PLACE

PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE

ROMA
Centro Congressi
di Confindustria
Auditorium
della Tecnica

9ª Edizione

30 Settembre
1 Ottobre
2022

Topics in cardiochirurgia e dintorni

DEGENERAZIONE DI PROTESI BIOLOGICHE

Greco C.A. , Lecce

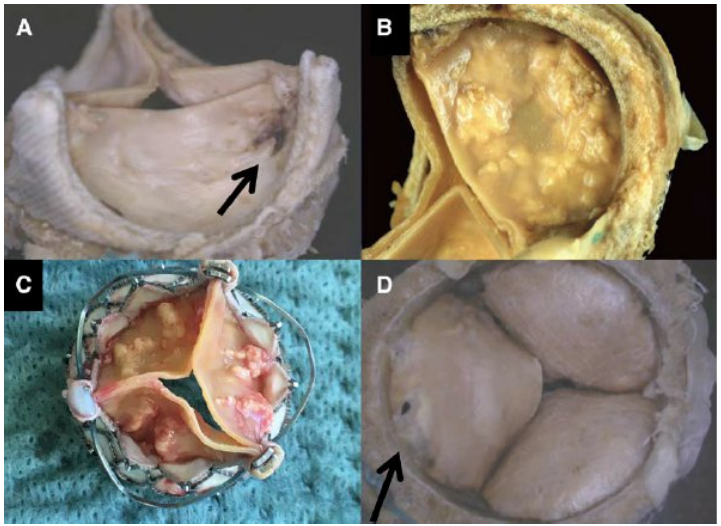
Standardized definitions of structural deterioration and valve failure in assessing long-term durability of transcatheter and surgical aortic bioprosthetic valves: a consensus statement from the European Association of Percutaneous Cardiovascular Interventions (EAPCI) endorsed by the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS)

European Heart Journal (2017) **38**, 3382–3390

PLACE
PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE

Standardized Definition of Structural Valve Degeneration for Surgical and Transcatheter Bioprosthetic Aortic Valves

Danny Dvir, MD*
Thierry Bourguignon, MD*
et al



A, Carpentier-Edwards Perimount valve: leaflet tear. **B**, Carpentier-Edwards Magna Ease valve: leaflet calcification. **C**, Engager THV (Medtronic): leaflet restriction and calcification. **D**, Carpentier-Edwards Perimount valve: leaflet tear (ventricular side).

SVD DEFINITION

SVD is an acquired intrinsic bioprosthetic valve abnormality defined as deterioration of the leaflets or supporting structures resulting in thickening, calcification, tearing, or disruption of the prosthetic valve materials with eventual associated valve hemodynamic dysfunction, manifested as stenosis or regurgitation. The pre-

Circulation. 2018;137:388–399.

Aortic Bioprosthetic Valve Durability

Incidence, Mechanisms, Predictors, and Management of Surgical and Transcatheter Valve Degeneration

Tania Rodriguez-Gabella, MD, Pierre Voisine, MD, Rishi Puri, MBBS, PhD, Philippe Pibarot, DVM, PhD, Josep Rodés-Cabau, MD

MECHANISMS OF SVD

SVD is a multifactorial process with 2 main consequences: calcification and leaflet degradation leading to valve stenosis or leaflet tear with ensuing valve regurgitation. Bovine pericardial valves have a

Aortic Bioprosthetic Valve Durability

Incidence, Mechanisms, Predictors, and Management of Surgical and Transcatheter Valve Degeneration

Tania Rodriguez-Gabella, MD, Pierre Voisine, MD, Rishi Puri, MBBS, PhD, Philippe Pibarot, DVM, PhD, Josep Rodés-Cabau, MD

MECHANISMS OF SVD

Several mechanisms have been identified in the pathogenesis of SVD. Bioprosthetic leaflet tissue is fixed in glutaraldehyde to cross-link and mask antigens, avoiding immune system rejection of the xenograft and making the bioprosthesis "immunologically inert." Free aldehyde groups resulting from this treatment, along with phospholipids and calcium ions in the circulation, result in a passive process of calcification (28). Hypercalcemia,

CURRENT OPINION

Incidence, risk factors, clinical impact, and management of bioprosthesis structural valve degeneration

Nancy Côté, Philippe Pibarot, and Marie-Annick Clavel

KEY POINTS

- SVD is the major cause of aortic and mitral valve bioprosthesis failure and is increasing over time.
- SVD is an active process characterized by leaflet mineralization, fibrosis caused by mechanical stress, lipid-mediated inflammation, and immune rejection processes.

Curr Opin Cardiol 2017, 32:123–129

PLACE 9ª Edizione

PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE

- **Bioprosthetic heart valves**
  - **Human tissue valves**
    - **Allografts** — Cadaveric aortic valve
    - **Autografts** — Ross procedure
  - **Xenografts**
    - **Stented**
      - **Porcine** — CE SAV, Medtronic Hancock II, Medtronic Mosaic, Abbott Biocor, Abbott Epic
      - **Pericardial** — CE PERIMOUNT line, Sorin Mitroflow PRT, Abbott Trifecta, Avalus Medtronic
    - **Stentless**
      - **Porcine** — Abbott Toronto SPV, Medtronic Freestyle
      - **Pericardial** — Sorin Solo Smart
      - **Sutureless** — Edwards INTUITY Elite, Sorin Perceval S
  - **Transcatheter heart valves**
    - **First generation iterations** — Edwards SAPIEN, Edwards SAPIEN XT, Medtronic CoreValve
    - **Newer generation Devices** — Edwards SAPIEN 3, Medtronic CoreValve Evolut R, Medtronic CoreValve Evolut Pro, Boston Scientific Lotus, Abbott Portico, Boston Scientific ACURATE neo, Edwards Centera, JenaValve

Stented porcine valves


Carpentier-Edwards SAV™


Medtronic Hancock® II


Medtronic Mosaic®


Abbott Epic™

Stentless porcine valves


Abbott Toronto SPV™


Medtronic Freestyle®

Sutureless valves


Edwards INTUITY Elite®


Sorin Perceval S®

Stented pericardial valves


Carpentier-Edwards Perimount Magna Ease™


Sorin Mitroflow™


Abbott Trifecta™


Avalus Medtronic™

Stentless pericardial valves


Sorin Solo Smart™

Transcatheter heart valves


Edwards SAPIEN® 3


Medtronic CoreValve® Evolut R®


Medtronic CoreValve® Evolut PRO®


Boston Scientific Lotus™


Abbott Portico ™


Boston Scientific ACURATE neo ™

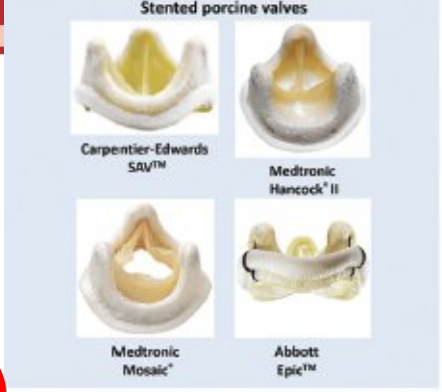

Edwards Centera®

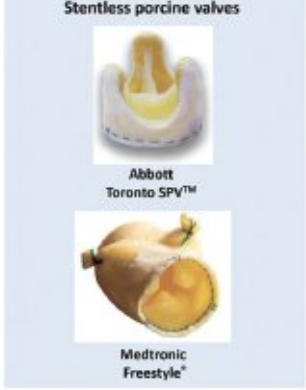

JenaValve™

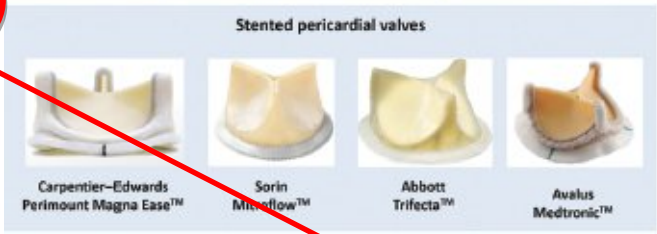
PLACE 9ª Edizione

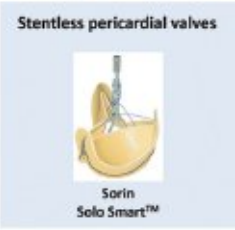
PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE

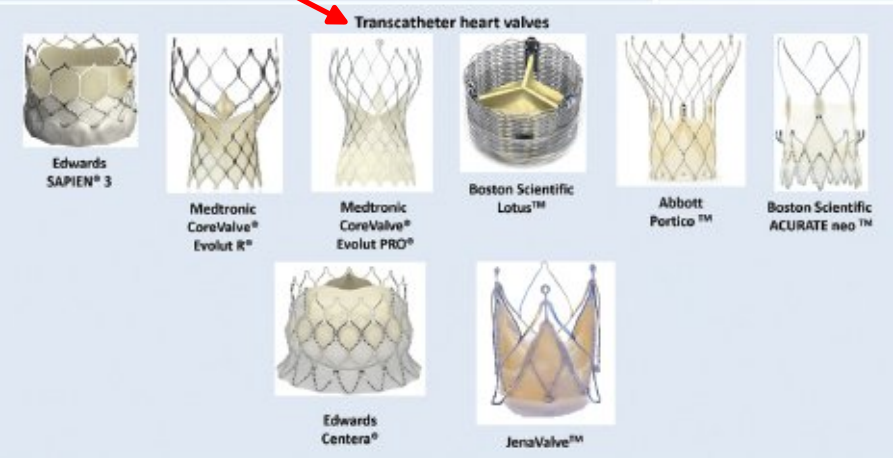
- **Bioprosthetic heart valves**
  - **Human tissue valves**
    - **Allografts**
      - Cadaveric aortic valve
    - **Autografts**
      - Ross procedure
  - **Xenografts**
    - **Stented**
      - **Porcine**
        - CE SAV
        - Medtronic Hancock II
        - Medtronic Mosaic
        - Abbott Biocor
        - Abbott Epic
      - **Pericardial**
        - CE PERIMOUNT line
        - Sorin Mitroflow PRT
        - Abbott Trifecta
        - Avalus Medtronic
    - **Stentless**
      - **Porcine**
        - Abbott Toronto SPV
        - Medtronic Freestyle
      - **Pericardial**
        - Sorin Solo Smart
    - **Sutureless**
      - **Sutureless**
        - Edwards INTUITY Elite
        - Sorin Perceval S
  - **Transcatheter heart valves**
    - **First generation iterations**
      - Edwards SAPIEN
      - Edwards SAPIEN XT
      - Medtronic CoreValve
    - **Newer generation Devices**
      - Edwards SAPIEN 3
      - Medtronic CoreValve Evolut R
      - Medtronic CoreValve Evolut Pro
      - Boston Scientific Lotus
      - Abbott Portico
      - Boston Scientific ACURITY neo
      - Edwards Centera
      - JenaValve











CURRENT OPINION

Incidence, risk factors, clinical impact, and management of bioprosthesis structural valve degeneration

Curr Opin Cardiol 2017, 32:123–129

Nancy Côté, Philippe Pibarot, and Marie-Annick Clavel

Risk factors previously found to be associated with bioprosthetic SVD are younger age, mitral valve position, end-stage renal disease, higher calcium-phosphorus product, hyperparathyroidism, hypertension,

thetic valve leaflets [6]. Prosthesis-related factors include small bioprosthesis size and PPM [13,51]. These factors may increase mechanical stress on the leaflets. Several studies suggest that the new generation of bioprostheses are more durable compared

(1) There should be clear distinction between SVD (the principal aetiology) and BVF (the clinical correlate).

