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Emotional prosody expression in acoustic analysis in patients with right hemisphere ischemic stroke

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ABSTRACT

Objectives: The role of the right cerebral hemisphere in nonverbal speech activities remains controversial. Most research supports the dominant role of the right hemisphere in the control of emotional prosody. There has been significant discussion of the participation of cortical and subcortical structures of the right hemisphere in the processing of various acoustic speech parameters. The aim of this study was an acoustic analysis of the speech parameters during emotional expression in right hemisphere ischemic strokes with an attempt to reference the results to lesion location.
Materials and methods: Acoustic speech analysis was conducted on forty-six right-handed patients with right-middle cerebral artery stroke, together with 34 age-matched people in the control group. We compared the results of acoustic studies between patients with varying infarct locations and the control group.
Results: Variations in fundamental frequency during verbal expression of joy, anger and sadness were significantly smaller in the patient group than in the control group. Cortical lesion caused more restrictions in fundamental frequency variation in the expression of joy and a lower voice intensity in expressions of anger and joy compared to those patients with subcortical lesions.
Conclusions: Cortical lesion was associated with a more impaired expression of emotional prosody than subcortical lesion. The results indicate the leading role of the cortical structures of the right hemisphere in the expression of emotional prosody.

1. Introduction

The right hemisphere plays an important role in verbal communication, as it is mostly responsible for speech prosody and its emotional aspects. The majority of studies have indicated the domination of the right hemisphere in expression of emotional prosody and the superiority of the left hemisphere in the expression of linguistic prosody (functional hypothesis). These functional differences may be connected to the selective dominance of the right or left hemisphere in particular acoustic parameters of speech (physical hypothesis). The right hemisphere predominates in control of the fundamental frequency, the variations of which determine the corresponding levels of

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the expression of emotions [1–9]. The left hemisphere predominates in the control of the duration of speech and pauses, which determine the appropriate structure of the utterance [5,10–15]. The role of the right hemisphere in control of voice intensity in emotional prosody expression remains uncertain. According to Ross [2,16,17] only Fo variation is a sensitive indicator of emotional prosody expression, while the intensity of the voice is less important. Other studies highlight the importance of voice intensity (mean values) especially during the expression of anger [9,18]. Similarly vague is also the influence of the location of damage to the non-dominant hemisphere on emotional expression and maintenance of acoustic parameters of speech, such as Fo, their intensity and duration. Shapiro and Danly [6] showed smaller fundamental frequency variations during emotional expression in patients with vascular damage of the right frontal lobe, compared to the control group of healthy people and patients with damage to the left hemisphere. Blonder et al. [19] evaluated a patient with prosodic impairment caused by an extensive cortico-subcortical right hemisphere ischemic stroke. Ross and Monnot [20] described disturbances of emotional prosody expression in patients with ischemic cortical and subcortical lesion. These abnormalities were more severe in patients with cortical damage. Based on his own results, Ross [21] hypothesized that the functional and anatomical organization of the right hemisphere in terms of emotional prosody is highly similar to the organization of language functions in the left hemisphere. Ross then proposed a distinction of emotional prosody analogous with the classification of aphasia. Therefore, he distinguished, among others, motor, sensory and global aprosodia, combining the types with damage to relevant part of the right hemisphere. However, a verification of Ross's hypothesis gave ambiguous results. Some of the studies confirmed the impairment of the expression of emotional prosody in patients with damage to the right frontal lobe as well as impairment of perception in cases of damage to the right temporoparietal lobe [22–25]. Although other studies did not show such a relationship, in the majority of patients disturbances in prosody could have been related to Ross's classification [26–29]. Other studies revealed the significant role of subcortical structures in prosodic emotional expression and perception. Cancelliere [27] described emotional dysprosody following subcortical damage without respect to cerebral laterality. Starkstein et al. [25] found disturbances in the perception of emotional prosody in patients with damage to the basal ganglia. Van Lancker Sidtis et al. [30] demonstrated reduced F0 variability in acoustic speech analysis of two patients with isolated basal ganglia damage. Moreover, the participation of subcortical structures in the control of prosody seems to be confirmed by disturbances in the melody of speech in other diseases of the extrapyramidal system, such as Parkinson's disease [31–33]. There are several concepts on the pathogenesis of dysprosody in damage of the subcortical structures. Subcortical aphasias are explained mainly by the occurrence of diaschisis, where the subcortical damage causes secondary lesions in the cortical areas responsible for linguistic functions. An analogous mechanism may concern the prosody disorders, where the subcortical focus influences the cortical areas of the right hemisphere in a depressive manner, inhibiting their activity. All this may cause restrictions of the fundamental frequency variations, which is one of the basic elements of dysprosody. Another considered mechanism of subcortical dysprosody relates to the pathomechanism of dysarthria, where the motor coordination of the articulatory apparatus may be impaired. This results in changes in the duration of the verbal sequences and impairment of the coordination of the motor functions of speech. Other authors point out the important role of the corpus callosum in the control of speech prosody, the supplementary motor area and the frontal part of the cingulum of the right hemisphere [34–36]. The damage to the corpus callosum may impair interhemispheric communication, which integrates the prosodic functions, controlled by the right hemisphere, with speech abilities, processed by the left hemisphere [17].

