PROS AND CONS OF PERICARDIAL CLOSURE AFTER HEART SURGERY

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Pericardial Repair

Clinical Importance
Pericardial Functions: Well documented, seldom appreciated

The majority of cardiac surgeons leave the Pericardium open after cardiac surgery
Pericardial Repair

How important is Pericardial Anatomy and Physiology?

Should Pericardial Repair be Standard after Cardiac Operations?
Pericardial Repair

Question:
Is this really the most physiological and “best” operative strategy for patients???
Management of The Pericardial Sac After Open Heart Procedures

Overwhelmingly, no “management” at all!

Most common technique:
  - Open sac
  - Forget about it!

Most common closure practice:
  - Re-approximate sac with several interrupted sutures

Limitations of direct reconstruction:
  - Large gaps
  - Very incomplete edge to edge reapproximation
  - Sometimes not possible due to technical constraints
Pericardial Repair

Attitudes about closing pericardium vary widely among heart surgeons

Should it be done?
How to do it / not to do it
Does it even matter?
Does it have any impact on clinical outcomes?
The benefits, drawbacks and technical aspects of this procedure have been complicated by:

- Conflicts between traditional believe and modern practice
- Lack of standard technique
- Lack of viable pericardial substitutes
- A virtual absence of convincing, high-quality prospective studies
Assumption 1: The pericardium in its normal anatomical state does not play a significant or meaningful role in healthy or pathological states.
Assumption 2:
The postoperative restoration of the pericardium to its normal anatomical state has a NO impact on early or late surgical outcomes.
My Notion about what’s important

Restore/maintain “normal” barrier of intact pericardial sac

Separate myocardium from sternum

- Safer re-entry at re-operation
- Discourage direct spread of infection from post-op sternal wound infection
- Protect myocardium and bypass grafts from acute injury in event of sternal dehiscence
- Prevent “contamination” of heart and pericardial sac from rundown of debris (blood, fat, necrotic tissue = “soup”) from the sternal wound

Compartmentalize and organize the surgical site

- Maintains proper lie of bypass grafts
- ? Decrease bleeding post-op
- ? Improve recognition of source of bleeding post-op

Reduced incidence of post-op complications? (AF, etc)
Pericardial Repair - objectives

1. Understanding Pericardial Physiology
2. Why closing pericardium after surgery is a good idea
3. Arguments against pericardial closure
4. Why pericardial closure may impact postoperative atrial fibrillation
5. Other possible benefits of pericardial closure
In order to understand the implications and make informed decisions about repair, pericardial physiology must first be understood.

What does the pericardium do?
Pericardial Functions: Well documented, seldom appreciated
Pericardium

Blood Supply

1. Primarily from internal thoracic arteries. Via the Musculo-phrenic branches

2. Directly from the aorta.
Pericardium

Venous Drainage

1. Via tributaries of the brachiocephalic veins
Pericardium

Nerve Supply

1. Vagal fibers from esophageal plexus
2. Sympathetic trunk (vasomotor)
3. Phrenic nerves primary source of sensory fibers (C3 – C5)
Pericardial Physiology

Three Main Functions

1. Mechanical
2. Membranous
3. Ligamentous
Normal Pericardial Function

**Mechanical**

- Physical protection of the heart
- Maintenance of normal position and orientation of heart within the mediastinum
- Secretion of pericardial fluid to reduce friction/work of contraction

**Hemodynamic**

- Intact sac discourages acute (+ chronic) disproportionate chamber distension
- Promotes atrial filling during diastole

**Neurohumoral**

- Fluid and neurohumoral exchange between pericardium and epicardium is constantly ongoing (many pharmacologic agents are directly absorbed across the epicardial surface)
- Neuroreceptors in sac contribute to autoregulation of cardiac cycle via the autonomic nervous system
Mechanical Functions

I. Relatively inelastic cardiac envelope.
   A. Maintenance of normal ventricular compliance (volume-elasticity relation).
   B. Defense of the integrity of the Starling mechanism which operates uniformly at all intra-ventricular pressures because presence of pericardium.
      1. Maintains ventricular function curves.
      2. Limits effect of increased LV end-diastolic pressure.
      3. Supports output responses to:
         a. Venous inflow loads and atrioventricular valve regurgitation (particularly when acute).
         b. Rate fluctuations.
Mechanical Functions

