## **Muco-ondulatory phonation theory**

By M.

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1° The fundamental problem of laryngeal physiology, the origin of the vocal cords vibration, is always discussed. The myo-elastic theory and the neuro-chronaxic theory share the preferences of the laryngologist and the phoniatre. A dozen years studying the phonation by stroboscopy, meditating the contemplation of high-speed films and especially the need to treat and cure voice patients, suggested me the following reflections.

2° The laryngologists who support the myoelastic theory speak about the vocal muscle vibration and the proponents of the neurochronaxic theory describe the polyphasic contraction of the ary-vocal and thyro-vocal muscles. But neither of them really see the vibration or contraction of these muscles.

What they actually observe is the anatomical structures movements that cover them. If the muscle of the vocal cord vibrates or not, is something that we suppose, but that we do not observe.

3° The only way, nowadays, to have an opinion on the vocal muscle role and action is electromyography. The studies carried out with this method by Fessard and Vallancien (1957), Spoor and Van Dishoeck (1958), Faaborg-Andersen and Edfeldt, Lafon (1958), Sawashima et al. (1958), Wustrow and Wieck (1960) and Barroilhet, Frank and Holmgren (1960), find action potentials that are not isochronous with the tone emitted by the studied larynx and therefore are not, the vibration cause.

4° We all know the extraordinary laxity of the larynx submucous cell tissue, which explains the origins of the dreaded acute subglottic laryngitis of childhood, Reinke's edema, laryngeal myxopathy, brightic, toxic edema, and Quinke, etc.

5° Laryngeal pathology offers us also valuable indications for inferring conclusions applicable to its physiology.

a) When the cords mucous is dry, due to climate changes or intense heating dysphonia occurs.

## JORGE PERELLÓ – MUCO-ONDULATORY PHONATION THEORY

b) Discrete acute laryngitis produces disproportionate dysphonia. It is difficult to believe that a slight catarrhal inflammation can produce paresis of the bulky thyroarythenoid muscle and even more affect the physiology of the recurrent nerve or the motor plates of the muscle.

c) Acute or sub-acute repeated laryngitis produces dysphonia, although no residual lesions are seen in laryngoscopy.

d) In dry laryngitis, which produces a varnish on the vocal cords, the voice is instantly improved by the "in situ" application of alpha-chemo-trypsin (Perelló 1958).

e) Perelló and Comas (1959 and 1960) have demonstrated that premenstrual dysphonia is produced by a thickening of the mucosa horny layer of the vocal cord edge.

f) The stroboscopic examination shows a normal vibration of the paralyzed cord by the recurrent nerve section.

g) Patients with paralysis of the vocal cords abduction by the recurrent nerves bilateral section, have a normal voice.

h) In the myxedema a matte, hoarse and deep voice occurs; but Mann (1928) instead sees normal strings, Leider and Matzker (1955) describe the strings slightly atrophic but with good mobility. Schönhärl (1954) observes a phase deviation in their vibration. The few manifestations in the vocal cords make the authors to say that the characteristics of the myxedema voice are due to the mucous membrane infiltration of the pharyngeal and oral resonator.

i) Mongolian dysphonia is due to the laryngeal mucosa infiltration.

6° All these facts make us think that perhaps it would be appropriate to take the opposite direction as Garde (1951) indicated always from the larynx to the brain, that is to say to direct us from the vocal muscle to the mucous membrane which covers it.

Already Gutzmann in 1933 sees the importance of the laryngeal mucosa for the phonation.

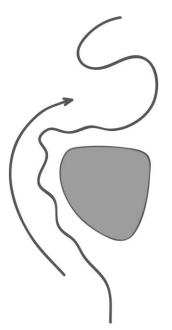
An attentive stroboscopic observation shows us what is called the vocal cords vibration as a rippling of the mucous membrane that covers them. These waves during phonation move from bottom to top and from front to back. The wave propagates from the sub-glottis, rises, crosses the cord edge, continues through the upper face and is slowly lost at the entrance of the ventricle. Before this wave disappears, we see the beginning of another in the subglottis. Sometimes we have the impression of seeing 2 waves or 2 free edges superimposed on the vocal string.

As the wave propagates upward due to the subglottis funnel shape, it comes into contact with the opposite side wave. Sometimes this contact is in one point and the rest of the rope does not come into contact. This rippling phenomenon of the mucosa is already mentioned by Merkel (1963), Smith (1957), Schönhärl (1960) and many other authors.

Farnswoorth (1938) calls it roll movement. This word gives the sensation of something heavy running, flattening the vocal cords. It is not so, in fact. Precisely, what is called vocal cord vibration, gives the impression of a passive, relaxed, flaccid manifestation which is agitated and waved by the expired airflow. It looks like a shaken rug.

This relaxation and passivity, which can be verified by stroboscopy, gives rise to the idea that it is not an active movement by muscular contraction like a snake movement or the path of a centipede. In fact, the vocal muscle does not have a segmental structure that facilitates this rolling contraction.

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It also seems that the vibration movement is favored by a suction of the subglottis walls. This can be explained by the mucous membrane laxity and the Bernouilli effect. So all the narrowest is the duct so much greater is the force suction that is exerted on these walls. (Van den Berg et al., 1957).

So here we are in front of the fact that the ocal cord vibration is nothing more than a ripple of the mucous membrane that slides on its very lax submucosa.

In a cough attack, this same wave movement is observed, and the mucosa increased to an intensity and extreme violence, as reported

by Von Leden (1960). We have yet another proof in the fact that in the sounds produced in inspiration, the wave of the vocal cords mucous surface propagates in the opposite direction, that is to say, from top to bottom.

The correct phonation requires that this mucosa be very flexible, moist and very movable on the deeper muscular planes. For a similar reason the trumpet player and the flutist wet their lips before playing their respective instruments.

Repeated laryngitis binds and scleroses the submucosal tissue. This one becomes less lax and does not allow the necessary mucosa displacement. It is for this reason that dysphonia occurs, although laryngoscopy shows nothing abnormal.

Nodules, polyps, monocordites, and submucosal hemorrhages are produced by edema or vascular rupture of this cellular tissue because of its excessive laxity and or exaggerated undulation.

The laryngeal muscles with their contraction and synergism vary the length, thickness and shape of the muscle of the vocal cord and force changes in the shape, speed, amplitude and frequency of the mucosa corrugation. This explains the differences in vocal production in tone, falsetto, yarns, pianos, etc. Intensity variations are produced by the tonic contraction of the thyro-arythenoid muscle which thins to emit the pianos, and grows to the strong ones. This tonic contraction of the vocal muscle has nothing to do with it, or at least it may not be synergistic or synchronic with the contraction of adductor muscles of the glottis. This fact explains how one can emit a high-pitched piano sound and a strong bass sound.

The muco-wave theory explains why the 2 strings can vibrate in different phase of the order of half a period. In reality, the mucosa of one cord can wobble with total independence from the other cord. Thus the mucosa undulation does not need to begin the phonation that the vocal cords are in adduction. The dysphonia in hyperkinesias is due to the exaggerated adduction that prevents the formation of waves in the mucosa. This theory can also explain the caisson dysphonia, an experiment carried out by Airon and Klein (1961).

It may be objected that it is very difficult to produce the ripple at the exact speed of the desired tone. We believe that this can be achieved with practice. What would serve the years of study of a singer? Does not the trumpeter attack exactly the desired tone with the vibration of his lips?

From all this, it seems to us that a more exact interpretation of reality will depend on a deeper study and experiments to prove that what is called vibration of the vocal cords is a rippling of this mucosa.

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