The aim of this study was the characterization of the basic parameters of speech – the fundamental frequency, duration of the test utterance and the intensity during controlled emotional expression in patients with acute ischemic damages to the right hemisphere and in the control group, with reference of the emotional prosody to the location of the stroke.

2. Materials and methods

2.1. *Participants*

Forty-six right-handed patients with right middle cerebral artery stroke, including 16 females (35%) and 30 males (65%), hospitalized in the Department of Neurology, Wroclaw Medical University between October 2003 and December 2008 were evaluated. The average age of the patients was 58 $\pm$ 12.31 years old (22–74 years old). The native language of all the patients was Polish. Diagnosis of the ischemic stroke was made based on clinical symptoms and brain CT image. The degree of neurological deficit was evaluated using the National Institute of Health Stroke Scale (NIHSS) on the day of the acoustic speech exam which was performed within 7–14 days after symptoms of the stroke were shown. It was assumed that acoustic speech analysis in an early ischemic stroke can detect potentially reversible emotional dysprosody. The cognitive functions were evaluated with the Mini-Mental State Examination (MMSE) scale, and a result below 24 points was one of the criteria for exclusion from the study. Communicative abilities were assessed with the Goodglass-Kaplan scale. Other excluding criteria were speech disorders hindering verbal communication, both aphasia and dysarthria alike, as well as a history of previous strokes, psychiatric disorders, dementia, Parkinson's disease and acute internal illnesses (circulatory and respiratory failures, renal and liver failures). The patients were divided into subgroups depending on the location of the stroke in the CT image – patients with: cortical stroke (lesion in the cortex or cortex with white matter but without damage to the basal ganglia or the internal capsule), subcortical stroke (lesion in the basal ganglia and/or the internal capsule) and cortico-subcortical stroke. Multifocal ischemic strokes, leukoaraiosis and hydrocephalus were other criteria for exclusion from the study.

The control group consisted of 34 right-handed people, including 12 females (35%) and 22 males (65%), aged 35 up to 81 years old (mean age was 54.7 $\pm$ 11.13 years old) and without any clinical symptoms of damage to the central nervous system. None of the patients in the control group was diagnosed with speech disorders, psychiatric diseases or acute internal illnesses (circulatory and respiratory failures, renal and liver failures).

