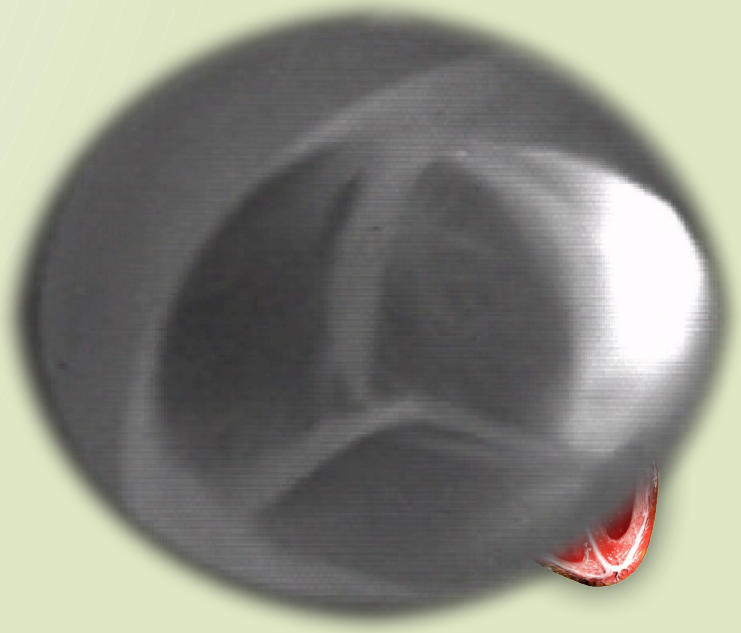
Prosthetic Heart Valves : Complications and Management

Shibikom Tamirat

General Surgeon

Adult and Pediatric Cardiac Surgeon



“Prosthetic heart valve is a device implanted in the heart of a patient with valvular heart disease”

Brunner and Suddarths



Early stages of cardiac surgery

- Elliott Cutler at the Peter Bent Brigham Hospital in Boston
  - In 1923. He inserted a valvulotome through a stab wound in the apex of the left ventricle of a twelve-year-old female patient with severe mitral stenosis and successfully incised both leaflets.
  - The patient survived and was improved but eventually died of additional cardiac problems four years later.
  - Cutler operated on six patients for mitral stenosis, but the only survivor was the first.
- In 1925 in London, Sir Henry Souttar
  - He used the left atrial appendage to approach the valve and inserted his finger through a purse-string suture
  - “It is of no use to be ahead of one’s time.”

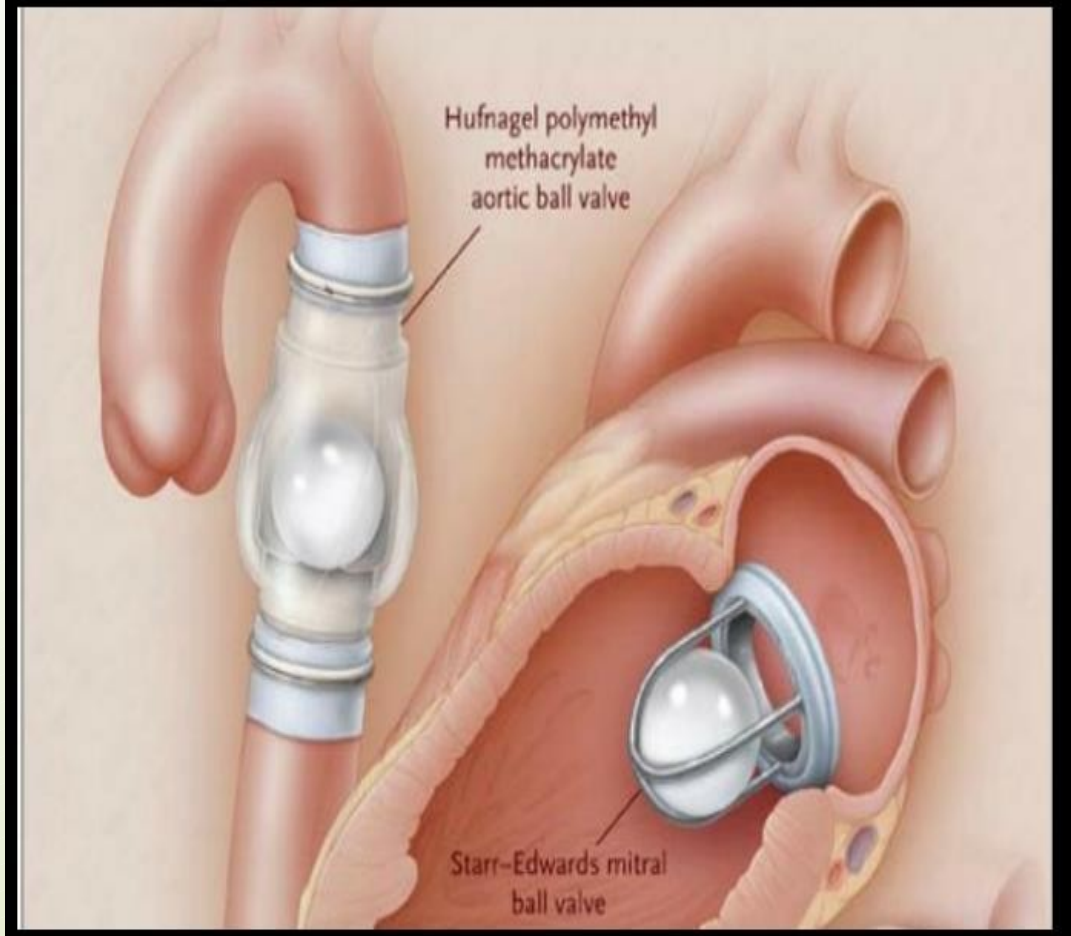


- Charles Bailey in Philadelphia
  - Devised a sheathed knife that could be attached to the index finger of the surgeon.
  - After four failures, this fifth attempt to open a stenotic valve was a total success—the patient lived another thirty-eight years.



