Efficiency of Various Cerebral Protection Techniques Used during the Surgical Treatment of Chronic Pulmonary Thromboembolism

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Abstract: Circulatory arrest during pulmonary thromboendarterectomy (PTE) for chronic pulmonary embolism leads to an increased risk of cerebral ischemia and neurological complications. This study aimed to assess the efficacy of various cerebral protection techniques used during the surgical treatment of chronic pulmonary thromboembolism. We prospectively studied 61 patients with chronic pulmonary thromboembolism who underwent PTE. We compared the dynamics of cerebral oxygen saturation (rSO2, %) during the surgical treatment and analyzed neurological complications during the early postoperative period in two groups of patients: 30 patients who underwent surgery under conditions of moderate hypothermia (23°C–24°C) combined with antegrade unilateral cerebral perfusion (ACP group) and 31 patients who underwent thromboendarterectomy under deep hypothermic circulatory arrest (18°C, DHCA group) combined with craniocerebral hypothermia. In the ACP group, regional rSO2 decreased by less than 20% from baseline during the course of PTE. In the DHCA group, a more profound reduction of cerebral oxygen supply (by >30% from baseline) was recorded compared with the ACP group (p < .05). During the early postoperative period, 29% of patients in the DHCA group exhibited neurological complications, compared with only 7% of patients from the ACP group. The results of logistic regression analysis indicated that the risk of progressive neurological deficit depended on the duration of the intraoperative period when the absolute values of regional rSO2 were <40%. The method with the best adjustment to human physiology in patients with chronic pulmonary thromboembolism was antegrade cerebral perfusion. This method provides a smaller decrease in cerebral oxygen supply during thromboendarterectomy and significantly reduces the risk of ischemia and neurological complications in the early postoperative period. Keywords: pulmonary embolism, cerebral oxygenation, cerebral protection, neurologic injury.
cerebral neurons, with subsequent ischemic cellular deterioration, is the main deleterious factor involved in the progression of cerebrovascular disturbances (5). This cellular deterioration arises from baseline hypoxia and worsening of cerebral tissue hypoxia in the course of surgery due to prolonged artificial perfusion and the application of circulatory arrest (6). Through this pathological process, patients who require circulatory arrest with total termination of cerebral blood flow have the highest incidence of global cerebral ischemia and progression of neurological deficits (7,8).

Nowadays, alternative methods are available for protecting the brain against cerebral ischemic damage (9,10). Our science institute has implemented a method for PTE performed under conditions of moderate hypothermia combined with antegrade unilateral cerebral perfusion (ACP). This method is one of the ways to prevent postoperative neurological complications and it has gained a positive reputation through its application in aortic arch repair surgery (11,12).

In this study, we tested the hypothesis that ACP combined with moderate hypothermia used during PTE is a more effective method of cerebral protection for patients with chronic pulmonary thromboembolism, compared with deep hypothermia combined with craniocerebral hypothermia under conditions of circulatory arrest.

**MATERIALS AND METHODS**

This was a prospective, randomized study. Every patient signed informed consent documents and the study was approved by the Institutional Review Board. Study identifier is AMC-1 10,773. We analyzed data from 61 patients, median age 49 (37–62) years (data are expressed as median [25th–75th percentile]), who were diagnosed with chronic pulmonary thromboembolism, based on clinical and instrumental examination, and underwent PTE between July 2011 and October 2013. Of them, 46 patients (75%) were men and 15 (25%) were women. The median duration of disease was 2.0 (1.3–3.3) years.

**Inclusion Criteria**
1. A diagnosis of chronic pulmonary thromboembolism with an indication for PTE
2. A history of lower limb deep vein thrombosis
3. An anatomically complete circle of Willis, as shown by computed angiography.

