

Contemporary Circulatory Arrest Conundrums

Phil Scott, CCP, FPP
Missouri Perfusion Society
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Debatable Issues...

- Hemodilution
- Temperature
- Steroids
- Qb Flow
- pH stat vs. alpha stat
- PO₂???
- ACP, RCP, Circulatory Arrest
- Cannulation sites, Etc...

Hemodilution

Should intentional dilution occur?



Prediction of safe duration of hypothermic circulatory arrest by near-infrared spectroscopy

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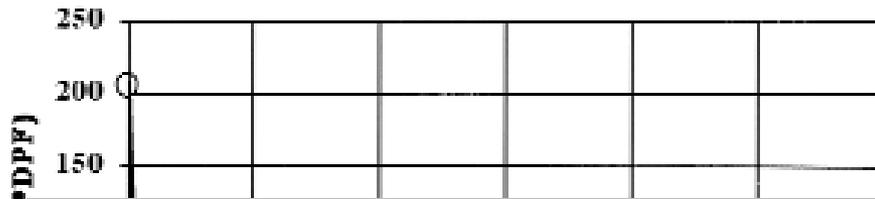
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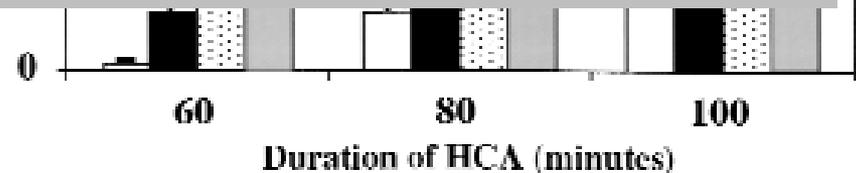
Thirty-six piglets (9.36 ± 0.16 kg) underwent circulatory arrest under varying conditions with continuous monitoring by near-infrared spectroscopy (temperature 15°C or 25°C, hematocrit value 20% or 30%, circulatory arrest time 60, 80, or 100 minutes). Each setting included 3 animals. Neurologic recovery was evaluated daily by neurologic deficit score and overall performance category. Brain was fixed in situ on postoperative day 4 and examined by histologic score.

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Oxygenated hemoglobin signal declined to a plateau (nadir) during circulatory arrest. Time to nadir was significantly shorter with lower hematocrit value ($P < .001$) and higher temperature ($P < .01$). Duration from reaching nadir until reperfusion (“**oxygenated hemoglobin signal nadir time**”) was significantly related to histologic score ($r_s = 0.826$), neurologic deficit score ($r_s = 0.717$ on postoperative day 1; 0.716 on postoperative day 4), and overall performance category ($r_s = 0.642$ on postoperative day 1; 0.702 on postoperative day 4) ($P < .001$). **All animals in which oxygenated hemoglobin signal nadir time was less than 25 minutes were free of behavioral or histologic evidence of brain injury.**

HbO2 decay curve**Time to nadir**

Oxyhemoglobin (*HbO2*) decay curve and normalized *HbO2* nadir time. During hypothermic circulatory arrest (*HCA*), the curve linear decrease in *HbO2* signal (*HbO2* decay curve) can be described by a logarithmic function, $HbO2 = a \log(t) + b$, $dHbO2/dt = a/t$, where t is the time after the onset of *HCA* and a and b are constants. We have defined that *HbO2* signal reaches the plateau state (nadir value) when the slope of fitting curve, namely the differential coefficient, $dHbO2/dt$, becomes more than -0.5 . *HbO2* nadir time was normalized by using the temperature coefficient of 2.5 for the young piglet.



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The aim of the present study was to determine a safe duration of HCA as assessed by neurologic and histologic examination in a survival piglet model with HCA and to study the influence of hematocrit value and temperature on safe duration.

Experimental Groups

Hematocrit value. During the cooling phase a hematocrit value of either 20% or 30% was maintained.

Duration of HCA. A circulatory arrest time of 60, 80, or 100 minutes was used.

Temperature during HCA. An esophageal temperature of either 15°C or 25°C was used.

Hematocrit

Before C

CPB prim

On CPB

Cooling 1

30 min

Rewarm

30 min

30 r

POI

Tempe

Bef

On

End

End

Rev

3 h

POI

0 ± 0.7

2 ± 0.4

2 ± 0.7

3 ± 0.4

2 ± 0.5

9 ± 1.6

29.4 ± 0.9

1.0

0.4

0.21

0.14

0.07

0.19

0.14

0.32

0.18

34.4 ± 0.7

27.4 ± 0.7

34.2 ± 0.7

29.4 ± 0.9

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Conclusion: Oxygenated hemoglobin signal nadir time determined by near-infrared spectroscopy monitoring is a useful predictor of safe duration of circulatory arrest. **Safe duration of hypothermic circulatory arrest is strongly influenced by perfusate hematocrit value and temperature during circulatory arrest.**

Problem? In the group with a hematocrit value of 20%, the CPB prime consisted of 400 mL of blood and 800 mL of crystalloid solution. The other group (hematocrit value of 30%) was prepared with 1200 mL of whole-blood prime.

