

Anesthetic Implications of Aortic Arch Aneurysm Repair with Deep Hypothermic Circulatory Arrest and Selective Cerebral Perfusion - A Case Report and an Updated Review

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Abstract: Deep hypothermic circulatory arrest (DHCA) is a well-established technique offering cerebral protection and optimal surgical conditions for complex cardiovascular and neurological procedures. By cooling the body to 18–20°C, DHCA reduces cerebral metabolic demand, allowing up to 40 minutes of safe circulatory arrest. Extended DHCA is supported by selective antegrade cerebral perfusion (SACP) or retrograde cerebral perfusion (RCP), with SACP preferred for its reliable global oxygenation. Continuous hemodynamic and neurological monitoring, including arterial and central venous pressure, cardiac output, and cerebral oxygenation (NIRS and BIS), is critical for patient safety. This case describes a 62-year-old female undergoing repair of a chronic type I aortic dissection. DHCA with bi-hemispheric SACP was employed, accompanied by hypothermia induction, pharmacological neuroprotection, and tight hemodynamic management. Postoperative outcomes were favorable, with no neurological deficits. Discussion highlights the importance of tailored acid-base, glycemic, and coagulation management strategies. Pharmacological agents, such as barbiturates and corticosteroids, show promise but require further evidence. Combining advanced monitoring techniques enhances safety and outcomes, emphasizing the need for individualized protocols to optimize DHCA efficacy and minimize complications.

Keywords: Aortic Arch Aneurysm; Deep Hypothermic Circulatory Arrest; Selective Cerebral Perfusion; Neuroprotection Strategies.



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1. Introduction

Proven safe for aortic surgeries since 1975, Deep Hypothermic Circulatory Arrest (DHCA) allows optimal surgical conditions while providing cerebral protection during complex cardiac, vascular, neurological, and urological procedures, including cerebral aneurysms and renal tumors with caval invasion [1]. This technique is based on the principle that cerebral metabolic rate is significantly impacted by hypothermia, decreasing by 6% for every degree below 37°C. In DHCA, the body is cooled to 18–20°C, which may be tolerated for up to 40 minutes without causing brain damage. Over 60 minutes, however, most patients will experience irreversible neurological sequelae. Systemic cooling during CPB is accomplished by circulating water from cooled reservoirs through a heat exchanger integrated into the CPB oxygenator, which may be improved with extra head cooling through ice packages [1]. In many centers, DHCA length is extended through retrograde cerebral perfusion (RCP), after cannulation of the superior vena cava, or selective antegrade cerebral perfusion (SACP), hemispheric or bi-hemispheric, after cannulation of

the right axillary artery, innominate artery, and/or left common carotid artery. SACP is the preferred technique for extended cerebral protection during complex aortic surgeries, as it prolongs the safe length of DHCA and offers a more reliable global oxygenation than RCP [2].

Hemodynamic monitoring is critical during surgeries involving DHCA to ensure adequate organ perfusion and minimize complications. Arterial pressure monitoring (IAP) is essential for assessing systemic blood pressure and collecting exams, while central venous pressure (CVP) offers insight into venous return and fluid status. Additionally, cardiac output monitors are vital to evaluate circulatory efficiency and adjust perfusion during CPB and reperfusion [1]. Measurement of right heart pressures may be performed using a pulmonary artery catheter or transesophageal echocardiography (TOE) in selected patients. Temperature is typically monitored at two sites: the nasopharynx to estimate brain temperature and the bladder to assess body temperature, ensuring accurate tracking of the patient's temperature throughout the procedure [1].

This report aims to describe the practical application and outcomes of advanced monitoring and cerebral protection strategies during DHCA, focusing on the use of bi-hemispheric selective antegrade cerebral perfusion in a complex aortic dissection surgery. The relevance of this case lies in demonstrating integrated approaches that combine hemodynamic and neurological monitoring techniques with pharmacological interventions and perfusion strategies to enhance patient safety and minimize complications. In addition to contributing to the understanding of the challenges and advancements in DHCA management, this report highlights the importance of individualized protocols for high-complexity surgeries.

2. Case Report

A 62-year-old female with a history of systemic hypertension and controlled asthma presented for elective repair of a chronic type I aortic dissection due to aortic aneurysm. Patient had a history of prior aneurysm repair with placement of a Dacron graft. In the operation room, after non-invasive monitoring and establishment of venous access, IAP via the left femoral artery and CVP via right internal jugular vein catheter were placed, as well as nasopharyngeal and bladder thermometers for core/body temperature monitoring and bispectral index (BIS) to verify cerebral electrical silence during DHCA. FloTrac® was used for monitoring cardiac output and Near-Infrared Spectroscopy (NIRS) informed cerebral oxygenation before, during and after DHCA (Figure 1).

