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# Cerebral microembolism in patients with segmental left ventricular wall motion abnormalities

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ABSTRACT

*Background and purpose:* The significance of segmental heart wall motion abnormalities for stroke is unknown. The aims of the study included (1) comparison of the frequency and type of embolic signals in the middle cerebral artery in patients with segmental left ventricular wall hypokinesis due to coronary heart disease with and without stroke, and (2) determination of the relationship between inflammatory parameters, fibrinogen level, dyslipidemia and microembolic signals in the middle cerebral artery in patients with segmental heart hypokinesis.

*Material and methods:* The study included 68 patients with segmental heart hypokinesis (33 without stroke [group I] and 35 with stroke [group II]), as well as 37 healthy volunteers and a reference group of 30 patients. Echocardiography and carotid/transcranial Doppler with detection of microembolic signals were performed. Patients from group I and II had erythrocyte sedimentation rate, leucocyte count, triglycerides, total cholesterol, HDL, and LDL examined.

*Results:* Embolic signals were detected in patients with segmental heart hypokinesis significantly more frequently than in the control and reference groups. The high number of embolic signals, signals of high intensity, hypokinesis of the distal part of the intraventricular septum, increased cholesterol levels, LDL and triglycerides were all found more frequently in patients from group II than in group I. Embolic signals were detected more frequently in patients with high fibrinogen levels and leukocytosis.

*Conclusions:* Embolic signals in the middle cerebral artery in patients with segmental left ventricular hypokinesis have to be considered as a risk factor of stroke. The following changes are observed in patients with cardiogenic stroke: hypokinetic intraventricular septum, high intensity embolic signals, increased serum fibrinogen levels and leucocyte count. It may indicate the importance of these factors in the aetiology of stroke.



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## 1. Introduction

Cerebral artery embolisms of cardiac origin are a clinically significant source of strokes. According to Warlow, they constitute the cause of 20% of cerebral infarctions. Thrombi developing in the heart are the embolic material in these cases [1]. According to Welch et al. [2], the causes of the absolute risk of cardiogenic cerebral embolism include: atrial fibrillation, mitral stenosis, mechanical heart valves, early myocardial infarction, atrial myxoma and the presence of thrombus in the left ventricle. The relative causes include, among other things, mitral valve prolapse, patent foramen ovale, interatrial septal aneurysm, calcific aortic stenosis and segmental left ventricular wall motion abnormalities post myocardial infarction [3]. The level of risk of cardiogenic stroke is still a controversial issue in such cases.

Literature data suggest a relationship between global left ventricular systolic function impairment in the form of a decrease in cardiac ejection fraction and the formation of cardiogenic embolism. Advanced systolic function impairment contributes to the formation of thrombus in the left ventricle. It is also assumed that the presence of hypokinetic segments in the cardiac wall may be conducive to the formation of thrombi [4]. The introduction and modification of methods of detecting embolic material (echocardiography and transcranial Doppler examination with embolism monitoring) over the past three decades are an argument in favour of revising the opinion on the risk of cerebral embolism secondary to various cardiac diseases.

In the literature, it is accepted that there is a relationship between the presence of emboli and the occurrence of clinically evident stroke [5–10]. So far, a number of reports on monitoring embolism in diseases constituting its potential cause have been published. Publications regarding the assessment of embolism in diseases considered to be relative causes of cardiogenic cerebral embolism are scarce. The combination of echocardiography and transcranial Doppler examination seems to be an optimal method in determining the significance of segmental left ventricular wall motion abnormalities for the occurrence of cardiogenic cerebral embolism.

The aims of the study included (1) comparison of the frequency and type of microembolic signals detected in the middle cerebral artery on transcranial Doppler ultrasonography in patients with segmental left ventricular wall motion abnormalities secondary to ischaemic heart disease without stroke and with cardiogenic stroke; and (2) determining relationships between selected indicators of inflammation (erythrocyte sedimentation rate [ESR], leucocyte count), fibrinogen levels as well as lipid disorders and the presence of microembolic signals in the middle cerebral artery on transcranial Doppler ultrasonography in patients with segmental left ventricular wall motion abnormalities.

## 2. Material and methods

Sixty-eight patients hospitalized at the 2nd Cardiology Department of the Medical University of Silesia or the Neurology Department of the Medical University of Silesia between 1999 and 2010 in whom segmental left ventricular wall motion abnormalities were found with the use of transthoracic echocardiography (TTE) were examined.