Results

- Mean arterial pressure was significantly higher in group 3 (30%/25°C) during the cooling phase ($P < .0001$)
- Osmolarity was significantly higher in groups 1 (30%/15°C) and 3 (30%/25°C) during the cooling phase ($P = .0003$)
- The changes in percentage body weight were significantly different between the groups on POD 1. The groups assigned to higher hematocrit values gained less weight. However, body weights of almost all animals returned to the preoperative level and there were no significant differences on POD 4
- Calculated percentage of total body water content showed marked edema in groups 2 (20%/15°C) and 4 (20%/25°C) after weaning from CPB

Results

- The HbO₂ signal increased significantly during the cooling phase in all groups. In groups 1 (30%/15°C) and 3 (30%/25°C), the HbO₂ signal continued to increase during the entire cooling phase, whereas the HbO₂ signal reached a plateau after about 20 minutes in groups 2 (20%/15°C) and 4 (20%/25°C).
- Tissue oxygenation index demonstrated an increase without plateau in all groups, and there was significant difference between groups 1 (30%/15°C) and 4 (20%/25°C) at the end of cooling

Results

- In groups 2 (20%/15°C) and 4 (20%/25°C), HbO₂ declined to a plateau within approximately 30 minutes, whereas in groups 1 (30%/15°C) and 3 (30%/25°C), the plateau occurred after approximately 60 and 40 minutes, respectively
- Time to reach plateau was calculated in each case. In groups 2 (20%/15°C) and 4 (20%/25°C), this was 28.7 ± 2.4 minutes and 23.1 ± 1.7 minutes, whereas in groups 1 (30%/15°C) and 3 (30%/25°C), this time was 51.3 ± 4.1 minutes and 41.4 ± 2.9 minutes

Results

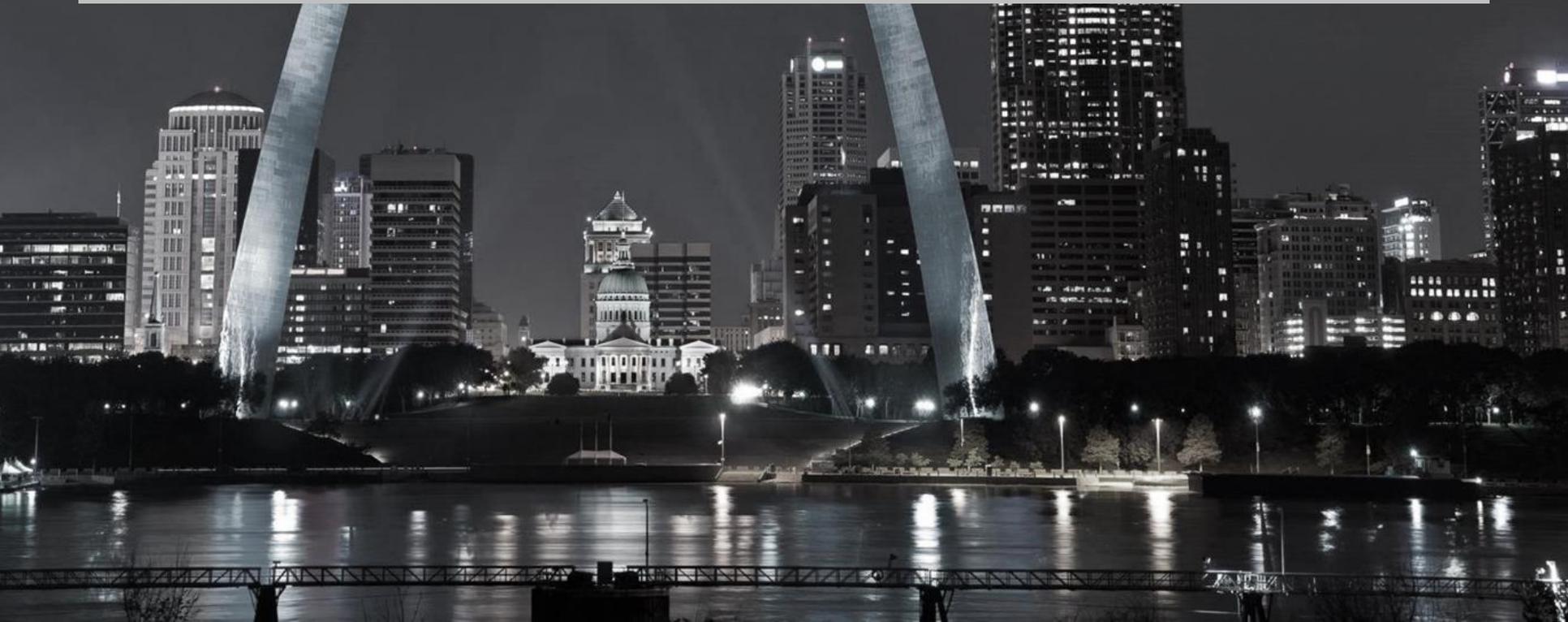
- Analysis of variance revealed a highly significant effect of hematocrit value ($P < .001$) and temperature ($P < .01$)
- Bonferroni t test indicated that group 1 (30%/15°C) is longer than group 2 (20%/15°C) and group 4 (20%/25°C) ($P < .001$ for each), and group 3 (30%/25°C) is longer than group 2 (20%/15°C) ($P = .02$) and group 4 (20%/25°C) ($P < .001$)
- No differences were detected between group 1 (30%/15°C) and group 3 (30%/25°C) ($P = .13$) or between group 2 (20%/15°C) and group 4 (20%/25°C) ($P = .98$)

Results

- Most animals assigned to 15°C recovered and showed normal performance without neurologic deficit by PODs 3 and 4. However, the animals assigned to 25°C with longer durations of HCA did not recover to normal.
- All 3 animals with the higher hematocrit value (30%), the lower temperature (15°C), and the shortest HCA (60 minutes) showed no brain damage.
- All animals with the lower hematocrit value (20%), the higher temperature (25°C), and the longest HCA (100 minutes) demonstrated severe brain damage
- There was a tendency for worse score when hematocrit value was lower, temperature was higher, and HCA was longer

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Conclusion: Oxygenated hemoglobin signal nadir time determined by near-infrared spectroscopy monitoring is a useful predictor of safe duration of circulatory arrest. Safe duration of hypothermic circulatory arrest is strongly influenced by perfusate hematocrit value and temperature during circulatory arrest.



