SNS COLLEGE OF ALLIED HEALTH SCIENCES

SNS Kalvi Nagar, Coimbatore - 35

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DEPARTMENT OF CARDIO PULMONARY PERFUSION CARE TECHNOLOGY

COURSE NAME : PRINCIPLES OF PERFUSION TECHNOLOGY II

III rd YEAR

TOPIC : Myocardial protection

AORTIC CROSS CLAMPING

Introduction

- Cardiac surgery is performed to restore cardiac function in a diseased heart
- By necessity it is accompanied by myocardial injury
- Myocardial protection is required to minimize the harmful effects on heart
- There is no universally accepted strategy for myocardial protection
- Minimize the difference between o2 delivery and utilization

Cardiac physiology

- Myocardium has a high rate of o2 consumption under normal circumstances
- Ischemia occurs when the supply of o2 is exceeded by the demand
- Infarction occurs when ischemia occurs for prolonged period of time

- O2 delivery to myocardium depends on
- 1 - hemoglobin
- 2- o2 saturation
- 3- blood flow

- Sub endocardium

  most vulnerable to injury

  flow occurs primarily during diastole

  flow depends upon transmural gradient

- Insufficient flow

  systemic hypotension

  aortic stenosis, CAD, VF, ventricular distension

- Heart depends on continuous supply of o2 to maintain full function
- In presence of o2 1 mole glucose – 36 moles ATP
- Under anaerobic conditions 1 mole glucose – 2 moles ATP, lactate and hydrogen accumulate in tissues

Physiology – myocardial o2 consumption

CONDITION	O2 CONSUMPTION
Normal working ventricular myocardium	8 ml of o2/100g per min
Empty beating heart	5.6 ml of o2/100g per min
Potassium arrested heart	1.1 ml of o2/100g per min
Myocardial cooling	0.3 ml of o2/100g per min

Objectives of the surgeon using cross clamp

- A still , bloodless field
- The heart is soft and can be retracted more easily
- Microvascular anastamosis can be more accurately constructed
- Cerebral air embolism can be prevented
- Certain cardiac anomalies can only be corrected with prolonged X-clamping of the aorta

Mechanisms of myocardial ischemic injury

- Depletion of high energy phosphates
- Intracellular acidosis
- Alterations in the intracellular calcium homeostasis
- Direct myocardial injury from ischemia

Ischemia – reperfusion injury

- Calcium overload
- Generation of oxygen derived free radicals
- Complement activation
- Adverse endothelial cell- leucocyte interactions
- Myocellular edema
- Damage to non-myocyte components

Consequence of ischemia –reperfusion injury

- Severity depends on length of ischemia, temperature of myocardium, conditions of myocardium before during and after the ischemia

1. No functional deficit
2. Myocardial stunning
3. Myocardial necrosis (stone heart)

Purpose of cardioplegia

- To provide cardiac quiescence
- Bloodless field, enhances visibility,
- Reduction in myocardial energy consumption, preservation of myocardial function
- Current methods allow rapid resumption of contractile activity at the end of the procedure

Protective strategies before the onset of ischemia

- Minimization of ongoing ischemia
- Rapid revascularization
- Nutritional repletion
- Prevention of ventricular distension
- Myocardial preconditioning

Protective strategies during ischemia

- Numerous interventions are possible
- Not all are required in every circumstances
- Must be individualized to situations

Elements of myocardial protection – well established strategies

1. Asystole
2. Hypothermia
3. Avoidance of edema
4. Buffering of acidosis
5. Calcium management

Asystole

- Prevents depletion of ATPs
- Good post ischemic functional recovery of myocardium

Hypothermia

- Myocardial o2 consumption decreases by 50% for every 10 ‘ C decrease in myocardial temperature (Q10 effect)
- Advantage : allows the interruption of myocardial blood flow for short period of time enabling the conduct of operation
- Disadvantages: alterations in cellular fluidity, transmembrane gradients, myocardial edema

TABLE 13.2. POSITIVE AND NEGATIVE PHYSIOLOGIC CONSEQUENCES OF HYPOTHERMIA

Positive	Negative
Decreases metabolic rate	Decreases rate of repair
Decreases oxygen requirements	Increases intracellular swelling, Na^+ accumulation
Temporarily decreases cell–cell interactions	Decreases *rate* of contraction; increases energy demands *per beat*
Decreases rate of degradative reactions	Induces ventricular fibrillation, arrhythmias
Increases tolerance to ischemia	Impairs oxygen dissociation
Reduces $[K^+]$ necessary for arrest	Impairs coronary autoregulation
Prolongs electrical silence, cardioplegia	Potential for phrenic nerve damage
Decreases cell deformability	Promotes rouleau formation(?)
Inhibits intracellular Ca^{2+} accumulation	Decreases membrane fluidity; decreases receptor transduction , transmembrane transport
Decreases rate of NF-κB (nuclear factor) translocation	Inhibits sarcoplasmic reticulum Ca^{2+} uptake

