

### Le protesi meccaniche

Si dice che funzionano per sempre ma non è così; anche senza endocardite, vanno incontro a distacchi tardivi, trombosi, pannus;

è necessario un follow-up attento e talora il reintervento





Le prime 1100 (1939-53)

La capostipite di tutte le "1100" fu la Fiat 508C «NuovaBalilla 1100»,

Grazie alla sua abitabilità, la 1100 ebbe in quegli anni un notevole successo specialmente come *TAXI* o come base per gli allestimenti ad *AMBULANZA*.



# Protesi meccaniche: (a palla); monodisco; doppio emidisco

Hufnagel



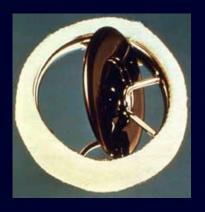
Starr



Kay-Shiley



**Bjork-Shiley** 



St.Jude



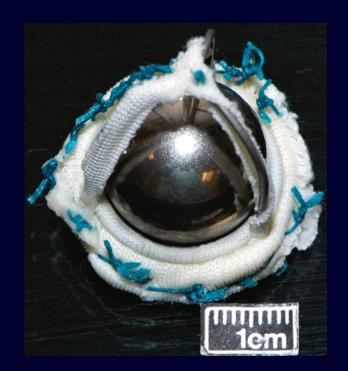
Sorin

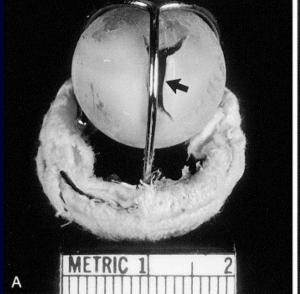


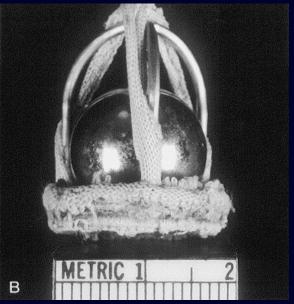


# Le "malattie" delle protesi: strutturale









Le "malattie" delle protesi: strutturale



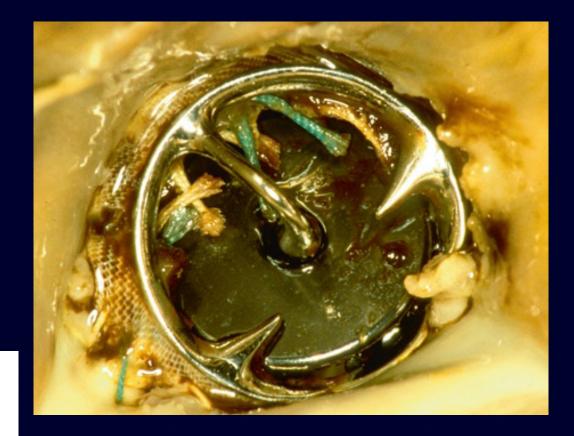


Le "malattie" delle protesi: distacco





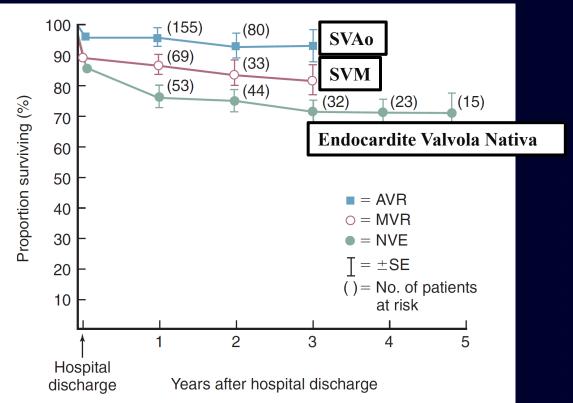
Le "malattie" delle protesi: tecnica







### Endocardite nativa vs Sost Valv Standard

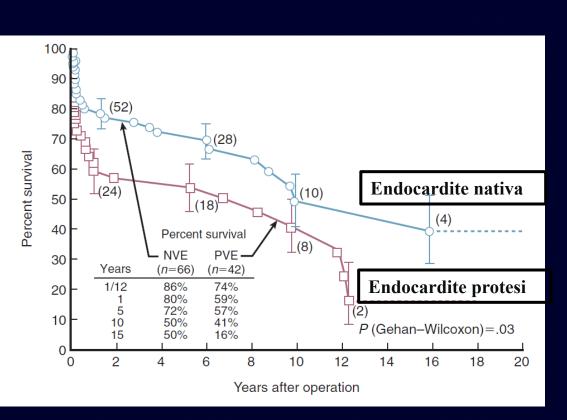


**Figure 15-12** Comparison of survival in patients operated on for native valve endocarditis with patients having isolated aortic or mitral valve replacement without infective endocarditis during the same era. Key: *AVR*, Aortic valve replacement; *MVR*, mitral valve replacement; *NVE*, native valve endocarditis; *SE*, standard error. (From Richardson and colleagues.<sup>R2</sup>)

Richardson JV, Karp RB, Kirklin JW, Dismukes WE. Treatment of infective endocarditis: a 10-year comparative analysis. Circulation 1978;58:589-97.

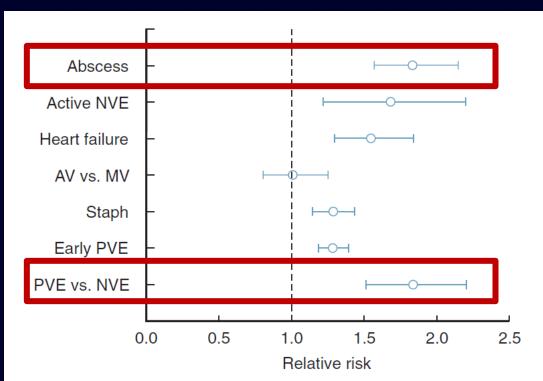
# Endocardite nativa vs protesi

**Figure 15-14** Survival according to initial native valve endocarditis *(NVE)* or prosthetic valve endocarditis *(PVE)* in 108 patients undergoing aortic valve replacement (P = .03). (From Haydock and colleagues.<sup>H3</sup>)



Haydock D, Barratt-Boyes B, Macedo T, Kirklin JW, Blackstone E. Aortic valve replacement for active infectious endocarditis in 108 patients. J Thorac Cardiovasc Surg 1992;103:130.

# Endocardite

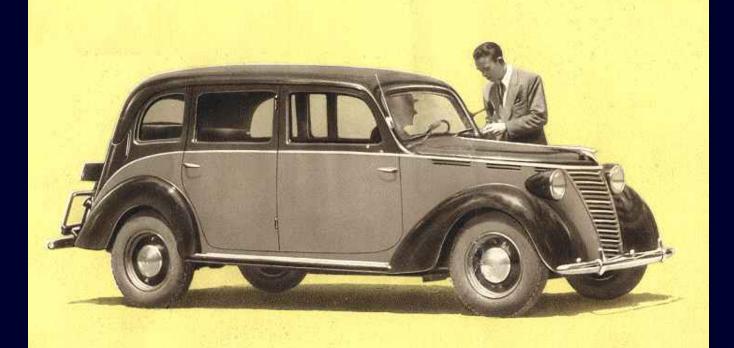


**Figure 15-15** Factors influencing operative mortality after surgical treatment of patients with infective endocarditis. Data are presented as relative risk (see Chapter 6 for definition) for each factor plus 95% confidence limits, as determined by meta-analysis of 30 major studies. Key: *AV*, Aortic valve; *MV*, mitral valve; *NVE*, native valve endocarditis; *PVE*, prosthetic valve endocarditis; *Staph*, *Staphylococcus aureus* or *Staphylococcus epidermidis*. (From Moon and colleagues.<sup>M13</sup>)

Moon MR, Miller DC, Moore KA, Oyer PE, Mitchell RS, Robbins RC, et al. Treatment of endocarditis with valve replacement: the question of tissue versus mechanical prosthesis. Ann Thorac Surg 2001;71:1164.

# Distacchi (275000 impianti/anno)

# Tassi FIAT 1100 BL



Tassi 1100 BL, a guida interna, 4-5 posti (di cui 2 su strapuntini), più il sedile anteriore per l'autista e altro strapuntino laterale al posto di guida per eventuale passeggero. In alternativa spazio per bagagli a mano. Scocca interamente metallica. Verniciatura nei colori regolamentari. Completezza di accessori.



# Distacchi (275000 impianti/anno)

Protesi Aortica 17%
Protesi Mitralica 22%

Fortunatamente >% benigni

Bioprotesi > Meccanica (??)

Rischio CCH elevato!

Favorenti leak: old age

Small BSA

Bioprotesi > Meccanica (??)

TEE - 3D ECO

Tecniche percutanee: Welcome!



