MANAGEMENT OF VOCAL FOLDS MOBILITY DISORDERS

ESSAY

Submitted in Partial Fulfilment for
Master Degree in
PHONIATRICS

By

GHADA ALAA EL-DIN GOBARA

SUPERVISED BY

Prof. Dr. *M. NASSER KOTBY*

Professor & Head of Unit Phoniatrics
Department of Otlaryngology,
Ain Shams University

Dr. ALIA MOHAMED EL-SHOBARY

LecturerPhoniatrics
Ain Shams University

Faculty of Medicine
AIN SHAMS UNIVERSITY

(1993)

ACKNOWLEDGMENT

First thanks for God the most merciful,

I can't find enought words to express my gratitude to ***Prof. NSER KOTPY,*** *whom I consider as a father to me, I thank him not only for his continous support, but also for his kind encouragement. This work is consider a very small part of his educational system.*

Thanks for all who helped in this work especially the most Remarkable personality ***Dr. ALIA El-SHOBARY,*** *without her help and direct supervision this work was impossible to come out.*

Finally, I wouldl ike to express my deepest thanks to my father, mother, and husband for their, help and encouragement.

بسم الله الرحمن الرحيم

CONTENTS

INTRODUCTION

INTRODUCTION

Vocal fold paralysis is not a diagnosis but it is a sign caused by many etiological factors. Interference with the nerve supply to the larynx leading to vocal fold paralysis, results in varying degrees of voice alternation, change in appearance of the larynx, dysfunction of swallowing, aspiration and airway obstruction, so it is important to know about the management of vocal fold paralysis.

Dysphonia is the presenting symptom of the vocal fold paralysis patient. The pathologic conditions may include various benign vocal fold lesions or diseases affecting the neuromuscular function of the larynx.

An accurate diagnosis must be accomplished by a skilled team. Careful assessment should be done to describe and localize the pathologic condition, this is essential to treatment planning.

Vocal fold paralysis may be treated by voice therapy or phonosurgery: implants, injections, framework surgery and reinnervation, the results depend on the technique, materials surgical skill, physiological and psychological status of the patient. There is no single procedure that is clearly best for each problem.

The aim of this work :

Is to throw the light on the various techniques of management of vocal folds mobility disorders in order to guide the choice of the optimal line of treatment.

NEUROANATOMY

NEUROANATOMY

Voice production relies on the highly coordinated functions of many laryngeal and respiratory muscles. Innervation of the larynx has three different types of nerve fibers and each type has a specific function, the motor nerve fibers that cause the muscle to act, the sensory nerve fibers that receive sensation and convey those sensations to the central nervous system and the autonomic nerve fibers that regulate the laryngeal vessels and glands *(Yashida et al., 1989).*

1. The Motor Nervous System:

Darley et al. (1975) has postulated that organized motor patterns are built into a hierarchy of 6 levels within the nervous system. These levels are integrated and interrelated. The higher levels function by activating, inhibiting, or modulating the patterns of the lower levels, the lowest level is organized into some reflexes that are integrated to perform purposeful movement.

The 6 levels of the control of the motor act can be summarized as follows:

1. Lower motor neuron (bulbar): this is reflexive in nature.

2. Vestibulo-reticular level: this regulates the lower motor neuron.

3. Extrapyramidal level: this is responsible for the automatic subconscious control of activities and adjustment of muscle tone.

4. Cerebellar: this is an error detector and error corrector, it controls the accuracy of the previous four levels.

5. Conceptual programming level, which is the highest level.

Laryngeal Neuro-Muscular Hierarchy :

The laryngeal motor control system may be divided into:

A) Cortical

The efferent innervation of the larynx starts at the foot of the frontal precentral gyrus. The axons of the primary neurons run to the medullary central via the corona radiata and travel in the internal capsule at the junction of the genu and the posterior limb. The fibers then descend mainly in the corticobulbar component of the pyramidal system. These fibers project through the corticobulbar component of the pyramidal system to the bulbar nuclei for layngeal control. i.e. the nucleus ambiguus (NA) of both sides. The few large diameter fibers derived from cells in layer V of the inferior precentral gyrus relay directly onto the largyneal motorneurons, whilst the many smaller diameter fibers relay indirectly through interneurons scattered through the laryngeal motroneuron pools. Most of these cortical projection fibers cross the midline within the pons and upper medulla oblongata traversing the medial lemnisci on the way to enter the contralateral motorneuron pools, but some remain ipsilateral so that each cerebral cortex project to the laryngeal motorneurons on both sides of the brain stem where the cell bodies of the secondary neurons lie *(Kotby and Haugen, 1970)*.