B. Defense of the integrity of any Starling mechanism:

4. Hydrostatic system (pericardium plus pericardial fluid) distribute hydrostatic forces over epicardial surfaces.
   a. Favors **equality of transmural end-diastolic pressure** throughout ventricle, therefore uniform stretch of muscle fibers (preload).
   b. Constantly **compensates for changes in gravitational** and inertial forces, distributing them evenly around the heart.
Normal Pericardial Physiology

Mechanical Functions

C. Limitation of excessive acute dilation.
D. Protection against excessive ventriculoatrial regurgitation (atrial support).
E. Ventricular interaction: relative pericardial stiffness.
   1. Provides a mutually restrictive chamber favoring balanced output from right and left ventricles integrated over several cardiac cycles.
   2. Permits either ventricle to generate greater isovolumic pressure from any volume.
   3. Reduces ventricular compliance with increased pressure in the opposite ventricle (e.g., limits right ventricular stroke work during increased impedance to left ventricular outflow.)
F. Maintenance of functionally optimal cardiac shape.
Normal Pericardial Physiology

Mechanical Functions

II. Provision of closed chamber with slightly subatmospheric pressure in which:
   A. The level of transmural cardiac pressures will be low, relative to even large increases in “filling pressures” referred to atmospheric pressure.
   B. Pressure changes aid atrial filling via more negative pericardial pressure during ventricular ejection.

III. “Feedback” cardiocirculatory regulation via pericardial servomechanisms.
   A. Neuroreceptors detect lung inflation and (via vagus): alter heart rate and blood pressure.
   B. Mechanoreceptors: Lower blood pressure and contract spleen.
Intact pericardium modulates left and right ventricular stroke volumes...
These results indicate that, in acute ischemia, the pericardium inhibits paradoxical systolic expansion of the ischemic region and increase in end-systolic length of non-ischemic segment. Thus, it is concluded that the pericardium modifies the regional myocardial systolic function in acute ischemia...
Normal Pericardial Physiology

Membranous Function

- Shields the heart by reducing external friction
- Production of pericardial fluid
- Generation of phospholipid surfactants
II. Membranous functions

I. Reduction of external friction due to heart movements.
   A. Production of pericardial fluid.
   B. Generation of phospholipid surfactants.

II. Buttressing of thinner portions of the myocardium (reciprocal variations in parietal pericardial thickness).
   A. Atria.
   B. Right ventricle.

III. Defensive immunologic constituents in pericardial fluid.

IV. Fibrinolytic activity in mesothelial lining.

V. Prostacyclin (PGE2, PGI2 and eicosanoids) released into pericardial sac in response to stretch, hypoxia and increased myocardial loading/work.

VI. Synthesis and release of endothelin, increased by angiotensin III stimulation.

VII. Barrier to inflammation from contiguous structures.
Normal Pericardial Physiology

Neuro-hormonal Feedback

- Prostaglandins and prostanoids constantly released by pericardial lining (mesothelium) in response to stretch

- Modulates caliber and tone of underlying coronary vessels (direct vasodilation by prostaglandin and indirectly by opposing coronary spasm by altering pericardial sympathetic neurotransmission)

- Since prostanoids inhibit efferent sympathetic effects may alter cardiac electrophysiology including reperfusion arrhythmias and myocardial contraction
Normal Pericardial Physiology

Neuro-hormonal Feedback

- Prostacyclins in the pericardium are a potent inhibitor of platelet aggregation: fibrinolysins in the intact mesothelial serosa oppose both intrapericardial clotting and formation of adhesions.
Prostaglandins in the pericardial fluid modulate neural regulation of cardiac electrophysiological properties.

“the pericardium produces prostaglandins that play a role in neural regulation of cardiac electrophysiological properties by modulating epicardial nerve effects...”

Miyazaki, T: Pride, H P: Zipes, D P

Circ-Res. 1990 Jan; 66(1): 163-75
Normal Pericardial Function

Summary

- Significant modulator of ventricular filling, heart deformation during diastole, the structural behavior of the septum and the LV and RV free walls
- Enhances RV function and is a strong modulator of left-right ventricular interdependence
- Protects against abrupt changes in volume, and subsequent, stretch-related electrical phenomena such as postoperative atrial fibrillation
Normal intra-cardiac pressures often result in markedly larger atrial and right ventricular volumes after the chest and the pericardium have been opened.