**Exclusion Criteria**
1. Arterial lesions in the brachiocephalic and intracranial arteries with a significant effect on hemodynamics
2. Any type of neurological dysfunction (including non-focal neurological symptoms, impairment of consciousness, diminution of cerebral competence, motor activity impairment, and focal cerebral damage)
3. A history of cerebrovascular disease

The primary endpoint of our study was the evaluation of the dynamics of cerebral oxygen supply by measurement of cerebral oxygen saturation (rSO2, %) at successive surgical stages of PTE under different methods of cerebral protection. The secondary endpoint was the percentage of patients with various neurological complications in the early postoperative period, in relation to the method of cerebral protection used during the surgery. Allocation of patients to study groups was carried out by simple blind randomization method using sealed opaque envelopes.

The first group included 30 patients, 24 men and six women, median age 51 (39–61) years. This group underwent surgery under conditions of moderate hypothermia (23°C–24°C) combined with ACP (ACP group), performed using right subclavian artery cannulation during thromboendarterectomy of the pulmonary artery branches.

The second group included 31 patients, 22 men and nine women, median age 48 (36–59) years, who underwent PTE under conditions of deep hypothermic circulatory arrest (DHCA group), performed with a decrease in body temperature to 18°C by perfusion and craniocerebral hypothermia established by packing the head with ice.

**Operative and Anesthetic Techniques**

In all patients, the surgical procedures were performed under conditions of CPB and nonpulsatile flow. The medical equipment included a membrane oxygenator (Affinity; Medtronic Inc., Minneapolis, MN) and a heart–lung machine (Stöckert™ SIII, Sorin Group, Munich, Germany). Volumetric blood flow was maintained at 2.5 L·min⁻¹·m⁻².

In the ACP group, PTE was performed to remove emboli from the pulmonary branches after clamping the brachiocephalic artery and starting ACP with a volumetric flow rate of 10 mL·kg⁻¹·min⁻¹ to maintain the right radial arterial pressure within 55–100 mmHg. After the removal of emboli from the right pulmonary branch, the clamp was removed from the brachiocephalic artery and volumetric flow rate was increased to the estimated value. Thereafter, the cold reperfusion stage was started and maintained for approximately 50% of the duration of ACP, after which the brachiocephalic artery was clamped once again and ACP was started during PTE to remove emboli from the left pulmonary branch. The nasopharyngeal temperature under conditions of moderate hypothermia was 23°C–24°C.

In the DHCA group of patients, PTE was performed to remove emboli from the pulmonary branches under conditions of DHCA and craniocerebral hypothermia (cloth helmet filled with ice is put on patient’s head immediately after anesthesia induction). Body temperature lowering was achieved with a thermal gradient of 7°C–8°C (heat-transfer
The nasopharyngeal temperature decreased to 18°C from the establishment of CPB to the time when nasopharyngeal temperature increased to 36°C warmed under conditions of CPB, and termination of CPB. The cooling time was defined as the period lasting approximately 50% of the circulatory arrest time with a full estimated rate. When body temperature increased to 34°C, 15% mannitol was administered in a dose of .25 g/kg body weight.

After the completion of PTE, the patient was slowly warmed under conditions of CPB until the nasopharyngeal temperature increased to 36°C. The termination of CPB was controlled by invasive monitoring of central and peripheral hemodynamics.

**Cerebral Oximetry**

The rSO2 of the right and left hemispheres during the intraoperative period was assessed using bilateral transcranial spectroscopy (cerebral oximeter INVOS 5100, Somanetics Corp., Troy, MI), with a probe attached to the right and left frontotemporal regions of the head surface. The rSO2 of the left and right hemispheres was recorded at different stages, as follows: 1) induction of anesthesia, 2) establishment of CPB, 3) PTE to remove emboli from the right pulmonary branch, 4) cold reperfusion, 5) PTE to remove emboli from the left pulmonary branch, 6) reperfusion before warming, 7) warming under conditions of CPB, 8) termination of CPB, and 9) termination of surgery.

All patients received routine perioperative monitoring, including continuous pulse oximetry and recording of the electrocardiogram, heart rate, and arterial and central venous pressures. The dynamics of blood glucose levels were monitored at different stages of surgery, as follows: induction of anesthesia, during the course of CPB, after PTE, warming under conditions of CPB, and termination of CPB.