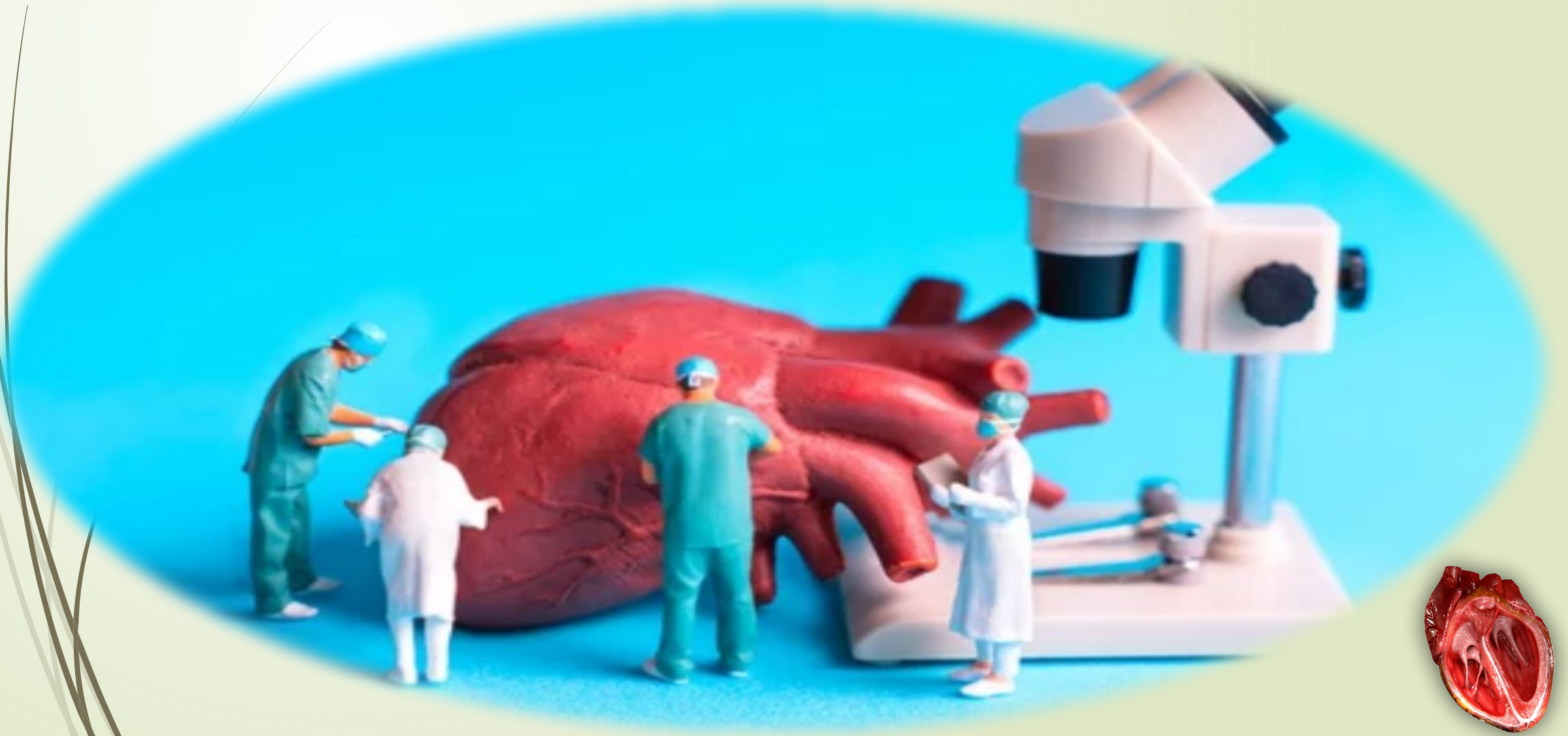


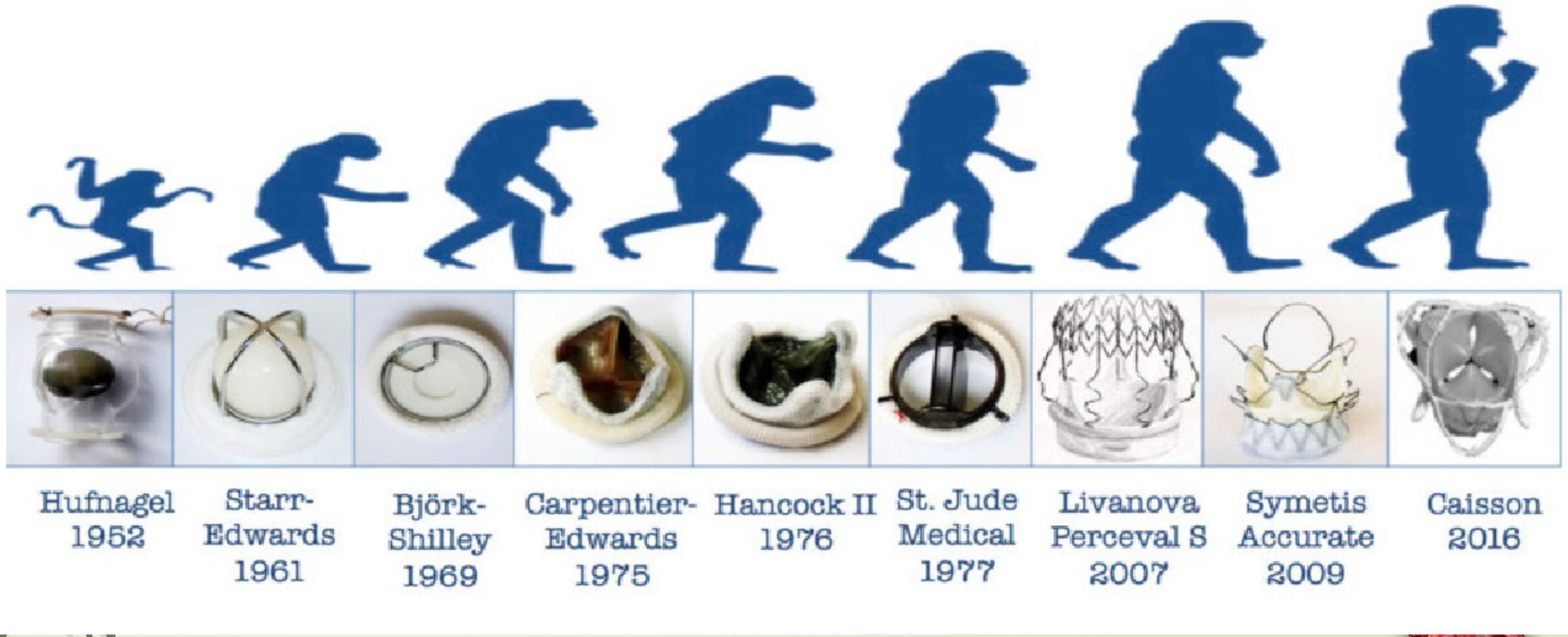
Valve de Hufnagel

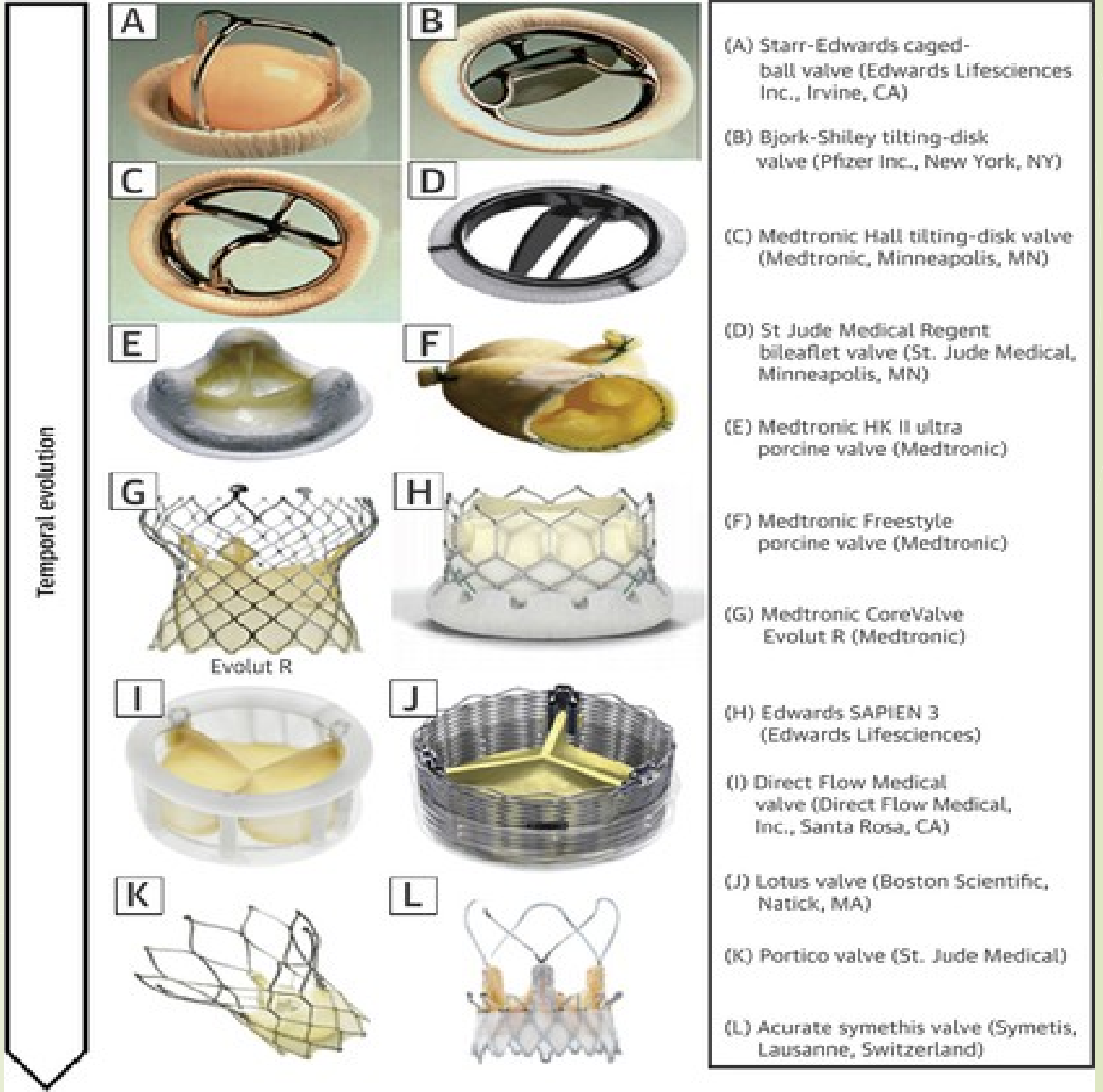









Hufnagel 1952
Starr-Edwards 1961
Björk-Shilley 1969
Carpentier-Edwards 1975
Hancock II 1976
St. Jude Medical 1977
Livanova Perceval S 2007
Symetis Accurate 2009
Caisson 2016



(A) Starr-Edwards caged-ball valve (Edwards Lifesciences Inc., Irvine, CA)

(B) Bjork-Shiley tilting-disk valve (Pfizer Inc., New York, NY)

(C) Medtronic Hall tilting-disk valve (Medtronic, Minneapolis, MN)

(D) St Jude Medical Regent bileaflet valve (St. Jude Medical, Minneapolis, MN)

(E) Medtronic HK II ultra porcine valve (Medtronic)

(F) Medtronic Freestyle porcine valve (Medtronic)

(G) Medtronic CoreValve Evolut R (Medtronic)

(H) Edwards SAPIEN 3 (Edwards Lifesciences)

