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„Mechanical model"of the pressure (compression) on the facial nerve as a hypothesis for the Bell's palsy– CASE STUDY

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INTRODUCTION

Facial Nerve is a mixed nerve with predominance of motor fibers supplying the mimic muscles of the face. Sensory (taste) fibres innervate the anterior 2/3 of the mucous membrane of the tongue, and the parasympathetic (secreting) fibres supply lacrimal gland, submandibular and sublingual gland as well as small glands in nasal cavity, soft palate and oral cavity (Bochenek A. 1989**,** Gołąb B. 1998, Mazur R. 1989).

Complete etiology of the peripheral facial palsy is a challenging enterprise, spurs controversy and remains so far an unsolved puzzle, thus it is commonly referred to as "idiopathic palsy". The most plausible cause to date seems to be a viral infection, triggered by Herpes Simplex Virus (HSV), which provokes the oedema of the nerve in the bony canal (Sportswood L, Spruance MD. 1994, Atolini N. Jr. et al. 2009, Patrik M. et al. 2001). Trauma within the splanchocranium and post-surgery complications are believed to be another potential causes. It is also plausible that nerve palsy is caused by the disturbance of blood supply, bacterial infection or tumour development. Therefore, an early, oral steroid treatment is the most frequent one (Patrik M. et al. 2001). Bell's idiopathic palsy is the most common peripheral facial palsy and amounts to 50% - 70% of the total number of paralyses (Janczewski G. 2007, Sportswood L, Spruance MD.1994), nonetheless, arriving at diagnosis of the Bell's palsy results from precluding other possible options, which is best confirmed by 6-month observation (Atolini N. Jr. et al. 2009, Janczewski G. 2007, Patrik M. et al. 2001).

Trauma or other pathology concerning the facial nerve may cause damage of axon or its myelin sheath and is measured in grades. Sunderland Classification (Kaye A. 1991, Janczewski G. 2007) is an universally adopted 5-grade scale which takes into account such criteria as clinical course or anatomical morphology of the damaged nerve. As far as the first grade is concerned (*neuropraxia)*, function recovery may be anticipated in 3 weeks. Second grade concerns disability of the feeding function of the nerve, as well as axon damage (*aksonotmesis)*. If it is a short-term process, return to complete or near-complete facial nerve functioning may be expected, although recuperation lasts

longer – from 3 weeks to 3 months. First and second grade can be observed in Bell's palsy, and in 70%-80% of the cases symptoms automatically subside (Janczewski G. 2007, Kryst L.1997, Patrik M. et al. 2001). Third grade equals *neurotmesis* and only partial nerve functioning may be restored. Grades four and five entail complete or partial loss of continuity, and there is no prognosis for regeneration, unless surgery is involved at an early stage.

Bell's illness is recorded among 20 - 40 patients out of 100 000 people per year. Children are less troubled by the Bell's palsy, it usually affects people in their twenties. This phenomenon can be observed on the same scale among males and females, although pregnant women are exposed to it three times as often. In 10 % of the cases, a detailed medical history reveals that illness occurred earlier within the family. (Janczewski G.2007, Kelly D. Sweeney M.D 1996, Patrik M. et al. 2001). Facial nerve impairment may have central or peripheral background. As far as central, e.g. cerebral paralysis is concerned, upper branch of the facial nerve, fed by both brain hemispheres, remains intact. Central paresis involves usually hemiplegia with various levels of intensity. Cerebral paresis may be linked to every intracerebral process damaging motor tracts or motor cortical areas. Peripheral paresis occurs when the nerve itself is damaged.

METHOD – CASE STUDY

During past four years, clinical picture of 6 patients who were diagnosed with idiopathic palsy of the facial nerve - I grade (*neuropraxia* - acc. to Suderland Classification) and V^0 grade (acc. to House Brackmann Scale - HBS) [Tab.1] – improved rapidly to III^0 HBS. In case of two patients, this improvement took several to dozen hours, for the other six – 2-3 days. The subsequent 7-10 days resulted in a complete recovery of the normal functioning of the face muscles (I^0 HBS). All patients were subjected to detailed general medical examination, including ENT consultation. Examination did not reveal inflammatory process resulting from viruses, bacteria, traumata or tumours. Physiotherapeutic treatment was introduced in all cases within 48 hours from the moment first symptoms developed.

Physiotherapeutic examination in the preliminary stage went according to a specifically prepared logarithm [Tab.2], with purpose of determining level of the facial nerve damage basing on the symptoms (Piercy J. 2005, Bernard L.M. 2005).

Cases under scrutiny revealed losses in the motor part of the facial nerve (group II.1. a-l) below the stylomastoid foramen. In the next stage of the examination, estimation of the face mimic muscles fitness followed and was given grade V^0 in House Brackmann Scale (Lewis B. Adour K.K. 1995, Mamikoglu B, et al. 2003, Rickemanann J. et al. 1997).

