Current perspective

Cerebral protection during surgery of the thoracic aorta: a review

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Operations on the aortic arch remain a major challenge for the cardiac surgeon and neurologic injuries represent the most feared complication. During the last decades, different cerebral protection techniques, including deep hypothermic circulatory arrest, and retrograde and antegrade cerebral perfusion have been introduced into clinical practice to reduce the incidence of such complications. All three methods present advantages and disadvantages. In this review, the theoretical impact of the current methods of brain protection is reported and discussed.

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The brain is an organ with a high-energy demand. Over 90% of the energy produced by the mitochondria in the brain is derived from oxygen and glucose carried by the circulation. Any decrease in oxygen causes a prompt fall in energy production and results in severe ischemic brain damage.

Since surgery of the aortic arch requires manipulation and exclusion of the cerebral circulation, brain protection is a primary concern and the utilization of optimal methods for the preservation of the cerebral function is necessary to avoid ischemic/embolic brain injuries.

It is suggested that two different forms of neurological injuries may occur after surgery of the thoracic aorta: permanent neurological dysfunction (PND) and transient neurological dysfunction (TND).

PNDs are defined as permanent focal or global neurological injuries clinically resulting in stroke or coma, respectively.

TNDs are defined as postoperative confusion, agitation, delirium, prolonged obtundation or transient Parkinsonism with a negative brain computed tomographic scan and complete resolution before discharge. However, more recent reports based on accurate neurocognitive testing before and after surgery have suggested that TND can no longer be considered as a benign self-limited condition, but rather as a long-lasting neurocognitive dysfunction capable of reducing the patients' quality of life by impairing memory and fine motor functions[1].

On the basis of the suggestions that TND is consequent to inadequate brain protection from ischemia during the period of aortic arch exclusion and that PND has an embolic origin[2] different strategies to protect the brain have been introduced in the last decade and have been continuously ameliorated in their clinical application. These include: a) methods based on the idea that when reducing cerebral oxygen consumption and/or maintaining cerebral blood flow (CBF), the cerebral tolerance to ischemia may be safely extended so as to exclude the cerebral circulation for the time required to perform the aortic arch reconstruction[3-5], and b) surgical techniques effective in reducing the embolic load to the brain by avoiding retrograde perfusion of the thoraco-abdominal aorta from the femoral artery[6-8].

Antegrade selective cerebral perfusion (ASCP) and deep hypothermic circulatory arrest (DHCA) with or without retrograde cerebral perfusion (RCP) are the methods currently employed for brain protection.

Alternative central arterial cannulation sites for the institution of cardiopulmonary bypass (CPB), such as the ascending aorta, right/left axillary artery, brachiocephalic trunk – aiming to avoid retrograde perfusion of the thoraco-abdominal aorta intended as a potential source of dislodged material destined to the cerebral circulation – have gained a wider consensus in many institutions and are very often preferred to the more traditional femoral artery[9,10].

The aim of the present article was to review the current strategies and techniques of cerebral protection employed during surgery of the thoracic aorta.

Historical notes

The first attempt to replace the transverse aortic arch for aneurysm was made by Schafer and Hardin[11] in 1952. During the procedure the aorta and the arch vessels were temporarily occluded keeping the lower body and the brain perfused by means of small bore polyethylene tubes. The patients did not survive the operation. Similar attempts[12] were subsequently made using different types of shunts. Unfortunately, the mortality was still unacceptably high mainly due to cerebral complications.

In 1955 Cooley et al.[13] introduced mild-surface-induced-hypothermia and temporary shunts extending from the ascending to the descending thoracic aorta with side arms to both the innominate and left common carotid arteries. These authors employed this technique to replace the transverse aortic arch in 2 patients. The first patient died of cerebral ischemia due to thrombosis of the innominate limb of the shunt on the sixth postoperative day. The second patient died of respiratory complications.

In 1957 DeBakey et al.[14] successfully replaced for the first time a transverse aortic arch using normothermic total CPB and cerebral perfusion involving several pumps and bilateral cannulation of both the subclavian and carotid arteries. The utilization of CPB with temporary or permanent shunts for cerebral arterial perfusion was then adopted by many surgeons but the mortality and the rate of adverse neurological outcome continued to be excessively high.