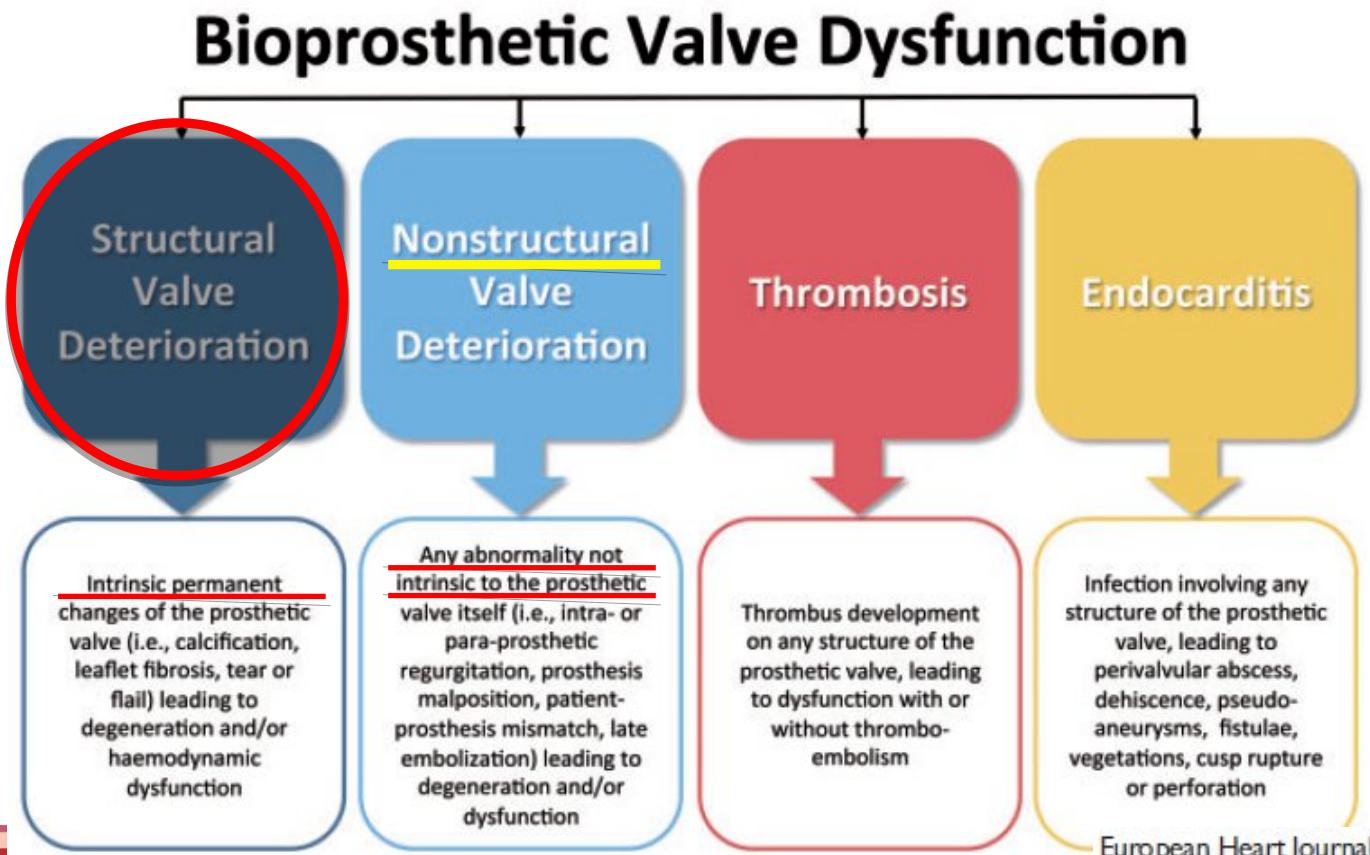
(2) Structural valve deterioration causes irreversible dysfunction whereas other pathological causes of bioprosthetic valve dysfunction (i.e. thrombosis, endocarditis) are potentially reversible and should be identified and categorized separately. However, the thrombotic or endocarditic process qualifies as a cause of BVF if it leads to lasting or permanent bioprosthetic valve dysfunction.

(3) Non-structural valve dysfunction (i.e. intra-prosthetic or paravalvular regurgitation, prosthesis malposition, patient-prosthesis mismatch, late embolization) may occur early after TAVI as a result of technical issues. Non-structural valve dysfunction resulting in valve-related death, reintervention, and haemodynamic dysfunction [i.e. severe new or worsening (>2+/4+) paravalvular aortic regurgitation] qualifies as a cause of BVF.

(4) Echocardiography is the principal imaging modality for the detection of SVD and the best and most accessible way to detect serial changes in valve function. Transprosthetic gradients should be determined in at least two consecutive measurements to account for detection bias and minimize inconsistencies related to the different types of bioprosthesis implanted. After TAVI and SAVR,

BVF: BIOPROSTHETIC VALVE FAILURE

Causes of bioprosthetic valve dysfunction.



European Heart Journal (2017) **38**, 3382–3390

PLACE
PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE

Standardized Definition of Structural Valve Degeneration for Surgical and Transcatheter Bioprosthetic Aortic Valves

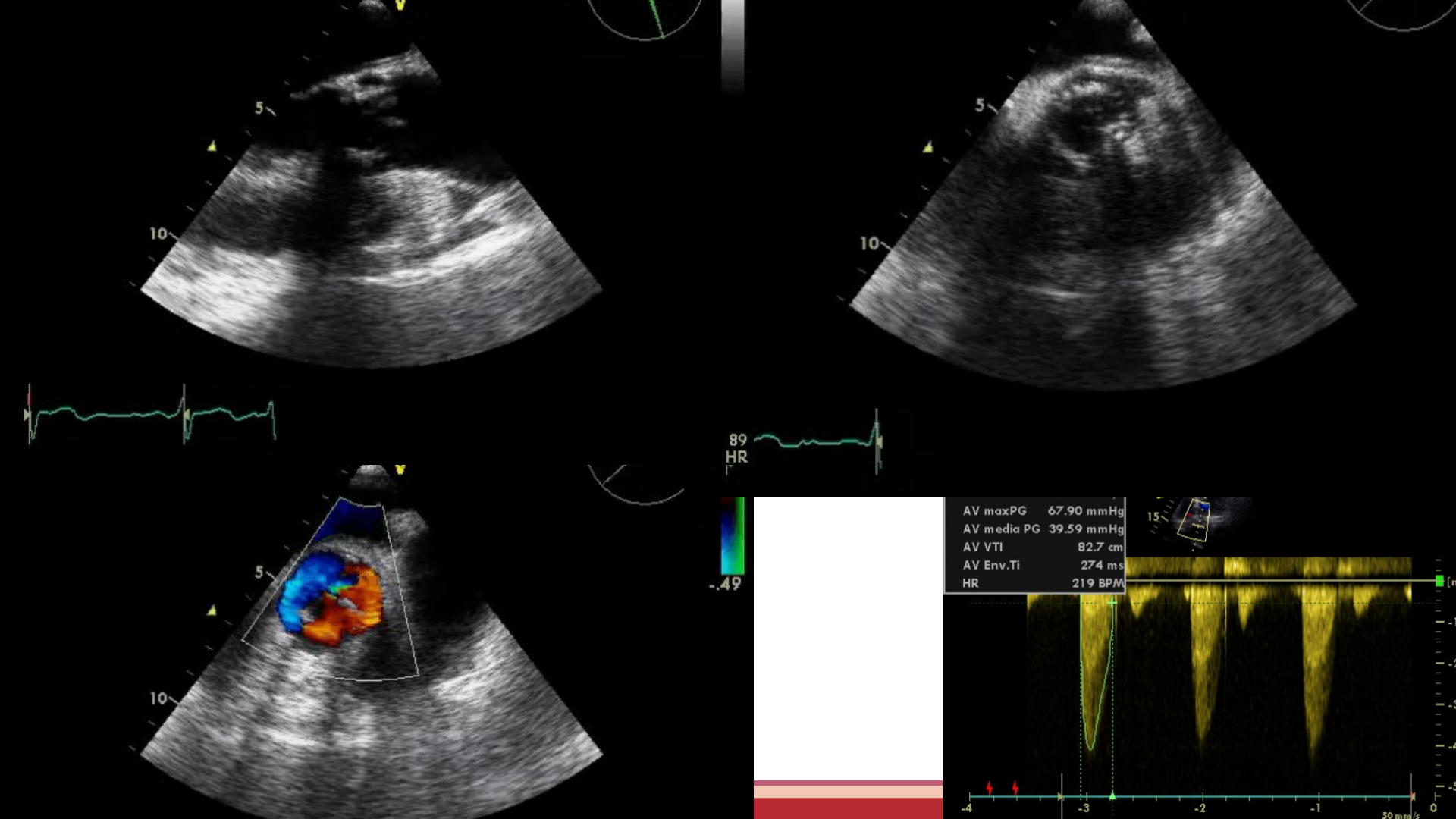
Danny Dvir, MD*
Thierry Bourguignon, MD*
et al