It is important to recognize, from more recent work on pericardial physiology, that the volume of any cardiac chamber depends upon its transmural pressure (i.e., intra-cardiac minus pericardial pressure), not upon intracardiac pressure per se.
Normal Pericardial Function

Summary

- Increased LV volume subsequent to pericardiotomy changes coronary blood flow distribution causing reduced coronary artery perfusion and decreased myocardial blood flow.

- The constraining effects of the native pericardium prevents the increased oxygen and coronary flow requirements produced by ventricular dilatation.
Why Close the Pericardium
Why Close Pericardium?

- Restore normal anatomy
- Restore normal physiology
- Re-compartmentalization of the intrapericardial microenvironment
- Re-compartmentalize mediastinum / thorax
- Maintenance of Retrosternal Distance
- Prevent complications (AF, adhesions, tamponade, CHF, etc)
Why close Pericardium?

Minimize Postoperative Adhesions

- Reoperations comprise 10% of adult cardiac surgeries performed annually.
- 50,000 resternotomies are performed in the US per year.
- The documented rate of catastrophic injury during resternotomy is approximately 1%.
- It can be estimated that a minimum of 5,000 catastrophic injuries are still occurring every year.
- Given the high risk of mortality associated with massive hemorrhage, many surgeons have advocated for closure of the pericardium or other protection of the epicardial surface whenever possible.
Why close Pericardium?

Maintenance of Retrosternal Distance

- Closure of the pericardium helps maintain a safe postoperative distance between the heart and the sternum.
- Maintenance of native cardiac geometry within the mediastinal space is important for preserving LV function after cardiac operation.
- Inadequate retrosternal distance has been cited as a potential risk factor for injury during re-sternotomy.
- Primary pericardial closure results in a significant and clinically meaningful increase in retrosternal distance immediately and long-term after operation.
Why Close Pericardium?

Re-compartmentalization of the intra-pericardial microenvironment

• Complete closure of the pericardium sequesters the heart from shed blood and its component cytokines and other pro-inflammatory mediators, as well as from infection.

• Inflammation has been implicated in the genesis of postoperative atrial fibrillation

• Exposure of the heart to pericardial and extrapericardial blood has been implicated in the development of cardiac adhesions and post-pericardiotomy syndrome
Why close Pericardium?

Re-compartmentalization of the intra-pericardial microenvironment

- Studies have demonstrated that repairing the pericardium may improve postoperative outcomes and facilitate patient management by enabling accurate differentiation between postsurgical bleeding of cardiac or mediastinal origin.
Closure of pericardium after open heart surgery. A way to prevent postoperative cardiac tamponade.

P Nandi, J S Leung and K L Cheung

“Absence of tamponade in the closed pericardium group can be explained by the fact that blood from extrapericardial sources of bleeding cannot collect round the heart because the pericardium is closed.”

British Heart Journal 1976;38:1319-1323
How do we do it?

Examples of Pericardial Closure
A pericardial relaxing incision has been performed on the right side; the lung can be seen through the intact mediastinal pleura. The dotted lines indicate additional or alternate sites for the incisions.
Clinical Implantation

Running suture down left and right sides

Opening for chest tubes

CorMatrix ECM®
Is this information convincing enough?
Pericardial Closure: Patient outcomes
CorMatrix ECM is Derived from Porcine Small Intestinal Submucosa

The SIS-ECM is obtained from the submucosa of the small intestine of pigs.
CorMatrix ECM™
Derived from porcine small intestinal submucosa
Special Properties of the ECM

- Immuno-modulatory
- Anti inflammatory
- Antimicrobial
- Site specific remodeling
- Chemo-attractant to progenitor cells
Retrospective review of isolated CABG patients undergoing ECM pericardial repair

