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A Case Report on DHCA

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Overview

Case Presentation

Case

A 74-years-old man with severe AI and Fusiform Aneursym in ascending thoracic aorta was scheduled for CABG and Bentall operation

Past Medical History

- HTN
- DM
- EF Rhythm
- Carotid Stenosis
- Renal Cyst

Drug History

- Tab Metformine 500/BD
- Tab Digoxin

Lab Test

- Na = 138
- K = 3.7
- FBS = 110
- BUN = 28
- Cr = 1.5
- COVID-19 PCR = Negative

Echocardiography

- Severe AI
- Ascending Aorta $= 3.4cm$
- Mild-to-moderate PI
- Moderate TR
- Mild-to-moderate MR
- Moderate LV enlargement
- EF $= 35 - 40\%$

Doppler U/S

In LCCA bulb and LICA origin a circumferential calcified, homogenous, regular plaque measured $7 \times 3.5mm$ with 40% stenosis is seen.

Spiral Chest CT

- Right side plevral effusion
- MPA dilatation
- A few atelectatic band in mid and lower zone

CT Angiography

- Aortic annulus $= 26mm$
- Aortic sinus $= 39mm$
- Fusiform aneurysm in ascending thoracic aorta with $54mm$ diameter
- Maximum diameter of arch in mid arch is $41mm$

Induction

- BP = 160/90
- O_2Sat = 97
- Cerebral Oximetery = 50-70

- Midazolam = $5mg$
- Etomidate = $20mg$
- Cisatra = $20mg$
- Maintenance = SAM

Lab Test after Induction

Air

- PH = 7.42
- $PO_2 = 78$
- $O_2Sat = 96$
- $PCO_2 = 41$
- $HCO_2 = 30$
- $TCO_2 = 31$
- BE = 5
- Na = 139
- K = 3.6
- Hb = 13.3
- HCT = 43
- BS = 90
- Lactat = 0.5
- Cl = 109
- Ca = 1.9

DHCA

From the second hour, the patient gradually became hypothermic to a temperature of 20 C for about an hour and a half and the patient's MAP decreased to about $20 mmHg$.

Cerebral Oximetery = 39/50 53/48 31/39

Medications

Medications that the patient received during hypothermia include:

- Phenobarbital $= 200mg$
- Thiopantal Sodium
- Dexamethazon $= 8mg$
- Topical Cerebral Cooling

Off Pump

- PH = 7.49
- O_2Sat = 100
- PCO_2 = 31
- HCO_2 = 24
- TCO_2 = 25
- BE = 2
- Na = 137
- K = 4.2
- Hb = 9.6
- BS = 166
- Lactat = 3.6
- Ca = 1.6

- Infusion Milrinone
- Infusion Epinephrin
- Fibrinogen $1gr$
- Calcium $10cc$
- Albumin $200gr$

Introduction to DHCA

Most cardiac surgical procedures can be accomplished using cardioplegia-induced cardiac arrest and cardiopulmonary bypass (CPB) to maintain perfusion of other organs. In some situations, however, the underlying pathology or the nature of the surgery proposed necessitates complete cessation of the circulation. The use of profound systemic hypothermia to preserve organ function during cessation of the circulation is termed deep hypothermic circulatory arrest (DHCA).

DHCA

DHCA provides excellent operating conditions while reducing the consequences of organ ischaemia. As the brain is the organ most susceptible to ischaemia during circulatory arrest, it follows that other organs will also be protected by this strategy.