After surgery, all patients were admitted to the intensive care unit (ICU). Patients were placed on mechanical ventilation according to a standardized protocol. The extubation criteria were clear consciousness, stable hemodynamics, an absence of signs of excessive drainage loss, and stabilization of electrolyte, acid-base, and respiratory parameters.

The institution of inotropic support was guided by hemodynamic data. Patients were transferred from ICU to hospital ward when they met the following criteria: stable hemodynamics without inotropic support or vasoactive drugs, urine output >.5 mL/kg/h, and minimal drainage.

**Neurological Status**

Before surgery, all patients underwent a clinical and instrumental assessment of their neurological state to identify any type of deficit, including non-focal neurological symptoms, diminution of cerebral competence, and focal cerebral deficits. Neurological status was examined with Mini-Mental State Examination (MMSE), 30-point questionnaire that is used extensively to measure state of cognitive functions and for screening of cognitive deterioration (13). The maximum score in that test is 30, which corresponds to high cognitive abilities. Instrumental methods included magnetic resonance imaging (MRI) and electroencephalography (EEG) to elucidate focal and functional changes in brain.

No neurological deficit was detected in any patient during the preoperative period; according to the MRI and EEG data there were no focal and functional changes in brain and according to the results of MMSE questionnaire, for every patient, composite score was 30.

During the early postoperative period, depression of consciousness was evaluated in the ICU according to the Glasgow Coma Scale (14). During the patients’ stay in the somatic department (at day 7–9), repeated clinical and instrumental assessments of neurological state were performed. We also recorded any other complications: pulmonary reperfusion syndrome, atrial fibrillation, acute myocardial infarction, renal and hepatic insufficiency, and need for cardiac pacing. The mortality rate was determined by registered in-hospital death.

**Statistical Analysis**

Statistical analyses were performed using Statistica ver. 6.1 (StatSoft Inc., Tulsa, OK). Because the criteria of normal distribution were not satisfied, data were expressed as median (25th–75th quartiles) or numerical values and percentages. The Wilcoxon signed-rank test was used to compare dependent variables, while the Mann–Whitney U test was used for independent variables.

The chi-square test with Yates’ correction or Fisher’s exact test was used for categorical variables, as appropriate. To study the correlation between variables, we used Spearman’s rank correlation coefficient. Univariate logistic regression analysis was performed to determine the risk factors for neurological complications during the early postoperative period. Odds ratios (ORs) are reported with 95% confidence intervals (CIs). All p values <.05 were considered statistically significant.

**RESULTS**

The clinical and functional characteristics of both groups of patients with chronic pulmonary thromboembolism are shown in Table 1. According to the intraoperative data, patients in the DHCA group had a longer warming time compared with the ACP group (p = .01).

**Parameters of Cerebral Oxygenation**

The changes in rSO2 values for the right and left hemispheres in patients with chronic pulmonary thromboembolism are shown in Figure 1, for both groups at different stages of surgery.
During PTE to remove emboli from the right pulmonary branch, in the DHCA group of patients a decrease in rSO₂ by 29% and 26% relative to previous values was registered in the right and left hemispheres, respectively. This was more pronounced than the changes in the ACP group, where rSO₂ values decreased by 14% and 17% in the right and left hemispheres, respectively. The differences between groups were statistically significant for both the right (\( p = .01 \)) and left (\( p = .02 \)) hemispheres. Furthermore, at this stage of surgery, the absolute rSO₂ values for

