Surgical Repair of Left Ventricular Aneurysms

- Review of Literature.
- Al Bassel Heart Institute Experience

Majed Othman M.D.,
Al Bassel Heart Institute, Damascus - Syria
Causes of Heart Failure

Non-Ischemic: 32%
Ischemic: 68%

Gheorghiade, 1998
Definition

Left Vent. Aneurysm:
- Distinct area of abnormal L.V. diastolic contour with systolic dyskinesia or paradoxical bulging.

Or
- Any large area of L.V. “akinesia or dyskinesia “ that reduce L.V. ejection fraction.
- May - Intraoperatively: an area that collapses upon left ventricular decompression.
INCIDANCE

- Left ventricular aneurysms develops in up to 30-35% of patients with Q wave MI.
- Left ventricle is involved in 95% of PTS. And right ventricle 5%
- Anterior wall and/or apex of the left ventricle 80% as common as those of inferior or infero-posterior wall.
The 3D architecture of the heart

The Ellissoid is the geometric form that most approaches the shape of the normal ventricle.

An elliptical shape allows a normal wall stress in the three directions (longitudinal, circumferential and radial) and an optimal direction of the blood stream at the inflow and the outflow tract.
LV Apex is anatomically damaged following MI

Impairing of apical function result in severe reduction of:
- Twisting and Untwisting
- EF


Apical Twisting and Untwisting
Pathophysiology

Development of a true L.V.A. involves two principal phases:

- Early expansion phase.
- Late Remodeling.
LV Remodeling

- acute MI (hrs)
- infarct expansion (hrs to days)
- global remodeling (days to months)
Early Expansion Phase

Start with onset of M.I.  
- 50% of pts. have L.V.A within 1st 48h.  
- 50% of pts. have L.V.A within 2 weeks.  
- Transmural infarction:  
- Myocyte Death begins 19mn post M.I.

Few days

Endocardial surface becomes smooth + loss of trabeculae + Deposition of fibrine +

Thrumbus 50%  
“Viable myocytes ± remain within the infarct zoon”
- 2-3 days

Inflammatory cells migration  
- 2-3 days

Lysis of necrotic myocytes  
- 10 days

Disruption of native collagen network.
- Bulging " infarct zoon " = loss of systolic contraction.
- Laplace low: $T = \frac{Pr}{2h}$.
  - $T$ = muscle fiber tension.
  - $P$ = ventricular pressure constant.
  - $r$ = Radius
  - $h$ = wall thickness

Deformation or stretch over time under constant load leads to wall stress "syst., diast." 

Infarct Expansion.
Infarct Expansion leads to:

- ↑ Diastolic stretch + ↑ Catecholamines
  - ↑ fibres Shortening → hypertrophy
  - ↑ O2 demand
- ↑ Regional wall stresses + ↑ O2 demand

↓ Cardiac output

So: L.V.A → Systolic and Diastolic Dysfunction.
II – Late Remodeling phase

Begins 2-4 weeks post M.I.
Nitrate Therapy ↓ Stretch
↓ Hypertrophy

A.C.E ↓ Expansion

* It is speculated that coronary reperfusion as late as 2 weeks after M.I. prevents aneurysm formation by improving blood flow and fibroblast migration into the infarcted myocardium.
Surgical Approach

- **Plication:**
  Without opening the aneurysm is reserved for only the smallest aneurysms that do not contain mural thrombus.

- **Linear closure.**

- **Circular patch.**

- **Endoventricular patch.**

- **Other Ventricular Remodling Techniques**
  Cellular Cardiomyoplasty.
Surgical approach

Problems:

- Volume
- Shape + geometry concity – elliptical
- Neurohumeral ventricular dysfunction
- Regional function
- Treat the disease, not symptoms return to normal.
- Dynamic function
- Cardiac architecture
- Safety and efficacy
The overall objective is to make our mental concept guide surgical activities, and thus go beyond evident pathology in corrective efforts. Our intent is to escape the boundary of the visible disease, and aim restoration toward the boundary of normality.
Early Results

Risks factors for hospital mortality:
- Age.
- Incomplete Revascularization.
- Gender (Females more than males).
- Ejection fraction less than 20%.
- Concurrent mitral valve replacement.
- Pre-operative cardiac index < 2 l/min/m.
- Mean pulmonary pressure > 33 mm hg
- Serum creatinine > 1.8 mg dl.
- Failure to use internal mammary artery.
In hospital complications

- Low cardiac out-put 22-39%
- Ventricular arrhythmias 9-19%
- Respiratory failure 4-11%
- Bleeding 4-7%
- Dialysis – dependant Renal failure 4%
- Stoke 3-4%
Indications

- Antero Septal infarction and dilated left ventricle (End diastolic volume index > 100m2/m2).
- Depressed ejection fraction (even below 20%).
- Left ventricular regional asynergy, either Dyskinesia or Akinesia, greater than 35% of the ventricular perimeter.
- Symptoms of Angina, heart failure, arrhythmias or combination of the three. Or
- Inducible ischemia on provocative tests in asymptomatic patients.