Debatable Issues...

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Temperature



The Facts!

- Although the brain accounts for only approximately 2% of the body weight, it utilizes 20% of the resting total body oxygen consumption and receives almost 15-20% of the total circulating blood volume from the heart.
- The brain's metabolic rate of oxygen and **glucose consumption** is multiple times faster than other human organs.
- Oxygen-dependent glucose metabolism produces ATP, which is the main source of intracellular energy for neurons. Unlike liver or muscle tissues, **the brain does not have “storage” for glucose**, thus a shortage of its delivery immediately impairs the neuronal function
- Changes in levels of oxygen and/or glucose delivery can be compensated by appropriate changes of the blood flow, a phenomenon known as “**autoregulation of cerebral flow**”.

The Facts!

- Mechanisms by which hypothermia provides neuroprotection are involved in **two main pathways** of ischemic neural injury which are closely interrelated and not independent of each other.
- 1: Amidst a lack of oxygen **ATP is synthesized through anaerobic glycolysis which is not sufficient to maintain normal neuronal function.** Concurrently, lactate accumulates in the neurons, lowering the intracellular pH. Such energy depletion and waste product accumulation within brain cells leads quickly to permanent damage and necrosis.
- 2: Calcium ion plays a central role in ischemic neuronal injury. Hypoxia leads to a release of excitatory neurotransmitters, such as glutamate, which in turn activates the *N*-methyl-D-aspartate (NMDA) channels. Once these channels are activated, calcium ions easily enter the cells and accumulate. Such **imbalance in the calcium level leads to activation of intracellular proteases and mitochondrial dysfunction, which result in neuronal cell death**

The Facts!

- **Hypothermia inhibits both of these injury-inducing pathways.** It is well established that hypothermia significantly decreases the global cerebral metabolic rate for glucose and oxygen.
- For every one degree Celsius drop in body temperature, cellular metabolism slows down by an average of 5-7%. Hence, hypothermia actually decreases the demand of the brain cells for oxygen.
- At 18 °C the metabolic rate of the human body is only 12% to 25% of the metabolic rate when at normal temperature.
- The lower the rate of anaerobic metabolism, the less lactate is accumulated and the less pronounced is any cellular acidosis. Lowering the temperature has been proven to reduce to a larger extent ATP breakdown than its synthesis in the brain, which increases cerebral ATP supply for energy-consuming processes .

The Facts!

- At the same time, hypothermia significantly reduces temperature-dependent release and extracellular levels of excitatory neurotransmitters such as glutamate, an NMDA receptor agonist. An important factor in activation of NMDA receptors is interaction with glycine, the levels of which are depleted in the brain during hypothermic conditions.
- Hence this is a dual mechanism for decreasing the activity of the NMDA channels, which significantly reduces the amount of calcium that is drawn into the neuronal cells. This provides a very effective neuroprotective effect, preventing irreversible neuronal injury.
- Other protective mechanisms of hypothermia are inhibition of the pro-apoptotic activity and reduction of free radicals and inflammatory cytokines.

Consensus on hypothermia in aortic arch surgery

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Yan et al

- Although voluminous studies have demonstrated benefits of HCA over the past three decades, pioneers in cardiac surgery continue to push the boundaries and seek to improve this procedure
- In conjunction with the recent increase **in the implementation of selective antegrade cerebral perfusion during HCA, a paradigm shift has been the use of warmer hypothermic temperatures**, which is argued to reduce re-warming time, minimize the degree of coagulopathy and improve survival outcomes

Yan et al

- Consensus on hypothermia classifications in aortic arch surgery
- Category: Nasopharyngeal temperature
- Profound hypothermia ≤ 14 °C
- Deep hypothermia¹ 14.1-20 °C
- Moderate hypothermia 20.1-28 °C
- Mild hypothermia 28.1-34 °C