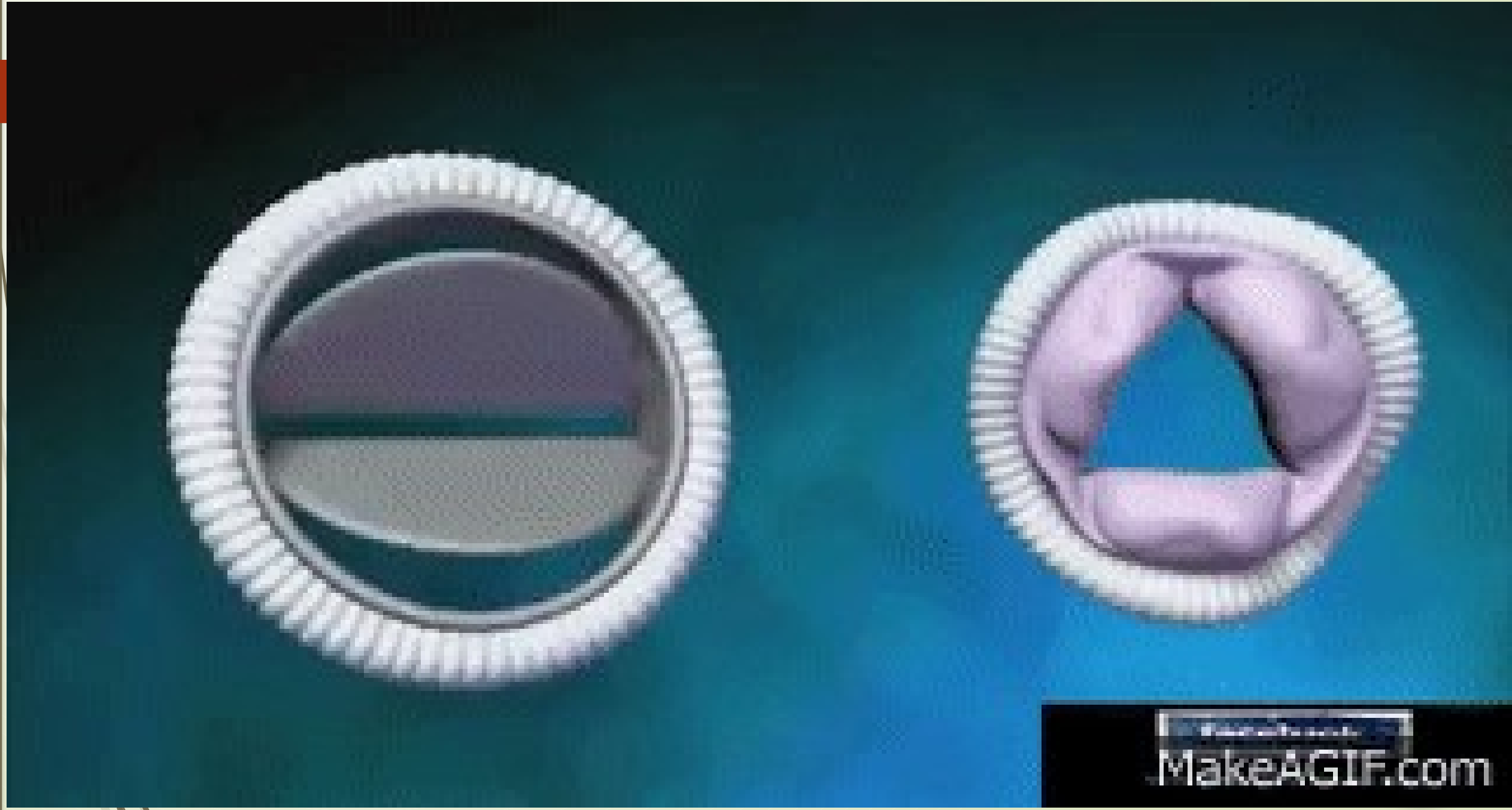
(I) Direct Flow Medical valve (Direct Flow Medical, Inc., Santa Rosa, CA)

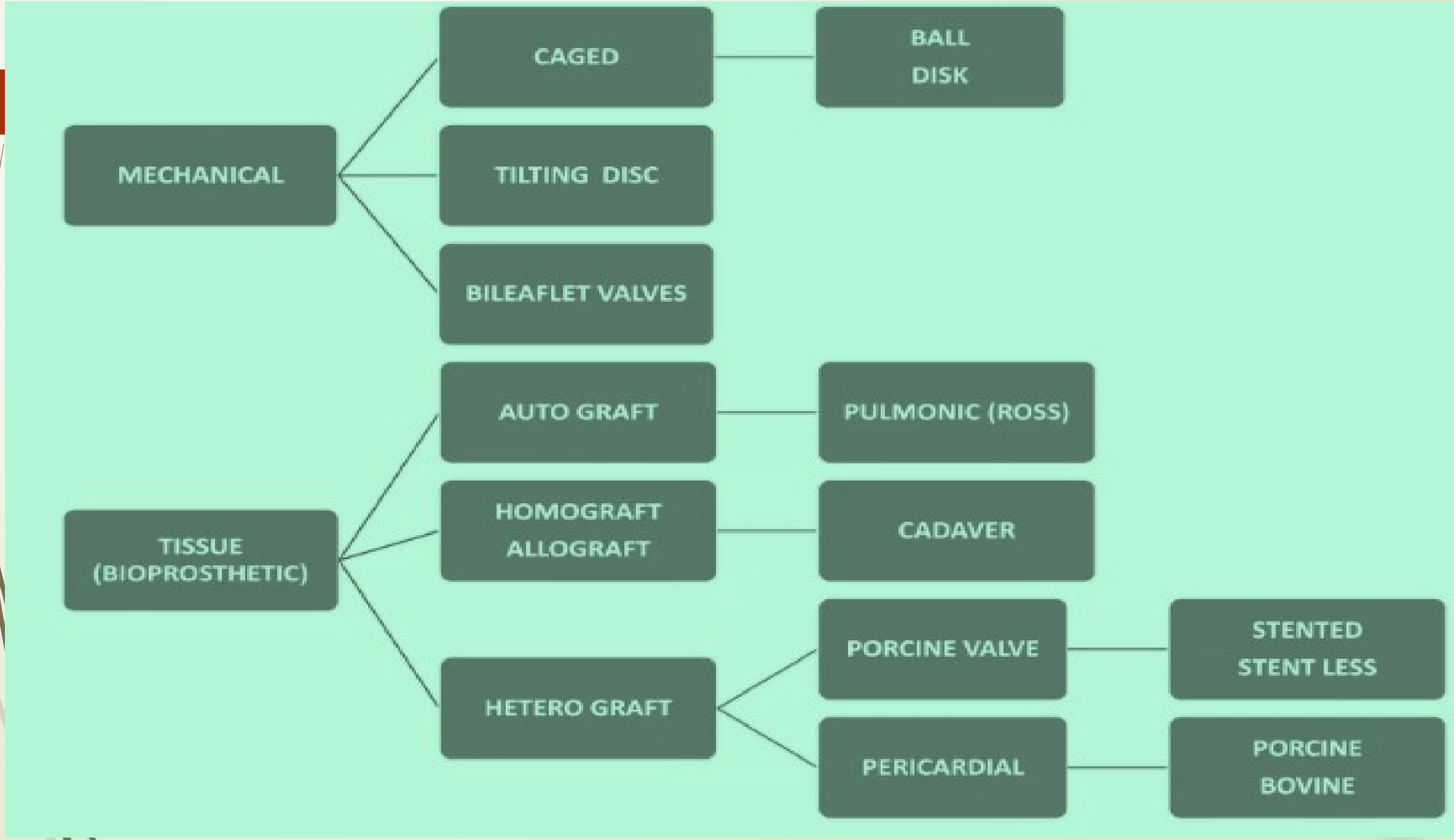
(J) Lotus valve (Boston Scientific, Natick, MA)

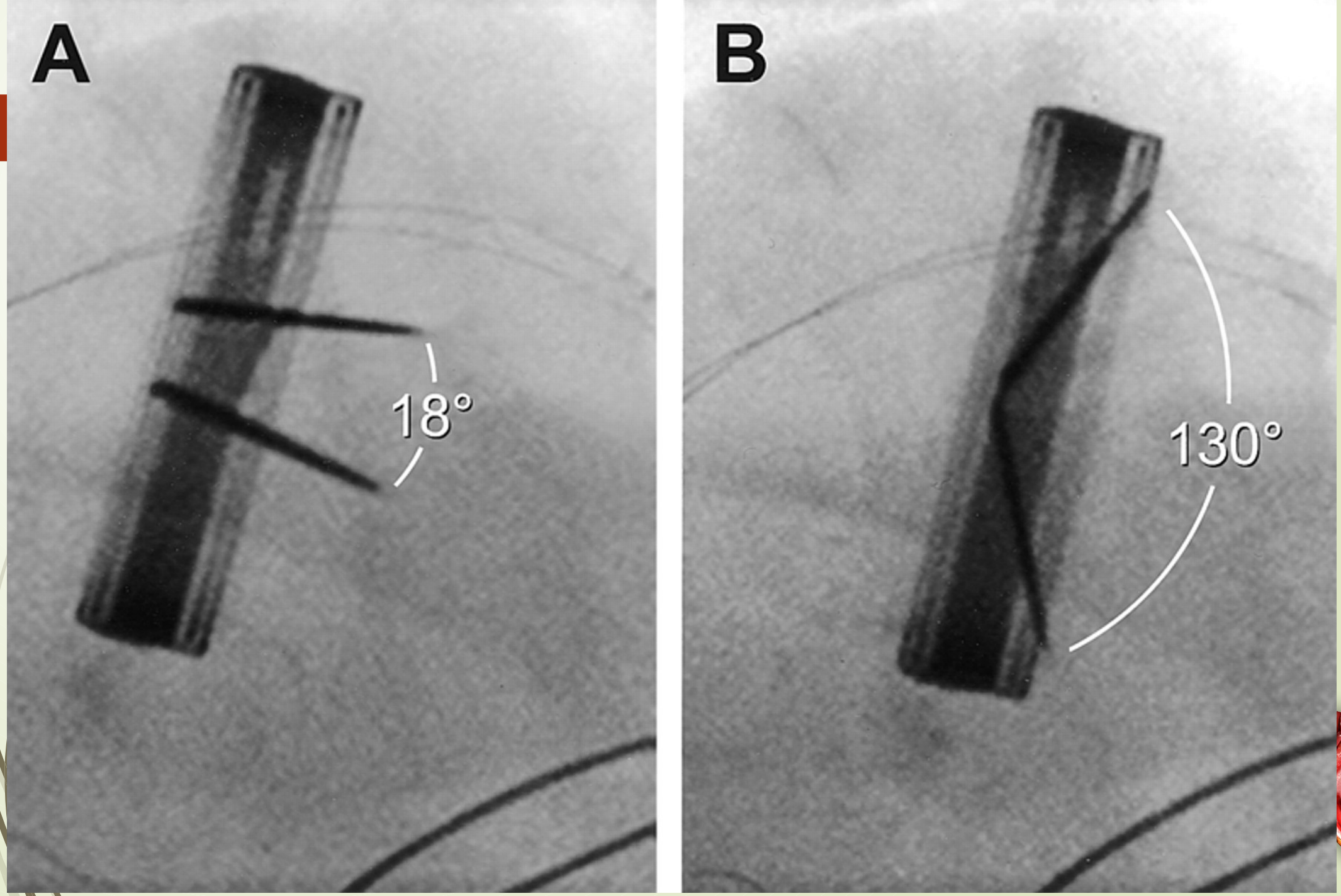
(K) Portico valve (St. Jude Medical)

