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Measurement of the depth of facial nerve at the level of stylomastoid foramen using MR imaging in Bell's palsy

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ABSTRACT

Objectives: To investigate whether the depth of the facial nerve from the skin surface as it exits the stylomastoid foramen differs between the paralyzed and unaffected sides in patients with Bell's palsy.
Methods: Forty-three patients (23 females, 20 males; mean age 43.8 ± 15.2 years) diagnosed with Bell's palsy between January 2014 and June 2017 were retrospectively reviewed and those who had a cranial MR imaging performed within 10 days upon admission to hospital were included in the study. The axial postcontrast CUBE sequence was utilized for the measurement of the facial nerve depth. Age, gender, and body mass index (BMI) as well as concomitant chronic diseases, were also noted. The severity of facial paralysis was graded using the House-Brackmann (HB) scoring system.
Results: The facial nerve depth was significantly lower on the paralytic side compared to the unaffected side (32.9 ± 5.4 mm vs. 36.9 ± 5.1 mm, respectively; $p = 0.007$). The facial nerve depth on the paralytic side was not statistically different in the female patients compared to male patients (31.2 ± 4.6 mm vs. 34.7 ± 5.7 mm, respectively; $p = 0.270$). The facial nerve depth on the paralytic side was not correlated with patients' age ($r = 0.288$; $p = 0.999$), BMI ($r = 0.215$, $p = 0.999$), and HB scores ($r = 0.031$; $p = 0.999$).
Conclusion: In our study cohort of patients with Bell's palsy, the facial nerve in the paralytic side is located more superficially as it exits the stylomastoid foramen when compared to the contralateral side. Therefore, the depth of the facial nerve may potentially play a key role in the etiology of Bell's palsy, which should be further evaluated.

1. Introduction

Bell's palsy is an acute-onset peripheral facial neuropathy characterized by lower motor neuron palsy; it constitutes the most common cause of facial paralysis [1]. The incidence varies among populations and ranges from 11.5 to 40.2 per 100,000 people. It equally affects men and women in middle and older age but is less common under 15 and over 60 years of age [2,3]. Clinical manifestations include rapid onset of unilateral facial weakness, lower motor neuron-type palsy, postauricular pain, impaired taste sensation, subjective change of sensory perception, and hyperacusis [1].

The etiology of Bell's palsy has not yet been clarified, and multiple factors have been implicated. Current literature advocates genetics, ischemic neuropathy, impaired microcirculation in the facial nerve vasa nervorum, metabolic impairment, autoimmune diseases, viral infection, and cold air [4–7]. The viral infection hypothesis focuses on HSV-1.

The patient contracts a primary infection with neurotropic herpes simplex virus in early childhood. The infection is thought to travel from oral or pharynx mucous membranes to ganglion cells through the afferent nerve. It remains an asymptomatic latent infection. But later on the infection may recur because of reactivating factors [2]. Studies conducted in the following years have supported the viral theory by demonstrating the presence of HSV-1 DNA in endoneurium fluid from the facial nerves of Bell's paralytic patients and in nerve biopsies [8]. In addition, the association of primary infection during facial decompression in animal models and facial paralysis by immunomodulation supported this theory [9,10]. On the other hand, various recent studies have failed to provide sufficient data on the etiological association between HSV-1 and Bell's palsy [11–14].

Other theories suggest a causal etiology of the cold-weather effect, which is well known but poorly explained [2,15]. It has been known that some clinical parameters such as hormones, blood pressure,

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platelet physiology, caloric metabolism, and weight can be affected by seasonal variations [14]. Stroke, myocardial infarction, and retinal vein occlusion are triggered by cold weather and the winter season [16–18]. Exposure to cold could aggravate atherosclerosis and acute thrombotic events by affecting the viscosity and coagulation of blood [18]. A possible effect of temperature on deeper segments of the facial nerve has been suggested for the vascular-ischemic and infectious etiology of Bell's palsy [19] Accordingly, the skin temperature may increase or decrease depending on the change of ambient temperature, whereas the internal temperature of deep tissues of the body remains largely constant between 36 °C and 37.2 °C as long as there is no pathological condition that causes fever [19]. Therefore, as the depth decreases, temperature changes in the more superficial tissues become more likely, and a more superficially located facial nerve would thus be more prone to temperature changes.