### Table 1. Clinical and functional characteristics of the patients with chronic pulmonary thromboembolism.

<table>
<thead>
<tr>
<th>Physiological Data, Measurement Units</th>
<th>First Group: 30 Patients, Antegrade Cerebral Perfusion Combined with Moderate Hypothermia</th>
<th>Second Group: 31 Patients, Deep Hypothermic Circulatory Arrest Combined with Cranio cerebral Hypothermia</th>
<th>( p ) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>48.1 (38–54)</td>
<td>49.0 (34–59)</td>
<td>.35</td>
</tr>
<tr>
<td>Male/female</td>
<td>21/9</td>
<td>21/10</td>
<td>.52</td>
</tr>
<tr>
<td>New York Heart Association class</td>
<td>2.8 (2.5–3.4)</td>
<td>2.9 (2.5–3.8)</td>
<td>.21</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>27.8 (21.1–30.2)</td>
<td>28.2 (22.3–30.5)</td>
<td>.35</td>
</tr>
<tr>
<td>History of myocardial infarction</td>
<td>1 (3%)</td>
<td>1 (3%)</td>
<td>.55</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1 (3%)</td>
<td>2 (6%)</td>
<td>.18</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1 (3%)</td>
<td>1 (3%)</td>
<td>.65</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>2 (6%)</td>
<td>2 (6%)</td>
<td>.51</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>5 (16%)</td>
<td>6 (19%)</td>
<td>.42</td>
</tr>
<tr>
<td>Chronic renal failure</td>
<td>1 (3%)</td>
<td>0</td>
<td>.65</td>
</tr>
<tr>
<td>Mean pulmonary arterial pressure (mmHg)</td>
<td>49.2 (38.6–61.3)</td>
<td>48 (36–56.8)</td>
<td>.54</td>
</tr>
<tr>
<td>Pulmonary arterial resistance (dynes-s-cm⁻¹)</td>
<td>671.0 (453–1157)</td>
<td>695.0 (410–1069)</td>
<td>.26</td>
</tr>
<tr>
<td>Right ventricular ejection fraction (%)</td>
<td>37 (26–55)</td>
<td>40 (35–52)</td>
<td>.62</td>
</tr>
<tr>
<td>Left ventricular ejection fraction (%)</td>
<td>65 (57–72)</td>
<td>68 (62–74)</td>
<td>.63</td>
</tr>
<tr>
<td>Cooling time (minute)</td>
<td>62.0 (52.0–73.0)</td>
<td>68.0 (55.0–80.0)</td>
<td>.36</td>
</tr>
<tr>
<td>Cold time (minute)</td>
<td>60.0 (49.0–75.0)</td>
<td>60.0 (51.0–74.0)</td>
<td>.67</td>
</tr>
<tr>
<td>Circulatory arrest time (minute)</td>
<td>41.0 (30.0–52.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antegrade cerebral perfusion time (minute)</td>
<td>38.0 (30.0–56.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Warming time (minute)</td>
<td>62.0 (55.0–68.0)</td>
<td>77.0 (63.0–84.0)</td>
<td>.01*</td>
</tr>
<tr>
<td>Cardiopulmonary bypass time (minute)</td>
<td>196.0 (177.0–227.0)</td>
<td>207.0 (194.0–238.0)</td>
<td>.18</td>
</tr>
<tr>
<td>Aortic cross-clamp time (minute)</td>
<td>114.2 (97–126)</td>
<td>115.5 (98–135)</td>
<td>.36</td>
</tr>
</tbody>
</table>

\( *p < .05 \) was considered as statistical significance.

![Figure 1](image.png)

**Figure 1.** Dynamics of cerebral oxygenation value of the right and left hemispheres during the course of surgery for chronic pulmonary thromboembolism, for both groups. (A) the ACP group, (B) the DHCA group. PTE, pulmonary thromboendarterectomy; CPB, cardiopulmonary bypass; rSO₂, %, cerebral oxygenation. \( *p < .05 \) was considered as statistical significance.
the right hemisphere differed significantly between the groups \((p = .03)\). These differences associated with the fact that circulatory arrest in the DHCA group of patients is accompanied by abrupt decrease of systemic arterial pressure and inevitably leads to pronounced cerebral hypoperfusion while in the ACP group cerebral circulation is supported by ACP.

