Quantification and Distribution of Cerebral Emboli during Cardiopulmonary Bypass in the Swine

The Impact of \( P_{a}CO_2 \)

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Background: Patients undergoing cardiac surgery have a substantial incidence of neurologic complications related to cerebral embolization during cardiopulmonary bypass. The purpose of this study was to determine if adjustments in the arterial carbon dioxide (\( P_{a}CO_2 \)) level can reduce cerebral and ocular embolization.

Methods: Twenty pigs underwent cardiopulmonary bypass at 38°C. At either hypercarbia (\( P_{a}CO_2 = 50–55 \) mmHg, group II, \( n = 10 \)) or hypocarbia (\( P_{a}CO_2 = 25–30 \) mmHg, group I, \( n = 10 \)), an embolic load of \( 1.2 \times 10^4 \) 67-μm orange fluorescent microspheres was injected into the aortic cannula. Before and after embolization, cerebral and ocular blood flows were determined at normocapnia using 15-μm fluorescent microspheres. After cardiopulmonary bypass was completed, the eyes were enucleated and brain tissue samples were collected. Microspheres were isolated and the fluorescence was measured.

Results: In groups II and I, the mean \( P_{a}CO_2 \) values at embolization were 52 ± 3 mmHg and 27 ± 2 mmHg, respectively (\( P < 0.0001 \)). Total and regional embolization were significantly less in hypocapnia than in hypercapnic animals: 14%±2% more emboli were detected in the brain in group II than in group I (\( P < 0.0001 \)). Cerebral blood flow after embolization was unchanged in both groups. Similarly, fewer ocular emboli occurred in hypocapnic animals than in hypercapnic animals (\( P = 0.044 \)), but in contrast to the brain, ocular blood flow decreased significantly in both groups after embolization.

Conclusions: Cerebral embolization is determined by the \( P_{a}CO_2 \) at the time of embolization. In cardiopulmonary bypass practice, reductions in \( P_{a}CO_2 \) during periods of embolic risk may reduce the risk for brain injury. (Key words: Brain; carbon dioxide tension; embolization; extracorporeal circulation.)

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CENTRAL nervous dysfunction can manifest as stroke, encephalopathy, or cognitive dysfunction after cardiopulmonary bypass (CPB). Depending on the patient population, 1% to 9% of patients may experience stroke or encephalopathy, and as many as 50% may have more subtle disorders of memory, vision, language, or coordination.1–5 The causes of this dysfunction are multifactoral and include regional cerebral hypoperfusion and cerebral embolization.2,4,5 Doppler ultrasonography can detect hundreds of embolic events during CPB,6–9 and cerebral embolization as measured by transcranial Doppler sonography is a predictor of brain injury after cardiac surgery.5,10 Importantly, cerebral embolization occurs during specific CPB surgical events,7–10 so simple physiologic interventions to reduce cerebral blood flow (CBF) during periods of embolic risk may reduce the extent of brain injury. Most embolization occurs with aortic cannulation, the onset of CPB, and after release of the aortic clamp and during the early phases of ventricular ejection.7–9 One practical intervention would be to decrease the arterial carbon dioxide tension (\( P_{a}CO_2 \)) during these periods. The aim of our study was to evaluate the influence of \( P_{a}CO_2 \) on regional cerebral and ocular embolization during normothermic CPB.

Methods

After we received approval from our institutional animal care and use committee, we studied 20 pigs that weighed 22 ± 2 kg. Pigs were given intramuscular injections of 4 mg/kg Telazol (Parke-Davis, Morris Plains, NJ) and 2 mg/kg Xylazine (Bayer, Shawnee Mission, KS). Anesthesia was induced using halothane, 2%, by mask and the pig tracheas were intubated. Peripheral intravenous access was secured and muscle relaxation was induced using 0.1 mg/kg pancuronium administered intravenously. Ventilation was controlled to maintain the \( P_{a}CO_2 \) level at 35 to 40 mmHg and an arterial oxygen
tension at 150 to 250 mmHg. Anesthesia was maintained with continuous intravenous infusions of 0.7 μg·kg⁻¹·min⁻¹ fentanyl and 28 μg·kg⁻¹·min⁻¹ ketamine. Pancuronium (0.5 μg·kg⁻¹·min⁻¹) was administered to provide muscle relaxation.