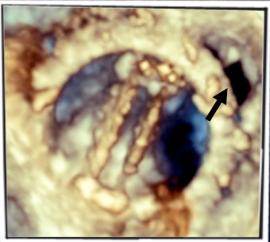




Circolare

Ovale





Incremento

### Il distacco per il CCH: inizia in SALA OPERATORIA

Protesi Aortica 6%

Protesi Mitralica 32%

> Complicanze post-op

> Degenza Intensiva e Degenza

Bioprotesi (63%) > Meccanica (36%)

TEE - 3D ECO

Favorenti leak: old age

CHF pre-op

IRC

> CEC > ClAo

Solo PVL moderati e severi

# Etiologia

Infezione
Stress meccanico
Fragilità anulare
Calcificazioni anulari
Deterioramento protesi

Canale tra 2 camere a pressione diversa 1-12.5% dei portatori di protesi

Sedi ben precise

Differenze importanti tra Ao e M

### Differenze importanti tra Ao e M

PVL aorta: IAo dipende da gradiente pressorio tra Ao ascendente e VSx in diastole

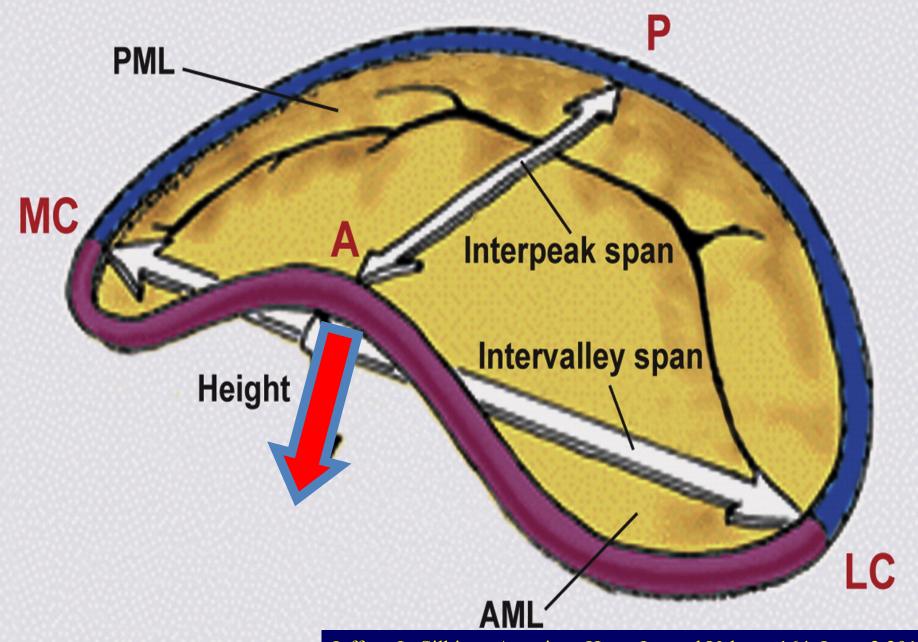
PVL Mitrale: IM dipende da gradiente pressorio tra VSx e Atrio Sx in sistole! (>)

Il gradiente pressorio di PVL mitralici causa flussi di velocità maggiori del jet di rigurgito che causa > sintomi e > emolisi

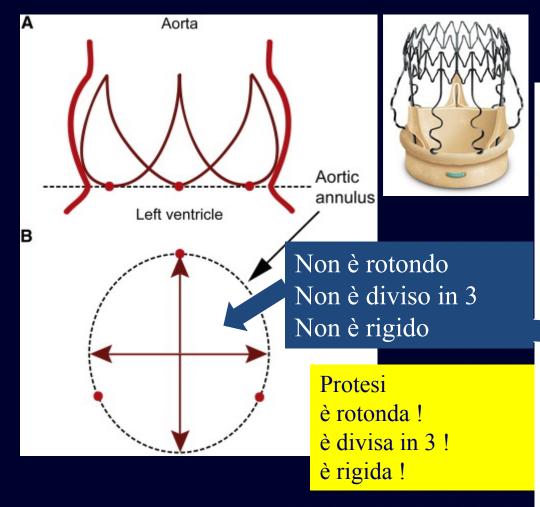
PVL mitralici evolvono più bruscamente



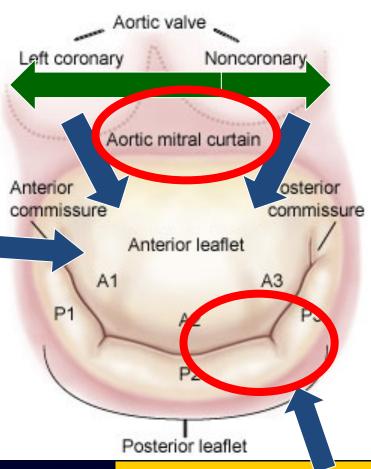
PVL aortici x lassità suture PVL mitralici x danno anulus ore 10-11 e mobilità ore 5



Jeffrey J. Silbiger. American Heart Journal Volume 164, Issue 2 2012 163 - 176



## Sedi ben precise

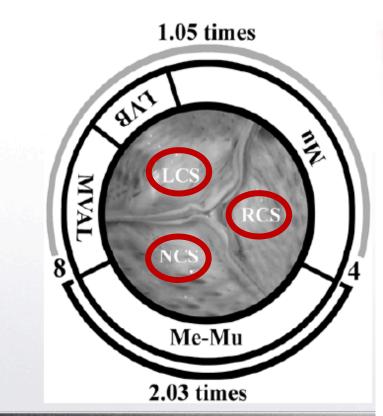


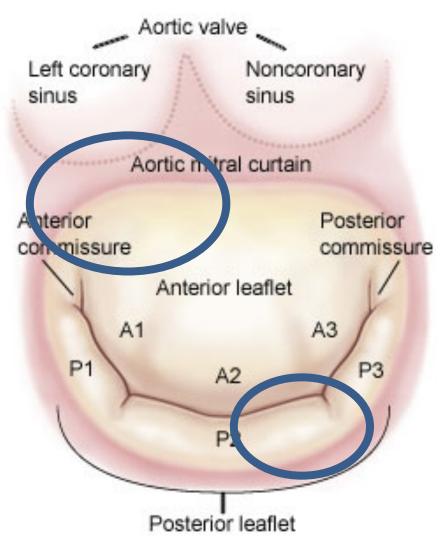
Zona molto mobile senza fibrosi

Mitrale: tenda mitroaortica tirata in basso

Aorta: seno non coronarico più basso, scende verso mitrale e durante SVAo lo *rettileinizzo*, tiro in su (traziono verso l'alto la tenda mitroaortica)!

# Sedi ben precise





Quando il distacco và rioperato?

La maggior parte PVL sono piccoli, asintomatici e seguono

un decorso benigno......ma se:

1 REDO mortalità 10%

2 REDO mortalità 15% 3 REDO mortalità 37%

Progressiva insufficienza cardiaca sintomatica

PVL

(M evoluzione più rapida di Ao)

50% SVAo o SVM 50% sutura PVL

Anemia emolitica incontrollata DOVUTA a PVL

Infezione persistente (indicazione per endocardite)



# TEE - 3D ECO CLINICA Cardiologo! EMATOLOGO!

# **EMATOLOGO!**

Non SOLO per diagnosi emolisi

Ma per escludere ALTRE CAUSE di emolisi

### Emolisi

Skoularigis and colleagues' criteria [10]

The patient has hemolysis if:

serum LDH > 460 U/L

and at least two of the following occur:

blood hemoglobin < 13.8 g/dL (male) or < 12.4 g/dL (female)

reticulocyte fraction  $\geq 2\%$ 

serum haptoglobin < 0.5 g/L

schistocytes in pheripheral blood present

Horstkotte criteria [11]