Anesthesia was induced with remifentanyl in target-controlled infusion system (TCI), 100 mg propofol and 36 mg rocuronium and maintained with isoflurane and remifentanyl for tight hemodynamic control. To ensure adequate cerebral and systemic perfusion during CPB, DHCA and SACP, systemic hypothermia was induced with cooling to 20°C using CPB. Additional ice packs were applied around the patient's head for enhanced cerebral cooling. Thiopental was administered to reduce cerebral metabolic demand, and methylprednisolone was used to prevent the inflammation produced by DHCA.

Total circulatory arrest lasted 25 minutes, when bi-hemispheric SACP was established via cannulation of the right axillary artery and the left carotid artery. Cerebral oxygenation was monitored continuously through NIRS (Figure 2). Flow was maintained within 6-10 mL/kg/min and pressure was maintained within 50-60 mmHg, adjusted by NIRS trends, to provide targeted perfusion to the brain during the reconstruction of the aortic arch. SACP lasted 38 minutes. After completing the aortic arch repair, gradual reperfusion was initiated with controlled rewarming to avoid reperfusion injury, maintaining the gradient between core and peripheral temperatures < 5°C.

This process was carefully monitored to maintain stable hemodynamics and prevent cerebral edema or injury, since the termination of SACP and the withdrawal of CBP are associated with significant vasodilation, changes in systemic vascular resistance, and hemodynamic instability. Vasopressor support was performed with 0.6 mcg/kg/min norepinephrine and 0.08 UI/min vasopressin to maintain a Mean Arterial Pressure (MAP) of 65

mmHg and a 10 mcg/kg/min dobutamine to enhance contractility during reperfusion. Coagulation management included reversal of heparin with protamine (1:1 ratio), and transfusion of platelets (2 pools), fresh frozen plasma (4 units), cryoprecipitate (8 units), 2,500 IU of prothrombin complex concentrate, and 450 mL of blood recovered by the cell salvage. Fluid, electrolyte, and acid-base imbalances were adequately managed. The heart resumed sinus rhythm post-CPB without significant arrhythmias. The patient received 2500 mL of Ringer's lactate, had a diuresis of 350 mL and a blood loss of 750 mL. The following table resumes trends in mean arterial pressure, NIRS, cardiac output, and core temperature at different time points (Table 1).

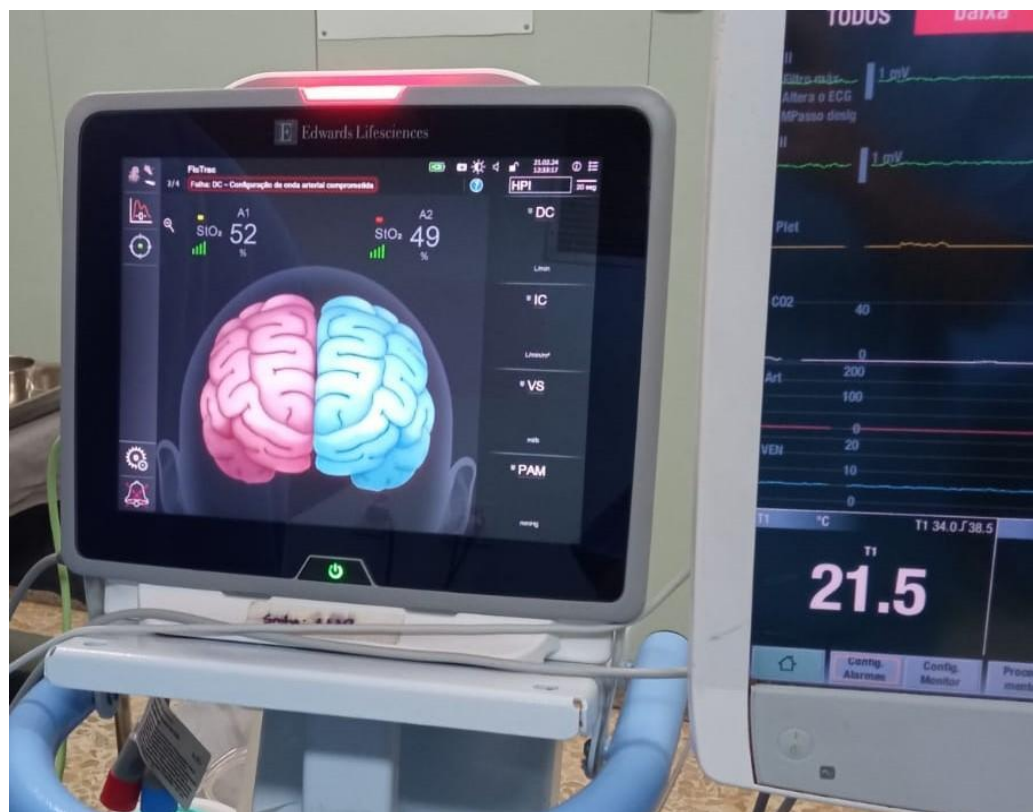
Figure 1. NIRS before entering CBP and DHCA.



