NEUROLOGICAL SAFETY IN CARDIAC SURGERY: OPTIMIZING CEREBRAL PROTECTION TECHNIQUES

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ABSTRACT

Cardiopulmonary bypass (CPB) is a crucial part of cardiovascular surgery, but it can lead to significant complications, most notably neurological complications. Thereby, making means for neurological protection a topic of focused research. This review of literature aims to explore the various strategies to mitigate these risks, including perioperative surgical modifications, pharmacologic interventions, and intraoperative monitoring techniques. The findings of this literature review show that key surgical strategies include maintaining systemic temperatures, slow rewarming, maintaining mean arterial pressure, and targeting a hematocrit of at least 25%. Pharmacological agents like barbiturates, propofol, and calcium channel blockers also have neuroprotective benefits. Intraoperative monitoring using transcranial doppler (TCD), electroencephalography (EEG), near-infrared spectroscopy (NIRS), and certain biomarkers can help assess cerebral blood flow and tissue oxygenation, enabling real-time intervention and risk stratification for long-term neurological outcomes. Hence, implementing these strategies can improve patient outcomes by reducing the incidence and severity of CPB-related neurological complications

KEYWORDS

Cardiopulmonary bypass, Cerebral protection, Neurological injury, Cardiac surgery

INTRODUCTION

The development of cardiopulmonary bypass (CPB), a device that could temporarily replace the heart's function and provide blood oxygenation, made it possible for surgeons to perform heart surgery while maintaining normal blood circulation and sufficient oxygenation of the brain and body. CPB offers cardiac surgery in a bloodless environment [1]. In order to offer physiological support, it integrates an extracorporeal circuit (ECC), where venous blood is drained to a reservoir, oxygenated, and then returned to the body via a pump. Surgeons, perfusionists, and anesthesiologists must work together as a team to successfully use CPB [2].

Despite being a crucial finding in cardiovascular surgery, CPB is linked to a number of morbidities, including those affecting the kidneys, lungs, and most significantly, the brain. One of the most feared side effects of heart surgery is still brain damage. The range of ailments is wide; more subtle injuries undoubtedly happen, even though a stroke may be quickly identified and diagnosed. Therefore, the focus of research has been on finding ways to safeguard the brain during cardiovascular surgery [3].

Reducing the sources of harm and improving brain tolerance to ischemic insults are the goals of brain protection during heart surgery [4]. First, identifying patients at high risk may help lower the incidence of problems in those groups, but this is likely not a good enough preventive measure. Therefore, three areas should be the focus of preventive strategies: first, technological advancements in cardiac surgery and brain protection; second, pharmacologic therapy; and third, the development of trustworthy methods for assessing cognitive impairment following cardiac surgery. Only with all of these tools may truly effective palliative or preventive measures be developed and implemented [5].

REVIEW OF LITERATURE

Since the beginning, there has been concern about neurologic damage after cardiac surgery, which can be broadly categorized as frank stroke, encephalopathy, and neurocognitive issues [6]. It's critical to recognize that a number of etiological risk variables are linked to the development of CPB-related cerebral dysfunction when examining cardiac surgical neurologic morbidity [7].

Regarding preoperative patient-related factors that raise the risk of neurologic morbidity, there is broad consensus. Advanced age, a history of previous neurological episodes, aortic atherosclerosis, low cardiac output states, atrial arrhythmias, hypertension, and diabetes are preoperative risk factors. The most significant risk factor, advanced age, seems to amplify the other risk variables [6].

The neurological results following CPB can also be influenced by a variety of operational circumstances. The operational elements that lead to brain injury include ischemia-reperfusion injury, thromboembolic events, inadequate perfusion, anesthetic-induced brain toxicity, CPB-mediated inflammation, hypoglycemia, electrolyte, or acid-base abnormalities, and hypotension. CPB procedures and tools that produce little or no oxygen flow to the brain for extended periods of time are among the main causes of intraoperative brain injury [8].

The majority of cardiac surgery-related neurological issues are secondary to CPB usage. The etiology is complex; neurologic adverse effects are linked to the inflammatory and embolic responses that occur with CPB, but they persist after off-CPB procedures [5].

It is likely that a number of distinct pathways contribute to the development of neurologic problems following CPB:

1. Embolism: micro- or macro embolism.
2. The blood-brain barrier (BBB) and the activation of the inflammatory response.

3. Modifications in neuronal metabolism brought on by vasogenic or cytotoxic edema or hypoxemia.
4. Hypoperfusion in the brain.
5. Hyperthermia.
6. Hyperglycemia [5].

