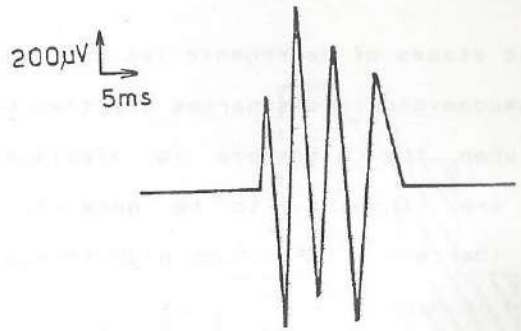


Fig.VII.4.: Polyphasic potential (schematic) : large size and long duration are characteristic, in relation with the fact that more muscle fibres of the same motor unit contribute to the action potential.

histogram of duration of motor unit potentials is helpful for the diagnosis (Fig. VII.5.).

If no regeneration occurs, fibrillation potentials and positive sharp waves may remain present for several years, although fibrosis develops.

In chronic stages of neurogenic lesions, spontaneous high frequency pseudomyotonic discharges are frequently observed, especially when the electrode is displaced inside the muscle.They are thought to be generated by ephaptic transmission (Mercelis, 1986).Such high frequency discharges are also noted in myopathies.

7.1.2.Clinical correlations

7.1.2.1.Position of the paralyzed vocal fold

Much has been written about the position of the paralyzed vocal fold.Dedo (1970) made a detailed description of the effects of experimental nerve sections in dogs :

1.Section of the inferior laryngeal nerve causes temporary spasm with subsequent paralysis of the vocal fold in the paramedian position, often with the arytenoid tilted forward.As the fold becomes completely immobile, it lengthens and adducts slightly with each subsequent

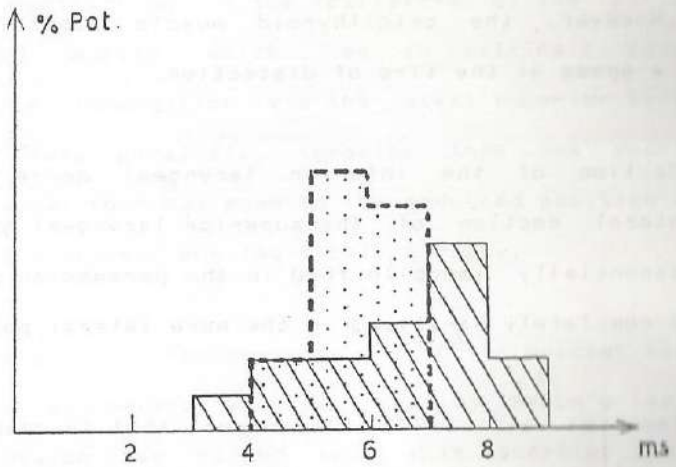


Fig.VII.5.: Comparative histograms of duration of motor unit action potentials in both thyroarytenoid muscles, one of them having been slightly denervated some months earlier.The latter shows globally an increased duration of motor unit action potentials.

phonation.

2.Section of the superior laryngeal nerve with an intact ipsilateral inferior laryngeal nerve always leads to little or no visible change in the contracture of the vocal folds.However, the cricithyroid muscle stops contracting after a spasm at the time of dissection.

3.Section of the inferior laryngeal nerve followed by ipsilateral section of the superior laryngeal nerve causes the essentially immobile fold in the paramedian position to become completely paralyzed in the more lateral position.

Gilmer et al. (1970) also report that in case of a high unilateral vagus nerve resection , with lesion of both the superior and the inferior laryngeal nerves, the vocal fold is immobilized in the intermediate position. In case of a lesion limited to one of the inferior laryngeal nerves, the paralyzed fold is seen in the paramedian position.

Sasaki et al. (1980) demonstrated that the physiologic inactivation of the cricothyroid muscle by tracheostomy is a key determinant of the lateralized vocal fold observed in acute low vagal and inferior laryngeal nerve paralysis.Tracheostomy abolishes the critical pressure changes necessary to trigger expiratory cricothyroid contraction.

Sasaki (1980) also reports that in early paralysis (less than one year) of one inferior laryngeal nerve, the involved vocal fold assumes a paramedian position caused by the unopposed contraction of the ipsilateral of the ipsilateral cricothyroid muscle which, as an extrinsic adductor, receives its innervation via the intact superior laryngeal nerve. In late paralysis, (greater than one year), the involved vocal fold may move to the abducted position as the ipsilateral intrinsic muscles slowly atrophy.

If paralysis is progressive, abductor muscles may lose their function before adductor muscles (Semon's law), but much discussion has raised about this assertion (Aubry et al., 1974). Murtagh and Campbell (1952) observed that adductor fibers continued to propagate action potentials despite a chemical or thermal blockade sufficient to paralyze abductor function.In addition, the chronaxy of adductor fibers, one of the most rapid thus far identified (0.1 ms) is tenfold shorter than that found in fibers subserving abduction.

7.1.2.2.Prognostic information

From the point of view of prognostic information, absent motor unit potentials, fibrillations or positive sharp waves indicate lack of recovery.If, however, polyphasic potentials

are observed, return to a satisfactory, or even normal function may be expected (Parnes and Satya-Murti, 1985). Limitations to this favourable prognosis are due to misdirected reinnervation (cf. Chapter IX), or to the fact that ankylosis of the cricoarytenoid joint may have occurred so that, despite reinnervation, the fixation impedes movement of the vocal fold (Parnes and Satya-Murti, 1985). In our own experience, nascent motor unit potentials appear several weeks before an eventual remobilization of the vocal fold.

7.2. Myogenic lesions

True myotonia, as well as other myopathies, are rarely observed in the field of laryngeal electromyography.Nevertheless, myotonic discharges may be observed even in vocal muscles in cases of Steinert's disease (Dejonckere, 1975). Myotonia is a disturbance of the muscle membrane resulting in an impaired relaxation . Myotonic discharges give a characteristic "dive-bomber"-sound in the loudspeaker.

In myopathies, a general characteristic is the loss of muscle fibers in the individual motor unit : This causes a decrease of the voltage . Further, the contribution of distant fibers is undetectable, so that the duration will be diminished.The patchy destruction of muscle fibers makes the

slight asynchrony more apparent and results in a "splintering" of the action potential.The total duration of the polyphasic unit does not exceed that of a normal motor unit.The spike compounds are all sharp (Fig.VII.6).A full interference pattern is observed at small or moderate effort (Goodgold and Eberstein, 1972; Mercelis, 1986).

The clinical picture suggests a paresis of both vocal folds.

7.3. Functional voice diseases

7.3.1.Functional dysphonia

Pioneering work in this field has been performed by Brewer et al. (1960) and Faaborg-Andersen et al. (1960) : They demonstrated that , while healthy phonation basically depends on flexible balances of the inter-related sets of intrinsic laryngeal muscles, unhealthy phonation or vocal abuse is regularly identified with imbalance of these same intrinsic muscles.Furthermore, electromyography provides a confirmation of the efficiency of voice therapy : existing intrinsic laryngeal muscle imbalance is relieved with a concurrent disappearance of symptoms and a return of normal laryngeal appearance.

Hyperfunctional dysphonia gives usually a stronger than

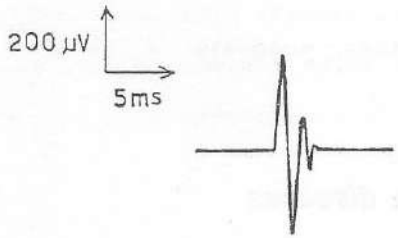


Fig.VII.6.: Myogenic potential (schematic) : short and small polyphasic motor unit potential, in relation with a drop-out of individual muscle fibers.

normal resting activity in the intrinsic even as extrinsic laryngeal musculature (Dejonckere, 1975).

Haglund et al. (1973) found slight neurogenic lesions in several cases first diagnosed as functional dysphonia : They concluded that some patients who were believed to be suffering from functional dysphonia were actually suffering from idiopathic vocal cord paresis, although their paresis was not severe enough to be detected at laryngoscopic examination.

Electromyographic analysis of all intrinsic laryngeal muscles in cases of so-called "internus paresis" or "transversus paresis", made by Tomita (1967), revealed no abnormality of the function of these muscles.

Stemple et al. (1980) recorded the "general laryngeal area" muscle tension, by means of a surface electrode applied onto the left thyroid lamina : they noticed that, in patients with vocal nodules, electromyographical activity was more important than in normal control subjects, both at rest and during phonation.They suggested biofeedback as a therapeutic method.

7.3.2.Spastic dysphonia

Spastic dysphonia, although not frequent, offers

characteristic electromyographic findings (Kittel et al., 1983).These authors investigated 12 patients : simultaneous electromyographical recordings were obtained from vocal muscle and from several articulatory muscles (musculus orbicularis oris; tongue; musculus masseter). In almost all of these muscles, the authors found phonatory hyperactivity with prolonged and enhanced pre-phonation and post-phonation phases.In some patients, the post-phonatory phase changed over directly into the next pre-phonatory phase without any interruption.The electromyography yielded such pertinent quantitative and qualitative objectivation that the authors suggested to document therapeutic results with its help.

Blitzer et al. (1985) related abnormal electromyographical findings in 50 % of the subjects : abnormally increased amplitude, asynchronous activity characteristic of a tremor disorder, synchronous bursts of activity also affecting other muscles.They also concluded that spastic dysphonia is not a "spastic" disease.

Abnormal spin-echo magnetic resonance imaging and abnormal auditory brain stem responses were found in several patients with spasmodic dysphonia by Schaefer et al. (1985) : infarcts within the basal ganglia and demyelinating lesions within the supralateral angles of the lateral ventricles were described.It may thus be suspected that this particular voice disease could be associated with slight

central nervous system disturbances, although a psychological component is usually present.

7.3.3.Psychogenic aphonia and dysphonia

Greiner et al. (1960) found a normal electromyographical activity in the thyro-arytenoid muscle.

Hirano (1981) observed that the incomplete glottic closure during phonation could be attributed to the neutralization of the activity of the adductor muscles as a result of the contraction of the posterior cricoarytenoid muscle : this muscle was active during phonation as well as during inspiration.

7.3.4.Stuttering

Thurmer et al. (1983) performed multi-channel electromyography in 42 patients with stuttering : Hooked wire electrodes were placed in vocal muscles and in some articulatory muscles.The authors objectivated special features of the tonic, clonic and combined disturbances of speech flow:

Tonic stuttering:

pre-phonatory hyperactivity in phonatory and articulatory

muscles.

Clonic stuttering:

pre-articulatory hyperactivity in the tongue and musculus orbicularis oris. Continuous activity in vocal muscles.

Tono-clonic stuttering:

this pattern combines characteristic electromyographical patterns of both tonic and clonic stuttering.

The authors also found significant differences between electromyographic patterns of spontaneous and rhythmic speech, for the same sentences out of standardized texts.In cases of rhythmic speech, the electromyographical tracings were clearly reduced in amplitude, and pre-phonatory hyperactivity was missing.Synkinetic activity was also reduced or absent.The authors evoked a close analogy with electromyographical patterns of phonatory and articulatory muscles in cases of spastic dysphonia.

Besides the possibility of electromyographical functional diagnosis in the phoniatric range, therapeutic possibilities offered by an electromyographical feedback therapy were also shown.

7.4. Central nervous disease

Clinicians have long been familiar with the fact that phonatory disturbances of laryngeal muscle function (in addition to articulatory disorders) are associated with the dyskinetic diseases (Parkinson's disease or athetosis for example), as well as with destructive lesions of the corticobulbar projections from either cerebral hemisphere or of the cerebellum, such as may occur in cerebrovascular or degenerative diseases (Wyke and Kirchner, 1976).These lesions cause incoordination and hyper- or hypotonicity of the laryngeal musculature, clinically as well as electromyographically, but no neurogenic manifestations.Krause (1884) first reported aphonia in dogs after bilateral extirpation of the gyrus precrucialis. Foerster (1936) postulated the necessity of a bilateral lesion as a pre-requisite to supranuclear laryngeal disturbances.

7.5. Disorders of rhythmic respiratory activity

7.5.1.Data from normal and pathological humans

In all of the intrinsic laryngeal muscles, there is, during quiet respiration, and even more so during forced respiration, a rhythmical increase and decrease of the electrical activity, thus linked to the respiratory cycle.Lucier et al. (1978) recorded respiratory-related

activity in single units of the inferior laryngeal nerve, and even in laryngeal motor neurons in the nucleus ambiguus (anesthetized cat).

The neurological mechanism of generation and control of this coordinated activity has been largely investigated in animals,but is still imperfectly understood.Several differences between animal species appear in the regulatory mechanism of ventilation (Dejours, 1963; Wyke and Kirchner, 1976), and as a consequence the mechanism in humans cannot be clearly deduced from animal experiments.

Here we report a set of electromyographic observations obtained in human subjects suffering from selected well identified peripheral neurologic disorders.Respiratory activity of the cricothyroid muscle is either preserved or altered to a variable degree.Critical analysis and comparison with results of experimental lesions in animals suggest complex neural mechanisms determining the phasic activity of the cricothyroid muscle, but similar contributing pathways are found in animals and humans.

Nineteen patients were selected,all suffering from well defined laryngeal neurological troubles.They first underwent a bilateral electromyographic recording of the cricothyroid muscle (innervated by the ramus externus of the nervus laryngeus superior).Electromyography of this muscle does not

require mucosal anesthesia.Subsequently,the other intrinsic laryngeal muscles (innervated by the nervus laryngeus inferior) were examined after topical xylocaine application.This procedure allowed objective estimation of the extent of lesion of each of the four laryngeal nerves.

Fifteen normal subjects have been included in the study,serving as a control group.All electromyographic recordings were performed using a concentric needle electrode.

All normal subjects showed electric activity in both cricothyroid muscles,consisting in a change in frequency of action potentials related to the respiratory cycle during normal breathing.Phasic activity was increased by forced ventilation.

Pathological cases are summarized in Table VII.I.

The following abbreviations are used : N.L.S.:nervus laryngeus superior ;N.L.I.:nervus laryngeus inferior ;N.L.S.muscles:muscles innervated by the nervus laryngeus superior ;N.L.I.muscles:muscles innervated by the nervus laryngeus inferior.

Analysis of the table reveals contributions of several pathways to the regulatory mechanism of rhythmic activity of the cricothyroid muscle :

Case	Aetiology	Laryngeal muscles affected	Respiratory activity in cricothyroid muscle	Conclusion
1	Amyotrophic lateral sclerosis	N.L.I.muscles: total denervation.Other muscles normal	Present bilateral	homolateral N.L.I. not essential
2	Oesophagus surgery	Left N.L.I.muscles:total denervation, then partial reinnervation.Other muscles normal	Left:absent	homolateral N.L.I. contributes
3	Carotid surgery	Left N.L.I.muscles:total denervation, then partial reinnervation	Present,bilateral,after temporary abolition	homolateral N.L.I. contributes
4	Thyroid surgery	Left N.L.I.muscles:total denervation:* other muscles normal	Absent bilaterally	N.L.I. contributes.Crossed connections
5	Post-radiotherapic neuritis	Left N.L.I. and N.L.S.muscles:partial denervation;other muscles normal	Left:absent	N.L.I. and/or N.L.S. contributes
6	Thyroid surgery	Left N.L.I.muscles:total denervation. Right N.L.I.muscles:reinnervation. Other muscles normal	Left:absent	Homolateral N.L.I. contributes
7	Thyroid surgery	Right N.L.S.muscle:total denervation, then reinnervation.Other muscles normal	Left and right:present after temporary bilateral abolition	N.L.S. contributes.Crossed connection
8	Thyroid surgery	Right N.L.I.muscles:partial reinnervation.Other muscles normal	Right:absent	Homolateral N.L.I. contributes
9	Thoracic surgery	Laryngeal muscles intact.Section of right nervus vagus intrathoracically	Right:absent,but resumes slightly during forced respiration	Homolateral nervus vagus contributes. Peripheral modulation

Table VII.1.(First part): Cases with various peripheral neurogenic lesions, electromyographically investigated from the point of view of the respiratory activity in the cricothyroid muscle.Abbreviations in text.

10	Toxic neuritis	Partial denervation of all muscles, then partial reinnervation	Absent during breathing through tracheotomy.Present during breathing through natural airways	Modulation by peripheral impulses
11	Thyroid surgery	Left N.L.S. and N.L.I.muscles:total denervation.Right muscles normal	Left:absent	Homolateral N.L.S. and/or N.L.I. contributes
12	Carotid surgery	Left N.L.I.muscles:total denervation Left N.L.S.muscle:partial denervation Right muscles:normal	Present,bilateral	Homolateral N.L.I. not essential
13	Viral neuritis	Left N.L.I.muscles:total denervation Other muscles normal	Absent bilaterally	Homo- and heterolateral N.L.I. intervene.Crossed connections
14	Neurofibromatosis Surgical section of right vagus nerve	Right N.L.I.muscles:total denervation Left N.L.I.muscles:subtotal denervation Right and left N.L.S.muscles normal	Weakly present,bilateral Increases in forced respiration	Homolateral N.L.I. not essential Peripheral modulation
15	Bulbar tumor	Right N.L.S. and N.L.I.muscles:subtotal denervation.left muscles normal.	Right:weakly present Left:absent	Heterolateral N.L.S. and/or N.L.I contribute
16	Traumatism	Right and left N.L.I.muscles:total denervation.Other muscles normal	Absent bilaterally	N.L.I. contributes
17	Amyotrophic lateral sclerosis	Partial denervation of all muscles	Present,bilateral,when breathing through tracheotomy.Increases on closure of canula	Peripheral modulation
18	Oesophagus surgery	Right and left N.L.I.:total denervation. Other muscles normal	Present,bilateral,during forced respiration	N.L.I. contributes but not essential
19	Acute methanol intoxication	Reinnervation after partial peripheral neurogenic lesion	Absent.Resumes during forced respiration	N.L.S. and/or N.L.I. contribute Peripheral modulation

Table VII.1.(Second part)

1. The superior laryngeal nerve is involved (case 7).This nerve carries afferent impulses from the supraglottic mucosa,proprioceptive receptors in the cricothyroid muscles and possibly (anastomoses) from pulmonary receptors (Jewett, 1964; Wyke and Kirchner, 1976).

2. The inferior laryngeal nerve is involved (cases 2,4 6,8,13,16,18); it is,however,not indispensable (cases 1,12).This nerve carries afferences from the subglottic mucosa,from laryngeal articular and muscular receptors and possibly from pulmonary receptors (Jewett, 1964; Wyke and Kirchner, 1976).

3. The vagus nerve (intrathoracic portion) is involved (case 9).It carries afferences from pulmonary receptors (Agostoni et al., 1957; Jewett, 1964).

4. There is evidence of crossed connections (cases 4,7,13,15).

5. Moreover,several cases reveal a modulation by peripheral impulses,in relation with the resistance of the respiratory airways (cases 9,10,14,17,19).

7.5.2.Comparison with evidence from animal experiments.

Several lines of evidence gained in animal experiments enlighten the observations of this study :

1.Primary central generation of rhythmic activity.

Rhythmic increase and decrease of frequency of action potentials in laryngeal motoneurones persist to a variable extent,even in decerebrated animals,after one of the following manipulations:

-total muscular paralysis by neuromuscular blocking agents (Stransky et al., 1973)

-diversion of the respiratory air-stream out of the larynx (Wyke, 1973)

-topical anesthesia of the laryngeal mucosa (Wyke, 1973)

-interruption of pulmonary afferent pathways by bilateral division of the vagus nerve distal to the origin of the inferior (recurrent) laryngeal nerves (Stransky et al., 1973; Wyke, 1973).

-complete deafferentation of the larynx (Stransky et al., 1973).

This suggests that the basic (but not exclusive) rhythmic respiratory of the intrinsic laryngeal muscles is central,mediated through relays from the respiratory nuclei in the lower brainstem to the nucleus ambiguus.

