Difficult Scenarios for Myocardial Protection

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Difficult Scenarios for Myocardial Protection

Stone Heart

- Nightmare
- Nightmare of the past?
- Myocardial Protection
- Difficult Scenarios
Ischemic Contracture of the Heart: “Stone Heart”

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Open heart surgery using cardiopulmonary bypass has enjoyed an exciting 15 year history during which many technical modifications were made and baffling complications encountered. For the past 8 years we have routinely employed a simplified technique using simple intravenous solutions to “prime” the extracorporeal circuit, maintained the patient and his heart at northermic levels and induced ischemic cardiac arrest to control or prevent myocardial injury during the period of bypass.1,2 Periods of induced ischemia have been tolerated by some patients for more than 2 hours without apparent myocardial damage. However, we have seen a relatively small number of patients who died in the operating room from an unusual type of myocardial failure. Rather than the poorly contracting, overstretched ventricle of myocardial failure, a small spastic heart, literally frozen in systole, develops. The ventricular chamber is decreased notably in volume because of the contracture, and even vigorous manual massage does not produce an adequate stroke volume. Changes in peripheral resistance, cardiotonic agents, electrolyte solute diastolic pressure could be obtained in stimulating agents and assist devices have not altered its inevitable course. On palpation the heart is in a contracted state similar to the uterine contraction ring or the tetanic contraction of striated muscle as seen in the laboratory. This characteristic state is recognized readily at the time of surgery. Ultimately, cardiopulmonary bypass cannot be discontinued without the patient dying because of the maintained contractile state. We have named this irreversible contracted ventricle the “stone heart.”

Fortunately, the stone heart is rare. Of the 4,782 patients (1,407 for congenital lesions and 3,375 for acquired) who have undergone open heart surgery at the Texas Heart Institute during the 5 years from July 1966 to July 1971, 51 patients (1 percent) died during operation from acute myocardial insufficiency. Of that group, less than 1 third (13 patients) experienced severe contracture of the heart and the criteria we recognize as stone heart. We have reviewed the clinical and pathologic characteristics of these 13 patients because of the frustration and failure in treating this entity. To our knowledge this syndrome has not been reported previously as such.

Clinical features: All patients with manifestations of the stone heart had acquired heart disease. The age range and sex differences were not significant. Preoperative symptoms were interesting since 11 of the 13 patients had congestive heart failure, 8 had angina pectoris and only 3 had previously documented myocardial infarction. All patients had advanced stages of acquired heart disease as evidenced by the functional classification, cardiac catheterization data and preoperative electrocardiogram. Of the 13 patients, 10 were in functional class IV and the remainder were in class III according to the New York Heart Association classification; Over half of the patients had electrocardiographic findings of left ventricular hypertrophy, and most had some type of conduction defect before operation. Cardiac catheterization revealed similar findings indicative of left ventricular failure. No measurement of left ventricular end-diastolic pressure could be obtained in 5 patients with severe aortic stenosis because of the inability to cross the densely calcified aortic valve with the catheter at the time of left heart catheterization. The left ventricular end-diastolic pressure was greater than 20 mm Hg in the remaining 8 patients, and in 3 of these patients it was greater than 40 mm Hg at rest. In patients with aortic stenosis, notable left ventricular hypertension was found with high gradients across the aortic valve in patients in whom these measurements could be made. Almost all of the patients had severe pulmonary hypertension.

Of the 13 patients, 11 had significant aortic valve disease and all had aortic valve replacement. Among this group, 5 patients had aortic valve replacement alone; 4 had aortic valve replacement plus mitral valve replacement; 1 had aortic valve replacement plus coronary artery bypass; and another had aortic valve replacement plus replacement of the ascending aorta for severe aortitis which extended proximally to exclude the right coronary artery. Two patients had undergone coronary artery bypass procedures for severe coronary artery disease.

Pathologic features: Autopsy of these patients demonstrated some common features. Universally,
Stone Heart: Definition

- Global ischemic contracture of the heart resulting in a firm myocardium and loss of intra-cavity volume.
- Stone heart is an irreversible ischemia-induced cardiac rigor mortis, in which the heart undergoes global spastic contraction in systole.
Stone Heart - Mechanism

- Calcium ions leak from extracellular fluid and the sarcoplasmic reticulum,
- The muscle contracts when the myosin shifts, but the lack of ATP prevents it from detaching, and the muscle remains contracted.
Difficult Scenarios for Myocardial Protection

1. Myosin cross bridge attaches to the actin myofilament

2. Working stroke—the myosin head pivots and bends as it pulls on the actin filament, sliding it toward the M line

3. As new ATP attaches to the myosin head, the cross bridge detaches

4. As ATP is split into ADP and P\textsubscript{i}, cocking of the myosin head occurs

Myosin head (high-energy configuration)