Four-inch 18-gauge cannulas were inserted surgically into a femoral artery for mean arterial blood pressure measurements and blood sampling. For CPB, a left-sided thoracotomy was performed. The bypass machine was primed with one unit of pig blood (~250 ml) and 750 ml 6% dextan 70. Venous drainage to the extracorporeal circuit was by a 36-French gauge cannula placed in the right atrium via the right atrial appendage. The blood was circulated by a centrifugal pump through a combined heat exchanger-oxygenator (Bentley Spiral Gold, Irvine, CA) and returned via a cannula (4.5-mm inner diameter) into the root of the aorta.

Cardiopulmonary bypass was performed, and nasopharyngeal temperature, measured with a thermocouple, was maintained at 38°C, hemoglobin at 7 to 8 g/dl, PaCO₂ at 35 to 40 mmHg, and arterial oxygen tension at 150 to 250 mmHg. The mean arterial blood pressure was maintained at 65 to 75 mmHg by altering the bypass pump flow rate. No vasoconstrictors or vasodilators were used. When steady state CPB conditions (as already defined) were reached, a preembolization CBF was determined. Afterward, the PaCO₂ was increased or reduced progressively in 30 min to reach target values of 50 to 55 mmHg (group H, n = 10) or 25 to 30 mmHg (group L, n = 10), respectively. Before the start of the experiment, pigs were assigned randomly to one of the groups. When the PaCO₂ had stabilized, the embolic load was administered progressively in 5 min. Approximately 30 min after embolization when PaCO₂ was normalized, a postembolic CBF was measured.

Cerebral blood flow measurements were performed with 15-μm red (excitation/emission wavelengths: 580/605 nm) and yellow-green (505/515 nm) fluorescent-labeled polystyrene microspheres (Molecular Probes, Eugene, OR), using the blood reference sample method. Four million microspheres (4 ml) were diluted in 6 ml 6% dextan 70 with 0.025% Tween 80, sonicated, vortexed, and microspheres were injected progressively in 60 s into the aortic inflow line distal to the arterial line filter. Beginning 30 s before microsphere injection, a reference blood sample was obtained in a period of 4 min. Blood was drawn from the femoral artery catheter into a glass syringe by a Harvard withdrawal pump at a rate of 4.9 ml/min. This was transferred into labeled vials, carefully rinsing syringes and extension lines. The embolic load consisted of 1.2 × 10⁵ 67-μm orange (540/560 nm) dyed fluorescent polystyrene microspheres (Molecular Probes). These microspheres (1 ml) were diluted in 9 ml 6% dextan 70 with 0.025% Tween 80, sonicated, and rigorously vortexed and injected progressively in 5 min into the aortic inflow line. After each injection, the syringe containing the microspheres was flushed with 20 ml 6% dextan 70.

After completion of the experiment, CPB was terminated, pigs were exsanguinated, and the brains were removed. Tissue samples (~1 g) of left and right temporal and occipital lobes, thalamus, internal capsule, and cerebellar hemispheres were obtained. Animals were enucleated by cutting the optic nerve at its entrance to the eyeball. Surrounding tissue (muscle and fat) was removed and the ocular bulb was processed as a whole. Blood and tissue samples were allowed to autolyze in the dark for 10 to 14 days. Thereafter microspheres were recovered by the sedimentation method.11 Tissue samples were digested in 2 ml ethanolic potassium hydroxide with 0.5% Tween 80 at 50°C for 48 h. Homogenized tissue samples were centrifuged (20 min at 2,000g) and the supernatant was removed carefully. One percent Triton X-100 (Sigma Chemical Co., St. Louis, MO) was added to each sample. Tubes were centrifuged again, the supernatant was removed, and the pellet was rinsed with phosphate buffer and distilled water. After a final centrifugation, 2-ethoxyethyl acetate (Aldrich Chemical, Milwaukee, WI) was added to the pellet to separate the fluorescent dyes from the microspheres. The recovery of microspheres from reference blood samples followed a commercially available protocol (NuFlow Extraction Protocol 9507.2, Interactive Medical Technology, West Los Angeles, CA). Blood and tissue samples (in 2-ethoxyethyl acetate) were placed in the dark for 5 days.