Indications for DHCA

Cardiac	Aortic surgery
	Pulmonary thromboendarterectomy
	Complex congenital surgery
Neurological	Cerebral aneurysms
	Arterio-venous malformations
Other	Renal cell carcinoma with caval invasion
	Other tumours with caval invasion

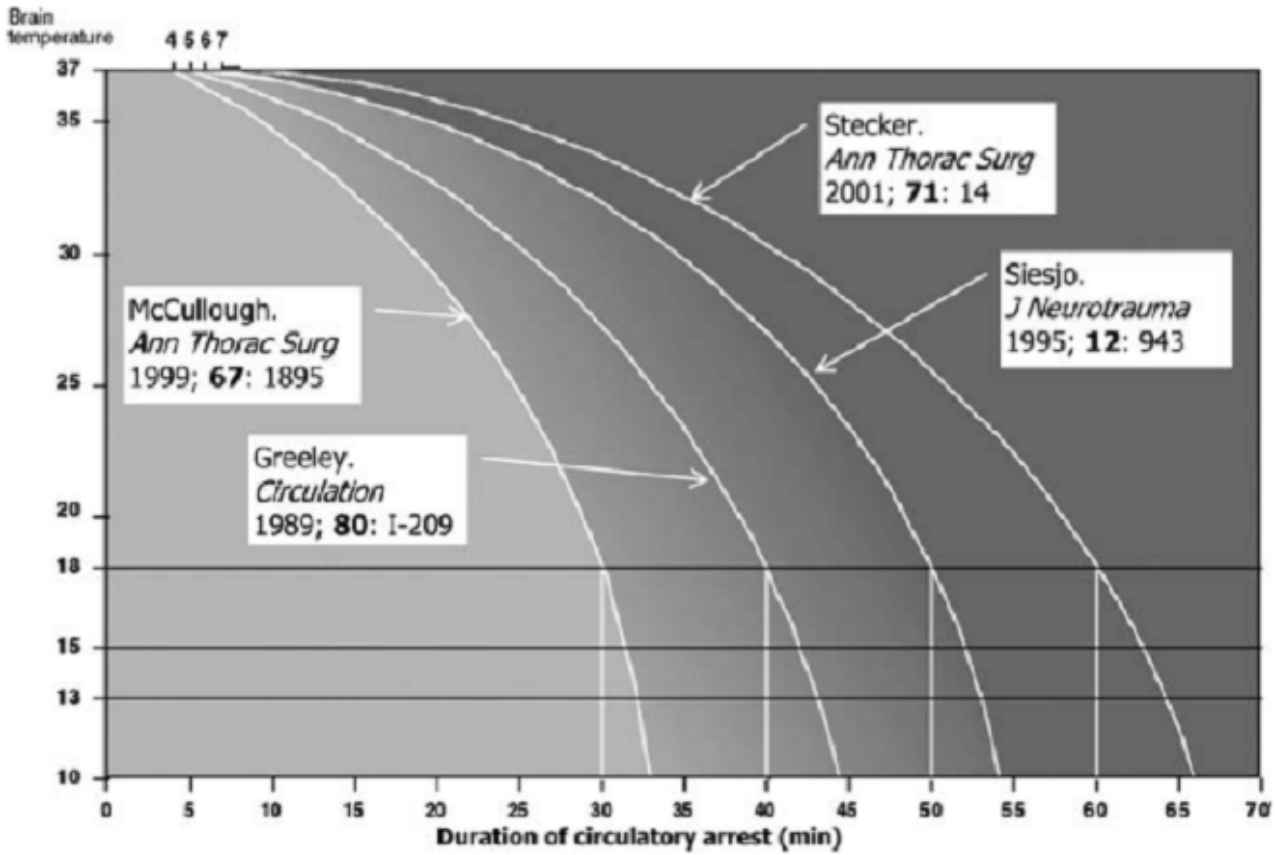
Table: Indications for DHCA

Adverse Consequences of Hypothermia

Cardiovascular	Arrhthymias secondary to potassium loss Increased plasma viscosity Vasoconstriction impairing microcirculation
Coagulation	Impaired coagulation Reduced platelet count
Renal and metabolic	Reduced glomerular filtration rate Metabolic acidosis Hyperglycaemia secondary to impaired glucose metabolism Effects on pharmacodynamics and pharmacokinetics
Cerebral	Vasoconstriction during cooling Brain injury from hyperthermia during rewarming