2.Existence of crossed connections

Direct electrical stimulation of the inspiratory region of the medullary reticular system on one side leads to bilateral activation of the vocal fold abductors,whilst stimulation of the expiratory region provokes bilateral adductor muscle activity (Kurozumi at al., 1971).

3.Laryngeal afferent systems (superior and inferior laryngeal nerves)

Respiratory-linked discharges can be recorded in different fibres in the superior laryngeal nerve. Furthermore,electrical stimulation of this nerve (or its internal branch) modifies laryngeal motor unit activity (Eyzaguirre et al., 1966).Direct mechanical stimulation of various parts of the supraglottic and subglottic laryngeal mucosa (including with gentle puffs of air) evokes afferent discharges in the related laryngeal nerves (Eyzaguirre et al., 1966; Kirchner, 1968, Wyke, 1973).

4.Pulmonary afferent systems (vagus nerve and partially laryngeal nerves)

Passive inflation of the lungs stimulates stretch-sensitive pulmonary mechanoreceptors innervated by myelinated vagal afferent fibres,and reflexly alters the activity of recurrent and external laryngeal motoneurones

(Barillot and Bianchi, 1971; Fukuda et al., 1973; Widdicombe, 1973).Moreover,a combination of degeneration and electrophysiological studies has shown that the laryngeal nerves contain afferent fibres originating from pulmonary mechanoreceptors (Agostoni et al.,1957; Jewett, 1964).Sasaki et al.(1980) have also demonstrated a peripheral control in dogs : the rhythmic activity of the cricothyroid muscle is directly controlled by a rise in subglottic pressure ; therefore,when tracheotomy abolishes the critical pressure changes necessary to trigger the activation of the cricothyroid muscle,it must inactivate this muscle as well.

7.5.3.Physiopathological conclusion

Phasic respiratory activity in cricothyroid muscle,primarily centrally generated, is under control of complex and intricated mechanisms,in which the superior laryngeal nerve,the inferior laryngeal nerve, the vagus nerve and cross connections all intervene.Furthermore,a peripheral modulation related somehow to the resistance of the airways is present.Despite differencies between animal species,it seems that qualitatively the same contributing elements are present in animals and humans (Dejonckere and Lebacq, 1984).

Chapter VIII

Etiopathogeny of laryngeal paralysis

8.1. Introduction

Laryngeal nerve paralysis is an important pathology in the field of otolaryngology and phoniatrics.Each patient with an immobile or less mobile vocal fold needs to undergo , besides a complete electrophysiological test, all possible medical investigations in order to define a precise etiological factor.This is of fundamental importance for applying, if possible, an etiological treatment, but also for treating ,at an early stage of development, a possibly related tumoral lesion.

At the present time, CT-Scan has become the most performant diagnostic procedure for compressive lesions of laryngeal nerves.Horizontal sections must explore head, neck and mediastinum.Further investigations are in the field of endoscopy, neurology, and biochemical analysis.

From another point of view, vocal function tests are necessary for assessment of the symptomatic treatment ,

which may be medical, functional or surgical : they include stroboscopy, acoustical analysis, airflow rate and spirometry, laryngography and phonetogram. More information about this subject can be found in Hirano (1976,1981) and Dejonckere (1985).

Unilateral vocal fold paralysis usually causes a dominant voice symptomatology, and it has been related that 7 % of all dysphonias are related to laryngeal paralysis (Ulrich, 1977).Nevertheless, innervation of the intrinsic laryngeal muscles is not a prerequisite for voice production (Buchthal, 1959).

Bilateral inferior laryngeal nerve paralysis more often results in breathing and swallowing difficulties (Dejonckere, 1980).

8.2. Review of recent literature

8.2.1.Main etiological factors

A large number of publications are devoted to etiological factors of vocal fold paralysis, from a clinical point of view .Fig.VIII.1. and VIII.2. give a comparison of statistics reported by five authors who consider separately unilateral and bilateral cases : Parnell and Brandenburg

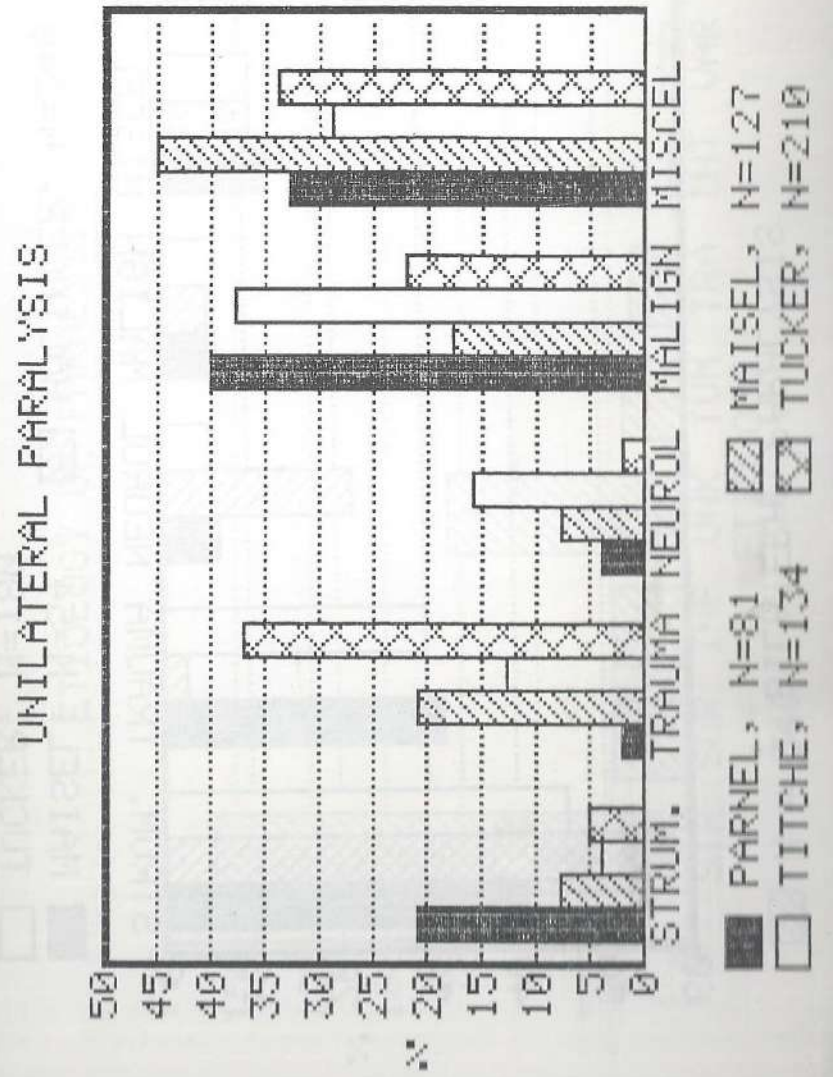


Fig.VIII.1.: Etiologic factors of unilateral vocal fold paralysis.
Strum. : Strumectomy
Malign : Malignant disease
Trauma : Traumatic injury (head, neck, thorax)
Neurol : Neurologic pathology
Miscel : Miscellaneous.

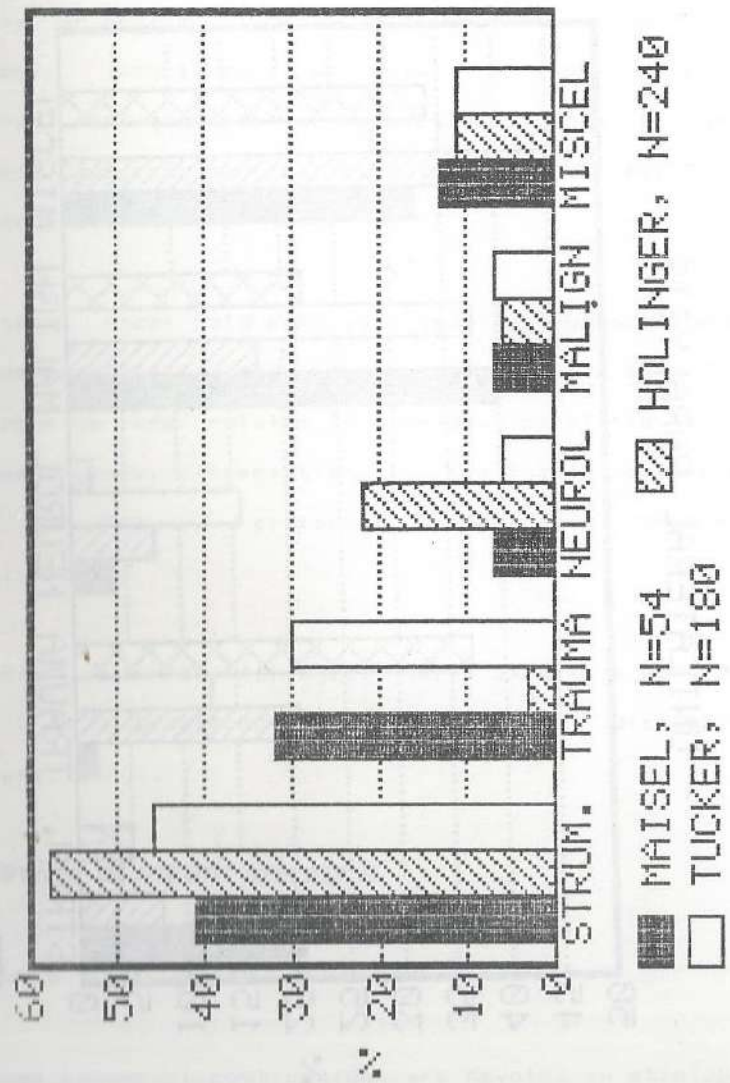


Fig.VIII.2.: Etiologic factors of bilateral vocal fold paralysis.
Strum. : Strumectomy
Trauma : Traumatic injury (head, neck, thorax)
Neurol : Neurologic pathology
Malign : Malignant disease
Miscel : Miscellaneous.

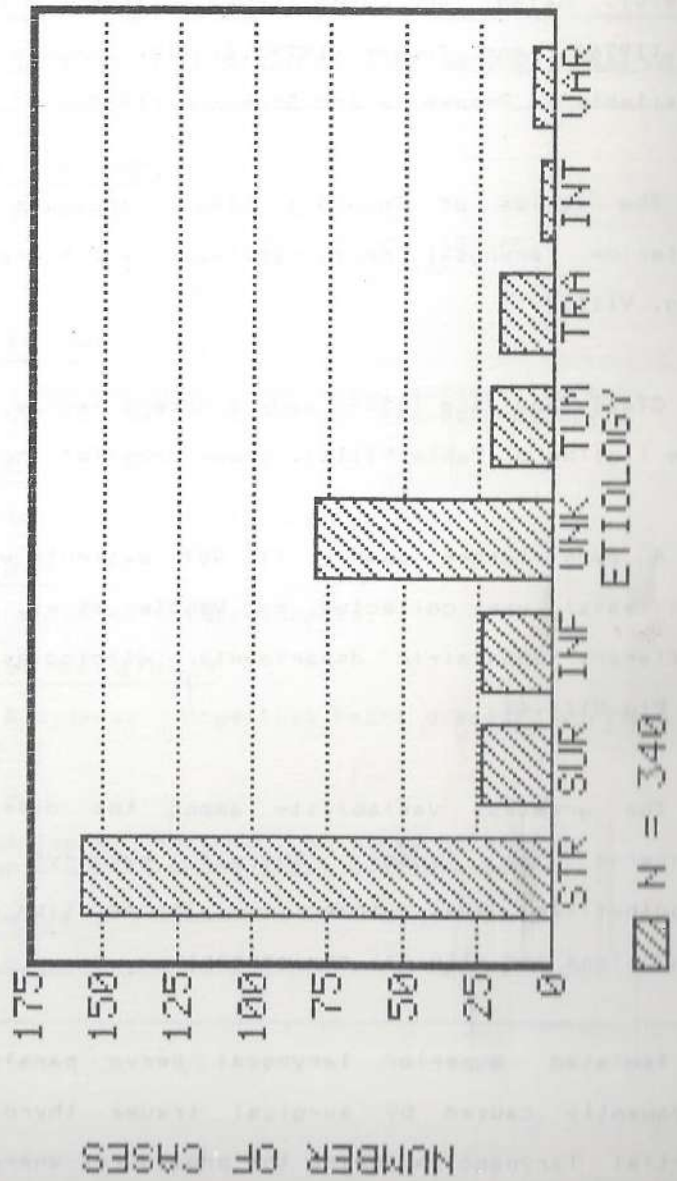


Fig.VIII.3.: Etiologic factors of inferior laryngeal nerve paralysis after Thumfart (1983) : Strumectomy, surgery, infection, unknown, tumors, trauma, intubation, varia.

(1970), Maisel and Ogura (1974), Titche (1976), Holinger et al.(1976), and Tucker (1979).Similar comparative tables are available in Paparella and Shumrick (1980).

The series of Thumfart (1983) includes 340 cases with inferior laryngeal nerve paralysis, which are summarized in Fig. VIII.3.

Claes and Jaco (1986) made a recent review of 631 cases : The listing of Table VIII.1. shows compiled incidences.

A even larger sample (1 087 patients with vocal fold paralysis) was collected by Wendler et al. (1984) from 16 different phoniatric departments : etiologies are presented in Fig.VIII.4.

The greatest variability among the different authors concerns the factor "Thyroid surgery" : It may be hypothetised that this is in relation with working conditions and clinical environment.

Isolated superior laryngeal nerve paralysis is most frequently caused by surgical trauma, thyroid surgery and partial laryngectomy being the procedures where the nerve is most likely to be exposed to trauma (Claes and Jaco, 1986).Its diagnosis may be clinically difficult.

ETIOLOGIES OF INFERIOR LARYNGEAL NERVE PARALYSIS

Malignant disease

24.5 % of which
- 50 % lung cancer
- 20 % oesophageal cancer
- 10 % thyroid cancer
- 20 % other

Surgical trauma

20.5 % : Pneumectomy, oesophageal surgery, surgery of heart, thyroid, neck dissection, mediastinoscopy.

Idiopathic

13 % : mononucleosis, influenza, etc...

Inflammatory

13 % of which 90 % tuberculosis.

Trauma (non surgical)

11 % : Accidents, congestive heart disease, aortic aneurysm...

Neurologic

7 % : Epilepsy, Parkinson, multiple sclerosis, alcoholic or diabetic neuropathy, Guillain-Barré.

Miscellaneous

11 % : Syphilis, rheumatoid arthritis, collagen disease...

Table VIII.1.: A compilation of etiologies of inferior laryngeal nerve paralysis , after Claes and Jaco (1986).

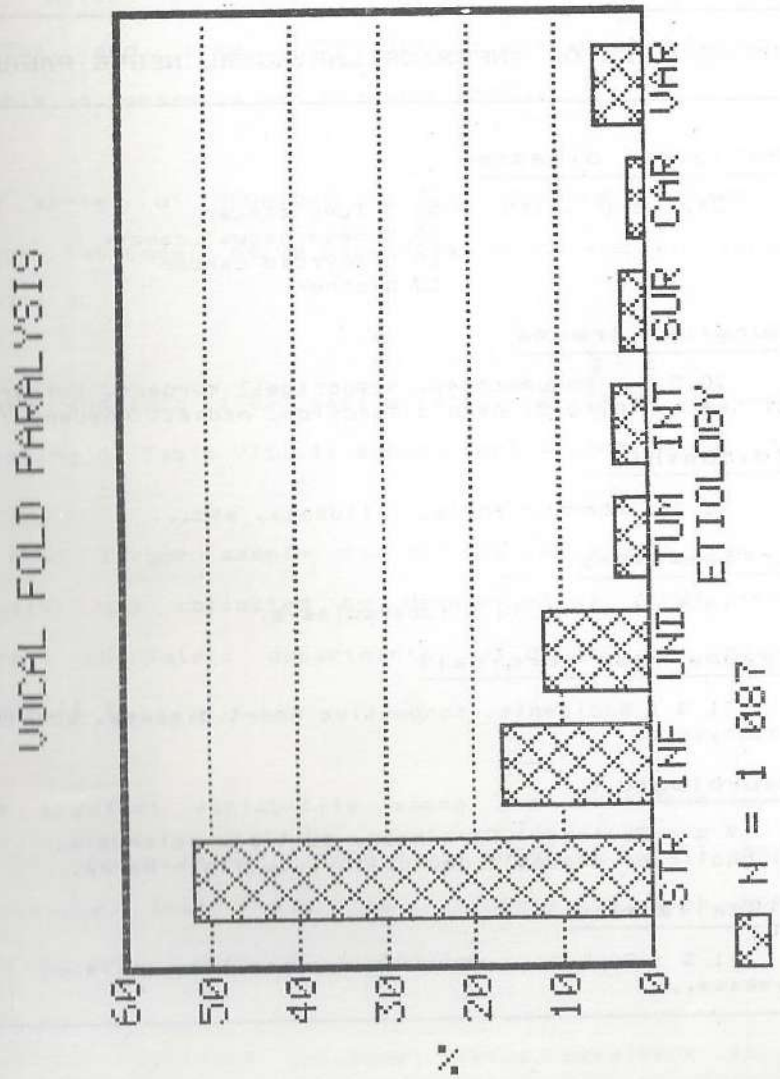


Fig.VIII.4.: Etiology of vocal fold paralysis, after Wendler at al.(1984).
Strumectomy, infection, undefined, tumors, intubation, neck and thorax surgery, hearth disease, miscellaneous.

It is important to notice that all these reviews deal with clinical (and not electromyographic) diagnosis.

Thyroid surgery may also cause paresis of extrinsic laryngeal muscles, especially the sternothyroid (Pahn et al., 1984).

In summary, we conclude that thyroid surgery and malignant disease are by far the most common etiologies of laryngeal paralysis.Besides, some specific, although rare, causes, are worthy of attention :

8.2.2.Peculiar etiologic factors

8.2.2.1.Intubation

All authors agree that paralysis is due to ischemia of the inferior laryngeal nerve.This can result from a stretching of the nerve when the neck is hyperextended for intubation, or from a trauma produced by the laryngoscope. But the most probable mechanism seems a compression by too large a tube, or an excessive pressure in the cuff. Cavo (1985) demonstrates that nitrous oxide diffuses into endotracheal tube cuffs causing a substantial increase in the intracuff pressure. The nerve can be compressed between

the endotracheal tube cuff and the cricoid, or between the thyroid ala and a dislocated arytenoid cartilage (Cros et al., 1981).A forced neck torsion during the surgical procedure increases the risk (Holley and Gildea, 1971).

An anatomical study has been realized by Cros at al. (1981) and shows that the ischaemic area induced by overpressure in the endotracheal cuff overlyes the nerve.

Clinically, the paralysis is generally unilateral, the vocal fold is in a paramedian position, and the dominating symptom is hoarseness.

The paralysis may appear even after a short duration intubation, but prolonged intubations increase the risk.Cros et al. (1981) reviewed 30 cases (of which 13 had electromyographic confirmation) : The average duration of intubation was 19 days for patients with unilateral lesion, and 60 days for patients with bilateral lesions.

Most of the cases of laryngeal paralysis due to short duration intubation evolve towards a total recovery.Electromyography is a valuable indicator for a good prognosis, when denervation is not complete (Cros et al., 1981).

8.2.2.2.Cardiopathy

Simultaneous occurrence of dysphonia and cardiac hypertrophy in patients with a valvular mitral stenosis was first described by Ortner in 1897 as "cardio-vocal syndrom".At the end of the nineteenth century, its frequency was estimated to be about 4 % of cardiovascular pathologies (Kittel et al., 1986).At present, with progress of cardiology, it has become much less frequent.Three hypothetical mechanisms have been proposed :

- pressure exerted on the left inferior laryngeal nerve by the dilated auricle

- pressure exerted on the left inferior laryngeal nerve by the Arteria pulmonalis.

