Deep hypothermia and circulatory arrest for surgery of complex intracranial aneurysms

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Abstract

Objective: Some intracranial aneurysms may not be operable by conventional neurosurgery due to their location or morphology. Cardiopulmonary bypass (CPB) and deep hypothermic circulatory arrest renders surgery of these complex aneurysms possible. Brain temperatures can be measured directly in this setting. Methods: Eight patients with complex intracranial aneurysms were operated on with the aid of CPB. Femoro-femoral bypass with heparin-coated circuit components was used in all cases. Venous drainage was augmented by a centrifugal pump in six patients and by a newly developed vacuum technique in two patients. Temperatures were monitored by probes in brain, tympanum, nasopharynx, bladder, rectum, arterial and venous blood. These measurements were recorded on-line together with those of cerebral oxygen saturation, AP, CVP and PAP. Blood gas analyses and an EEG were also performed continuously. Results: Outcome was excellent in seven patients, in one patient moderate neurological disability occurred. Mean time on cardiopulmonary bypass was 160 (117–215) min, for cooling to a brain temperature of 18°C 33 (20–47) min, and for total circulatory arrest 27 (15–45) min. Additionally, terminal brain arteries were clamped for up to 68 min in four patients. No cardiac complications were observed. Actual brain temperatures were best reflected by the tympanum probes (max. deviation 2°C), whereas temperatures measured in bladder or rectum exhibited deviations of up to 10°C. EEG activities were arrested between brain temperatures of 19 and 26°C. Conclusions: Complex intracranial aneurysms can be treated successfully using deep hypothermic circulatory arrest. Extensive monitoring adds to the speed and safety of the procedure. The resulting comparative measurements of temperatures at different body sites including brain, EEG, and other variables may be of general relevance for operations employing deep hypothermia and circulatory arrest. © 1998 Elsevier Science B.V. All rights reserved.

Keywords: Intracranial aneurysms; Extracorporeal circulation; Deep hypothermia; Brain temperature

1. Introduction

Treatment of some intracranial aneurysms is either not, or only with high risks, feasible by conventional neurosurgical or endovascular embolisation techniques. The complexity of these aneurysms may be due to calcification, partial thrombosis or size and location. This renders exposure and clipping of their neck dangerous or impossible without previous exsanguination or incision of the aneurysm sack. Surgery of these complex intracranial aneurysms may be performed using deep hypothermia and circulatory arrest [1–5]. These operations pose various problems to an interdis-
ciplinary team. For cardiac surgeons and perfusionists, careful performance of cardiopulmonary bypass (CPB) and coagulation management is of primary importance. Intraoperative exposure of the brain creates unique possibilities for monitoring during the different phases of operations with deep hypothermia and circulatory arrest. We report on a consecutive patient series operated on at our institution with recently accessible refinements of CPB techniques.

2. Patients and methods

Eight patients were diagnosed by preoperative radiological examinations (6 patients) or during a previous exposure (2 patients) to suffer from intracranial aneurysms not amenable to endovascular embolisation or conventional neurosurgery. Pertinent data relating to patients and aneurysms are shown in Table 1. All patients received preoperatively, in addition to the usual neurosurgical work-up, a transthoracic echocardiography and laboratory examinations for exclusion of aortic insufficiency and preformed cold agglutinines, respectively.

Anesthesia was induced by fentanyl, etomidate and pancuronium; for maintenance, isoflurane was added. Leads for ECG, EEG, transcranial measurement of oxygen saturation (Invos 3100, Somanetics Medilab, Estenfeld, Germany) and external defibrillation pads were placed. Temperature probes were positioned in tympanum, nasopharynx, bladder and rectum. Hemodynamic monitoring included a radial artery cannula and a pace port Swan-Ganz catheter. The head, thorax, abdomen and both groins were prepped and draped.