Whyke and Kirchner (1976) showed that the activity of the laryngeal motorneurons located in the nucleus ambiguus is influenced by projections that reach them from the cerebral cortex and from the mesencephalic tectum and cerebellum, as well as by reflexogenic inputs entering the lower brain stem from various receptor systems located within the larynx itself and within the lungs. Many studies proved the role of the mid brain periaqueducted gray matter (PAG) in vocal control and vocalization, as *Magoun et al. in (1937)* elicited natural-sounding vocalization by electrical stimulation of the PAG in anesthetized monkeys. *Holstege (1989)* stated that the nucleus retroambiguus (NRA) may play a role in coordinating laryngeal and respiratory muscles during vocalization as there are fibers from it to NA and between it and ventrolateral PAG so it may be suggested that the dorsolateral PAG neurons project to ventrolateral PAG cells which may then project to the NA throw NRA.

The role of the cerebellum in laryngeal control has been suggested but not clearly demonstrated *(Larson, 1975)*. The exact pathway is unknown. *Larson (1978)* reported that when lesions of the cerebellum are large they caused many gross motor changes in vocalization.

B. Brain Stem :

The cell bodies of the second order neurons lie in the nucleus ambiguus in the caudal part of the brain-stem. The cranial third of the nucleus ambiguous is considered to be the somata-motor center of the pharyngeal and some of the palatal muscles. These groups of muscles are closely related together in their central connections as well as in function. The center of the cricothyroid muslce is closely related to the pharyngeal and palatal muscles, thus illustrating how the cricothyroid muslce functionally incorporates with the external laryngeal muscles. The caudal parts of the nucleus ambiguus regulate the intrinsic laryngeal muslces attached to the arytenoid cartilage. All these muscles hae been shown to act in intimate harmony *(Kotby and Haugen, 1970 a).*

The intrinsic laryngeal muscles are innervated by the neurons of the nucleus ambiguus (NA) which lies deep in the reticular formation of the medulla oblongata, the NA contribute efferent fibers to the glossopharyngeal nerve which sends fibers to the stylopharyngeus, to the vagal nerve which is distributed to the pharyngeal and esophageal striated muscles and the cricothyroid muscle (CT) and to the cranial root of the accessory nerve which supplies the intrinsic laryngeal muscles except CT *(Yashida et al., 1992).*

In the nucleus ambiguus the cells of origin of the cricothyroid muscles (CT) are lying in the rostral cell group of the nucleus while motoneurons for the inferior laryngeal nerve (ILN) are situated in the caudal cell group. ILN innervate the posterior cricoarytenoid (PCA), the interarytenoid IA, the lateral cricoarytenoid LCA, and the thryoarytenoid TA muscles, so that the laryngeal motneurons of these muscles are situated cadually in the order of, PCA motoneurons are situated in the most rostral region, the TA, LCA and IA are located more caudally *(Yoshida et al., 1992).*

C. Peripheral Nerves :

The motor axons of the nerve cells leave the nucleus ambiguus (NA) on its dorsal aspect, then curve laterally and ventrally to the medulla in the rootlets of the ipsilateal mainly vagus and cranial accessory nerves . Most of the fibres except those for the cricothyroid muscle, leave the medulla by the latter route and transfer to the vagal nerve trunk in the jugular foramen. The fibers to the cricothyroid muscle enter the vagal rootlets directly *(Du Bois and Foley, 1936).*

The vagus nerve emerges from the medulla oblongata between the olive and inferior cerebellar peduncle. It presents two enlargements: the jugular (superior) and nodose (inferior) ganglia, at the level of the jugular foramen shortly after it emerges into the neck through the foramen. These ganglia contain the first order neurons in the afferent pathways of the vagus nerve, including the larynx. The cranial accessory nerve communicates with the superior (jugular) ganglion and joins the main trunk of the vagus just distal to the inferior or nodose ganglion, its motor fibers are distributed through the pharyngeal and recurrent laryngeal branches of the vagus nerve *(Bowden, 1973; Dixon et al., 1982).*

In the neck the vagus nerve has several branches that control the voice and speech mechanism. These branches are the pharyngeal nerve, superior laryngeal nerve, and the recurrent laryngeal nerve.

Pharyngeal nerve :

The pharyngeal nerve emerges from the upper part of the inferior (nodose) ganglion of the vagus. It descends to the level of the middle pharyngeal constrictor muscle. At that point, it divides into filaments that receive branches from the sympathetic trunk and glossopharyngeal and external laryngeal nerves to form the pharyngeal plexus. The plexus provides nerve fibers to the pharynx and to all the muscles of the soft palate except the tensor palati muscle.