Surgical and Technical Neuroprotective Strategies.

Hypothermia: Hypothermia remains crucial in any cerebral protection strategy [9]. It can be defined as a body temperature below the normal physiological level of 37º C [10]. Tissue temperature, which is typically comparable to body temperature, affects cellular metabolic rate and, consequently, oxygen needs. According to the Q10 rule, which estimates tissue metabolic rate in relation to temperature, cellular metabolism decreases by 50% for every 10°C that a patient's body temperature is lowered below normal. Cooling protects tissues at times when oxygen delivery is minimal or nonexistent because of this significant decrease in oxygen demand. Because hypothermia makes gases more soluble, it also has the benefit of reducing air embolism [9].

Hypothermia can be classified into

a) Mild hypothermia: 28.1-34º C
b) Moderate hypothermia: 20.1-28º C
c) Deep hypothermia: 14.1-20º C
d) Profound hypothermia: ≤ 14º C [11].

Lowering the body temperature to 30°C results in a 50% decrease in the overall oxygen demand of the body and a 54% decrease in the oxygen demand of the brain. During cardiac surgery, moderate hypothermia maximizes the reduction of metabolic demands while posing the least risk of problems. However, when hypothermia falls below 27.5°C, the risk of complications increases. Adults with thoracic aortic aneurysms and infants with congenital abnormalities can benefit from surgical treatment through the use of deep hypothermia with circulatory arrest

(DHCA) and deep hypothermia with CPB. Following a time of perfusion cooling to a preset target temperature, the CPB flow is completely and continuously stopped [10,12].

Hypothermia during cardiac surgery is induced by systemic hypothermic CPB, topical cooling of the myocardium and cold cardioplegia [10].

Despite of the beneficial effects of hypothermia, it is important to remember that it greatly alters virtually every physiologic process of the body. Nursing assessment of the hypothermic patient is improved when nurses are aware of the physiologic effects of hypothermia in postoperative cardiac patients. It's critical to monitor body temperature appropriately and provide prompt nursing and medical measures [10].

Finally, prevention of cerebral hyperthermia, that is, cerebral temperatures exceeding 37ºC, during the rewarming phase must be avoided. Yosef et al demonstrated that this can be done using two approaches a) Reduce the temperature differential between the blood and water in the CPB heat-exchanger apparatus to delay the rate of rewarming, and b) set the goal rewarming temperature between 34°C and 35°C [13].

Cerebral Perfusion Strategies: Antegrade cerebral perfusion (ACP): During HCA, ACP is a technique that perfuses only the brain using specific cannulation techniques. Therefore, it has the potential benefit of shielding the brain against hypoxic ischemia damage [14]. Even after extended periods of cardiac arrest, users of ACP have reported less transient neurological impairments in the post-operative period, demonstrating that the approach improves oxygen transport to the cerebral capillaries [15].

An axillary or innominate conduit is used in the most popular method of treating ACP. The right carotid can be perfused, supplying blood to the brain, by pinching the base of the innominate artery [9]. At temperatures between 23 and 28 °C, the majority of surgical groups employing ACP perfuse the brain at a rate of roughly

8–12 ml/min/kg of body weight, with a perfusion cerebral oximetry pressure of 40–60 mmHg [16].

In terms of brain protection, the goals of ACP during HCA are to satisfy the needs of brain metabolism, maintain the brain's metabolic waste away during ischemia, and restore brain temperature to specific levels. Ensuring sufficient back bleeding through the cannula, avoiding large cannulas, avoiding inserting the cannula more than a few centimeters into the artery, and tying the purse string suture after decannulation are all crucial for the successful use of this technique [15].

In order to preserve the cerebral oxygen supply and to purge the atherosclerotic material and air produced during the procedure, retrograde cerebral perfusion (RCP) entails the retrograde perfusion of cold, oxygenated blood into the superior vena cava [17]. More uniform cerebral cooling, the removal of air bubbles, embolic debris, and metabolic waste products, the inhibition of cerebral blood cell microaggregation, and the provision of oxygen and nutritional substrates to brain tissue are some of the ways that RCP may provide neuroprotection [18].

