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NEUROSONOGRAPHIC ASSESSMENT OF THE FACIAL NERVE IN PATIENTS WITH HEMIFACIAL SPASM

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ABSTRACT

Ultrasound and neurosonographic examination of the facial nerve was performed in 60 patients with hemifacial spasm and 40 healthy controls. The aim was to identify structural and hemodynamic changes of the nerve associated with neurovascular conflict. Patients with hemifacial spasm showed a significant decrease in facial nerve diameter (1.9 ± 0.3 mm vs. 2.4 ± 0.2 mm; $p < 0.01$), reduced echogenicity, and altered perineural blood flow. These findings indicate compressive changes and early demyelination of nerve fibers. A positive correlation was established between the degree of neurosonographic changes and the severity of spasm ($r = 0.62$; $p < 0.05$), confirming the diagnostic value of the method.

KEYWORDS: *Hemifacial Spasm, Facial Nerve, Neurosonography, Neurovascular Conflict, Echogenicity.*

RELEVANCE

Hemifacial spasm (HFS) is a chronic neurological disorder characterized by involuntary contractions of the facial muscles on one side of the face, which significantly reduces patients' quality of life [1,2]. The prevalence of the disease ranges from 9 to 15 cases per 100,000 population, predominantly among middle-aged women. The main pathogenetic mechanism of HFS is considered to be neurovascular compression, in which an arterial or venous loop compresses the root exit zone of the facial nerve at its emergence from the brainstem. Chronic compression leads to focal demyelination of axons, the formation of ectopic excitation zones, and the development of pathological impulses that spread to adjacent fibers, which clinically manifests as a series of spasmodic twitching of the facial muscles.

Modern neuroimaging techniques, particularly magnetic resonance imaging (MRI), make it possible to verify neurovascular conflict and determine the anatomical relationships between the vessel and the nerve. However, despite the high diagnostic value of MRI, this method has limitations in assessing the peripheral segment of the facial nerve and the microcirculation within its structure. At the same time, neurosonography is becoming an increasingly valuable method for visualizing peripheral nerves, providing real-time assessment of their diameter, echogenicity, structural integrity, and perineural blood flow.

Early diagnosis of structural and hemodynamic changes in the facial nerve using neurosonography is of great importance for determining the stage of the pathological process, predicting the course of the disease, and selecting a personalized treatment strategy — ranging from neuroprotective pharmacotherapy to microvascular decompression. Thus, the study of neurosonographic characteristics of the facial nerve in hemifacial spasm represents a relevant and promising direction in modern neurology and functional neuroimaging.

OBJECTIVE

To study the neurosonographic characteristics of the facial nerve in hemifacial spasm and determine their diagnostic and prognostic significance.

MATERIALS AND METHODS

The study included 84 participants: 44 patients with hemifacial spasm (main group) and 40 practically healthy individuals (control group) matched by sex and age.

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Table 1
Characteristics of Study Participants

Parameter	Main group (n=44)	Control group (n=40)	Total (n=84)
Sex			
Males	18 (40,9%)	16 (40,0%)	34 (40,5%)
Females	26 (59,1%)	24 (60,0%)	50 (59,5%)
Age, years			
20–29	10 (22,7%)	8 (20,0%)	18 (21,4%)
30–39	15 (34,1%)	14 (35,0%)	29 (34,5%)
40–49	12 (27,3%)	12 (30,0%)	24 (28,6%)
≥50	7 (15,9%)	6 (15,0%)	13 (15,5%)
Disease Duration / Observation , years			
<1	8 (18,2%)	—	8 (9,5%)
1–3	20 (45,5%)	—	20 (23,8%)
>3	16 (36,3%)	—	16 (19,0%)

The groups did not differ by sex: in the main group, there were 18 men (40.9%) and 26 women (59.1%), while in the control group, there were 16 men (40.0%) and 24 women (60.0%). Most participants belonged to the 30–39 years age category (34.1% and 35.0%, respectively). Patients aged 20–29 years accounted for 22.7% and 20.0%, those aged 40–49 years — 27.3% and 30.0%, and those over 50 years — 15.9% and 15.0%. The duration of disease in patients with hemifacial spasm was less than 1 year in 8 individuals (18.2%), 1–3 years in 20 (45.5%), and more than 3 years in 16 (36.3%).