ADP

P\textsubscript{i}

Thin filament

ADP and P\textsubscript{i} (inorganic phosphate) released

Myosin head (low-energy configuration)

ATP

ATP hydrolysis

Thick filament

As ATP is split into ADP and P\textsubscript{i}, cocking of the myosin head occurs
Stone Heart: Definition

- Stone heart syndrome was described as a complication of cardiopulmonary bypass in the early years of cardiac surgery.
- The use of modern cardioplegia approaches prevent this complication in cardiac surgery in most cases.
When uncontrolled reperfusion is initiated after global myocardial ischemia in cardiac surgery, the response may be only myocardial stunning. A more severe response consists of reperfusion arrhythmias, particularly ventricular tachycardia and ventricular fibrillation. The more prolonged and the larger the area of myocardial ischemia, the more frequent, severe, and intractable the arrhythmias.\(^{19}\) A still more severe response is the hard and fibrillating heart, sometimes termed *stone heart.*\(^{18,17,2,18}\) The *stone heart phenomenon* may involve only some regions of the heart, typically the basilar portion of the left ventricle and the subendocardium. This phenomenon indicates that the heart has undergone severe damage and may be considered to have approached the critical “point of no return.” It has not necessarily reached this point, because the stone heart is, at least under some circumstances, capable of recovery. The histologic features of these advanced forms of reperfusion damage include disruption of the regular myofibrillar pattern and evident contraction bands.\(^{5}\)
Difficult Scenarios for Myocardial Protection

1. Acute ischemic dysfunction
2. Stunning
3. Hibernation
4. Necrosis (stone heart)
5. Apoptosis
1. Acute Ischemic Dysfunction

- Reversible contractile failure
- O₂ supply
- Immediate recovery
2. Myocardial Injury - **Stunning**

- Partially reversible
- May be accompanied by endothelial dysfunction (NO) causing reduced coronary flow
- Result of ischemia-reperfusion insult
- Mediated by increased intracellular Ca accumulation
- Recovery: Hs, weeks
3. Myocardial Injury - Hibernation

- Partially reversible
- Related to poor myocardial blood flow
- Chronic
- Recovery: weeks, months
4. Myocardial Injury – Necrosis

- Irreversible
- Hyper contracture - “contracture band necrosis” (stone heart)
- Osmotic/ionic dysregulation, membrane injury
- Cell swelling and disruption
- Lysis
5. Myocardial Injury - Apoptosis

- Irreversible
- Death signal
- Cell shrinkage
- Phagocytosis
Myocardial Stunning

Reperfusion Arrhythmias (VT / VF)

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Myocardial Protection

Difficult Scenarios

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Is it still important in 2017?

- Based on working:
  - With many good surgeons
  - In many good centers
  - In many countries
- My opinion:
  - More safety margins
  - Be ready to difficult scenarios (1%-5%)
Difficult Scenarios for Myocardial Protection

The main tools

- Short ischemic time
- Cooling
- Cardioplegia type
- Antegrade - Retrograde
- IABP
- Decisions!
The Effects of Hypothermia on Myocardial Oxygen Consumption and Transmural Coronary Blood Flow in the Potassium-arrested Heart

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Hypothermia remains the primary adjunct employed to lower cellular metabolism during various cardiac procedures. In these experiments, left ventricular myocardial oxygen consumption (MVO₂) and transmural blood flow (TBF) were measured during cardiopulmonary bypass with the range of temperatures used clinically. Determinations were made in empty beating normothermic hearts and after potassium cardioplegia at 37, 32, 28, 22, 18, and 15° (K⁺ = 15–37 meq/L; Hct 25 volumes %). Oxygen content of the total coronary sinus collection was compared with a large volume arterial sample using a Lex-O₂-Con-TL analyzer (vs Van Slyke, R = 0.98). Transmural blood flow was measured at each temperature using microspheres (8μ), and perfusion was maintained at 80 mmHg. Asystole (37°) alone decreased MVO₂ from 5.18 ± 0.55 to 1.85 ± 0.20 ml O₂/min/100 g of left ventricle or approximately 65% (p < 0.001). With progressive cooling to 15° an additional 82% decrement in oxygen uptake occurred during asystole (p < 0.001). During asystole at 37° the decrease in MVO₂ was reflected mainly by a large decrement (p < 0.01) in TBF (1.27 ± 0.19 to 0.74 ± 0.17 ml/min/g of mean left ventricular flow). However, with cooling below 32°, the arteriovenous oxygen difference narrowed progressively (p < 0.001) while TBF paradoxically returned to control levels. Endocardial/epicardial flow ratios were not altered by cooling. These data not only confirm earlier reports describing a sequential drop in MVO₂ with incremental myocardial cooling, but also establish MVO₂ levels for perfused hearts arrested by potassium at lower temperatures (18–15°). Moreover, as transmural blood flow becomes independent of metabolic necessity during hypothermia, coronary autoregulation appears to be impaired, possibly affecting detrimental tissue over perfusion.
Myocardial Protection - Difficult Scenarios