- downward traction of the hypertrophic hearth.

(Kittel et al, 1986)

In contrast , cardiovascular surgery has gained a considerable importance in the last decades, and has become a more significant causative factor of vocal fold paralysis.

The following percentages of vocal fold paralysis after cardiac surgery are available :

Stenborg (1973) : 3 cases among 57 patients with vocal fold paralysis.
Titche (1976) : 4 cases among 134 patients
Holinger et al. (1976) : 3 cases among 389 patients
Hirose (1978) : 42 cases among 600 patients
(Kittel et al..1986)

From another point of view, Kittel et al. (1986) observed 6 left-sided pareses in 434 patients operated because of cardiovascular malformations.

8.2.2.3.Lung cancer

Lung cancer is, in Europe, the most frequently observed malignant tumor in men.Survival rate at five years is not higher than 10 % (Ries et al., 1983), because only a few of all patients are operable (20 %), and long time survivals are, at the present time, practically inexistant with other treatments.

Our personal statistics are made on 994 cases of lung cancer (1970-1983) (Carrillo et al., 1986). 30 cases of laryngeal paralysis are observed (3.3 %).In 16 cases, dysphonia was the first or dominating symptom at the time of the first consultation.

In 27 cases, the left vocal fold was involved, in 2 cases the right vocal fold, and in 1 case both vocal folds.

The position of the paralyzed fold is

- paramedian : 5 cases
- intermediate : 5 cases
- in abduction : 3 cases
- in adduction : 3 cases.

In the remaining cases, no sufficient precision could be obtained.

After complete assessment, all patients were directly considered as inoperable: 15 because they had metastases, 15 in relation with mediastinal extension of the tumor. 24 patients had a regular follow-up : mean survival was 9 months.

It must be considered that a vocal fold paralysis due to a lung cancer is a sign bad prognosis.

8.2.2.4.Metabolic disease

This group of etiological factors has been emphasized by

Georgopoulos et al. (1981): Among 39 patients with idiopathic laryngeal paralysis, they found :

- 14 cases of diabetes mellitus
- 4 subjects with a pathological oral glucose tolerance test
- 3 subjects with uremia
- 1 subject with hyperlipidemia
- 1 subject with hyperuricemia.

Further, among 296 subjects suffering from diabetes mellitus, 15 showed disturbances of laryngeal mobility.

A neuritis-mechanism is to be suspected, with probable demyelination and axonal degeneration.

8.2.2.5.Idiopathic vocal fold paralysis

Frequency of idiopathic vocal fold paralysis is very high in some series, and very low in other ones. Capps (1948) found 172 cases among 455 subjects with unilateral vocal fold paralysis (Paparella and Shumrick, 1980).In our own statistics (366 cases of peripheral neurogenic lesion of the larynx) , a precise etiology lacks in 17 cases.

Ward and Berci (1982) observed that, in all of their 39

idiopathic cases (among 238), both the superior and inferior laryngeal nerves were paralyzed, indicating that the lesion could involve the nuclear lower motor neurons.The authors thought that, in all probability, so-called idiopathic vocal fold paralysis is similar in origin to other idiopathic cranial nerve palsies such as facial paralysis, loss of olfaction, and sudden deafness.

8.2.2.6.Vocal fold paralysis in infancy

The causes of laryngeal paralysis in the pediatric population are different from those in adults. In children, the major causes of vocal fold paralysis, both unilateral and bilateral, are birth trauma, central nervous system disease, meningomyelocele with Arnold-Chiari malformation and hydrocephalus, idiopathic, surgical trauma, and disease of the heart and great vessels.Statistics of Cohen et al. are reported in Fig.VIII.5.(100 cases).

The authors did not perform an electromyographic investigation, but each child had a direct laryngoscopy even as a bronchoscopy.Cricoarytenoid fixation was excluded by direct palpation of the joint.

Male children are slightly more often involved (59 / 100).

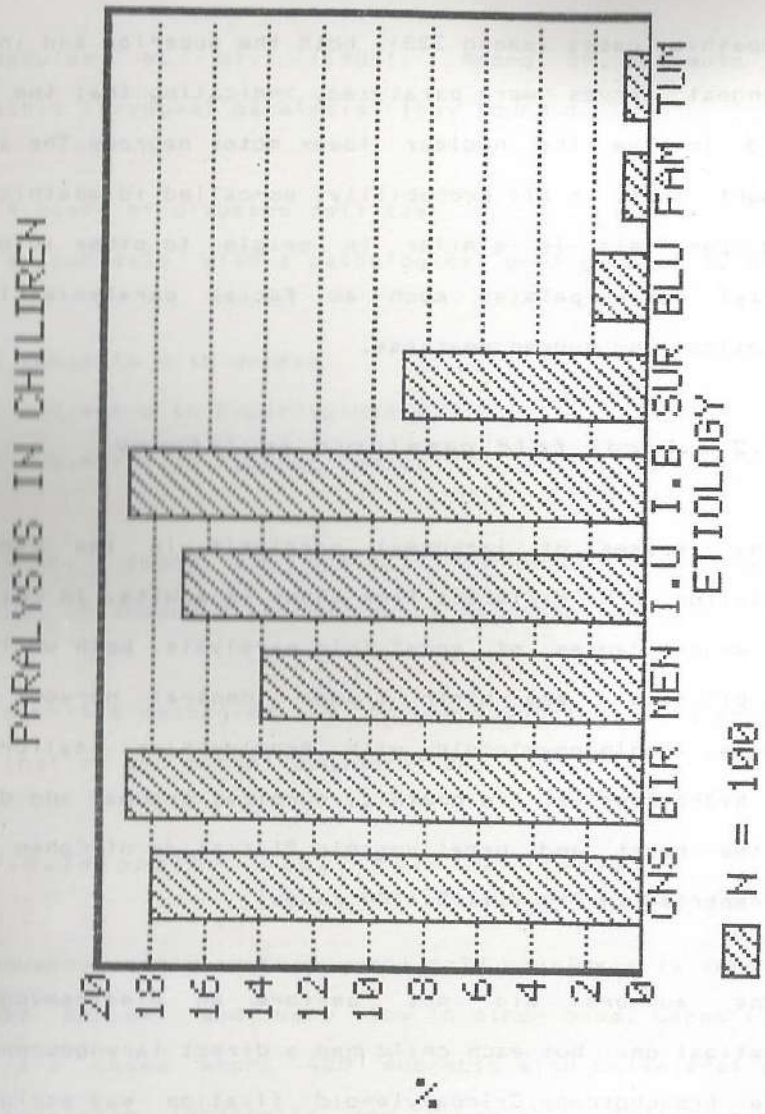


Fig.VIII.5.: Etiology of laryngeal paralysis in children, after Cohen et al., 1982.
Central nervous system disease, birth trauma, meningomyelocele with Arnold-Chiari malformation and frequently hydrocephalus, idiopathic unilateral, idiopathic bilateral, surgery, blunt trauma to the neck, familial, tumor.

The earliest evidence of laryngeal paralysis is in the group of children with birth trauma.Children with central nervous system disease (e. g. Möbius' syndrom) show evidence of laryngeal paralysis within the first ten days of life.However, in the group of patients with meningomyelocele with Arnold-Chiari malformation and hydrocephalus, the mean age of onset is later (47 days).This delay in onset of symptoms may be related to the progressive development of hydrocephalus which followed the repair of the meningomyelocele.Uncomplicated hydrocephalus does not produce laryngeal paralysis.

Complicated delivery with abnormal presentation or positioning of the infant and the need for cesarean section birth was associated with laryngeal paralysis in almost 20 % of the children.

Sixty-six percent of the patients had a bilateral abductor paralysis: Most of them are due to a meningomyelocele with Arnold-Chiari malformation or a central nervous system disease.Kinking and herniation of the brainstem is thought to cause impairment of its blood supply and the resultant ischaemia leads to bilateral abductor vocal fold paralysis.In those with unilateral paralysis, the left side is most frequently involved.

Inspiratory stridor is the most common presenting symptom.Severe obstruction to the airway, aspiration and dysphagia frequently occur.Dysphonia is an uncommon symptom whether the paralysis is unilateral or bilateral.

Intubation and tracheotomy are often necessary.

(Cohen et al., 1982)

Hereditary bilateral vocal fold paralysis has been described.Swallowing difficulties and other neurological symptoms are usually associated.Tracheotomy is frequently required during the neonatal period.

Localized delay in neurologic maturation is suspected as the etiology.

(Gacek, 1976; Grundfast and Milmoe, 1982)

8.3. Personal statistics

8.3.1.Etiology

Among 369 patients with a peripheral laryngeal electromyographic pathology, we found 366 cases with a

peripheral neurogenic syndrome, and 3 cases with a myogenic syndrome.In 17 of these cases (4.6 %), no specific etiology could be demonstrated, in spite of a careful clinical and paraclinical investigation. Table VIII.2. lists the etiological categories.

8.3.2.Sex

Fig.VIII.6. separates, for each group of etiologies, the respective number of male and female patients concerned.

Globally, no statistically significant difference appears between sexes (175 men and 195 women).Nevertheless, some etiologic factors are much more frequent in one sex.

On the one hand, strumectomy is much more frequent in women ($p < .000001$).On the other hand, neck surgery (except the thyroid gland) ($p < .01$) and thoracic tumors ($p = .00001$) appear significantly more frequently in men as a cause of laryngeal denervation.The overwhelming majority of thoracic tumors are bronchial carcinomas with hilum metastases.

Other causes seem approximately equally common in both sexes, and no significant difference is detected.

ETIOLOGY OF PERIPHERAL NEUROGENIC SYNDROM OF THE LARYNX (personal statistic)	Number of cases
Strumectomy	159
Cervical surgery	36
Thoracic tumors	41
Neurologic pathology	32
Trauma	33
Thoracic surgery	18
Cervical tumors	12
Neuritis	11
Cardiopathies	7
Undefined	17

Table VIII.2.: Personal statistic of etiological factors in 366 cases of peripheral neurogenic syndrom of the larynx, investigated by electromyography.

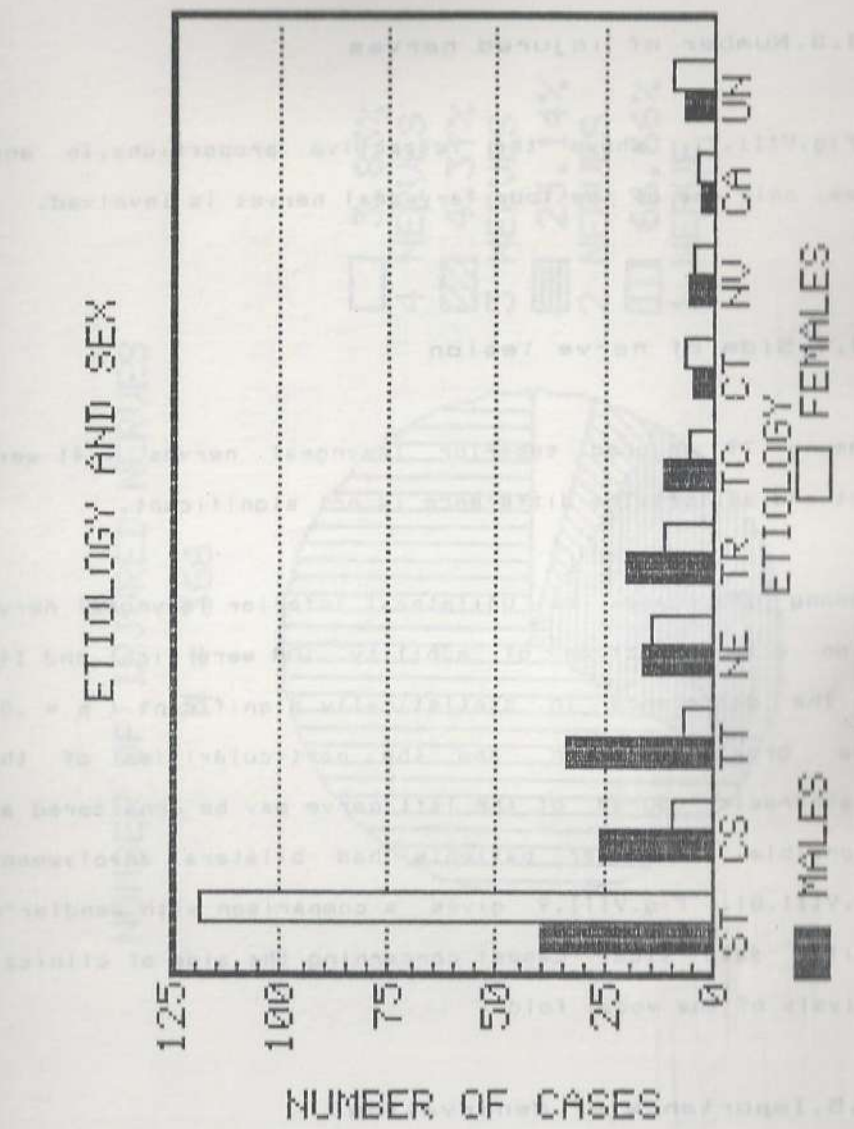


Fig.VIII.6.: Etiology of peripheral neurogenic syndrom of the larynx, with, for each etiologic group, the respective number of male and female patients.
Strumectomy, cervical surgery, thoracic tumors, neurologic pathology, trauma, thoracic surgery, cervical tumors, neuritis, cardiopathies, undefined.

8.3.3.Number of injured nerves

Fig.VIII.7. shows the respective proportions.In most cases, only one of the four laryngeal nerves is involved.

8.3.4.Side of nerve lesion

Among 79 injured superior laryngeal nerves , 41 were right and 38 left.The difference is not significant.

Among 253 cases of unilateral inferior laryngeal nerve lesion with reduction of mobility, 108 were right and 145 left.The difference in statistically significant (p = .02).The greater length and the particularities of the intrathoracic course of the left nerve may be considered as responsible. 71 other patients had bilateral involvement (Fig.VIII.8). Fig.VIII.9 gives a comparison with Wendler's compiled data (1087 cases) concerning the side of clinical paralysis of the vocal fold.

8.3.5.Importance of denervation

Among 79 injured superior laryngeal nerves, 52 had a partial degeneration, and 27 a total denervation.Partial denervation is significantly more frequent (p < .01).

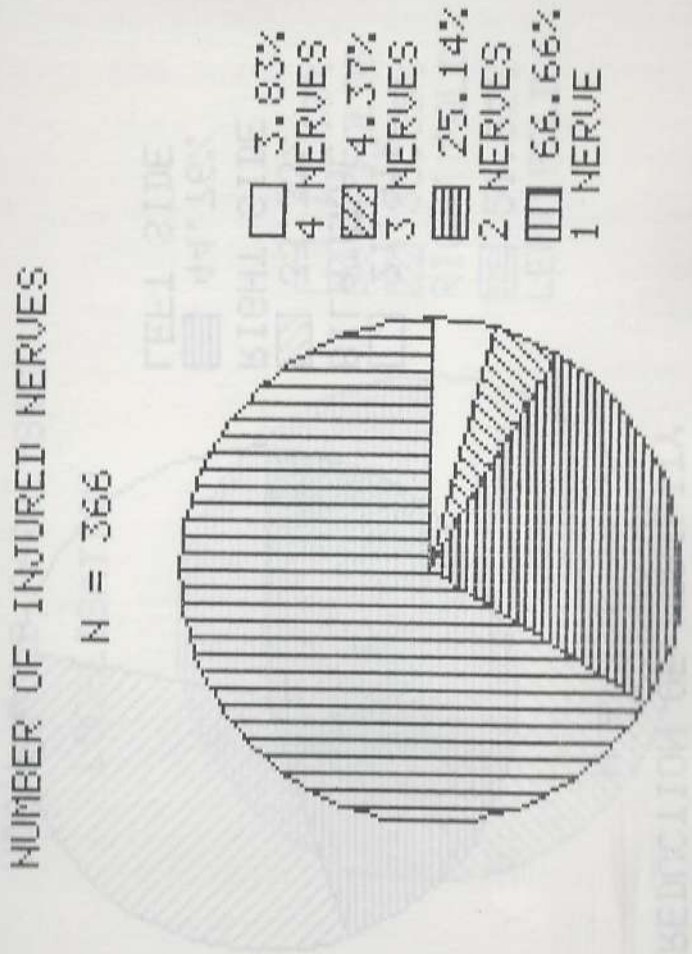


Fig.VIII.7.: Number of injured laryngeal nerves, in cases of peripheral neurogenic syndrome of the larynx.

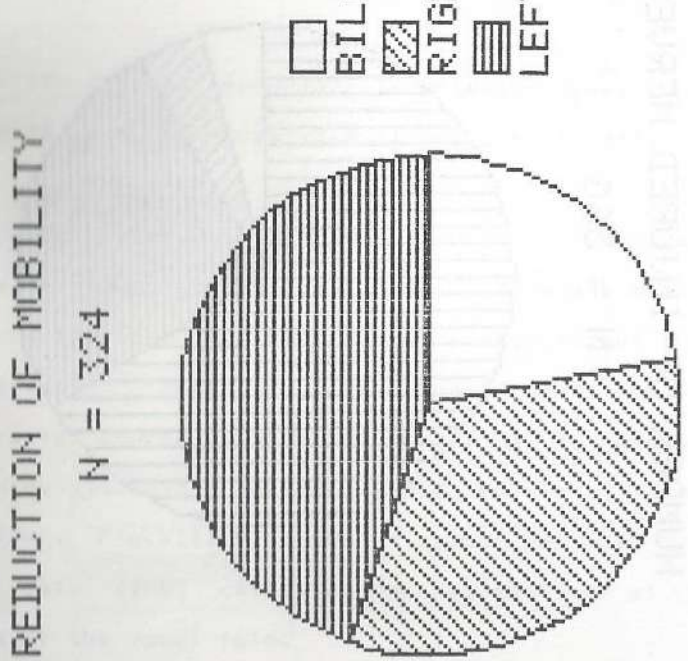


Fig.VIII.8.: Side of reduction of mobility, in cases of inferior laryngeal nerve lesion.

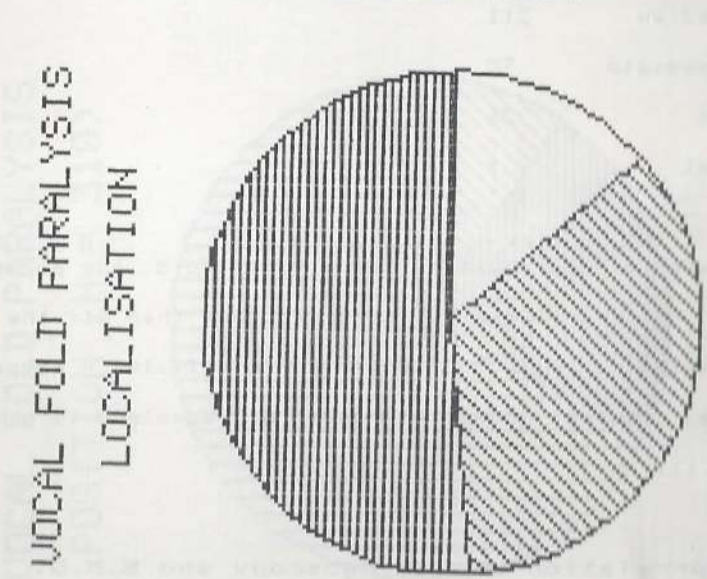


Fig.VIII.9.: Side of clinical paralysis of the vocal fold, after Wendler et al. (1984) (1087 compiled cases).

Among 324 injured inferior laryngeal nerves, 183 had a partial denervation, and 141 a total denervation.Partial denervation is also significantly more frequent (p = .02).

8.3.6.Position of the paralyzed vocal fold

Paramedian	211
Intermediate	70
Median	26
Lateral	3

In case of denervation of a vocal fold, the paramedian position is significantly more frequent than all the other eventualities (p < .000 000 1) (Fig VIII.10).A comparison with the clinical compiled data of Wendler is given in Fig.VIII.11.