Shortly thereafter, on the basis of a series of investigations on adult dogs documenting profound inhibition of the cerebral metabolism at lower brain temperatures and of the successful utilization of profound hypothermia during complex congenital heart lesion repair, Pierangeli et al.[3] demonstrated that the use of DHCA for cerebral protection during aortic arch repair in adult patients was a far simpler and safer approach to the treatment of aortic arch pathology. The advantages of the hypothermic technique were immediately apparent and included a quiet, dry, motionless surgical field, which was unencumbered by temporary bypass grafts or perfusion cannulae. Excessive manipulation and dissection of the brachiocephalic vessels was no longer necessary. Since that time, the efficacy of DHCA has been widely accepted. However, the increasing utilization of DHCA revealed some of its limitations especially when longer durations for complex aortic arch repairs were required. In the largest clinical experience reported to date, Svensson et al.[15] demonstrated that patients treated with periods of circulatory arrest > 40 and > 65 min had an increased risk of stroke and early mortality, respectively.

Thus, in an effort to extend the safe period of hypothermic circulatory arrest, adjunctive methods of brain protection aimed at maintaining some oxygenated CBF have been recently explored. These include RCP and ASCP.

RCP was first described by Mills and Ochsner[16] in 1980 as a treatment for massive air embolism during CPB. In 1982, Lemole et al.[17] reported on the intermittent use of this technique during the repair of a dissected thoracic aorta. Intermittent and later continuous RCP was adopted by Ueda et al.[5] as a method of cerebral protection during procedures involving the aortic arch. Since then, RCP has gained wide acceptance and good results have been reported.

The recent new interest in ASCP was spearheaded by Frist et al.[4] in 1986. They described a simplified technique of CPB and unilateral selective cerebral perfusion under conditions of moderate hypothermia for the treatment of selected patients with aortic arch aneurysms. However ASCP did not gain widespread acceptance until Kazui and colleagues[18-20] indicated, with a series of clinical and experimental investigations, important details of the perfusion technique including the site, volume, pressure, and temperature of the perfusion.

Deep hypothermic circulatory arrest

The main hypothesis is that hypothermia depresses the cerebral metabolism *enough* to allow a safe period of total circulatory arrest characterized by the absence of detectable functional or organ derangements during the early or late postoperative period.

Hypothermia and oxygen consumption, considered as a measure of cerebral oxygen metabolism and CBF, are strongly coupling variables of paramount importance for brain protection. Thus, cerebral metabolism is reduced by approximately 5 to 7% per degree centigrade, which results in a prolonged tolerance to ischemia. In humans CBF is directly related to the temperature, cerebral metabolism, and arterial carbon dioxide tension. CBF (together with cerebral oxygen metabolism) decreases as the brain temperature decreases. During CPB the influence of temperature on cerebral perfusion is dependent on carbon dioxide management. Alpha-stat management (temperature-uncorrected blood gases) maintains a better CBF to metabolism ratio during hypothermia than pH-stat management (temperature-corrected blood gases) but has the potential disadvantage of cerebral hypoperfusion because of a low arterial carbon dioxide tension[21]. Alternatively, pH-stat management with strongly uncoupled CBF and metabolism has the theoretical advantage of increasing cerebral perfusion and enhancing brain cooling, but may be laden by an increase in intracranial pressure and hence brain edema and brain embolism.

DHCA is a well-established method of brain preservation during cardiovascular operations. However, brain damage as a consequence of hypothermic circulatory arrest remains a frequent subtle complication. The optimal conditions for maximizing cerebral protection during DHCA are not well established and the utilization of this technique varies among institutions. There is still controversy about basic aspects of DHCA such as the optimal temperature at arrest, cooling and warming rates, pH strategy, hemodilution, and the duration of circulatory arrest that may be considered as safe.

The available information does not allow the formulation of a table or of a rigorously derived equation relating the safe duration of total circulatory arrest to various temperatures on the basis of rigorously derived rules.