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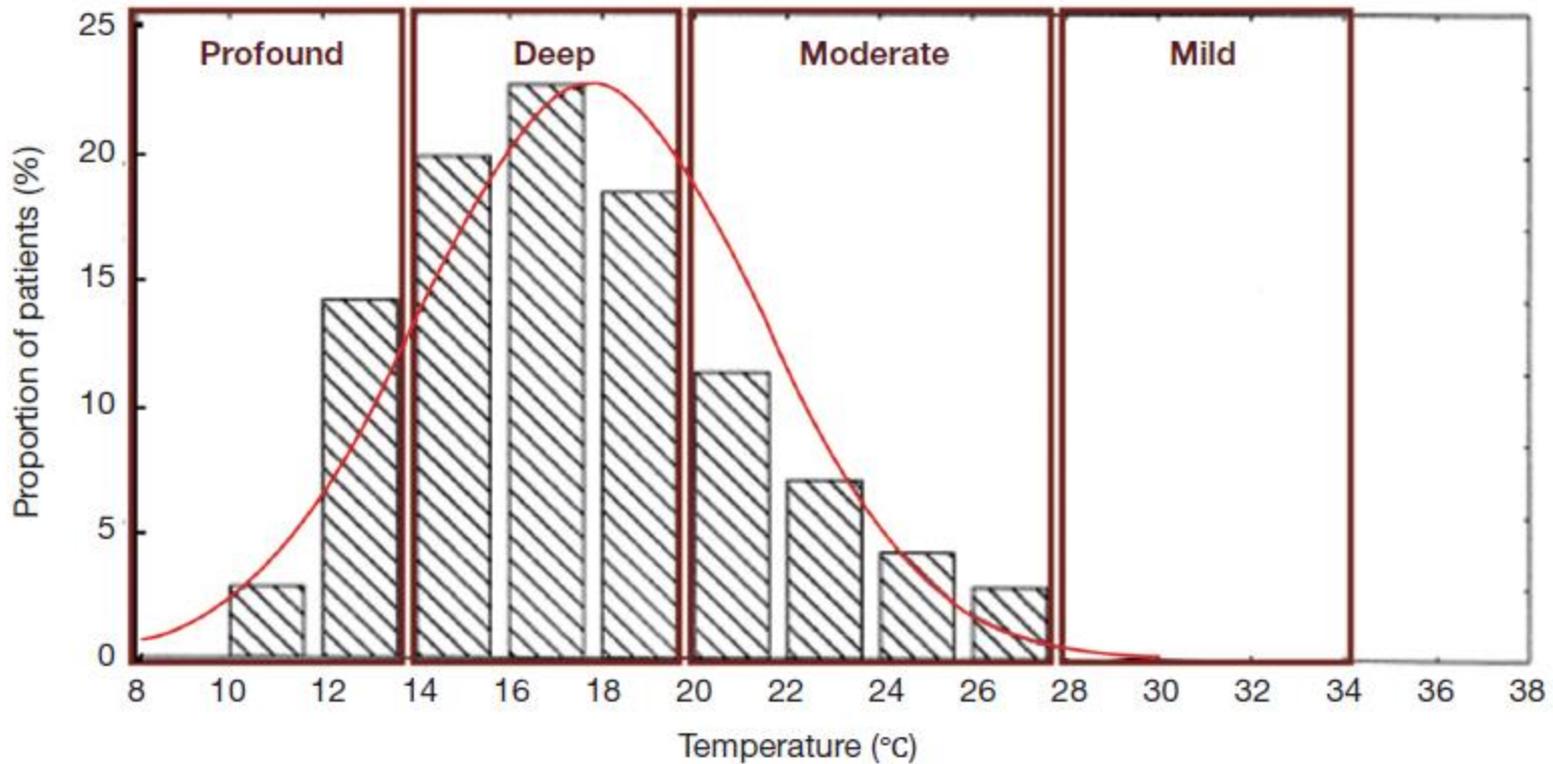


Figure 1 The proportion of patients for whom electrocerebral silence is achieved at a given nasopharyngeal temperature. Proposed categories are superimposed in dark red. (Modified from Stecker *et al.*)

Yan et al

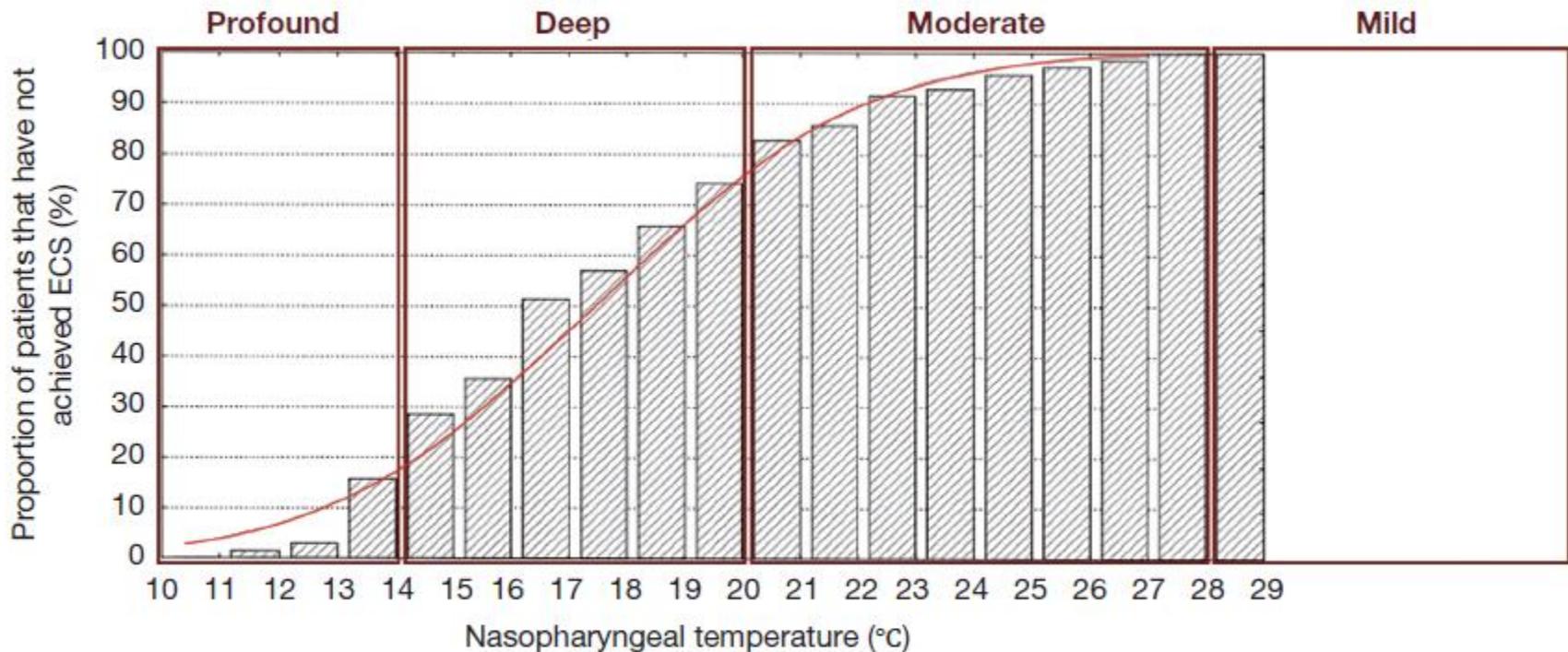


Figure 2 Cumulative probability representation that electrocerebral silence (ECS) is not achieved for temperatures *above* that indicated. For example, at 20 °C, 75% of patients have not achieved ECS. Proposed categories are superimposed in dark red. (Modified from Stecker *et al.*)