Table 1. Trends in mean arterial pressure, NIRS, cardiac output, and core temperature at different time points.

Time Point	Mean Arterial Pressure (mmHg)	NIRS (%)	Cardiac Output (L/min)	Core Temperature (°C)
Pre-DHCA	75-85	70-75	3.5-4.0	36.5
During DHCA	50-60	45-55	0.0	21.0
Rewarming	60-70	60-65	1.5-2	32.0
Postoperative	65-75	65-70	2-2.5	36.0

Figure 2. NIRS and core temperature during SCP.



Regarding the postoperative course, the patient remained intubated for 30 hours postoperatively and was successfully extubated without complications. She remained in the ICU for a total of 6 days, during which low-dose vasopressor support was weaned off. The total hospital stay was 8 days. She presented a KDIGO 2 acute kidney injury, which was managed without renal replacement therapy, with prompt recovery. No major complications such as coagulopathy, infections or arrhythmias were observed.

Neurological evaluation was performed through a formal neurological examination postoperatively, including assessment of motor and sensory function, cranial nerves, and deep tendon reflexes. In addition, the patient underwent cognitive screening using the Mini-Mental State Examination (MMSE), which revealed no deficits. Brain computer tomography was also performed postoperatively to rule out subclinical ischemic injury, confirming the absence of lesions. Longitudinal consults in our service confirmed the absence of neurological deficits, reassuring the neurological integrity following DHCA.

3. Discussion

The duration of SACP and DHCA is the most important aspect related to neurological prognosis in patients undergoing aortic surgery. In this case, DHCA lasted 25 and SACP lasted 38 minutes, due to the technical complexity of the surgical procedure, since the patient already had prior aortic surgery. While most literature suggests that prolonged DHCA beyond 40 minutes is associated with increased neurological risk, a study in a specialized center with 490 participants demonstrated that the isolated use of DHCA can be safe for up to 50 minutes, without significant neurological sequelae [3]. However, the use of SACP allows for longer periods of cerebral protection. A meta-analysis showed that, although there was no difference in the incidence of permanent or transient neurological deficits between SACP and DHCA versus DHCA alone, mortality was significantly lower with the use of SACP compared to DHCA alone [4]

Deep hypothermia (<20°C) provides robust neuroprotection by reducing metabolic demand; however, there is an ongoing debate regarding the optimal degree of cooling. Some studies suggest that moderate hypothermia (20-28°C) may be sufficient, potentially reducing rewarming-related complications, such as neuroinflammation and loss of cerebrovascular autoregulation [5]. Controlled rewarming is equally important, as rapid temperature elevation has been linked to ischemia-reperfusion injury and cerebral edema. Protocols emphasizing gradual rewarming, often at rates <0.5°C/min, may mitigate these risks and improve neurological recovery.

Pharmacological neuroprotection has been investigated to further reduce the risk of neurological injury, through metabolic suppression, anti-inflammatory effects, and antioxidative mechanisms. Burst suppression could be achieved with barbiturates such as thiopental, reducing cerebral metabolic rate and oxygen consumption, despite their potential hemodynamic side effects [2]. Propofol, which presents neuroprotective properties, induces burst suppression like barbiturates and has antioxidative properties that may mitigate ischemia-reperfusion injury. High-dose corticosteroids are frequently administered to attenuate the inflammatory response associated with DHCA and reperfusion, although it lacks evidence in reducing neurological injury. Magnesium sulfate, mannitol and lidocaine are other less used agents with a theoretical benefit of reducing excitotoxicity [2]. The use of thiopental and glucocorticoids is standard in our service for surgeries with total circulation arrest.