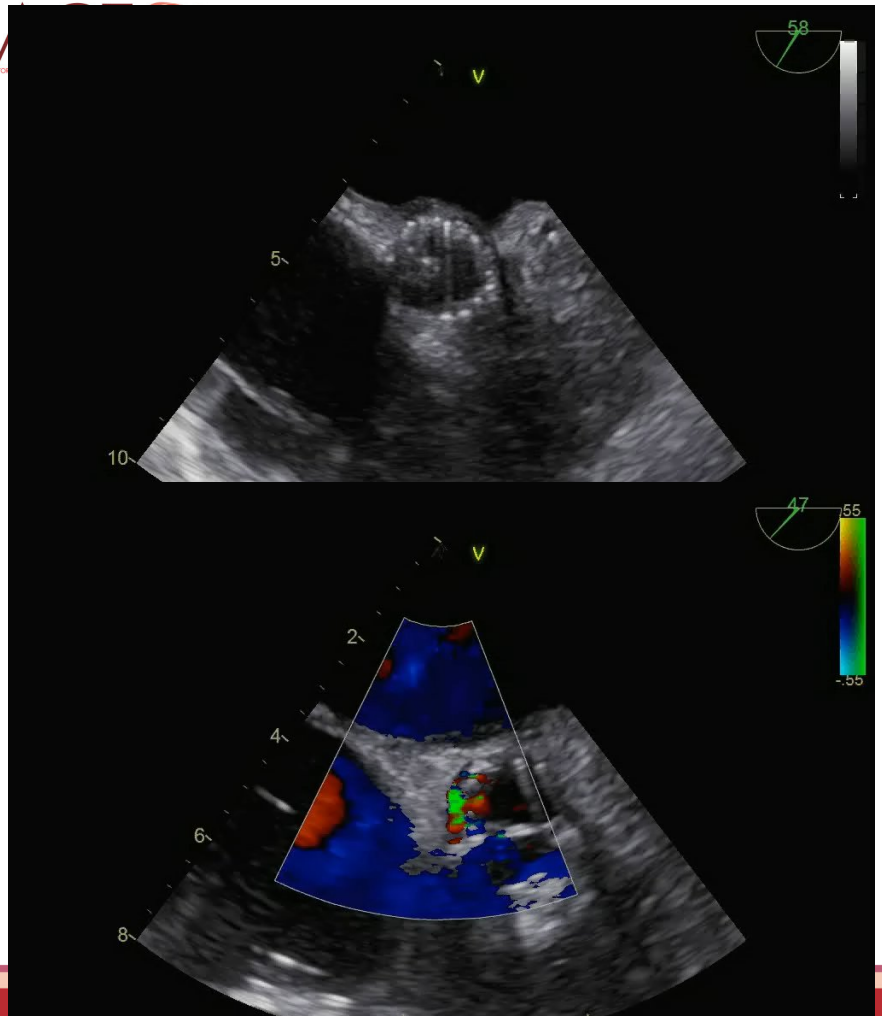
SVD DEFINITION

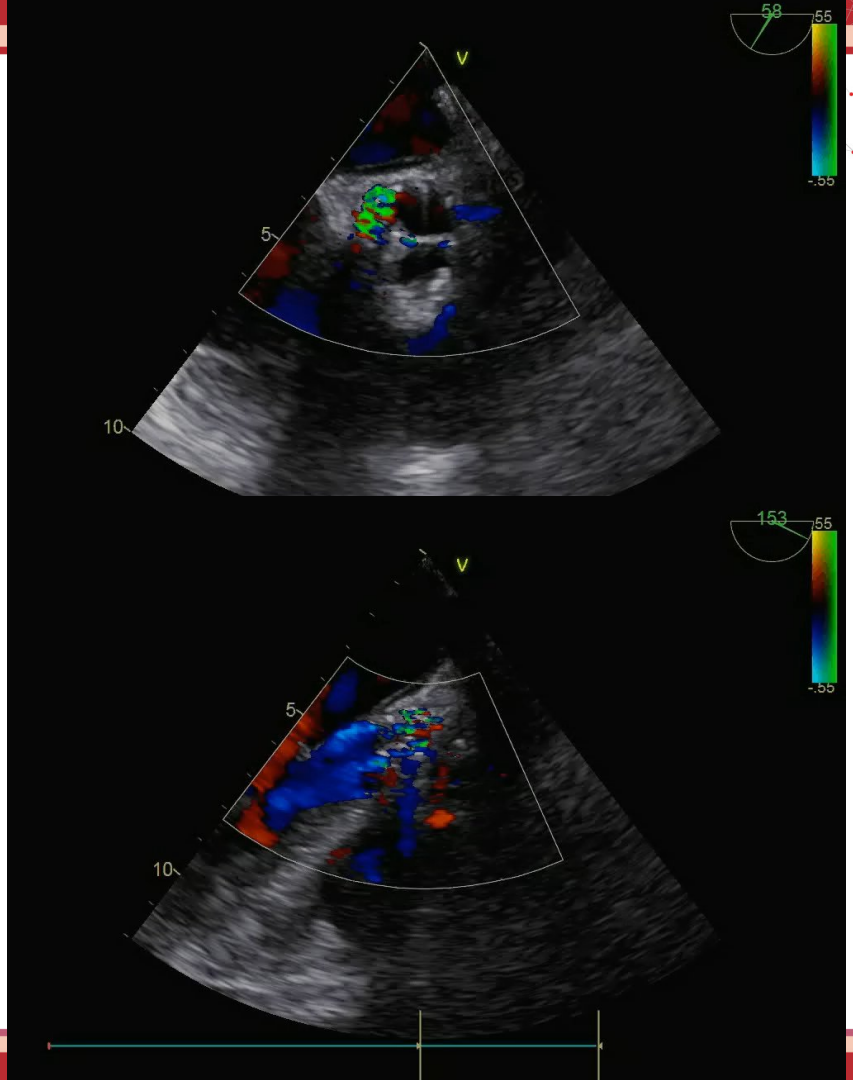
Other clinical valve abnormalities that are not because of deterioration of the valve tissue are not included in the definition of SVD. These include patient-prosthesis mismatch, device malposition, paravalvular regurgitation, and abnormal frame expansion, although these may be associated with early SVD. In particular, patient-

up. Prosthetic valve thrombosis and infective endocarditis also are not included in the definition of SVD, but

Circulation. 2018;137:388–399.


5
10
89
HR
-.49
AV maxPG 67.90 mmHg
AV media PG 39.59 mmHg
AV VTI 82.7 cm
AV Env.Ti 274 ms
HR 219 BPM
-4
-3
-2
-1
0
50 mm/s


58
V
5
10
47
55
-55
V
2
4
6
8


58
55
-55
V
5
10
153
55
-55
V
5
10

Standardized Definition of Structural Valve Degeneration for Surgical and Transcatheter Bioprosthetic Aortic Valves

Danny Dvir, MD*
Thierry Bourguignon, MD*
et al

SVD Definition

SVD Stage 0	• No significant change from immediate post implantation*
SVD Stage 1	• Morphological leaflet abnormality without significant hemodynamic changes†
SVD Stage 2S	• Moderate stenosis‡
SVD Stage 2R	• Moderate regurgitation§
SVD Stage 2RS	• Moderate stenosis and moderate regurgitation
SVD Stage 3	• Severe stenosis and/or severe regurgitation

Circulation. 2018;137:388–399.

PLACE 9ª Edizione

Standardized Definition of Structural Valve Degeneration for Surgical and Transcatheter Bioprosthetic Aortic Valves

DIAGNOSIS OF SVD

Echocardiography

Transthoracic echocardiography (TTE) is the primary imaging modality for the assessment of SAVR and TAVR valves, although transesophageal echocardiography (TEE) may be helpful when standard imaging is suboptimal.[7,58] All patients should have a complete diagnostic TTE postimplantation study after SAVR or TAVR at discharge to provide a baseline for comparison with future studies, including comprehensive imaging of the valve prosthesis components, measurement of antegrade velocity, mean gradient, aortic valve effective orifice area, and evaluation of any (transvalvular or paravalvular) prosthetic regurgitation. In patients followed af-

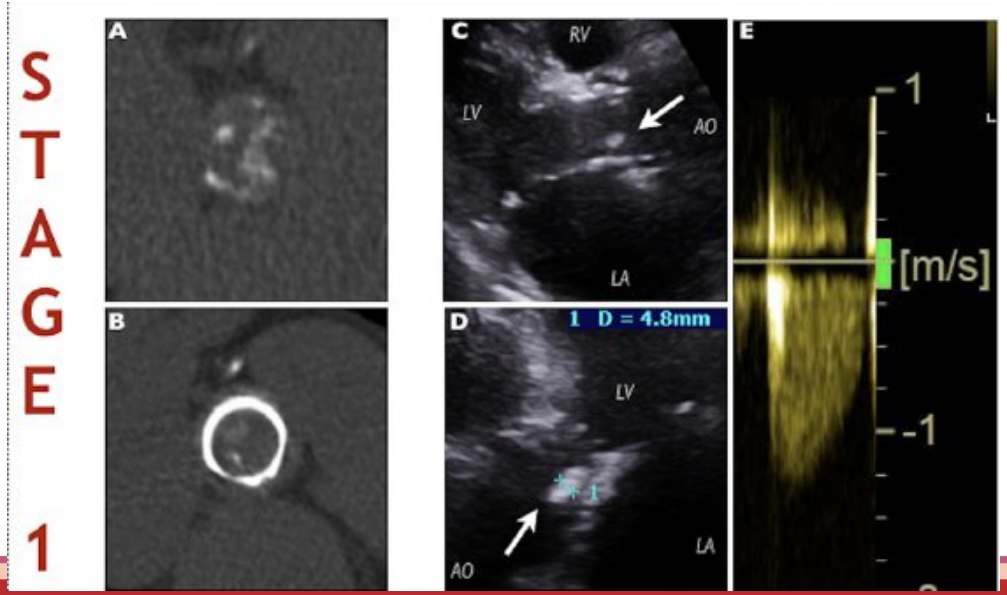
Circulation. 2018;137:388–399.

DIAGNOSIS OF SVD

SVD Stage 1

- **Morphological leaflet abnormality without significant hemodynamic changes†**

Examples include leaflet fluttering, leaflet thickening, and limited, delayed, or asymmetrical leaflet opening or closure on echocardiography or other types of imag-



DIAGNOSIS OF SVD

Computed tomography predictors of structural valve degeneration in patients undergoing transcatheter aortic valve implantation with balloon-expandable prostheses

Marco Guglielmo[1] · Laura Fusini[1] · Manuela Muratori[1] · Gloria Tamborini[1] · Valentina Mantegazza[1] · Daniele Andreini[1,2] · Andrea Annoni[1] · Mario Babbaro[1] · Andrea Baggiano[1] · Edoardo Conte[1] · Serena Carriero[3] · Alberto Formenti[1] · Andrea Igoren Guaricci[4] · Elisabetta Mancini[1] · Rocco Mollace[1] · Giuseppe Muscogiuri[1] · Saima Mushtaq[1] · Francesca Ricci[1] · Alexia Rossi[5,6] · Stefano Scafuri[1] · Brunilda Alushi[7,8,9] · Claudio Cau[10] · Riccardo Cau[10] · Margherita Cesarano[11] · Luca Saba[10] · Mark Rabbat[12,13] · Mauro Pepi[1] · Gianluca Pontone[1]

Conclusions Female sex and AoA D_{min} < 19.9 mm are associated with SVD in patients undergoing TAVI with balloon-expandable valves. When implanting large prostheses in order to avoid paraprosthetic regurgitation, caution should be observed due to the risk of excessive stretching of the AoA $D_{min,}$ which may play a role in SVD.

SVD Stage 2S

• Moderat

Stage 2 SVD refers to morphological abnormalities of valve leaflets associated with hemodynamic dysfunc-

Semiquantitative parameters	
Acceleration time, ms	
Acceleration time/LV ejection time ratio	
Quantitative flow-dependent parameters	
Peak velocity, m/s	3–4
Mean gradient, mm Hg	20–40
Increase in mean gradient during follow-up associated with decrease in EOA and DVI	10–20
Quantitative flow-independent parameters	
Doppler velocity index	0.25–0.35
EOA for BSA ≥1.6 m²	1.0–1.2
EOA for BSA <1.6 m²	0.8–1.1

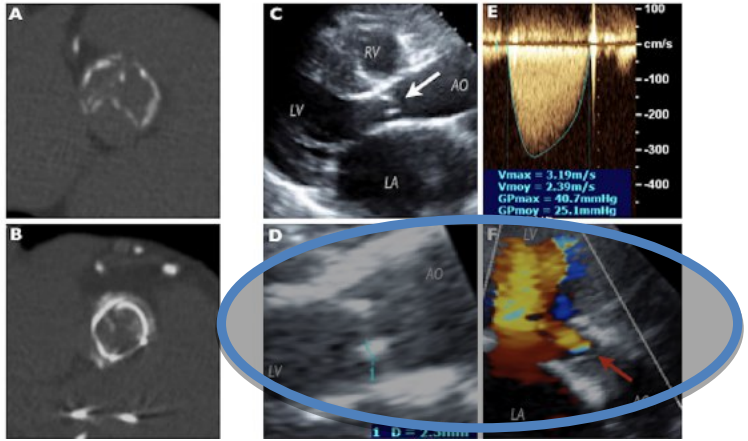
tion are graded Stage 2R. Important considerations in the determination of Stage 2S include an increase in transvalvular mean gradient of ≥10 mm Hg with a concomitant decrease in valve area, which is not the result of isolated leaflet thickening. Many bioprosthetic valves have baseline postprocedural gradients of 15 to 19 mm Hg (because of prosthesis-patient mismatch), and a mild increase in gradients often related to an increase in flow should not be viewed as SVD. This possible confusion is the reason that the documentation of abnormal leaflet morphology and deterioration in valve hemodynamics (increase in gradients and decrease in valve rea) is crucial for the definition of SVD. High-quality

Circulation. 2018;137:388–399.

SVD Stage 2R

- Moderate regurgitation§

STAGE 2



tion should be followed by assessment of the location of the leakage (optimally with transesophageal echo-cardiography) to exclude paravalvular regurgitation.

