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The Role of Electromyography in the Diagnosis of Velopharyngeal Insufficiency: Our Experiences

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The subject of this study is the electromyographic investigation of the velopharyngeal sphincter structures. Seventy-five patients underwent examination, both patients with symptoms of velopharyngeal insufficiency and patients who were thought to have latent pathological sphincter changes based on local findings. A control group of 10 healthy examinees was also investigated. On the basis of electromyographic findings we divided patients into 2 groups: 57 patients without neuromuscular disorders and 18 patients with neuromuscular disorders of the velopharyngeal sphincter. Twelve patients from the latter group had acute, and 6 had chronic lesions of the velopharyngeal sphincter. Particular cases of illness within these 2 groups were investigated further. This study shows the usefulness of electromyography for diagnosing the exact causes of velopharyngeal insufficiency and for choosing the best approach to treatment.

Key words: electromyography, velopharyngeal sphincter, velopharyngeal insufficiency, soft palate, hypernasality

The velopharyngeal sphincter (VPS) is formed by the structures of the soft palate and the corresponding parts of the lateral and posterior pharyngeal wall. When necessary, the communications between the nose and pharynx, or the nose and lower airway are reduced or closed by sphincter contraction. Thus, food is prevented from entering the nose, and airflow from the pharynx into the nose is regulated. Normal anatomical structure and adequate functioning of the associated musculature (1) are necessary for the VPS to function correctly.

Velopharyngeal sphincter insufficiency (VPI) and its most significant symptom hypernasality, or even regurgitation of food can be the result of anatomic anomalies of the sphincter structures, neuromuscular disturbances of the sphincter muscles, or a combination of these causes.

A number of investigative methods are used in the diagnosis of patients with VPI. Clinical and radiological methods as well as endoscopy are most often used (2-11).

The clinical features of VPI caused by anatomical abnormalities and VPI caused by neuromuscular disorders are very similar, sometimes even identical. It is not possible to differentiate between the causes of the insufficiency using the diagnostic methods mentioned above. These methods only reveal the morphological changes in the sphincter structure during activity or at rest (12-18).

The aim of this study is to produce criteria whereby the physician can differentiate between the different etiologies of VPI by examining the electromyographic features of the VPS. In doing so, we hope to demonstrate the usefulness of electromyography (EMG) for the diagnosis of VPI.

Materials and Methods

An Amplaid EMG 15 electromyograph (Milan, Italy) and concentric needle electrodes were used for the EMG of the VPS muscles. The EMG data were recorded and stored in an IBM personal computer.

During the last 12 years, EMG of the VPS was performed on 75 of our patients, aged 1 month to 15 years. These patients included patients with symptoms of VPI (hypernasality or regurgitation of food through the nose), and patients who based on local clinical findings were thought to have latent pathological changes of the VPS (shorter, less movable palate). As a control, we

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also performed EMG of the VPS on 10 normal individuals, aged 7 to 19 years. The results of both groups were compared.

Epimucosal anaesthesia was used in most patients (17). Electromyographic activities of the sphincter muscle were registered systematically. Electrodes were first applied to several points of the soft palate. Particular attention was paid to the examination of levator dimple area (dimple forming on the soft palate with the contraction of m. levator), the posterior half of the palate, and the area along the medial line of the palate. The depth of the electrode puncture was up to 10 mm. Furthermore, the activities of the posterior palatopharyngeal arc, lateral and posterior wall of the pharynx at the level of the palate were examined with 2-3 punctures (at a depth of 2-3 mm).

Electromyographic activities were recorded while the examinee was pronouncing the sounds "a" and "e", and in small children while they were crying.

The following electromyographic features of the VPS were analyzed: Pattern during relaxation; pattern during voluntary movement (vocalization); analysis of motor unit action potentials; and mapping of activity (searching for an area of no apparent electrical activity, known as a "silent zone"). In analysis of motor unit action potentials we analyzed the following: Amplitude (range); duration (range); discharge frequencies of individual units; and percentage of polyphasic potentials (potential in which the base line is crossed more than 4 times).

Results

By examining the different aspects of the electromyographs of the VPS mentioned in the Materials and Methods section, we arrived at criteria for distinguishing between the normal VPS, VPS with acute lesions, and VPS with chronic lesions (Table 1). The electric potential parameters of the posterior pharyngeal wall, soft palate and posterior tonsillar arc were calculated separately.

The normal VPS had the following electromyographic features: During relaxation, there were no signs of electrical activity; during vocalization, there were mixed or interference-pattern electrical potentials; amplitude range of motor unit action potentials was within 150-900 μV, and their duration range was 3-5 ms; the percentage of polyphasic potentials was less than 12 % (Fig. 1). The electrical potential characteristics of the posterior pharyngeal wall and posterior tonsillar arc corresponded to those of the soft palate. Only the amplitude of the motor unit action potentials was much lower (range 50-150 μV).