The intensity of fluorescence in tissue and blood samples was determined using a spectrofluorometer (SLM 8100; SLM-AMINCO, Rochester, NY). The fluorescence of each sample was measured at its specific excitation-emission wavelength. The optimal excitation—emission wavelength of each color was determined before each period of spectrofluorometric analysis. Cerebral blood flow was calculated from the intensity of fluorescence in the blood and tissue samples using the following formula:

\[
\text{CBF (ml·g}^{-1}·\text{min}^{-1}) = \frac{(R·I_R)·(W_t)}{I_R·W_t}
\]

where \( R \) = rate at which the reference blood sample was withdrawn (4.9 ml/min), \( I_R \) = the fluorescence intensity of the tissue sample, \( I_R \) = the fluorescence intensity of
Table 1. Systemic Physiologic Values of Both Groups for the Three Study Periods

<table>
<thead>
<tr>
<th>Study Period</th>
<th>Group</th>
<th>Temp (°C)</th>
<th>MAP (mmHg)</th>
<th>Pump Flow (L.min⁻¹.m⁻²)</th>
<th>Hgb (g.dl⁻¹)</th>
<th>PaCO₂ (mmHg)</th>
<th>PaO₂ (mmHg)</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preembolization</td>
<td>H</td>
<td>38.0 ± 0.2</td>
<td>69 ± 4</td>
<td>2.0 ± 0.6</td>
<td>7.5 ± 0.7</td>
<td>205 ± 52</td>
<td>36 ± 4</td>
<td>7.36 ± 0.07</td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>38.0 ± 0.2</td>
<td>70 ± 2</td>
<td>2.3 ± 0.8</td>
<td>7.8 ± 0.7</td>
<td>221 ± 53</td>
<td>37 ± 3</td>
<td>7.37 ± 0.05</td>
</tr>
<tr>
<td>Embolization</td>
<td>H</td>
<td>38.1 ± 0.3</td>
<td>67 ± 3</td>
<td>2.2 ± 0.4</td>
<td>7.4 ± 0.6</td>
<td>210 ± 68</td>
<td>52 ± 3*</td>
<td>7.24 ± 0.07*</td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>38.1 ± 0.1</td>
<td>68 ± 4</td>
<td>2.5 ± 0.6</td>
<td>7.6 ± 0.8</td>
<td>235 ± 55</td>
<td>27 ± 2</td>
<td>7.53 ± 0.04</td>
</tr>
<tr>
<td>Postembolization</td>
<td>H</td>
<td>38.0 ± 0.1</td>
<td>69 ± 4</td>
<td>2.2 ± 0.5</td>
<td>7.5 ± 0.8</td>
<td>222 ± 54</td>
<td>37 ± 3</td>
<td>7.38 ± 0.06</td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>38.0 ± 0.2</td>
<td>69 ± 3</td>
<td>2.3 ± 0.7</td>
<td>7.7 ± 0.9</td>
<td>211 ± 34</td>
<td>37 ± 3</td>
<td>7.39 ± 0.06</td>
</tr>
</tbody>
</table>

Values are mean ± SD (n = 10 in each group).
Temp = nasopharyngeal temperature; MAP = mean arterial blood pressure; Hgb = hemoglobin.
* P < 0.05 between groups by repeated measures ANOVA.

the blood sample, and Wt = the weight of the tissue sample (in grams).

Ocular blood flow was determined according to a similar procedure and is expressed as ml · 100 g⁻¹ · min⁻¹. This represents the blood flow to the whole ocular bulb without distinguishing between structures with high flow (choroid), low (sclera), or no flow (corpus vitreum).

To determine the amount of 67-μm orange fluorescent microspheres per tissue sample, a standard curve with known concentrations of orange microspheres was constructed. The relation between the intensity of fluorescence and microsphere number is essentially linear in dilute samples. Before each spectrophotometric analysis, a standard curve was constructed by analyzing serial dilutions of 67-μm orange fluorescent microspheres. The fluorescence intensity of the tissue sample was determined and the standard curve was defined by the following equation: I = m · C + b.

From this equation the concentration of microspheres was calculated as C = (I - b)/m

where C = the concentration (number of microspheres/ml solvent), I = the fluorescence intensity of the tissue sample, b = the Y intercept, and m = the slope.