The subjects were divided into two groups. Group I consisted of 33 patients (9 women and 24 men) aged 21–71 years (mean: 53.7; standard deviation [SD]: 10.85 years) with ischaemic heart disease diagnosed by a cardiologist (on the basis of anginal pain in patient history and the signs of past ischemia in electrocardiogram [ECG]), without the clinical signs of stroke. Group II included 35 patients (9 women and 26 men) aged 37–87 years (mean: 62.7; SD: 12.6 years) with ischemic disease who were in the acute phase of embolic ischaemic stroke during the study. Cardiogenic stroke was diagnosed on the basis of ischaemic stroke classification criteria proposed by Adams et al. [11]. The study did not include patients with past stroke, another concomitant or past central nervous system (CNS) disease, valvular heart disease and implanted valves, cardiac dysrhythmias, thrombus present in the heart, myocardial infarction in the past 6 months as well as stenosis or occlusion of carotid and intracranial arteries diagnosed on the basis of the results of a Doppler examination.

The types of left ventricular systolic function impairment and their prevalence are presented in Table 1.

The following diseases co-occurred in the subjects: arterial hypertension in 40 patients: 17 from group I (51.5%) and 23 from group II (65.7%) (non-significant, NS); type 2 diabetes in 22 subjects: nine from group I (27.3%) and 13 from group II (37.1%) (NS); lipid disorders in 23 subjects: one from group I (3.03%) and 22 from group II (62.8%) ($p < 0.001$).

The reference group consisted of 30 patients treated at the Cardiology and Diabetology Outpatient Clinic of the Independent Teaching Hospital No. 7 in Katowice (20 women and 10 men) aged 38–74 years (mean: 56.5 years; SD: 9.7) without left ventricular function impairment (normal results of TTE), in whom the main risk factors for atherosclerosis (arterial hypertension, hyperlipidaemia and diabetes), similar to those in groups I and II, occurred: Arterial hypertension occurred in 10 subjects (33.3%), type 2 diabetes in 23 subjects (76.7%), and lipid disorders in 28 subjects (93.3%). Ischaemic heart disease was found in 6 patients.

The control group consisted of 37 healthy volunteers (23 women and 14 men) aged 41 to 71 years (mean: 54.1 years; SD: 8.98), who were patients with radicular syndromes treated at the Neurology Department and Neurology Outpatient Clinic. Physical and neurological examinations along with recent laboratory test results allowed us to exclude cardiovascular and central nervous system diseases in them.

All patients from groups I and II as well as the reference and control ones were informed of the objective of the study and consented to it. The study was approved by the Bioethical Commission of the Medical University of Silesia.

The following measures were carried out in the patients with segmental left ventricular wall motion abnormalities (groups I and II) and in the reference group: (1) Interview and physical examination; (2) TTE and, additionally in 11 patients in whom TTE did not allow an unequivocal exclusion of the presence of thrombus, transesophageal echocardiography (TEE) was performed; and (3) 12-lead ECG.

The following were additionally performed in groups I and II: ESR and leucocyte count, total cholesterol, triglycerides, HDL

**Table 1 – Types of left ventricular wall motion abnormalities in groups I and II.**

| Type of left ventricular systolic function impairment | Patients with systolic function impairment | Group I $n = 33$ | | Group II $n = 35$ | | $p$-Value |
|---|---|---|---|---|---|---|
| | | $n$ | % | $n$ | % | |
| Hypokinesis of whole IVS | 10 | 4 | 12.1 | 6 | 17.1 | NS |
| Hypokinesis of distal part of IVS | 18 | 14 | 42.4 | 4 | 11.4 | 0.0061 |
| Asynchronism of IVS contraction | 2 | 2 | 6.1 | 0 | 0 | NS |
| Hypokinesis of anterior wall | 2 | 0 | 0 | 2 | 5.7 | NS |
| Hypokinesis of side wall | 17 | 5 | 15.1 | 12 | 34.3 | NS |
| Hypokinesis of apex | 16 | 7 | 21.2 | 9 | 25.7 | NS |
| Hypokinesis of inferior wall | 3 | 1 | 3.03 | 2 | 5.7 | NS |

IVS – intraventricular septum; NS – not significant.

cholesterol, LDL cholesterol, serum fibrinogen levels. All the included patients underwent examination of blood flow velocity in extra- and intracranial blood vessels, in the middle cerebral artery with the Doppler method – patients with ischaemic stroke in the first 24–48 h of the disease, patients without the signs of stroke during their stay at hospital, and in the reference and control groups on an outpatient basis.