All participants underwent neurosonographic examination of the facial nerve using a high-frequency linear probe (7.5–12 MHz) on an expert-class ultrasound machine. The following parameters were assessed:
• Nerve diameter (mm) — to detect signs of compression or deformation;
• Nerve echogenicity — assessed using a visual scale from 1 to 5 points (1 — markedly decreased, 5 — normal structure);
• Perineural blood flow — evaluated using color Doppler scanning (CDS).
The clinical severity of the spasm was assessed using the Facial Spasm Severity Scale (FSSS), which scores from 0 (no spasms) to 4 (constant intense spasms with pronounced facial asymmetry).

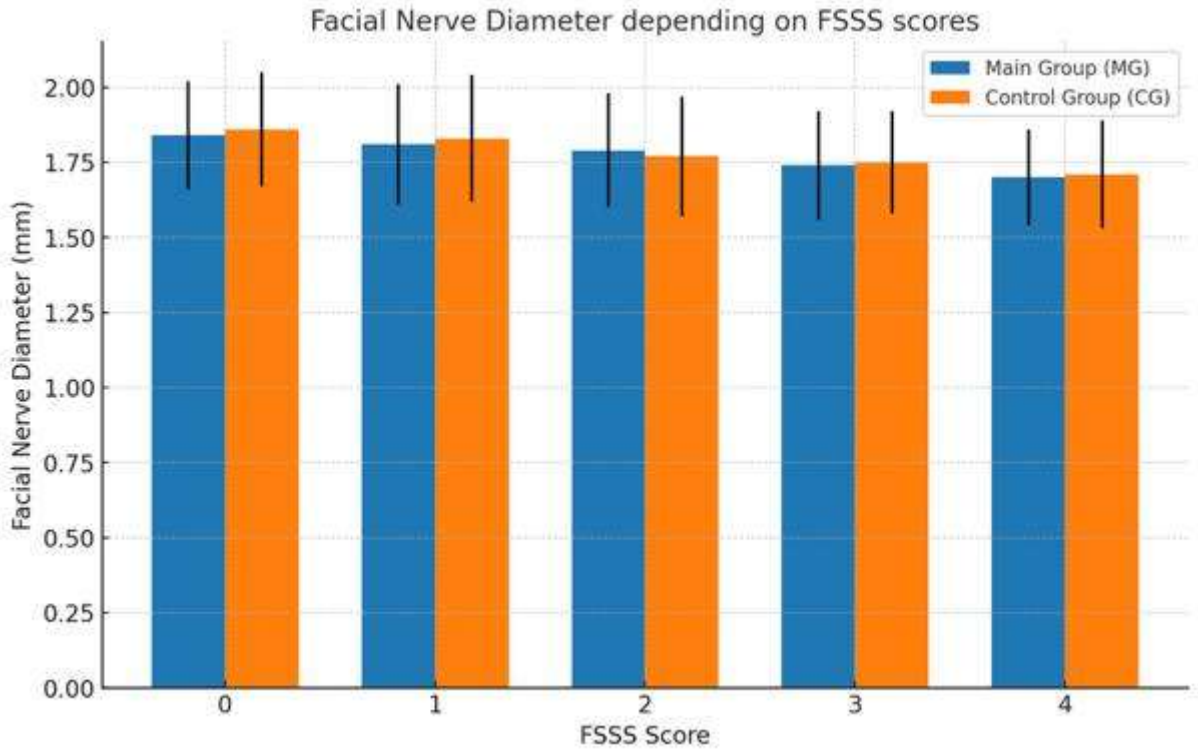
Statistical analysis was performed using SPSS 26.0. The t-test was used to compare parameters between groups, while correlations between neurosonographic indicators and spasm severity were determined using Pearson's correlation coefficient. Values of $p < 0.05$ were considered statistically significant.

Thus, the combination of neurosonography and clinical scales allowed for an objective assessment of the structural and functional changes in the facial nerve and their relationship with the clinical manifestations of hemifacial spasm.