8.3.7.Correlation of laryngoscopy and E.M.G.

The data, with respect either to the total or the partial character of the injury, are presented in Table IX.Clinically, the criterion is either immobility or reduced mobility of the vocal fold; electromyographically, the criterion is either presence or absence of voluntary motor unit potentials.Diagnosis significantly differs depending on

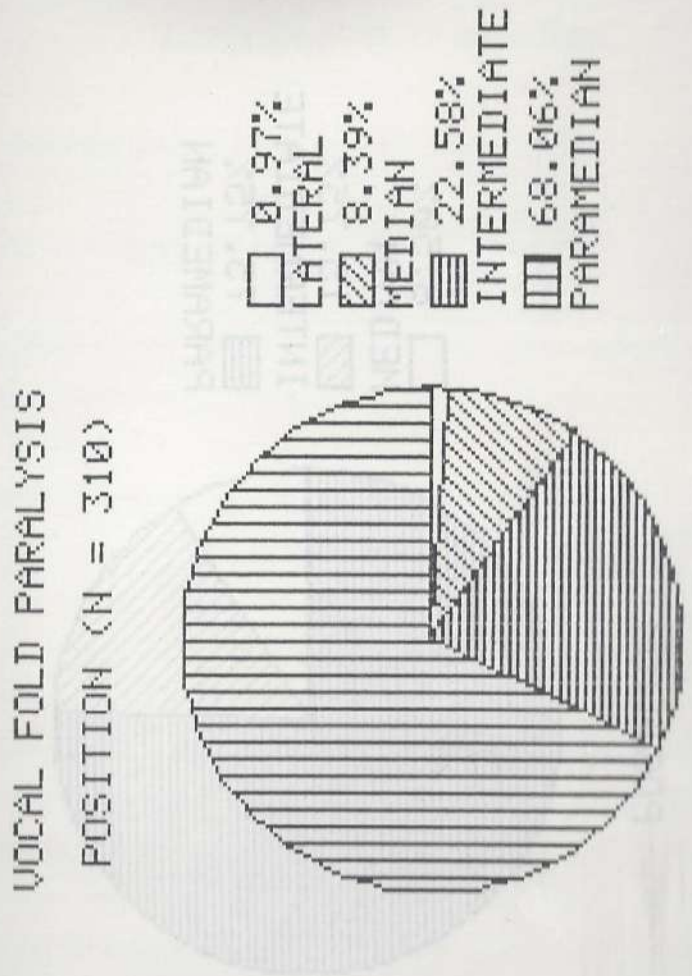


Fig.VIII.10.: Position of the paralyzed vocal fold.

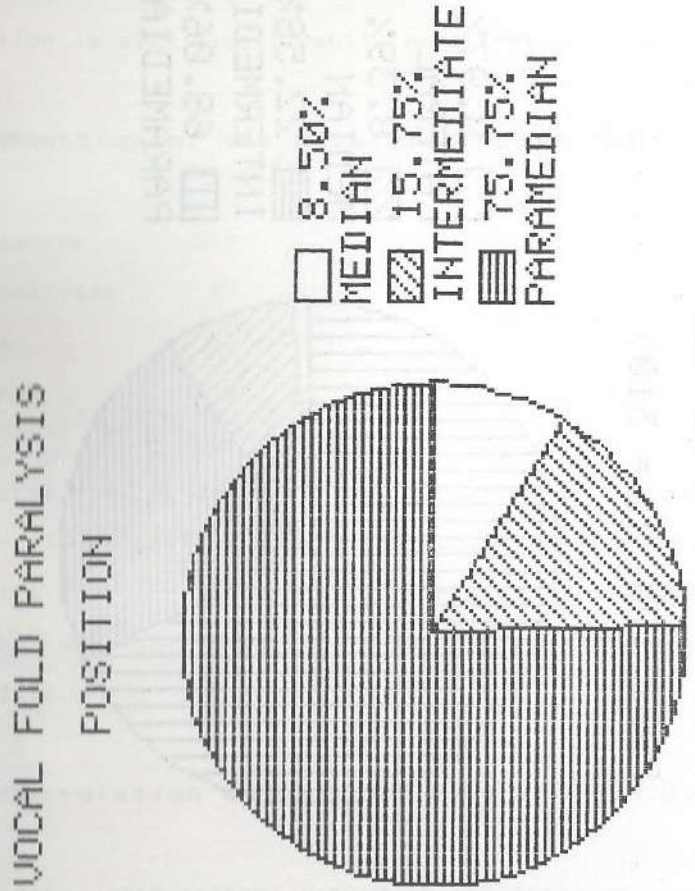


Fig.VIII.11.: Position of the paralyzed vocal fold, after Wendler et al. (1984) (1087 compiled cases).

the method ($p < .0001$) (Everitt, 1980).

Chapter IX

Laryngeal reinnervation

9.1. Neurophysiological basis

9.1.1.Types of lesions

Reinnervation may occur either after axonal failure or nuclear neuronal disease.

Axonal failure is usually classified in:

1.Neurapraxia : it may be considered as the first and early response to nerve compression, realizing a physiological block of conduction.

2.Axonotmesis : The axon is sectioned , and a wallerian degeneration occurs, but the perineural sheath is preserved, allowing axonal regeneration.

3.Neurotmesis : it corresponds to a physiological section of the nerve.

Ryu (1986) investigated histochemical changes in the characteristics of the intrinsic laryngeal muscles 1 to 6 months after damage of the inferior laryngeal nerve in dogs.Three different techniques were used : removal of the nerve, section and crushing of the nerve. Following the

removal of the recurrent nerve. the activity of phosphorylase decreased rapidly and disappeared almost completely within one month after the operation.The activities of nicotine-amide adenine dinucleotide dehydrogenase and succinic dehydrogenase decreased gradually. but they did not disappear within six months. ATPase did not change throughout the period of six months. When the nerve was crushed, the level of enzyme activity did not change significantly. Enzyme activities after the section of the nerve also changed, and the degree of the changes was weaker than in case of removal of the nerve. Grouping of the same type of muscle fibers was observed predominantly in case of section of the nerve.

9.1.2.Electromyographic data

When recovery from injury occurs, the reinnervating axons are immature and of diameter smaller than normal.Consequently, their conduction velocity is considerably reduced.The electomyographic patterns reflect the disintegration of the motor unit and the recruitment phenomenon.In early phases of reinnervation, motor unit potentials have a low amplitude and a richly notched shape.

In later stages of reinnervation, the polyphasic potentials increase in amplitude. These large potentials, sometimes called giant potentials, indicate that abnormal

motor units have been built up by the process of reinnervation.

9.1.3.Axon sprouting

Early after denervation, the membrane of the muscle fibre becomes hypersensitive to acetylcholine (Miledi and Potter, 1971), and is most receptive to reinnervation by regenerating axons (Hatsuyama, 1966).The membrane of a denervated muscle fiber also becomes sensitive to acetylcholine over its entire surface, whereas normally innervated fibers are only sensitive at the motor end plates.

Elsberg (1917) demonstrated that a normal muscle does not accept any additional innervation (hyperneurotization), but, if the muscle's original nerve is injured when the foreign nerve is implanted, then the muscle may become reinnervated by both normal and foreign nerves (dual innervation) (Guth, 1962).Local motor nerves sprout axons freely near denervated motor endplates in response to

1.the production of "neurocletin" described by Hoffman (1950), and nerve growth factor-like substances reported by Lundberg and Hanson (1979), Kiernan (1979), and Spiro (1978), or

2.the absence of "axon-sprouting inhibitor factor", a substance normally found near intact distal axons motor endplates (Diamond et al., 1976).

Active nerve growth factors that stimulate axon sprouting originate from the most distal region of the degenerating axon, near the motor endplate (Diamond et al., 1976; Horiuchi and Sasaki, 1978), or from denervated muscle fibres (van Harreveld, 1945).As a consequence, the neuromuscular pedicle procedure of surgical reinnervation , with preservation of continuity between distal terminations of the nerves and their motor endplates, probably prevents axon sprouting , a crucial phase of muscle reinnervation (Crumley, 1982).

A denervated muscle adjacent to a normally innervated muscle is able to stimulate nerve fibers within the innervated muscle to produce axon sprouts.Sprouting activity seems to be maximal in partially denervated muscles, while a muscle more than half denervated will show less vigorous sprouting (van Harreveld, 1945; Edds and Small, 1951) (Fig.IX.1.).

9.1.4.Muscular atrophy

To be efficient, reinnervation must take place during a ill-defined time period before the muscle fibre undergoes

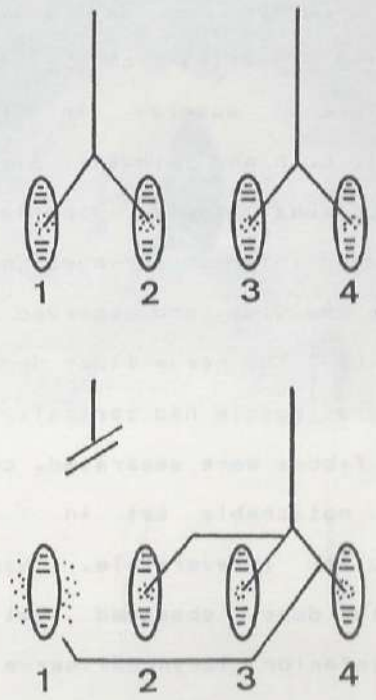


Fig.IX.1.: Above : Schematic muscle innervation.Two axons innervate each two muscle fibers.Axon sprouting inhibiter factor is present in normal muscle.
Below : The first axon is cut.After denervation, nerve growth factor and absence of axon sprouting inhibitor factor cause sprouting of the reinnervating axon toward the denervated motor end plates.

atrophy from denervation.For normal function, the muscle needs a frequent stimulation by the alpha and gamma fibers, as well as neurotrophic substances delivered from the neuron cell body (Ducker and Kaufmann, 1977).The time necessary for atrophy varies from species to species, and even among different muscles in the same subject (Denny-Brown, 1951; Guth and Zalewski, A.,1963; Sunderland, 1978)(Fig.IX.2.). Iizuka (1966) ,experimenting with dogs, pulled up the exposed inferior laryngeal nerve with a small piece of ivory on one side, and observed that, after three days of pulling up, the nerve fiber degeneration and the atrophy of the vocal muscle had partially set in.After one month, the muscle fibers were separated, cut and collapsed, and necrosis had noticeably set in : In this time, the changes were quite irreversible. Nagashima (1970), experimenting with dogs, observed that, after partial removal of the inferior laryngeal nerve with no possible regeneration, the weight of the denervated muscles rapidly decreased, becoming about a half of that of the unaffected side in three months.The muscle fibers progessively were replaced with connective tissue. Slow type I fibres seem to be more prone to atrophy than the more hardy fast-twitch fibres (Dreyfus and Shapira, 1948).Other factors may affect the duration of denervated muscles' survival , as exposure to cold or passive motion during the denervation period.

Atrophy of the intrinsic laryngeal muscles is most

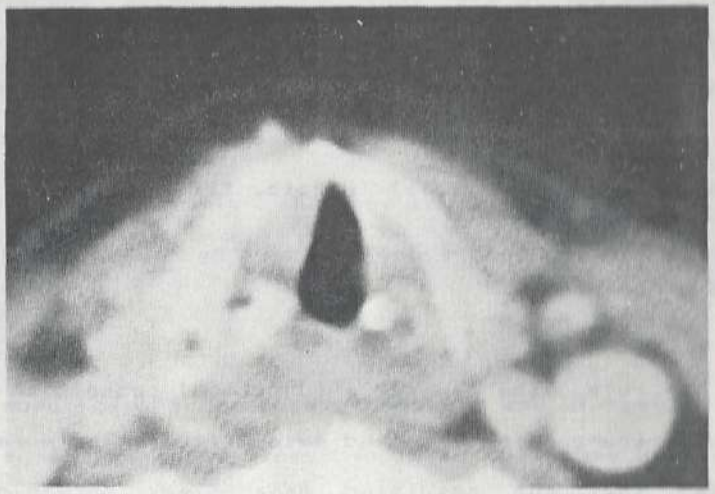


Fig.IX.2.: Muscular atrophy, as it is shown by a CT-scan of larynx, at the level of the right paralyzed vocal fold.In the present case, total denervation occured five years before, and was due to strumectomy.

evident in cases where no voluntary electrical activity is found.When the inferior laryngeal nerve is cut in dogs, the weight of the muscle is reduced to 50 % of the healthy one: however, when the electrical activity is restored , muscle weight returns to normal. In the contrast laryngogram, the vocal fold on the paralyzed side is thinner than on the healthy side (see Fig.V.7.) (Hiroto, 1976).

9.1.5.Fibrosis

As a rule, the sooner a functional connection is made between regenerating axon and muscle fiber, the more likely it is that the resulting metabolism, morphology and function of the muscle cell will return to normal.If there is a delay in reinnervation, fibrosis appears in the muscle, which diverts sprouting axons and prevents them from reaching motor endplate .During the first three weeks following denervation, motor endplates undergo several histological changes and may fill up with collagen if reinnervation does not occur (Bowden and Gutmann, 1944; Crumley, 1982).Long lasting immobilization also causes fibrosis of cricoarytenoid articulation.

9.2. Spontaneous reinnervation

Reinnervation is quite frequent in cases of idiopathic paresis of laryngeal nerves (Haglund et al., 1973) : two

types of potentials are characteristic :

1."Nascent potentials" : they are of low amplitude and polyphasic shape, sometimes richly notched, and indicate early reinnervation.

2."Giant potentials" : they are also polyphasic, but of extremely large amplitude, typical of later stages of reinnervation.

If some of the recorded potentials are of normal type in a case of paresis, they indicate that some of the motor axons remain viable.Thus , some muscle fibers may have been reinnervated through bifurcation of these viable axons.Indications as to the the mode of reinnervation may be obtained from the analysis of the shape of the polyphasic potentials : Those of more or less normal duration and consisting only in a series of repetitive spikes are to be considered as deriving from recently reinnervated muscle fibers.Others are of long duration and consist in smooth waves, accompanied by notches and spikes, suggesting that they derive from a normal motor unit which ,by a sprouting mechanism, has included previously denervated muscle fibers (Haglund et al., 1972 , 1973).

Satoh (1978) observed that, in two cases of spontaneous nerve regeneration after idiopathic paralysis , the

potential evoked by electrical stimulation was of elongated latency and abnormal form.In the two cases, both the superior and the inferior laryngeal were involved.The author suggested that ordinary electromyography and evoked electromyographic test are capable of describing the quality of nerve regeneration.

From a clinical point of view, Wendler et al.(1984) noticed that , considering all cases together, a respiratory motion of the paralyzed vocal fold is fully-recovered in 10-15 % of the subjects, partially in 30-40 %, and not at all in 30-40 %.After strumectomy, partial or complete recovery occurs in 40 % of the cases; after neuritis in 50 %, and in case of undefined etiology in 80 %. Most of the the patients recover mobility of the paralyzed fold, even as a better voice, after 3 months.Nevertheless, some patients recover after one year, and even after two years (Fig. IX.3. and IX.4).

9.3. Misdirected regeneration

In 1927, Colledge and Ballance first described the phenomenon of paradoxical glottic movements of expiratory abduction and inspiratory adduction after inferior laryngeal nerve repair.

Tomita (1967) published electromyographic investigations

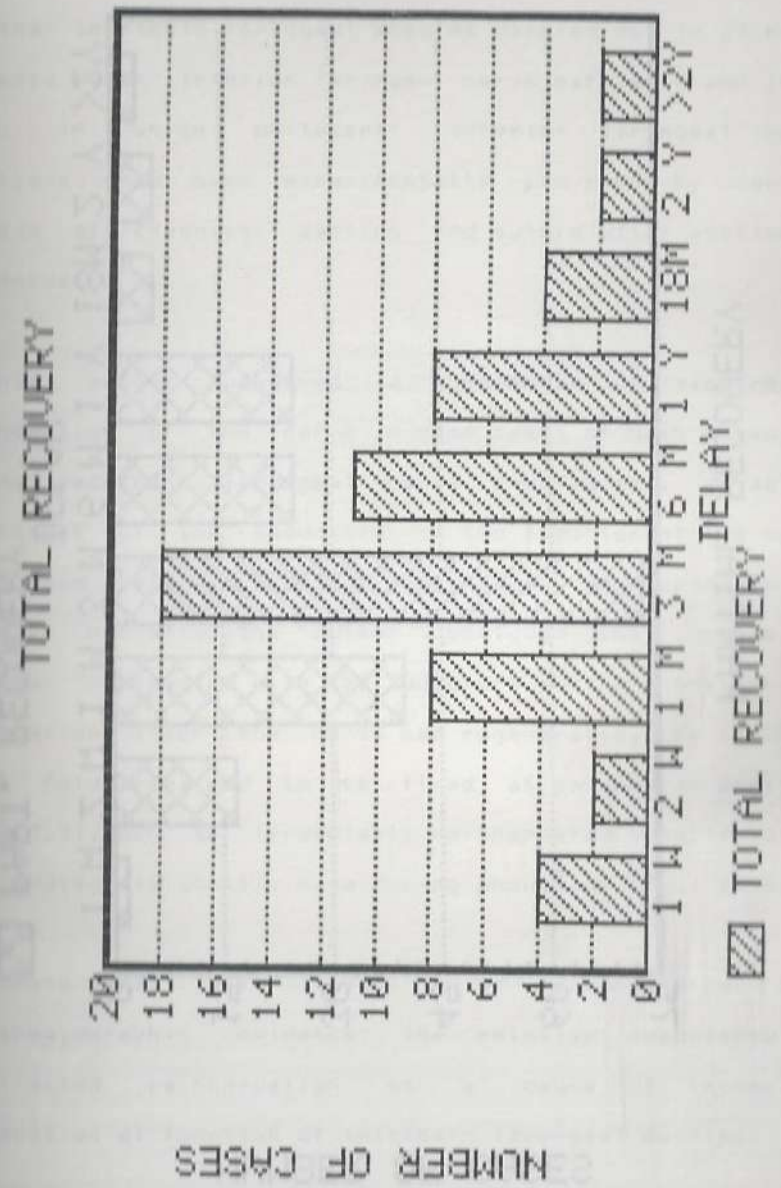


Fig.IX.3.: Time delay for clinical complete recovery of vocal fold motion after paralysis, after Wendler's et al. compiled data (1984).Most of remobilisations of paralyzed vocal folds occur after 3 months, but favourable evolution may still be observed after one or even two years.