Advantages of DHCA include:
• quite dry, motionless operative field;
• avoidance of clamping and manipulation of the aorta with reduced brain embolic risk;
• less complicated and no need for additional equipment.

Disadvantages of DHCA include:
• limited "safe" time of circulatory arrest. In the largest clinical experience published to date, Svensson et al.[15] reported on a series of 656 patients (median DHCA time 31 min, range 7-120 min) overall stroke and early mortality rates of 7 and 10% respectively. At univariate analysis (but not at multivariate analysis), periods of circulatory arrest > 40 and > 65 min were indicated as predictors of stroke and early mortality respectively.

Reich et al.[22], in agreement with the results of others[23], demonstrated that a DHCA time ≥ 25 min is associated with memory and fine motor deficits and with prolonged hospital stay. Sakamoto et al.[24], in a laboratory investigation, indicated that a DHCA time < 25 min was free of behavioral or histological evidence of brain injury.

McCullough et al.[25] recently demonstrated that the human cerebral metabolic rate is still 17% of baseline at 15°C and that at this temperature circulatory arrest is safe so long as it lasts for a maximum of 29 min. The same authors[26], in an animal model, demonstrated that metabolic activity is still 19 and 11% of baseline values at 18 and 8°C respectively. Thus, despite the dramatic reductions in cerebral metabolism and oxygen requirements with profound hypothermia, there is still an ongoing, continuous need of substrates to the brain. Circulatory arrest will lead to a continuous temperature-dependent depletion of energy stores and to an increase in intracellular acidosis that is due to anaerobic glycolysis and production of lactate. This cerebral ischemic insult is aggravated by hyperglycemia[27]. The observation that significant residual metabolic activity is still present at 18°C is somewhat alarming and suggests that further metabolic suppression may be achieved by deeper levels of hypothermia. However, for brain temperatures between 18 and 8°C, a loss of autoregulation[26] has been demonstrated and the so-called "luxury perfusion" that results when CBF exceeds metabolic demands is theoretically associated with an increased risk of brain embolism. These findings strongly suggest that circulatory arrest times of 45-60 min were too optimistically indicated as safe;
• reperfusion injury mediated by activated constituents of blood, including leukocytes especially neutrophils, platelets, and complement, may produce vascular, tissue, and neurological damage, with cardiac and pulmonary dysfunction[28,29];
• prolonged CPB time required to cool down and rewarm the patients. It has been suggested that prolonged periods of CPB and hypothermia may result in an increased number of pulmonary, renal, cardiac, and endothelial dysfunctions and in an increased microembolism. In a recent study[23] comparing the postoperative morbidity in patients undergoing ascending aorta/hemiarch replacement, it has been suggested that the postoperative recovery of the pulmonary and renal functions is better in patients having ASCP and moderate hypothermia than in those having DHCA;
• clotting complications. It has been documented that deep hypothermia causes alterations in the normal coagulation mechanism. Hypothermia may shorten the coagulation time. Antithrombin activity shows a marked rise after rewarming, while prothrombin and factor VII remain at a low level for several days. An increase in fibrinolytic activity has been reported in experimental animals. Moreover, prolonged periods of perfusion for cooling and rewarming have been shown to exhaust clotting factors and interfere with blood coagulation[30]. Connolly et al.[31] observed a reduction in the platelet count to below 15 000/mm^3 during cooling, as well as an increased fibrinolytic activity on rewarming.

Retrograde cerebral perfusion

RCP is commonly used as an adjunct to DHCA during surgery of the thoracic aorta as a method to improve the neurological outcome.