The aneurysm was exposed as far as safely possible by the neurosurgeon and a needle probe was inserted for direct measurement of brain temperature. Then aprotinin was administered (2 Mio units before and after CPB, 1/2 Mio during each hour of CPB). Heparin was given for an ACT >450 s (Kaolin tube) and the femoral cannulae were placed using surgical vessel exposure. The tip of a long heparin-coated venous cannula (21 or 23 Fr, Medtronic Biomedicus, Eden Prairie, MN) was placed high into the right atrium. CPB was started slowly with a preset water temperature of 5°C and flow was increased to 2.6–3.0 l · min⁻¹ · m⁻² (Figs. 1 and 2). Thiopental was infused in a dose sufficient to achieve EEG burst suppression. In case of ongoing EEG activities at a brain temperature of ≈ 20°C, thiopental administration was repeated. Sodium nitroprusside was given in case mean arterial pressure exceeded 50 mmHg. Continuous arterial and venous blood gas analyses were performed (CDI 400, Sarns 3M, Tustin, CA) in the frame of a-stat pH management. Arterial pO₂ was adjusted to values between 120 and 150 mmHg. Heparin-coated circuit components were used in all cases (oxygenator: Quadrox Bioline LT, Jostra, Hirrlingen, Germany; arterial filter: CBM 40 Medtronic Carmeda, Anaheim, CA). Arterial and venous blood temperatures, pressures and flows were measured with appropriate probes (DP 38, Medtronic Biomedicus; DPT 6000, PVB Medizintechnik, Kirchsecon, Germany). In six patients, venous return was augmented with a centrifugal pump (Medtronic Biomedicus). Vacuum suction was applied to the venous reservoir by means of a precision vacuum regulator (Dräger, Lübeck, Germany) in two patients. Central venous pressure was adjusted to ≈ 0 mmHg (Fig. 1). When the hearts started to fibrillate, an iv potassium bolus of 20 to 40 mval was applied to induce cardiac arrest. If ventricular fibrillation returned before rewarming, potassium administration was repeated. Maximal cooling was performed with high pump flows until a brain temperature of 18°C was reached. Then CPB was stopped and the venous blood actively drained into the venous reservoir to allow for a bloodless field and collapse of the aneurysm. Exposure and clipping of the aneurysms was performed during one or several episodes of total circulatory arrest. To reduce the time of complete circulatory arrest, terminal brain arteries were clamped during the procedure in four patients. Intermittently, CPB was restarted to test for appropriate position of the clips using α-agonists to raise arterial blood pressure (Fig. 1). After final testing, the patients were rewarmed using full flow, lowering of vascular resistance by sodium nitroprusside, and maximum heat exchange capacity. To compensate for intraoperative hemodilution, a hemofilter integrated in the CPB setup was used. After termination of bypass at a bladder temperature of 37°C and protamine administration, residual blood in the circuit was concentrated in a cell saver and retransfused. Intraoperative measurements of the various parameters were recorded on-line.

3. Results

Data of CPB are shown in Table 2. In addition to total circulatory arrest, terminal brain arteries were clamped in four patients for a mean of 41 (range 33–68) min. Exposure and clipping of aneurysms was performed rather in repeated brief episodes of total circulatory arrest (mean number 3; range 1–6) and clamping of terminal brain arteries with ongoing CPB than in a single period of prolonged circulatory arrest. Even a short intermittent pump run restored decreased cerebral oxygen saturation (Fig. 1). Active venous drainage using a centrifugal pump or vacuum assistance rendered full flows possible throughout the operations. Cardiac arrest could be achieved by systemic cooling and potassium administration in all patients. No cardiac complications were observed and in six patients,
Table 1
Summary of patient data