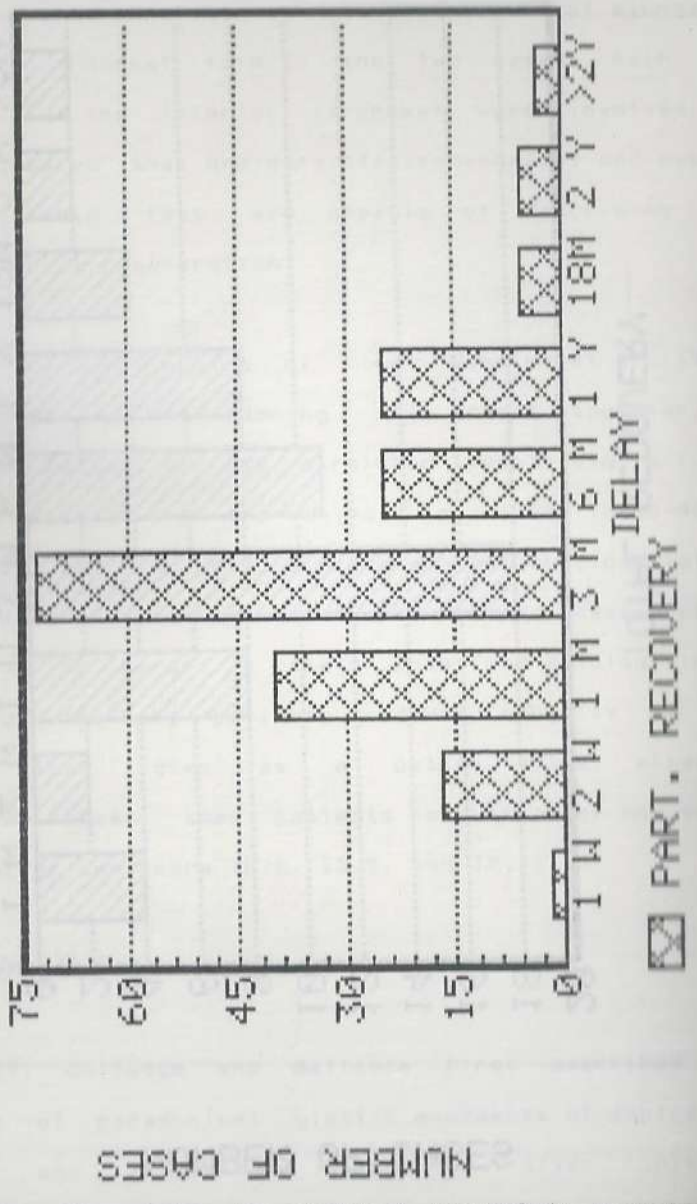


Fig.IX.4.: Time delay for clinical incomplete recovery of vocal fold motion after paralysis, after Wendler's et al. compiled data (1984).Even as for cases of complete recovery, most of remobilisations occur after 3 months, but late recoveries are not exceptional.

of the intrinsic laryngeal muscles carried out in 29 human subjects with inferior laryngeal nerve paralysis and in 14 dogs, in which unilateral inferior laryngeal nerve paralysis had been experimentally provoked by various methods of crushing, section and suture after section of the nerve.

This author observed a phenomenon of misdirected regeneration of the nerve in some cases of both human and canine recurrent laryngeal nerve paralysis:i. e. active discharges of the adductors or the abductor of the vocal fold were recorded during inspiration and expiration as well as phonation.The author concluded that , since the abductor contracted with the adductors during phonation and respiration after the nerve had regenerated, the involved vocal fold remained to be fixed at paramedian position (Fig.IX.5).Such an irregularly reinnervated vocal fold may show a discrete glottic wave during phonation (Fex, 1970).

Hirota et al. (1968) also demonstrated with electromyographic evidence the relative importance of misdirected reinnervation as a cause of incomplete restoration of function of intrinsic laryngeal muscles.

Satoh et al. (1974) applied sectioning and repairing , as well as crushing inferior laryngeal nerves in dogs, and observed in some cases abnormal motion patterns , which

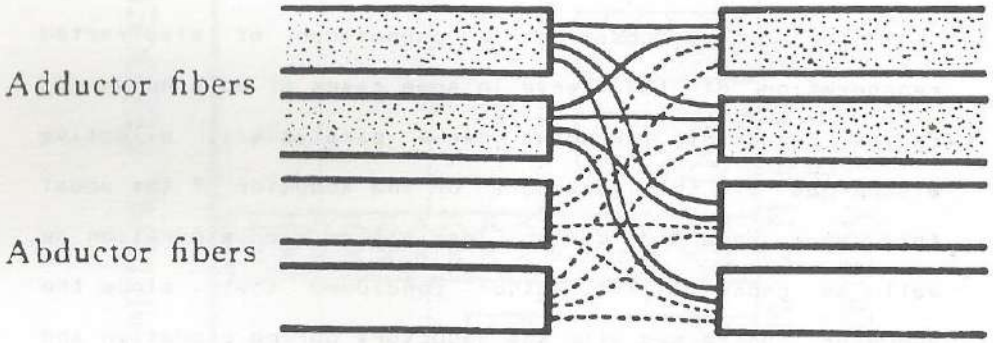


Fig.IX.5.: Schematic misdirected regeneration after inferior laryngeal nerve section (Tomita, 1967).This phenomenon is responsible for lack of remobilisation of the vocal fold, and even for abnormal motion patterns (Fig.IX.3).

they attributed to misdirected reinnervation.

Hiroto (1976) observed that, in some cases of misdirected regeneration in humans, the vocal fold on the paretic side abducted slightly during phonation when the healthy fold adducted.

A personal example of electromyographic evidence of misdirected regeneration is shown in Fig. IX.6.

Therefore, selective treatments to adductors or abductors have been designed (Murakami and Kirchner, 1971 ; Iwamura, 1974 ; Matsui, 1976 ; Sato and Ogura, 1978).

9.4. Surgical reinnervation

Early in the 20 th century, Horsley (1909) presented a successful case of simple neurorrhaphy of the left inferior laryngeal nerve in a woman, who had been shot by a pistol.Both nerve stumps were approximated after excision of the diseased portion.About 15 months postoperatively, the vocal folds had a good mobility, the left one lagging slightly behind the normal side on adduction (misdirected regeneration ?).

In 1924, Frazier used the ansa hypoglossi for end-to-side nerve anastomosis with the inferior laryngeal

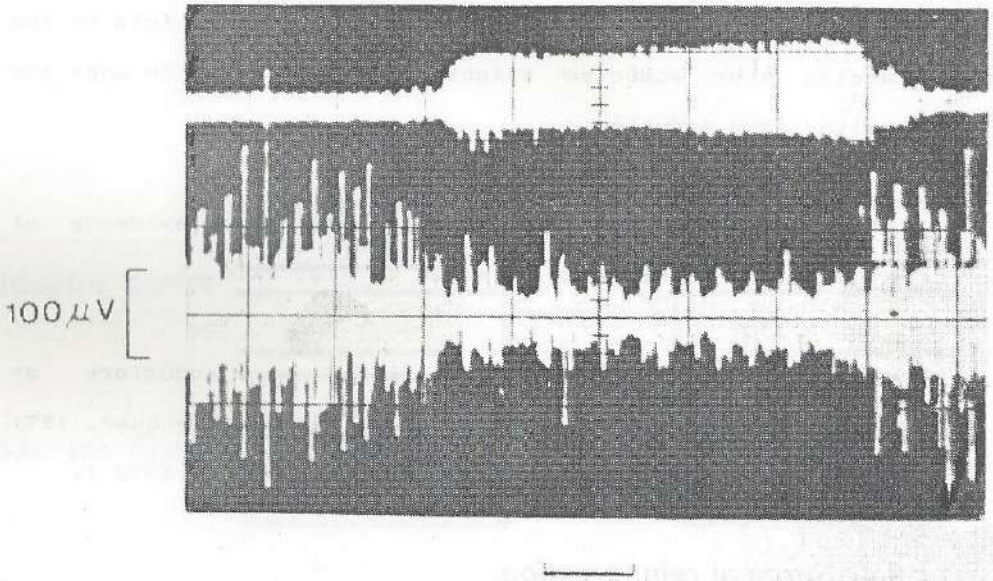


Fig.IX.6.: Electromyographic pattern in a thyroarytenoid muscle, during phonation, after misdirected reinnervation.Above : sound oscillogram; Below : Electromyogram.A paradoxical decrease of muscular activity is observed during sound emission.Clinically, the trophic vocal fold remained immobilized in a paramedian position.Time calibration is 10 ms.

nerve.

A detailed review of historical aspects of surgical reinnervation is given by Sato and Ogura (1978).

Satoh et al. (1974) sectioned and repaired the inferior laryngeal nerve in dogs, and compared muscular functions with that of animals in which the nerve had been crushed or subjected to sustained pressure : Reinnervated intrinsic laryngeal muscles showed impaired function .Besides the misdirected reinnervation of muscle fibers by inappropriate components of the regenerating fibers of the nerve, the authors considered three reasons as contributing more or less to this reduction of function of the reinnervated laryngeal muscles :

1.The sprouting nerve fibers often fail to reach the muscle fibers.

2.Connective tissues developes between muscle fibers at the same time as the muscle fibers atrophy.

3.There is an obstruction of mechanisms in the process between electrical and mechanical changes leading to contraction, which may be caused by chemical changes in the muscle fibers (electro-mechanical uncoupling).

Shin (1971) showed that , in dogs, after section and surgical suture, the regenerated nerve fibers were less in number and that there was considerable development of fibrous tissue.

A complete recovery of the laryngeal function , with cyclic adequate abduction and adduction is thus not to be expected after regeneration of the inferior laryngeal nerve (Crumley, 1982).

Such considerations could also be a plausible explanation for the discrepancy between the good quality of electrophysiological reinnervation and the poor clinical mobilization of the vocal fold.

Doyle (1964) and Doyle et al. (1967) pioneered the selective reinnervation of the posterior cricoarytenoid muscle by implantation of the inferior laryngeal nerve.Miglets (1974) reported a clinical success with this technique.

Tucker (1976, 1978, 1979) obtained good results with a nerve-muscle pedicle technique in cases of bilaterally paralyzed vocal folds with insufficient airway.He dissected free a small portion of muscle (2-3 mm square) from the anterior belly of the omohyoid, so as to include the nerve (a branch of the ansa hypoglossi).This prepared

nerve-muscle pedicle was sutured to the posterior cricoarytenoid muscle.Appropriate abduction of the reinnervated side was noted, after 6 - 12 weeks, at rest, in approximately 50 % of the cases.Some of the remainder were observed to abduct after a modified Master two step test.This technique does not involve cutting most of the terminal nerve fibers, thus avoiding neural degeneration.A nerve branch, not derived from the vagus nerve but spontaneously firing during inspiration is used (the sternothyroid muscle is also convenient).Finally, because only the abductor muscle is selectively reinnervated, misdirected reinnervation, inherent to nerve anastomosis techniques is avoided.Other authors confirm the good results (May et al., 1980).Nevertheless, actual axon sprouting is questionable (cf.supra) : Experiments made by Crumley (1982) and Rice et al. (1983)suggest that , although the ansa hypoglossi neuromuscular pedicle may enlarge the glottic airway during dyspnea, it does not do so by actually reinnervating the posterior cricoarytenoid muscle : The mechanism of action should be an enhancement of he accessory vocal fold "abduction" produced by accessory muscles of respiration, while assisting the cricothyroid muscle with enlargement of the glottis in the antero-posterior dimension. Electromyographic data and muscle histology reveal that the posterior cricoarytenoid muscle may be reinnervated by adjacent inferior constrictor muscle following the procedure.A recent study by Shyue-Yih

Chang (1985) shows that , three months after nerve-sternohyoid muscle pedicle graft in paralyzed posterior cricoarytenoid muscle in dogs, two thirds of the animals demonstrate excitable evoked muscle action potentials in the operated muscle.The wave form is dispersed and in some dogs the latency is longer.However, this experiment does not provide direct evidence of reinnervation : Other neural regeneration could have caused a similar effect.On histological examination, the grafted neuromuscular pedicle shows severe degeneration of muscle fibers : Almost the whole grafted muscle is replaced by fibrous tissue.In some dogs, neural proliferation is seen at the junction of the neuromuscular pedicle and the posterior cricoarytenoid muscle.There are no obvious nerve fibers growing across the junction.

Neal et al. (1983) performed omohyoid nerve-muscle pedicle tranposition in subhuman primates : after 6 to 8 week, galvanic stimulation of the omohyoid nerve pedicle caused lateralization of the paralyzed vocal fold, although no inspiratory abduction was noted. Electron microscopy uniformly showed the presence of both myelinated and unmyelinated nerve fibers in the posterior cricoarytenoid muscle on the denervated side. The attempts to show horseradish peroxidase transport via the ansa hypoglossi to the cervical spinal cord motor neurons have never been successful.However, there was transport of peroxydase from

the posterior cricoarytenoid muscle to the nucleus ambiguus on the same side as the inferior laryngeal nerve section.

Fex (1966), Taggart (1971) and Morledge et al. (1973) implanted the phrenic nerve into the posterior cricoarytenoid muscle, in cats and dogs.This nerve conducts inspiratory impulses to the diaphragm a few milliseconds after the inspiratory discharges of the posterior cricoarytenoid muscle (Sato and Ogura, 1978), but the physiological similarities between the diaphragmatic and posterior cricoarytenoid muscles are so apparent that it has been suggested that both muscles are innervated by the same group of neurons in the brainstem (Wyke, 1968).Some of the operated animals demonstrate inspiratory vocal fold abduction.However, the function of the diaphragm should not be sacrified, although unilateral sectioning of the phrenic nerve seems to be very well tolerated (Fackler et al., 1967; Robertson, 1979).

Crumley et al. (1980) improved the technique by splitting the phrenic nerve, and routing half of the fibres to the posterior cricoarytenoid muscle via a free nerve graft.With this technique, cyclic inspiratory contraction of the paralyzed posterior cricoarytenoid muscle is obtained in dogs (Crumley, 1982).

9.5. Artificial long-time stimulation of denervated posterior cricoarytenoid muscle

Considering the problems occurring with surgical reinnervation techniques, some authors (Obert et al., 1984; Zrunek et al., 1986) experienced implanting stimulation electrodes into the posterior cricoarytenoid muscle after section of the inferior laryngeal nerve (only animal experimentation).Long-time interrupted stimulation could prevent as well abductor muscle atrophy as arytenoid ankylosis.Some problems with electrode stability and corrosion still need adequate solution.

Chapter X

Neuromyography and reflexmyography

10.1. Introduction

In detection electromyography, the recorded muscle action potentials are either spontaneous or volitional.Muscle action potentials can also be evoked by electrical stimulation, using surface or needle electrodes.Moreover, electrical stimulation of afferent nerves initiates reflex evoked waves in concerned muscles (Goodgold and Eberstein, 1972; Mercelis, 1986).Laboratory experimentation has been performed in cats by Suzuki and Sasaki (1976).

Neuromyography is the stimulation of a nerve with recording of the electrically induced muscle activity of the innervated muscle.Reflexmyography is the stimulation of an afferent nerve with recording of the reflex muscular response.A well known example of the latter is the blink reflex : The supraorbital nerve is stimulated and the reflex response is picked up in the orbicularis oculi.In the case of the larynx, the afferent branch of the superior laryngeal nerve is stimulated; after switching over to the vagal nuclei, an efferent reflectory activity is evoked in

the vagus nerve and the laryngeal motor nerves, with a muscle response potential in the intrinsic laryngeal muscles (Thumfart, 1986). Furthermore, late contralateral evoked potentials suggest a crossed pathway, with possible thalamo-cortical relays.

In case of injury of the neuro-muscular system, the evoked electromyographic test is in general considered as the most useful test in estimating the degree of injury at an early stage after the onset.It contributes to the prognosis and the setting up of the policy of treatment (Satoh, 1975).Shin (1971) has demonstrated in dogs that, in case of a regenerated inferior laryngeal nerve, the evoked potentials reveal prolonged latency and a characteristic pattern.

10.2. Methods of stimulation and recording

Classically, when the internal branch of the superior laryngeal nerve is stimulated, a reflexive closure of the glottis results (Arnold, 1961).
Abo-El-Enein and Wyke (1966) also show that afferent discharges provoked from muscular mechanoreceptors (spiral nerve endings and muscle spindles) by graduated passive stretches applied to individual laryngeal muscles are capable of producing sustained, reciprocally co-ordinated changes in the tone of the intrinsic muscles of the larynx.

Peytz et al. (1965) stimulated the inferior laryngeal nerve with stainless steel needles (diameter 0.7 mm, length 50 mm), insulated except for a length of 3 mm at the tip.They applied the stimulus

1. To the inferior laryngeal nerve near the trachea : Cathode I was placed paratracheally at a distance of 2.5 cm below the most prominent point of the cricoid arch.

2. To the vagus nerve just before the branching of the recurrent nerve : Cathode II was inserted at the same level as cathode I at the posterior edge of the sternocleidomastoid muscle.

The depth of the electrodes was varied until the lowest threshold of the action potential was obtained.

Anode I was placed in the subcutaneous tissue of the jugular fossa, Anode II in the subcutaneous tissue , anterior to the sternocleidomastoid muscle.

The stimuli were rectangular current pulses of 0.1 or 0.2 msec duration.

The action potential of the vocal muscle is detected by a concentric needle electrode (external diameter 0.45 mm)

.The electrode is introduced either through a direct laryngoscope (local anaesthesia and pethidine intravenously), or during a total laryngectomy.

Atkins (1973) used a similar method.

Satoh (1978) stimulated the internal branch of the superior laryngeal nerve, vagus nerve and inferior laryngeal nerve with electrodes made of copper wire measuring 80 u in diameter coated with enamel except for* 5 mm along the edge (Fig.X.1.). In order to stimulate the internal branch of the superior laryngeal nerve, the electrodes are inserted approximately 1 cm deep at the distance of 1 cm so that the region where this nerve penetrates the thyrohyoid membrane may be the center of the electrodes.To stimulate the recurrent nerve, the anode is inserted 3 cm below the lower margin of the cricoid cartilage and the cathode 1 cm above the anode at the depth of approximately 2.5 cm for both electrodes.To stimulate the vagus nerve, the anode and the cathode are inserted approximately 2.5 cm deep at the external margin of the sternocleidomastoid muscles, the anode being above and the cathode below, at the same height of the electrodes as in stimulating the recurrent laryngeal nerve.

The bipolar concentric needle electrode used for recording is inserted percutaneously into the cricothyroid

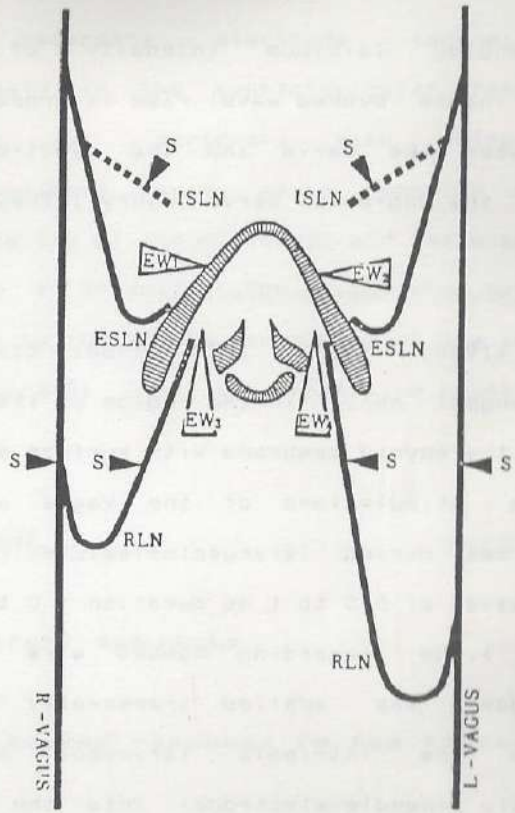


Fig.X.1.: Location of stimulation and recording electrodes, according to Satoh (1978).
ISLN: Internal branch of the superior laryngeal nerve.
ESLN: External branch of the superior laryngeal nerve.
RLN: Recurrent laryngeal nerve.
Arrow: Stimulation.
EW1: Evoked waves induced from the right cricothyroid muscle.
EW2: Evoked waves induced from the left cricothyroid muscle.
EW3: Evoked waves induced from the right thyroarytenoid muscle.
EW4: Evoked waves induced from the left thyroarytenoid muscle.

muscle and the thyroarytenoid muscle.

The thresholds (minimum intensity of stimulation required to induce evoked wave) rise in proportion of the distance between the nerve and the electrodes but also according to the degree of nerve injury : They vary from 2 to 20 volts.

Thumfart (1980, 1981, 1983, 1986) stimulated the superior laryngeal nerve in the region of its penetration through the thyrohyoid membrane with surface electrodes.He also applied stimulations of the vagus and inferior laryngeal nerves during laryngectomies.Electric impulses were square waves of 0.5 to 1 ms duration (0 to 10 mA ; 50 to 100 V).The recording hooked wire (or bipolar needle-electrode) was applied transorally under optic control into the intrinsic laryngeal muscles, or transcutaneously (needle-electrode) into the cricothyroid muscle.