The facial nerve has courses in the anterior, inferior, and lateral directions after exiting the stylomastoid foramen. In newborns and children up to two years old, it is located just below the subcutaneous tissue when it exits the skull. After two years of age, it takes a deeper course when the mastoid tip and the tympanic ring is formed. In adults, it may be located as deep as 5 cm from the skin surface [1].

In this study, we hypothesized that the facial nerve, in relation to the skin surface as it exits the stylomastoid foramen, has a more superficial course in the paralytic side than the healthy side in patients with Bell's palsy. Therefore, we aimed to investigate whether the depth of the facial nerve from the skin surface as it exits the stylomastoid foramen differs between the paralyzed and unaffected sides in patients with Bell's palsy. Additionally, the effects of patients' age, gender, body mass index (BMI), and severity of paralysis on the depth of the facial nerve were evaluated.

2. Material and methods

Clinical and demographic data of the patients with Bell's palsy who were admitted to the Haydarpaşa Numune Training and Research Hospital between January 2014 and June 2017 were retrospectively reviewed. Patients diagnosed with Bell's palsy and, who had a cranial magnetic resonance (MR) imaging performed within ten days upon admission to hospital were included in the study. Patients younger than 18 years, patients with abnormal ear anatomy, patients with a history of ear cosmetic surgery and patients with bilateral and/or recurrent Bell's palsy were excluded.

Eighty-five patients met the study criteria. Cranial MR images of these patients from the hospital's picture archiving and communication system was examined to determine the course of the facial nerve and to see if it could be located at its exit from the stylomastoid foramina on both sides. This determination was only possible in 43 of these patients, who constituted our final study cohort.

Age, gender, height, weight, and BMI of patients at initial admission, as well as concomitant chronic diseases, were also noted. The severity of facial paralysis was graded using the House-Brackmann (HB) scoring system, which uses a grading from one to six ranging from normal function to total paralysis, respectively [20].

The study was approved by the Institutional Ethics Committee of Haydarpaşa Numune Training and Education Hospital (date 22.05.2017, no HNEAH-KAEK2017/KK/65) and conducted in accordance with the latest version of the Helsinki Declaration. The requirement for an informed consent form was waived by the Ethics Committee due to the retrospective design of the study.

2.1. MR imaging protocol

MR examinations were performed using a 1.5 T General Electric optima 450w MR unit (GE Healthcare; Milwaukee, Wisconsin, USA) using an 8-channel phase array head coil. MRI imaging for evaluation of the facial nerve included an internal acoustic canal (IAC) protocol with the following sequences: a noncontrast, axial, 5-mm, whole-brain T2-weighted sequence; an axial, 3-mm, noncontrast T1 sequence of IAC angled perpendicular to the dorsal aspect of the brainstem; and an axial, contrast-enhanced, non-fat-saturated, 3D, fast-spin echo T2-FLAIR (CUBE) sequence from the occipital bone to superior petrous ridge (Table 1). Twenty milliliters of intravenous gadolinium chelate (Dotarem, Guerbet; Paris, France) was administered before the exam with a delay of 30 s.

Table 1
MR protocol

	CUBE (axial)	T1W (axial)	T2W (axial)
FOV	28.8	24	24
ST	1.4	5	5
TR	5500	460	5300
TE	90	10	104
ETL	150	2	30
Resolution	288 × 256	218 × 192	288 × 192
NEX	1	1	1.5
BW	62.5	31.25	53

CUBE: 3D T2-FLAIR sequence; T1W: T1 weighted; T2W: T2 weighted; FOV: field of view (cm); ST: slice thickness (mm); TR: repetition time (ms); TE: Echo time (ms); ETL: Echo train length (ms); NEX: number of excitations; BW: bandwidth (kHz).

2.2. Measurement of the facial nerve depth

The axial postcontrast CUBE sequence was utilized for the measurement of the facial nerve depth. A straight line was drawn between the nasion and the external occipital protuberance. Another anteroposterior straight line (A) was drawn parallel to this line, which passed through the outermost skin surface of the preauricular region on both sides. The facial nerve was traced and located at the point of its exit from the stylomastoid foramen. A third line (B) passing through this point was drawn at 90 degrees to line A, and the distance of facial nerve to the outer skin was measured in mm on line B (Fig. 1). A radiologist (L.S.) with more than ten years of experience in facial nerve imaging and who was unaware of the patient's clinical findings performed all measurements.