Acute neurogenic lesions of the soft palate had the following characteristics: Fibrillations and positive sharp waves during relaxation; discrete activity or reduced interference pattern during vocalization; amplitude range of motor unit action potentials was within 100-350 μV, and their duration range was within 3-5 ms; the percentage of polyphasic potentials was more than 12 % (Fig. 2).

Soft palate chronic neurogenic lesions had the following characteristics: Fibrillation and positive sharp waves during relaxation; reduced interference pattern and increased discharge frequencies of individual units during vocalization; amplitude range of motor unit action potentials was within 100-2,000 μV, and their duration range was within 4-7 ms; the percentage of polyphasic potentials was more than 12 % (Fig. 3). We recorded no pathological neuromuscular activity of the posterior pharyngeal wall and posterior tonsillar arc during our investigation.

Table 1 Electromyography criteria for differetiating the normal velopharyngeal sphincter (VPS), VPS with acute lesions and VPS with chronic lesions

	Normal VPS	Acute lesion	Chronic lesion
Pattern during relaxation	No activity	Fibrillation and positive sharp waves	Fibrillation and positive sharp waves
Pattern during voluntary activation (vocalization)	Mixed pattern or interference pattern	Discrete activity or reduced interference pattern	Reduced pattern, increased discharge of individual units
Motor unit action potentials			
Amplitude range	150-900 μV	100-350 μV	100-2,000 μV
Duration range	3-5 ms	3-5 ms	4-7 ms
Percentage of polyphasic potentials (> 4 phases)	< 12	> 12	> 12

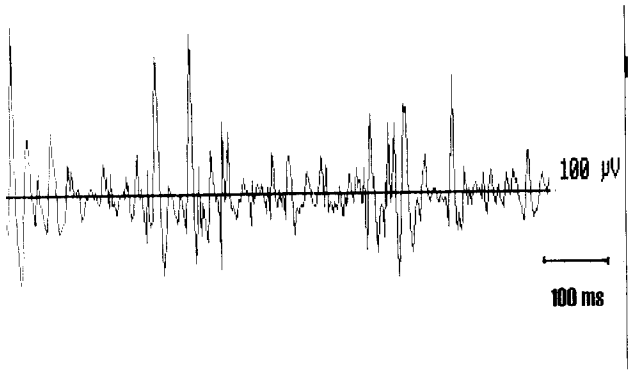

Fig. 1 Normal electromyographic findings of the soft palate (patient of the control group).

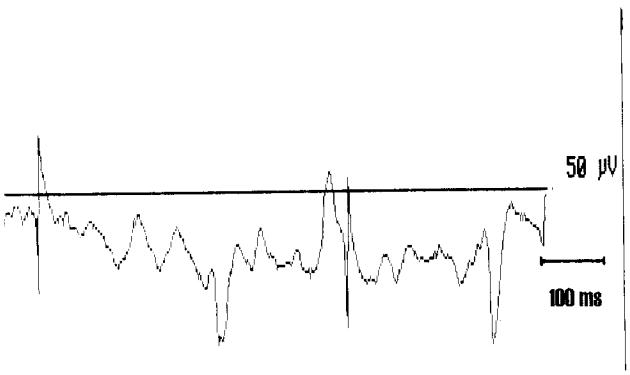

Fig. 2 Acute neurogenic lesion of the soft palate (idiopathic paresis of half of the soft palate).

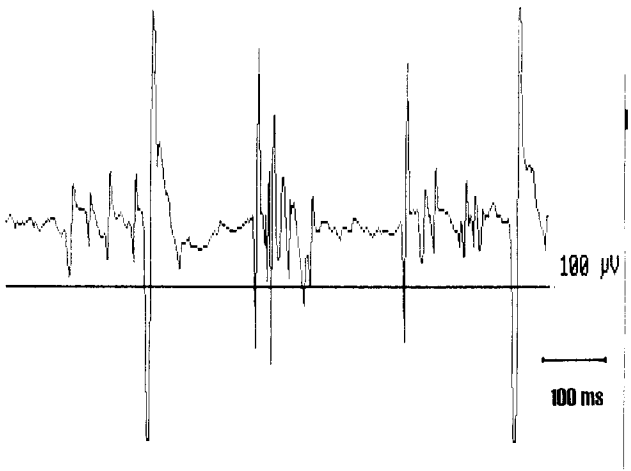

Fig. 3 Chronic neurogenic lesion of the soft palate (permanent soft palate paresis following tonsillectomy).