“From: The journal of thoracic and cardiovascular surgery vol.: 124, No 5 889
Lorenzo Menicanti / Marisa Di Donato”
Tissue heterogeneity increases susceptibility to ventricular arrhythmia

The presence of infarcted tissue or scar forms the substrate for malignant reentrant arrhythmias. Infarcts can have marked spatial heterogeneity, with areas of necrosis interspersed with bundles of viable myocytes, particularly in the border zones and periphery of the infarct.

Interaction among dispersion of regional wall motion, repolarization, and arrhythmia is another manifestation of the importance of Mechano Electric Feedback in arrhythmogenesis.

Ischemic Dilated Cardiomyopathy
Surgical Options

CABG alone?

CABG plus MV repair?

CABG plus Surgical Ventricular Restoration and MV repair?

CABG plus Surgical Ventricular Restoration?
Contraindications

Relative:

- Systolic pulmonary artery pressure more than 60 mmHg. not associated with severe mitral regurgitation.
- Sever right ventricular, dysfunction.
- Regional asynergy without dilation of the ventricle “Risk of too small residual chamber“.
S.V.R to Re-size and Re-shape patient selection

To whom?

Why?

How?
Al Bassel Heart Institute experience
March 2005

Before
Endoventricular Circular Patch

After
Endoventricular Circular Patch, assisted by Manican TR3. I.S.V.R

Linear Reconstruction Assisted by Manican TR3. I.S.V.R

70 pts
25 pts

Pts: 95 pts
Excluded: ventricular aneurysm type III – IV

Sex: ___ 70 M 73.6%
25 F 26.3%

Age: 35 – 73 yrs.
Pathology: All pts.
Ischemic Cardiomyopathy
Angiographic LV silhouettes - RAO 30

Type I: AB, AL, PB, INF

Type II

Type III

Type IV

M Di Donato, AHA Orlando, 2003
Type of Surgery

Type I  ---------  15
Type II  ---------  80
Type III  ---------  Excluded
Type IV  ---------  Excluded
Symptoms & Finding

N.Y.H.A ---------------- III ------------ 77 --------- 81%
IV -------------------18pts -----18.9%

M.R ( 0 – 4+ ) --------+2 ----- 65 pts ------ 68.4%
+3 ------ 20 pts ------ 21%
+4 ------ 10 pts ----- 10.5%

Arrhythmia : Sinus tachycardia
Supra ventricular tachycardia
Nuclear Scanning : done for all pts.
Type III
Functional Mitral Regurgitation

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Results

Mortality: 4/95 ------- (4.2%)

Influenced by:
- P.A.P
- M.R
- Dilated L.V.
- Intra Aortic balloon pump x8

Short Term Results:
- N.Y.H.A= ok
- M.R = O
- EF= 20% Improvement.
### Hemodynamic Review

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CASE REPORT I

PT.  F.D  57 Y.   Male

1ST admission in urgency  23/2/2006  Ant. M.I.
Cath. : LAD occluded    Reopening & stenting   Discharged

2nd admission 16/03/2006
Dyspnea +++    Chest pain +++

Echo : "apical aneurysm"
Ruptured into pericardium + sever mitral regurgitation.

Recath : LAD = good flow
Huge aneurysm and free apical wall rupture

Decision : Surgery in urgency
- Reconstruction of L.V.
- Mitral plasty

Now : Good condition
### Hemodynamic Calculations

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27 Mar 06 14:07
CASE REPORT II

Pt. : F.B.  30 y  M
- Chest pain + abdominal pain for 2 days. Diagnosis “Irritable Cologne”
- Referred to hospital
- E.C.G. : Extended Acute Anterior well infarction
- Chest : X.Ray : pleural effusion I
- Echo : Bid
  LviDd : 6.2 Lvids : 4.7  EF = 28%
  R.E.F = 40%
  End. Systolic volume  126 ml
  End. Diastolic volume 175 ml
- Cath. in urgency :  LAD = occluded + huge apical aneurysm
- Indicated to surgery in urgency
- Follow up : Very well
- Results : Going very well
CONCLUSIONS

Arrest the Progression of CHF

Reduce the SIZE of the ventricle
- Reduces Wall Tension
- Reduces/Eliminates expansion of the akinetic zone
- Restores Proper Volume

Restore the elliptical SHAPE of the ventricle
- Increases Contractility
- Increases Filling Rate

Restore the ORIENTATION of the ventricle
- Align all components to work Efficiently
- Reorient papillary muscles
There must be larger and earlier diagnoses for large wall motion abnormality after myocardial infarct, mainly when revascularization is indicate, in order to prevent secondary deterioration.