Perfusate is used to administer flow through the cannula in RCP at temperatures between 10°C and 12°C, with pressures of 25 mmHg and flow rates between 300 and 500 mL/min. In RCP, the SVC is cannulated, and oxygenated blood is infused retrogradely [9,19].

Since RCP does not significantly provide glucose or oxygen to the brain, it is frequently used in conjunction with DHCA to give broader tissue protection. However, RCP de-airs the arterial system while maintaining arterial pressurization and rapidly cools the brain with little flow resistance [9].

Atheroma Management- Atheromatous aortic debri, which can embolize to several arterial beds, including the brain, is a major source of harmful embolic material from the aorta itself. Aortic atheromatous plaque damage during heart surgery may put a patient at risk for postoperative atherothromboembolism, which could help to explain why delayed stroke occurs after heart surgery [17,20].

To reduce the amount of atheromatous material that is released from the aorta wall and embolizes into the cerebral circulation, several strategies might be employed. These include using customized cannulae that result in less "sandblasting" of the aorta wall and placing the aortic cannula in a location that is comparatively free of plaque. utilizing all arterial grafts to prevent proximal anastomosis, conducting circulatory arrest and replacing the ascending aorta, preventing aorta cross clamping by employing fibrillatory arrest, and avoiding CPB by switching to "off pump" surgery. The atheromatous ascending aorta can now be "knowledgeably avoided" in terms of cannulation, clamping, and the location of proximal vein graft anastomosis with respect to the use of TEE and epiaortic scanning [17, 21, 20].

Acid- Base Management- As the temperature drops, a gas becomes more soluble in blood. As a result, after blood gas samples are adjusted for temperature during hypothermic bypass, the patient seems to have respiratory alkalosis, with a high pH and low PaCO2. By raising the PaCO2, the pH can be brought back to the normal range; this technique is called pH-stat analysis. Another option is to leave it uncorrected; this is referred to as the alpha-stat analysis [22].

Alpha-stat, the most often used blood gas management standard during adult CPB, reduces the risk of emboli while preserving normal cerebral blood flow (CBF) autoregulation by linking cerebral metabolism to CBF. This enables for an adequate oxygen supply. The most recent findings support the use of pH-stat control in congenital cardiac surgery because it can maintain brain cooling homogeneity prior to circulation stoppage. However, during pH-stat control, the pH exceeds what is necessary for the brain's metabolic demands [18,20].

Study	n	Findings
Murkin et al.	316	Using primary endpoints, the frequency of cognitive impairment is the same for alpha-stat and pH-stat management. A advantage with alpha-stat was indicated by further analysis when the CPB length was more than 90

		minutes. It is unclear whether numerous comparisons were adjusted for
Stephan et al.	65	Neurologic deficits more common 7 days after surgery with pH-stat versus alpha-stat management. Psychometric testing was not performed and long-term results were not reported [23].

Table 1: Randomized clinical studies examining how acid-base control during CPB affects neurological issues.

Glycemic Control- Up to 75 percent of patients experience hyperglycemia during CPB, which is characterized as a blood glucose level of greater than 180–200 mg/dl. In people who already have diabetes mellitus, the prevalence is even higher. The stress response to surgery and CPB, which is characterized by a marked increase in circulating catecholamines and cortisol, the exogenous administration of glucose-containing solutions (dextrose-containing cardioplegia and pump prime), and hypothermia-induced insulin resistance because of its capacity to maintain brain cooling homogeneity before circulating arrest are some of the underlying mechanisms [18,20].

Long-term hyperglycemia increases the risk of death and morbidity, increases the frequency of infections, impairs hemodynamics, increases platelet aggregation, and results in longer and more costly hospital admissions. Appropriate blood glucose monitoring, hyperglycemia prevention, and treatment are necessary to prevent this. Urine and arterial and mixed venous blood samples are taken before CPB (and before heparin is administered), at regular intervals during CPB, at the end of CPB, and when the patient first arrives in the intensive care unit. Hemofiltration replacement therapy with buffered 0.9% normal saline or insulin administration is the clinical treatment for hyperglycemia during CPB [24,25].

In addition to this, as suggested by Murkin, avoidance and limitation of some of the following will effectively limit hyperglycemia with expectation of enhanced neurologic outcomes: 1) glucose containing intravenous, cardioplegic, and pump-

priming solutions; 2) enhanced awareness and treatment of catecholamine-induced hyperglycemia; and 3) more aggressive insulin dosing strategies [26].