2.2. *Procedure*

The acoustic speech evaluations were conducted in constant conditions – in the Laboratory of Speech Acoustics, Department of Neurology, Wroclaw Medical University. The recordings were made within 7–14 days after the symptoms of the stroke were shown. During the exam, patients were sitting and the microphone was placed 5 cm form their mouths. Speech was recorded on the hard drive of a desktop computer with a Pentium 3.00 GHz processor, with 1GB RAM and a Sound Blaster Audity sound card. For the speech analysis and audio editing two programs were used: Medicom ''Iris'' and Steinberg WaveLab 6, both modified for the needs and purposes of the Laboratory of Speech Acoustics.

Before the recording, patients were given a list of the test sentences and subsequently oral instructions were given detailing the performance of particular phases of the study. The initial action was the longest possible vocalization of the sound ''a'', which was used for evaluation of the technical conditions of the exam (the quality of the recorded sound, appropriate settings for the technical parameters of the acoustic program and the positioning of the microphone). The exam task was to repeat the phase ''there is no'' (in Polish ''nie ma'') with different emotional coloring four-fold in each emotion. The first statements were supposed to be neutral, the following ones expressing anger, sadness and joy, with the strongest possible emotional involvement. None of the phases of the test, including the oral instructions, indicated how the exam task was supposed to be performed, and this was aimed at elimination of any kind of model imposition by the investigator. The recorded samples were stored on the hard drive of the desktop computer and later used in the computer acoustic analysis. All the calculations conducted on the collected material were done automatically by the computer program. From each of the four recorded repetitions, only recordings number 2 and 3 were used in the acoustic analysis. In each variant of the test utterance, the following acoustic parameters were analyzed:

a) the variation of the fundamental frequency (Fo), determined by the standard deviation of Fo (Hz)

   We did not perform a log on transformation of Fo (such as converting the Hz data into semitones) or a co-efficient of variation measurement, because of the similar gender distribution among the patients and in the control group.

b) the mean voice intensity in decibels (dB)
c) the duration of the utterance.

For each of the evaluated parameters in every patient subgroups and in the control group mean values with standard deviation (SD) were calculated. For verification of the statistical differences between the patients (with cortical, subcortical and cortico-subcortical strokes) and the control group the following tests were employed:

1. Kolmogorov–Smirnov Test with Lilliefors's correction in order to evaluate whether the distribution of the assessed characteristics was consistent with the normal distribution
2. Other tests comparing the expected values for characteristics consistent with the normal distribution in order to find the statistically important differences between the patients and the control group:

- The Fisher–Snedecor Test – to compare the compatibility of variances
- The Student *t*-Test – to compare the expected values (equal variances)
- The Cochran–Cox Test – to compare the expected values (different variances).

To verify the statistical differences of particular acoustic parameters obtained during the utterances made with various emotional expressions by the patients and controls the one-way ANOVA test was employed. In cases of statistically important differences, the next step was to use the Schiffe post hoc test which enabled the identification of which of the compared pairs differed significantly in the expected (mean) values. $p \leq .05$ was assumed as the statistically important level. For the statistical analysis STATISTICA 7.0 PL was used.

3. Results

3.1. *The clinical characteristics*

In the analyzed group of patients the average point score on the NIHSS was 4.26 (range between 2 and 9 points). The CT brain images showed: cortical stroke in 22 patients (48%), subcortical stroke in 20 patients (43%) and cortico-subcortical stroke in 4 patients (9%).

3.2. *The acoustic parameters of speech in patients and the control group*

In order to evaluate the influence of the ischemic damage of the right hemisphere on the expression of emotional prosody, variations of fundamental frequency, intensity of speech and time parameters were compared in patients and the control group.

Variations in fundamental frequency during verbal expression of joy, anger and sadness were significantly smaller than in the control group (Table 1). The alternations of Fo during the neutral utterance did not differ significantly in either group.

Analysis of the fundamental frequency during emotional expression showed that both in patients and in controls the direction of the changes of the voice frequency was consistent with the expression of the particular emotional state. Variations in the fundamental frequency in the expression of joy were significantly larger in both groups compared to the neutral and sad utterance. Changes in the Fo in the expression of anger were bigger compared to the neutral utterance (Fig. 1). Moreover, in the control group significant variations in Fo were found in the expression of anger compared to sadness, which was not found in those patients with damage to the right hemisphere. Changes in fundamental frequency in expressions of the remaining emotions were similar.