(L) Acurate symethis valve (Symetis, Lausanne, Switzerland)

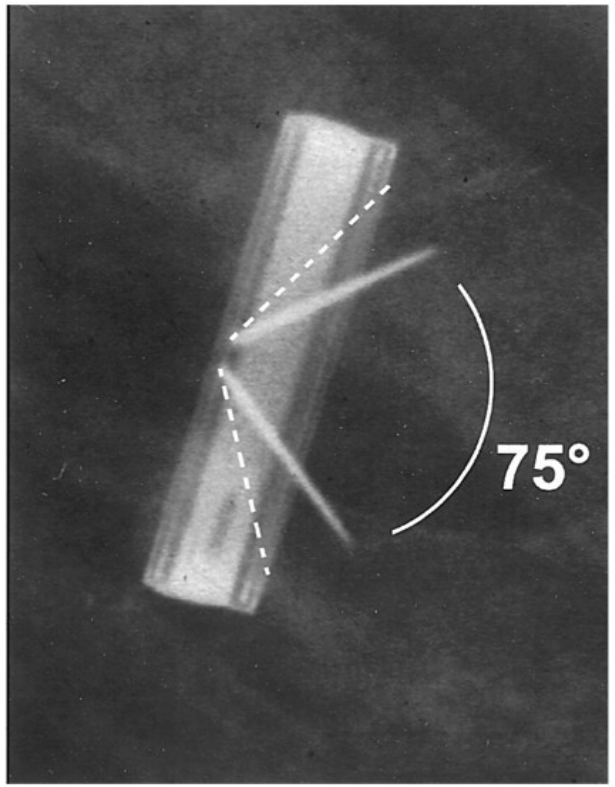



MakeAGIF.com

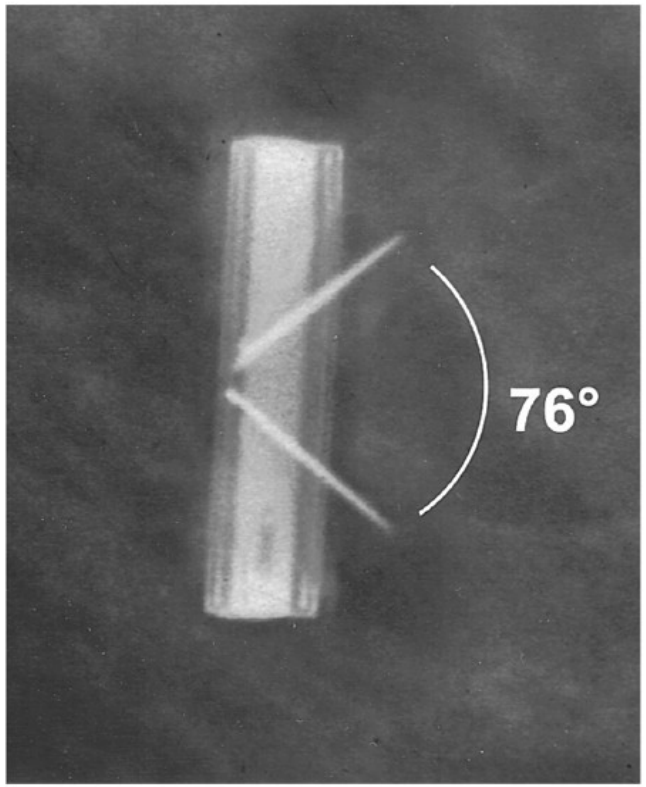

MECHANICAL
CAGED
BALL
DISK
TILTING DISC
BILEAFLET VALVES
TISSUE
(BIOPROSTHETIC)
AUTO GRAFT
PULMONIC (ROSS)
HOMOGRAFT
ALLOGRAFT
CADAVER
HETERO GRAFT
PORCINE VALVE
STENTED
STENT LESS
PERICARDIAL
PORCINE
BOVINE