no hemolysis

 $LDH\left( U/L\right)$ 

< 220

Haptoglobin (mg%

> 37

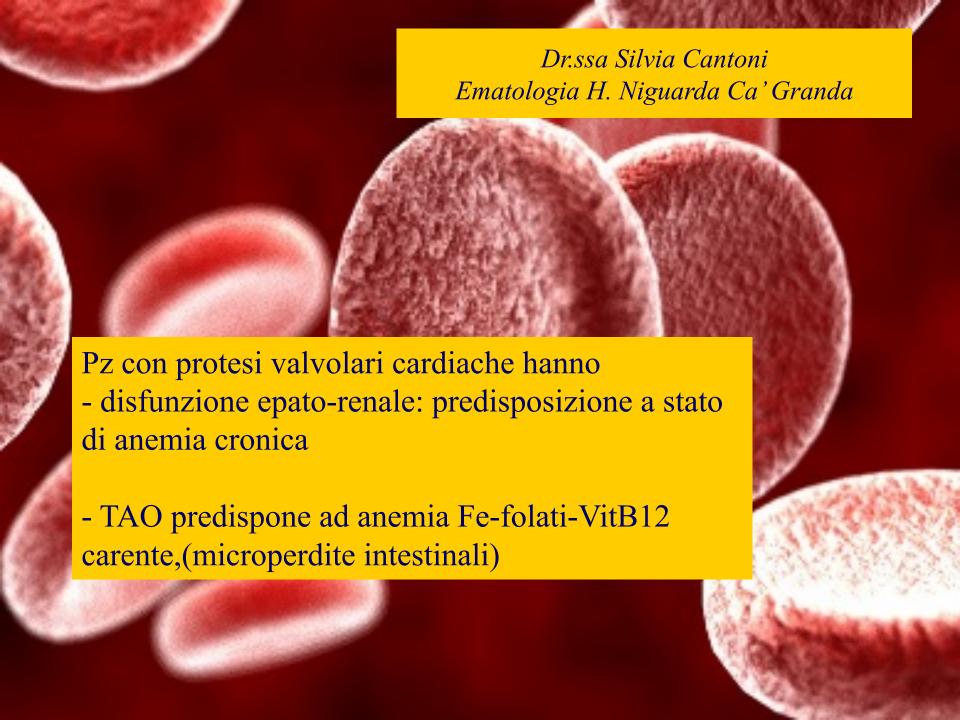
mild hemolysis 220–400 < 37

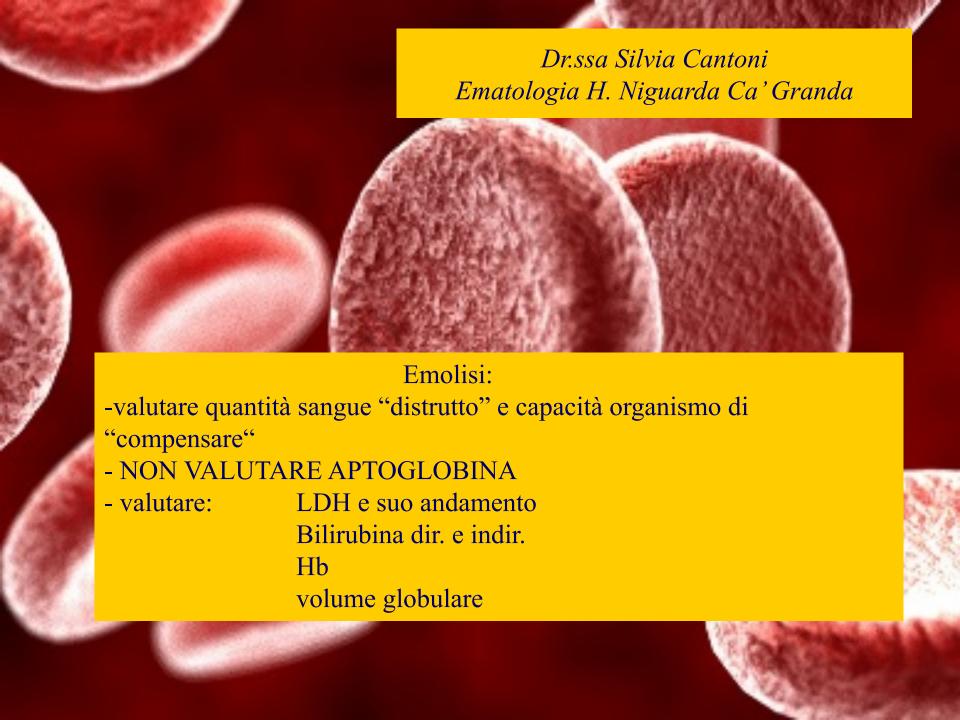
moderate hemolysis 400–800 < 10

severe compensate hemolysis > 800

decompensated hemolysis > 1000







### Dr.ssa Silvia Cantoni Ematologia H. Niguarda Ca' Granda

valutare: LDH e suo andamento

Bilirubina dir. e indir.

Hb

volume globulare

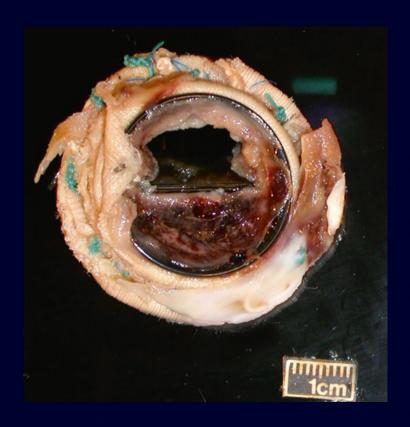
No aptoglobina LDH 1000 Bilirubina bas

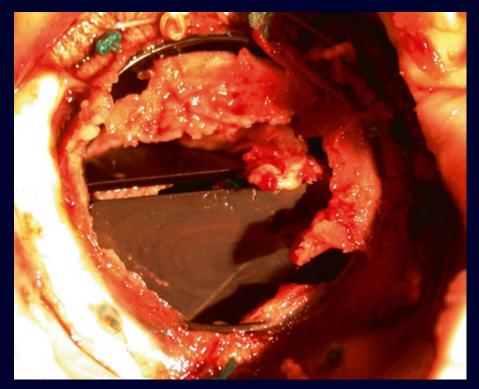
Tutti insieme!

NO schistociti allo striscio SI frammentociti coerenti con LDH elevati AST + IRC + LDH >2000 + RDW (37 pz 8 aa tutti M) No aptoglobina
LDH 1000
Bilirubina alta
Hb ridotta normocitica
(eritropoiesi inefficace)

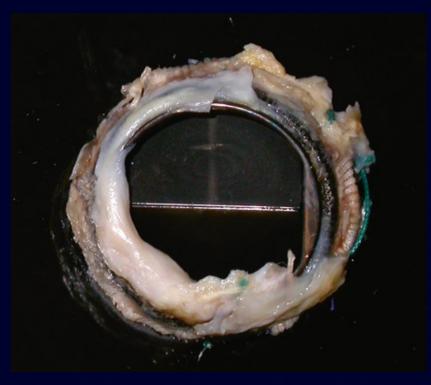


Le "malattie" delle protesi: panno + trombosi



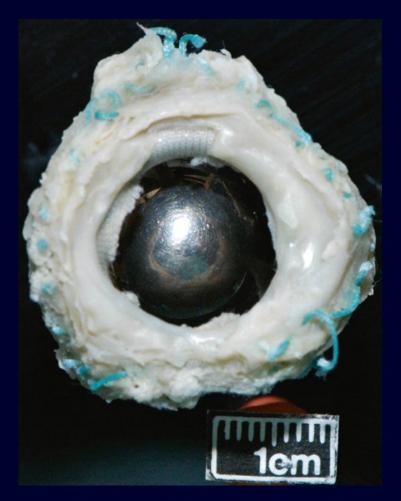


Le "malattie" delle protesi: panno





# Le "malattie" delle protesi: panno





# Panno- Trombosi

Ostruzione protesi meccaniche possono essere causate da panno, trombosi o entrambi

Differenziare panno da trombosi è difficile in quanto presentazioni cliniche similari

### Incidenza trombosi

0.3%-1.3%/Pz/anno in paesi sviluppati,

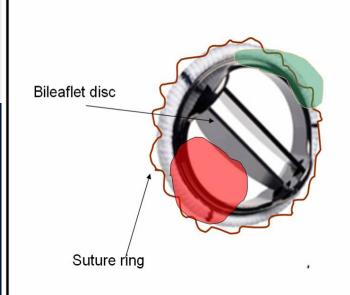
6.1% in paesi in sviluppo

> Cuore dx (tricuspide sino 20%) protesi mitralica 2-3 vv > protesi Ao

Sospetto Trombosi > se Tao inadeguata sintomi progrediscono rapidamente



4%/pz/anno se non TAO. 2%/pz/anno se antiaggregante. 1%/pz/anno se corretta TAO.



### **Pannus**

- ·Small, less dense mass, growing from
- Within suture line
- ·Along the plane of valve

#### **Thrombus**



- Large
- ·Less dense
- Mass project away from valve disc

	Panno	Trombosi
Cronologia	Minimo 12 mesi, solitamente 5 aa post-op	Sempre!! Se tardiva può associata a panno
Relazione TAO	Scarsa	Elevata
Localizzazione	Mtr>Ao	Mtr=Ao
Morfologia	Massa piccola coinvolge le linee di sutura, crescita centripeta cresce vicino ai dischi	Massa più grande  Movimento indipendente da  strutture fisse  crescita centrifuga  Proiettato in atrio sx
Echo density	Maggiore	Minore
Impatto sul gradiente	Ao>Mtr	Mtr>Ao
Impatto su area valvolare	Ao>Mtr	Mtr>Ao
Onset sintomi	Graduali	Rapidi

# Panno - Trombosi

# Forme non ostruttive

- Nel 10% dei casi reperto occasionale ECO
- Asintomatica
- Episodi embolici

### Forme ostruttive

- Raramente asintomatica
- EPA/scompenso
- Episodi embolici

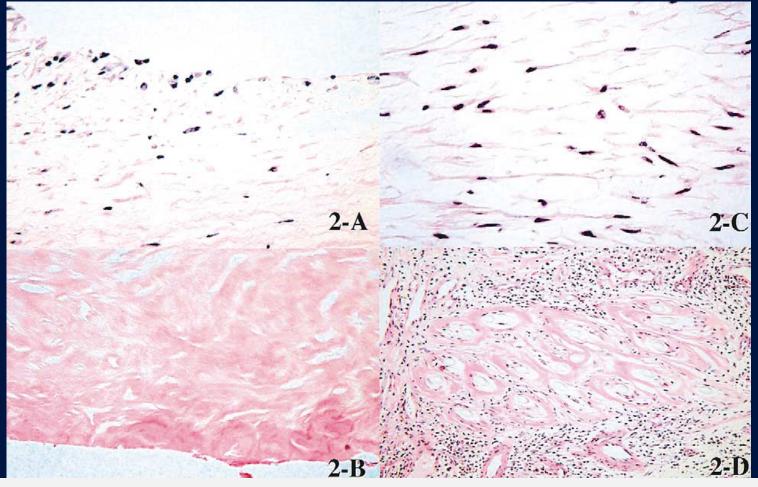
#### **Panno**

Histologic examination revealed a structure of fibroconnective tissue consistent with pannus formation.