In our experience, stimulation of the inferior laryngeal nerve is difficult to realize in clinical routine, because this nerve is difficult to find and to access.Deep insertion of a rigid electrode may be painful, and is not without danger.The superior laryngeal nerve is easy to stimulate efficiently and with minimal discomfort for the patient : The tip of the rigid active electrode (cathode)

is inserted through the skin, just under the major horn of the hyoid bone , at approximately 1 cm depth under the skin.The reference electrode (anode) is located subcutaneously in the supraclavicular fossa. Electrical stimulation is performed with pulses of 1 ms duration.Amplitude varies as a function of the distance between the tip of the electrode and the superior laryngeal nerve (5 to 50 Volts).The stimulation current intensity extends up to 100 mA. The frequency of the impulses is 1 or 2 per second.At least ten repetitive responses need to be recorded.

10.3. Results

10.3.1.Normal subjects

10.3.1.1.Evoked response in the thyroarytenoid muscle

With our technique, the first response obtained in the thyroarytenoid muscle after stimulation of the superior laryngeal nerve is a mechanical artifact, due to the contraction of the cricothyroid muscle, with passive stretching of the vocal fold.Its latency is about 2-3 milliseconds.The second response is a true evoked muscular potential, occuring after about 15 ms in the right-hand side, 17 ms in the left-hand side (Fig.X.2.and X.3). The

latency increases with the heigth of the subject.The difference (1.5 to 2.5 ms) is to be related to the difference of length between the two inferior laryngeal nerves (+/- 11 cm in a normal adult subject). Thumfart relates somewhat longer latencies, and does not observe a mechanical artifact, but the latter may be due to the fact that hooked wire electrodes are used rather than concentric needle electrodes.A controlaterally evoked potential may also be recorded, and in our experience, its latency is considerable (about 60 ms).Its neurophysiological and clinical significance requires further investigations.

10.3.1.2.Evoked response in the cricothyroid muscle

With a same method of stimulation, a direct muscular reponse is obtained in the cricothyroid muscle, with a short latency (2 - 3 ms) : The distal efferent nerve length is estimated to be about 8 cm. Simultaneously, afferent fibers of the superior laryngeal nerve are also stimulated, and provoke a second, reflex response, about 12 ms after the beginning of stimulation.The same values are found in both sides (Fig.X.4. and X.5.). This has also been reported by Thumfart (1983, 1986), who mentions again somewhat longer latencies, as well for the first potential (4 - 7 ms) as for the second one (16 - 18 ms) . The evoked response may be also controlateral, while the direct muscular response is only ipsilateral.

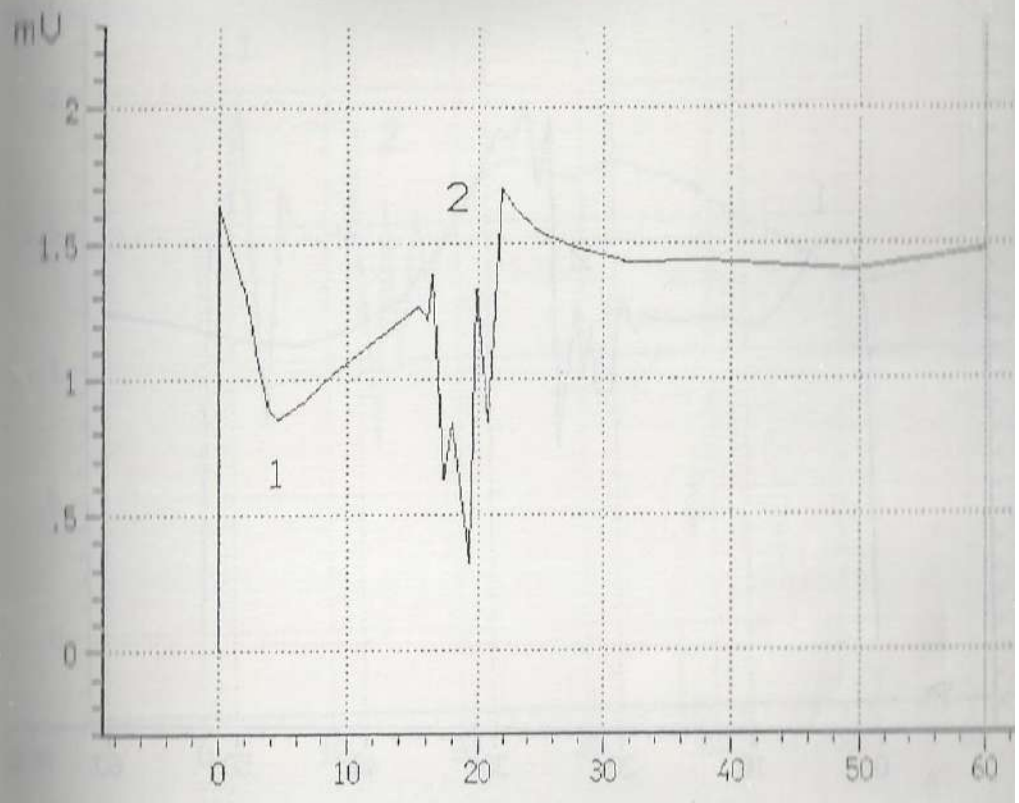
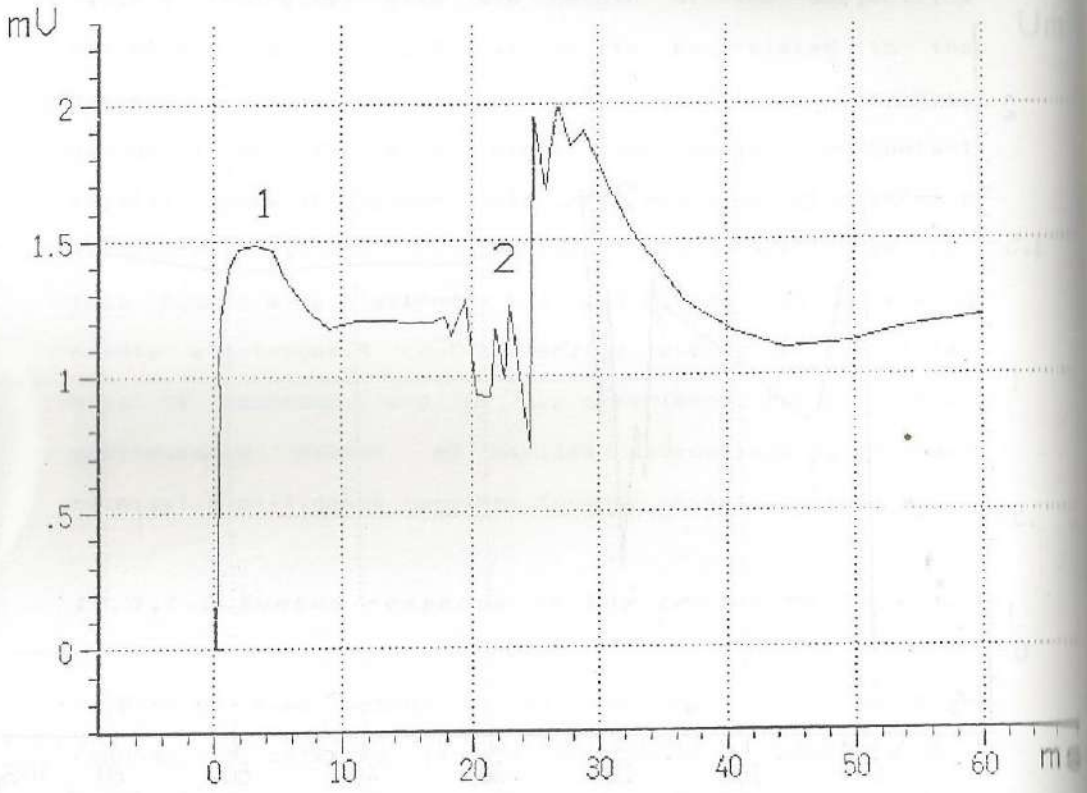


Fig.X.2.: Evoked response in the right thyroarytenoid muscle after stimulation of the right superior laryngeal nerve.

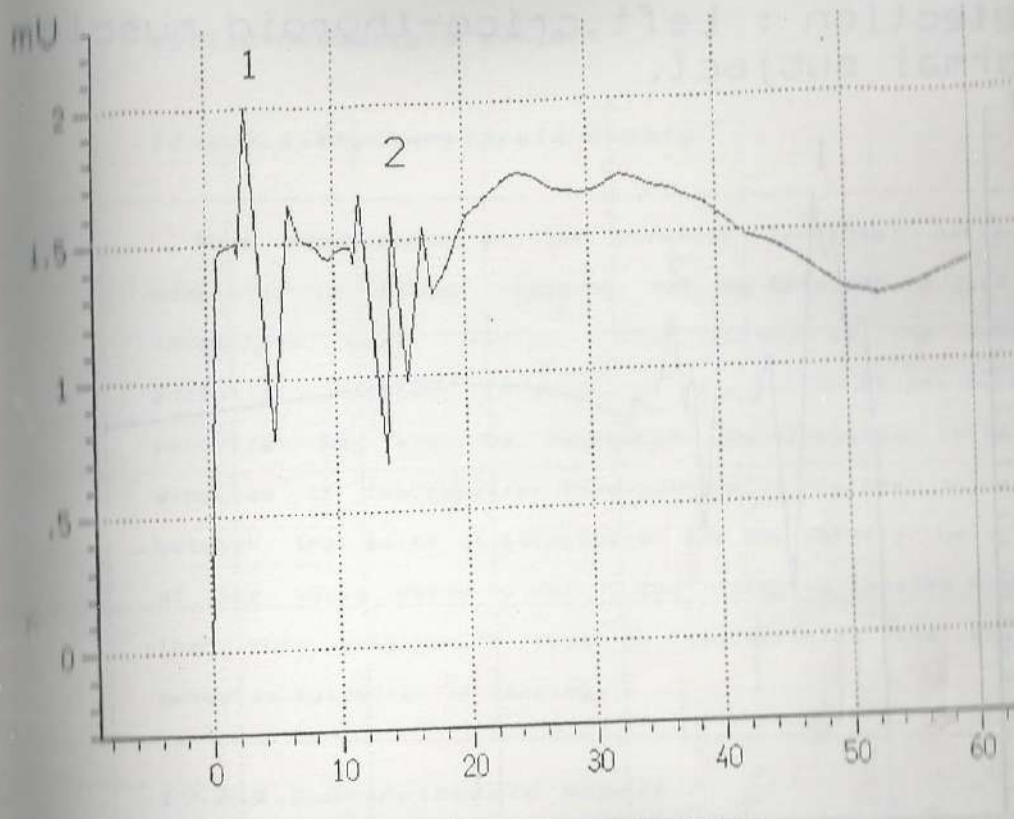
Normal subject.



1 : Mechanical artefact (Contraction of crico-thyroid muscle)

2 : Evoked muscular potential
Latency : 17.5 ms.

Fig.X.3.: Evoked response in the left thyroarytenoid muscle after stimulation of the left superior laryngeal nerve.Latency is about 2 ms longer than on the right side.



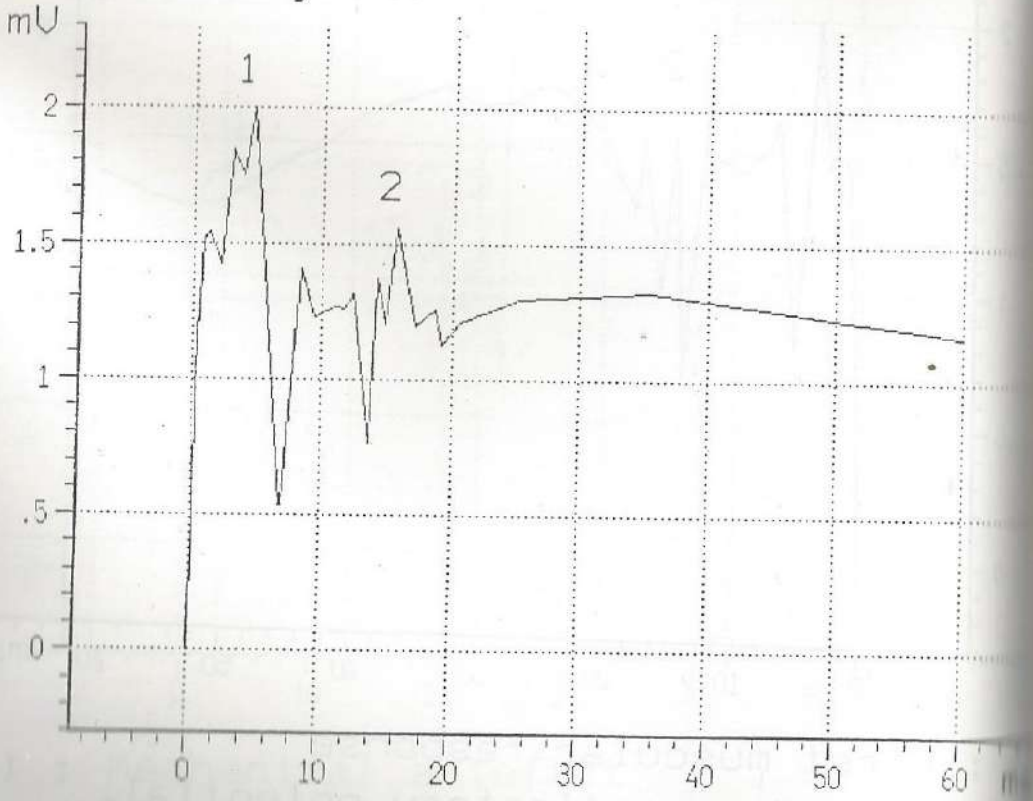
1 : First muscular response

2 : Second or reflectory potential.

Latencies : 2.8 and 11.8 ms.

Fig.X.4.: Direct and evoked response in the rig cricothyroid muscle after stimulation of the right superi laryngeal nerve.

Detection : Left crico-thyroid muscle
Normal subject.



1 : First muscular response
2 : Second or reflectory potential.
Latencies : 2.2 and 12 ms.

Fig.X.5.: Direct and evoked response in the left cricothyroid muscle after stimulation of the left superior laryngeal nerve.

10.3.2.Pathologic subject

10.3.2.1.Thyroarytenoid muscle

When denervation of the inferior laryngeal nerve is complete, no evoked response can be obtained.In case of incomplete nerve lesion, the latency of the evoked potential increases (Fig.X.6.). The duration of the evoked potential may also be increased. No response is to be expected if the superior laryngeal nerve has been injuried between the point of stimulation and the inferior ganglion of the vagus nerve , nor if the lesion is located higher than this ganglion.In case of neurapraxia, the evoked muscular potential is lacking.

10.3.2.2.Cricothyroid muscle

Surgical trauma is frequently more distal than the stimulation level: it may either be partial or total, and alters the direct response.More proximal lesions cause a rapid wallerian degeneration, again either partial or total.In some cases, the evoked response is delayed and highly polyphasic. In one case with a partial nuclear lesion, we found only a direct response : The evoked response was lacking.

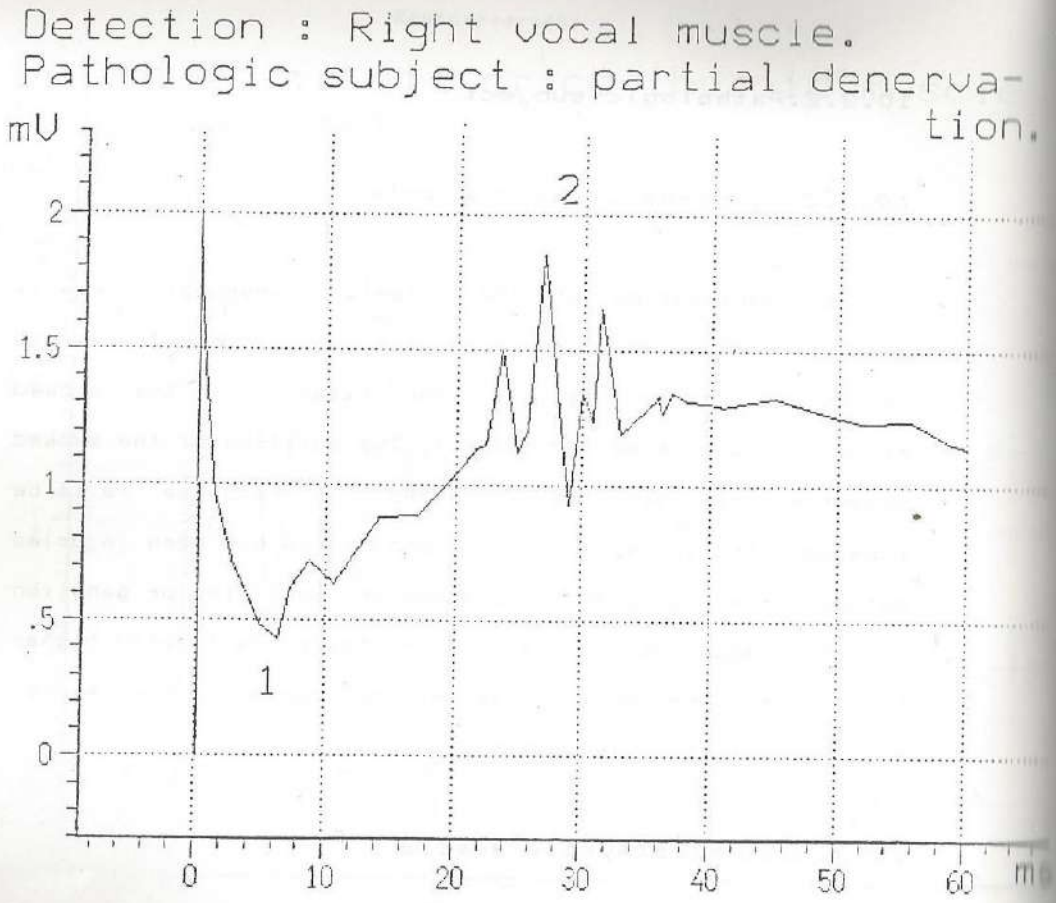


Fig.X.6.: Evoked response in the right vocal muscle after stimulation of the right superior laryngeal nerve.Pathologic subject with slight denervation of the muscle after strumectomy.An evoked response is present, but with a longer latency than normal.

As a general rule, it is interesting to compare the responses with those of the contralateral side.

These findings are in agreement with those of Thumfart (1983, 1986).

10.4. Conclusions

With the use of neuromyography and reflexmyography, it has become possible to test the complex polysynaptic reflex pathways of laryngeal innervation, and to obtain valuable information about the site and the degree of the nerve injury, as well as about the state of nerve regeneration.Further neurophysiological and clinical investigation in this fiels seems very promising.

E.M.G. – ATLAS

100 μV

100 ms

Subject:

Male, aged 34.

Muscle:

Right thyroarytenoid.

Description:

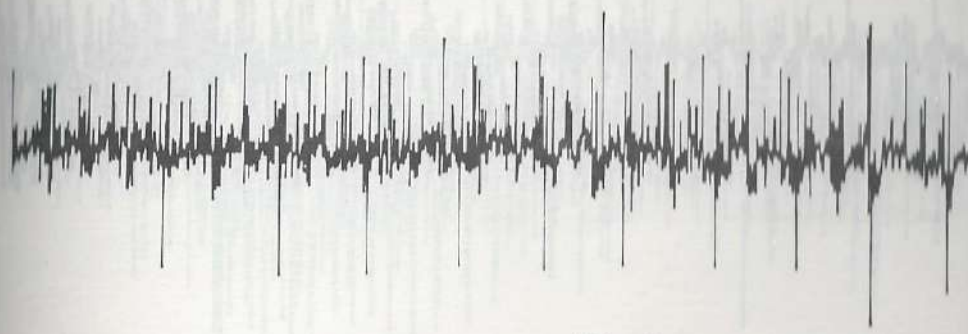
Single motor unit potential pattern.