A
18°
B
130°

10/21/1995

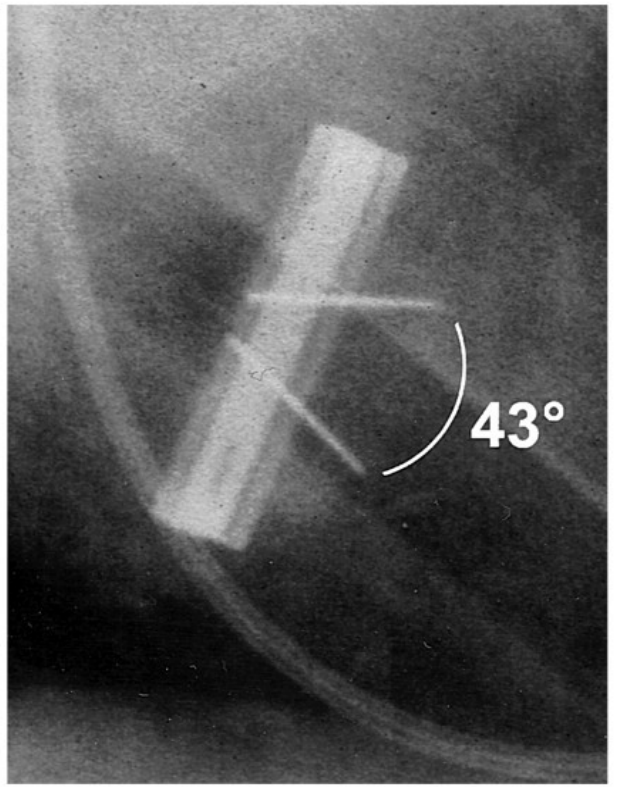


10/25/1996



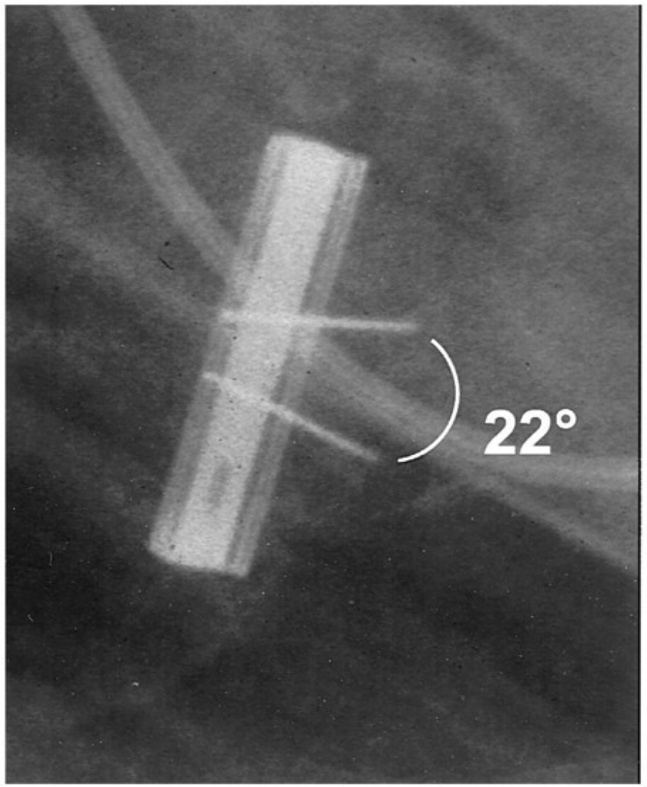
MPG:10 mmHg

After 3 h
rtPA 100 mg

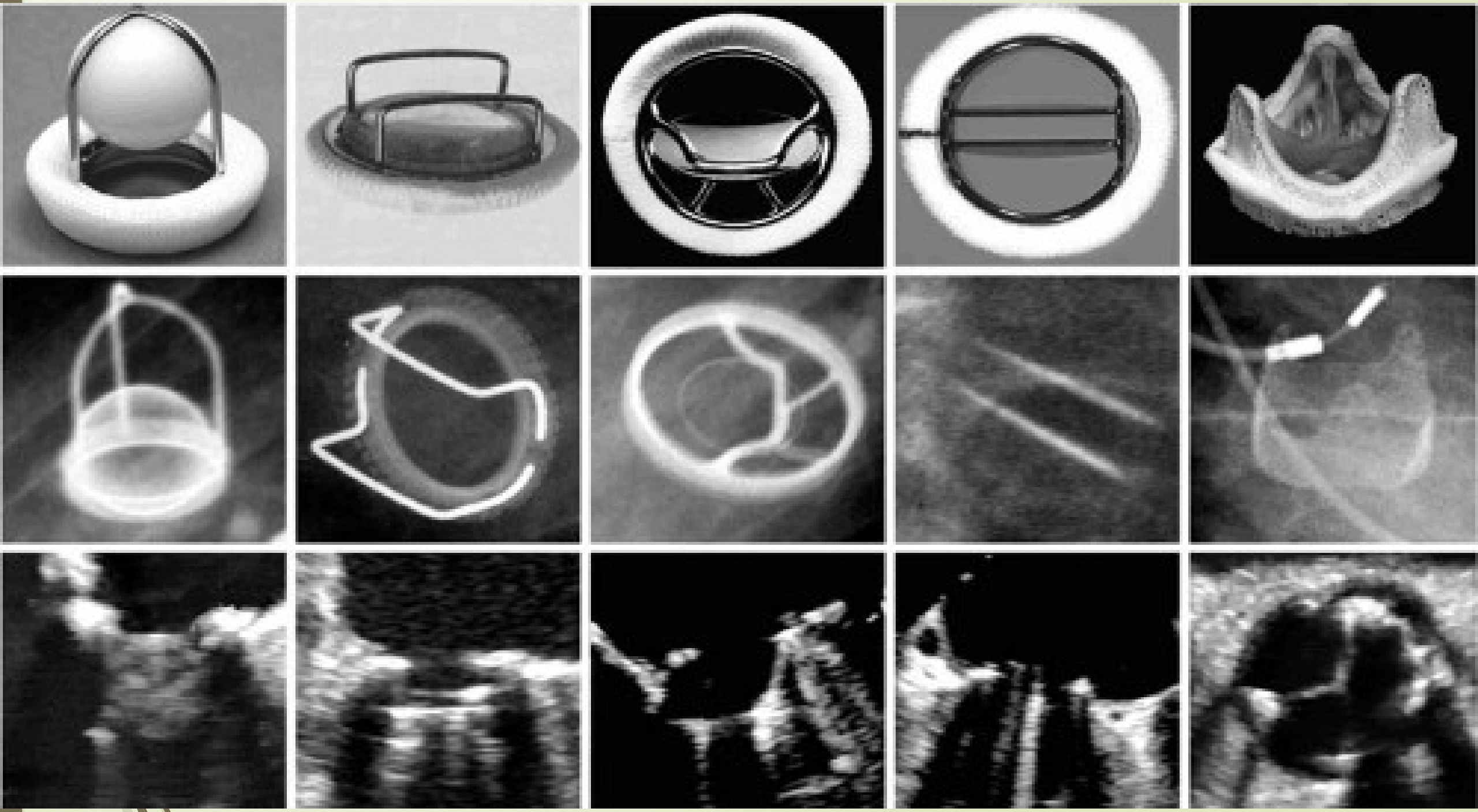


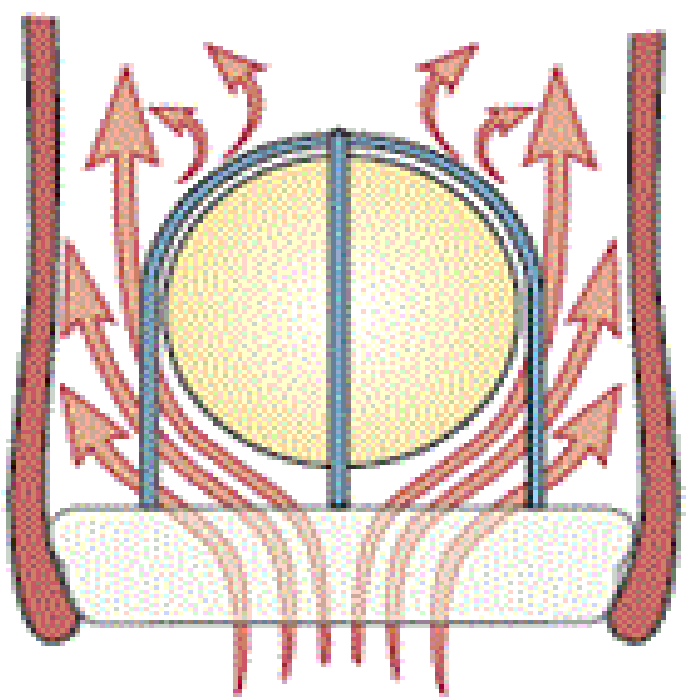
MPG: 6 mmHg

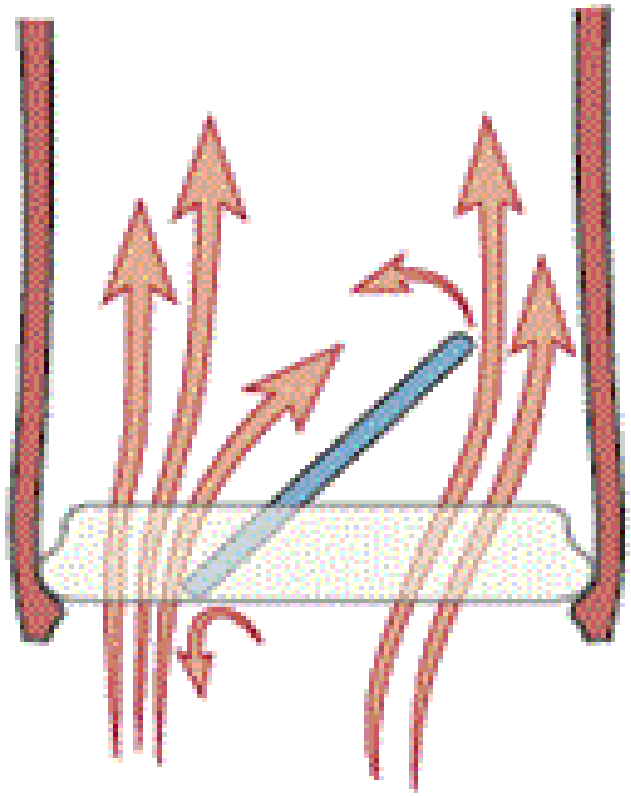
After 24 h
i.v.heparin

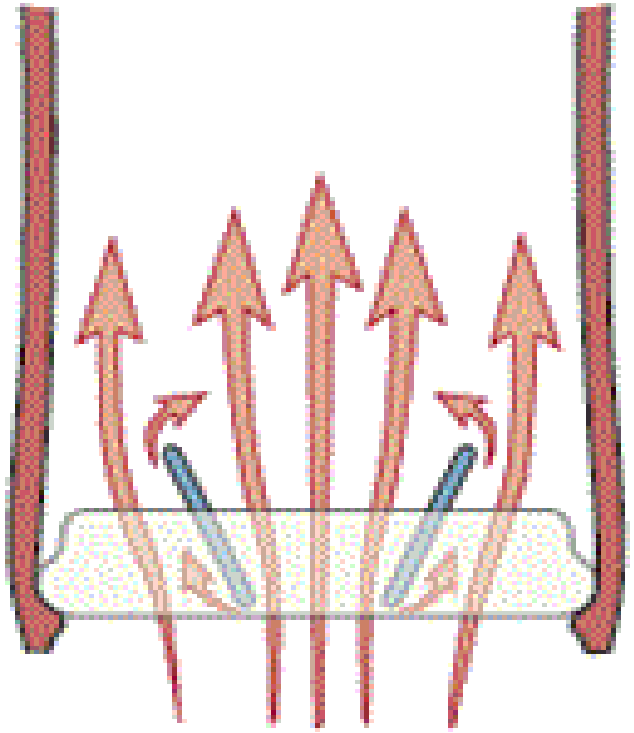


MPG: 5 mmHg

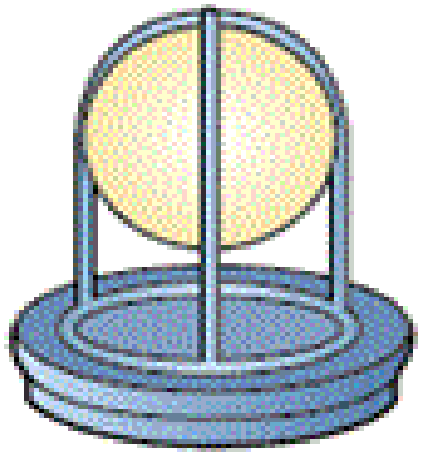


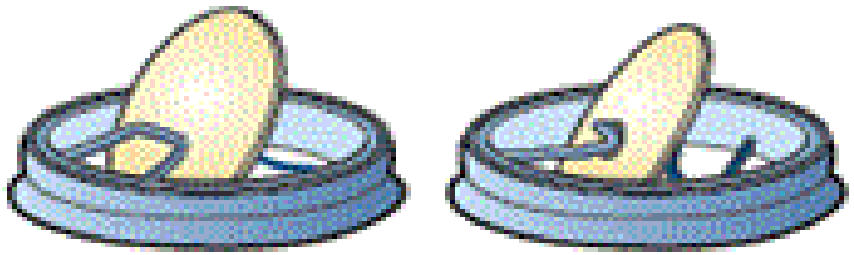


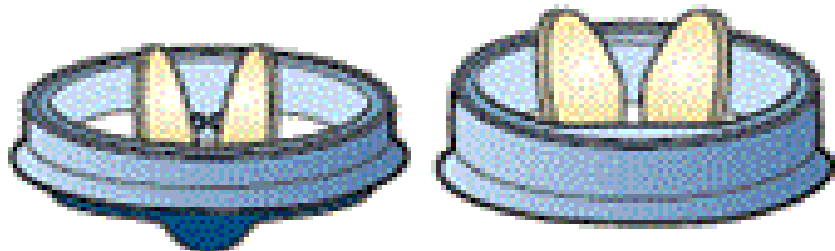




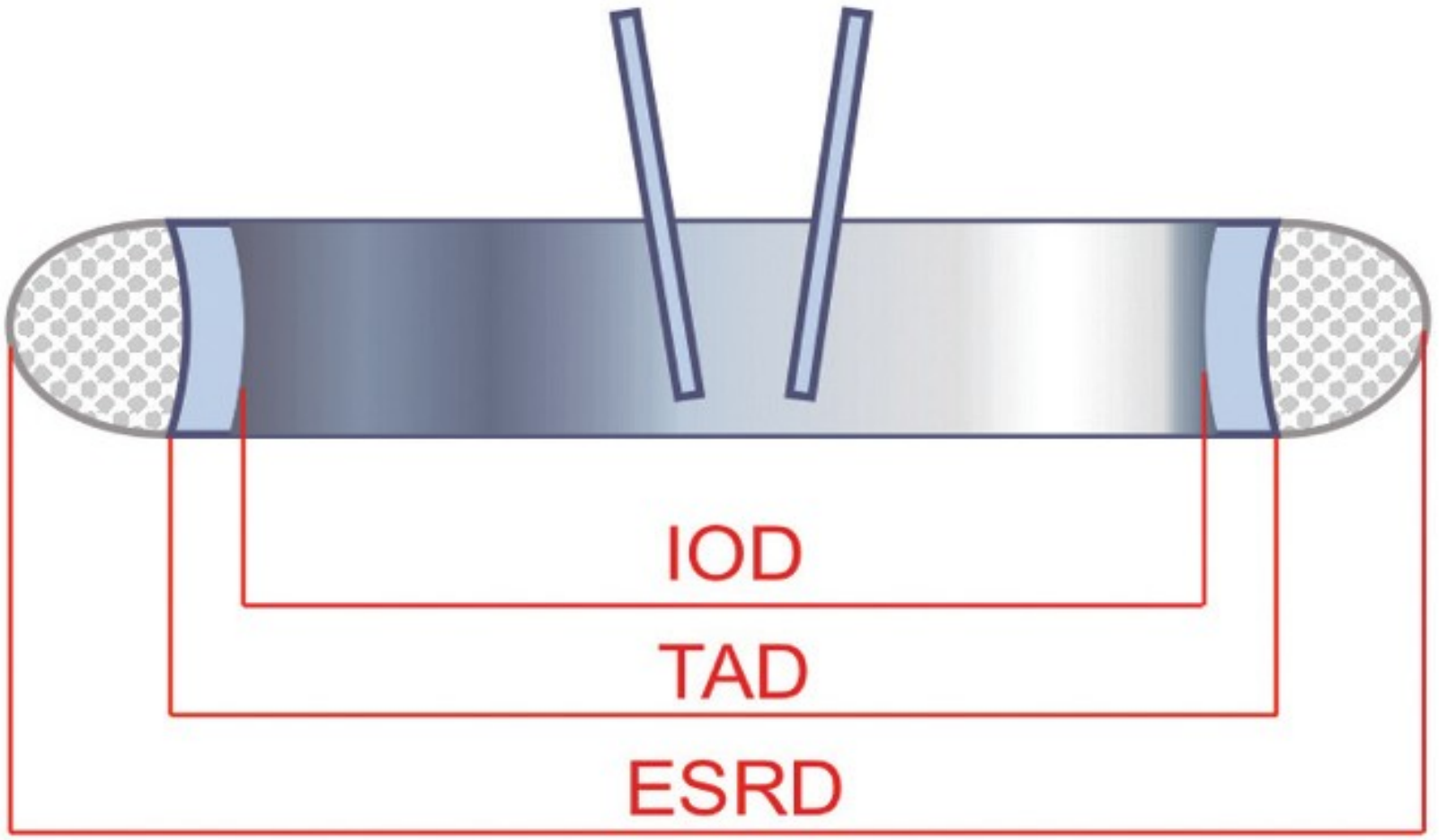
(B)





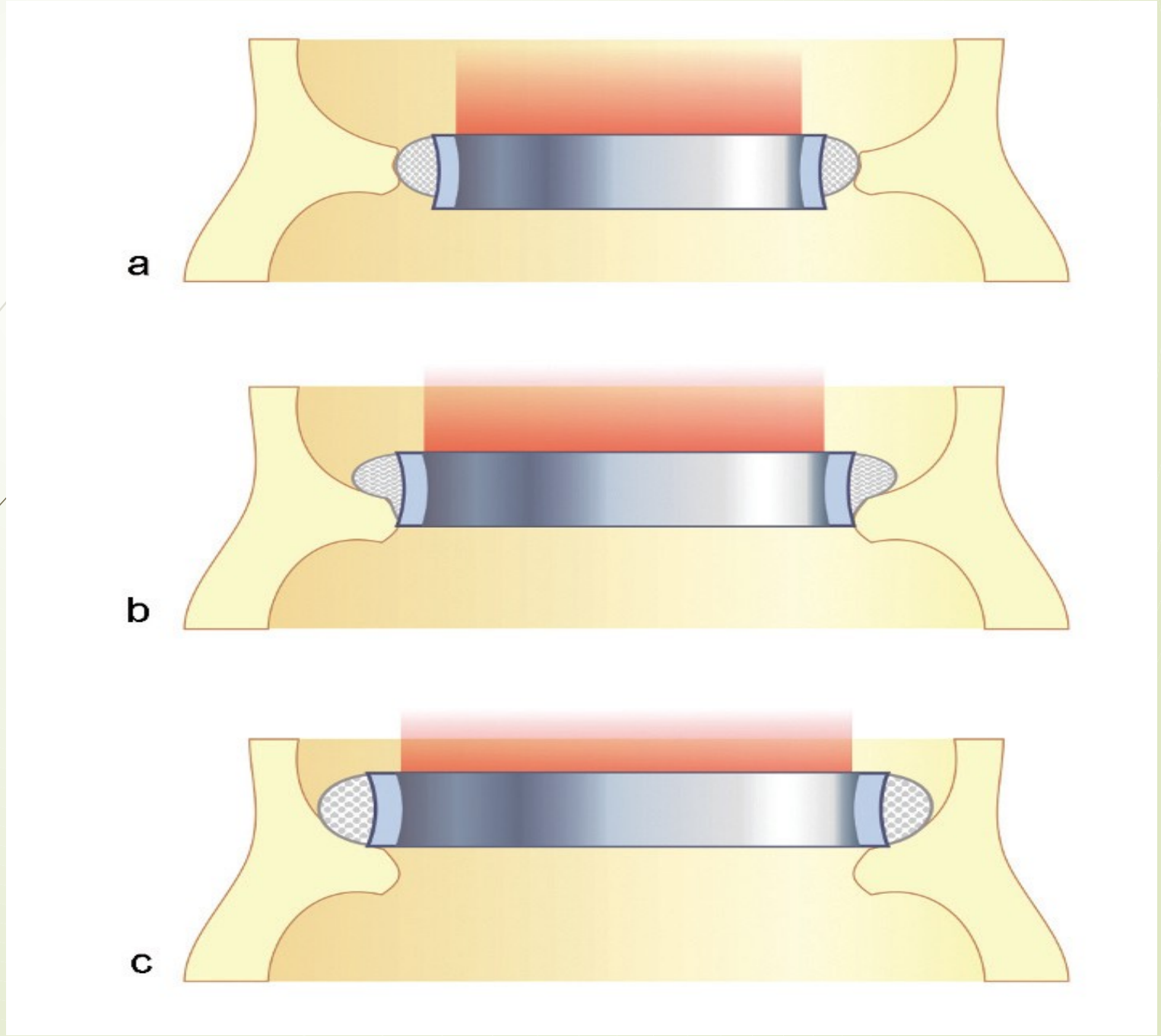


VALVE	OPENING ANGLE	CLOSING ANGLE
SJM	<24	>130
Carbomedics	<29	>148