Table: Adverse consequences of hypothermia

Duration of Circulatory Arrest



Safe period for DHCA

At normothermia, brain injury occurs after around 4 min of circulatory arrest. Cerebral metabolism decreases by 6–7% for every 1 ℃ decrease in temperature from 37 ℃; therefore, brain cooling results in a reduction in oxygen requirements. Circulatory arrest is typically undertaken at 18–20 ℃ and a range of safe periods for DHCA have been reported at this temperature. Most patients tolerate 30 min of DHCA without significant neurological dysfunction, but when this is extended to longer than 40 min, there is a marked increase in the incidence of brain injury. Above 60 min, the majority of patients will suffer irreversible brain injury, although there are still a small number of patients who can tolerate this. Longer periods of DHCA are tolerated in neonates and infants compared with adults.

Anaesthesia for DHCA I

All patients should undergo thorough preoperative assessment and be prepared for the extent of invasive monitoring and the potential for prolonged postoperative intensive care. When a patient requires emergency surgery, however, detailed assessment and prolonged discussion may be neither practical nor possible. Sedative premedication may be appropriate for patients undergoing an elective procedure involving DHCA, although care should be taken in patients in whom respiratory depression and hypoxia may induce haemodynamic disturbance—for example, patients with severe pulmonary hypertension.

Anaesthesia for DHCA II

The use of corticosteroids should be considered, as there is some evidence—albeit inconclusive— that they are neuroprotective. This is thought to occur by decreasing the release of inflammatory cytokines and preventing lysosomal breakdown during hypothermia. To be effective, corticosteroids (e.g. prednisolone 1 mg kg^{-1}) should be administered at least 6–8 h before surgery, any resultant hyperglycaemia is probably best treated using insulin.

Induction and Monitoring I

In addition to routine non-invasive monitoring, all patients undergoing surgery with DHCA require invasive arterial and central venous pressure monitoring. In some instances, femoral or bilateral radial arterial monitoring may be appropriate. In addition to allowing measurement of central arterial pressure, placement of a femoral arterial cannula may aid the surgeon should an intra-aortic balloon pump be required. Measurement of right heart pressures using a pulmonary artery catheter or the use of transoesophageal echocardiography (TOE) may be indicated in some patients. Temperature monitoring at two sites, typically the nasopharynx and bladder, is used to estimate brain and body temperatures, respectively. Studies have shown that bladder and tympanic temperatures correlate most closely with brain temperature. There is poor correlation between pulmonary artery catheter and rectal temperature measurements, and brain temperature.

Induction and Monitoring II

Despite limited evidence of outcome benefit, neurological monitoring is routinely used in many centres. Broadly speaking, these fall into one of two categories: monitors of cerebral substrate delivery [jugular bulb oximetry, transcranial Doppler sonography, and near infrared spectroscopy (NIRS)] and monitors of cerebral function [quantitative electroencephalography (qEEG) and evoked potential monitoring].

Induction and Monitoring III

Measurement of jugular venous oxygen saturation (Sj_{O_2}) provides an indication of the balance between cerebral oxygen supply and demand. Its use is limited as Sj_{O_2} reflects global cerebral blood flow and may be unaffected by regional cerebral ischaemia. Low Sj_{O_2} before the start of DHCA has been shown to be associated with adverse neurological outcome.

Induction and Monitoring IV

Transcranial Doppler is used to measure cerebral blood flow velocity (a surrogate for cerebral blood flow) in the basal cerebral arteries and to detect microemboli. Using paired optical sensors placed on the scalp, NIRS measures the oxygen saturation of blood in all vessels to a depth of 20–40 mm. Ease of use and limited potential for harm have prompted the increasing use of NIRS monitoring.

Induction and Monitoring V

qEEG is a sensitive monitor of cerebral ischaemia but may be affected by electromagnetic interference, hypothermia, and anaesthetic agents. In some centres, the EEG is used to assess the adequacy of cooling before DHCA. However, the temperature at which the EEG becomes isoelectric varies widely between patients—as low as 12.5 $^{\circ}\mathrm{C}$ in some studies—and appears to be higher during cooling than rewarming.