<table>
<thead>
<tr>
<th>No.</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Clinical presentation</th>
<th>Aneurysm</th>
<th>Size (cm)</th>
<th>Morphology</th>
<th>Localisation</th>
<th>Previous exposure</th>
<th>Postoperative complications</th>
<th>Outcome GOS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>47</td>
<td>Hemianopsia, vertigo</td>
<td>3</td>
<td>Saccular/fusiform</td>
<td>Left ICA (PCoA) aplasia of proximal ICA</td>
<td>No</td>
<td>None</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>45</td>
<td>Headache, diplopia, acute hydrocephalus</td>
<td>3.5</td>
<td>Saccular/fusiform</td>
<td>Left VA (PICA)</td>
<td>No</td>
<td>Surgical revision (epidural hematoma)</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>41</td>
<td>SAH, (3 times)</td>
<td>4.5</td>
<td>Saccular, partial thrombosis</td>
<td>Right MCA (bifurcation)</td>
<td>No</td>
<td>None</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>63</td>
<td>Hemiparesis, headache</td>
<td>3</td>
<td>Saccular/fusiform, partial thrombosis</td>
<td>Right PCA (P1-segment)</td>
<td>No</td>
<td>Small ischemic area (thalamic perforator)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>49</td>
<td>SAH</td>
<td>3.5</td>
<td>Saccular, trilobulated</td>
<td>BA (left SCA)</td>
<td>Yes</td>
<td>None</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>41</td>
<td>SAH</td>
<td>3</td>
<td>Saccular/fusiform, partial calcification</td>
<td>ACoA (fenestrated)</td>
<td>No</td>
<td>None</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>25</td>
<td>Headache</td>
<td>2</td>
<td>Saccular/fusiform, partial thrombosis</td>
<td>Right ACA (pericallosal artery)</td>
<td>Yes</td>
<td>Ipsilateral hemiparesis (complete remission)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>31</td>
<td>Hemianopsia</td>
<td>2.5</td>
<td>Saccular, partial calcification</td>
<td>Left ICA (ophthalmic artery)</td>
<td>No</td>
<td>None</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

ACA, anterior cerebral artery; ACoA, anterior communicating artery; BA, basilar artery; GOS, Glasgow outcome scale 6 months postoperatively; ICA, internal carotid artery; MCA, middle cerebral artery; PCA, posterior cerebral artery; PCoA, posterior communicating artery; PICA, posterior inferior cerebellar artery; VA, vertebral artery; SAH, subarachnoid hemorrhage.
Hearts resumed spontaneously normal sinus rhythm during rewarming. In two cases, external defibrillation was performed. For control of venous and cardiac filling, CVP proved to be more reliable than pulmonary artery pressure. This is demonstrated in Fig. 1 during the second episode of circulatory arrest; when using active venous drainage with negative CVP and an arterial pressure \( \sim 0 \) mmHg a mean PAP of 10 mmHg was measured.

In one patient, we observed a rapid increase in pressure drop across the arterial cannula during the cooling phase. The short arterial canula was replaced emergently by a long cannula reaching up into the aorta during a short period of circulatory arrest. No other problems with CPB were encountered in any of the patients. All patients were weaned without difficulties from CPB using minor to moderate doses of dopamine and sodium nitroprusside for blood pressure control.

During cooling, arterial blood temperature fell within a few minutes to 15°C (Fig. 2). Temperature gradients of up to 10°C between bladder or rectum and brain were observed (Fig. 2). Tympanic temperatures reflected best actual brain temperatures throughout the course of CPB with maximum deviations of 2°C. Nasopharyngeal temperatures were found to be second best in many patients in this respect, with deviations up to 4°C. However, in some cases, deviations occurred

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Table 2
Data of cardiopulmonary bypass (CPB)

<table>
<thead>
<tr>
<th></th>
<th>CPB (min)</th>
<th>Cooling (min)</th>
<th>Total circulatory arrest (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>160</td>
<td>33</td>
<td>27</td>
</tr>
<tr>
<td>Range</td>
<td>117–215</td>
<td>20–47</td>
<td>15–45</td>
</tr>
</tbody>
</table>

* Cooling time down to a brain temperature of 18°C.
which approximated those of bladder temperatures. EEG activities were arrested between brain temperatures of 19 and 26°C.