Yan et al

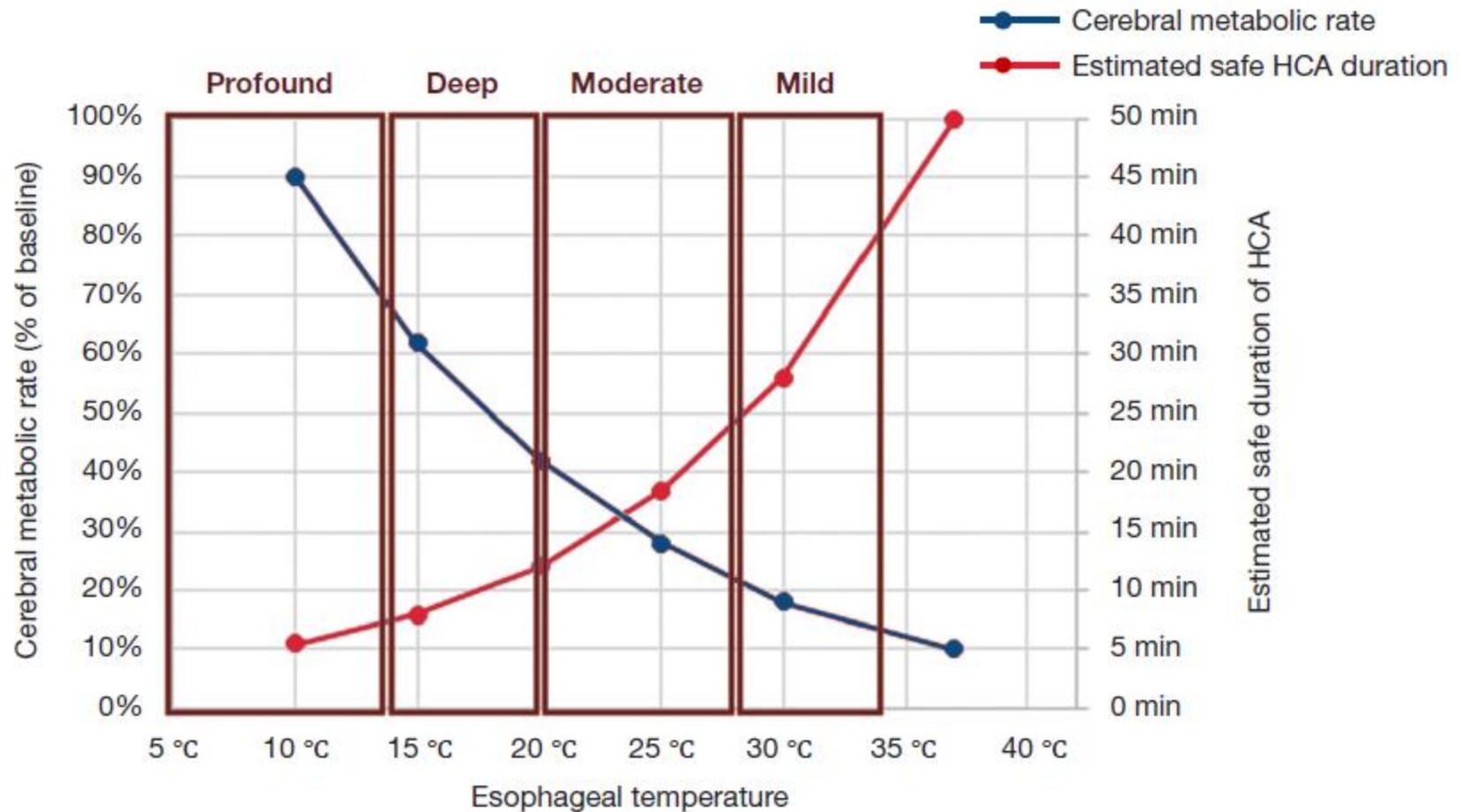


Figure 3 Cerebral metabolic rate, as percentage of baseline, at various esophageal temperatures, and estimated safe duration of HCA. Proposed categories are superimposed in dark red. (Modified from McCullough *et al.*)

Antegrade Cerebral Perfusion With Mild Hypothermia for Aortic Arch Replacement: Single-Center Experience in 245 Consecutive Patients

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Division of Thoracic and Cardiovascular Surgery, Johann Wolfgang Goethe University, Frankfurt/Main, Germany; and Division of Cardiovascular Surgery, Mesa Hospital, Ankara, Turkey

- Cooling was limited to 28°C to 30°C rectal temperature.
- The innominate and left carotid artery were snared with silicone elastomer loops and occluded at the time of initiation of the ACP
- The ACP was conducted with a perfusate temperature of 30°C in a pressure-controlled manner.

Mild hypothermia (32°C) and antegrade cerebral perfusion in aortic arch operations

Farhad Bakhtiary, MD, Selami Dogan, MD, Omer Dzemali, MD, Peter Kleine, MD, PhD, Anton Moritz, MD, PhD, and Tayfun Aybek, MD, PhD, Frankfurt/Main, Germany

- Brain oxygen consumption is reduced by 50% of baseline values if the patient is cooled systemically to 28°C core temperature.
- Regional cerebral blood flow with antegrade perfusion decreases more rapidly at less than 28°C than between 36°C and 30°C.
- Thus a perfusate temperature of greater than 28°C should be optimal for ACP to meet the lowered metabolic demands of the brain.
- We have started to use 32°C mild systemic hypothermia together with ACP for operations on the aortic arch.

Debatable Issues...

- Hemodilution
- Temperature
- **Steroids**
- Qb Flow
- pH stat vs. alpha stat
- PO₂???
- ACP, RCP, Circulatory Arrest
- Cannulation sites, Etc...