PLACE 9ª Edizione
PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE

Aortic Bioprosthetic Valve Durability

Incidence, Mechanisms, Predictors, and Management of Surgical and Transcatheter Valve Degeneration

Tania Rodriguez-Gabella, MD, Pierre Voisine, MD, Rishi Puri, MBBS, PhD, Philippe Pibarot, DVM, PhD, Josep Rodés-Cabau, MD

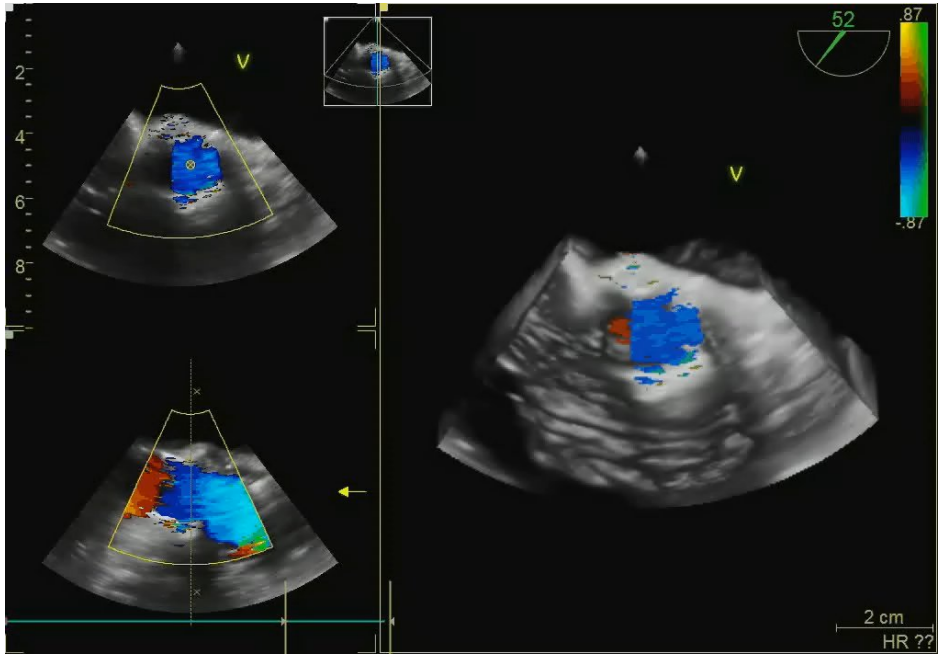
DIAGNOSIS AND DEFINITION OF SVD

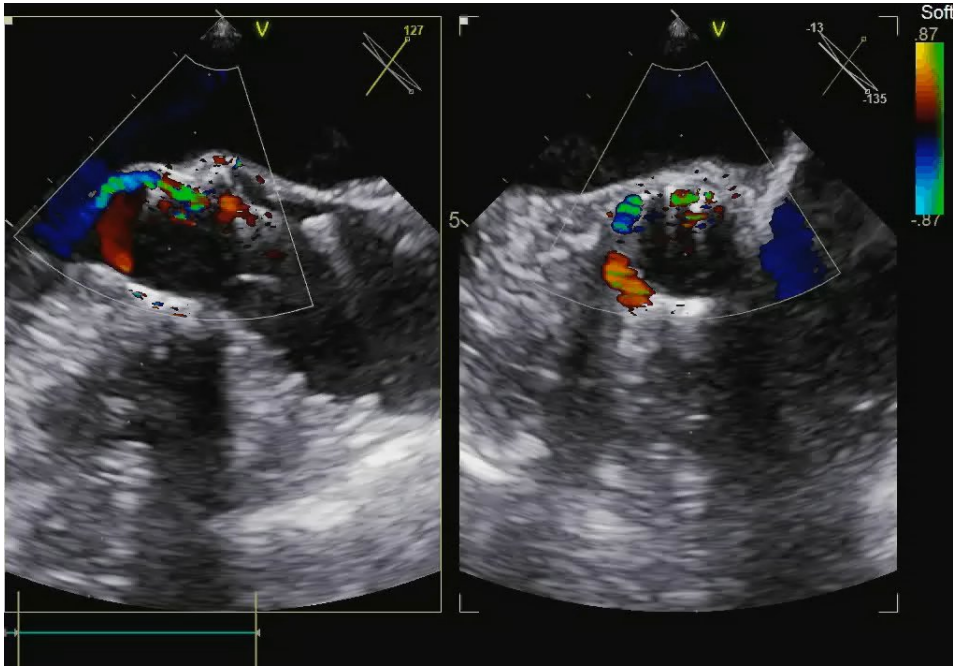
The higher resolution makes TEE an even more helpful imaging technique for evaluating and identifying the mechanism of biological SVD (leaflet thickening/calcification or leaflet flail or tear) [Its higher resolution makes transesophageal echocardiography (TEE) a helpful imaging technique when TTE has technical limitations. The role of TEE remains essential for assessing aortic insufficiency and distinguishing between transvalvular and paravalvular regurgitation. The 3-dimensional echocardiographic en face surgical view of the valve is extremely useful for determining the presence, origin, direction, and extension of regurgitant jets

rosthetic dysfunc-

PLACE 9ª Edizione

PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE





DIFFICOLTA' DIAGNOSTICHE

01 • ANALISI MORFOLOGICA
- Ispessimento/Trombosi
- Trombosi/Panno

02 • ANALISI FUNZIONALE
- Ostruzione/Mismatch pr-pz

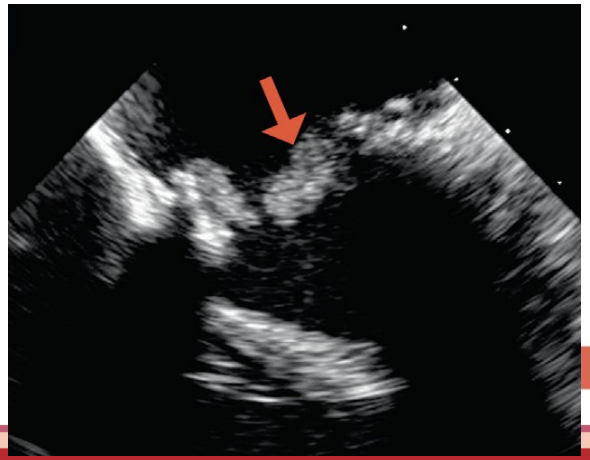
Bioprosthetic Valve Thrombosis Versus Structural Failure

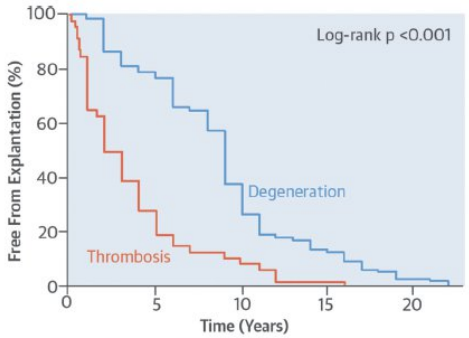
Clinical and Echocardiographic Predictors

Alexander C. Egbe, MD, MPH,* Sorin V. Pislaru, MD, PhD,* Patricia A. Pellikka, MD,* Joseph T. Poterucha, DO,* Hartzell V. Schaff, MD,† Joseph J. Maleszewski, MD,‡ Heidi M. Connolly, MD*

BACKGROUND Bioprosthetic valve thrombosis (BPVT) is considered uncommon; this may be related to the fact that it is often unrecognized. Recent data suggest that BPVT responds to vitamin K antagonists, emphasizing the need for reliable diagnosis.

CONCLUSIONS BPVT is not uncommon and can occur several years after surgery. A combination of clinical and echocardiographic features can reliably diagnose BPVT. (J Am Coll Cardiol 2015;66:2285–94)





COMPETENCY IN MEDICAL KNOWLEDGE: Thrombosis of bioprosthetic heart valves can occur several years after implantation. Likely predictors of this complication include a >50% increase in mean echo-Doppler transvalvular gradient within 5 years, paroxysmal atrial fibrillation, subtherapeutic INR in patients anticoagulated with vitamin K antagonists, increased cusp thickness, and abnormal cusp mobility.

Bioprosthetic Valve Thrombosis Versus Structural Failure

Clinical and Echocardiographic Predictors

Alexander C. Egbe, MD, MPH,* Sorin V. Pislaru, MD, PhD,* Patricia A. Pellikka, MD,* Joseph T. Poterucha, DO,* Hartzell V. Schaff, MD,† Joseph J. Maleszewski, MD,‡ Heidi M. Connolly, MD*

CONCLUSIONS BPVT is not uncommon and can occur several years after surgery. A combination of clinical and echocardiographic features can reliably diagnose BPVT. (J Am Coll Cardiol 2015;66:2285–94)

TABLE 5 Test Performance Characteristics for the Diagnosis of BPVT

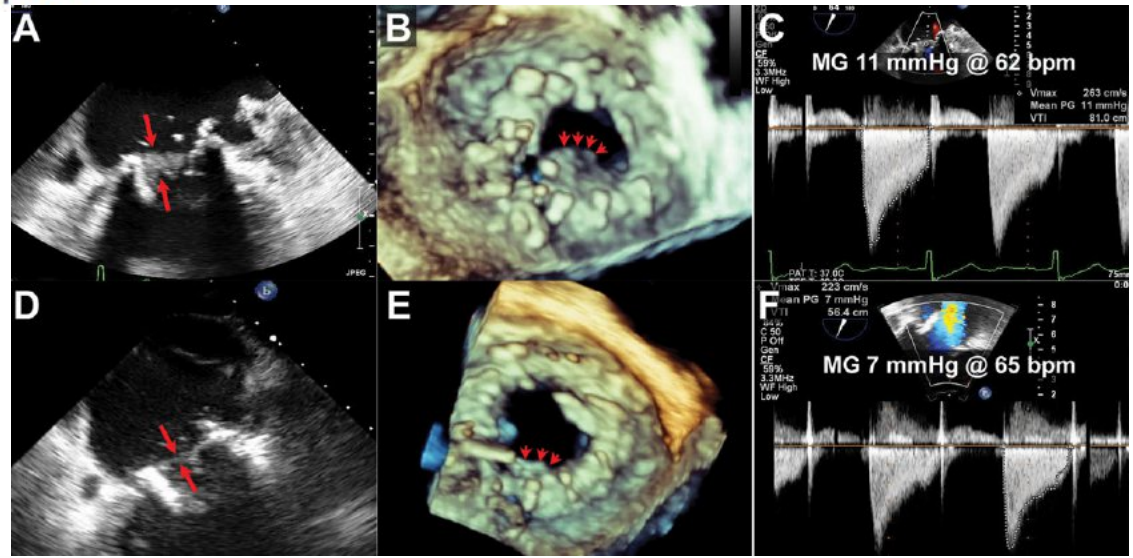
	Total Score	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Variables					
A. 50% mean gradient increase	1	45	89	68	77
B. Increase cusp thickness	1	74	69	55	84
C. Abnormal cusp mobility	1	63	70	51	81
D. Paroxysmal AF	1	63	73	54	80
E. Subtherapeutic INR	1	30	92	67	73
Combination of variables					
A and B	2	86	57	50	89
A, B, and C	3	72	90	78	87
A, B, C, and D	4	70	94	87	86
A, B, C, D, and E	5	66	93	85	89

PLACE 9ª Edizione

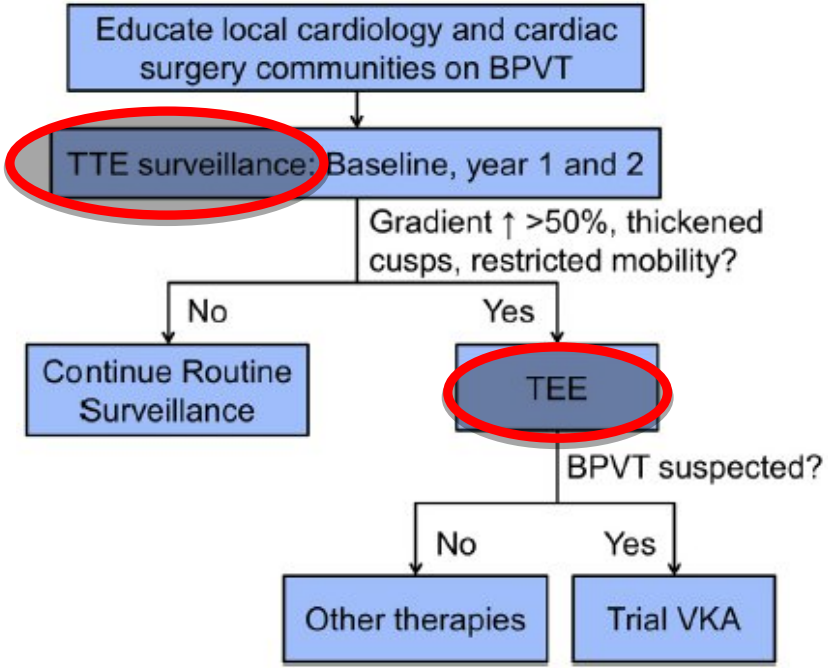
Misconceptions, diagnostic challenges and treatment opportunities in bioprosthetic valve thrombosis: lessons from a case series

Sorin V. Pislaru[a,†,*], Imad Hussain[a,†], Patricia A. Pellikka[a], Joseph J. Maleszewski[b], Richard D. Hanna[a], Hartzell V. Schaff[c] and Heidi M. Connolly[a]

CONCLUSIONS: BPVT may occur late after surgical implantation. VKA therapy resulted in haemodynamic and clinical improvement with minimal risk, and should be considered the first-line therapy in haemodynamically stable patients. Echocardiographic criteria for improving BPVT diagnosis are proposed.