Grade	Description	Characteristics
I	Normal	Normal function of all facial muscles
II	Slight paresis	Slight paresis on close inspection; normal symmetry and tone at rest; Complete closure of the eyelid with minimum effort; mouth: slight asymmetry
III	Moderate paresis	Visible asymmetry during movement; synkinesis is moderately noticeable; normal symmetry and tone at rest; forehead: muscles function impairment; eye: complete closure with effort; mouth: slight weakness during movement
IV	Moderately severe paresis	Obvious asymmetry during movement; normal symmetry and tone at rest; forehead: no movement; eye: incomplete eye closure; mouth: asymmetrical with maximum effort
V	Severe paresis	Only slight, barely noticeable, movement, asymmetrical facial appearance at rest; forehead: no movement; eye: incomplete eye closure; slight movement
VI	Total paralysis	No movements and tone at rest of mimic muscles

Table 1. Facial nerve damage scale acc. to House Brackmann (HBS). Janczewski G. 2007

No.	Damage type	Disorder description	Yes	No
I.	Cerebral damage	a. Possible wrinkling of the forehead on		

		the paralysed side with simultaneous slack of other facial muscles.	Y	N
		b. Various intensity of hemiplegia	Y	N
	Peripheral damage			
II.	1. below the stylomastoid foramen	a. Face asymmetry resulting from smoothing of the nasopharyngeal fold	Y	N
		b. Drooping of the corner of the mouth and eye	Y	N
		c. Bulging of the eye	Y	N
		d. eyeball rotation upwards on attempt of the eyelid closure (Bell Symptom)	Y	N
		e. no conjunctiva reflex, no possibility of eyelid closure	Y	N
		f. No forehead wrinkling	Y	N
		g. No grinning	Y	N
		h. No whistling	Y	N
		i. No wings of the nostril movement while breathing	Y	N
		j. Speech disturbance, unclear words	Y	N
		k. Food stays excessively long in the paralysed part		
		l. Drinking disturbance, spilling the liquids		
	2. branching of the chorda tympani	Apart from the symptoms described above		
		a. loss or impairment of the taste in anterior 2/3 of the tongue (sweet and salty)	Y	N
		b. disturbance in secretion of saliva by submandibular and sublingual gland (feeling of dryness in the throat)	Y	N
	3. branching of the nerve to the stapedius	Apart from the symptoms described above		
		a. Excessive sensitivity to sound, especially low tones	Y	N
	4. on the level of the greater petrosal nerve	Apart from the symptoms described above		
		a. Disturbance in the reception of smell sensations	Y	N

Table 2. Evaluation of the facial nerve damage basing on the displayed symptoms

As medical history shows, in 5 out of 6 cases, 24 hours before the symptoms developed, intense headaches radiating from suboccipital area through to the eye on the paralysed side appeared and lasted several days. First symptoms developed just after the awakening and grew more intense throughout the day (4 cases). In two other cases, first paresis symptoms developed during the daily professional activities.

Manual examination revealed that all patients have structural disorders in short suboccipital muscles on the paralysed side (change in consistency and pain when pressured - reproducing the pain symptoms as described in medical history). In three cases, limited mobility of the first cervical vertebra in the lateral glide in direction of paralysed side was noted.

Physiotherapeutic treatment in all described cases was case oriented. During the first appointment, however, paralysed soft tissue was treated, and pulsatory mobilization of the $C_0 - C_1$ segment was introduced. The mobilization had the following structure: 10 sets of 20 impulses, with ca. 30 seconds break between the sets.During the next appointments, restoring of, and, in the later stage, facial mimic muscles activity training was introduced.

ANALYSIS-CONCLUSIONS

Very rapid change in the medical picture, observed directly after the manual techniques were implemented (no other treatment, including steroid oral treatment, was involved – patients did not have time to buy medicines despite them being prescribed), allows to construct a hypothesis that one of the reasons of the facial nerve palsy may be a mechanical pressure (compression) of the nerve below the styromastoid foramen caused by the continuous pathology within the area of suboccipital muscles or/and limited mobility of the first cervical vertebra. (In this case it is not possible to asses whether the reduced mobility influenced the increased muscle tone or just the opposite – that is, increased muscle tone reduced the mobility in $C_0 - C_1$ segment). It can be presumed that, after two conditions are met: precluding all the known

pathologies (HSV, bacterial infections, trauma, tumours etc.) and revealing losses only in the motor part of the facial nerve (group II.1. a-l) below the styromastoid foramen, assuming the model of mechanical pressure on the facial nerve may, even if in a small way, provide explanation for 50 -70% of the unidentified causes of the facial nerve palsy. At present, it is not possible to defend this thesis owing to the lack of the objective measuring instruments. Choice of the studied group is another major problem. Although the incidence rate is high (20-40 cases per each 100 000), only few patients in the early stage (up to 48 hours) find their way to physiotherapists, since, as it was mentioned, oral steroid treatment is the preferential one (Patrik M. et al. 2001). Regardless of the paresis cause, nerve ischemia lasting several weeks provokes its permanent damage amounting to III^0, IV^0 or V^0 in Sunderland Classification, moreover, positive effects of the therapy, and, as such, the evidence for the mechanical model hypothesis, may be observed only on the early stage (several days) when the paralysis reaches I^0 or II^0 in the Sunderland Classification.

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