Detection of cerebral embolism in the middle cerebral artery (at the depth of 60 mm for 30 min on each side) was performed in patients with ischaemic stroke in the first 24–48 h of the disease, in patients without the signs of stroke during their stay at the hospital and in the reference and control groups on an outpatient basis. The total number of microembolic signals per hour and the intensity of the microembolic signals were determined in each patient. In the study, we use the term 'signal intensity', frequently utilized in English-language literature, which is equivalent to the term ''signal frequency'' expressed in decibels. Microembolic signals were analyzed with the use of commercially available software developed by EME. Doppler examinations and detection of cerebral embolism were performed at the Doppler Laboratory of the Neurology Department of the Independent Public Teaching Hospital No. 7 in Katowice. A Pioneer 2002 TC device, manufactured by EME, equipped with 2- and 4-MHz transducers as well as a two-gate transducer for detecting cerebral embolism was used. The detected microembolic signals met the Spencer criteria [12].

### 2.1. *Statistical analysis*

Statistical analysis was carried out with the use of licensed versions of: Microsoft Excel 2003 and Statsoft Statistica 7.1 PL software. At the first stage of the statistical analysis, the basic descriptive statistics were determined (arithmetic means, medians, quartiles, variances, standard deviations, etc.). The $\chi^2$ test for independence or Fisher exact test was used to compare the distribution of nonparametric variables. Nonparametric variables were compared with the use of Student $t$-test (age – normal distribution) or the Mann–Whitney $U$-test (the duration of coronary heart disease not meeting the condition of normal distribution). The null hypothesis concerning the conformity of a given parameter to normal distribution was assessed with the use of the Kolmogorov–Smirnov or Shapiro–Wilk tests. In the statistical analysis, the accepted level of statistical significance (type I error) was $p(\alpha) < 0.05$.

## 3. Results

Microembolic signals were detected in 10 patients in group I, 17 patients in group II and in one individual in the reference group. They were not found in individuals in the control group. The difference between groups I and II was statistically insignificant. Statistical significance was achieved when comparing results obtained in patients with segmental left ventricular wall motion abnormalities (groups I and II) with that of the control group ($p < 0.01$) as well as the reference group ($p < 0.01$).

Larger numbers of microembolic signals (3–7 signals) were found in patients in group II. The statistical difference was significant in comparison with patients from group I ($p < 0.05$) (Table 2).

Higher-intensity microembolic signals (7–11 dB) were found in patients in group II. The statistical difference was significant in comparison with patients from group I ($p < 0.01$) (Table 3).

Microembolic signals in group II, i.e. in patients with stroke, were recorded more frequently in comparison with group I

**Table 2 – Number of persons with the presence of 1, 2 or 3 microembolic signals (MES) recorded in groups I and II.**

| | Number of patients with MES | Patients with: | | | | No of patients with 1–2 MES | No of patients with 3–7 MES |
|---|---|---|---|---|---|---|---|
| | | 1 MES $n$ (%) | 2 MES $n$ (%) | 3 MES $n$ (%) | 7 MES $n$ (%) | $p = 0.047$[a] | |
| Group I ($n = 33$) | 10 | 4 (12.1%) | 6 (18.2%) | | | 10 | 0 |
| Group II ($n = 35$) | 17 | 2 (5.7%) | 9 (25.7%) | 5 (14.3%) | 1 (2.9%) | 11 | 6 |

[a] Fisher test.

**Table 3 – The frequency of the signals of different intensities in groups I and II.**

| | Patients with MES of different intensities *n* (%) | | | | | | | Number of patients with MES | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | 5 dB | 6 dB | 7 dB | 8 dB | 9 dB | 10 dB | 11 dB | 5–6 dB | 7–11 dB | |
| Group I ($n = 33$) | 9 (27%) | 1 (3%) | | | | | | 10 | 0 | $p < 0.01$ |
| Group II ($n = 35$) | 8 (22.8%) | 8 (22.8%) | 4 (11.4%) | 2 (5.7%) | 1 (2.8%) | 1 (2.8%) | 1 (2.8%) | 16 | 9 | |

MES – microembolic signals.