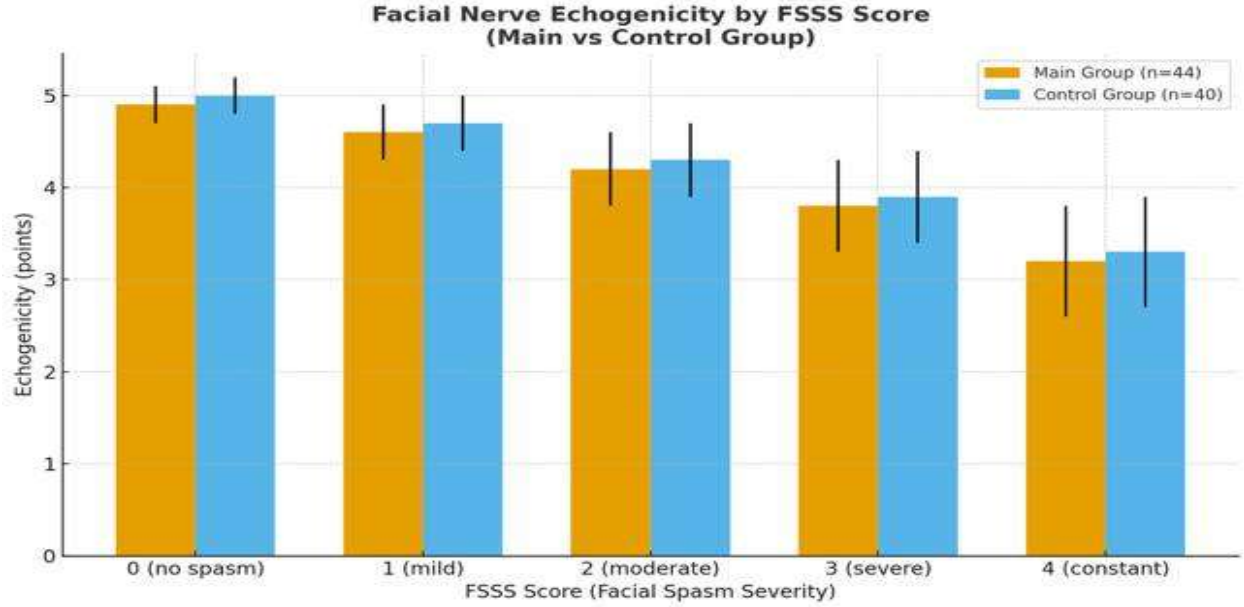
Study Results
Figure 2 shows the distribution of facial nerve diameter depending on the severity of hemifacial spasm, assessed using the Facial Spasm Severity Scale (FSSS), in patients of the main group (MG, n=44) and individuals of the control group (CG, n=40). Measurements were performed using a high-frequency linear probe (7.5–12 MHz), which allowed for the detection of signs of narrowing and deformation characteristic of compression and demyelination**.** In the main group, a gradual decrease in facial nerve diameter was observed with increasing spasm severity according to the FSSS:
• 1.84 ± 0.18 mm at 0 points (no spasms),
• 1.81 ± 0.20 mm at 1 point (mild spasms),
• 1.79 ± 0.19 mm at 2 points (moderate spasms),
• 1.74 ± 0.18 mm at 3 points (severe spasms),
• 1.70 ± 0.16 mm at 4 points (constant intense spasms).

In the control group, a similar trend was observed; however, the average nerve diameter values were slightly higher in participants without spasms or with minimal manifestations (e.g., 1.86 ± 0.19 mm at 0 points) and gradually approached the values of the main group at higher FSSS scores.

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The data obtained demonstrate an inverse relationship between facial nerve diameter and the severity of hemifacial spasm: as the FSSS score increases, a gradual decrease in nerve diameter is observed. The differences between the main and control groups are not pronounced, indicating comparability of the morphometric parameters of the facial nerve among study participants. These results suggest that the reduction in nerve diameter may reflect both compensatory and pathological changes occurring with spasm progression.



Note: Echogenicity was assessed using a visual scale from 1 to 5 points (1 — markedly decreased, 5 — normal structure); values are presented as mean ± SD.

Table 3 shows the relationship between facial nerve echogenicity and the severity of hemifacial spasm according to the FSSS in the main group (MG, n = 44) and control group (CG, n = 40). In both groups, echogenicity gradually decreased with increasing FSSS scores.