Emboli Reduction- The brain is subjected to an almost continuous shower of macro- and micro-particulate matter during CPB. These macro and microemboli come from a variety of sources and are believed to be factors in the genesis of postoperative cerebral dysfunction [27]. Emboli that develop within the extracorporeal perfusion systems can be divided into three broad categories: gaseous, blood-derived, and foreign material [28].

TYPE	SOURCE
Gaseous emboli	Bubble oxygenator, opening a beating heart, temperature gradients, intravenous solutions, emptying of reservoir, improper priming of the CPB circuit, reversal of vent or perfusion lines in pump head.
Blood-derived emboli	Operative field cardiotomy suction, muscle/connective tissue fragments, inadequately anticoagulated blood, activation of inflammatory response.
Foreign material emboli	Antifoam compounds in oxygenators, spallation from roller pumps, CPB circuit, inorganic debris in crystalloid solutions, banked blood [27,28].

Table 2: Table representing the type of emboli and its source

The strategies to prevent or minimize embolism during CPB include:

1. Anticoagulation regimens tailored to each patient to reduce platelet activation and coagulation factor consumption during CPB.
2. Utilizing CPB circuits coupled to covalent heparin reduces cellular aggregation formation, complement activation, and inflammation.
3. The use of membrane oxygenators, which, in contrast to bubble oxygenators, offer better oxygenation, cause less damage to the blood components, and lessen the occurrence of microembolism.

4. Insufflation of CO2 into the surgical field displaces air decreasing the nitrogen content of the gaseous emboli.
5. Filter and bubble detector inclusion in the arterial line, to prevent the introduction of air into the aorta.
6. Reduction in CPB time and use of centrifugal pumps for longer duration surgeries to avoid spallation.
7. Minimization of cardiotomy suction, since the cardiotomy blood contains a variety of substances including bone wax, marrow, in addition to RBCs, plasma, and inflammatory mediators.
8. The cardiotomy suction aspirate must be processed through a cell saver before returning it to the CPB circuit to reduce lipid embolism.
9. Large temperature differences (>14º C) between arterial blood and patient should be avoided during both cooling and rewarming.
10. Improving temperature regulation for cerebral reperfusion and rewarming following severe hypothermia. preventing the temperature from rising above 37°C, as this could exacerbate cerebral ischemia.
11. Proper priming procedures can reduce the number of foreign particles entering the system. Crystalloids, colloids, and blood should be filtered to remove foreign particles.
12. The perfusionist must always be alert for membrane tears and holes during priming and operation and must be careful not to have poor connections or open stop cocks through which air can enter the system.
13. Use of certain safety devices like bubble detectors, bubble traps, level sensors and level alarms also help to prevent the introduction of air into the patient.
14. Prevention of air entering the heart is best accomplished by not opening cardiac chambers while the heart is beating.
15. Aortic venting must be done before the termination of CPB for effective evacuation of air from the ascending aorta [5,27,28,29,30,31].

Cerebral Blood Flow (CBF) Management- CPB can seriously disrupt the autoregulation of CBF. The mean arterial pressure (MAP) determines CBF. Therefore, hypoperfusion and eventual cerebral ischemia may result from a low MAP. Increased intracerebral pressure brought on by a high MAP may result in cerebral edema [19]. The autoregulatory curve still "breaks" at a pressure of about 55 mmHg, despite the fact that hemodilution linked to CPB raises CBF for any given MAP; below this, CBF is compromised. Therefore, keeping MAP between 50 and 60 mmHg improves collateral flow and perfusion of the peri-ischemic area when embolic events do occur and helps to sustain CBF even in the context of carotid, cerebral, and penetrating vascular illness [32].

Hemodilution And Hematocrit Management- Two crucial variables affecting brain oxygen delivery are CBF and arterial oxygen content. The concentration of oxygen saturation in arterial hemoglobin, in turn, effectively determines the arterial oxygen content. By reducing hemoglobin concentration (and thus hemocrit) by a third, hemodilution in CPB raises CBF while decreasing blood viscosity and vascular resistance. Hemodilution is frequently used in hypothermic CPB because blood viscosity increases with hypothermia. This rheologically-mediated increase in CBF promotes cerebral oxygen supply as haematocrit decreases, therefore the cerebral metabolic rate of oxygen stays independent of haematocrit throughout a wide range. However, as hemodilution progresses, cerebral oxygen demand and, eventually, oxygen consumption are hampered when the CBF response can no longer compensate for the loss in arterial oxygen content and oxygen extraction capacity is depleted, potentially leading to cerebral ischaemia. Hence, it is reasonable to aim a haematocrit of less than 30% if the temperature is dropped to 30ºC. Lower haematocrits, often below 25%, are desired if temperatures are reduced to 25ºC. Furthermore, as demonstrated by Rosengart and colleagues, the use of retrograde autologous priming (RAP) of the CPB circuit is an efficient technique of reducing considerable hemodilution and frequency of RBC transfusion during cardiac procedures [5,23,26,33,11].