**Table 4 – The frequency of microembolic signals (MES) in groups I and II, depending on the location of segmental left ventricular wall motion abnormalities.**

| Localization of left ventricular impairment | No of patients | | | | | | *p*-Value |
|---|---|---|---|---|---|---|---|
| | Group I + II $n = 68$ | | Group I $n = 33$ | | Group II $n = 35$ | | |
| | All patients | Patients with MES | All patients | Patients with MES | All patients | Patients with MES | |
| Hypokinesis of whole IVS | 10 | 7 (70%) | 4 | 4 (100%) | 6 | 3 (50%) | NS |
| Hypokinesis of distal part of IVS | 18 | 6 (33.3%) | 14 | 3 (21.4%) | 4 | 3 (75%) | <0.05 |
| Asynchronism of IVS contraction | 2 | 1 (50%) | 2 | 1 (50%) | 0 | 0 | NS |
| Hypokinesis of side wall | 17 | 5 (24.9%) | 5 | 0 | 12 | 5 (41.6%) | NS |
| Hypokinesis of anterior wall | 2 | 0 | 0 | 0 | 2 | 0 | NS |
| Hypokinesis of inferior wall | 3 | 1 (33.3%) | 1 | 0 | 2 | 1 (50%) | NS |
| Hypokinesis of apex | 16 | 7 (43.7%) | 7 | 2 (28.6%) | 9 | 5 (55.5%) | NS |

IVS – intraventricular septum; NS – not significant.

**Table 5 – Selected inflammatory and lipid variables in groups I and II.**

| Variable | Range | Group I | | Group II | | *p* |
|---|---|---|---|---|---|---|
| | | *n* | % | *n* | % | |
| ESR | ≤5/10 mm/h | 22 | 66.6 | 16 | 45.7 | NS |
| | >5/10 mm/h | 11 | 33.3 | 19 | 54.3 | |
| WBC | $\leq 10 \times 10^3/\mu L$ | 27 | 81.8 | 28 | 80 | NS |
| | $>10 \times 10^3/\mu L$ | 6 | 18.2 | 7 | 20 | |
| Cholesterol | ≤200 mg/dL | 32 | 96.9 | 19 | 54.3 | <0.001 |
| | >200 mg/dL | 1 | 3.03 | 16 | 45.7 | |
| Triglycerides | ≤150 mg/dL | 33 | 100 | 23 | 65.7 | <0.05 |
| | >150 mg/dL | 0 | 0 | 12 | 34.3 | |
| LDL-cholesterol | ≤100 mg/dL | 33 | 100 | 22 | 62.8 | <0.001 |
| | >100 mg/dL | 0 | 0 | 13 | 37.1 | |
| HDL-cholesterol | >40 mg/dL | 33 | 100 | 34 | 97.1 | NS |
| | ≤40 mg/dL | 0 | 0 | 1 | 2.8 | |
| Fibrinogen | ≤3.5 g/L | 30 | 90.9 | 27 | 77.1 | NS |
| | >3.5 g/L | 3 | 9.1 | 8 | 22.9 | |

ESR – erythrocyte sedimentation rate; WBC – white blood cell; GR – group; NS – not significant.

only in the cases of hypokinesis located in the distal part of the interventricular septum ($p < 0.05$) (Table 4).

Elevated total cholesterol (>200 mg/dL), LDL (>100 mg/dL), and triglycerides (>150 mg/dL) levels were found significantly more frequently in group II in comparison with group I (Table 5).

Among patients with segmental left ventricular wall motion abnormalities (groups I and II), microembolic signals occurred significantly more frequently in patients with elevated fibrinogen levels (>3.5 g/L) and in patients with elevated leucocyte count ($>10 \times 10^3/\mu L$) in comparison with patients with normal fibrinogen levels and leucocyte counts ($p < 0.001$ and $p < 0.02$, respectively). No statistically significant differences between patients with normal and abnormal parameters: ESR, total cholesterol, LDL, HDL, triglycerides levels as well as ejection fraction (<40% vs. >40%) were observed (Table 6).