**Statistical Analysis**

Systemic physiologic data for the preembolization, embolization, and postembolization periods were analyzed using two-factor repeated-measures analysis of variance. For these models the physiologic variable was the dependent variable, treatment group was the independent cross-classification factor, and time was the repeated factor. Linear regression was used to test the adequacy of microsphere mixing and equal distribution of microspheres between the left and right sides of the brain. To determine whether the regression line was different from the identity line, we performed individual t tests of intercept = 0 and slope = 1 along with the simultaneous F test for the identity line. All subsequent analyses for regional CBF and embolization were performed using the mean across the left and right sides of the brain. Regional blood flow and embolization values for the occipital and temporal lobes were combined and are presented as values for the neocortex. In addition, the mean numbers of emboli delivered to the circumferential arterial territories (neocortex and cerebellum) and the penetrating arterial territories (internal capsule and thalamus) were determined. For each brain region, preembolistic and postembolistic blood flows were compared using the paired t test, and the two treatment groups were compared using the two-sample t test. The two-sample rank-sum test was used to compare embolization between groups H and L. In addition, with PaCO₂ treated as a continuous variable, linear regression was used to assess the association of embolization and PaCO₂. All data are presented as the mean ± SD. A P value < 0.05 was considered significant.

**Results**

Table 1 shows the systemic physiologic data for the three study periods. With the exception of the controlled variable, PaCO₂ (and pH), physiologic variables did not differ within or between groups during any of the three study periods.

Cerebral blood flow and embolization values did not differ between the left and right brain regions. Paired left and right preembolic (r = 0.98) and postembolic (r = 0.98) CBF values and the emboli counts (r = 0.93) were well matched (Y intercepts not significantly different

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Table 2. Cerebral and Ocular Blood Flow before and after Embolization

<table>
<thead>
<tr>
<th>Region</th>
<th>Group H (ml · 100 g⁻¹ · min⁻¹)</th>
<th>Group L (ml · 100 g⁻¹ · min⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Preembolic Flow</td>
<td>Postembolic Flow</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neocortex</td>
<td>61 ± 17</td>
<td>52 ± 18</td>
</tr>
<tr>
<td>Internal capsula</td>
<td>32 ± 9</td>
<td>33 ± 15</td>
</tr>
<tr>
<td>Thalamus</td>
<td>55 ± 15</td>
<td>57 ± 30</td>
</tr>
<tr>
<td>Cerebellum</td>
<td>60 ± 17</td>
<td>58 ± 22</td>
</tr>
<tr>
<td>Mean brain</td>
<td>54 ± 14</td>
<td>50 ± 17</td>
</tr>
<tr>
<td>Eye</td>
<td>43 ± 18</td>
<td>27 ± 11*</td>
</tr>
</tbody>
</table>

Values are mean ± SD (n = 10 in each group).

* P < 0.05 by paired t test, comparing preembolization to postembolization flow.

from 0, slope not significantly different from 1), indicating adequate mixing of microspheres in the aortic root. Therefore, only the mean values for regional blood flows and embolization are presented.

Before embolization, at normocapnia CBF did not differ between the groups (table 2). For all brain regions measured, the mean preembolic CBF was 54 ± 14 ml · 100 g⁻¹ · min⁻¹ in group H and 49 ± 15 ml · 100 g⁻¹ · min⁻¹ in group L. Predicted differences in blood flow between brain regions such as gray and white matter structures were shown in both groups (neocortex \(vs\). internal capsula: \(P < 0.0001\) for groups H and L; table 2).