During the cold reperfusion after PTE from the right pulmonary branch, \(rSO_2\) values for the left hemisphere were significantly higher \((p = .02)\) in the DHCA group than in the ACP group. This could have been related to a more profound decrease in cerebral metabolic demands under conditions of low body temperature and craniocerebral hypothermia.

Considering the ACP group during PTE to remove emboli from the left pulmonary branch, the decrease in \(rSO_2\) to the right and left hemispheres was greater (by 18% and 20%, respectively) than that observed during PTE from the right pulmonary branch \((p = .007\) and \(p = .04\), respectively) which would be due to incomplete correspondence of maintained ACP to cerebral metabolic demand. In addition, a reduction in \(rSO_2\) was observed in patients from the DHCA group for the right and left hemispheres (by 34% and 31%; \(p = .03\) and \(p = .02\), respectively) during PTE to remove emboli from the left pulmonary branch compared with the stage of removing emboli from the right pulmonary branch. More profound decrease of \(rSO_2\) value in the DHCA group during PTE to remove emboli from the left pulmonary branch is caused by the fact that there is more marked reduction in cerebral metabolic activity in the setting of prolongation of hypothermic condition. This leads to further increase of \(rSO_2\) values. Therefore, percent of decrease in \(rSO_2\) value for DHCA group members during repeated circulatory arrest is augmented due to both higher \(rSO_2\) values at the cold reperfusion stage and greater suppression of cerebrovascular adaptational and regulatory mechanisms under abrupt decrease of arterial pressure in major cerebral vessels.

The decrease in \(rSO_2\), expressed as a percentage of the value at the stage of cold reperfusion, was significantly greater in the DHCA group than in the ACP group for both right and left hemispheres \((p = .03\) and \(p = .04\), respectively). At the warming stage in the DHCA group, \(rSO_2\) values for the right and left hemispheres remained higher compared with the ACP group \((p = .002\) and \(p = .01\), respectively). This could be explained by the incomplete restoration of brain metabolic activity after DHCA and craniocerebral hypothermia.

A statistically significant inverse correlation was observed between nasopharyngeal temperature at different stages of surgery and \(rSO_2\) values; this could be explained by downregulation of cerebral metabolic demands during hypothermia. For this relationship the correlation values ranged from \(r = -.33\) to \(r = -.45\).

During anesthesia induction, glucose concentrations in the ACP group and the DHCA group were 5.6 (5.1–5.8) and 5.4 (5.0–5.9) mmol/L, respectively. Over time, at the complete warming stage after termination of CPB, we observed a statistically significant increase in blood glucose level for both groups, to values of 9.3 (8.0–10.6) mmol/L \((p = .007)\) and 10.7 (8.8–13.0) mmol/L \((p = .0001)\), respectively. Therewith, in the DHCA group, glucose level at the stage of complete warming was statistically significantly higher than in ACP group \((p = .029)\).

### Neurological Complications

The results of the univariate regression analysis showed that risk of neurological complications during the early postoperative period was dependent on the length of the period during PTE when absolute \(rSO_2\) values were <40%. For each one-minute increment in the duration of this period, the incidence of neurological complications increased by 8% \((OR, 1.08; 95\% CI, 1.00–1.16; p = .009)\).

Details of the clinical outcomes are shown in Table 2.

<table>
<thead>
<tr>
<th>Table 2. Postoperative complications and clinical course.</th>
</tr>
</thead>
<tbody>
<tr>
<td>First Group: 30 Patients, Antegrade Cerebral Perfusion Combined with Moderate Hypothermia</td>
</tr>
<tr>
<td>Neurologic complications: All</td>
</tr>
<tr>
<td>Encephalopathy</td>
</tr>
<tr>
<td>Acute stroke</td>
</tr>
<tr>
<td>Ventilation time, hour</td>
</tr>
<tr>
<td>Lung reperfusion syndrome</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td>Cardiac pacing</td>
</tr>
<tr>
<td>Inotropic support</td>
</tr>
<tr>
<td>Intensive care unit, days</td>
</tr>
<tr>
<td>Hospital stay, days</td>
</tr>
<tr>
<td>In-hospital mortality</td>
</tr>
</tbody>
</table>