2.3. Statistical analysis

The NCSS (Number Cruncher Statistical System) 2007 (Kaysville; Utah, USA) software was used for the statistical analysis. The study data were summarized with the descriptive statistics (mean, standard deviation, range, frequency, percentage, etc.). For comparison of the quantitative data, an independent samples test was used for the two group comparisons of the variables with normal distribution. Spearman's correlation analysis was used to evaluate inter-parameter relationships. Correction for multiple testing was applied. Significance was evaluated at $p < 0.05$.

3. Results

Forty-three patients with Bell's palsy (23 females, 20 males; mean age 43.8 ± 15.2 years) were included in the study. Bell's palsy affected the right side in 18 patients (41.9%) and the left side in 25 patients (58.1%). The mean BMI was 28.5 ± 5.4 kg/m^2. According to BMI, most patients (n = 33, 76.7%) were overweight or obese (BMI ≥ 25 kg/m^2). While 29 patients (67.4%) did not have any chronic disease, 13 patients had hypertension, 11 had diabetes mellitus, and 10 had both of these conditions. According to the HB facial nerve grading system, one patient had grade five, five patients had grade four, 26 patients had grade three, and 11 patients had grade two Bell's palsy. The mean HB score of patients was 2.9 ± 0.7. The vast majority of patients were seen