Table 1 – Acoustic parameters (mean ± SD) of emotional expression in patients and the control group.

	Fundamental frequency variation (Hz)		Voice intensity (dB)		Utterance duration (s)	
	Patients	Controls	Patients	Controls	Patients	Controls
Neutral	15.66 ± 12,486	17.69 ± 11,044	54.91 ± 5966	54.52 ± 6201	0.61 ± 0.141	0.59 ± 0.111
Joy	*30.62 ± 20,710	64.25 ± 35,200	56.87 ± 5607	57.27 ± 6076	0.63 ± 0.166	0.64 ± 0.214
Sadness	*20.06 ± 13,206	26.45 ± 15,657	55.03 ± 5642	54.54 ± 6534	0.67 ± 0.194	0.68 ± 0.198
Angry	*28.13 ± 16,712	48.46 ± 25,692	58.93 ± 5825	60.43 ± 5048	0.56 ± 0.115	0.59 ± 0.118

* $p \leq .05$ in comparison to control group.

Mean voice intensity values during expression of particular emotions did not differ significantly between the groups (Table 1). However, smaller – statistically insignificant ($p = .0985$) – differences were found between voice intensity during expression of anger in patients and the control group.

Analysis of the sound intensity showed that both patients and controls were able to change it according to the type of emotion (Fig. 2). Voice intensity during expression of anger was statistically significantly bigger compared to sadness and neutral expression. However, no significant difference between the remaining utterances was found.

Moreover, no differences were found between the duration of the phrase "there is no" with different emotional expressions in patients and controls (Table 1).

Nevertheless, in patients the duration of the expression of sadness was longer than the expression of anger (Fig. 3). The durations of the remaining utterances in patients and all utterances in controls were similar.

3.3. *Changes in the acoustic parameters of speech in patients with vascular damage to the right hemisphere with reference to the location of stroke in the CT brain image*

In order to evaluate the influence of the location of the stroke on the expression of emotional prosody, variations of the fundamental frequency, the intensity of speech and time parameters were analyzed in patients with cortical, subcortical and cortico-subcortical strokes and in the control group.

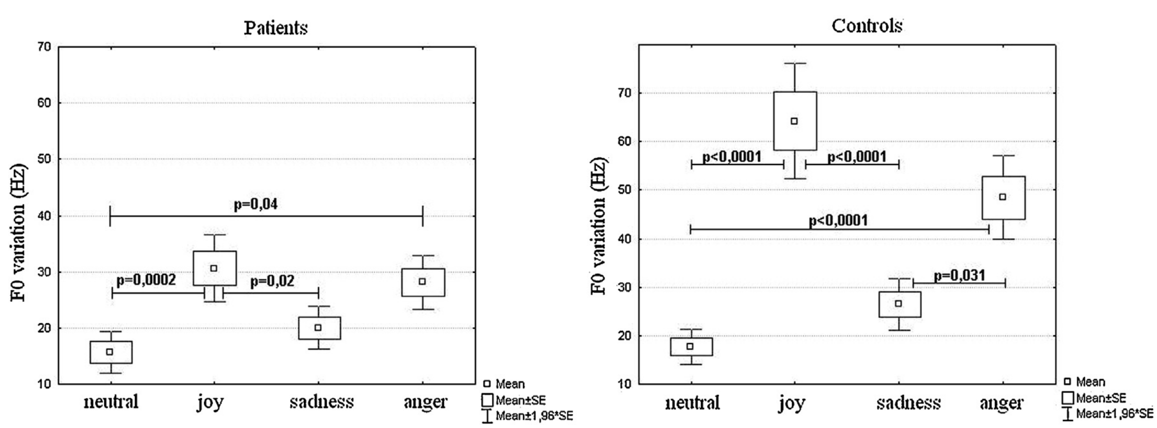

Fig. 1 – Fo variation in utterances with different emotional coloring: patients and control group.