Pulsatility- The lack of pulsatile flow, like the abrupt changes in temperature and haematocrit that can occur during CPB, is a physiological phenomenon specific to CPB, as nonpulsatile flow is the most commonly employed kind. Nonpulsatile CPB has been shown to produce a loss in haemodynamic energy, leading in capillary collapse, microvascular shunting, and activation of inflammatory mediators; in CPB patients, this is associated with anaerobic metabolism, lactate buildup, and multiple organ failure. Nonpulsatile CPB has also been linked to increased cerebral ateriovenous oxygen gradient, cerebral oedema, higher cerebrospinal fluid creatine kinase, and an increase in acute cerebral cellular damage. The pulatile perfusion approach, on the other hand, resembles the pulsatile character of human blood flow and has been shown to produce haemodynamic energy levels that improve vascular bed patency and blood flow to essential organs. It has also been demonstrated to reduce postoperative mortality by ensuring capillary patency, lowering systemic vascular resistance, and increasing erythrocyte velocity inside capillaries. Therefore, pulsatile CPB has been advocated for better postoperative results [11, 20,33,34].

Anti-Inflammatory Therapies- Systemic inflammatory response syndrome (SIRS) is thought to be caused by humoral and cellular activation brought on by interactions between the patient's circulating blood and the artificial material surfaces of the CPB circuit. The SIRS is becoming more widely acknowledged as an unavoidable reaction to cardiac procedures involving CPB. The adhesion, aggregation, and eventual microembolism of neutrophils and other blood cellular elements are characteristics of these disorders [16]. Several commercially available extracorporeal devices, such as leukocyte-depleting filters, condensed extracorporeal circuits, and biocompatible surface coatings, have shown better results and a reduction in the inflammatory response to extracorporeal circulation [5,26]. For instance, the decrease in incidence of SIRS with the use of biocompatible surface technology is well documented by Satio and associates [26].

Pharmacologic Neuroprotection

The development of pharmaceutical treatments for neuroprotection is the result of numerous advancements in our understanding of the fundamental mechanisms behind brain injury. Pharmacologic neuroprotection can take at least three forms: antiadhesive drugs, medicines that block various stages of the cellular ischemia cascade, and metabolic depressants [6].

Metabolic suppressants- Consideration of metabolic suppression as a neuroprotective strategy is immediately prompted by the basic notion of ischemia as a condition of cerebral O2 delivery that is insufficient to meet O2 demand [6]. Consideration of metabolic suppression as a neuroprotective strategy is immediately prompted by the basic notion of ischemia as a condition of cerebral O2 delivery that is insufficient to meet O2 demand [19,20].

Agents that inhibit ischemic pathways- Calcium antagonists: By encouraging microembolism and favoring platelet aggregation and BBB damage brought on by phopholipase A2 activation, intracellular calcium buildup is one of the main causes of cell death in cerebral ischemia. Hence, the use of calcium channel blockers such as nimodipine has been shown to prevent cerebral ischemia associated with transmembrane calcium flux [5,6,19,20].

N-methyl-D-aspartare (NMDA) receptor antagonists: NMDA receptors which bind to glutamate can lead to neuronal excitotoxicity. This can be reduced by using glutamate NMDA antagonists such as magnesium, s(+)-ketamine, remacemide, xenon and dextromethorphan [5,20].

Anti-inflammatory and Antiadhesive agents- SIRS is thought to have a significant role in the spread of ischemia-mediated brain damage. Because corticosteroids can lower the inflammatory response, they have long been thought of as possible neuroprotective drugs. Aprotinin which is a non specific serine protease enzyme inhibitor with broad spectrum anti-inflammatory properties has also been shown to decrease white cell activation and transmigration significantly [6,20].