- Myocardial oxygen consumption

Fig. 3. Myocardial oxygen consumption (MVO$_2$) was determined for 11 dog hearts during empty beating normothermic conditions and following potassium arrest at several temperature levels (37°C–15°C).
Difficult scenarios

- Poor LVF
- Significant coronary disease
- Long cross-clamp time
- Hypertrophic heart (AVR)
- Combined procedure (CABG+AVR)
- Aortic insufficiency
- Poor RV
- Unpredictable scenario!
Difficult Scenarios - Poor LVF

- Reversible damage?
- No reserve to survive the stunning
- Contraindication in the past
- Awareness needed today
  - Cooling
  - IABP (preventive)
  - Focus
Significant Coronary Disease

- Ante-grade cardioplegia only?
- Predictable?
  - Retro-grade cardioplegia
  - Cooling
  - Short ischemic time
Hypertrophic Heart (AVR)

• More muscle to protect
• Less efficiency cardioplegia
• Predictable?
  • Cool down the heart (32⁰ c)

Combined procedure (CABG + AVR)

• Combined problem
Aortic insufficiency
- Ante-grade cardioplegia
- Left ventricle distension
  - Retrograde

Poor RV
- Poor prognosis in general
- Retrograde is unpredictable to the RV
- Graft to RV coronary?
Unpredictable scenario!

- Unpredictable scenario – the worst!
- 95% or even 99% success
- Is it enough?
- More safety margins!?!?
- For 1%?
- The harm to the rest 99%
- Philosophy
- Academic - teaching
Cardioplegia in Rambam Health Care Center

- Antegrade and retrograde
- Micro (not diluted)- Blood cardioplegia:
  - Loading dose: 12 mg Adenosine (300 ml/1 min.)
    3 gr Magnesium
    20 mEq potassium
    100 mg lidocaine
  - Maintenance: 2 gr Magnesium
    60 mEq potassium

Blood flow: 140 ml/min
Plegia flow: 15 ml/h

Adjustment between the blood and the solution flow
Monitoring during Surgery

• Blood gas (A, V) is taken every 15 minutes: pH, Pco2, Po2, K, Na, Ca, Lactate, Glu, HCO₃⁻, BE.

• If potassium concentrations are too high, K⁺ flow can be decreased to 10 ml/h.
High risk patients?

- Cold blood cardioplegia
- Cool down to 32
- Cool down to 28?
- Cardioplegia through the veins graft
- Retrograde over 60 minutes?
Summary

- Stone heart
- However, patients are still dying
- Difficult scenarios
- Unpredictable scenarios
- Philosophy!
- Suitable to your practice, your patient
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Difficult Scenarios for Myocardial Protection
How to Avoid Stone Heart Every Time

SAHA

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Cardioplegia in Rambam Health Care Center

- Cardioplegia is induced by stopping electrical activity, which reduces myocardial oxygen consumption by 90%, by creating a condition when no action potentials can be induced and activate myocytes.

- This is most commonly performed by creating extracellular hyperkalemia, causing persistent blockade of fast natrium channels and an inability to create action potentials.

- The same effect can be achieved with extracellular hyponatremia or a high concentration of lidocain.

- All other components are minor, and while the scientific literature has shown some beneficial in-vitro effects, there are no human studies indicating survival advantage.
Cardioplegia in Rambam Health Care Center

- Adenosin induces very rapid A-V block, so the ventricles can be arrested very quickly. Adenosin also helps preserve endothelial function.
- Magnesium acts like a calcium antidote thus helping relax the myocites.
- Lidocain helps block the natrium channels.
- May other substances and drugs has been tried, such as steroids and beta-blockers, but none has really proved significant.
Stone Heart - Mechanism

• Anoxia cause rapid depletes glycogen and ATP.

• The muscles rely on anaerobic metabolism.

• This lack of ATP prevents the myosin heads detaching from the actin.
Cooling

Normo-thermic Ischemia

• 20 minutes - completely reversible
• 40 minutes – half the cells are necrotic
• 1 hour - lethal for all cells