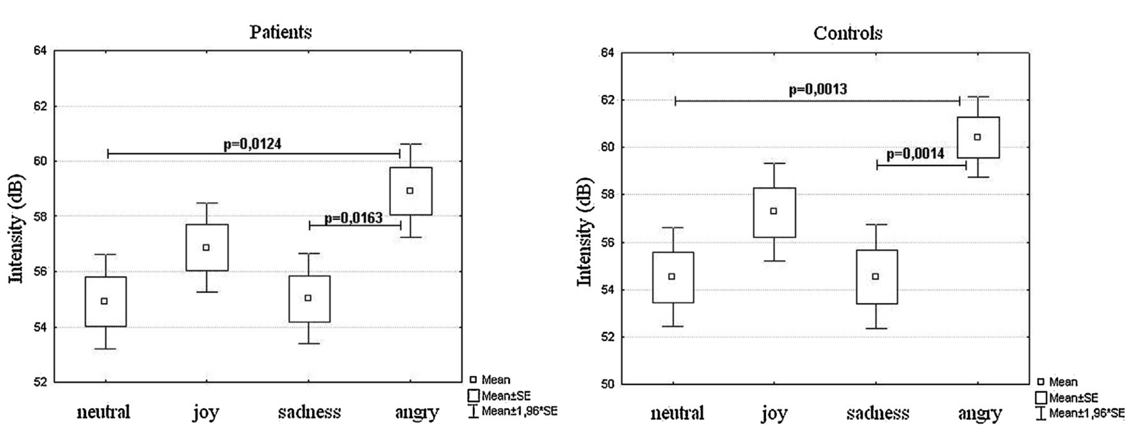

Fig. 2 – Voice intensity of utterances with different emotional coloring: patients and control group.

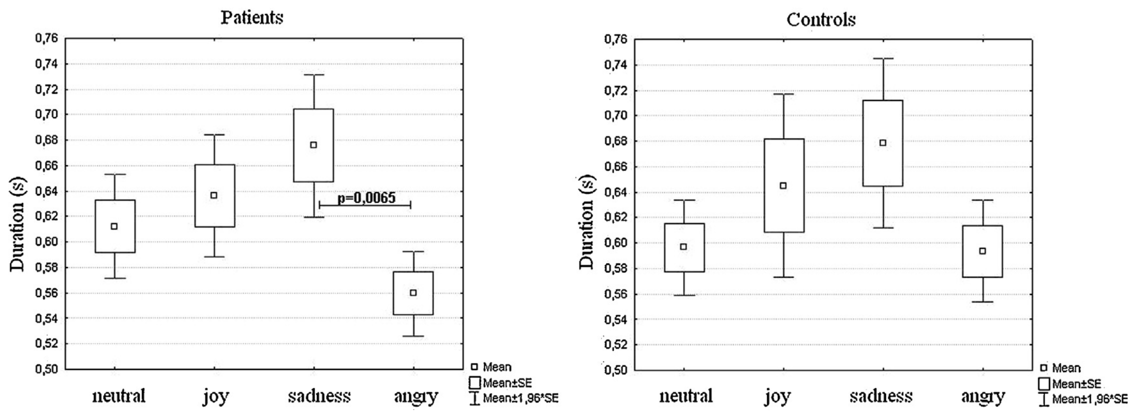

Fig. 3 – Duration of utterances with different emotional coloring: patients and control group.

Variations of Fo were significantly smaller in patients with subcortical strokes during expression of joy, anger and sadness compared to the control group. Variations of Fo were significantly smaller in patients with cortical strokes in the expression of joy and anger and in patients with cortico-subcortical strokes – in the expression of joy, compared to the controls (Table 2). Moreover, in patients with cortical strokes smaller variations in the fundamental frequency were observed in the expression of joy in comparison to those patients with subcortical strokes.