- Methods of producing myocardial hypothermia

1. Administration of cardioplegia, given at a temp of 4-10”C will produce myocardial cooling to 15-16”C
2. Systemic hypothermia
3. Topical cooling

Avoidance of edema

- Controlled delivery pressure
- Composition – mannitol and glucose

Buffering of acidosis

- Episodic reinfusion of solution- washout
- Composition-- blood, histidine, bicarbonate, THAM

Calcium management

- Extreme hypercalcemia and hypocalcemia- suboptimal myocardial protection
- Calcium chelation
- Drugs

Composition of cardioplegia

1. Crystalloid cardioplegia
2. Blood cardioplegia

Crystalloid CP

- Uncommonly used in adult patients
- Preservation of donor heart during transplantation
- Do not contain hemoglobin and deliver only dissolved o2

Blood CP

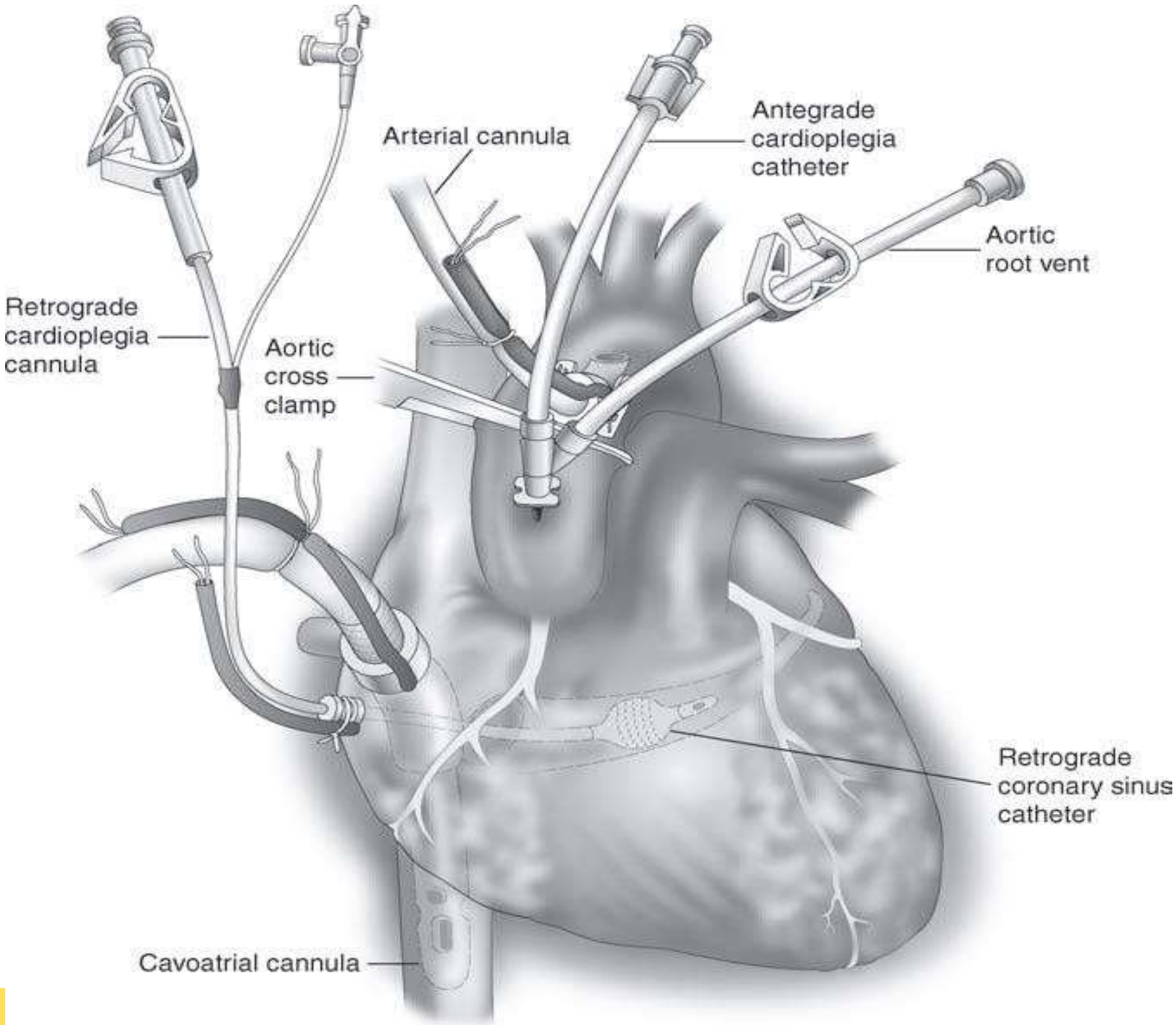
- Mixing blood to crystalloid with a final hematocrit of 16-20%
- High o2 carrying capacity
- Contains buffers, free radical scavengers, colloids and other benificial substarces
- Microplegia – blood minimally diluted with elements necessary for achieving cardiac arrest