Sorin Bicarbon	<24	>135
Edwards Duromedic	<13	>120
Medtronic Hall	70-75	-
TTK Chithra	72	-



Valve diameters. *IOD* internal orifi ce diameter, *TAD* tissue annulus diameter, *ESRD* external sewing ring diameter



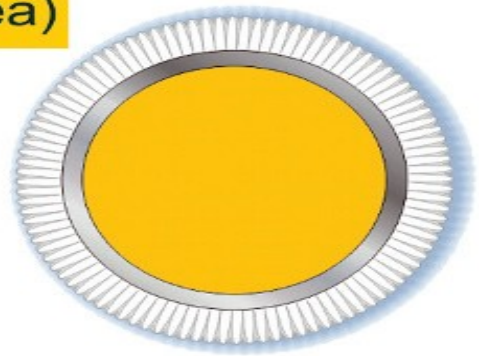

a
b
c



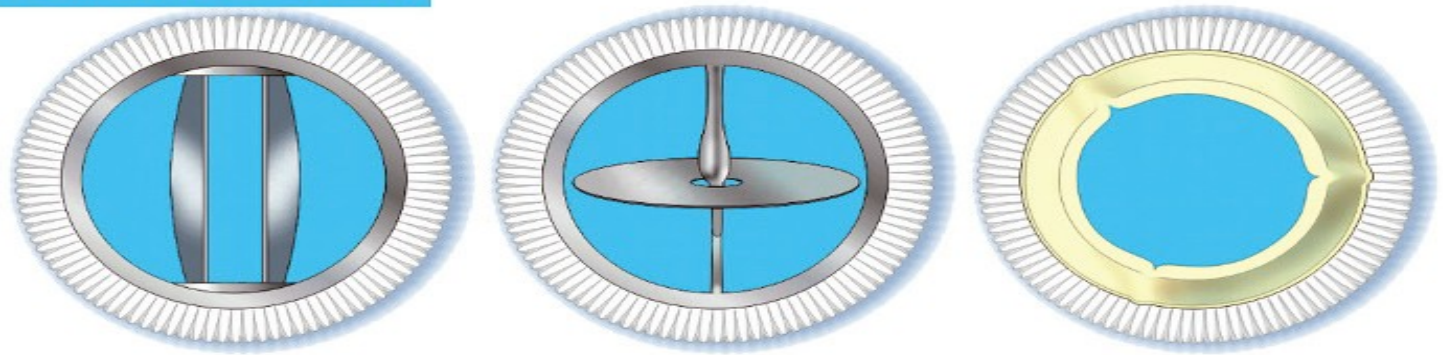
- Geometric Orifice Area
  - The whole inner area of the valve including the area occupied by the opened discs or leaflets, struts, and other mechanisms of the valve
- Clear Orifice Area
  - By subtracting the area of opening components of the valve from GOA
- Effective Orifice Area
  - That portion of the valve orifice area through which the blood really flows