- Primary Isolated CABG Patients
- Repair Group treated with CorMatrix ECM
  - Complete circumferential suturing of CorMatrix ECM to native pericardium
- Control Group not treated, pericardium left open
- 222 patients, 3 institutions
<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Control Group</th>
<th>Repair Group</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>N</strong>=111</td>
<td></td>
<td>N=111</td>
<td></td>
</tr>
<tr>
<td>Age range, years</td>
<td>35-85</td>
<td>39-86</td>
<td>0.13</td>
</tr>
<tr>
<td>Mean age, years (SD)</td>
<td>64.5 (11.2)</td>
<td>62.3 (10.7)</td>
<td>0.13</td>
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<tr>
<td>Mean height, inches (SD)</td>
<td>67.7 (3.9)</td>
<td>67.9 (3.9)</td>
<td>0.69</td>
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<tr>
<td>Mean weight, pounds (SD)</td>
<td>193.2 (40.9)</td>
<td>197.7 (38.4)</td>
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<tr>
<td>Sex, male</td>
<td>64.90%</td>
<td>67.60%</td>
<td>0.78</td>
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<tr>
<td>Ethnicity2, white</td>
<td>85.2% (92/108)</td>
<td>81.7% (89/109)</td>
<td>0.59</td>
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<tr>
<td><strong>Medical History</strong></td>
<td></td>
<td></td>
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<tr>
<td>Left ventricular hypertrophy3</td>
<td>5.0% (5/101)</td>
<td>6.9% (7/103)</td>
<td>0.77</td>
</tr>
<tr>
<td>Left bundle branch block3</td>
<td>2.0% (2/101)</td>
<td>3.0% (3/103)</td>
<td>1</td>
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<tr>
<td>Previous revascularization</td>
<td>11.70%</td>
<td>3.60%</td>
<td>0.04</td>
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<tr>
<td>Congestive heart failure</td>
<td>9.00%</td>
<td>8.10%</td>
<td>1</td>
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<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>15.30%</td>
<td>22.50%</td>
<td>0.23</td>
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<tr>
<td>Hypertension</td>
<td>80.20%</td>
<td>83.80%</td>
<td>0.6</td>
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<tr>
<td>Peripheral vascular disease</td>
<td>9.90%</td>
<td>18.00%</td>
<td>0.12</td>
</tr>
<tr>
<td>Bleeding</td>
<td>0.00%</td>
<td>0.00%</td>
<td></td>
</tr>
<tr>
<td><strong>Medications</strong></td>
<td></td>
<td></td>
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<tr>
<td>Beta-adrenergic blocker</td>
<td>47.3% (52/110)</td>
<td>49.60%</td>
<td>0.79</td>
</tr>
<tr>
<td>Statin</td>
<td>61.0% (67/110)</td>
<td>54.10%</td>
<td>0.34</td>
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<tr>
<td>Anti-arrhythmic</td>
<td>3.6% (4/110)</td>
<td>3.60%</td>
<td>1</td>
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<tr>
<td>Calcium channel blocker</td>
<td>32.7% (36/110)</td>
<td>19.80%</td>
<td>0.03</td>
</tr>
<tr>
<td>Angiotensin converting enzyme inhibitor</td>
<td>28.2% (31/110)</td>
<td>28.80%</td>
<td>1</td>
</tr>
<tr>
<td>Angiotensin II receptor blocker</td>
<td>3.6% (4/110)</td>
<td>5.40%</td>
<td>0.75</td>
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<tr>
<td>Nonsteroidal anti-inflammatory Drug</td>
<td>38.2% (42/110)</td>
<td>33.30%</td>
<td>0.49</td>
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<tr>
<td>Steroids</td>
<td>2.7% (3/110)</td>
<td>1.80%</td>
<td>0.68</td>
</tr>
<tr>
<td>Magnesium</td>
<td>0.9% (1/110)</td>
<td>0.90%</td>
<td>1</td>
</tr>
</tbody>
</table>
Retrospective review of isolated CABG patients undergoing CorMatrix ECM pericardial repair