Steroids



FORUM

Pharmacological agents as cerebral protectants during deep hypothermic circulatory arrest in adult thoracic aortic surgery

A survey of current practice*

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A nighttime photograph of a cityscape featuring a large fountain with two tall, illuminated towers. In the foreground, a bridge spans across a body of water, with city lights reflecting on the surface. The background shows a dense urban skyline with various buildings and lights.

Only 16% of the respondents supported the use of steroids.

Systemic Steroid Pretreatment Improves Cerebral Protection After Circulatory Arrest

Dominique Shum-Tim, MD, Christo I. Tchervenkov, MD, Al-Maleek Jamal, BS, Toni Nimeh, MD, Chwan-Yau Luo, MD, Edgar Chedrawy, MD, Eric Laliberte, CCP, Anie Philip, PhD, Colin P. Rose, MD, and Josee Lavoie, MD

Division of Cardiovascular Surgery, The Montreal Children's Hospital, and Divisions of Plastic Surgery, Cardiology, and Anesthesia, Montreal General Hospital, McGill University Health Center, Montreal, Quebec, Canada

- This study evaluates whether systemic steroid pretreatment enhances neuroprotection during deep hypothermic circulatory arrest (DHCA) compared with steroid in cardiopulmonary bypass (CPB) prime.
- The benefit of steroids during extracorporeal circulation, the medication was administered directly into the patients before initiation of CPB
- Methylprednisolone pretreatment before surgery was associated with better pulmonary functions as compared with steroid in pump prime or no steroid groups

Debatable Issues...

- Hemodilution
- Temperature
- Steroids
- **Blood Flow**
- pH stat vs. alpha stat
- PO₂???
- ACP, RCP, Circulatory Arrest
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Qb Flow?

- A majority of published literature promotes CI of $2.4 \pm .2$
 - Realization of the importance of measured flow with roller pumps
- Higher blood flow reduces vasoactive pharmaceutical demands
- Higher blood flow reduces temperature gradients
- Oftentimes vasodilator proves necessary which benefits balanced cooling and warming



Debatable Issues...

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pH Stat/Alpha Stat



https://www.openanesthesia.org/hypothermia_ph_stat_management/

- Definition of pH-Stat
- During pH-stat acid-base management, the patient's pH is maintained at a constant level by managing pH *at the patient's temperature*. **pH-stat pH management is temperature-corrected.** Compared to alpha-stat, pH stat (which aims for a pCO₂ of 40 and pH of 7.40 at the patient's actual temperature) leads to higher pCO₂ (*respiratory acidosis*), and *increased cerebral blood flow*. CO₂ is deliberately added to maintain a pCO₂ of 40 mm Hg during hypothermia.

https://www.openanesthesia.org/hypothermia_ph_stat_management/

- Definition of alpha-Stat
- During alpha-stat acid-base management, the ionization state of histidine is maintained by managing a standardized pH (measured at 37C). Alpha-stat pH management is *not* temperature-corrected – as the patient’s temperature falls, the partial pressure of CO₂ decreases (and solubility increases), thus a hypothermic patient with a pH of 7.40 and a pCO₂ of 40 (measured at 37C) will, in reality, have a lower pCO₂ (because partial pressure of CO₂ is lower), and this will manifest as a relative *respiratory alkalosis* coupled with *decreased cerebral blood flow*. During alpha-stat management you have no idea what the patient’s pCO₂ is, your goal is to maintain a constant dissociation state of histidine.

Alpha Stat vs. pH Stat

- A study by Kiziltan et al., in which 52 patients were randomized to alpha-stat versus pH stat management, showed that **pH stat management led to increased jugular venous oxygen concentrations**, implying increased CBF. A study by Sakamoto et al., comparing pH stat to alpha stat during repair of cyanotic neonatal congenital heart disease, demonstrated that pH stat management led to less pulmonary collateral circulation as well as higher oxyhemoglobin and lower deoxyhemoglobin levels on cerebral near-infrared spectroscopy, suggesting greater cerebral oxygenation through improved oxygen delivery with pH stat. A prior study by Murkin et al. comparing pH stat to alpha stat showed that **during pH stat, CBF and CMRO₂ become uncoupled** (CBF is pressure-dependent), whereas **during alpha-stat CBF is related to metabolic needs (CMRO₂) and *not* to cerebral perfusion pressure.** **The major concern with pH stat is the potential for increasing the cerebral embolic load.**

Kiziltan et al

- *H Tarik Kiziltan, Mehmet Baltali, Ahmet Bilen, Gülşah Seydaoglu, Muzaffer Incesoz, Atilay Tasdelen, Sait Aslamaci* **Comparison of alpha-stat and pH-stat cardiopulmonary bypass in relation to jugular venous oxygen saturation and cerebral glucose-oxygen utilization.** *Anesth. Analg.:* 2003, 96(3);644-50, table of contents [[PubMed:12598237](#)]

Kiziltan et al

- Jugular venous oxygen saturation (SJVO(2)) reflects the balance between cerebral blood flow and metabolism. This study was designed to compare the effects of two different acid-base strategies on jugular venous desaturation (SJVO(2) <50%) and cerebral arteriovenous oxygen-glucose use. We performed a prospective, randomized study in 52 patients undergoing cardiopulmonary bypass (CPB) at 27 degrees C with either alpha-stat (n = 26) or pH-stat (n = 26) management. A retrograde internal jugular vein catheter was inserted, and blood samples were obtained at intervals during CPB. There were no differences in preoperative variables between the groups. SJVO(2) was significantly higher in the pH-stat group (at 30 min CPB: 86.2% +/- 6.1% versus 70.6% +/- 9.3%; P < 0.001). The differences in arteriovenous oxygen and glucose were smaller in the pH-stat group (at 30 min CPB: 1.9 +/- 0.82 mL/dL versus 3.98 +/- 1.12 mL/dL; P < 0.001; and 3.67 +/- 2.8 mL/dL versus 10.1 +/- 5.2 mL/dL; P < 0.001, respectively). All episodes of desaturation occurred during rewarming, and the difference in the incidence of desaturation between the two groups was not significant. All patients left the hospital in good condition. **Compared with alpha-stat, the pH-stat strategy promotes an increase in SJVO(2) and a decrease in arteriovenous oxygen and arteriovenous glucose differences. These findings indicate an increased cerebral supply with pH-stat; however, this strategy does not eliminate jugular venous desaturation during CPB.**