# Pannus is an abnormal layer of fibrovascular tissue or granulation tissue.

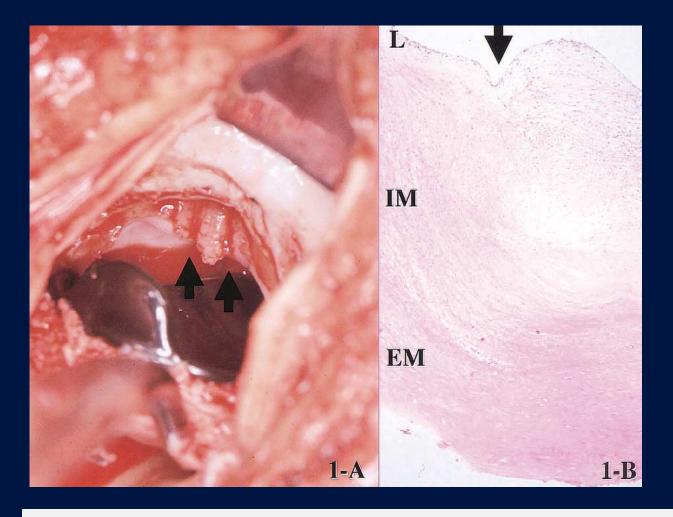
Granulation tissue è nuovo tessuto connettivo e fini vasi sanguigni che si forma sulla superficie di ogni ferita durante il processo di guarigione.

Granulation tissue cresce dalla base della ferita ed è in grado di riempire ferite di ogni superficie!



The specimens consisted of the endothelial cells in the lamina layer (Figure 2, *A*), *chronic inflammatory cell infiltration* (neutrophils, lymphocytes, plasma cells, macrophages, foreign body giant cells, and mast cells), pleomorphic spindle cells such as *myofibroblasts*, *and capillary vessels* (Figures 1, *B*; *2*, *A*, *C*, *and D*). *Neither malignant cells*, *calcifications*, *nor cavitations* were found in the pannus specimens. *Bacteria and fungi were also not found* in the pannus specimens with HE and Grocott's staining.





Histological analyses showed that pannus tissue was comprised of the following 3 distinctive layers and a lesion (Figure 1, *B*): a lumen layer (L) in the surface of pannus, an internal medial layer (IM), an external medial layer (EM), and a stump lesion located between the autologous annular tissue and the prosthetic valve

"The cause of pannus is generally recognized as a bioreaction to the prostheses."

### **Panno**OVERGROWTH

"overgrowth of periannular pannus may be associated with persistent inflammation caused by the prosthesis."

Fattori predisponenti

TAO inadeguata

Shear stress

Disegno protesi

Tecnica chirurgica

Diametro valvola se anulus piccolo

Turbolenze flusso ematico

Biocompatibilità (??)



# Obstruction of St Jude Medical valves in the aortic position: Histology and immunohistochemistry of pannus

, אוס (ס), אוס (ט), anu אבטע (ט).

TABLE 1. Results of immunohistochemistry

Stain	Target	Lumen	I. Media	E. Media	Stump
TGF-β	TGF-β	+	•	•	++
TGF-β-R1	TGF-β	+	+++	++	++
α-SMA	SMC, MFB	+	+++	++	+
Desmin	SMC	+	++	+	+
EMA	EpC, MFB, PC	•	++	+	+
CD34	EnC, FB	++	+	+	•
Factor VIII	EnC	++	•	•	•
CD68KP1	Mp, Mo	•	•	•	++
MMPs	MMPs	•	•	•	•

I. Media, Internal medial layer; E. Media, external medial layer;  $TGF-\beta$ , transforming growth factor-beta;  $TGF-\beta$ , matrix metalloproteinases;  $TGF-\beta$ , smooth muscle cell;  $TGF-\beta$ , myofibroblast;  $TGF-\beta$ , epithelial cell;  $TGF-\beta$ , endothelial cell;  $TGF-\beta$ , fibroblast;  $TGF-\beta$ , fibroblast;  $TGF-\beta$ , myofibroblast;  $TGF-\beta$ , epithelial cell;  $TGF-\beta$ , endothelial cell;  $TGF-\beta$ , fibroblast;  $TGF-\beta$ , fibroblast;  $TGF-\beta$ , myofibroblast;  $TGF-\beta$ , epithelial cell;  $TGF-\beta$ , endothelial cell;  $TGF-\beta$ , plasma cell;  $TGF-\beta$ , fibroblast;  $TGF-\beta$ , monocyte;  $TGF-\beta$ , represents a proportion of one third or less of positively stained cells;  $TGF-\beta$ , represents a proportion of two thirds; and  $TGF-\beta$ , no expression.

The Journal of Thoracic and Cardiovascular Surgery ● Volume 126, Number 2 401

#### Trombosi

Virchow's triad, *fattori predisponenti formazione trombo:* 

**Endotelial factors:** biocampatibilità protesi

interazione tra protesi e zone sutura

cicatrizzazione e endotelizzazione necessitano alcune settimane

**Haemodinamical factors:** protesi related e emodinamica del cuore in toto (low-flow)

Haemostatic factors: adeguatezza TAO

Perchè TAO deve essere ottimizzata?

Interazione componenti plasmatiche e superficie della protesi

Assorbimento di fibrinogeno da parte dei mateiali estranei che provoca adesione piastrinica

Turbolenze transprotesiche provocano aree di stasi

#### Trombosi

Virchow's triad, fattori predisponenti formazione trombo: Endotelial factors: biocampatibilità protesi interazione sede mitralica, Fattori di rischio: une settimane Haemod v-flow) doppia protesi, età >70aa, Haemost deficit contrattile del VSx storia di tromboembolia stato ipercoagulativo Le protesi biologiche non sono esenti da questa complicanza. -panno cresce lentamente, Inter rotesi difficile da visualizzare e distinguere da DS-......ogeno da parte dei mateiali estranei che provoca Asso adesione piastrinica

Turbolenze transprotesiche provocano aree di stasi

# Circulation. published online of Circulation.

Circulation. published online March 3, 2014;

Practice (

Even in clinical trials, the time in therapeutic range for patients on VKA varies from only 60% to 70%.

However, embolic events do occur even in patients who are in the therapeutic range at every testing interval.

However, the prosthetic valve should be considered the source of thromboembolism even if echocardiographic findings are unchanged

**Anticoagulation for Prosthetic Mechanical valves** 

AVR and no risk factor Long-term warfarin, INR 2.0-

Bileaflet valves Long-term warfarin, INR 2.5–3.5

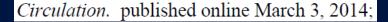
Monoleaflet or ball-cage valves Long-term warfarin, INR 2.5-3.5

AVR + risk factor\* Long-term warfarin, INR 2.5–3.5

MVR Long-term warfarin, INR 2.5–3.5

Solo SVAO no RF

Whitlock RP, Sun JC, Fremes SE, et al. Antithrombotic and thrombolytic therapy for valvular disease: antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians Evidence- Based Clinical Practice Guidelines, Chest, 2012;141:e576S-e600S.





2014 AHA/ACC Guideline for the Management of Patients With Valvular Heart Disease: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

#### Class I LoE: A

Aspirin 75 mg to 100 mg daily is recommended in addition to anticoagulation with a VKA in patients with a mechanical valve prosthesis

TAO rischio tromboembolico 1-2%

TAO ASA embolie maggiori da 1.9 a 8.5%

stroke da 4.2 a 1.3% mortalità da 7.4 a 2.8%

sanguinamenti maggiori da 6.6 a 8.5%



NO Non indicato se rischio sanguinamento elevato o intolleranza

#### sı Pz con pregressi episodi trombo-embolici, polivasculopatia o stato ipercoagulante

Whitlock RP, Sun JC, Fremes SE, et al. Antithrombotic and thrombolytic therapy for valvular disease: antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians Evidence- Based Clinical Practice Guidelines. Chest. 2012;141:e576S-e600S.

Little SH, Massel DR. Antiplatelet and anticoagulation for patients with prosthetic heart valves. Cochrane Database Syst Rev. 2003;CD003464.