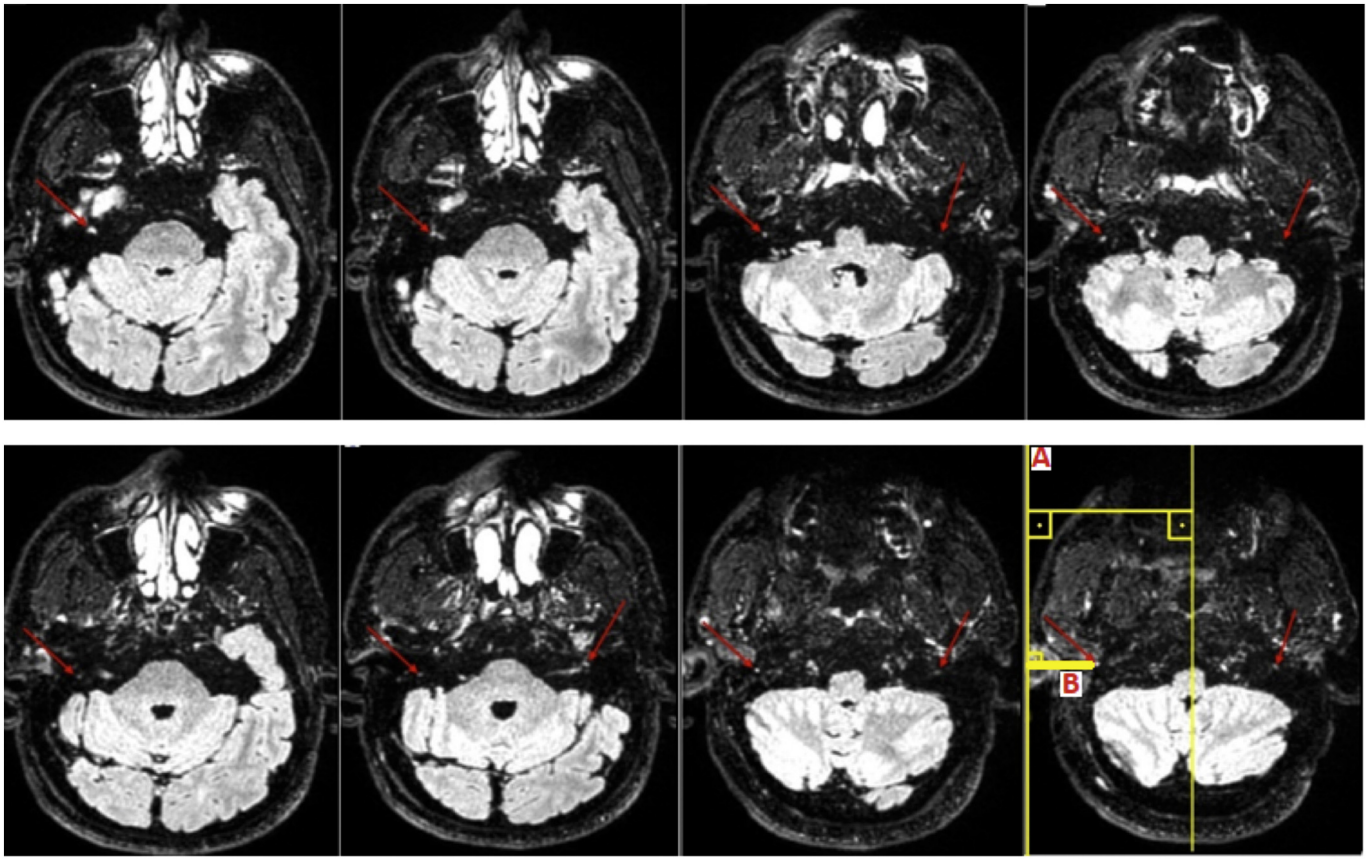

Fig. 1. Tracking the course of facial nerve on both sides (red arrows) and measurement of the depth at its exit from stylomastoid foramen on the axial cranial MR images. For the measurement of the facial nerve depth, a straight line is drawn between the nasion and the external occipital protuberance. Another anteroposterior straight line (A) is drawn parallel to this line which passes through the outermost skin surface of the preauricular region on both sides. The facial nerve is traced and located at the point of its exit from the stylomastoid foramen. A third line (B) passing through this point is drawn at 90° to line A and the distance of facial nerve to the outer skin is measured on line B. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Table 2
Demographic and clinical characteristics of the study cohort

Parameter	Result (n = 43)
Age (years), *mean ± SD*	43.8 ± 15.2 (20–79)
Gender, n (%)	
Female	23 (53.5%)
Male	20 (46.5%)
Laterality, n (%)	
Right	18 (41.9%)
Left	25 (58.1%)
House-Brackmann score, *mean ± SD*	2.9 ± 0.7 (2–5)
BMI (kg/m^2), *mean ± SD*	28.5 ± 5.4 (18–41)
BMI (kg/m^2), n (%)	
Normal	10 (23.3%)
Overweight (≥25 kg/m^2)	33 (76.7%)
Season at admission, n (%)	
Spring	14 (32.6%)
Summer	1 (2.2%)
Autumn	14 (32.6%)
Winter	14 (32.6%)
Concomitant chronic diseases, n (%)	
Diabetes mellitus	21 (48.8%)
Hypertension	23 (53.5%)

SD: standard deviation; BMI: body mass index.

during the transition seasons and winter season. Only one case was seen in the summer. The demographic and clinical characteristics of the study cohort are summarized in Table 2.

The facial nerve depth was significantly lower on the paralytic side compared to the unaffected side (32.9 ± 5.4 vs. 36.9 ± 5.1, respectively; $p = 0.007$). The facial nerve depth on the paralytic side was not statistically different in the female patients compared to male patients (31.2 ± 4.6 vs. 34.7 ± 5.7, respectively; $p = 0.270$) (Table 3).

Table 3
Facial nerve depth (mm) with respect to the presence of Bell's palsy and patient's gender

		Mean ± SD (min–max)	$p^{\dagger}$
Side	Paralytic side	32.9 ± 5.4 (23.1–48.0)	0.007
	Unaffected side	36.9 ± 5.1 (28.1–50.1)	
Gender[‡]	Female	31.2 ± 4.6 (21.0–79.0)	0.270
	Male	34.7 ± 5.7 (20.0–64.0)	

SD: standard deviation. Correction for multiple testing was applied.
† Independent samples test.
‡ For paralytic side.

The facial nerve depth on the paralytic side was not correlated with patients' age ($r = 0.288$; $p = 0.999$) or BMI ($r = 0.215$, $p = 0.999$). On the unaffected side, there was also no significant correlation between facial nerve depth and BMI ($r = 0.313$, $p = 0.280$). No significant correlation was found between facial nerve depth and HB scores on the paralytic and unaffected sides ($r = 0.031$ and $r = -0.018$, respectively; $p > 0.05$) (Table 4).