Induction and Monitoring VI

The choice of anaesthetic drugs is largely a matter of personal and institutional preference. Theoretically, avoiding volatile agents, which uncouple cerebral blood flow from cerebral metabolism (autoregulation), may confer some benefit. The influence of hypothermia on drug metabolism and elimination should be considered and doses altered accordingly.

Anticoagulation

Unfractionated heparin (3–5 mg kg^{-1}) is typically used to maintain an activated clotting time (ACT) > 400 s. Inadequate anticoagulation during CPB increases the consumption of clotting factors and may paradoxically worsen postoperative coagulopathy. Lysine analogues (e.g. tranexamic acid, ϵ-aminocaproic acid) are frequently used to reduce fibrinolytic haemorrhage. It should be borne in mind that tranexamic acid may cause postoperative seizures.

General considerations

The duration of surgery conducted with DHCA mandates careful attention to the prevention of pressure sores and damage to the eyes, nerve plexuses, and peripheral nerves. Particular attention should be paid to cannulation sites, monitoring lines, airway connectors, and TOE probe which may cause pressure necrosis of the skin.

Measures should be taken to assist rewarming and prevent hypothermia after separation from CPB. An i.v. fluid warmer, heated mattress, and forced-air blanket should be considered in all cases.

Cerebral protection I

Systemic cooling is achieved during CPB by pumping water from a heater/cooler reservoir through a heat exchanger in the CPB oxygenator. The temperature gradient between water and blood is typically maintained at ,$< 10^{\circ}\mathrm{C}$. Additional cerebral cooling can be achieved using a head-cooling jacket through which iced water is circulated or ice packed around the head. Although topical cooling of the head has been shown to be of some benefit in animal models, there is as yet no evidence of outcome benefit in humans.

Cerebral protection II

When CPB is resumed after a period of DHCA, hypothermic perfusion should be maintained for 10–20 min before rewarming commences. This is thought to reduce the risk of raised intracranial pressure which can occur during this period. Once rewarming commences, the gradient between core and peripheral temperatures should be ,$< 5^{\circ}\mathrm{C}$. Excessively rapid rewarming with perfusion temperatures $> 37^{\circ}\mathrm{C}$ may induce cerebral ischaemia secondary to an imbalance between oxygen supply and demand. Similarly, cerebral hyperthermia should be avoided as this may exacerbate neurological injury and increase the risk of adverse neurological outcomes.

Haemodilution

During hypothermia, the combination of increased plasma viscosity, erythrocyte rigidity, and progressive vasoconstriction leads to impairment of the microcirculation. Haemodilution, typically to a haematocrit of 20%, is thought to improve flow in the microcirculation.
Excessive haemodilution (e.g. haematocrit $<$10%) significantly reduces oxygen carrying capacity and causes tissue ischaemia.
Normovoaemic haemodilution is often achieved by removing heparinized blood via the arterial cannula immediately before commencement of CPB. Any blood removed is labelled and stored for retransfusion during rewarming.

Pharmacological Neuroprotection I

Drugs used for this purpose included thiopental (59%), propofol (29%), and others (48%), most commonly corticosteroids.
Thiopental and propofol have both been investigated at doses sufficient to cause burst suppression. Propofol does not appear to be associated with cardiac depression or delayed emergence from anaesthesia.
Although the use of thiopental is associated with dose-dependent myocardial depression and delayed emergence, it does not adversely impact on the ability to separate from CPB. There is no evidence that either drug improves neurological outcome in adults.

Glycaemic Control

There is evidence that hyperglycaemia during periods of hypothermia worsens the impact of ischaemia through increased glycolysis and intracellular acidosis. Virtually, all patients undergoing DHCA develop impairment of glucose metabolism and will require control of glucose with insulin. Concurrent corticosteroid administration is likely to increase the risk of perioperative hyperglycaemia.

The End