On the basis of electromyographic findings, the patients were divided into 2 basic groups as follows:

Group 1: Patients without neuromuscular disorders. Fifty-seven patients were included in this group, none of whom were shown to have neurogenic lesions of the sphincter by EMG.

By further examination and systematic insertion of electrodes into particular sphincter structures, we noted areas without electromyographic activity, so-called "silent zones" without muscular structures, in 17 patients. Four types of "silent zones" can be distinguished according to their location, each of which corresponds to a specific pathological condition.

Eleven patients had triangular "silent zones" in the middle of their soft palates (submucosal or occult submucosal cleft palate) (18-20). Three patients had "silent zones" in the posterior third of their soft palates (short *i.e.* hypoplastic palate) (2). Two patients had "silent zones" in irregularly located areas of the soft palate (associated with scar tissue) which developed after surgery on the palate (22). And in 1 of our patients, we used EMG to reveal the lack of muscle in the palatopharyngeal arc (aplasia of the palatopharyngeal muscle) (23). Using EMG, we could not prove with certainty anatomic anomalies of VPS in other patients. In our opinion, these are marginal cases or patients in which hypernasality belongs to the phoniatric domain.

Group 2: Patients with neuromuscular disorders. The second group which we would like to discuss in detail contained 18 patients with neurogenic lesions of the soft palate; 12 with acute and 6 with chronic lesions (Table 2).

Acute neurogenic lesions were found in 6 patients with hypernasal speech following tonsillectomy. EMG was performed 8 days post surgery. Lesions were bilateral in 2 patients, and unilateral in 4. After 3 weeks, they all showed normalization of speech and normal EMG findings.

Acute neurogenic lesions were found also in 4 patients with so-called "idiopathic transitory paresis" of the palate. Three patients had unilateral lesions, while 1 patient had a symmetrical lesion. All 3 patients with unilateral paresis completely recovered. Three months from the onset of the first symptoms in the patient with symmetrical palatal paresis we registered signs of a chronic neurogenic lesion.

In 2 of our patients with acute neurogenic lesion of half of the palate, these lesions were only the first symptoms of a bulbar brain tumor (the EMG findings were

Table 2 Causes of neurogenic lesions of velopharyngeal sphincter in our patients

	Number of patients
Acute neurogenic lesions	12
Idiopathic paresis	4
Paresis following tonsillectomy	6
Paresis due to bulbar brain tumor	2
Chronic neurogenic lesions	6
Idiopathic paresis	2
Paresis following tonsillectomy	1
Paresis following palatoplastics	3
Total number of patients	18

consistent with those of an acute lesion of the soft palate).

A chronic neurogenic lesion was found in 1 patient with hypernasality following tonsillectomy (EMG was performed 9 years after surgery). Identical findings were noted in 3 patients with hypernasality after palatoplastic surgery. The lesions were found in different areas of the palate.

Two patients showed chronic neurogenic lesions of unknown cause. One had a unilateral and the other a symmetrical lesion.

Three infants formed a special group, in which regurgitation through the nose accompanied by swallowing disorders was the only symptom. Using EMG, signs showing either anatomic anomalies or neurogenic lesions of the VPS were not found. These findings are believed to indicate supranuclear disorders of the central nervous system.

Discussion

VPI has two main causes: Anatomical abnormalities of the VPS (with normal muscular functioning) and neuromuscular disorders of the VPS. The clinical features of both of these types of VPI may be very similar or even identical in many cases.

In the present study, we performed EMG on a significant number of patients with VPS dysfunction. By examining the EMG features of these patients, we arrived at criteria for distinguishing between the normal VPS, VPS with acute lesions and VPS with chronic lesions. Earlier studies on this subject were performed on smaller groups of patients. These studies describe the normal EMG features of specific structures of the VPS and some anatomical abnormalities (13–15, 17, 22, 23).

By recognizing the different EMG characteristics of VPI induced by neuromuscular abnormalities and VPI induced by anatomical abnormalities, the physician will be able to choose the optimum treatment in each case.

Acute neurogenic lesions of either half or all of the palate require further neurological analysis to determine whether the condition is idiopathic paresis or a symptom of another neurological disorder.

By EMG analysis of acute or chronic neurogenic lesions which developed after tonsillectomy in our patients, we could eliminate abnormalities of the sphincter structure as a cause of post-operative hypernasality. By EMG analysis of chronic neurogenic lesions which developed after plastic surgery in our patients, we could eliminate inadequate surgical techniques as a cause of VPI. We could also determine whether neuromuscular disorder was reversible or irreversible.

Furthermore, by performing EMG on cases with chronic neurogenic lesions, we could differentiate real paresis from pseudoparesis of the palate caused by occult anomalies. Lastly, using EMG, we were able to differentiate paresis of half the palate from congenital asymmetry of the palate.

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