GOA
(geometric orifice area)



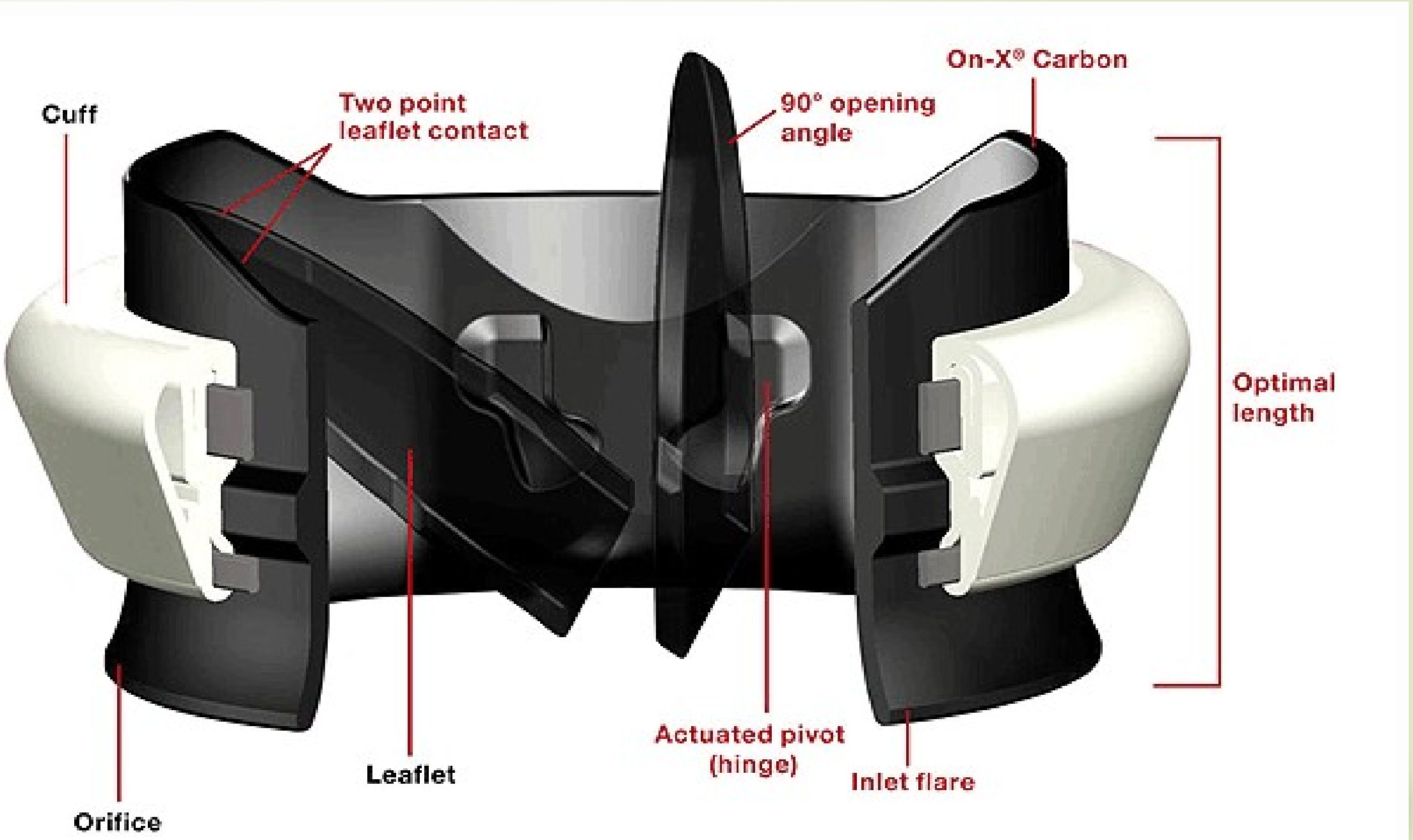
COA
(clear orifice area)



EOA
(effective orifice area)






Cuff
Two point leaflet contact
90° opening angle
On-X® Carbon
Optimal length
Orifice
Leaflet
Actuated pivot (hinge)
Inlet flare

- Prosthetic heart valve is associated with improved survival and relief of symptoms.
  - Valve replacement is not a curative procedure but introduces a new disease process with prosthetic valve-related complications



Prosthesis-related complications

- Structural failure (bioprosthetic valves)
- Thrombosis and thromboembolism
- Pannus formation
- Paraprosthetic regurgitation
- Intravascular haemolysis
- Endocarditis