Laffort P, Roudaut R, Roques X, et al. Early and long-term (one-year) effects of the association of aspirin and oral anticoagulant on thrombi and morbidity after replacement of the mitral valve with the St. Jude medical prosthesis: a clinical and transesophageal echocardiographic study. JAm Coll Cardiol. 2000:35:739-46.

Pengo V, Palareti G, Cucchini U, et al. Low-intensity oral anticoagulant plus low-dose aspirin during the first six months versus standard intensity oral anticoagulant therapy after mechanical heart valve replacement: a pilot study of low-intensity warfarin and aspirin in cardiac prostheses (LIWACAP). Clin Appl Thromb Hemost. 2007;13:241-8.

Altman R, Boullon F, Rouvier J, et al. Aspirin and prophylaxis of thromboembolic complications in patients with substitute heart . J Thorac Cardiovasc Surg. 1976;72:127-9.



Circulation. published online March 3, 2014

Pz con protesi meccaniche

2014 AHA/ACC Guideline for the Manager Long in the Long valvular Heart Disease: A Report of the American College College Guidelines

Class I LoE: C

Continuation of TAO a valore terapeutico se sottoposti a procedure invasive minori: (estrazioni dentarie, cataratta, dermatologia)

Class I LoE: C

Bridging TAO con eparina ev o LMWH sc se INR basso preop di interventi invasivi in Pz

- 1) SVAo e RF tromboembolici,
- 2) SVAo older-generation, or
- 3) SVM.

Class I LoE: C

Temporary interruption of VKA anticoagulation, senza bridging TAO preop di interventi invasivi in Pz SVAo senza RF tromboembolici.



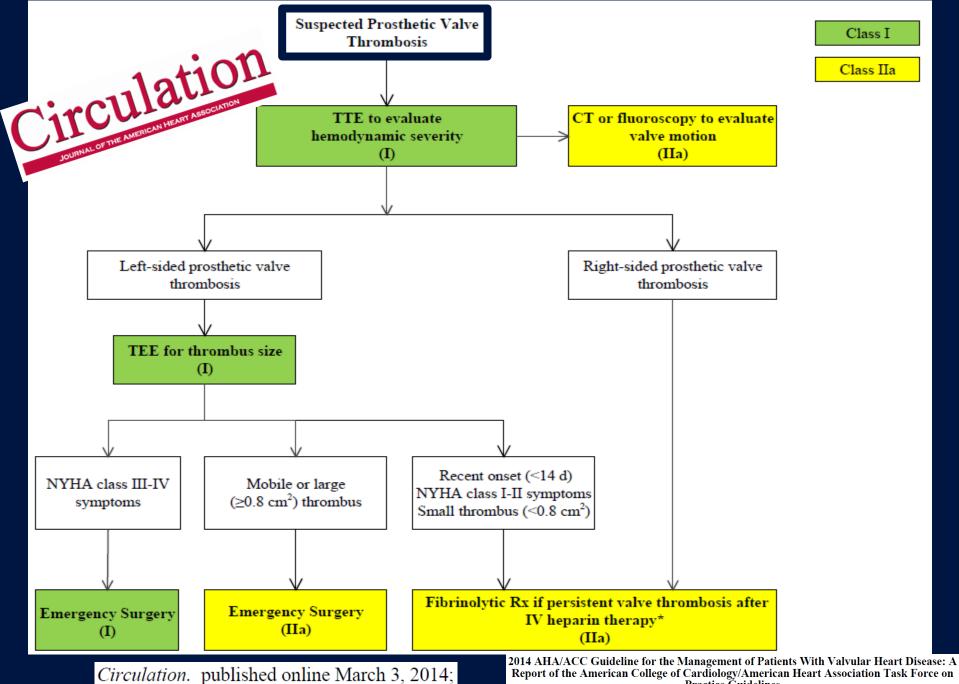
Le protesi meccaniche endocardite, distacchi tardivi, trombosi, pannus.