Voice intensity was significantly smaller during expression of anger in patients with cortical and cortico-subcortical strokes compared to the control group. In patients with cortical strokes a smaller voice intensity was also found in expressions of anger and joy in comparison to patients with subcortical strokes (Table 3).

The duration of the neutral utterance was longer in patients with cortico-subcortical strokes compared to the controls. Patients with subcortical strokes showed a longer duration in the expression of joy than patients with cortical strokes (Table 4).

4. Discussion

Previous studies on the acoustics of speech suggest that impairment of the expression of emotions in injury to the right hemisphere is caused mostly by reductions of variations of the

Table 2 – Fundamental frequency variation (mean ± SD) during emotional expression in patients with cortical, subcortical, cortico-subcortical strokes and the control group.

	Fundamental frequency variation (Hz)			
	Neutral	Joy	Sadness	Anger
Subcortical stroke (*n* = 22)	14.41 ± 12,026	*37.25 ± 25,113	*19.31 ± 1677	*27.20 ± 13,057
Cortical stroke (*n* = 20)	17.64 ± 14,022	$^{*,\#}$24.63 ± 12,730	21.21 ± 15,478	*28.57 ± 20,431
Cortico-subcortical stroke (*n* = 4)	12.68 ± 5611	*24.10 ± 19,357	18.48 ± 11,310	31.02 ± 18,261
Controls (*n* = 34)	17.69 ± 11,044	64.25 ± 35,200	26.45 ± 15,675	48.46 ± 25,692

* $p \leq .05$ in comparison to control group
$^{\#}$ $p \leq .05$ in comparison to patients with subcortical strokes

Table 3 – Voice intensity (mean ± SD) during emotional expression in patients with cortical, subcortical, cortico-subcortical strokes and the control group.

	Voice intensity (dB)			
	Neutral	Joy	Sadness	Anger
Subcortical stroke (*n* = 22)	56.74 ± 6043	58.72 ± 5601	56.66 ± 5864	61.11 ± 5290
Cortical stroke (*n* = 20)	53.77 ± 5657	$^{\#}$55.56 ± 5419	53.93 ± 5514	$^{*,\#}$57.37 ± 6038
Cortico-subcortical stroke (*n* = 4)	50.60 ± 4215	52.76 ± 2744	52.12 ± 2792	*54.75 ± 2785
Controls (*n* = 34)	54.52 ± 6201	57.27 ± 6076	54.54 ± 6534	60.43 ± 5048

* $p \leq .05$ in comparison to control group.
$^{\#}$ $p \leq .05$ in comparison to patients with subcortical strokes.

Table 4 – Utterance duration (mean ± SD) during emotional expression in patients with cortical, subcortical, cortico-subcortical strokes and the control group.

	Utterance duration (s)			
	Neutral	Joy	Sadness	Anger
Subcortical stroke (n = 22)	0.59 ± 0.151	#0.68 ± 0.215	0.7 ± 0.256	0.56 ± 0.128
Cortical stroke (n = 20)	0.61 ± 0.114	0.59 ± 0.096	0.65 ± 0.113	0.55 ± 0.105
Cortico-subcortical stroke (n = 4)	*0.76 ± 0.150	0.63 ± 0.048	0.69 ± 0.118	0.62 ± 0.081
Controls (n = 34)	0.6 ± 0.111	0.64 ± 0.214	0.68 ± 0.198	0.59 ± 0.118

* $p \le .05$ in comparison to control group.
\# $p \le .05$ in comparison to patients with cortical strokes.