Drugs	Proposed primary mechanism	Study	n	Type of surgery	Main findings
Thiopental	↓ CMRO2	Nussmeier et al.	182	Valvular	Reduced cognitive issues ten days following surgery
		Zaidan et al.	300	CABG	Neurologic results from thiopental and placebo were same
Propofol	↓ CMRO2	Roach et al.	225	Valvular	Cognitive problems do not change five to seven days or fifty to seventy days following surgery
Nimodipine	Ca2+ channel blockers	Legault et al.	150	Valvular	The study ended early because there was no proof that nimodipine improved cognitive outcomes and the treatment group had a higher mortality rate than the control group
Remacemide	NMDA receptor antagonist	Arrowsmith et al.	171	CABG	Remacemide improved global cognitive function and performance on three of ten psychometric tests

Aprotinin	Mechanism unknown; may be due to ↓ inflammatio-n/↓ pericardial aspirate	Levy et al.	287	CABG	No strokes in 'high' and 'low' dose aprotinin groups versus controls and 'pump' prime only groups.
		Harmon et al.	36	CABG	Six weeks following surgery, the lower aprotinin group experienced cognitive losses compared to the placebo group
		Mangano et al.	4374	CABG	An observational research that used propensity-adjusted, multivariable logistic regression revealed an 181% increase in the risk of stroke or encephalopathy
Lidocaine	Na+ channel blockade; membrane stabilization/ ↓EAA release	Mitchell et al.	55	Valvular	The lidocaine group had a superior neurocognitive outcome ten days and ten weeks following surgery than the placebo group, but not at six months
		Wang et al.	42	CABG	Nine days following

						surgery, neurocognitive function improved when lidocaine was used instead of a placebo.
Pexelizumab	↓C5a and C5b-9		Mathew	800	CABG	It had no effects on global cognition but did lower decline in the visuospatial domain compared with placebo [20,23].

Table 3: Pharmacologic neuroprotection trials for people undergoing CPB

Neuromonitoring: For patients having heart surgery, neuroprotective measures should include real-time neurologic monitoring. Monitoring should ideally make it simple, dependable, and repeatable to identify harmful neurologic occurrences. Although there are several monitoring techniques available, they are not always used [21].

Electroencephalography (ECG): During heart surgery, this method has been employed in several centers to track electrocortical activity. The primary source of cerebral oxygen consumption and the main focus of tactics meant to reduce the cerebral oxygen metabolic rate is synaptic activity. During surgery, EEG can be used to continuously check the effectiveness of this type of synaptic inhibition. However, EEG has certain limitations as a monitor of cerebral function during surgery. One major drawback is its inability to capture nonsynaptic metabolic activity, which can continue even after the EEG becomes isoelectric. Furthermore, EEG gives limited insight into subcortical neural activity and largely reflects surface cortical activity. However, EEG may offer important insights on electrophysiologic recovery and seizure incidence in the early post-operative phase [33].

Doppler Sonography: A portable, noninvasive method for measuring the blood flow velocity (BFV) in the middle cerebral artery (MCA) continuously and in real time is transcranial Doppler (TCD) sonography [10]. Additionally, it offers a numerical evaluation of cerebral perfusion. Cerebral autoregulation, chemoreactivity, and flow-metabolism coupling can all be evaluated by TCD sonography, which also offers continuous data on MCA blood flow. TCD sonography can also be used to identify and measure embolic events. The major drawback with TCD sonography is that its utility in judging cerebral blood flow is predicated on the assumption that the that the diameter of the MCA is constant, which has not been adequately established [27].

Near-Infrared Spectroscopy (NIRS): The brain hemodynamics and oxygenation may be continuously monitored at the bedside thanks to this newly created in vivo technique. It is predicated on two fundamental ideas: (a) that near-infrared light may readily pass through bone, soft tissues, and skin to reach the brain; and (b) that hemoglobin and cytochrome aa3, the last enzyme in the electron transport chain, absorb near-infrared light in an oxygen-dependent manner. The capacity to concurrently measure intracellular (cytochrome aa3) and intravascular (oxyhemoglobin) cerebral oxygenation at the bedside is what distinguishes this method. This is conceivable as mitochondrial oxygen availability affects cytochrome aa3's oxidation state. It has been demonstrated that this method may successfully examine the intricate alterations in brain hemodynamics and metabolism that follow heart surgery in both adults and children [27]. One of NIRS's drawbacks is that it only tracks the frontal cerebral cortex; it cannot evaluate deeper brain regions or other parts of the cortex [21].