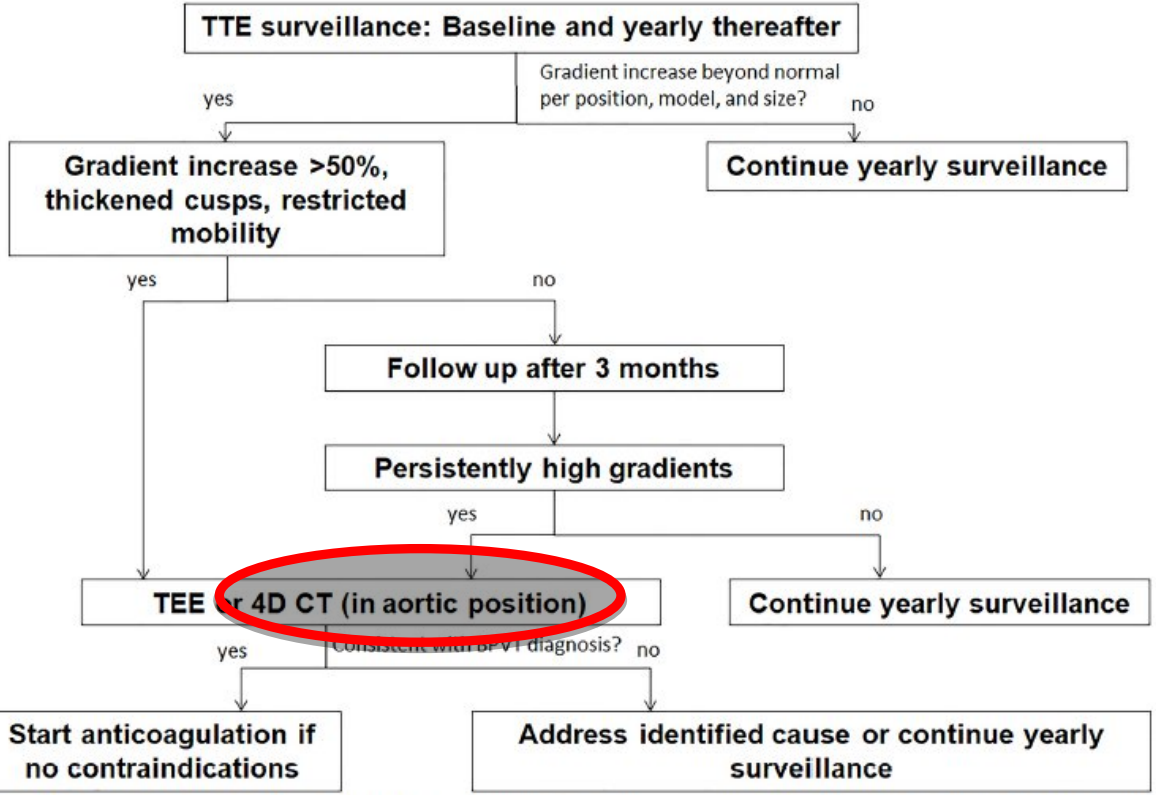


Bioprosthetic valve thrombosis: The eyes will not see what the mind does not know



Bioprosthetic valve thrombosis (BPVT) is thought to be a rare, but potentially life-threatening complication, with reported incidence varying from 0.1 per 100 valve-years[1] to as high as 6%.[2] In this issue of the *Journal*, Dohi

Gradient changes in bioprosthetic valve thrombosis: duration of anticoagulation and strategies to improve detection



Suggested approach for diagnosis of bioprosthetic valve thrombosis. TEE, transoesophageal echocardiography.

Naser JA, *et al. Open Heart* 2021;**8**:e001608.

CLINICAL MANAGEMENT ACCORDING TO SVD STAGE

Baseline echocardiographic examinations, both immediately post-implant and after 30-days.

Education regarding the need to promptly seek medical evaluation when symptoms occur.

Stage 0	*Yearly clinical and echocardiographic follow-up*
Stage 1	*Repeat echocardiography at 3-6 months** *Trial of anticoagulation if subclinical leaflet thrombosis is suspected*
Stage 2	*Clinical evaluation and repeat echocardiography at 3-6 months** *Consider intervention if symptomatic and symptoms are related to SVD*
Stage 3	*Consider intervention†* *If asymptomatic with preserved LVEF consider clinical and echocardiographic evaluation every 3-6 months*