Neurological monitoring during DHCA can be performed through cerebral blood flow (e.g., jugular venous oximetry, transcranial Doppler, and near-infrared spectroscopy) or brain activity (e.g., quantitative EEG, BIS and evoked potentials). While jugular venous oxygen saturation provides insight into cerebral oxygen balance, its reliability is limited. Quantitative EEG is sensitive to ischemia but affected by interference (1). BIS, which measures the depth of anesthesia and cerebral electrical activity, showed a decrease to zero during DHCA, indicating complete suppression of cerebral electrical activity. A review of the literature demonstrated that BIS, as a quantitative indicator, helps to adjust anesthesia and improve cerebral perfusion. It can identify cerebral hypoperfusion or anomalous brain activity during surgery, allowing for rapid adjustments in anesthesia [6]. However, BIS does not provide direct confirmation of cerebral ischemia, is susceptible to interference by artifacts and its correlation with burst suppression on EEG has not been fully established.

NIRS, on the other hand, is a reliable tool to monitor intraoperative frontal lobe cerebral oxygen saturation. However, its use to predict postoperative stroke remains limited [7]. To ensure the reliability of NIRS readings, careful placement of the sensor on the forehead, avoiding direct light exposure, and consistent calibration are necessary. Despite the lack of strong evidence of significant benefits for neurological outcomes like stroke, these tools are gaining use due to their practicality and minimal harm risk [8,9]. In this case, the use of both BIS and NIRS allowed complementary data, with NIRS providing real-time information on cerebral oxygenation and BIS verifying that the brain. In terms of available technology in our hospital, we consider that our patient received an adequate neurological assessment, within the limitations of each method, as both NIRS and BIS provided complementary data that ensured optimal cerebral protection during DHCA and SACP [3].

Acid-base management and glycemic control are critical to optimizing neurological and systemic outcomes. Hypothermia significantly alters gas solubility and ionization, necessitating tailored strategies to maintain physiological homeostasis. Two main approaches are employed for acid-base management: the alpha-stat strategy, which maintains a pH of 7.40 and a partial pressure of carbon dioxide (pCO₂) of 40 mmHg at 37°C regardless of the patient's actual temperature, and the pH-stat strategy, which adjusts pH and pCO₂ to measured patient temperature by adding carbon dioxide to the circuit [2]. While alpha-stat is preferred in adults for preserving cerebral autoregulation, pH-stat is more commonly used in pediatric patients to increase cerebral blood flow despite a potentially higher embolic risk. Glycemic control is also crucial, as hypothermia and cardio-

pulmonary bypass often induce hyperglycemia due to insulin resistance and stress responses. Elevated glucose levels exacerbate ischemic injury and lactate production, requiring regular monitoring and insulin administration to maintain normoglycemia. Individualized management strategies integrating these considerations are essential for minimizing complications and improving outcomes during DHCA [2].

DHCA can induce coagulopathy, particularly impairing maximum clot elasticity due to platelet dysfunction in ROTEM analysis [4]. Therefore, coagulation management and insurance of proper hemostasis during CPB and reperfusion is vital to minimize ischemic injury and guarantee the success of the surgery. The administration of antifibrinolytic agents during CPB has been explored to mitigate coagulopathy. The Society of Cardiovascular Anesthesiologists (SCA) recommends the use of antifibrinolytics, and autologous blood recovery to limit exposure to allogeneic transfusions in patients under cardiac surgery, aiming to minimize blood loss, reduce transfusions, and improve patient outcomes. ROTEM can provide a detailed understanding of coagulation status, aiding in tailored management strategies [5]. Unfortunately, ROTEM was not available in our service at the time of the case. This is a weakness of our work, since the results of these tests could guide a more targeted transfusion of blood components.

4. Conclusion

Hypothermia, SCP and continuous hemodynamic and neurological monitoring are crucial for maximizing patient safety in DHCA. The use of pharmacological agents such as barbiturates and corticosteroids, though promising, still lacks solid evidence to conclusively demonstrate their efficacy in preventing neurological injury. Rigorous monitoring of cerebral oxygenation and brain activity, with the combination of NIRS and BIS, allows for comprehensive evaluation, enabling early interventions and ensuring the best possible neurological outcome. Management of acid-base balance, glycemic control, and coagulation is critical and directly impacts systemic and neurological results. Ultimately, personalized management strategies, tailored to each patient's unique needs, are essential for optimizing outcomes in surgeries under DHCA. The advancement of monitoring techniques and ongoing research into pharmacological and therapeutic protocols are vital to improving safety and effectiveness, minimizing complications, and enhancing long-term prognosis.

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Research Ethics Committee Approval: We declare that the patient approved the study by signing an informed consent form and the study followed the ethical guidelines established by the Declaration of Helsinki.

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Conflicts of Interest: The authors declare no conflicts of interest.

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