*\(p < .05\) was considered as statistical significance.
In the ACP group, neurological complications were observed in 7% of cases during the early postoperative period. One patient was diagnosed with moderate post-hypoxic encephalopathy (composite score by MMSE scale was 26) and the other with an acute cerebrovascular event (ACE) in the left and right carotid arterial territories. In the latter patient, a reduction in rSO2 to <40% was recorded during PTE. In the ACP group, a reduction in rSO2 values to <40% was recorded only in three patients, a statistically significantly lower incidence than in the DHCA group (10% vs. 35%, \( p = .01 \)).

In the DHCA group, neurological complications were observed in 29% of cases. An ACE was identified in two patients and was recorded for the left and right carotid arterial territories. Moderate post-hypoxic encephalopathy was recorded in seven patients. Neurological disorders included disturbances of higher mental functions at the clinical level, such as psychomotor agitation, spatial and temporal disorientation, personality disorder, noncritical thinking regarding self-sentiment and the environment. According to the MMSE scale for this patient group, mean composite score was 26 (24–27). The consciousness score was 15 on the Glasgow Coma Scale. In this group of patients, 11 subjects (35%) demonstrated a reduction of absolute rSO2 values to below the critical limit (<40%) during the course of PTE, and these subjects included patients with neurological abnormalities.

In the ACP group, a lethal outcome during the early postoperative period was recorded in one 40-year-old female patient; the cause of death was multiple organ failure combined with ACE affecting the left and right carotid territory. For this case, during PTE we recorded absolute rSO2 values <40%.

In the DHCA group, three lethal outcomes were recorded (two women, one man). Two lethal outcomes were recorded in 30-year-old and 48-year-old female patients, due to multiple organ failure combined with brain coma. In both those patients we observed absolute rSO2 values <40% during PTE. Another lethal outcome was recorded in a 48-year-old male patient, due to multiple organ failure complicated by disseminated intravascular clotting syndrome. In this patient we did not observe any pronounced decrease in rSO2 indices during PTE.

**DISCUSSION**

The main finding of this study is that the use of ACP combined with moderate hypothermia for pulmonary end-arterectomy offers better cerebral protection against ischemia than the use of DHCA combined with craniocebral hypothermia. Furthermore, the use of ACP reduces the risk of neurological complications in the early postoperative period.

During circulatory arrest, arterial pressure in the major cerebral vessels decreases dramatically. In our study, pronounced decrease of cerebral perfusion during circulatory arrest was reflected by significant reducing of rSO2 readings compared with preceding values. Under conditions of hypoperfusion, the distal cerebral vascular regions are most vulnerable, and ischemia in these vascular regions occurs earlier and has more severe manifestations. Carbon dioxide accumulates and acidosis is established. The accumulation of suboxidized metabolites causes edema, which may cause cerebral perfusion abnormalities.

Clinical studies show that a reduction in rSO2 values by more than 30% from baseline contributes to the development of post-hypoxic ischemia, leading to neurological dysfunction.

Deep hypothermia reduces the cerebral metabolic demands, whereas the requirement for nutrients is maintained (17,18). In our study, we noted a greater increase in rSO2 values during CPB in patients who underwent surgery under conditions of deep hypothermia compared with patients who underwent surgery under conditions of moderate hypothermia. Despite deep body cooling and craniocebral hypothermia, the most profound reduction in cerebral oxygen supply during PTE (by more than 30% from baseline) was recorded under conditions of circulatory arrest.

Regression analysis showed that the duration of the period during which absolute rSO2 values were <40% in the course of surgery was associated with the progression of neurological disorders. In the DHCA group, a reduction of rSO2 values to <40% occurred more frequently compared to the group of patients who underwent PTE under ACP combined with moderate hypothermia. Consequently, the former group had a larger proportion of patients with neurological complications.