CPB is usually instituted by means of bicaval cannulation and arterial cannulation with a shunt between the arterial and venous lines that is clamped during antegrade CBF. Central cooling is carried out within 20-30 min to produce profound hypothermia with core temperatures ranging from 10-20°C. Depending on the institution criteria, as soon as electroencephalographic silence, jugular venous saturation or cooling for a specific time interval or temperature are achieved, the superior vena cava is snared, circulatory arrest is induced, and the arterial line is clamped while the shunt line is opened to allow the oxygenated blood to be diverted to the cannula of the superior vena cava. Unsaturated venous blood is returned to the heart/lung machine via a

cardiotomy suction placed in the open thoracic aorta and via the cannula of the inferior vena cava (Fig. 1). After completion of the distal aortic anastomosis and reattachment of the arch vessels, deairing of the open aortic arch is achieved by RCP and backflushing via the femoral artery. Resumption of conventional CPB and rewarming are then accomplished. Means of RCP flow monitoring generally include either a central venous catheter or a jugular bulb catheter. Near-infrared spectroscopy and transcranial Doppler assessment of the flow in the middle cerebral artery and central retinal artery have also been used. The flow (100-500 ml/min) is usually adjusted to maintain the central venous pressure in the range of 15 to 25 mmHg.

Hypothetical RCP neuroprotective mechanisms include the maintenance of cerebral hypothermia, washout of embolic air or debris, cerebral perfusion and metabolic support. However, on the basis of the conflicting results of clinical and animal laboratory studies, none of the proposed mechanisms of cerebral protection noted above may be held as being definitely established.

Debate on the effectiveness of RCP has in part focused on the presence of competent valves in the venous circulation as well as of a dominant collateral circulation via the azygos system circumventing the brain in human beings[32,33].

Furthermore, experimental studies investigating retrograde CBF in animal models are difficult to interpret because of interspecies differences in cerebral venous anatomy and widely varying experimental protocols. Canine models are limited by the presence of venous valves in the neck and head rendering bilateral cannulation of the maxillary veins or perfusion of the superior sagittal sinus necessary. Such a step would not be very relevant in clinical practice[34,35]. More conventional cannulation is possible in the pig because there are no venous valves. However, there still are some differences in the proportion of flow going to the brain versus extracranial structures with respect to humans.

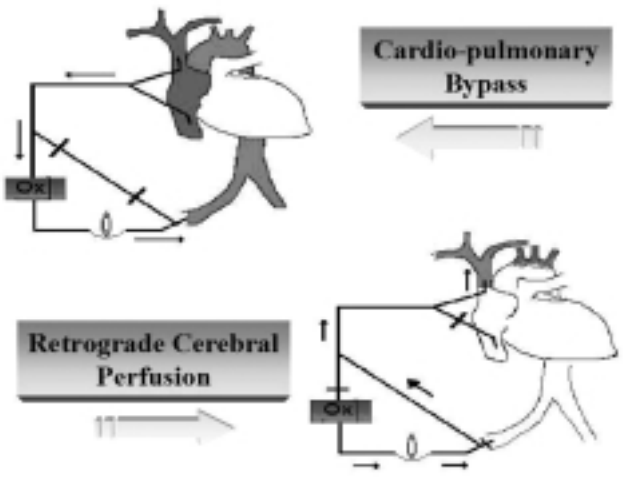

Figure 1. *Retrograde cerebral perfusion.*

The experimental literature seems to suggest that the overall retrograde intracranial flow is in the range of 20 to 60% of values achieved with hypothermic CPB[36-38]. However, on the basis of capillary flow data, it is unlikely that RCP provides sufficient perfusion to satisfy cerebral metabolic oxygen and substrate demands[39,40].

Human and laboratory studies of cerebral metabolism evaluating the cerebral oxygen consumption, pH and high-energy phosphate level modifications using magnetic resonance and/or spectrophotometry suggest that RCP provides inadequate metabolic support when compared to normothermic baseline conditions, CPB and ASCP but appears to be superior to DHCA alone[38,41-43].

Reports on the effects of RCP on the histopathology of the brain in animal models are numerous in the literature and mostly comparative with DHCA and ASCP.

Midulla et al.[43] demonstrate that although by all outcome measures ASCP affords the best cerebral protection, RCP is clearly better than DHCA.

Juvonen et al.[42] reported that although effective washout of particulate emboli from the brain may be achieved with RCP obtained by occluding the inferior vena cava, no advantage of RCP with inferior vena cava occlusion after embolization is seen at behavioral scores, electroencephalographic recovery or histopathologic examination; RCP with inferior vena cava occlusion results in greater fluid sequestration and mild histopathologic injury even in control animals.