<table>
<thead>
<tr>
<th>Adverse Event</th>
<th>Control Group</th>
<th>Repair Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Postoperative Complications</td>
<td>29/111</td>
<td>37/111</td>
</tr>
<tr>
<td>Intra-operative complications</td>
<td>2/111</td>
<td>6/111</td>
</tr>
<tr>
<td>Acute inflammation</td>
<td>1/111</td>
<td>0/111</td>
</tr>
<tr>
<td>ARDS</td>
<td>6/111</td>
<td>3/111</td>
</tr>
<tr>
<td>Allergic reaction</td>
<td>0/111</td>
<td>1/111</td>
</tr>
<tr>
<td>Angina</td>
<td>0/111</td>
<td>0/111</td>
</tr>
<tr>
<td>Anti-coagulation complications</td>
<td>1/111</td>
<td>1/111</td>
</tr>
<tr>
<td>Bleeding</td>
<td>0/111</td>
<td>0/111</td>
</tr>
<tr>
<td>Chronic inflammation</td>
<td>0/111</td>
<td>0/111</td>
</tr>
<tr>
<td>Cognitive sequelae</td>
<td>1/111</td>
<td>0/111</td>
</tr>
<tr>
<td>Infection</td>
<td>0/111</td>
<td>0/111</td>
</tr>
<tr>
<td>ECM migration</td>
<td>0/111</td>
<td>0/111</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>0/111</td>
<td>0/111</td>
</tr>
<tr>
<td>Pericarditis</td>
<td>0/111</td>
<td>0/111</td>
</tr>
<tr>
<td>Renal failure</td>
<td>2/111</td>
<td>1/111</td>
</tr>
<tr>
<td>Stroke</td>
<td>1/111</td>
<td>1/111</td>
</tr>
<tr>
<td>Tamponade</td>
<td>1/111</td>
<td>1/111</td>
</tr>
<tr>
<td>Death</td>
<td>1/111</td>
<td>0/111</td>
</tr>
<tr>
<td>Other</td>
<td>22/111</td>
<td>27/111</td>
</tr>
</tbody>
</table>
3 year clinical experience with CorMatrix ECM

- Provides an environment for tissue specific cardio-vascular remodeling
- Safe, reliable and cost-effective
- No adverse complications
- Provides structural integrity during remodeling
- Pericardial closure restores the normal anatomy, facilitates perioperative patient care
- Facilitates surgical approach for redo operations
- Protects underlying grafts and cardiac structures
Pericardial Repair
Pericardial Structure Repaired
3 YEARS POST-OP
Other important advantages

Decreased Postoperative Atrial Fibrillation???
Atrial Fibrillation After Cardiac Surgery

- Occurs in about 30% of patients

- Associated with increased mortality (both early and late), increased morbidity, increased cost

- Incidence is reduced but not eliminated by the use of pre-operative antiarrhythmic agents — B-Blockers and Amiodarone most effective
Pathogenesis of POAF

Pre-disposing factors:
- Advanced age
- Hypertension
- Diabetes
- Obesity
- Metabolic syndrome
- Left atrial enlargement
- Diastolic dysfunction
- Left ventricular hypertrophy
- Genetic predisposition

Intraoperative factors:
- Surgical atrial injury
- Atrial ischemia
- Pulmonary vein vent
- Venous cannulation
- Acute volume changes

Post-operative factors:
- Volume overload
- Increased afterload
- Hypotension

- Inflammation
- Oxidative stress

Triggers:
- Atrial premature contractions
- Imbalance of autonomic nervous system
- Electrolyte imbalance (hypomagnesemia, hypokalemia)

Atrial structural substrate

Dispersion of atrial refractoriness

Multiple re-entry wavelets

Atrial electrophysiological substrate

Additional Trigger
- Transepicardial absorption of inflammatory cytokines

POAF
Circumferential Pericardial Reconstruction Using an Extracellular Matrix Implant Correlates with Reduced Risk of Postoperative Atrial Fibrillation in Coronary Artery Bypass Surgery Patients

W. Douglas Boyd, MD, William E. Johnson, III, MD, Parvez K. Sultan, MD, Thomas F. Deering, MD, FACC, FACP, FHRS, Robert G. Matheny, MD, FACS

University of California Davis Medical Center, Davis, California, USA; Mobile Infirmary Medical Center, Mobile, Alabama, USA; Trinity Medical Center, Birmingham, Alabama, USA; Piedmont Heart Institute, Atlanta, Georgia, USA; CorMatrix Cardiovascular, Atlanta, Georgia, USA

- Conclusions: In this retrospective study, circumferential pericardial reconstruction with the ECM implant contributed directly to a statistically significant and clinically meaningful reduction in the rate of postoperative AF in primary isolated CABG patients. A prospective, multicenter, randomized trial has been planned to further test this approach.
Methods:

A retrospective comparison of the incidence of postoperative AF in 111 patients who underwent a circumferential pericardial reconstruction procedure with the CorMatrix ECM for Pericardial Closure (CorMatrix Cardiovascular Inc., Atlanta, GA) following primary isolated CABG, versus a control group of 111 patients who did not undergo pericardial reconstruction.
Results:

Postoperative AF occurred in 43 of 111 control patients (38.74%, LCL 29.64, UCL 48.45) but only 20 of 111 treated patients (18.02%, LCL 11.37, UCL 26.45) representing a 53.8% relative risk reduction in the treatment group (P=0.0003). There was a small but not statistically significant decrease in hospital LOS in treated patients. The two treatment groups exhibited a similar postoperative complication profile.
Circumferential pericardial reconstruction with CorMatrix ECM after cardiac operations is:

- Safe
- Effective in re-establishing normal pericardial function in the immediate and long term post-operative periods
- Increases the safety of sternal re-entry
- Appears to reduce the incidence of post-op atrial fibrillation
How Might ECM Pericardial Reconstruction Discourage The Occurrence of Post-op Atrial Fibrillation?

- Prevent acute chamber distension
- Prevent rundown of “soup” from wound onto epicardial surface
- Inhibits inflammatory response to surgical wound
- Induces remodeling response in the surgical wound
CorMatrix for Pericardial Closure
Potential Impact on Reducing New Onset Post-Op Atrial Fibrillation

Published Article reporting a 54% reduction in post-op Atrial Fibrillation following pericardial reconstruction using CorMatrix

Prospective Multi-center FDA approved study underway to confirm impact on reducing post-op Atrial Fibrillation
Arguments against closing the pericardium
Should the pericardium be closed routinely after heart operations?

Vivek Rao, MD, PhD\textsuperscript{a}, Masashi Komeda, MD, PhD\textsuperscript{a}, Richard D. Weisel, MD\textsuperscript{a}, Gideon Cohen, MD\textsuperscript{a}, Michael A. Borger, MD\textsuperscript{a}, Tirone E. David, MD\textsuperscript{a}

The cardiac index and stroke work index were lower in the closure group compared to the open group ($P<0.001$), however, these difference were only present for one hour post operatively and at 4 h and 8 h post operatively no difference could be determined.
Pericardial Closure: Arguments against

Adverse changes in hemodynamics

- A transient reduction in cardiac index and stroke work index following primary pericardial closure has been documented in humans in one small, prospective randomized clinical trial.

- Several clinical case series have also reported mild-to-moderate cardiac constriction, reduction in cardiac output and a fall in arterial pressure resulting from primary pericardial closure.
Pericardial Closure: Arguments against

Adverse changes in hemodynamics

- Are consistent with the combined effects of contraction of the pericardium, dilation of the cardiac chambers in the absence of the pericardium, and the adverse effects of CPB.
- Studies have shown these effects to be transient in nature.
- No study has ever reported an adverse clinical outcome.
Pericardial Closure: Arguments against

Perceived risks of graft compression

- Several authors have asserted that pericardial closure could lead to kinking of bypass grafts or IMA conduits
- No published studies regarding this phenomenon
- Not observed in >40,000 closures
Pericardial Closure: Arguments against

Perceived risks of graft compression

- Pericardial substitutes can be tailored to allow circumferential closure that approximates normal pericardial constraint around the ventricles, while avoiding adverse effect on grafts.
Pericardial Closure: What I have learned

- There are no real contraindications to pericardial closure
- The details are important!!!!
- How you open pericardium can facilitate closure
- Preservation of Thymus, pre-closure of notches, early CT suction
- No gaps, separate chest tubes
- Blake tubes have to be frequently “milked”
- In my experience, hemodynamic compromise after closure has virtually NEVER been related to pericardial closure
- Until comfortable, do early post-op / pre-discharge echoes and CT scans to evaluate the pericardial sac.
Top Reasons to Close Pericardium

1) Restores normal anatomy
2) Allows normal function
3) Protects underlying bypass grafts early / late reentry
4) Helps guide postoperative patient management
5) Helps prevent tamponade
6) Potential for improving RV function
7) Normalizes postoperative hemodynamics
8) Takes minimal time, is reimbursable and cost effective
9) May prevent post-op AF
10) Improvement of patient outcomes
11) No evidence of adverse effects