At the time of embolization, the mean \(P_{aCO_2}\) was 52 ± 3 mmHg in group H and 27 ± 2 mmHg in group L (table 1). This resulted in more emboli in all brain regions in group H (fig. 1). The mean number of emboli per gram of sampled brain was 46 ± 20 in group H \(versus\) 19 ± 7 in group L (\(P < 0.0001\)). A strong correlation existed between the number of emboli delivered to the brain and the \(P_{aCO_2}\) level (\(r = 0.72, P < 0.001\); fig. 2). As the mean brain weight was 69 ± 4 g, the estimated total count of emboli delivered to the brain was 1,300 to 3,200 emboli (1% to 3% of the emboli injected into the aortic root). In both groups, more emboli were detected in brain regions with a high preembolic CBF than in brain regions with a low pre-embolic CBF. Within groups, more emboli were found in circumferential arterial territories (neocortex and cerebellum) than in penetrating arterial territories (internal capsule and thalamus) (fig. 3). In group H, the mean number of emboli per gram was 62 ± 25 in circumferential and 22 ± 15 in penetrating arterial territories (\(P < 0.01\)). In group L, the mean number of emboli per gram was 27 ± 10 in circumferential and 8 ± 3 in penetrating arterial territories (\(P < 0.01\)). Trapping of emboli in the circumferential territories is evident in the data for the neocortex and thalamus. Before embolization, the two tissues had equivalent blood flows (table 2), but embolization to the thalamus represents one half the embolization to the neocortex (fig. 1). Similarly, internal capsular blood flow is approximately one half that of the flow of the neocortex, and embolization to the internal capsule is less than one quarter the embolization to the neocortex. These relations were observed in both groups (table 2, fig. 1). Embolization did not alter postembolic CBF in either group (fig. 1). The mean global postembolic CBF was 50 ± 17 ml · 100 g⁻¹ · min⁻¹ in group H and 45 ± 12 ml · 100 g⁻¹ · min⁻¹ in group L. Neither global nor regional CBFs decreased significantly after embolization, and the groups did not differ in their CBF values after embolization (table 2).

During the experiment, ocular blood flow did not differ between the groups (table 2). At normocapnia, preembolic ocular blood flow was 43 ± 18 ml · 100 g⁻¹ · min⁻¹ in group H and 37 ± 12 ml · 100 g⁻¹ · min⁻¹ in

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*Fig. 1. The regional distribution of cerebral emboli in group H (■) and in group L (□). Values are the mean ± SD emboli per gram (n = 10 in each group). \(P < 0.05\).*
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ocular blood flow decreased in both groups after embolization. In groups H and L, ocular blood flow decreased by 37% and 32%, respectively, after embolization (table 2).

Discussion

Cerebral embolization is thought to be a primary cause of neurologic injury after cardiac surgery.1,2,4,5 Cerebral emboli have been detected in 100% of patients undergoing CPB1, and recent reports indicate that total cerebral embolization is an important predictor of cognitive dysfunction.5,10 Technical interventions such as membrane oxygenators11 and arterial in-line filters5,6 probably decrease the number of cerebral embolic events. Physiologic interventions also could be relevant. It has been suggested that increases in CBF, as occur with pH-stat management or normothermic CPB, may result in a greater incidence of cerebral embolization.11,15 We might also predict that decreases in CBF during periods of embolic risk might reduce total cerebral embolization.

The fundamental importance of PaCO₂ in the cerebral vasculature is well described. With out without bypass, a change of 1 mmHg in PaCO₂ results in a change in the CBF of approximately 3% or 4%.16,17 In CPB practice, the debate about the importance of PaCO₂ has focused primarily on the advantages or disadvantages of α-stat or pH-stat management during hypothermia.14 However, our results suggest that PaCO₂ during bypass may have
broader implications and might be used specifically as a physiologic intervention to reduce the cerebral embolization during periods of embolic risk.

This is the first study to show clearly that the $\text{Pa}_{\text{CO}_2}$ significantly alters the magnitude of cerebral embolization. These findings have two important implications. First, the results favor the $\alpha$-stat approach. Although this study was conducted during normothermia, the findings are applicable to hypothermic bypass conditions because, during hypothermia, $\text{Pa}_{\text{CO}_2}$ remains a primary determinant of CBF\textsuperscript{14,16,18} and should have a similar effect on the total degree of cerebral embolization. However, the issue of $\alpha$-stat or pH-stat is of only modest interest for various reasons. pH-stat management is not used widely. Bypass temperatures are increasing, so the carbon dioxide solubility issue is less and less relevant. And most importantly, cerebral embolization occurs most often during late CPB, when the body temperature approximates normothermia.