## 4. Discussion

The objective of the study was to determine, by monitoring embolisms with the use of TCD, whether the presence of segmental left ventricular wall motion abnormalities can be the source of embolic material. Patients with cerebrovascular disease had microembolic signals more often than patients

**Table 6 – The presence of microembolic signals (MES) and the results of selected laboratory tests for the whole group of 68 patients with segmental left ventricular dysfunction.**

| Variable | Range | No of patients | MES | | *p*-Value |
|---|---|---|---|---|---|
| | | | *n* | % | |
| ESR | ≤5/10 mm/h | 38 | 12 | 31.6 | NS |
| | >5/10 mm/h | 30 | 15 | 50 | |
| WBC | $\leq 10 \times 10^3/\mu L$ | 55 | 18 | 32.7 | <0.02 |
| | $>10 \times 10^3/\mu L$ | 13 | 9 | 69.2 | |
| Total cholesterol | ≤200 mg/dL | 51 | 19 | 37.2 | NS |
| | >200 mg/dL | 17 | 8 | 47.1 | |
| Triglycerides | ≤150 mg/dL | 56 | 22 | 39.3 | NS |
| | >150 mg/dL | 12 | 5 | 41.6 | |
| LDL-cholesterol | ≤100 mg/dL | 55 | 21 | 38.2 | NS |
| | >100 mg/dL | 13 | 6 | 46.1 | |
| HDL-cholesterol | >40 mg/dL | 67 | 26 | 38.8 | NS |
| | ≤40 mg/dL | 1 | 1 | 100 | |
| Fibrinogen | ≤3.5 g/L | 57 | 17 | 29.8 | <0.001 |
| | >3.5 g/L | 11 | 10 | 90.9 | |
| Left ventricle ejection fraction | ≥40% | 64 | 26 | 40.6 | NS |
| | <40% | 4 | 1 | 25 | |

ESR – erythrocyte sedimentation rate; WBC – white blood cell; NS – not significant.

without cerebral events, which had been indicated in earlier reports by Babikian et al. [14], Markus et al. [15] and Kumral et al. [16]. In the presented study, in the group of patients with segmental left ventricular wall motion abnormalities without the clinical symptoms of stroke, microembolic signals were detected in almost 1/3 of patients. Similar results were obtained by Sliwka et al. [17] and Tong et al. [18]. The difference in the incidence of microembolic signals between groups I and II was statistically insignificant in the presented study. They occurred, however, significantly more often in patients with segmental left ventricular wall motion abnormalities (groups I and II) in comparison with the control and reference groups. The obtained results, therefore, confirm the hypothesis that segmental left ventricular wall motion abnormalities are associated with the presence of microembolic signals.

The incidence of microembolic signals in patients with segmental left ventricular wall motion abnormalities in whom no other possible causes of cardiogenic embolism have been found was analyzed in the presented study. The results of the presented study suggest that embolism can also be found when only segmental left ventricular wall motion abnormalities without a simultaneously detectable thrombus are detected in a patient. The frequency of detection of microembolic signals in these cases is similar to the results obtained by Kuznetsow et al. [19] in patients with cardiogenic ischaemic stroke with one cause of cardiogenic cerebral embolism.

More microembolic signals are detected in patients with stroke and a cardiac cause of embolism than in patients with stroke without a determined potential source of embolism [20,21]. The number of signals increases in cardiac diseases associated with a higher risk of the occurrence of embolism [22,23]. The presented results are comparable with those obtained by other authors and suggest that the number of microembolic signals increasing to at least 3 in patients with segmental left ventricular wall motion abnormalities should become a signal warning against the possibility of the occurrence of ischaemic embolic stroke [24].

The analysis of the intensity of microembolic signals in the study groups allowed us to determine that only low-intensity signals were detected in patients without stroke. In turn, in the group of patients with stroke, higher-intensity microembolic signals occurred as well. It has been proven that the higher intensity of microembolic signals is dependent on the density of embolism more than on its size [22]. In the literature, it has been demonstrated that solid emboli consisting of various platelet aggregates cause weaker (lower intensity) microembolic signals than atherosclerotic particles of the same size [25]. The cardiogenic causes of embolism are the source of signals of both higher and lower intensity [26]. These differences can be associated with the heterogeneity of cardiogenic embolic material. The results of the presented study suggest that patients with confirmed presence of segmental left ventricular wall motion abnormalities and the presence of high-intensity microembolic signals are at a particularly high risk of clinically evident stroke.