Ye et al.[44] suggested that antegrade cerebral perfusion prevented ischemic damage to the brain and that RCP provided more protection than DHCA alone but that quite severe damage still occurred.

Boeckxstaens and Flameng[45] reported that non-human primates undergoing RCP had greater glial edema than those having DHCA and morphologic studies conducted by Mohri et al.[46] revealed the development of focal infarctions in the brain, destruction of the blood-brain barrier, and cerebral edema following RCP.

The relationship between the use of RCP and the clinical outcome is also unclear. Some authors reported the duration of RCP to be a predictor of death and of an adverse neurological outcome[47,48], whereas others did not[49-51].

Studies comparing the clinical outcome obtained with RCP, DHCA and ASCP also yielded mixed results. Coselli and LeMaire[52] and Ehrlich et al.[53] reported that the use of RCP resulted in less mortality and fewer neurological disorders than DHCA. In a prospective comparative study, Okita et al.[54] reported similar results in terms of hospital mortality and postoperative new strokes in patients operated with RCP and ASCP. However, the prevalence of TND was significantly higher in patients with RCP. In a recent study by Hagl et al.[55] including a group of patients requiring a period of "total cerebral protection" lasting between 40 and 80

min, ASCP was associated with a lower incidence of TND than RCP or DHCA.

In summary, on the basis of human and laboratory investigations, the neuroprotective mechanisms of RCP still remain controversial. When compared to ASCP, RCP seems to be less affective but still provides somewhat more brain preservation than DHCA probably due to the continued cerebral cooling via the veno-arterial and veno-venous collateral circulations.

Antegrade selective cerebral perfusion

The earliest attempts to repair the aortic arch relied on complex methods of extracorporeal cerebral perfusion. In 1957, DeBakey et al.[14] reported a successful resection of an aortic arch aneurysm using normothermic CPB achieved by means of several pumps and bilateral cannulation of both the subclavian and carotid arteries. In 1958, these same authors reported a 75% overall mortality in a group of 24 patients undergoing ascending aorta and aortic arch repair[56].

The CPB perfusion technique was simplified by Dubost et al.[57] and Pearce et al.[58] using a single pump with an Y-connection of the arterial line.

However, since then, extracorporeal cerebral perfusion during aortic arch replacement was overshadowed by the growing interest and utilization of circulatory arrest and deep hypothermia.

More recently, the recognition that a) deep hypothermia provides a reduced safe time of circulatory arrest (40-65 min)[15], b) by combining ASCP with moderate hypothermia much lower flow rates may be used, and c) ASCP provides better cerebral protection than either DHCA or RCP[20,39,43,59], led to a renewed interest in ASCP.

The new ASCP "era" was initiated in 1986 by Frist et al.[4]. These authors described a simplified CPB technique with partial brachiocephalic perfusion, a low CPB flow (30 to 50 ml/kg/min) and moderate systemic cooling (26 to 28°C). The arterial line from a single pump head had an Y-shape to perfuse the femoral artery and either the innominate or left carotid artery. Eight patients (out of 10) survived the operation and no postoperative stroke occurred.

In 1991, Bachet et al.[60] introduced a procedure employing cold "cerebroplegia" and the "open aortic anastomosis technique". They perfused the innominate and left common carotid arteries with blood between 6 and 12°C through separate pumps and heat exchangers. In 54 patients with arch aneurysms, the mortality was 13% and only one episode of severe neurological injury occurred[60].

Kazui et al.[18] introduced the use of separate arterial pump heads for the cerebral and systemic circulations in order to provide individual hypothermic perfusion at 25°C to each system. Cerebral perfusion was achieved by means of endoluminal cannulation of the brachiocephalic and left common carotid arteries while the left subclavian artery was clamped or occluded with a Fogarty catheter to avoid the steal phenomenon (Fig. 2).