The periods of embolic risk during cardiac operations are well characterized. Transcranial and carotid Doppler and transesophageal echocardiography indicate that most embolization occurs at the time of aortic cannulation, at the onset of CPB, after release of the aortic clamp, and during the early phases of ejection after cross-clamp removal.\textsuperscript{7–9} After release of the aortic cross clamp, as many as 50% of all the Doppler-detected emboli may occur.\textsuperscript{7–9} Second, our findings indicate that a modest reduction in $\text{Pa}_{\text{CO}_2}$ before the release of the aortic cross clamp could reduce greatly the amount of emboli delivered to the brain. Although hypocapnia vasocostriction may reduce cerebral oxygen delivery under conditions of hypotension,\textsuperscript{19} we speculate that the risk from hypocarbria-associated reductions in blood flow would be offset by a reduction in cerebral embolic load. In addition, experimental work indicates that hypocapnia alone ($\text{Pa}_{\text{CO}_2} = 21$ or 22 mmHg) does not reduce CBF to a point that is likely to produce ischemia, even during induced hypotension.\textsuperscript{19,20} Of greater concern than inducing cerebral ischemia with aggressive ventilation might be the development of respiratory alkalosis and hypokalemia; however, with current oxygenators, $\text{Pa}_{\text{CO}_2}$ can be reduced and normalized rapidly, and because the period of embolic risk is relatively brief, these concerns should be of little consequence. Although hypocarbria may be associated with a certain transient physiologic changes, ultimately the potential benefits of reducing cerebral embolization with selective hypocarbria are probably justified.

Although the $\text{Pa}_{\text{CO}_2}$ determined the number of emboli delivered to the brain, it did not alter their distribution. Regional embolization is a function of tissue blood flow and embolus size. To a large extent, brain structures with the highest blood flow levels also receive the greatest number of emboli. In addition, larger emboli are more likely to be trapped in the circumferential arteries,\textsuperscript{21} so structures supplied by penetrating arteries may have an embolization that is low relative to their flow. This is evident in the data for the neocortex and thalamus. Before embolization, the two tissues have equivalent blood flows, but embolization to thalamus is one half the embolization to neocortex. Similarly, internal capillary blood flow is approximately one half the blood flow of the neocortex, but embolization to the internal capsule is less than one quarter the embolization to the neocortex. These data reflect the independent effect of vascular territory on regional embolization. The results also indicate that, at moderate levels of embolization, global CBF can be maintained. Although more cerebral emboli were detected in the brains of animals exposed to hypocarbria than in those exposed to hypocarbria, neither group showed significant decreases in their postembolic CBF. This indicates that the number of microspheres used in this cerebral embolization model was not overwhelming. We would speculate that global CBF may be maintained after moderate embolization, because the cerebral hemispheres contain a network of arteriolar collaterals, so shunting of blood may help protect the brain from the consequences of embolization.

However, this result would not be expected with a greater total embolization or obstruction of major arteries or arterioles by large emboli. Our findings on ocular embolization are also valuable. A significant portion of patients may experience visual abnormalities after cardiac surgery, and a greater proportion may have occult visual field defects.\textsuperscript{2} This can be explained readily by ocular embolization. In a small but sophisticated clinical study using retinal fluorescein angiography, Blauth \textit{et al.}\textsuperscript{1} found that all patients undergoing CPB probably experience transient retinal microvascular occlusions resulting from embolization.

The eye is supplied by the ophthalmic artery, with flow distributed primarily to the choroid and in small part to the retina.\textsuperscript{22} However, both the choroid and the terminal branches of the central retinal artery must be intact to maintain retinal activity. As in the brain, $\text{Pa}_{\text{CO}_2}$ is a primary determinant of uveal and retinal blood flow,\textsuperscript{22} and we found that ocular embolization can be reduced significantly by decreases in $\text{Pa}_{\text{CO}_2}$ during embolization. However, in contrast to the brain, both groups showed...
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alter their distribution within the cerebral circulation, emboli size would probably not influence the total amount that enters the cerebral circulation. It is reasonable to expect that the dominant effect of $P_{a\text{CO}_2}$ on CBF would affect a wide range of emboli types and sizes to a similar extent.

In conclusion, our study shows clearly that $P_{a\text{CO}_2}$ is an important determinant of cerebral embolization. A high $P_{a\text{CO}_2}$ increases the number of cerebral and ocular emboli, and a low $P_{a\text{CO}_2}$ decreases the numbers. Thousands of emboli may enter the cerebral circulation during CPB, and technical interventions can alter the total degree of cerebral embolization. Our study proves that a physiologic intervention, the manipulation of the $P_{a\text{CO}_2}$, can further attenuate cerebral embolization. This has important implications for clinical CPB practice. During periods of embolic risk, a moderate reduction in $P_{a\text{CO}_2}$ may decrease the number of cerebral emboli. This simple intervention has the potential to improve neurologic outcome after cardiac operations.

References


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