Jugular Venous Oxyhemoglobin Saturation (Sjvo2): This technique measures cerebral venous oxygen saturation globally directly from the jugular bulb. Because jugular saturation depends on both cerebral blood flow and cerebral oxygen extraction, it can be used as an indirect measure of cerebral oxygen metabolism. Careless use of any technique that measures cerebral parenchymal oxygenation using intravascular oxygenation can lead to misinterpretation. In circumstances of

severe hypothermia, setting a high jugular saturation could be deceptively reassuring, for example, because the rise in oxyhemoglobin affinity makes it more difficult to interpret jugular saturation readings. However, a declining jugular saturation can be a precursor to rising cerebral oxygen extraction and, thus, imminent cerebral hypoxia-ischemia when blood temperature and pH remain constant [21].

Biochemical Markers: The idea behind the detection of biological markers in serum and cerebrospinal fluid (CSF) is that these substances arise outside of the central nervous system (CNS) when neuronal damage disrupts the normal blood-brain barrier [35]. The following biomarkers have been the most often researched:

1. Adenylate kinase: up to 50% of patients had elevated CSF levels, which are linked to cerebral hypoperfusion, hypothermic surgery, and cognitive impairment.
2. Thymic tissue, lipid mediastinum cells, Schwann cells, and astrocytes all contain the calcium-linked protein S100β. White matter cell death from severe trauma, Alzheimer's illness, Down syndrome, acute ischemic or hemorrhagic stroke, or coma from prolonged cardiac arrest can all increase blood levels of S100β. S100β levels only rise when CPB is used during heart surgery. It has a two-hour plasma half-life and is eliminated by the kidneys.
3. N-acetyl aspartate (NAA): Only neural tissue contains NAA. Magnetic resonance (MR) spectroscopy is used to measure NAA, which assesses mitochondrial function. Following CPB, there is a direct correlation between cognitive decline and a decline in the NAA/creatine ratio, which may recover during follow-up. Neuronal damage from stroke or cranial trauma is associated with a drop in NAA values [5].

These biomarkers have various limitations even though they might be very helpful in identifying brain impairment while using CBP. Until 24 hours following surgery, blood levels might not be correlated with the degree of neurologic impairment; assays are typically expensive, and several assays would be needed. These

indicators typically have complicated kinetics, and it can occasionally be challenging to interpret their values. Finally, research on the relationship between serum biomarkers and cognitive decline has yielded mixed findings, and no link to the disability has been discovered as of yet [5].

DISCUSSION

Cardiac surgery carries severe and unavoidable risks of neurologic and cognitive impairment. CPB is associated with a number of physiological factors that either predispose or exacerbate neurologic problems. Prominent among these factors are perfusion flows and pressures, the inflammatory response to CPB, changes in CNS temperature, acid-base management, metabolic disturbances such as hyperglycemia, and embolic phenomena [6]. To create measures to lower the incidence of these issues, it is crucial to identify those who are at risk for perioperative neurologic adverse events. There are two primary approaches to preventive measures aimed at preventing neurologic damage: Changes to surgical methods and the application of neuroprotective medications [5].

Slow rewarming with input temperatures no higher than 37°C and keeping the systemic temperature between 33°C and 34°C, keeping a MAP above 50 mmHg to ensure a normal cerebral perfusion pressure, ideal acid-base management, aiming for a hematocrit of more than 25% during CPB, avoiding atherosclerotic ascending aorta manipulation with appropriate surgical technique, preserving euglycemia, using biocompatible circuits, using various filters to prevent embolic injury, and avoiding retransfusion of unprocessed cardiotomy suction blood are all examples of enhanced perioperative neuroprotective surgical strategies [20].

It has been demonstrated experimentally that pharmacologic therapies involving substances such as lidocaine, corticosteroids, propofol, barbiturates, and calcium channel blockers can lessen the neurologic damage caused by heart surgery [21].

Lastly, biomarkers like S100β protein and intraoperative neurologic monitoring techniques like TCD, EEG, and NIRS can be used to determine intraoperative CBF and tissue oxygenation. They can also be used to identify patients who are more likely to sustain long-term damage and can serve as markers of hemodynamic instability [36].

CONCLUSION

It can be observed that there are a variety of interventions available to decrease cognitive impairment related with cardiac surgery. Intraoperative monitoring combined with improved operative methods and pharmacological interventions, helps detect, prevent, and control neurological damage following cardiac surgery.

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