However, ASCP was not considered safe and its use varied among institutions until Tanaka et al.[19] revealed, in an elegant experimental study, important perfusion

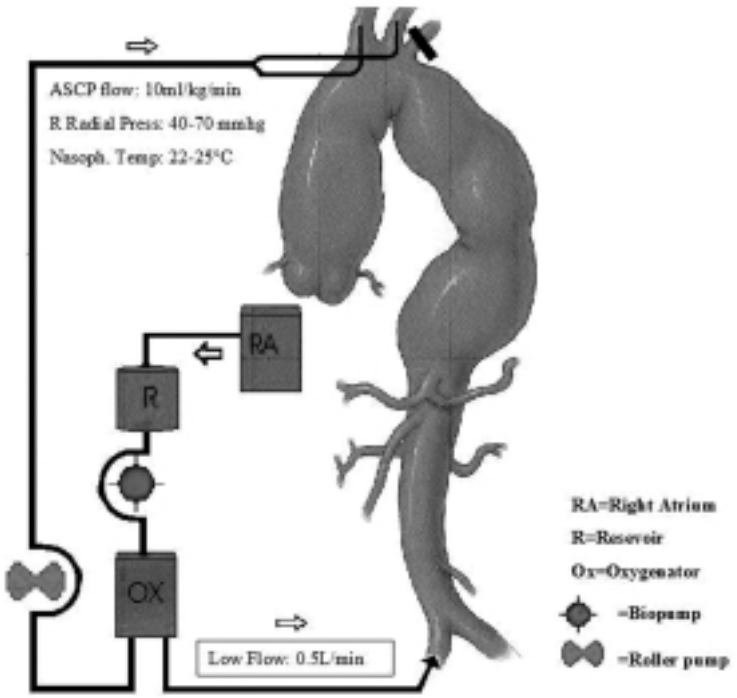

Figure 2. *Antegrade selective cerebral perfusion (ASCP).*

details such as the optimum perfusion flow rate, pressure, and temperature. On the basis of somatosensory, cerebral metabolism and histopathologic findings, these authors suggested that the safe range of flow rates for cerebral perfusion during moderate hypothermia (25°C) is > 50% of the physiologic level with a perfusion pressure of no less than 30 mmHg. In the clinical setting, a perfusion volume of 10 ml/kg/min is considered to be ≥ 50% of the physiologic flow rate of the cerebral circulation.

The relationship between the use of ASCP and clinical outcome has been widely reported in the literature.

In a recent study by Kazui et al.[61] including a series of 330 patients, the hospital mortality was 11.2% (falling to 3.2% for the last 124 patients), the PND rate 2.4%, and the TND rate 4.2%.

In a multicenter study, Di Eusanio et al.[62] reported, for the largest series of patients published to date (n = 588), a hospital mortality of 8.7% with PND and TND rates of 3.8 and 5.6% respectively. At logistic regression analysis, urgent operation, a recent neurological event, tamponade, unplanned bypass surgery and the pump time were indicated as independent predictive risk factors for hospital mortality. The same authors, in a previous series of 413 patients, clearly indicated that the extent of aortic replacement and the duration of ASCP (up to 90 min) were not associated with an increased risk of hospital mortality and of an adverse neurological outcome[63]. This means that when ASCP is used, under elective or urgent conditions, the predicted risk of hospital mortality and adverse neurological outcomes is similar regardless of whether a total thoracic aorta replacement or only a hemiarch replacement is performed, the duration of circulatory arrest no longer being a primary concern during this kind of surgery. Indeed, when using ASCP, the surgeon is given the luxury of an "unlimited time" to perform the aortic reconstruction which seems to be particularly important during complex and time-consuming procedures such as total arch replacement and acute type A dissection repair.

Manipulation and cannulation of the arch vessels have been indicated as important drawbacks of ASCP since they a) increase the embolic risk in patients with atherosclerotic aortic arch aneurysms, b) increase the risk of damage of the arch vessels in patients with acute aortic dissection, and c) render a complex procedure even more complex due to the reduced surgical visibility and to the necessity of preparing the arch vessels and installing the ASCP system. Thus, the usefulness of ASCP during less time-consuming procedures (ascending aorta/hemiarch replacement) and acute type A dissection repair is more debated. Indeed, by some authors, DHCA with or without RCP is preferred during these aortic procedures since manipulation of the aortic arch and arch vessels may be avoided and the procedure may be safely accomplished within 25-30 min of circulatory arrest with deep hypothermia.