Periapical left ventricular wall motion abnormalities found with the use of TTE are important factors predisposing the formation of left ventricular thrombus [27,28]. The results of the presented study concerning patients without fresh myocardial infarction and only with segmental wall motion abnormalities confirm these observations. In the group of patients without an ischaemic cerebral event, segmental left ventricular wall motion abnormalities were associated with the occurrence of microembolic signals only in cases with apical hypokinesis. In the group of patients post-ischaemic stroke more than a half of patients with apical hypokinesis also had microembolic signals on TCD, i.e. more frequently than with another location of segmental left ventricular wall motion abnormalities. This result seems to be in agreement with the opinion, suggested in the newest reports, that the basic factors increasing the risk of cardiogenic embolic complications include the location of the formation of embolic

material, and not only the qualitative and quantitative characteristics of microembolic signals [22]. According to the results of the presented study, the distal part of the septum seems to be a place of easier formation of dangerous embolic material as well. The occurrence of microembolic signals in patients with stroke was significantly more common in comparison with individuals without stroke in cases of hypokinesis located in the distal part of the interventricular septum.

The causal relationship between hypercholesterolaemia and stroke is not so well documented as in the case of coronary artery disease. It is doubtless that there is a direct relationship between hyperlipidemia and carotid artery atherosclerosis, which is currently a sufficient argument in favour of considering hyperlipidemia to be a risk factor for ischaemic stroke. In the discussed study, occurrence of elevated total cholesterol, LDL and triglycerides as well as decreased HDL levels was found more frequently in patients with the symptoms of acute stroke in comparison with group I. However, the elevated lipid metabolism parameters did not exhibit a relationship with the occurrence of microembolic signals. It is worth mentioning that platelets are considered to be the main cellular element of particles generating microembolic signals. It has been proven that the platelets of patients in the acute phase of ischaemic stroke are activated [29]. However, it is still unknown if platelet activation is an independent risk factor or a result of the impact of hyperlipidemia, diabetes and arterial hypertension, which affect their functions [30]. Among the potential haemostasis disorders, only plasma fibrinogen concentration has been verified as an independent risk factor for arterial thrombosis in prospective epidemiological studies [31–33]. A summary report concerning 7 studies confirmed that plasma concentration of fibrinogen that is within the upper third of the normal range increases the risk of cardiovascular diseases by 2.3 times in comparison with the group of individuals in whom such concentration was in the lower third of the normal range [34]. This risk is particularly increased by the co-occurrence of arterial hypertension. Increases in fibrinogen levels and blood viscosity are more significant for prognosis than hypercholesterolaemia. High fibrinogen concentration worsens prognosis in patients after ischaemic stroke [35]. In the presented study, elevated fibrinogen concentration was significantly associated with the occurrence of microembolic signals, which seems to suggest the importance of elevated fibrinogen concentration for the formation of emboli. Leukocytosis was also significantly associated with the occurrence of embolism. Therefore, hyperfibrinogenaemia was perhaps – at least in some patients – stimulated by infection and these two factors were significant for the formation of emboli in these patients. In the literature, it has been demonstrated that when several causes of thrombus formation within the heart co-occur, its embolic potential increases [19,22]. Combined abnormalities, therefore, increase the risk of cerebral embolism. In the presented study, in patients with segmental left ventricular wall motion abnormalities, it was also found that the risk of cerebral embolism depends on the location of wall motion abnormalities, the intensity and number of microembolic signals as well as fibrinogen concentration.

## 5. Method limitations

The long period of patient recruitment was caused by difficulties in selecting a group that would closely reflect the inclusion and exclusion criteria. It was particularly difficult to find patients with stroke in whom it was possible to assume, at a level of probability close to certainty, that embolism was solely caused by segmental left ventricular wall motion abnormalities, that is, patients in whom other causes of embolism were unequivocally excluded. In the second group, the causes of ischaemic heart disease were not established. Because in the general population more than 98% of its cases are caused by coronary artery atherosclerosis, this aetiology can be assumed at a high level of probability in the subjects from group II as well. Throughout the entire study period, a protocol established before its beginning (1999) was observed and it was impossible to modify it in the following years in spite of the increased availability of laboratory tests, e.g. CRP.

## 6. Conclusions

1. Microembolic signals detected in the middle cerebral artery with the use of Doppler ultrasonography in individuals with segmental left ventricular wall motion abnormalities can be clinically significant as a risk factor for stroke.
2. Patients with cardiogenic stroke are frequently found to have septal hypokinesis, higher-intensity microembolic signals, elevated fibrinogen concentration and an increase in serum leucocyte count, which can suggest that the listed factors are significant for the aetiology of stroke.

## 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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