After the main stage of surgery, attention should be paid to blood glucose levels, because prolonged CPB, hypothermia, and the massive release of anti-insulin hormones contribute to the progression of acidosis. In this study we observed an increase in blood glucose levels in both groups and marked hyperglycemia in patients who underwent PTE with DHCA.

Recent studies conducted to determine a safe duration for DHCA have considered essentially only aortic surgery. Some authors maintain that limitation of the DHCA period is important for several reasons. For elderly patients, duration of DHCA longer than 25 minutes may cause transient neurological dysfunction, mild motor deficit, and contribute to a prolonged hospital stay. A still more prolonged period of circulatory arrest may be connected with functional insufficiency of other organs: e.g., postoperative hepatic and kidney disorders (19). Some studies found that DHCA without cerebral perfusion is a safe method of cerebral protection if the duration of arrest is less than 30 minutes (20). Other authors concluded that the majority...
of aortic surgeries can be performed under circulatory arrest with a duration no longer than 40 minutes (21).

In our study, the total duration of circulatory arrest for PTE to remove emboli from the pulmonary branches was no more than 40 minutes; however, the duration of circulatory arrest was not associated with the risk of neurological disorders. The difference in the surgical approach in relation to the need for reperfusion must also be taken into consideration, not only after the circulatory arrest, but also during the period between the PTE procedures to remove emboli from the right and left pulmonary branches. The use of DHCA is accompanied by the progression of cerebral ischemia, with subsequent ischemia–reperfusion damage. All these factors result in a loss of blood–brain barrier integrity and effusion of liquid into the cerebral tissue (22,23). Taking into account particularity of surgery method including at least two deep hypothermic circulatory arrests during PTE for patients with chronic pulmonary thromboembolism, it should be remembered about deterioration of cerebrovascular adaptational and regulatory mechanisms. It has been reflected in our study as more profound decrease of rSO2 values during repeated circulatory arrest.

Presently, there is no consensus regarding the selection of the best method of cerebral protection. Studies of retrograde cerebral perfusion have determined limitations of this method, such as the impossibility of defining the optimal blood volume for the supply of brain tissue through the inferior vena cava. However, an undeniable advantage of this method is the possibility of washing away microscopical emboli of biologic aggregates and air bubbles from small-caliber cerebral arteries under conditions of cerebral vascular embolism (24).

ACP helps to extend the “safe” period of an operation without deep hypothermia, because it provides 1) metabolic supply directed to the brain during circulatory arrest, 2) removal of acidic metabolites released by the process of anaerobic metabolism, and 3) removal of activating neurotransmitters involved in the pathogenesis of ischemic brain damage (25,26).

Further research of patients with chronic pulmonary thromboembolism who were operated using different methods of cerebral protection during surgical treatment is essential because that allows us to find new solutions to improve cerebral protection including not only technical equipment but use of medications to improve patients life quality. Taking into account changes in cerebral oxygenation during intraoperative period, it is possible in the future to evaluate the long-term results of surgery for this cohort of patients and to trace dynamics of cerebral complications as well as survival rates.

This study has several limitations. First, authors have not conducted analysis of dynamics of lactate concentration collected from jugular vein at various stages of the surgery. Second, we have not considered neurospecific markers associated with cerebral tissue injury. Because of a relatively small number of patients in every group and age variability in both groups, which could make a contribution to postoperative complications, there is a need to expand the samples of patients. Those measures allow elucidating wider spectre of predictors of postoperative complications including taking into account various comorbidities and age factor.

This study demonstrates that ACP during the course of PTE is an effective method of cerebral protection that is well adjusted to human physiology. The use of ACP in the treatment of chronic pulmonary thromboembolism obviates the need for DHCA. This method entails a lesser reduction in oxygen supply during PTE and substantially decreases the risk of ischemia and neurological deficits affecting the central nervous system.

REFERENCES