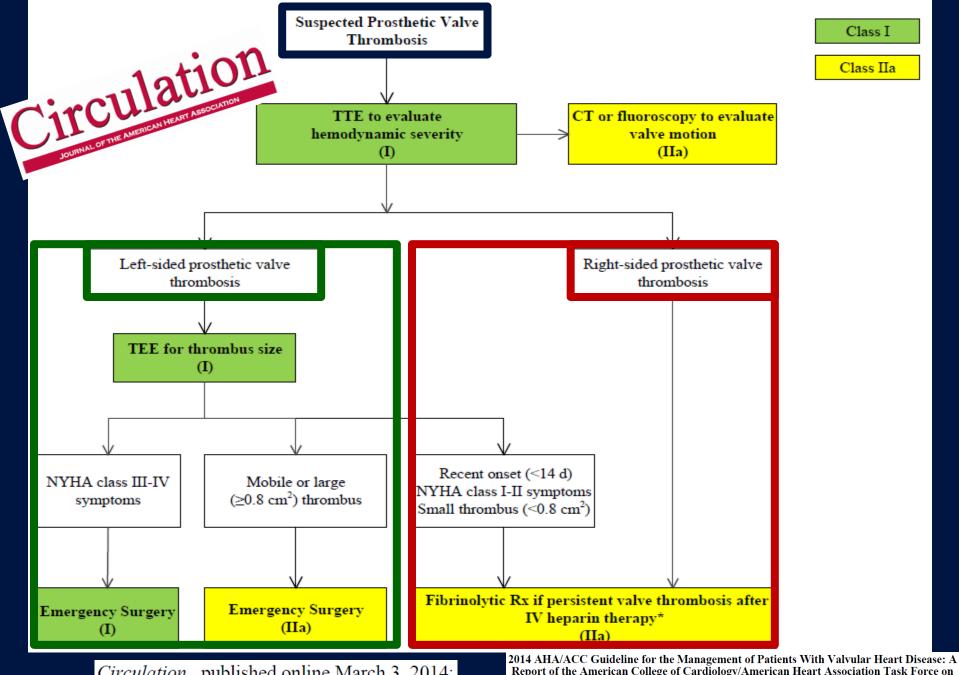






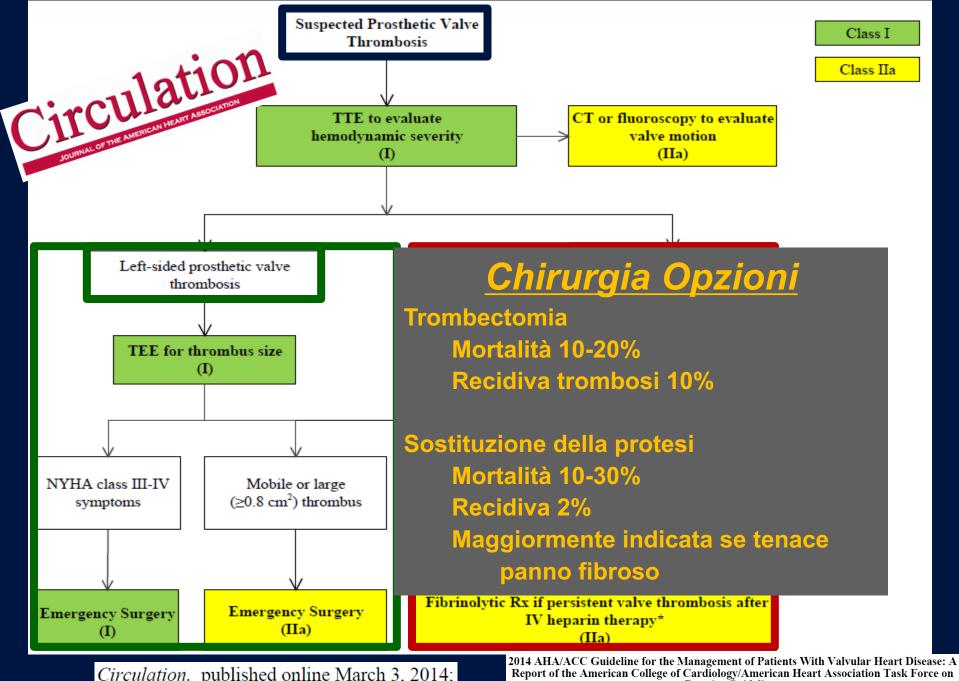


Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines



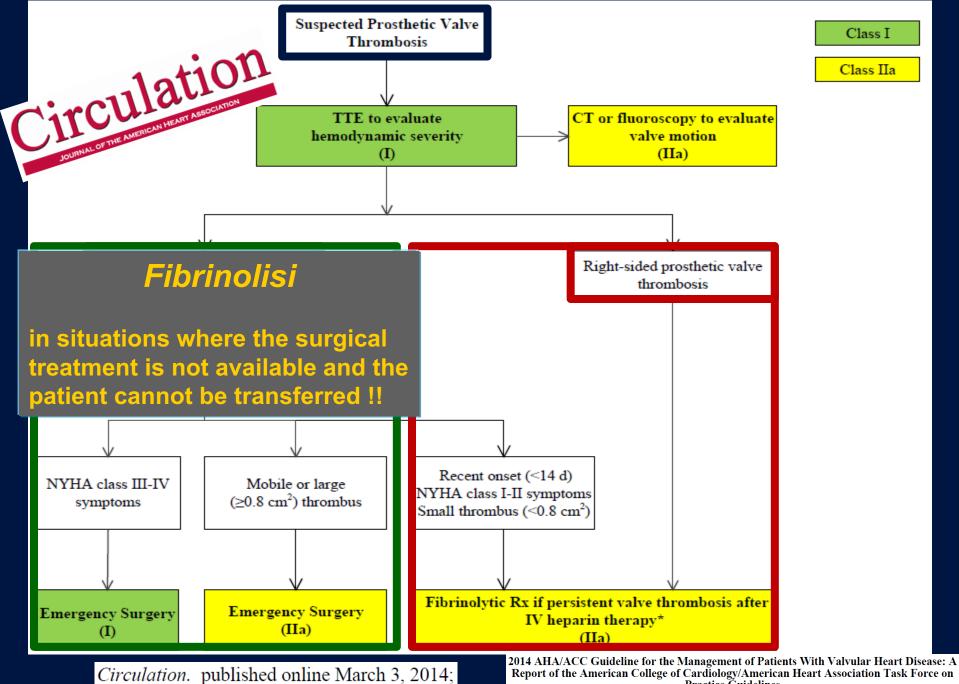
Circulation. published online March 3, 2014;

Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

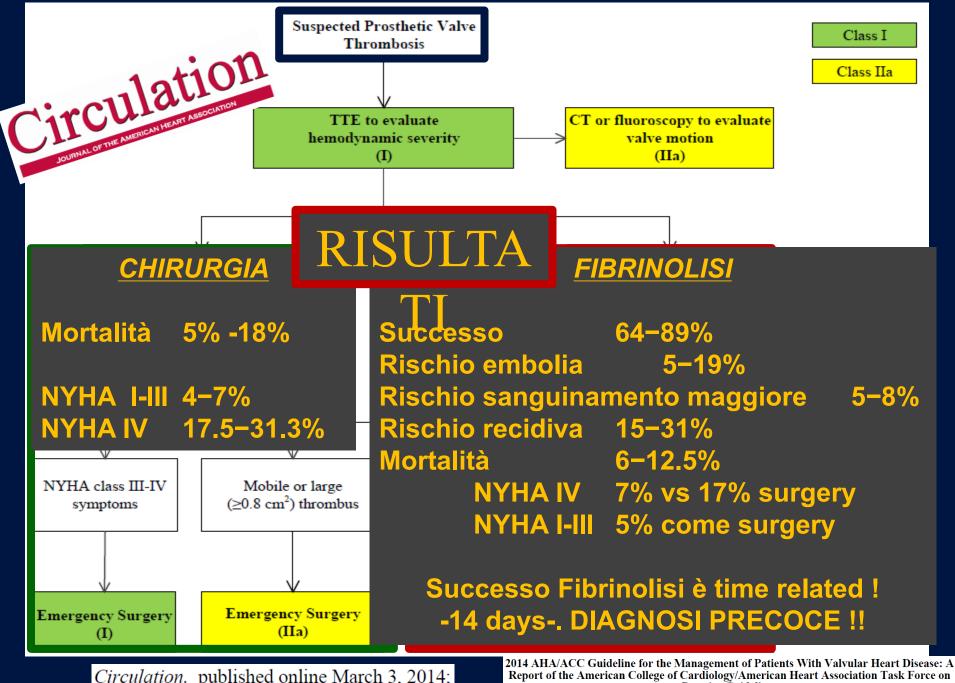


Circulation. published online March 3, 2014;

Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines



Practice Guidelines



Sostituzione valvolare non è una cura bensì un cambiamento di condizione:



Più anni da primo intervento

Deteriormanto funzionale repentino

Scompenso importante (trombosi-endocardite)

Scompenso recente

> % Urgente

# L'intervento non è a rischio zero

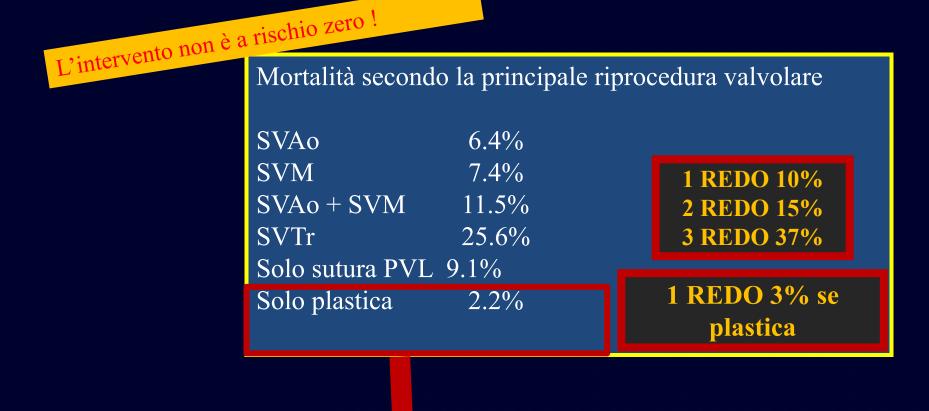
Plastica fallita/altra valvola nativa 3.0%

Disfunzione protesica/PVL

10.6%

Endocardite o trombosi protesi

29.4%



Rischio direttamente proporzionale ad invasività



# KNOW YOUR ENEMY





#### Considerazioni

- Rimossa una protesi è difficile reimpiantarne una di stessa misura (spesso una misura inferiore)

- Su anulus aortico è possibile ampliare anello per inserire protesi uguale o più grande, su mitrale NO!

- Se PVL in biologica di 8 anni che funziona correttamente cosa faccio?

Suturo distacco o cambio protesi?



L'intervento non è a rischio zero!

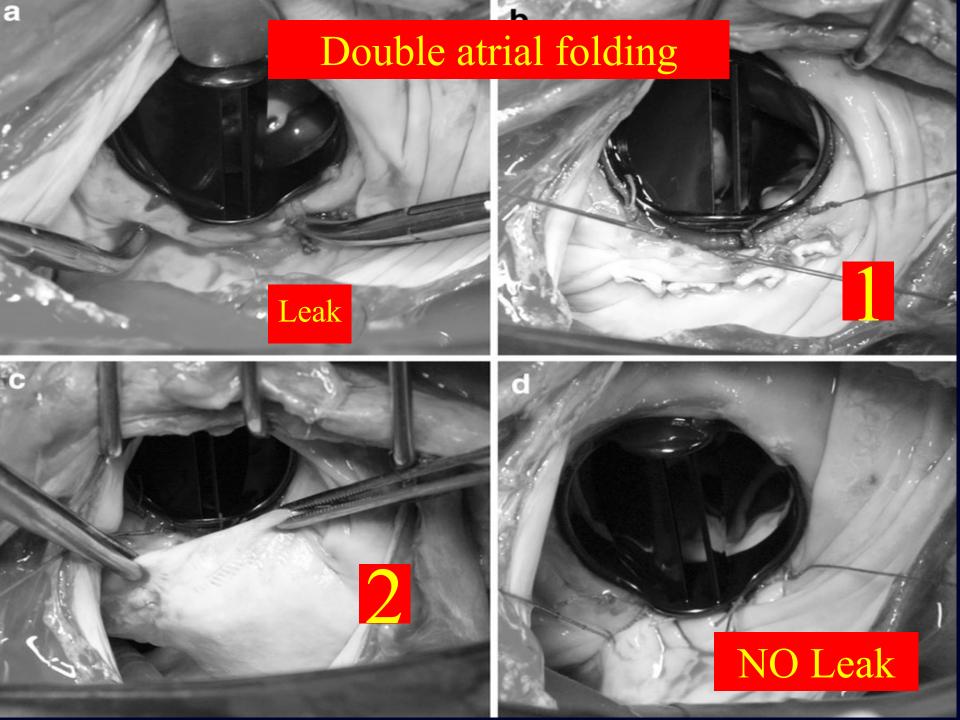
Come stà il Paziente? Emolizza? È stabile o peggiora? (Pz e leak!) Ma soprattutto.....