Sakamoto et al

- *Takahiko Sakamoto, Hiromi Kurosawa, Toshiharu Shin'oka, Mitsuru Aoki, Yukihisa Isomatsu* **The influence of pH strategy on cerebral and collateral circulation during hypothermic cardiopulmonary bypass in cyanotic patients with heart disease: results of a randomized trial and real-time monitoring.** *J. Thorac. Cardiovasc. Surg.*: 2004, 127(1);12-9 [[PubMed:14752407](https://pubmed.ncbi.nlm.nih.gov/14752407/)]

Sakamoto et al

The optimal pH strategy during hypothermic cardiopulmonary bypass remains controversial. Systemic pulmonary collateral circulation may develop in patients with cyanotic anomalies. The purpose of this study was to evaluate the effect of pH strategies on cerebral oxygenation and systemic pulmonary collateral circulation during hypothermic cardiopulmonary bypass in cyanotic patients with heart disease.

Sakamoto et al

Forty cyanotic patients (age > 1 year) with heart disease were prospectively randomized into 2 groups. **Group 1** (n = 19, 14.3 +/- 1.5 kg) underwent hypothermic cardiopulmonary bypass with **alpha-stat** strategy and **group 2** (n = 21, 12.5 +/- 0.9 kg) with **pH-stat**. Cardiopulmonary bypass was established with pump-assisted drainage. Cerebral oxygenation was assessed by near-infrared spectroscopy and the systemic pulmonary collateral circulation was calculated by pump flows [% systemic pulmonary collateral circulation = perfusion flow - drainage flow)/perfusion flow x 100]. **Lactate was measured as an index of systemic anaerobic metabolism.**

Sakamoto et al

There were no significant differences in preoperative hematocrit, oxygen saturation, Qp/Qs, cardiopulmonary bypass duration, minimum temperatures, perfusion flow and pressure, urine output, and depth of anesthesia between the groups. **Oxyhemoglobin signal and tissue oxygenation index of near-infrared spectroscopy monitoring were significantly lower in group 1 compared with group 2** (P =.008 and P <.0001, respectively), suggesting **inadequate cerebral oxygenation with alpha-stat**. Deoxygenated hemoglobin signal was significantly higher in group 1 relative to group 2 (P <.0001). The % systemic pulmonary collateral circulation was significantly lower in group 2 compared with group 1, suggesting a reduced pulmonary collateral circulation with pH-stat (P <.0001, average; group 1, 20.1% +/- 1.2%; group 2; 7.7% +/- 0.7%). **Serum lactate was significantly lower in group 2** (P <.0001).

Sakamoto et al

- **CONCLUSIONS:**

The pH-stat strategy results in an improved environment, including sufficient cerebral oxygenation, decreased systemic pulmonary collateral circulation, and lower lactate level during hypothermic cardiopulmonary bypass in cyanotic patients with heart disease. Future studies should investigate the long-term neurological outcome.

Murkin et al

- *J M Murkin, J K Farrar, W A Tweed, F N McKenzie, G Guiraudon* **Cerebral autoregulation and flow/metabolism coupling during cardiopulmonary bypass: the influence of PaCO₂.** *Anesth. Analg.:* 1987, 66(9);825-32 [[PubMed:3113288](#)]

Murkin et al

Measurement of ^{133}Xe clearance and effluent cerebral venous blood sampling were used in 38 patients to determine the effects of cardiopulmonary bypass, and of maintaining temperature corrected or noncorrected PaCO_2 at 40 mm Hg on regulation of cerebral blood flow (CBF) and flow/metabolism coupling. After induction of anesthesia with diazepam and fentanyl, mean CBF was $25 \text{ ml}\cdot 100 \text{ g}^{-1}\cdot \text{min}^{-1}$ and cerebral oxygen consumption, $1.67 \text{ ml}\cdot 100 \text{ g}^{-1}\cdot \text{min}^{-1}$. Cerebral oxygen consumption during nonpulsatile cardiopulmonary bypass at 26°C was reduced to $0.42 \text{ ml}\cdot 100 \text{ g}^{-1}\cdot \text{min}^{-1}$ in both groups. CBF was reduced to $14\text{--}15 \text{ ml}\cdot 100 \text{ g}^{-1}\cdot \text{min}^{-1}$ in the non-temperature-corrected group ($n = 21$), was independent of cerebral perfusion pressure over the range of $20\text{--}100 \text{ mm Hg}$, but correlated with cerebral oxygen consumption. In the temperature-corrected group ($n = 17$), CBF varied from 22 to $32 \text{ ml}\cdot 100 \text{ g}^{-1}\cdot \text{min}^{-1}$, and flow | metabolism coupling was not maintained (i.e., CBF and cerebral oxygen consumption varied independently). However, variation in CBF correlated significantly with cerebral perfusion pressure over the pressure range of $15\text{--}95 \text{ mm Hg}$. This study demonstrates a profound reduction in cerebral oxygen consumption during hypothermic non-pulsatile cardiopulmonary bypass. **When a non-temperature-corrected PaCO_2 of approximately 40 mm Hg was maintained, CBF was lower, and analysis of pooled data suggested that CBF regulation was better preserved, i.e., CBF was independent of pressure changes and dependent upon cerebral oxygen consumption.**

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Hyperoxia with Circulatory Arrest?