Extension of diagnosis towards akinesia and some global ischemic akinesia, in order to stabilize the physiology of the remaining contractile muscle.

Cardiologists must give attention in view of an extended ventricular scar (akineti or dyskinetic ) after myocardial infarction and not wait for the natural evolution (LV Remodeling) “Vincent Dor”
THANK YOU
Left ventricular apex occluder. Description of a Ventricular Partitioning Device

Hugh Sharkey1*; Serjan Nikolic1, PhD; Alexander Khairkhahan2; Michael Dae2, MD

1. Cardiokinetix Inc., Redwood City, California; 2. University of California, San Francisco, California, USA

Description

Implant device and delivery system.

The Ventricular Partitioning Device (VPD) is an intraventricular implant that is deployed in the Left Ventricle (LV) in patients with regional wall motion abnormalities following myocardial infarction (MI). The implant device is delivered via a guiding catheter and delivery system introduced percutaneously from the femoral artery by the standard techniques for left heart catheterization. The implant device is an ePTFE membrane bonded to an expanded NiTi frame in the configuration of an inverted umbrella. (Figure 1) The stem of the umbrella is positioned in the apex of the LV and has a collapsible foot designed to hold the implant off the endocardium a preset distance. The struts that support the membrane have a parabolic configuration that allows the rim of the membrane and the passive anchors located at the tip of each of the struts to engage the endocardium and maintain the position of the implant by spring tension against the endocardial wall (Figure 2). The guide catheter is a braided catheter with a preformed distal tip that is designed to orient it directly into the LV apex. A Dilator is placed within the guide catheter during placement, and accommodates standard intravascular guidewires to assist in placement of the guide catheter. The guide catheter with dilator in place is advanced retrograde across the aortic valve with the guidewire positioned beyond the tip and is placed at the apex of the LV. The guide catheter has a side port and haemostatic valve that allows injection of contrast around the delivery catheter. The delivery catheter is used to advance the VPD implant within the guide catheter following placement of the guide catheter in the LV. It has a distal screw mechanism for engaging and disengaging the implant. The delivery catheter has a compliant balloon located just...
Figure 3. Delivery Catheter and Implant.

Figure 4. Placement of the VPD implant device in the LV.
EKG Changes Associated With MI

Before coronary occlusion
Heart muscle normal

Onset and first several hours
Subendocardial injury and myocardial ischemia. No infarction (cell death) yet

First day
Ischemia and injury extend to epicardial surface. Subendocardial muscle dying in area of most severe injury

First and second days
Transmural infarction nearly complete. Some ischemia and injury may be present at borders

After two or three days
Transmural infarction complete

After several weeks or months
Infarcted tissue replaced by fibrous scar, sometimes bulging (ventricular aneurysm)
**Understanding the Silhouette Method of Assessment**

The silhouettes were inspired by the centerline method which is used to model regional wall motion in the ventricle. The four silhouette types were developed by Marisa Di Donato, M.D., San Donato Hospital, Milan, Italy, based on analysis of over 1100 post MI patients.

The **RED** line depicts endocardial wall motion at peak systole and **BLACK** line depicts endocardial wall motion at peak diastole. The **PURPLE** intersecting lines dissect the territories of involvement, and the **BLUE** vertical lines illustrate the area of contraction. The silhouette is applicable when comparing images from most diagnostic imaging modalities.
Contractile Silhouette Types

Type I

Type II

Type III
Correlates involving mechanical changes

- Dilated heart, and poor ejection fraction: intramyocardial stress and strain
- Systemic overload (Hypertension, Aortic Stenosis)
- Regional ventricular stress (dyskinesia)
- QT interval and U wave
- QRS complex
- Heart rate variability
- Alternans (etherogeneity, T wave)
- Electrolyte disturbances
- The autonomic nervous system
A chamber Size and Shape factor is addressed during surgical ventricular reconstruction (SVR) where the procedure includes:

- resection of scar,
- septal exclusion,
- cavity reduction by endoventricular patch
- coronary grafting and
- mitral repair when needed.
CONCLUSIONS

The high success rate of SVR is not linked to one single factor
But to the integrated components of the entire surgical procedure that:

- Relieves chronic ischemia (Complete revascularization)
- Reduces Ventricular dimensions (Stretch and stress)
- Excludes the scar (Patch)
- Interrupts the functional re-entry circuit that can develop ventricular arrhythmias (Cryoablation?)
Results of LV Remodeling

- Enlarged Ventricle
- Elliptical shape lost
- Fiber orientation lost
- Misdirected vector forces of contraction
- Misalignment of valves
- Loss of force and twisting action of contraction
- Misalignment of valves
- Reduced cardiac output
WHY SVR WORKS?

- Relieves ischemia through complete revascularization
- Reduces wall stress and stretch by reducing volume
- Reduces volume overload by reducing mitral regurgitation
- Improves LV geometry by re-shaping the ventricle
The SVR: Unresolved

- What is the perfect size and how to calculate it?
- To what extent is cardiac efficiency restored?
- Can the operating point for the reshaped ventricle be mathematically defined?
- Does reverse remodeling occur at the cellular level if the heart failure syndrome is reversed by an operation that reshapes it?
INDICATIONS for SVR (2)

-HF symptoms are the first indication for SVR but also pts presenting with ventricular arrrhythmias and/or angina who need surgical revascularization represent an indication for SVR if the previous conditions are present.

- For pts who are asymptomatic despite post-infarction LV dysfunction, serial echocardiographic studies to detect the first signs of deterioration (i.e. LV progressive enlargement or decline in EF) should be performed.
THANK YOU
found that the severity of the regurgitation was associated with the degree of sphericity of the patient's ventricle (KONO, JACC 1992; 20: 1594-1598).

**TR'ISVR surgical ventricular restoration**

SVR has evolved over 20 years and more than 2,500 procedures. This experience has shown the importance of the size, shape and orientation of the ventricle. The TR'ISVR™ procedure is the next evolution in ventricular restoration. The system features the Mannequin™, endoventricular shaping device, which serves as a template to produce the optimum size, shape and orientation of the patient's ventricle. For the first time, this procedure achieves consistent, repeatable, and accurate reshaping of the ventricle into an ellipse. The TR'ISVR System also includes the TR'ISVR endoventricular patch, patch sizing guide and procedural accessories. The system has FDA marketing clearance and has received the CE mark.

*Mitral regurgitation is related to the degree of sphericity of the ventricle.*
Surgery

Endoventricular patch as prescribed by Dor and developed by The Team of Milano, St. Donato by using TRISUR.
History

1st Angiographic diagnosis 1951
- 1944 : BECK Fascialata placation.
- 1955 : Likoff & Baily – Thoractomy clamping without C.P.B.
- 1958 : Cooly modern treatment linear repair + C.P.B.
- Geometric Reconstruction : Dor, Jatene, Cooly.
In hospital complications

- Low cardiac output
- Ventricular arrhythmias
- Respiratory failure
- Bleeding
- Dialysis dependant Renal failure
- Stroke
SVR to Re-size and Re-shape

To WHOM?

Patient selection
The Rationale to Reshape

WHY SVR?
SVR to Re-size and Re-shape

HOW?
### Pathology

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<tr>
<td>ANT. Latero – basal .MI</td>
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- **LAD**: Attent in all pts, or occluded by retrograde filling.
- **EF**: all PTS > 35%
  - Range: 14% —— 35%
- **Mean Delay from infarction**: 6 months —— 5 yrs
- **All PTS are chronic (except 2 pts acute)**
LV Volume Curve

EDV = 257 ml  EF = 23%
 ESV = 275 ml

ED VLA Slice (frame = 8)
ES VLA Slice (frame = 4)
LV Volume Curve

EDV = 250 ml
ESV = 201 ml

ED VLA Slice
(frame = 7)

ES VLA Slice
(frame = 3)
Al Bassel Heart Institute

experience

March 2005

Before

Endoventricular Circular Patch

After

Endoventricular Patch, assisted by Manican TR3. I.S.V.R

Linear Reconstruction Assisted by Manican TR3. I.S.V.R

70 pts

25 pts

Pts : 95 pts

Excluded: ventricular aneurysm type III – IV

Sex : ___ 70 M 73.6%

25 F 26.3%

Age : 35 – 73 yrs.

Pathology : All pts.

Ischemic Cardiomyopathy