- In patients without spasms (0 points), the mean echogenicity was 4.9 ± 0.2 in the MG and 5.0 ± 0.2 in the CG;
- With mild spasms — 4.6 ± 0.3 and 4.7 ± 0.3;

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- With moderate spasms — 4.2 ± 0.4 and 4.3 ± 0.4;
- With severe spasms — 3.8 ± 0.5 and 3.9 ± 0.5;
- With constant intense spasms — 3.2 ± 0.6 and 3.3 ± 0.6, respectively.

An inverse relationship between facial nerve echogenicity and hemifacial spasm severity was established. The decrease in echogenicity reflects progressive structural changes (demyelination, compression, etc.) and can be considered an objective marker of nerve damage severity.
Assessment was performed using color Doppler scanning (CDS); values are presented as the number of patients and the percentage of the group.

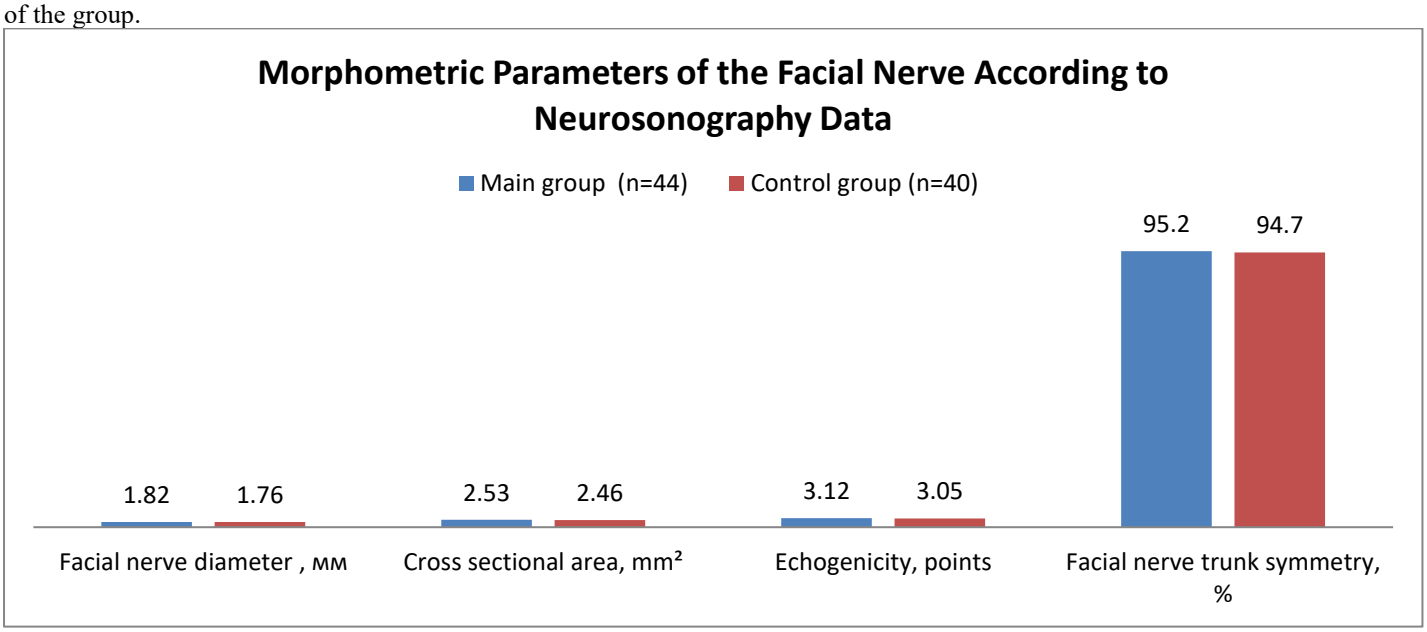


Figure 4 illustrates the distribution of perineural blood flow in the facial nerve depending on the severity of hemifacial spasm according to the FSSS in the main group (MG, n = 44) and control group (CG, n = 40). In both groups, the frequency of detected blood flow increased with greater spasm severity.

In the MG, perineural blood flow was observed in:
- 12.5% of patients without spasms,
- 25% with mild spasms,
- 40% with moderate spasms,
- 71.4% with severe spasms,
- 100% with constant intense spasms.