The clinical course of patients is summarized in Table 1. All patients were sedated until the morning of the first postoperative day when a CT-scan of the head was performed. A generalized brain edema was not observed in any of the patients. Edema at the site of surgical access was found to be not greater than in cases operated without CPB. In the second patient of our consecutive series, early revision for intracranial bleeding was required. Afterwards, all patients received 4–8 U of fresh frozen plasma after termination of CPB and protamine administration. With one exception, 2–3 U of red cells were given to the patients perioperatively, no other blood products were required. No wound healing problems, thromboembolic complications or renal insufficiencies were observed. Neurological outcome was excellent in seven patients according to Glasgow Outcome Scale (GOS 1) [6]. The patient who had required early revision for bleeding was moderately disabled at six months postoperatively (GOS 2) but lived independently.

4. Discussion

Deep hypothermic circulatory arrest as an adjunct for treatment of complex intracranial aneurysms is not a new technique and has been used for many years. [1–5,7]. The interdisciplinary effort and the side effects of cardiopulmonary bypass pose major challenges to the operative team and have hindered a more widespread use of this technique [8]. Increased perioperative bleeding and coagulopathy have been problems in the past and resulted in major complications leading to temporary abandonment by many centers [1,3,8]. Therefore, the main goal for cardiac surgeons and perfusionists has to be careful and diligent performance of CPB.

Groin cannulation is preferable over sternotomy because of reduced surgical trauma and wider separation of the different surgical fields [3,8]. This can only be accomplished if venous drainage is sufficient allowing enough flow and avoiding distention of the heart. Therefore, like others groups, we used an additional centrifugal pump in the venous line [4,5]. Recently we developed a technique to augment venous drainage by applying vacuum suction to the venous reservoir using a precision regulator. This also resulted in ample venous return and we did not observe any technical difficulties or differences compared to patients operated on with the aid of a centrifugal pump. We did not use intraoperative transesophageal echocardiography to monitor for cardiac distension as recommended by others [4,5,8]. However, cardiac distention has rarely been a problem in the past, when transesophageal echocardiography was not yet available [1]. None of our patients had preoperatively more than trace aortic insufficiency and we felt comfortable with our hemodynamic surveillance and augmented venous return. Additionally, the operating theatre appeared, even without echocardiography, more than crowded with personnel, instrument tables, monitoring devices, CPB and cell saving machinery. However, echocardiography may be useful in the presence of cardiac pathology.

One always has to be prepared to perform sternotomy if cardiac distention is suspected or in case of insufficient flow of the groin cannulae [8]. This was not required in any of our patients. In one case we experienced a rapidly increasing pressure drop across a short femoral artery cannula, possibly due to a hypothermic vascular spasm during cooling. Replacement with a long cannula designed for transthoracic placement which was advanced into the aorta immediately restored normal flow parameters. We now use this type of cannula routinely. For hemodynamic monitoring, particularly of venous return and cardiac filling, a Swan-Ganz catheter was always used. When we had observed an unexplained increased mean PAP value concomitantly with negative CVP and otherwise normal CPB hemodynamics for the first time, cardiac distension was suspected and external cardiac massage was performed. However, high PAP values persisted in spite of exsanguination and we attributed it to clinging of the catheter tip to the vascular wall. Since then, we encountered this phenomenon repeatedly without any interventions or overt consequences.