fundamental frequency [2,6,37,38]. In our materials these observations were confirmed, importantly showing a narrower range in the variations of the fundamental frequency during verbal expression connected to particular emotions in patients with ischemic damage to the right hemisphere, compared to the control group. The biggest difference in the variations of the fundamental frequency in patients and in controls revealed itself during expression of joy, and a bit smaller in expression of anger. Nevertheless, the direction of the variations to fundamental frequency was maintained, i.e. patients with strokes in the right hemisphere were able to change the fundamental frequency according to the assumed emotional coloring of the test sentence. However, it was done with significantly lower efficiency than by the controls, which indicates the quantitative, not qualitative, nature of emotional expression dysprosody in patients. On the basis of the CT brain image, patients with cortical and subcortical lesion in the right hemisphere were distinguished, showing a correlation between the locus of the stroke and the expression of emotional prosody. The largest deficit in the expression of the emotional prosody was caused by the cortical damage, which was presented by reduction of the Fo variations during expression of joy compared to the patients with subcortical strokes. Earlier studies that used a computer acoustic speech analysis were conducted on small groups of patients with various locations of the strokes in the right hemisphere. To our knowledge, no previous studies have compared the expression of emotional prosody in patients with isolated cortical and subcortical lesions. In this situation, it was impossible to establish a correlation between the location of the ischemic lesion (cortical vs subcortical) and expressive dysprosody. The biggest of the acoustic studies on affective prosody published so far, where the patient group was homogenous in terms of the location of the stroke, was conducted by Ross and Monnot [20]. The authors revealed expressive dysprosody deficits in patients with mainly cortical ischemic strokes of the right hemisphere. However, there were no patients with isolated subcortical damage. Our comparative analysis of Fo variability in patients with isolated cortical and isolated subcortical strokes showed larger deficits in patients with cortical damage.

The role of the right hemisphere in the control of voice intensity remains unclear. This parameter is rarely the subject of clinical trials, acoustic studies or functional neuroimaging [16,39–41]. According to Ross et al. [17], this parameter plays a less important role than Fo in the expression of emotional prosody. Gandour et al. [42] did not identify changes in the voice intensity (RMS energy measurement) during expression of joy and anger. Similarly, in our own studies no significant difference in voice intensity during emotional expression was observed either in patients, or in controls. The highest voice intensity was registered during expression of anger in both groups. However, analysis of the intensity in patients with damage to cortical and subcortical structures showed a lower intensity of the voice during the expression of anger and joy in patients with cortical strokes compared to the patients with subcortical strokes. Moreover, the intensity of the voice in patients with subcortical strokes was not significantly different compared with the control group. A dominant role of the right hemisphere cortex in the expression of voice intensity, which was shown in own studies, has not been evaluated previously.

Previous studies argue against the important role of the right hemisphere in the control of the durational features (e.g. phrase length, tempo, rhythm) [37,43]. Similarly, in our study, there were no significant differences in the duration of the test tasks between the patients and controls. In the cases of subcortical lesion, the duration of the joyful utterance was longer than in patients with cortical strokes, but similar to that in the control group. These changes are not sufficient to indicate the predominance of cortical or subcortical structures in the control of speech timing.

Our own studies indicate that cortical and subcortical structures of the right hemisphere have an important role in the control of emotional prosody. Injury to these structures caused the impairment of the fundamental frequency variation that was more pronounced in patients with cortical lesions. Furthermore, only damage to cortical structures produced a deficit in voice intensity.

5. Conclusions

The presented study, based on computer acoustic speech analysis, confirmed the important role of the right hemisphere in the expression of emotional prosody. In a precise and objective manner, the directions of changes of prosodic parameters of speech were shown, also in correlation to the location of ischemic damage to the non-dominant hemisphere. In particular, attention was drawn to the dominant role of the fundamental frequency in modulation of emotional expression. The results obtained from acoustic analysis indicate the integrative nature of the cortico-subcortical activities connected to the verbal expression of emotional prosody. Nevertheless, the dominant role in this process belongs to the cortical structures of the right hemisphere.

Conflict of interest

None declared.

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None declared.

Ethics

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans; Uniform Requirements for manuscripts submitted to Biomedical journals.

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