Certainly, cannulation of the arch vessels represents an important phase of the procedure and great care has to be taken. However, in our experience it always took < 3 min to insert the ASCP cannulae into the arch vessels and establish cerebral perfusion. Surgical visibility can be adequate if the ASCP catheters are placed towards the patient's head. They are minimally traumatic and positioned, under direct visualization, quite distally in the arch vessels where the likelihood of plaque, atheroma, clots or dissection is very low. It was always easy, during dissection repairs, to identify the true lumen of the arch vessels through the aortotomy, and our cerebral monitoring tools always allowed us to confirm the correct positioning of the catheters and the symmetric distribution of blood to the two hemispheres. In cases of severe atheroma, clots or dissection involving both the aortic arch and the proximal segments of the arch vessels the separated graft technique may be effective in reducing the risk of brain embolization and improving the neurological outcome of patients by entirely replacing the aortic arch and proximal segments of the arch vessels[64].

Furthermore, in a previous study comparatively investigating the results in terms of postoperative pulmonary and renal function recovery in patients undergoing ascending aorta/hemiarch replacement with ASCP and DHCA, we observed both longer intubation times and higher creatinine values in patients receiving DHCA as compared to those receiving ASCP and moderate hypothermia as a method of brain protection[23]. It may be speculated that the better pulmonary and renal function recovery after ASCP was due both to the use of moderate hypothermia (instead of deep hypothermia) and to the reduced periods of CPB required to cool down and rewarm the patient. Moreover, if it is true that in cardiac surgery in general and in aortic surgery in particular, CPB time is the most important risk factor for hospital mortality and morbidity, all possible efforts should be made to reduce it: ASCP (as compared to DHCA) unquestionably requires shorter periods of extracorporeal circulation since moderate hypothermia is sufficient to achieve adequate cerebral protection.

In summary, ASCP provides numerous advantages:
- "unlimited time" of safe circulatory arrest;
- the extent of aortic replacement has no impact on the hospital mortality and neurological outcome;
- lower TND rates;
- better pulmonary function recovery after surgery;
- moderate instead of deep hypothermia may be used;
- shortened periods of CPB;
- better brain cooling.

Avoidance of stroke

It has been suggested that during surgery of the aortic arch PND (stroke or coma) is due to embolism. Consequently, reducing the risk of brain embolism, in addi-

tion to adequate cerebral protection, is of paramount importance.

Thus, in recent years, alternative cannulation sites such as the ascending aorta, the right/left axillary/subclavian artery and the brachiocephalic trunk with or without the interposition graft technique, have been employed and often preferred to the more traditional femoral artery which still remains the most common cannulation site for reoperation and type A dissection repair[7,9,10].

These central cannulation sites share in common the avoidance of retrograde perfusion of the thoraco-abdominal aorta intended as a potential source of embolic material such as plaques, clots and calcifications destined to the cerebral vasculature.

Furthermore, during acute type A dissection repair, these alternative cannulation sites may be effective in reducing the risk of brain/organ malperfusion always considerable at the time of institution of CPB, clamping of the aorta and ventricular fibrillation during the cooling phase.

When the ascending aorta is cannulated, as we do in most patients with degenerative aortic aneurysms, computed tomographic scan, manual palpation, transesophageal or epiaortic scan may help to find an optimal plaque-free site for cannulation.

As reported in the literature, the quality of the aortic arch wall is an important risk factor for PND, the risk being obviously higher in patients with large atheromas, clots, and calcifications.

A frequent source for this potentially mobile material is the origin or proximal segments of the arch vessels. The separated graft technique, by replacing the entire aortic arch and proximal segment of the arch vessels with a three-branched prosthetic graft is thought to be effective in reducing embolic injuries in patients with atherosclerotic aneurysms[65]. If only a partial arch replacement is performed, all the remaining material should be carefully removed by means of thromboendarterectomy.

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