High pump flows were maintained throughout CPB. Prerequisites are liberal administration of nitroprusside and exact blood gas management. Nitroprusside is needed to counterbalance temperature related vasoconstriction during rapid cooling, thus allowing full pump flows and avoiding peaks of high blood pressure which could increase the risk of aneurysm rupture. During rewarming, it serves in decreasing arterial resistance and opening of low perfused vascular beds like the splanchnic area. With increasing temperatures and oxygen consumption, continuous measurement of venous pO2 with values > 35 mmHg protects against a mismatch of oxygen supply and demand [7]. Due to the course of the oxygen dissociation curve at low temperature, venous pO2 reflects more accurately oxygen consumption than venous oxygen saturation. Since arterial pO2 is kept between 120–150 mmHg, the risk of gaseous bubble formation during rapid rewarming is reduced. With increasing confidence in this set-up, CPB time can be shortened. Our last patient, a woman weighing 70 kg, was cooled in 20 min down to a brain temperature of 18°C, rewarming required 33 min. To avoid the possible adverse effects of prolonged circulatory arrest, the resulting decrease of cerebral oxygen...
saturation, and total body oxygen debt [7], repeated briefer episodes of circulatory arrest and clamping of terminal brain arteries were preferred to a single long episode of total circulatory arrest. This can only be accomplished in the context of a real team effort with close intraoperative and perioperative communication between team members.

Even small amounts of postoperative intracranial hemorrhage may be disastrous to patients. For control of bleeding, a meticulous operative technique by the neurosurgeon is essential. However, CPB related coagulopathy has often been a problem [1,3,8]. We used aprotinin with full systemic heparinisation which has been shown to be safe and effective in deep hypothermic circulatory arrest [9,10]. Since hypercoagulation has been described in conjunction with aprotinin and circulatory arrest, and the ongoing debate on the safety of the drug in this respect [11,12], we did not reduce heparin dosage in spite of the heparin-coated circuit components. Residual blood in the bypass circuit was washed by cell saver to avoid a heparin rebound after protaminisation. After we had observed one cranial revision for bleeding, fresh frozen plasma was administered to all patients and we did not encounter this problem again. No platelets or concentrated coagulation factors, as advocated by others [5], were given. Heparin-coated circuit components were used because of the reported reduction of coagulopathy and damage to blood cells [13,14]. In our own experience, heparin-coated oxygenators and arterial filters exhibit a lower increase of resistance during deep hypothermic perfusion compared to conventional devices and may therefore reduce direct blood trauma (unpublished data). This observation may be the hemodynamic correlative of decreased material adherence to heparin-coated surfaces [15].

For cerebral protection during circulatory arrest, actual brain temperatures are of pivotal importance. Like others [5,16], we observed large gradients between different body sites. The performance of neurological operations in deep hypothermic circulatory arrest offers the possibility of direct measurements of brain temperatures. In this setting, tympanum probes exhibited by far the best reflection of actual brain temperatures throughout the course of CPB. During rapid cooling, bladder and rectum temperatures appeared completely unreliable coming in some patients already down to 18°C while brain and tympanum were still at 28°C. Nasopharyngeal probes were second best in estimation of actual brain temperatures, however, in some patients larger differences occurred. This may be due to lack of direct contact to mucous membranes or dislocation during positioning of the head. If the probe comes to lie in close proximity to great vessels, this may result in measurements reflecting blood rather than brain temperature. During rewarming, increase of rectal temperatura was delayed, probably due to high vascular resistance of splanchnic vessels after profound hypothermia. Bladder temperature increased much faster and almost parallel to venous blood, since with returning urine output after termination of circulatory arrest, the bladder probe comes into contact with urine of plasma temperature. Therefore, we share the opinion [16] that multiple body temperatures have to be monitored in operations employing circulatory arrest after rapid cooling. This appears particularly true in the setting of femoral artery cannulation where until the onset of ventricular fibrillation, some blood of higher temperature may recirculate in the upper parts of the body. Otherwise, the low brain temperatures required for longer periods of circulatory arrest may actually not be reached. In our experience, the EEG was also not reliable in indicating low brain temperature. This may in part be due to our aggressive thiopental management. However, large doses of this drug may also be administered during operations on the heart and aorta employing deep hypothermic circulatory arrest.

Operations on complex aneurysms using deep hypothermia and circulatory arrest can be performed with relative safety. Key for success is a team effort with all partners prepared to respond to the special needs and problems of the other team members. Extensive monitoring adds to the speed and safety of the procedure. The resulting comparisons between different parameters may be useful for other cardiovascular operations employing deep hypothermia and circulatory arrest.

References