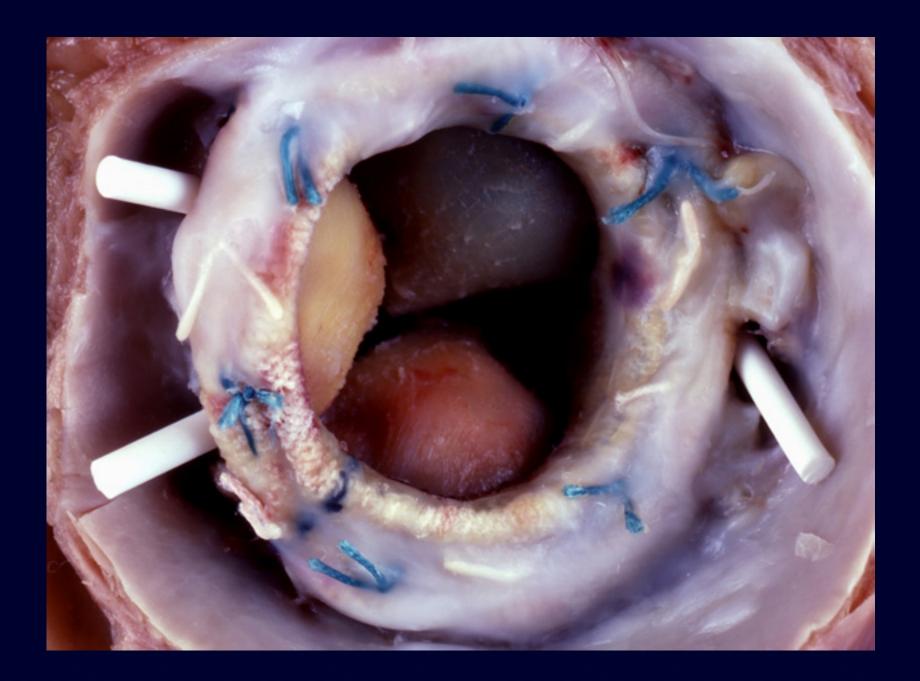


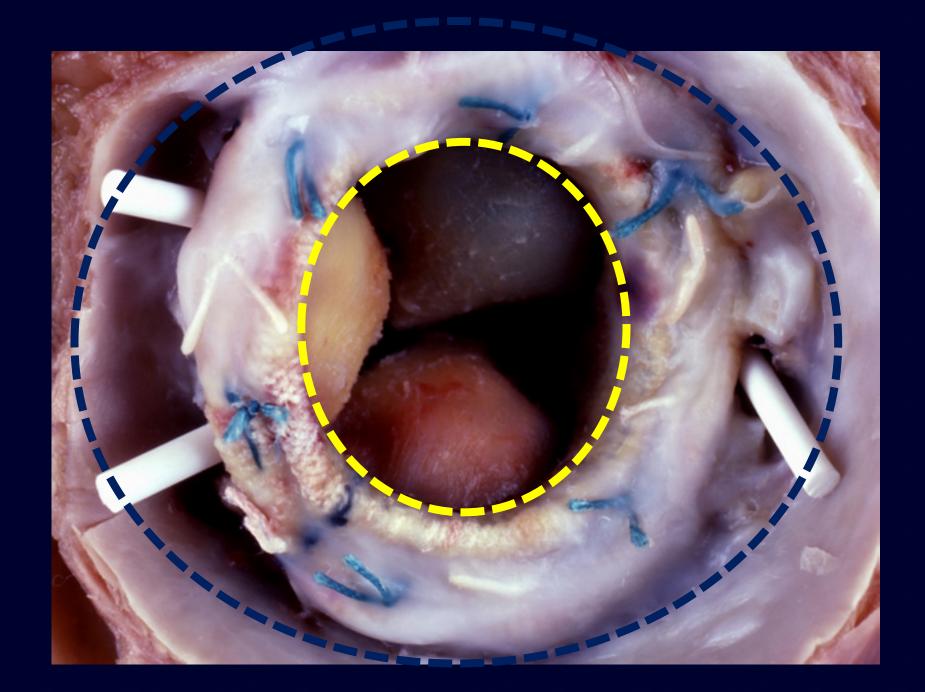
## CAUTION KEEP AWAY

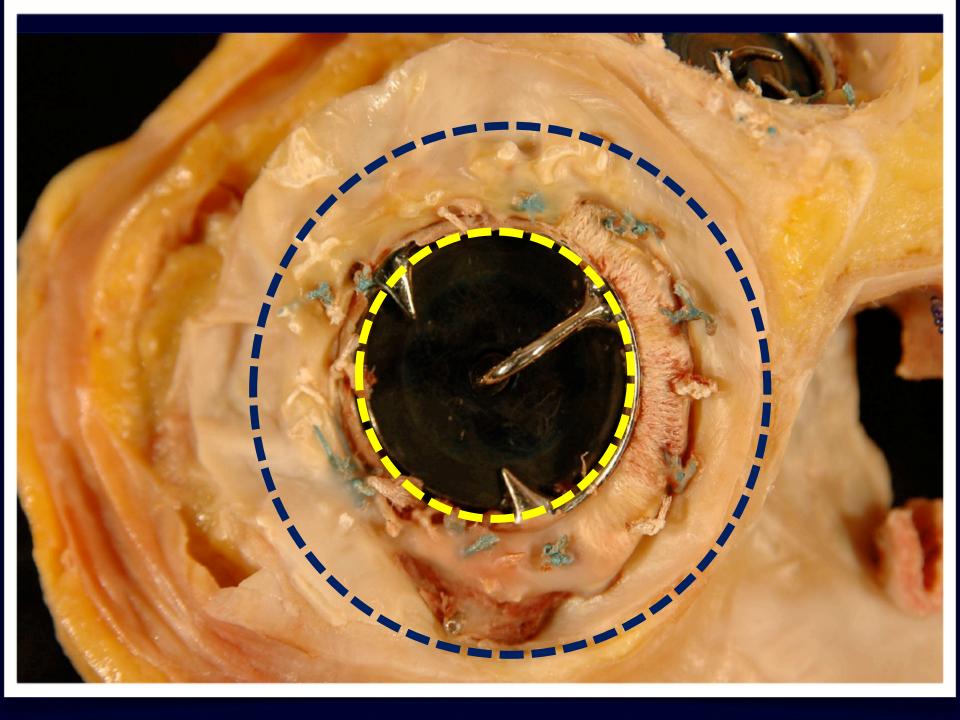
Calcificazioni anulari	•
Fragilità tessuti	•
Patch pericardio	
Punti di rinforzo	