Thrombosis of Bioprosthetic Valves

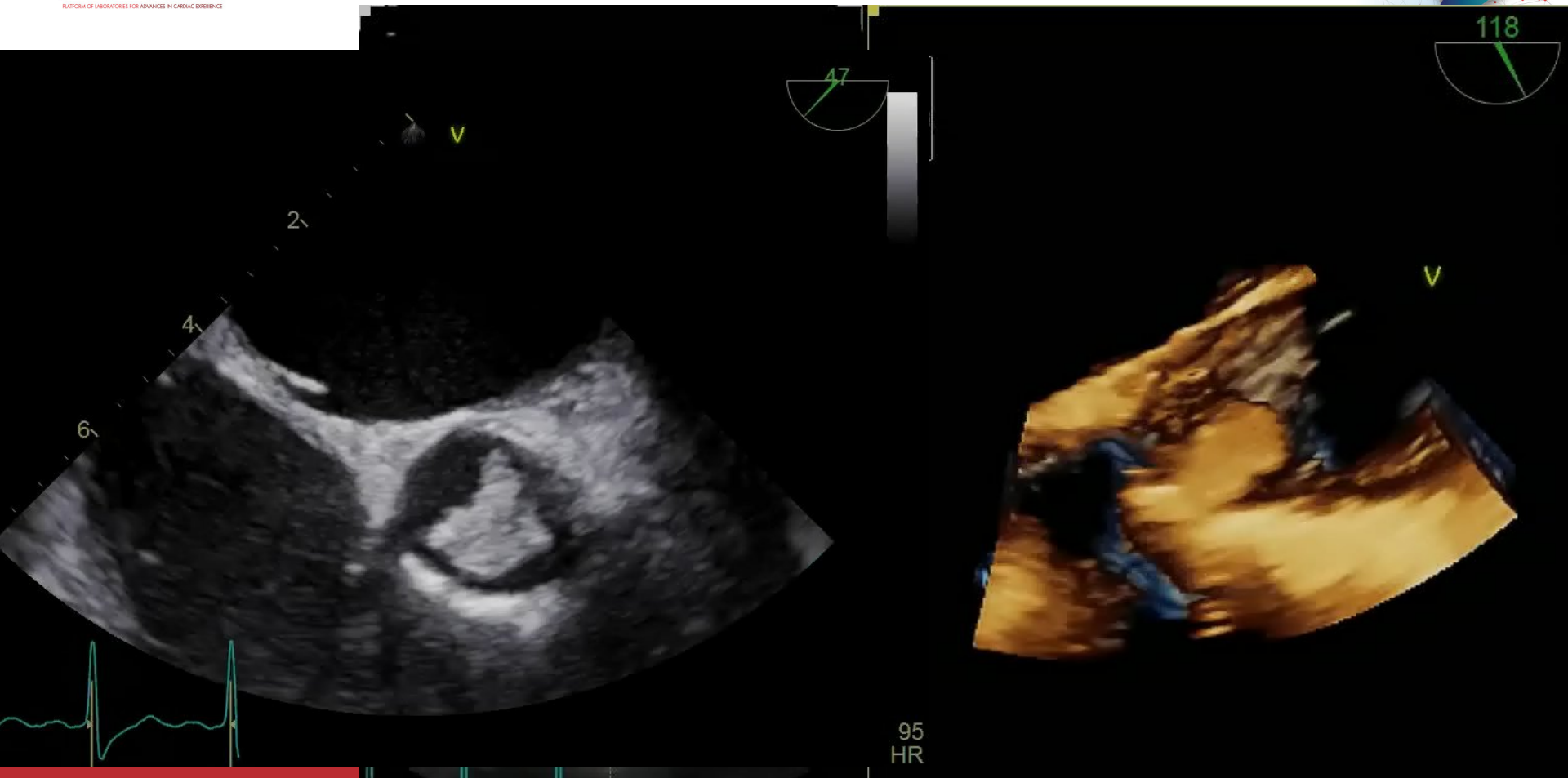
Can We Afford to Ignore It?*

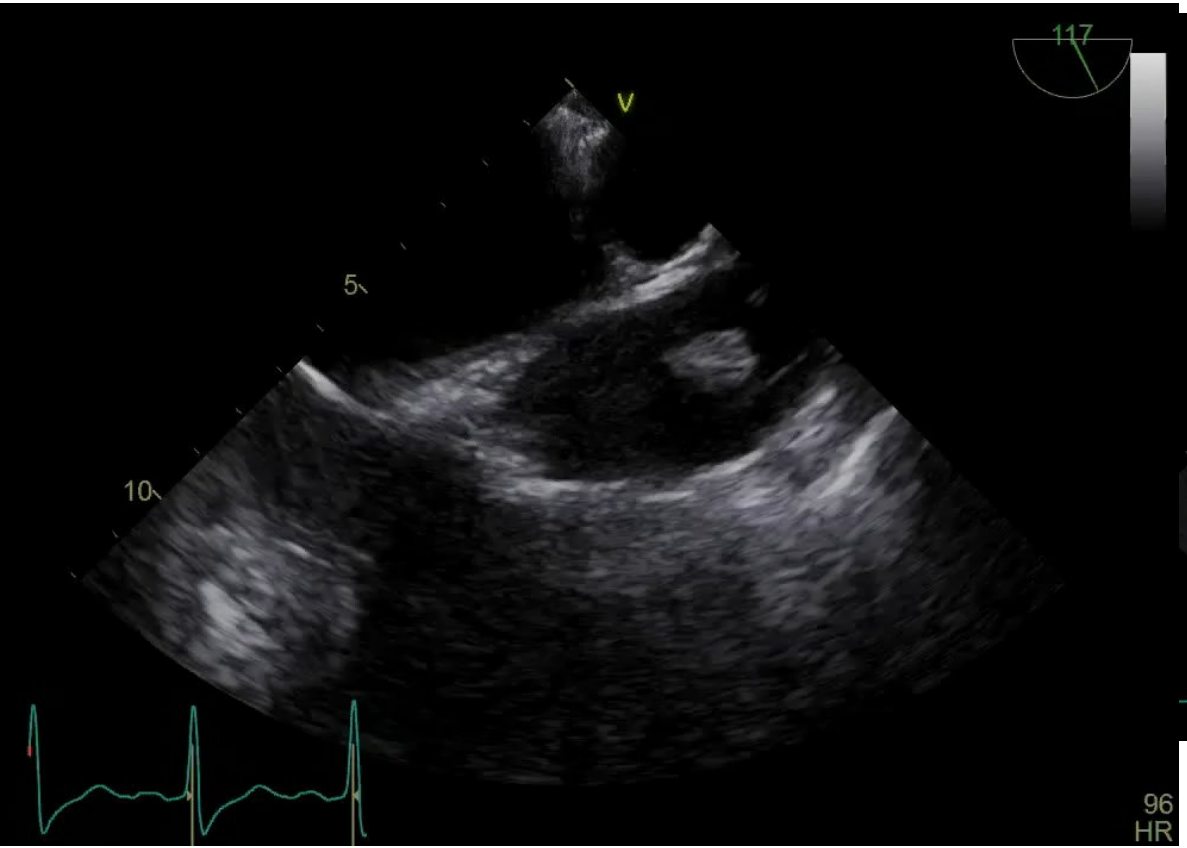
William J. Stewart, MD

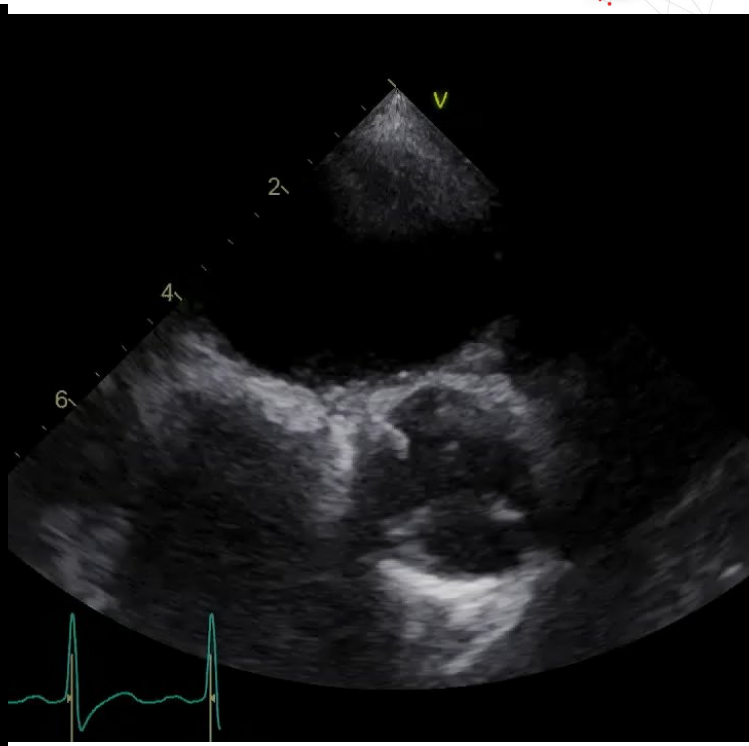
JACC VOL. 66, NO. 21, 2015

PLACE 9ª Edizione

PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE




117
V
5
10
96
HR

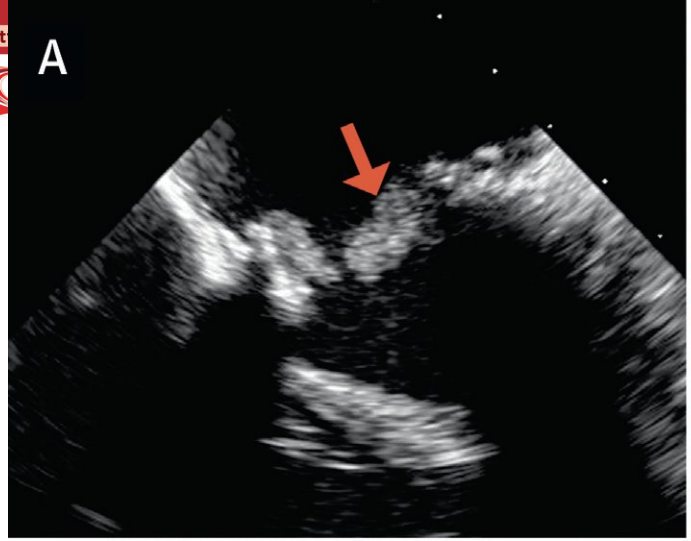

V
2
4
6

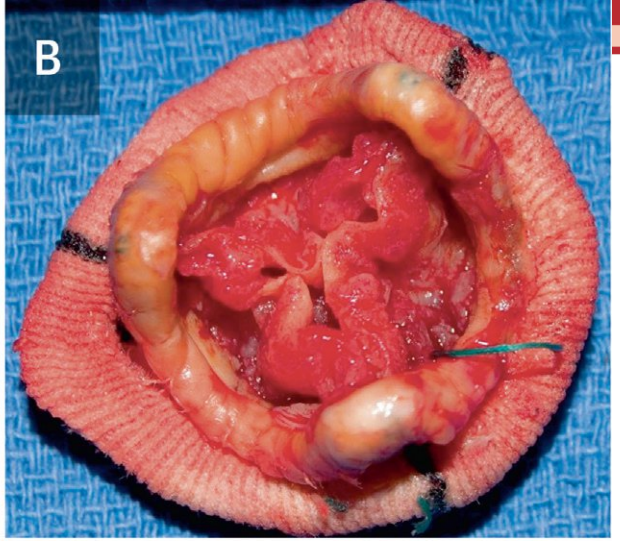
DIFFICOLTA' DIAGNOSTICHE

01 • ANALISI MORFOLOGICA
- Ispessimento/Trombosi
- Trombosi/Panno

02 • ANALISI FUNZIONALE
- Ostruzione/Mismatch pr-pz

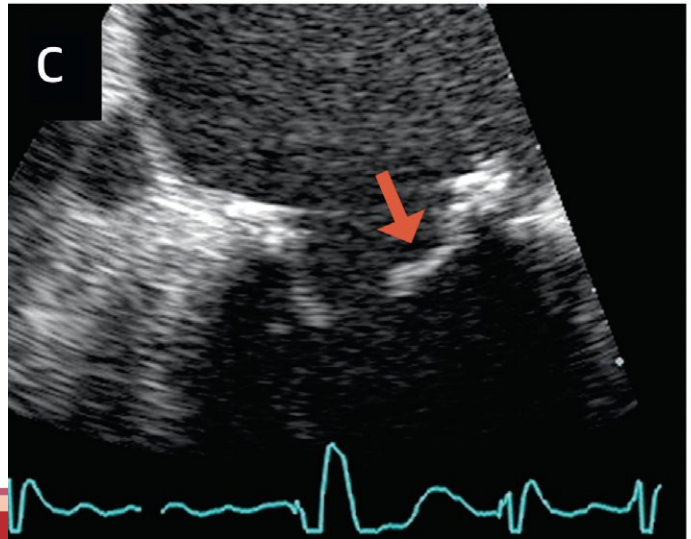
TROMBOSI

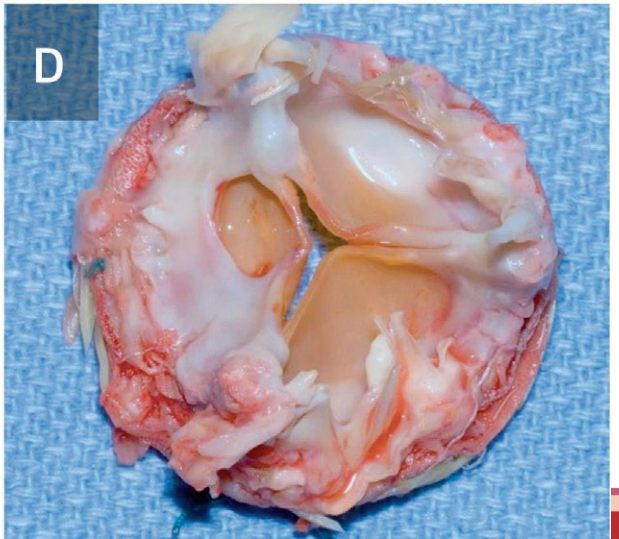




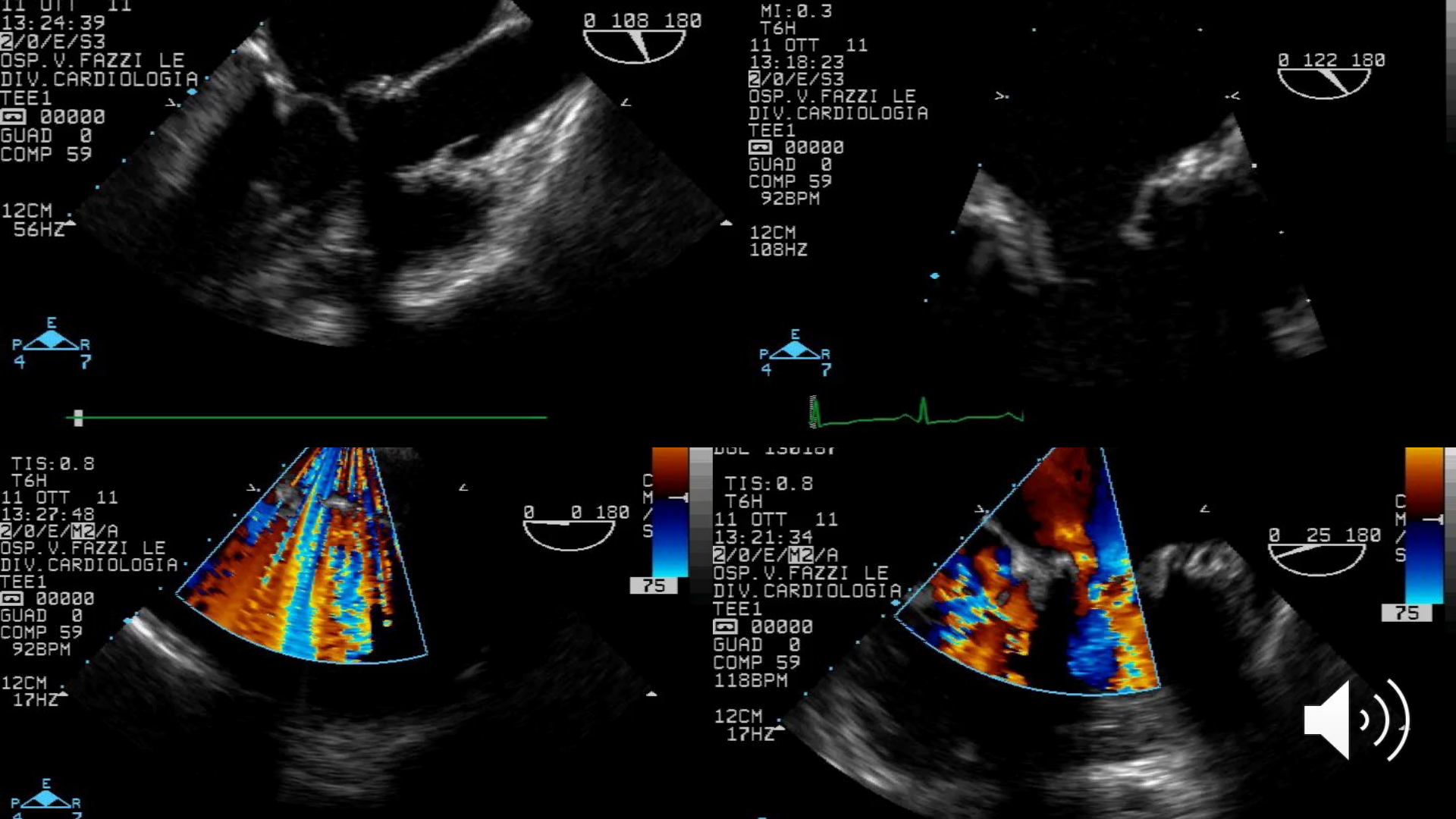
TROMBOSI

PANNO





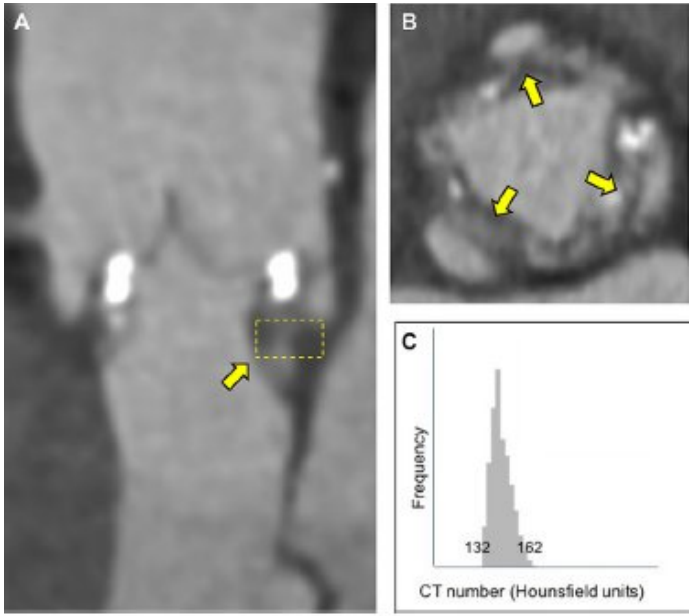
PANNO

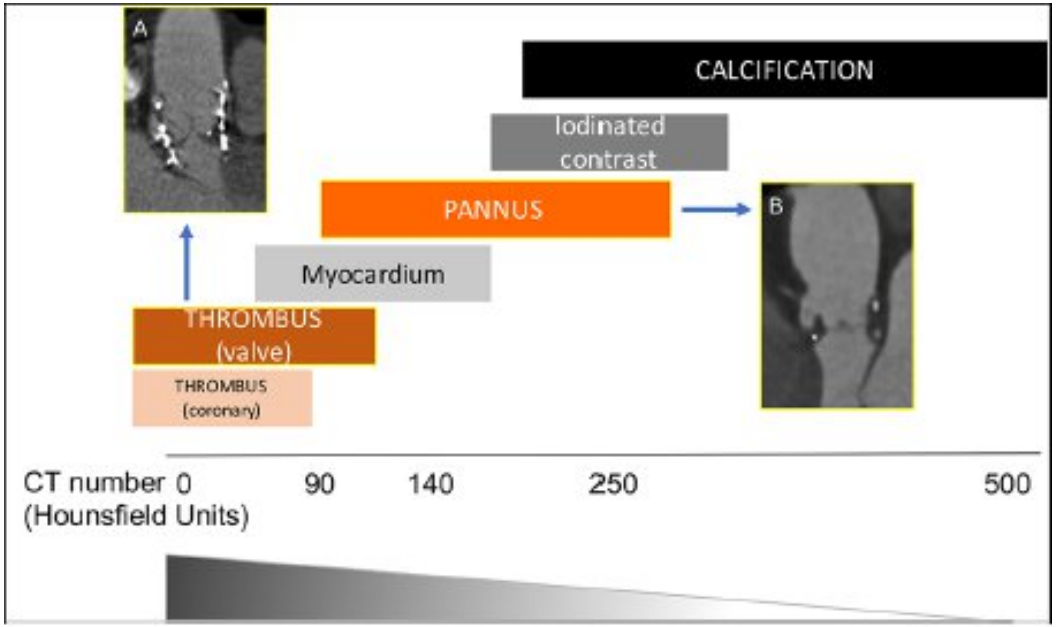

0 108 180
13:24:39
2/0/E/S3
OSP.V.FAZZI LE
DIV.CARDIOLOGIA
TEE1
00000
GUAD 0
COMP 59
12CM
56HZ
E
P R
4 7
MI:0.3
T6H
11 OTT 11
13:18:23
2/0/E/S3
OSP.V.FAZZI LE
DIV.CARDIOLOGIA
TEE1
00000
GUAD 0
COMP 59
92BPM
12CM
108HZ
0 122 180
E
P R
4 7
TIS:0.8
T6H
11 OTT 11
13:27:48
2/0/E/M2/A
OSP.V.FAZZI LE
DIV.CARDIOLOGIA
TEE1
00000
GUAD 0
COMP 59
92BPM
12CM
17HZ
0 0 180
CM/S
75
E
P R
TIS:0.8
T6H
11 OTT 11
13:21:34
2/0/E/M2/A
OSP.V.FAZZI LE
DIV.CARDIOLOGIA
TEE1
00000
GUAD 0
COMP 59
118BPM
12CM
17HZ
0 25 180
CM/S
75

PLACE 9ª Edizione

Cardiac CT in prosthetic aortic valve complications

JACK PATRICK MORRELL ANDREWS, MD, MRCP, TIMOTHY RG CARTLIDGE, MD, MRCP,
MARC ROBERT DWECK, MD, PhD and ALASTAIR J MOSS, MD, MRCP





Br J Radiol 2019;

PLACE 9ª Edizione

PANNUS vs THROMBOSIS

Table 10 Differential diagnosis: pannus vs. thrombosis

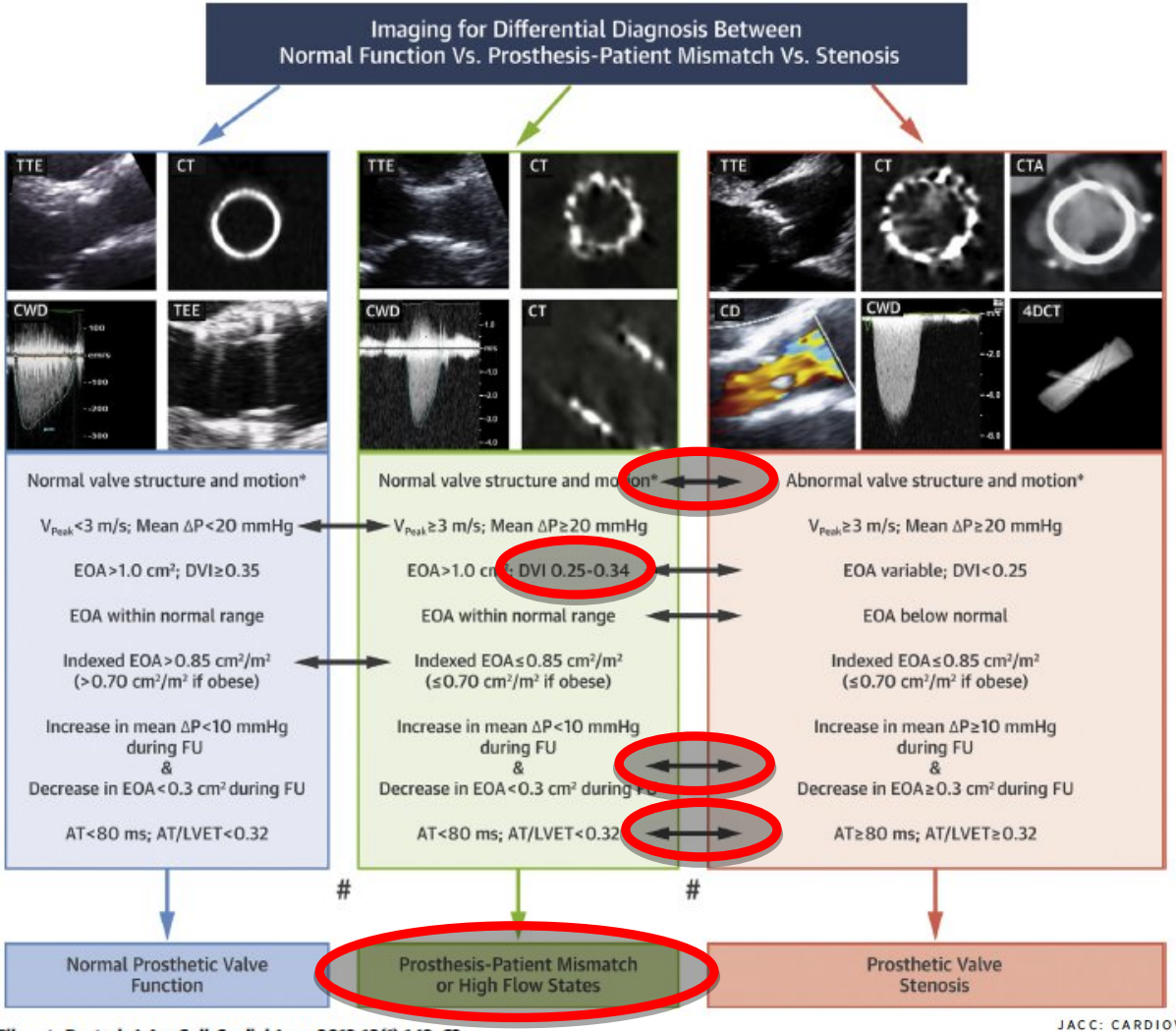
	Pannus	Thrombosis