Case history:

Normal subject.

Comment:

Spontaneous normal resting activity in a vocal muscle.



100 μV

100 ms

Subject:

Male, aged 46.

Muscle:

Right thyroarytenoid.

Description:

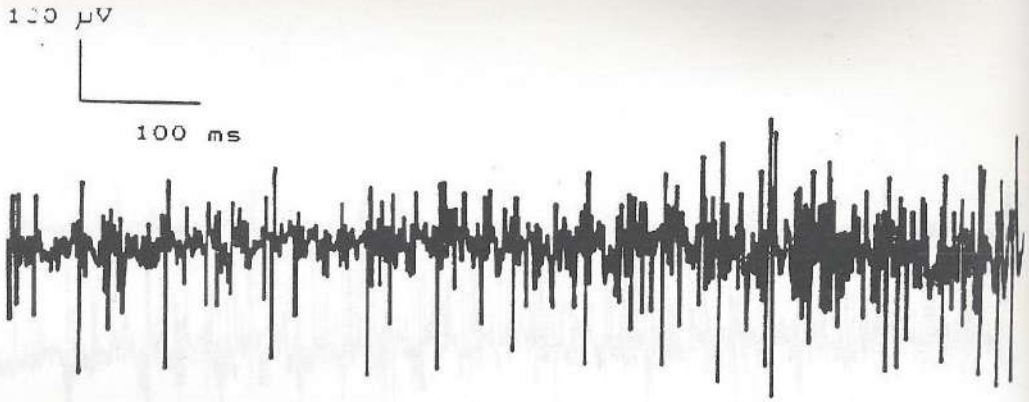
Intermediate pattern.

Case history:

Normal subject.

Comment:

Vocal emission of low pitch and moderate intensity. Several motor units are activated, and fire at various frequencies.



Subject:

Male, aged 43.

Muscle:

Right thyroarytenoid.

Description:

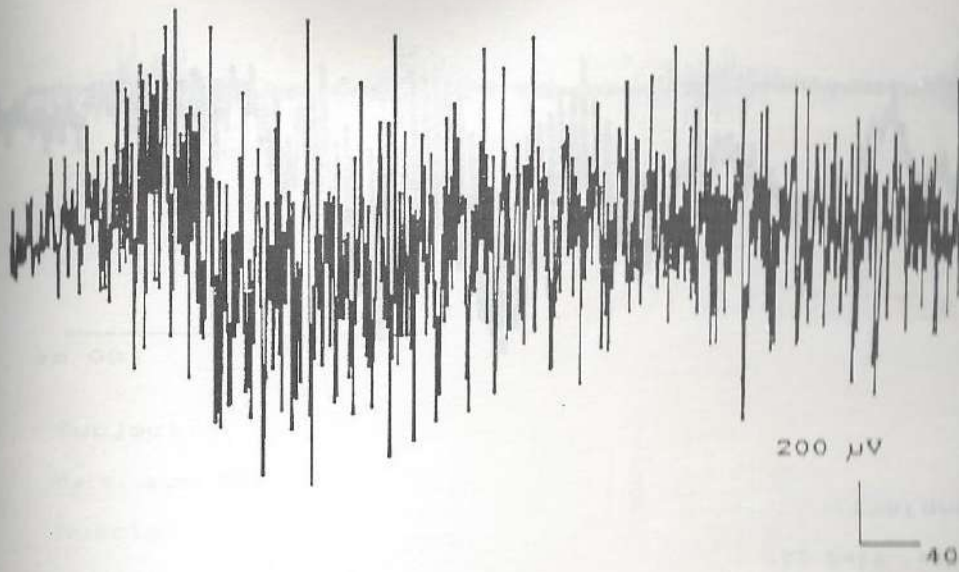
Normal motor unit potentials. As greater tension is developed in the vocalis muscle, the rate of discharge of active motor units increases, and also additional motor units are recruited (partial unterference pattern.

Case history:

Normal subject during voicing.

Comments:

Continuous regulation of pitch and intensity requires adequate adjustments of muscle activity of the intrinsic laryngeal muscles.



Subject:

Male, aged 34.

Muscle:

Right thyroarytenoid.

Description:

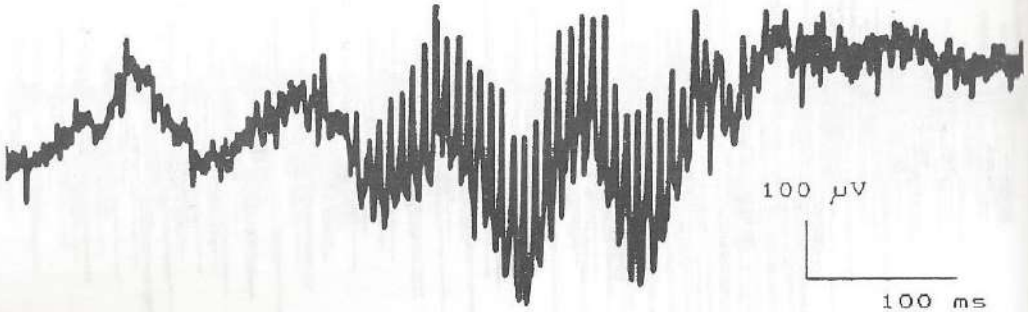
Normal voluntary action potentials. Interference pattern.

Case history:

Normal subject during singing.

Comments:

Submaximal contraction of the muscle (high pitched and rather loud emission).Many potentials, firing at a high frequency, interfere with one another so that single potentials can no longer be discriminated.



Subject:

Male, aged 55.

Muscle:

Left thyroarytenoid.

Description:

Microphonic effect, synchronous with vocal fold phonatory oscillation, and of same pitch.These sinusoidal waves are not to be taken for action potentials.

Case history:

Important bilateral Reinke's oedema, in relation with smoking habits.Slight mechanical impairment of vocal fold motion, but absence of neuromuscular pathology.

Comments:

The tip of the concentric needle electrode is not positioned into the vocalis muscle, but submucosally into the gelatinous substance of Reinke's space.

Subject:

Male, aged 37.

Muscle:

Right thyroarytenoid.

Description:

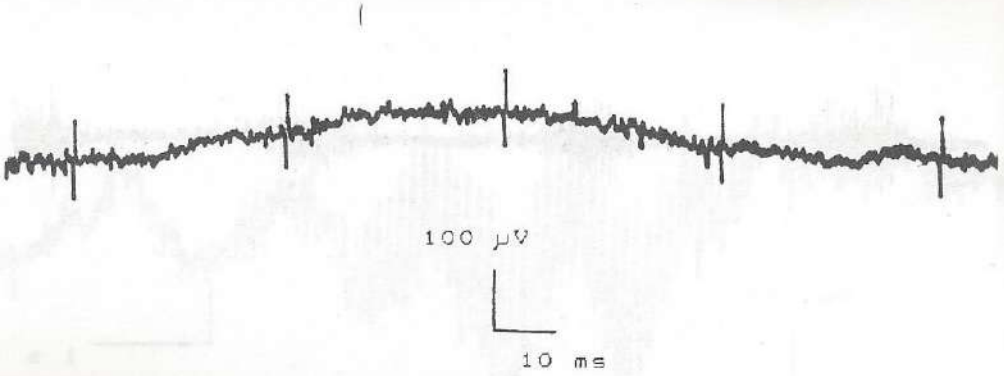
Spontaneous rythmicity, synchronous with breathing.Activation occurs during inspiration, and is more important than normally.

Case history:

Severe head and neck trauma one year ago.Moderate laryngeal stenosis due to fibrosis, caused by long term intubation.No nerve injury.

Comment:

Increased variations of rhythmical electromyographic activity may be seen in normal subjects during forced breathing.This subject has a permanent increase of resistance to the air flow at the level of his larynx.



Subject:

Female, aged 39.

Muscle:

Left thyroarytenoid.

Description:

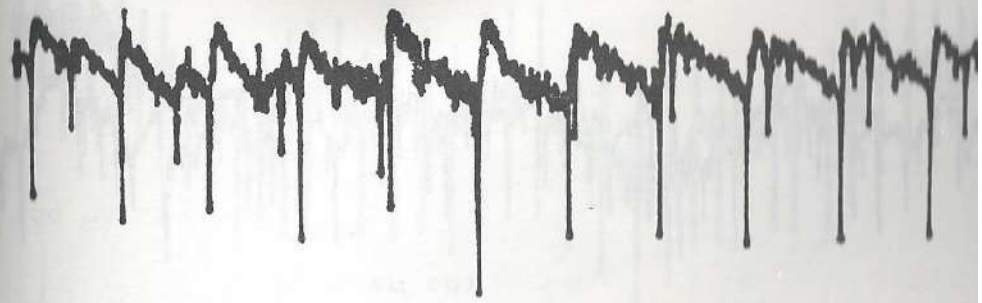
Five fibrillation potentials.

Case history:

Unilateral total denervation after strumectomy.

Comment:

Pathological spontaneous activity after complete nerve section.Fibrillation potentials may appear as soon as 5-6 days after denervation, and remain present for several years.Duration (with concentric needle electrode) is about 1-3 msec and amplitude low: usually less than 0.15 mV.The sound they produce in the loudspeaker is typical, even as their regular rhythm (1 to 30 / second).They are usually associated with positive sharp waves, but appear here isolated.Absence of voluntary action potentials.



Subject:

Female,aged 56.

Muscle:

Left thyroarytenoid muscle.

Description:

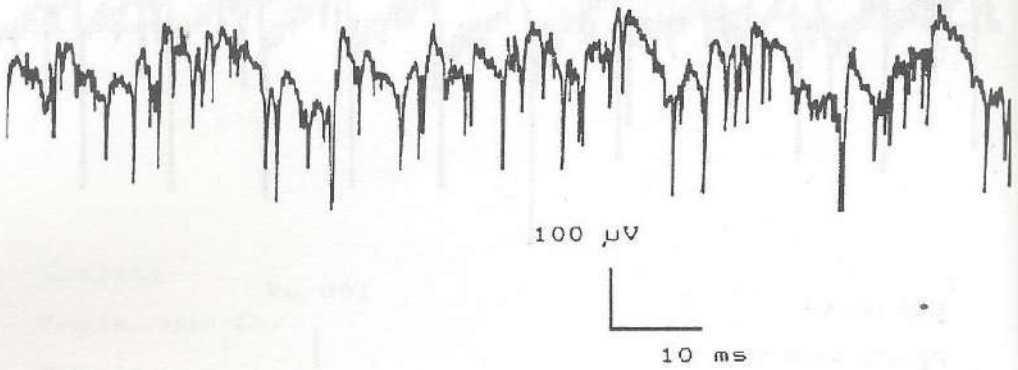
Positive sharp waves and fibrillation potentials.Absence of motor unit potentials.

Case history:

Strumectomy 2 months before.Paralysis of the left vocal fold.

Comments:

Pathological spontaneous activity.Positive sharp waves are diphasic potentials characterized by a sharp initial positive deflection followed by a slow decay into the negative direction.This negative phase is lower in amplitude, but prolonged in duration, sometimes continuing for 100 msec.Peak-to-peak amplitude is usually 50 uV to 1 mV.Frequency of discharge may range from 2-100 / sec.



Subject:

Male, aged 65.

Muscle:

Right cricothyroid.

Description:

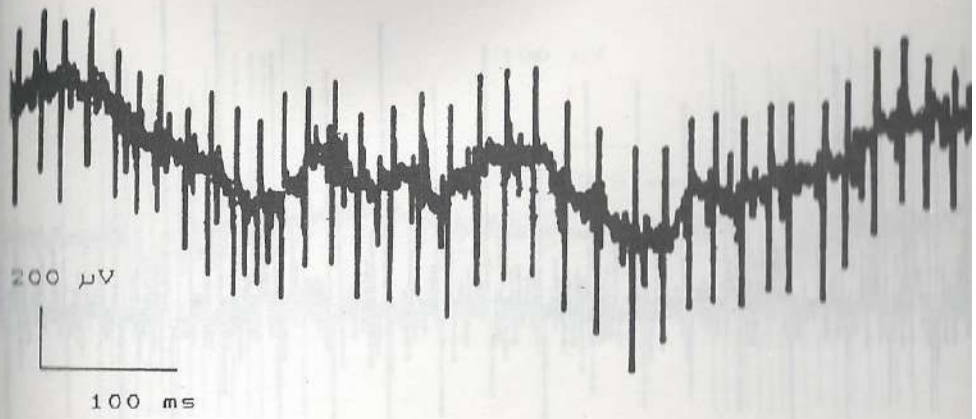
Abundant positive sharp waves and fibrillation potentials.

Case history:

Partial laryngectomy, 2 months ago.Complete nerve section of the right superior laryngeal nerve.

Comments:

Important pathological spontaneous resting activity, without motor unit potentials.



Subject:

Female, aged 56.

Muscle:

Right thyroarytenoid.

Description:

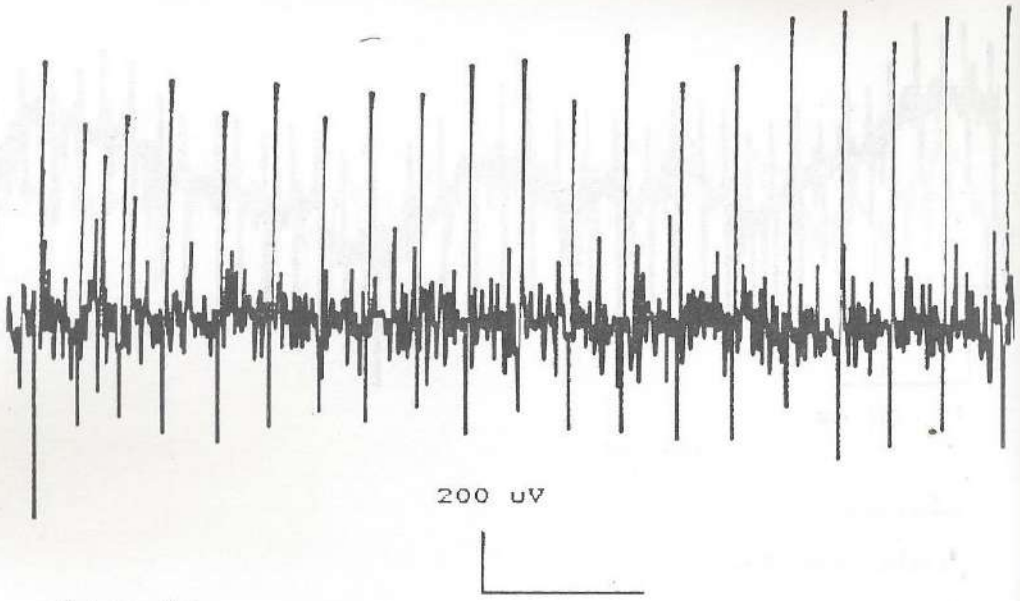
Single motor unit firing at about 40 - 50 per second.The baseline further shows a small superimposed microphonic effect.

Case history:

Neck trauma with knife, 2 months before.Paralysis of the right vocal fold.

Comments:

In partial neurogenic lesions, with a decreased number of functional motor units, the few remaining motor units fire at high frequencies (> than 25 per second).



Subject:

Male, aged 68.

Muscle:

Right thyroarytenoid.

Description:

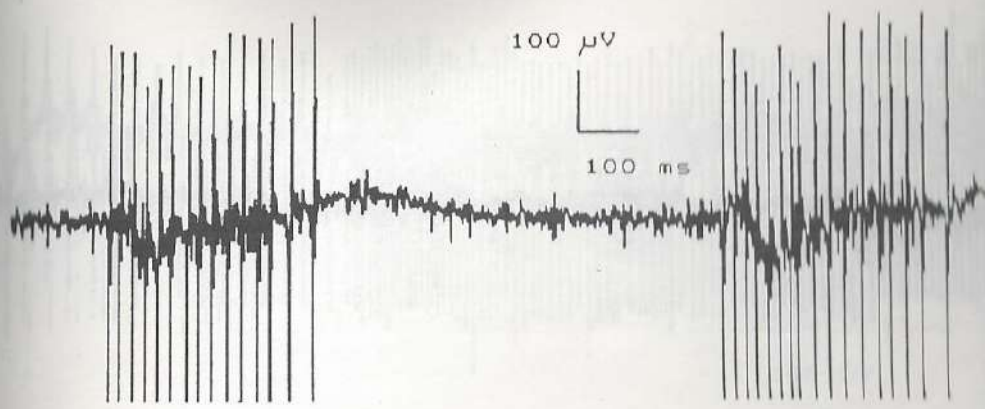
Single motor unit pattern during high pitch phonation.Frequency of firing is about 30 per second.A small microphonic effect is perceptible.

Case history:

Severe paresis of both vocal folds.Lateral amyotrophic sclerosis.

Comments:

Lack of spatial recruitment of motor units suggests a partial primary axonal failure.



Subject:

Male, aged 34.

Muscle:

Right thyroarytenoid.

Description:

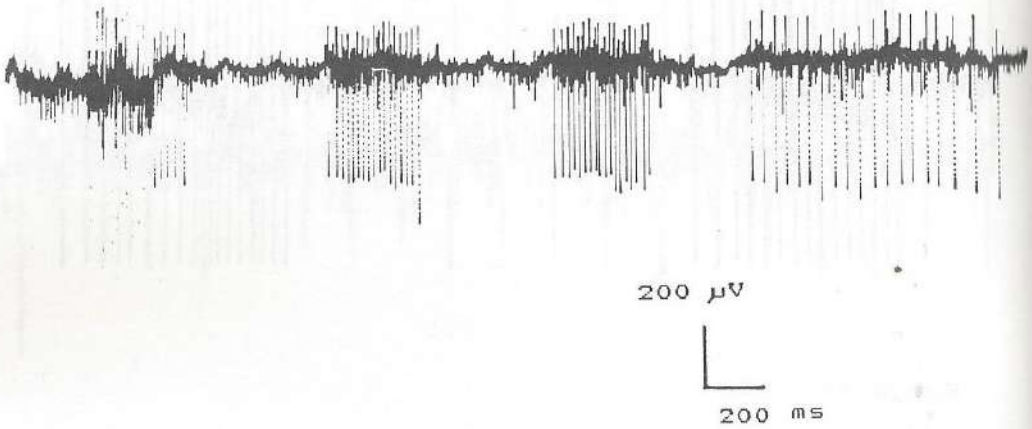
Two short high frequency discharges.

Case history:

Syringobulbia.Incompetent larynx.Paresis of both vocal folds.

Comment:

These bizarre spontaneous discharges consist of trains of potentials with a firing rate of about 50 to 70 per second.Such high frequency discharges are not specific ! They may be observed in any irritation lesion of anterior horn cells, peripheral nerves, and even in myopathies.



Subject:

Male, aged 34.

Muscle:

Left thyroarytenoid.