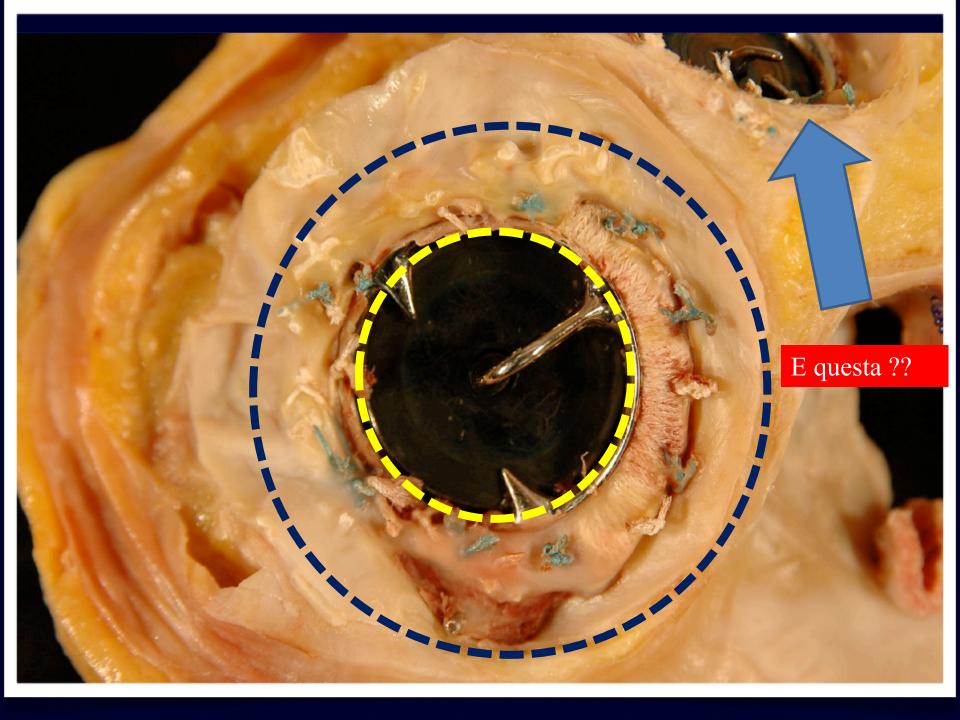


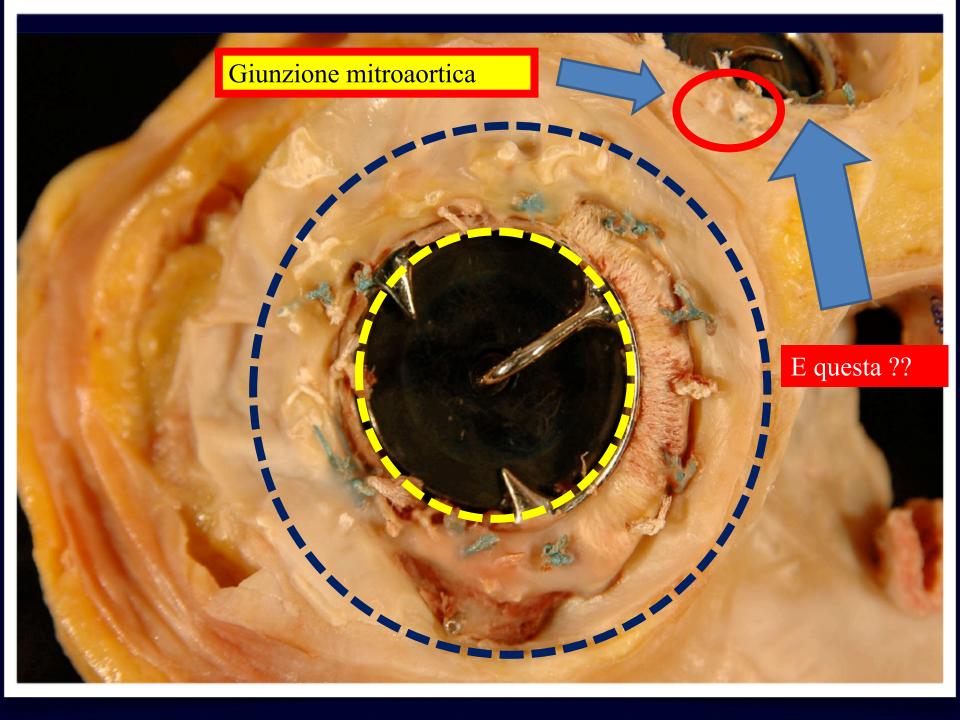


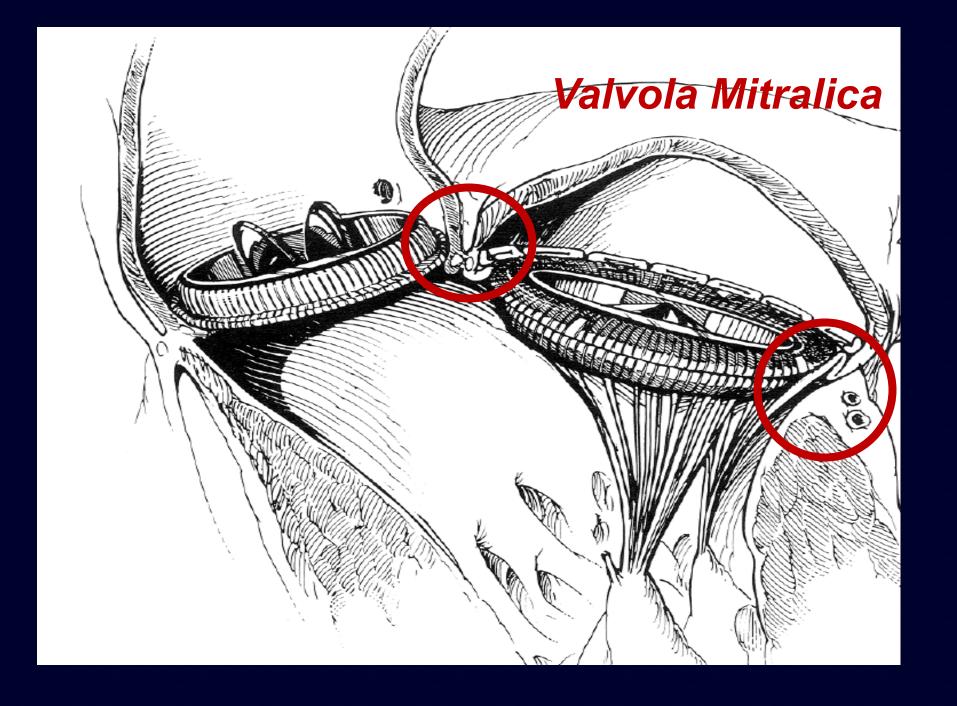


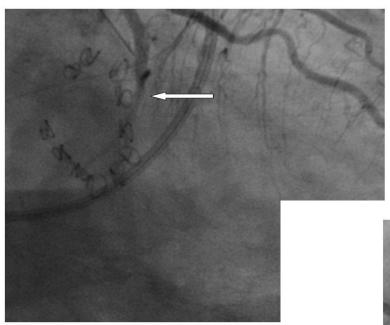




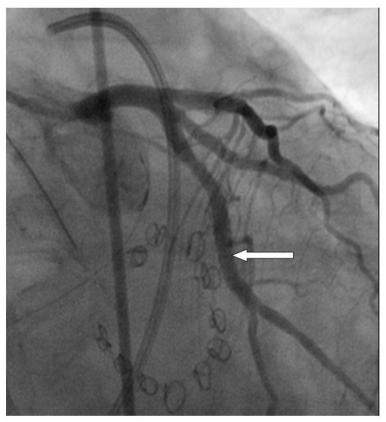




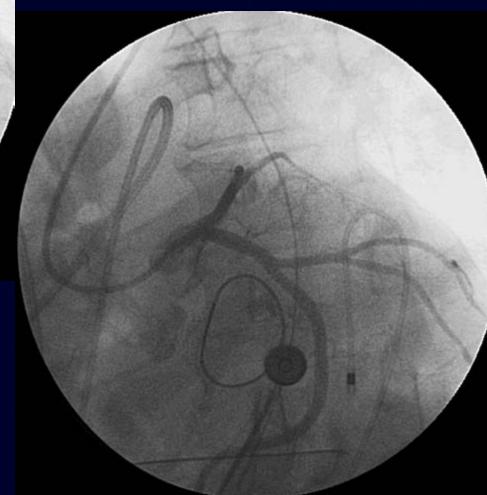


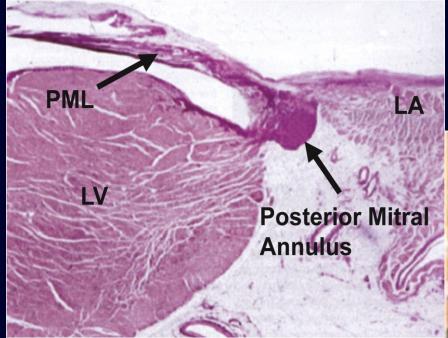


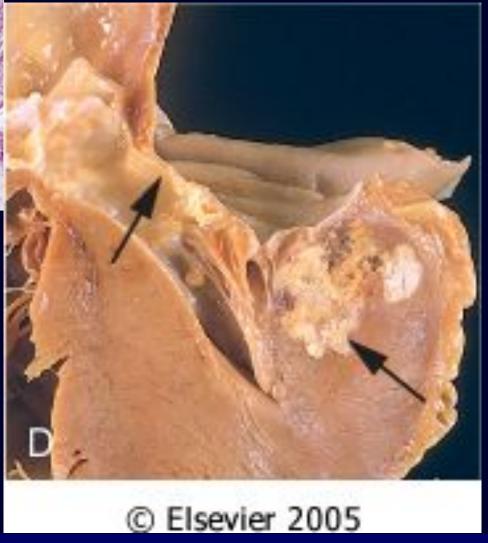
Ischemia



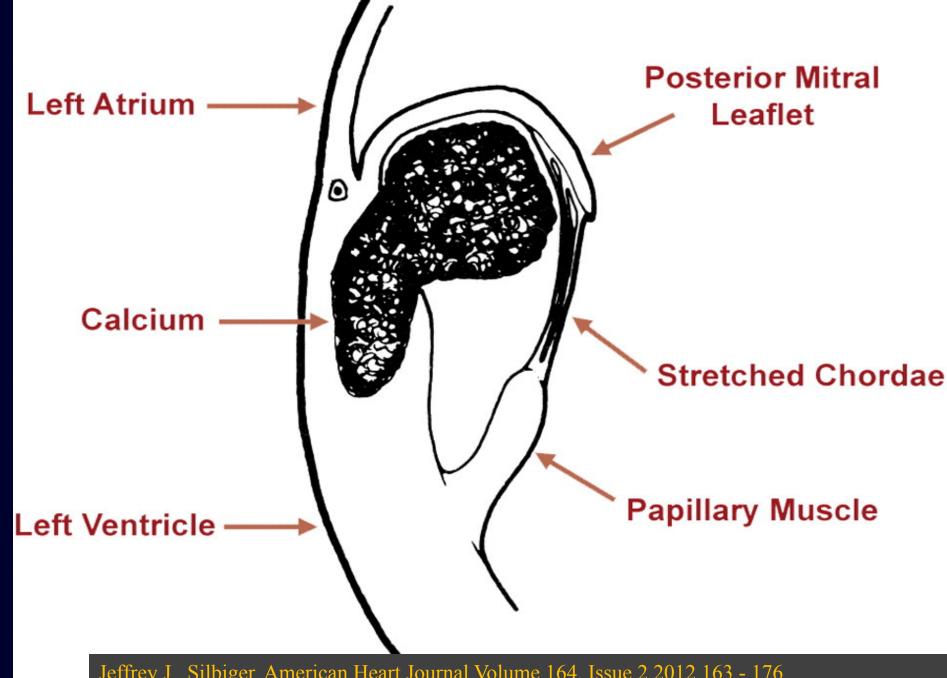








Jeffrey J. Silbiger. American Heart Journal Volume 164, Issue 2 2012 163 - 176





## Rottura di cuore **BI-LOBED** ANEURYSM В LAD OM, ICT An OM<sub>T</sub> C