Chronology	Minimum 12 months, commonly > 5 years from surgery date	Occurs at any time (if late usually associated with pannus)
Relation to anticoagulation (low INR)	Poor relationship	Strong relationship
Location	MV > AV	TV >> MV = AV
Morphology	• Small mass • Mostly involve suture line (Ring) • Centripetal growth • Confine to the disk plane • Growth beneath disc	• Larger mass than pannus • Independent motion common • Thin outer ring maybe visible • Project into LA for MV position • Mobile elements
Echo density (video-intensity ratio)	More >0.7 (100% specific)	Less (<0.4)
Cardiac CT: attenuation value	>200 HU	<200 HU
Impact on gradient	AV > MV	MV > AV
Impact on valve orifice	AV > MV	MV > AV
Impact on disc motion	Yes/no	Yes

AV, aortic valve; LA, left atrial; MV, mitral valve; TV, tricuspid valve.

DIFFICOLTA' DIAGNOSTICHE

01 • ANALISI MORFOLOGICA
- Ispessimento/Trombosi
- Trombosi/Panno

02 • ANALISI FUNZIONALE
- Ostruzione/Mismatch pr-pz



Pibarot, P. et al. J Am Coll Cardiol Img. 2019;12(1):149-62.

PLACE 9ª Edizione
PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE

Predictors and Outcomes of Prosthesis-Patient Mismatch After Aortic Valve Replacement

Victor Dayan, MD, PhD,[a] Gustavo Vignolo, MD,[a] Gerardo Soca, MD,[a] Juan Jose Paganini, MD,[a] Daniel Brusich, MD,[a] Philippe Pibarot, DVM, PhD[b]

JACC: CARDIOVASCULAR IMAGING, VOL. 9, NO. 8, 2016

Outcomes	Studies	Patients	Odds/HR/MD	P
Perioperative Mortality	33	25956	1.54 [1.25, 1.91]	<0.0001
Overall Mortality	38	29560	1.26 [1.16, 1.36]	<0.0001
PNC	9	7690	1.59 [1.05, 2.44]	0.003
ΔLVEF	6	1174	-1.02 [0.24, -2.28]	0.067
ΔLVMI	14	2927	18.38 [24.99, 12.06]	<0.0001

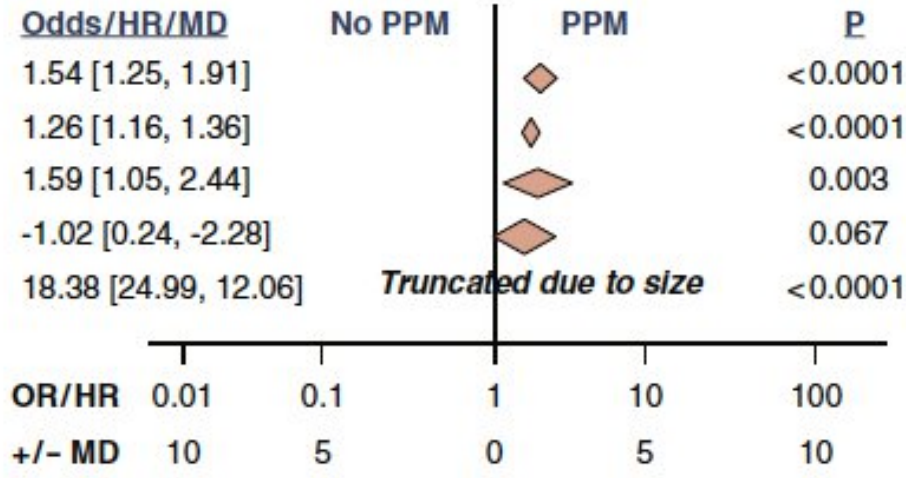


TABLE 1 Imaging Criteria for the Differential Diagnosis of Normal Prosthetic Valve Function vs. PPM vs. Valve Stenosis