Route of delivery

1. Antegrade
2. Retrograde
3. Antegrade and retrograde

Antegrade cardioplegia

- Cannula placed in the aortic root
- Flow- 150ml/ min.m2
- Perfusion pressure 70-100mmHg
- Low perfusion pr – uneven distribution
- High perfusion pr – endothelial damage
- Root perfusion cannot be given in aortic valve regurgitation


Arterial cannula
Antegrade cardioplegia catheter
Aortic root vent
Retrograde cardioplegia cannula
Aortic cross clamp
Retrograde coronary sinus catheter
Cavoatrial cannula

Retrograde delivery

- Through coronary veins by way of coronary sinus through a special cannula
- Pressure less than 40mm hg
- High pressure – damage to coronary sinus
- Improper placement – injury to CS
- May not adequately protect RV
- Advantages: aortic valve procedures, coronary

History

- Until early 1950”s myocardial global ischemia by cross clamping the aorta and reperfusion was followed without any protective measures
- This some times cause irreversible severe myocardial damage (stone heart)

Melrose in 1955

- Rapid cardiac arrest using high dose potassium over 200mM
- Disadvantage – intracellular calcium overload and reperfusion injury

Hearse

- Concept of cold chemical cardioplegia
- Three components

1. Chemical arrest
2. Hypothermia
3. Additional protection- energy substrates, steriod, buffers etc

1970”s

- Intracellular and extracellular CP solutions
- Bretschneider solution- intracellular
- St thomas hospital no2 solution - extracellular

Blood cardioplegia

- Proposed by Buckberg
- Greater o2 supply capacity
- Higher osmotic pressure
- Greater acid base balance capacity

Elements of myocardial protection – emerging strategies

- Oxygen radical therapy
- Amino acid enhancement
- Adenosine

Elements of myocardial protection – experimental strategies

- Nitric oxide
- Specific anti neutrophil therapy
- Complement related therapy
- Hyper polarising agents
- Sodium – hydrogen exchange inhibitors

Myocardial preconditioning

- Concept: myocardium that has undergone a brief limited period of ischemia may be better able to tolerate a subsequent, longer period of ischemia
- Hearts exposed to brief ischemic stimulus sustain a smaller area of necrosis following a second longer period of ischemia
- Eg: ischemia, hyperthermia, drugs

del NIDO CARDIOPLEGIA

- 1 liter plasma-lyte solution to which the following are added

1. Mannitol 20%, 16.3 ml
2. Magnesium sulfate 50%, 4ml
3. Sodium bicarbonate 8.4%, 13ml
4. Potassium chloride (2meq/ml), 13ml
5. Lignocaine 1%, 13ml

Custodial HTK cardioplegia (Bredschnider's solution)

- 1 L contains

1. Sodium chloride- 15 mmol/L
2. Potassium chloride - 9 mmol/L
3. Magnesium chloride – 4mmol/L
4. Histidine hydrochloride- 18mmol/L
5. Histidine – 180mmol/L
6. Tryptophan – 2mmol/L
7. Mannitol- 30mmol/L
8. Calcium chloride – 0.015mmol/L

9.Potassium hydrogen 2-ketoglutarate- 1mmol/L

Osmolarity- 310mOsm/kg, pH- 7.02-7.20

Key points

- Intraoperative myocardial injury can occur before, during or after CPB
- The degree of permanent myocardial injury after ischemia is a function of severity and duration of ischemia which can be modified
- Reperfusion injury is defined as additional myocardial injury incurred after restoration of blood flow to ischemic myocardium
- Impaired microvascular bloodflow (no-reflow response) can result from these injuries

- Reperfusion injuries can cause atrial and ventricular dysarrhythmias, reversible systolic and diastolic LV dysfunction(stunning), myocardial necrosis.

- A variety of strategies for myocardial protection have been identified

1. Established strategies
2. Emerging strategies
3. Experimental strategies