4. Discussion

In this retrospective study, we primarily showed that in patients

Table 4
Spearman's correlation coefficient (r) for the correlation between the facial nerve and age, BMI, and Hausebreakman score

	Facial nerve depth			
	Paralytic side		Unaffected side	
	r	p	r	p
Age (years)	0.288	0.999	–	–
BMI (kg/m^2)	0.215	0.999	0.313	0.280
House-Brackmann score	0.031	0.999	−0.018	0.999

BMI: body mass index. Correction for multiple testing was applied.

with Bell's palsy, the facial nerve in the paralytic side was located more superficially as it exited the stylomastoid foramen when compared to the contralateral side, suggesting that the depth of facial nerve has a key role in the etiology of Bell's palsy.

Several studies have explored the relationship between cold air and Bell's palsy. Campbell et al. [21] studied the seasonal, climatic, and latitudinal factors on the incidence of Bell's palsy and revealed that the incidence of Bell's palsy was higher in the cold season when compared to other seasons of the year. Diego et al. [2] examined 1906 Bell's palsy patients and reported that the number of cases seen during the summer season was significantly lower than those seen in other seasons. Blunt [22] showed that the disease occurred more frequently during periods of cold weather. Tovi et al. [23] also detected seasonal differences where Bell's palsy cases were seen more frequently in winter. Hsieh et al. [24] showed that seasonality was significantly associated with the incidence of Bell's palsy among men, with the incidence increases during the cold months. On the other hand, some studies failed to show a seasonal association of Bell's palsy [25,26]. In the present study, the majority of Bell's palsy patients were seen during the transition and winter seasons, supporting the possible relationship between cold air and Bell's palsy.

Cold air exposure has also been suggested to cause HSV-1 reactivation since some mutations of HSV-1 have been shown to have higher replication rates at low temperatures [27]. Sudden, frequent, and prolonged exposure to cold air temperature may lead to edematous neuritis with reflex ischemia caused by vasomotor changes in the facial region and/or HSV-1 reactivation in ganglion cells [28,29]. The reduction of temperature with superficialization of the facial nerve may cause HSV-1 reactivation. In our study, this is supported by the fact that the vast majority of patients developed the disease during the cold months compared with the warm months, and the facial nerve on the paralytic side was significantly more superficial than the healthy side.

There are some studies in the literature examining the relationship between facial nerve contrast enhancement and paralysis. When evaluating the results of our study, we could not obtain a reliable quantitative value of contrast enhancement because of inadequate image resolution. Respective studies in the literature suggested that even if the image resolution is sufficiently, high contrast enhancement should only be assessed by researches who have gathered experience in this field [30,31].

There is no consensus on the sex predisposition to Bell's palsy. As some studies reported that women have a greater tendency for the occurrence of Bell's palsy than men, some studies showed no gender difference [2,26]. In our study, although there was not a statistically significant difference between men or women in the study cohort, the facial nerve depth was shallower in female patients than male patients, indicating that the facial nerve was located more superficially at its exit from stylomastoid foramen in female patients when compared to male patients. This finding may explain why Bell's palsy is seen more frequently in females in some studies, which needs to be confirmed by further large-scale research [2,26].

Our study has its limitations. First of all, the study cohort had a small sample size, which decreased the statistical power of the study. Secondly, we measured the distance of depth of facial nerve only at a single level. Nevertheless, although the depth of the facial nerve at the level of stylomastoid foramen is known in the healthy population, no study has measured the facial nerve depth in patients with Bell's palsy. Thus, this is the first study to demonstrate the effect of the depth of the facial nerve from the skin surface at the level of the stylomastoid foramen in facial paralysis. Considering the limitations of the current study, further studies with larger cohorts evaluating the depth of facial nerve and other potential etiological factors are needed to validate our results and to clarify the etiopathogenesis of Bell's palsy.

5. Conclusion

In conclusion, in our study with patients showing signs of Bell's palsy, we found that the facial nerve at the level of its exit from the stylomastoid foramen is located more superficially on the paralytic side as compared to the unaffected side. Therefore, the depth of the facial nerve may potentially play a key role in the etiology of Bell's palsy. Since Bell's palsy has multiple and controversial etiological factors, we suggest that the depth of facial nerve may be an important factor in the development of Bell's palsy, which should be evaluated in further studies.

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Declaration of Competing Interest

None.

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