	Normal	Moderate PPM	Severe PPM	Mild/Moderate Stenosis	Severe Stenosis
Leaflet morphology and mobility by TTE/TEE or MDCT*	Normal	Normal	Normal	Often abnormal	Abnormal
Doppler echo parameters					
Peak velocity, m/s	<3	3-3.5	≥3.5	3-4	≥4
Mean gradient, mm Hg	<20	20-30	≥30	20-35	≥35
Doppler velocity index	≥0.35	≥0.30	≥0.30	0.25-0.35	<0.25
EOA, cm^2	>1.00	>1.00	>1.00	Variable	<0.80
Indexed EOA, cm^2/m^2	>0.85	0.66-0.85	≤0.65	0.66-0.85	≤0.65
If BMI ≥30 kg/m^2	>0.70	0.56-0.70	≤0.55	0.56-0.70	≤0.55
Difference (normal EOA – measured EOA), cm^2	<0.30 (<1 SD)	<0.30 (<1 SD)	<0.30 (<1 SD)	0.30-0.60 (1-2 SD)	>0.60 (>2 SD)
Contour of the transprosthetic jet†	Triangular, early peaking	Triangular, early peaking	Triangular, early peaking	Triangular to intermediate	Rounded, symmetrical
Acceleration time, ms†	<80	<80	<80	80-100	>100
Acceleration time/LV ejection time ratio†	<0.32	<0.32	<0.32	0.32-0.37	>0.37
Changes in Doppler echo parameters during follow-up					
Increase in mean gradient, mm Hg	<10	<10	<10	10-19	≥20
Decrease in EOA, cm^2	<0.30	<0.30	<0.30	0.30-0.60	>0.60
Percent decrease in EOA, %	<25	<25	<25	25-49	≥50
Percent decrease in DVI, %	<20	<20	<20	20-39	≥40
Hybrid (Doppler CT) parameters					
Indexed hybrid EOA, cm^2/m^2	>1.00	0.81-1.00	≤0.80	0.81-1.00	≤0.80
If BMI ≥30 kg/m^2	>0.85	0.71-0.85	≤0.70	0.71-0.85	≤0.70

Prosthesis-Patient Mismatch After Aortic Valve Replacement in the PARTNER 2 Trial and Registry

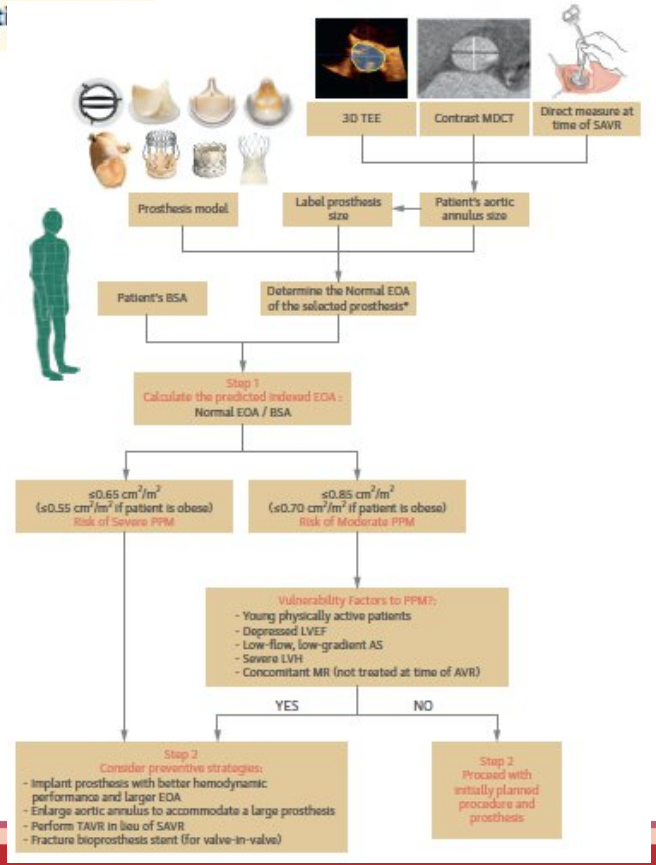
JACC: CARDIOVASCULAR INTERVENTIONS VOL. 14, NO. 13, 2021

PPM is often defined with the use of the EOAi directly measured by transthoracic echocardiography at the pre-discharge or 30-day echocardiogram. However, the measured EOAi has several limitations: 1) it (and thus the determination of PPM) is not available in patients with missing echocardiograms; 2) it is subject to echocardiographic technical pitfalls and measurement errors; and 3) it is flow dependent and may thus overestimate the severity of PPM in patients with a low-flow state. To overcome this limitation, the use of the predicted EOAi has been proposed to define PPM; this parameter is calculated by dividing the normal reference value of EOA for the implanted model and size of prosthesis by the patient's BSA

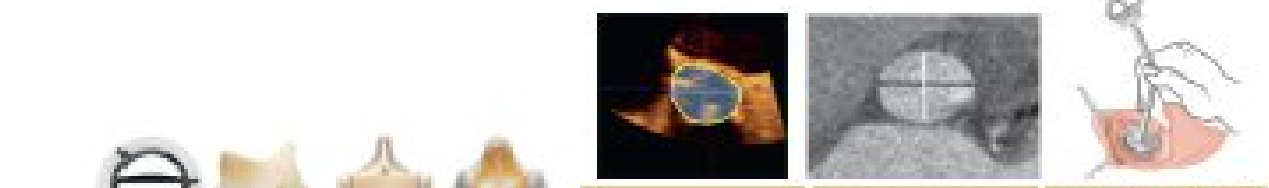
Imaging for Predicting and Assessing Prosthesis-Patient Mismatch After Aortic Valve Replacement

Philippe Pibarot, DVM, PhD,[a] Julien Magne, PhD,[b,c] Jonathon Leipsic, MD,[d] Nancy Côté, PhD,[a] Philippe Blanke, MD,[d] Vinod H. Thourani, MD,[e] Rebecca Hahn, MD[f]

Algorithm for the Prediction and Preventi



JACC: CARDIOVASCULAR IMAGING, VOL. 12, NO. 1, 2019
JANUARY 2019:149–62



3D TEE | Contrast MDCT | Direct measure at time of SAVR

Prosthesis model

Label prosthesis size

Patient's aortic annulus size

Patient's BSA

Determine the Normal EOA of the selected prosthesis*

Step 1
Calculate the predicted indexed EOA :
Normal EOA / BSA

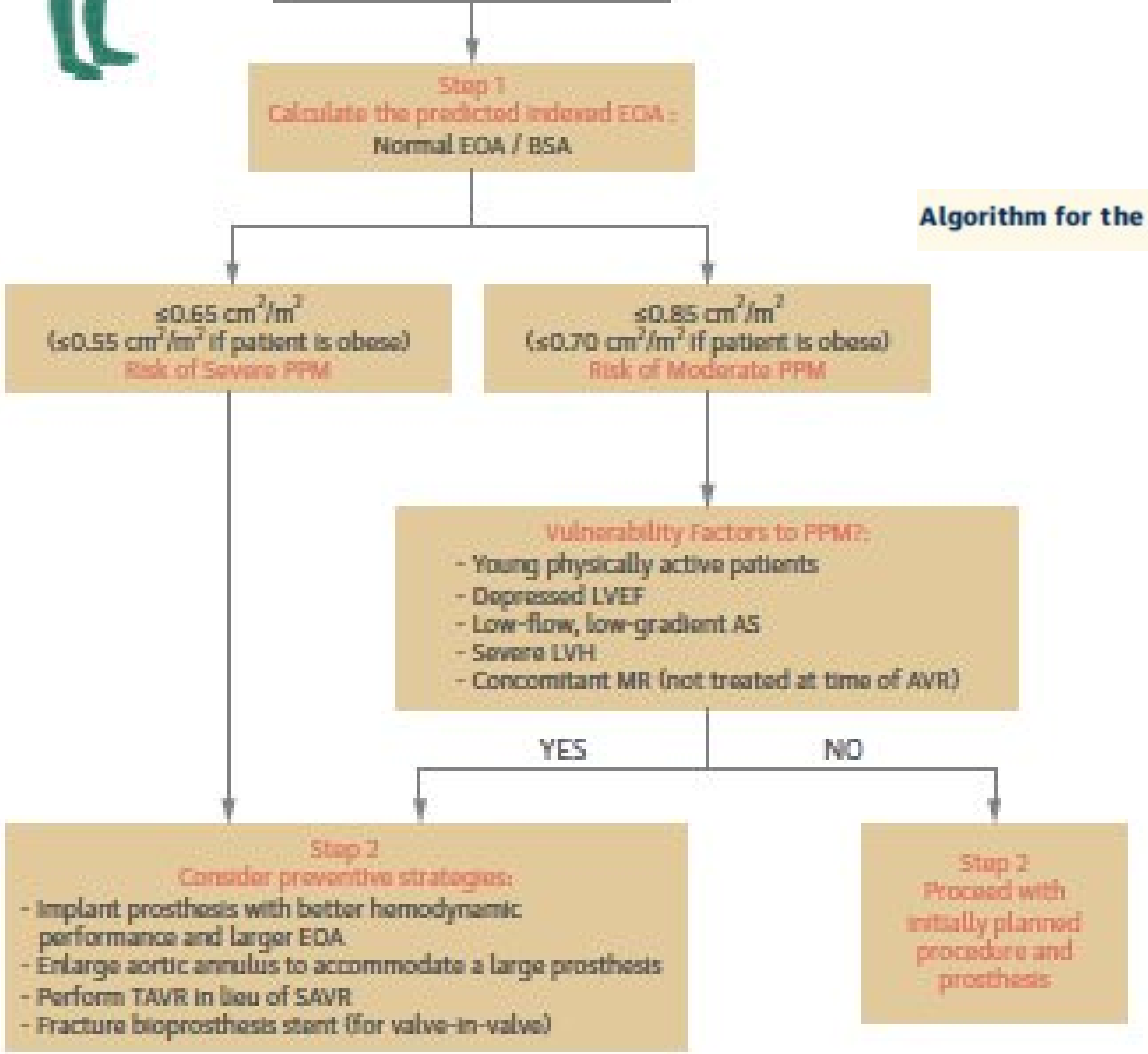
≤0.65 cm^2/m^2
(≤0.55 cm^2/m^2 if patient is obese)
Risk of Severe PPM

≤0.85 cm^2/m^2
(≤0.70 cm^2/m^2 if patient is obese)
Risk of Moderate PPM

Algorithm for the Prediction and Prevention of PPM

JACC: CARDIOVASCULAR IMAGING, VOL. 12, NO. 1, 2019
JANUARY 2019:149–62

Algorithm for the Prediction and Prevention of PPM



CONCLUSIONI

- La degenerazione continua ad essere uno dei principali limiti delle bio-protesi e ne condiziona la durata, nonostante i progressi tecnologici.
- Il significativo incremento nell'uso delle bioprotesi aortiche (specie TAVI) porterà inevitabilmente ad un aumentato numero di pazienti portatori di degenerazione nei prossimi 10 anni
- I principali FdR sono correlati alle caratteristiche dei pazienti (età più giovane, maggiore BMI, FdR CV) e a variabili tipiche delle bioprotesi (profilo emodinamico, MPP).
- Per la diagnosi di degenerazione, l'ETT è l'esame di prima linea, ma il TEE e, sempre più spesso, la MDCT sono utili nei casi di difficile definizione diagnostica e nella prevenzione delle disfunzioni protesiche.

CONCLUSIONI 2

Al fine di posticipare nel tempo la sua insorgenza ed aumentare la durata delle bioprotesi occorre un adeguato trattamento dei fdr CV (ipertensione…), ma anche una corretta selezione dei pazienti, considerando, prima dell'impianto, il rischio di degenerazione precoce.

THANK YOU

THANK YOU