In the CG, a similar trend was noted: 7.1%, 15.4%, 40%, 60%, and 66.7%, respectively. A direct relationship was established between the severity of hemifacial spasm and the presence of perineural blood flow. The increase in the frequency of vascular response reflects the functional activity of the nerve and may serve as an objective marker of compensatory or pathological changes associated with spasm progression. Values are presented as mean ± standard deviation; the p-value was calculated for comparisons between the main and control groups.

Table 2
Morphometric Parameters of the Facial Nerve According to Neurosonography Data

Parameter	Main group (n=44)	Control group (n=40)	Total (n=84)	p-meaning
Facial nerve diameter, mm	1,82 ± 0,21	1,76 ± 0,19	1,79±0,20	0,14
Cross sectional area, mm²	2,53 ± 0,38	2,46 ± 0,35	2,50±0,37	0,18
Echogenicity, points	3,12 ± 0,51	3,05 ± 0,48	3,08±0,50	0,23
Symmetry of the facial nerve trunk, %	95,2 ± 4,8	94,7 ± 5,0	94,9±4,9	0,67

Note: Values are presented as mean ± standard deviation. The p-value was calculated for comparison of parameters between the main and control groups.

Table 2 presents comparative data on the morphometric characteristics of the facial nerve in participants of the main group (n = 44) and control group (n = 40). The mean nerve diameter was 1.82 ± 0.21 mm in the MG and 1.76 ± 0.19 mm in the CG (p = 0.14); the cross-sectional area was 2.53 ± 0.38 mm² and 2.46 ± 0.35 mm², respectively (p = 0.18). Echogenicity was comparable (3.12 ± 0.51 vs. 3.05 ± 0.48; p = 0.23), and trunk symmetry remained high (95.2 ± 4.8% vs. 94.7 ± 5.0%; p = 0.67). Morphometric parameters

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of the facial nerve in the main and control groups did not differ statistically, indicating structural homogeneity and stability of morphological characteristics in the studied population.

To analyze the relationships between facial nerve morphometric parameters and the severity of hemifacial spasm (according to the FSSS), Pearson's correlation coefficient (r) was used, and statistical significance was assessed by the p-value. Correlation analysis revealed a significant inverse relationship between nerve diameter and hemifacial spasm severity ($r = –0.62$, $p < 0.05$): as FSSS scores increased, nerve diameter decreased, reflecting progressive morphological changes. A similar negative correlation was observed between echogenicity and FSSS scores ($r = –0.55$, $p < 0.05$), indicating structural alterations of the nerve (demyelination and compression).

At the same time, a direct correlation was noted between perineural blood flow and spasm severity ($r = +0.48$, $p < 0.05$), reflecting reactive vascular remodeling with progression of the pathological process. With increasing spasm severity, nerve diameter and echogenicity decreased, while the frequency of detected perineural blood flow increased. Differences in morphometric parameters between the main ($n = 44$) and control ($n = 40$) groups were not statistically significant, demonstrating comparability of anatomical and functional characteristics. Nevertheless, the observed dynamics were consistent in both groups, confirming the generalizability of the identified patterns.

DISCUSSION

The results of this study confirm that the morphometric and functional parameters of the facial nerve are closely associated with the severity of hemifacial spasm. A decrease in nerve diameter and echogenicity, accompanied by an increase in perineural blood flow, reflects both degenerative and compensatory processes arising from chronic nerve compression [1–3,6,8]. These findings are consistent with previous studies indicating that ultrasound assessment of the facial nerve can detect early structural changes in hemifacial spasm [7,9,11]. The positive correlation between spasm severity and perineural blood flow supports the hypothesis of vascular adaptation of the nerve to chronic irritation [4,5,10]. Thus, neurosonographic features — reduced diameter, decreased echogenicity, and increased blood flow — can be considered objective markers of facial nerve damage severity [8,12].

CONCLUSIONS

1. With increasing severity of hemifacial spasm, there is a significant decrease in facial nerve diameter and echogenicity, accompanied by an increase in perineural blood flow.
2. All correlations are statistically significant ($p < 0.05$) and reflect morphofunctional changes of the nerve.
3. Neurosonography is a promising method for the objective assessment of lesion severity, early diagnosis, and dynamic monitoring of patients with hemifacial spasm.

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