Description:

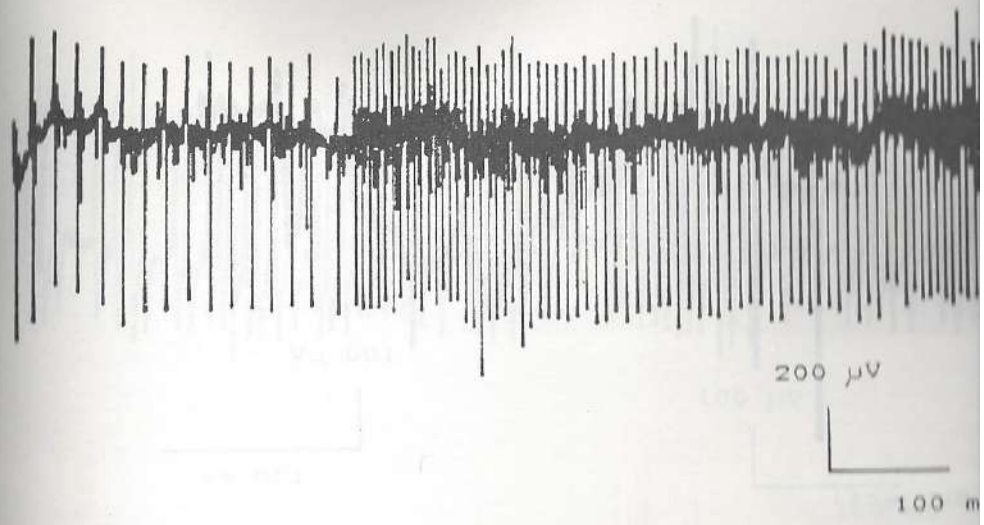
Several short high frequency discharges.

Case history:

Same case as the precedent.

Comment:

Same as the precedent.



Subject:

Female, aged 42.

Muscle:

Left thyroarytenoid.

Description:

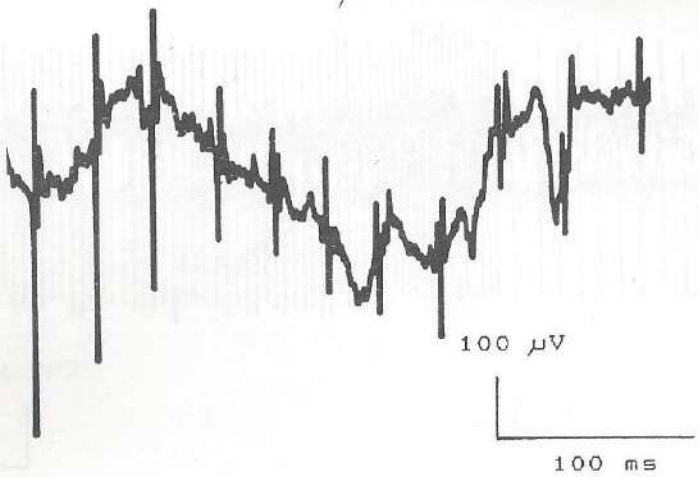
High frequency discharge, at variable rates of frequency.

Case history:

Oesophagectomy, 4 months earlier.

Comment:

Such trains of potentials are not pathognomonic of myopathy (cf. Steinert's disease), but are a distinctly abnormal phenomenon, occurring also in peripheral neuropathies. Some authors have suggested that true electromyographic myotonia is identified by a fluctuation of both amplitude and frequency, wich is less frequently observed in other cases.



Subject:

Female, aged 3.

Muscle:

Left thyroarytenoid.

Description:

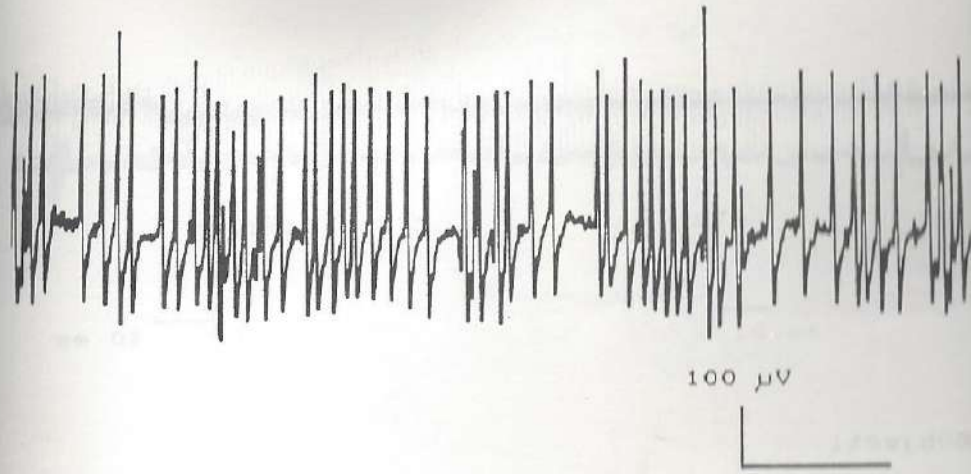
Single motor unit potentials, pulsing at high frequency, with myasthenic decrementation.

Case history:

Congenital paresis of facial,pharyngeal and laryngeal muscles.Mobius' syndrom.

Comments:

A single motor unit potential pulsing at more than 25 cycles / second, without recruitment of other motor units, suggests a partial nerve or nucleus lesion.In the present case, repetitive stimulation demonstrates a myasthenic decrementation, but the changes are not as impressive as in myasthenia gravis.



Subject:

Male, aged 48.

Muscle:

Left thyroarytenoid.

Description

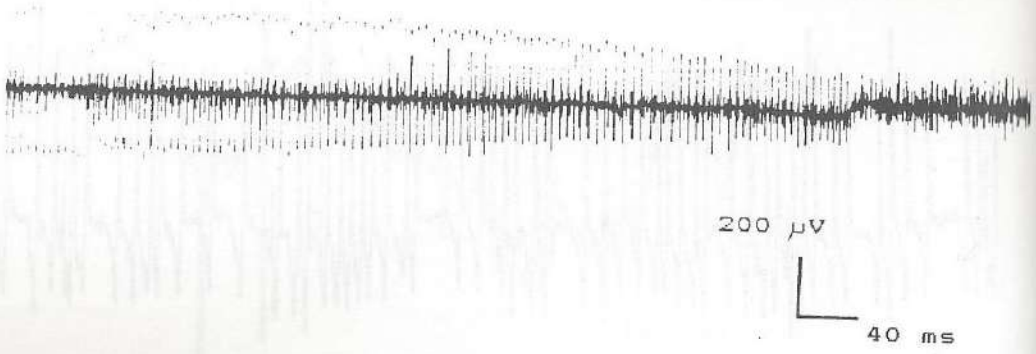
High frequency discharges, somewhat dysrhythmic, provoked by electrode insertion.

Case history

Radiotherapy on neck for Hodgkin's disease two years before.Paresis of both vocal folds.

Comments:

These high frequency discharges are to be considered as a manifestation of hyperirritability, associated here with degenerative changes.Increased insertional activity is frequently associated with neuropathic disease.



Subject:

Male, aged 56.

Muscle:

Left thyroarytenoid.

Description:

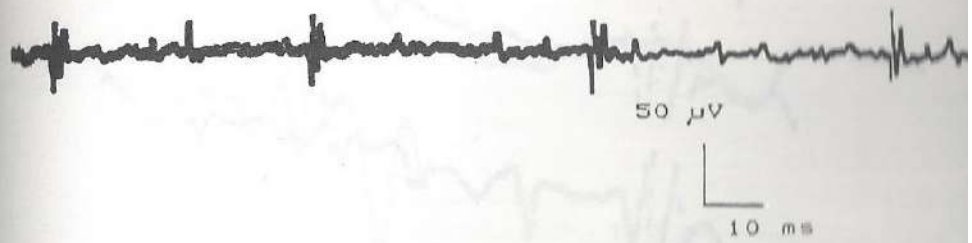
High frequency discharge,evoked by needle movement.

Case history:

Inherited myotonia dystrophica (Steinert's disease) with facial, pharyngeal and laryngeal involvement.

Comments:

In true myotonia, high frequency repetitive discharges usually show a recurrent fluctuation of frequency and amplitude.The sound is typical.A myotonic response may be evoked either by voluntary movement, or by mechanical (or electrical) stimulation.



Subject:

Male, aged 60.

Muscle:

Right thyroarytenoid.

Description:

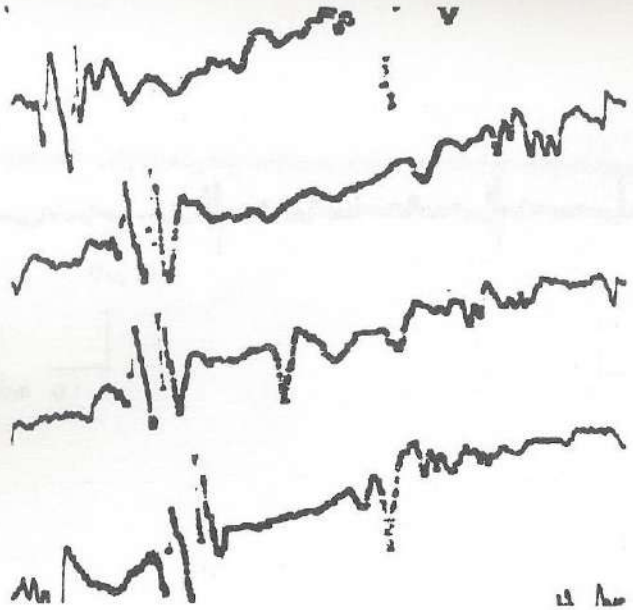
Small and short polyphasic motor unit potential, rhythmically firing during breathing.

Case history:

Right carotid artery surgery, 4 months before.Right vocal fold immobilized in paramedian position.

Comments:

This is the earliest variety of low amplitude and relatively short duration potentials, formerly called nascent units, which indicate reinnervation and precede clinical recovery.



Subject:

Female, aged 32.

Muscle:

Left cricothyroid.

Description:

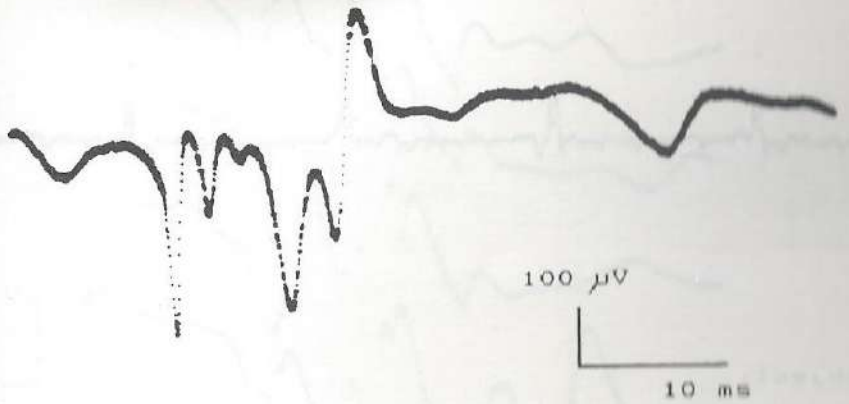
Small and rather short polyphasic potential.Nascent motor unit.

Case history:

Strumectomy 3 months before.

Comments:

These nascent motor units are here the first sign of reinnervation.Polyphasia, low amplitude and relatively short duration are characteristic, and reflect immaturity, small diameter and slow conduction velocity of reinnervating axons.



Subject:

Male, aged 63.

Muscle:

Right thyroarytenoid.

Description:

Polyphasic motor unit potential with a rather normal amplitude.

Case history:

Oesophagectomy for neoplasm, 6 months earlier.Paralysis of right vocal fold.

Comments:

Partial peripheral nerve lesion.

100 µV

40 ms

Subject:

Male, aged 51.

Muscle:

Left thyroarytenoid.

Description:

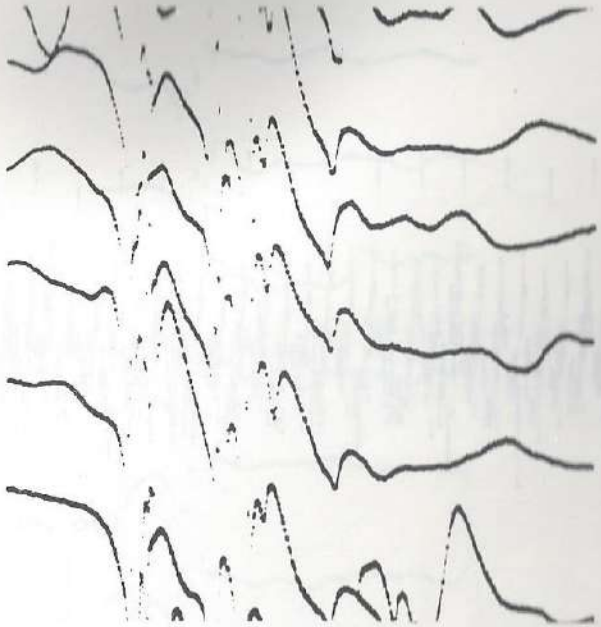
Single polyphasic potential, rhythmically firing.

Case history:

Neuritis of viral origin.Clinically paralyzed left vocal fold.Reinnervation.

Comment:

This potential has increased duration, with polyphasia, but still low amplitude.In the present case, after about six months of complete denervation, it indicates reinnervation.Nevertheless, the vocal fold will remain immobile.



100 µV

2 ms

Subject:

Female, aged 48.

Muscle:

Left thyroarytenoid.

Description:

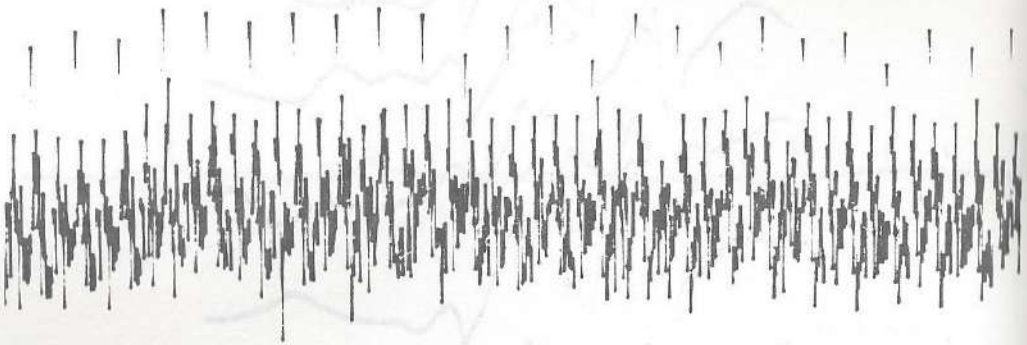
Polyphasic motor unit potential, with late components ("linked potentials"). Increased duration and amplitude.

Case history:

Strumectomy, 10 months before.Paresis of left vocal fold.Initially the vocal fold was paralyzed in paramedian position.

Comment:

In early stages of recovery of partial nerve lesions, long duration polyphasic potentials are observed (collateral reinnervation).In later stages, potentials become larger in size and shorter.Giant motor units appear.



Subject:

Female, aged 36.

Muscle:

Right thyroarytenoid.

Description:

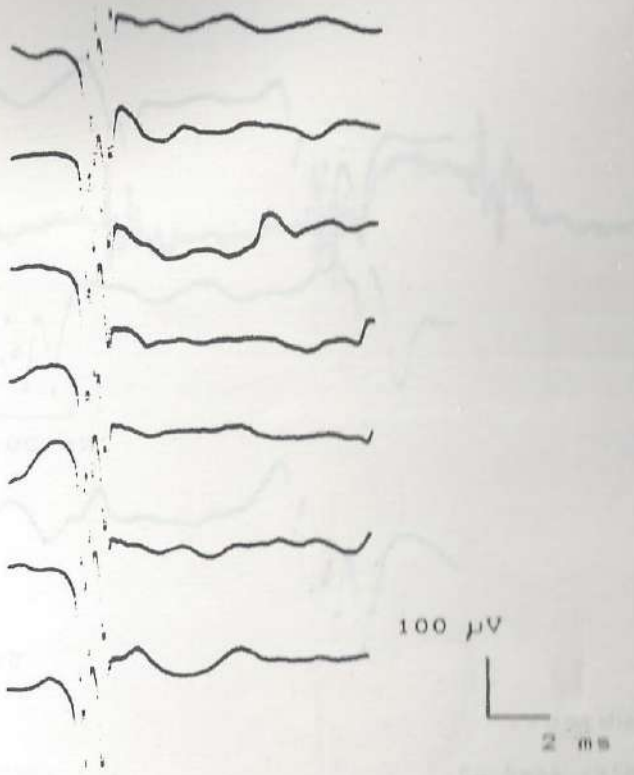
Abnormal spontaneous breathing activity.Short duration and high frequency polyphasic potentials.

Case history:

Dermatomyositis.Muscular and vocal weakness was the cardinal clinical symptom.

Comment:

In myopathy the total duration of the polyphasic unit does not exceed that of a normal motor unit, and the spike compounds are all sharp.The peak-to-peak amplitude is rather low.A full interference pattern at small or moderate effort is also characteristic of myopathy.



Subject:

Female, aged 36.

Muscle:

Left cricothyroid.

Description:

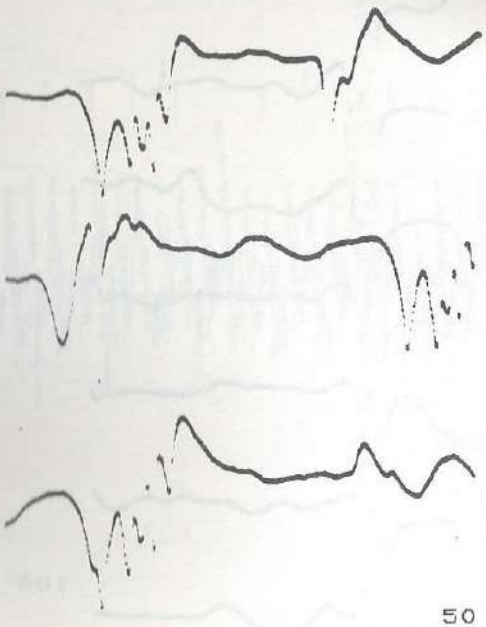
Single, short duration and low amplitude polyphasic potential.

Case history:

Dermatomyositis.Cf. supra.

Comment:

Such potentials may be considered as pathognomonic of myopathic disease.



Subject:

Male, aged 63.

Muscle:

Right cricothyroid.

Description:

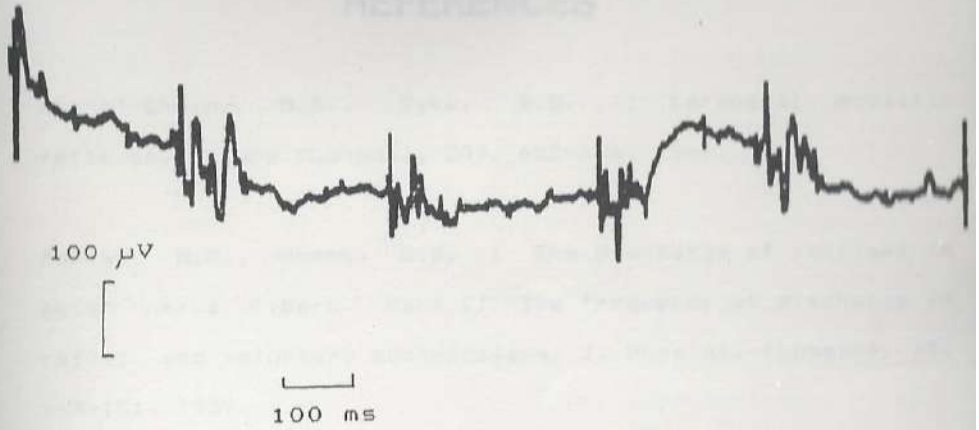
Short duration and low amplitude polyphasic potential.

Case history:

Severe polymyositis considered as a paraneoplastic syndrom of an advanced prostatic cancer.Slight paresis of both vocal folds.

Comments

With respect to the clinical context, this abnormal potential is to be considered as myopathic, but there is some analogy with a reinnervation potential.



Subject:

Female, aged 25.

Muscle:

Right thyroarytenoid.

Description:

Rhythmical, myoclonic, unvoluntary contractions of the right hemilarynx, with "nystagmus" of the vocal fold, at a frequency of about 3-4 per second.

Case history

Multiple sclerosis, with brainstem and cerebellum involvement.

Comments

Laryngeal myoclonus is very rarely observed.It is associated with extrapyramidal symptoms, and with central nervous system disorder.

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