[ASAIO J.](#) 2012 Jul-Aug;58(4):330-6. doi: 10.1097/MAT.0b013e318251dfab.

Hyperoxia management during deep hypothermia for cerebral protection in circulatory arrest rabbit model.

[Wang Q](#)¹, [Yang J](#), [Long C](#), [Zhao J](#), [Li Y](#), [Xue Q](#), [Cheng L](#), [Cheng W](#).

- During cooling to deep hypothermia, increasing hemoglobin (Hb) oxygen affinity causes a progressive impairment of oxygen transfer from Hb to plasma, with subsequent decreases in transfer to cerebral interstitium and cells.
- This impairment of oxygen transport is minor at 27°C but can be substantial at 17°C.
- Hyperoxia management during deep hypothermia provided substantial dissolved oxygen and demonstrated better cerebral protection over normoxia management.

Hyperoxia with Circulatory Arrest?

Hyperoxia for Management of Acid-Base Status During Deep Hypothermia With Circulatory Arrest

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Children's Hospital Medical Center, Cincinnati, Ohio, and Children's Mercy Hospital, Kansas City, Missouri

- The pH values were lower after DHCA in patients managed with the normoxia strategy regardless of pH strategy
- Only patients in group IV (hyperoxia and pHstat) had normal pH levels after DHCA
- The base deficit was least depressed immediately after DHCA for group IV compared with all other groups

Hyperoxia with Circulatory Arrest?

OXYGENATION STRATEGY AND NEUROLOGIC DAMAGE AFTER DEEP HYPOTHERMIC CIRCULATORY ARREST. II. HYPOXIC VERSUS FREE RADICAL INJURY

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Objectives: Laboratory studies suggest that myocardial reperfusion injury is exacerbated by free radicals when pure oxygen is used during cardiopulmonary bypass. In phase I of this study we demonstrated that normoxic perfusion during cardiopulmonary bypass does not increase the risk of microembolic brain injury so long as a membrane oxygenator with an arterial filter is used. In phase II of this study we studied the hypothesis that normoxic perfusion increases the risk of hypoxic brain

In the setting of prolonged deep hypothermia and circulatory arrest with membrane oxygenators, normoxic cardiopulmonary bypass significantly increases histologically graded brain damage with respect to hyperoxic cardiopulmonary bypass. Near-infrared spectroscopy suggests that the mechanism is hypoxic injury, which presumably overwhelms any injury caused by increased oxygen free radicals.

Debatable Issues...

- Hemodilution
- Temperature
- Steroids
- Qb Flow
- pH stat vs. alpha stat
- PO₂???
- **ACP, RCP, Circulatory Arrest**
- Cannulation sites, Etc...

Deep hypothermic circulatory arrest

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Deep hypothermic circulatory arrest

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At our Aortic Institute at Yale, straight DHCA has been the method of choice for the absolute majority of complex procedures involving the aortic arch. We believe that straight DHCA is an especially appealing method of cerebral protection because of its simplicity and effectiveness. In this article, we will provide evidence that straight DHCA is an effective method of brain protection during aortic surgery without any adjunctive cerebral perfusion.

Typically DHCA is required for partial arch replacement (hemiarch) requiring an open distal anastomosis, and for total arch replacement. As soon as CPB is initiated, the patient is cooled down to 19 °C (for hemiarch) or to 18 °C (for total arch).

Temperature monitoring is conducted solely via a probe in the urinary bladder. The head is packed in ice to achieve topical cooling. Steroids are routinely administered for all patients before CPB is initiated and alpha-stat management is used for acid-base balance.

After termination of DHCA, the rewarming usually takes about 60 minutes. We prefer gentle rewarming (gradient between blood and bath temperature less than 10 °C) in order to prevent potential protein denaturation.

For ascending and arch replacement, the mortality rate for elective cases was only 2%, and for all cases (including emergent cases) only 2.2%

However, in our experience, the overall stroke rate for patients undergoing ascending and arch operations with DHCA was 3.1%

Among the few patients with DHCA time longer than 45 minutes, the stroke rate was 13.1%. While it may be tempting to attribute this higher stroke rate in long arrest cases to inadequacy of cerebral protection, a closer look argues otherwise.

Two-thirds of strokes in DCHA were embolic on CT scan (not ischemic), and thus not directly ascribable to the method of protection.

Debatable Issues...

- Hemodilution
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- ACP, RCP, Circulatory Arrest
- Cannulation sites...

Cannulation Controversies

Cannulation of the axillary artery provides several benefits, including:

It must be acknowledged that there is a lack of conclusive evidence that clearly endorses axillary over femoral cannulation and some of the superior results from axillary cannulation may be related to maintenance of continuous antegrade cerebral perfusion. Clinical decision on axillary versus femoral cannulation is ultimately dictated by patient factors and surgeon preference.

- Potentially redirecting flow into the true lumen in dissections, and decompressing the expanded false lumen;
- Artery is usually free from atherosclerotic plaques.

My Circulatory Practice at Mayo Clinic

- High Blood Flow (~3.0 CI)
- No Vasoconstrictive Drugs
- pH stat cooling until blood temp of 20° C then convert to alpha stat management
- Hyperoxia to drive nitrogen out
- No steroids in the pump. If treated, prior to bypass initiation
- No intentional hemodilution
- Mannitol as a diuretic
- EEG on all elective cases
- 1 stroke in last 254 cases (.3%)

Contemporary Circulatory Arrest Conundrums

Phil Scott, CCP, FPP
Missouri Perfusion Society
June 8th, 2019