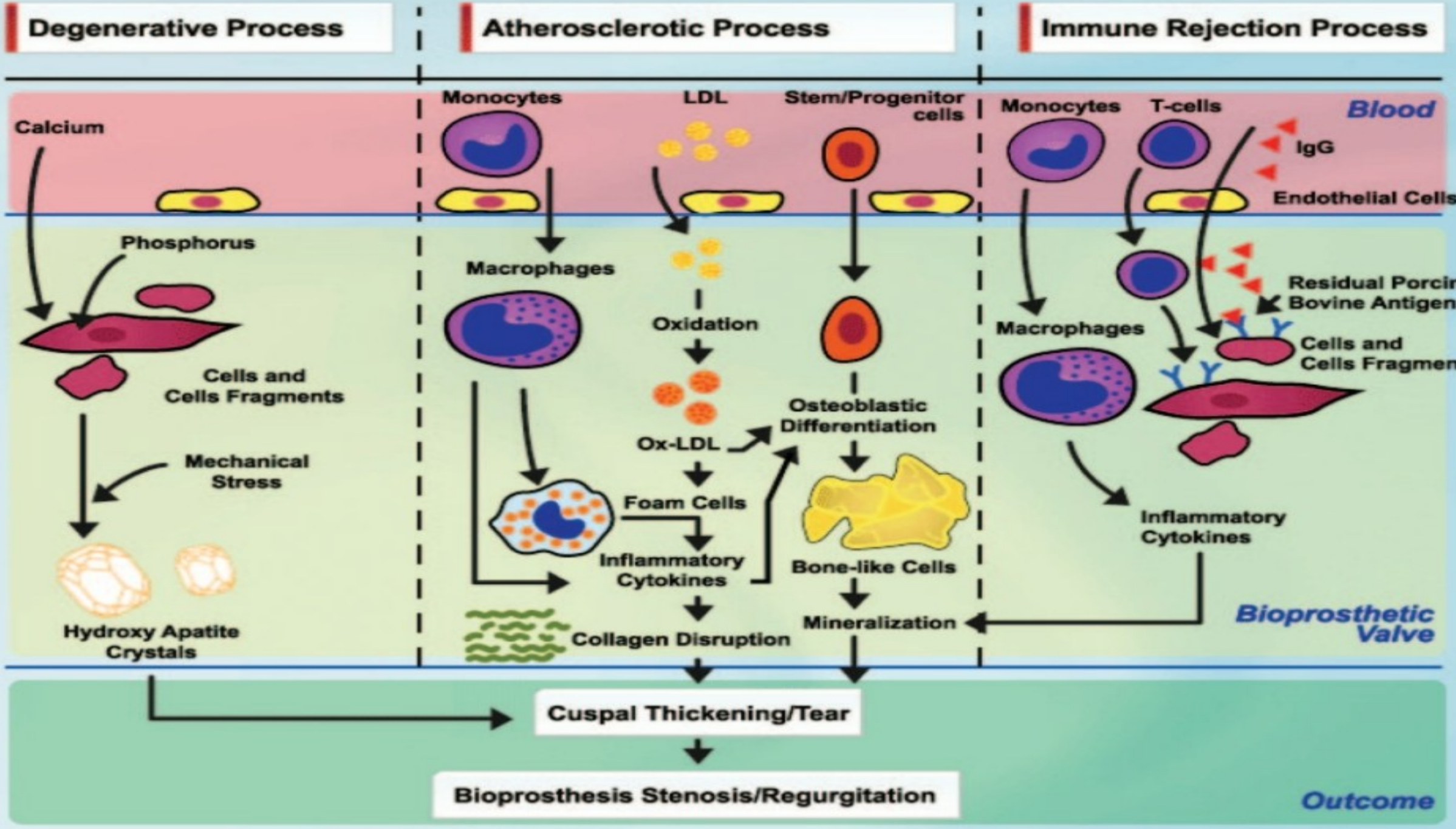
Structural valve degeneration

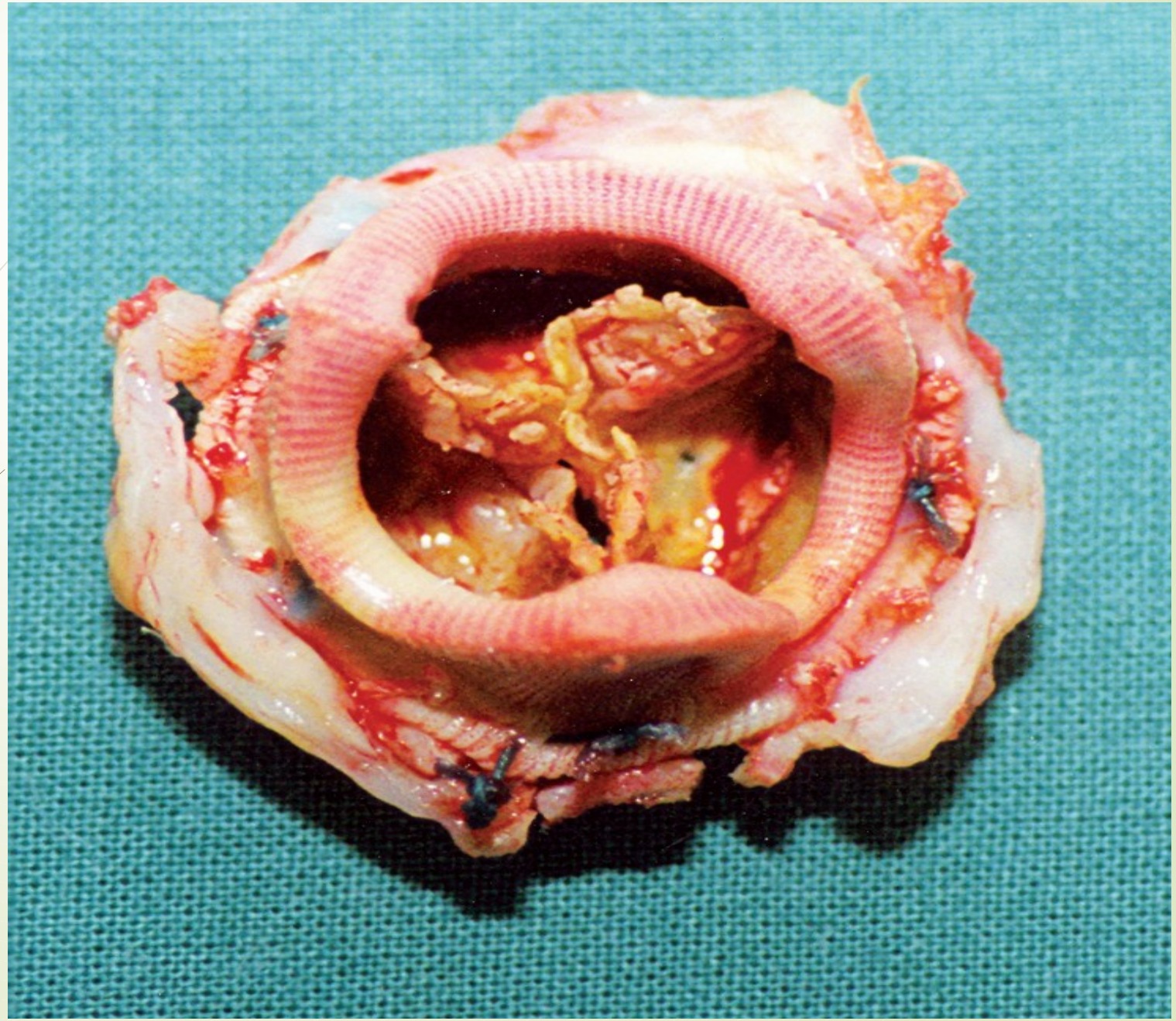
- Progressive deterioration of the leaflets or supporting structures, resulting in thickening, calcification, tearing, or disruption of the prosthetic valve with eventual hemodynamic dysfunction, manifesting as stenosis, regurgitation or a combination of both
- Types (overlapping processes)
  - Leaflet calcification
  - Non-calcific tissue degeneration
- Active disease process at the cellular and molecular levels



- Accelerated calcification
  - Young age
  - End-stage renal disease
  - Disorders of the calcium metabolism
- Mitral >> Aortic
  - Systole vs Diastole
- Glutaraldehyde fixation
- Host, Mechanical and Valve factors




Degenerative Process
Atherosclerotic Process
Immune Rejection Process
Blood
Calcium
Monocytes
LDL
Stem/Progenitor cells
Monocytes
T-cells
IgG
Endothelial Cells
Phosphorus
Macrophages
Oxidation
Macrophages
Residual Porcine/ Bovine Antigens
Cells and Cells Fragments
Cells and Cells Fragments
Osteoblastic Differentiation
Ox-LDL
Mechanical Stress
Foam Cells
Inflammatory Cytokines
Bone-like Cells
Inflammatory Cytokines
Hydroxy Apatite Crystals
Collagen Disruption
Mineralization
Bioprosthetic Valve
Cuspal Thickening/Tear
Bioprosthesis Stenosis/Regurgitation
Outcome



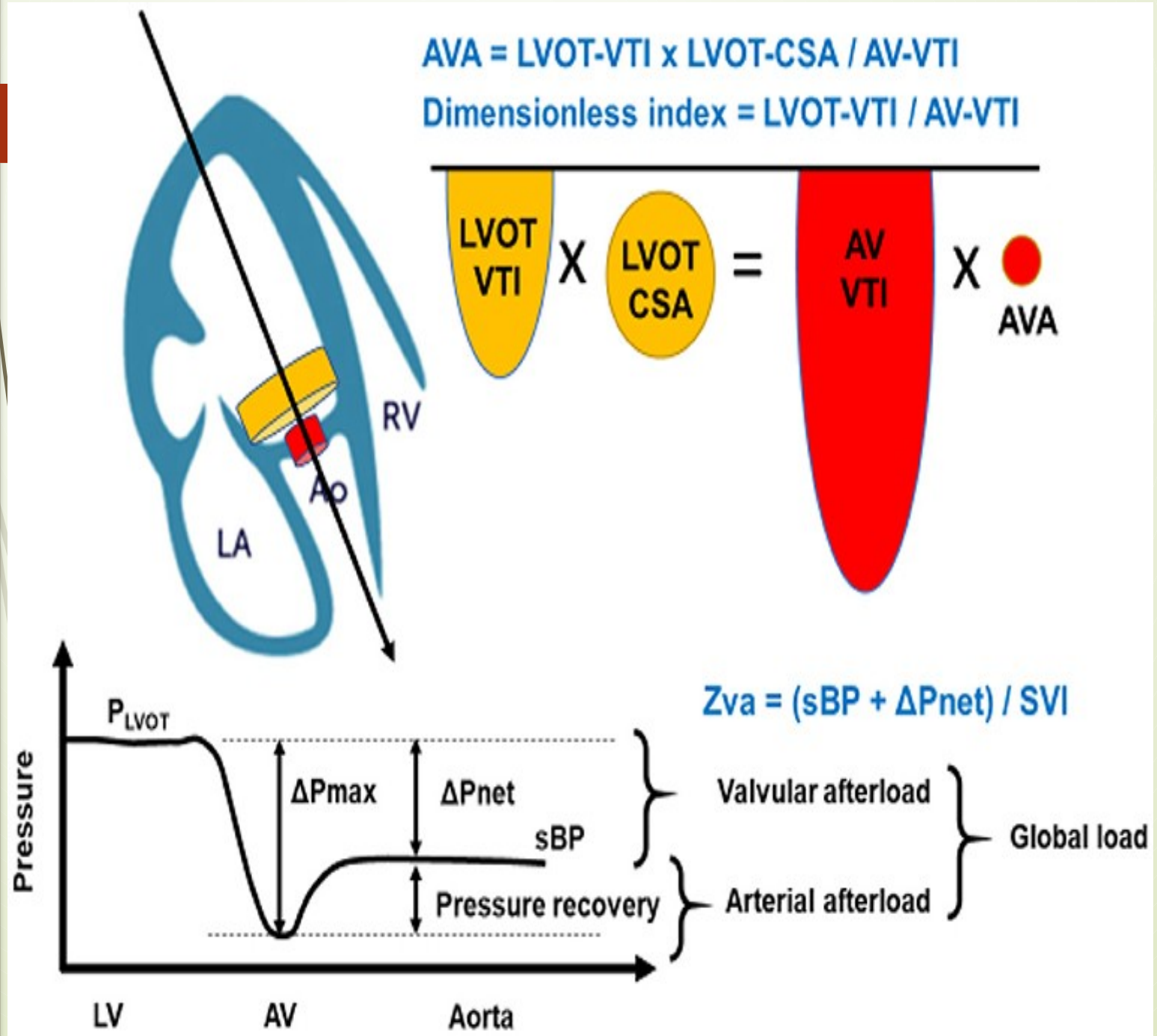


- Symptom onset can be acute or progressive depending on the rate and degree of valve dysfunction
- Physical examination is less sensitive
- No single echocardiographic parameter can accurately assess prosthetic valve function
- The more parameters are abnormal, the more the probability of prosthetic valve obstruction increases



- American Society of Echocardiography (ASE)
  - Possible aortic BPV stenosis
    - Peak prosthetic aortic jet velocity 3-4 m/s
    - Mean gradient 20-35 mmHg
    - EOA 0.8-1.2 cm^2
    - Doppler velocity index (DVI) 0.25-0.29.
  - Significant aortic BPV stenosis
    - Peak prosthetic jet velocity >4 m/s
    - Mean gradient >35 mmHg
    - EOA <0.8 cm^2
    - DVI <0.25



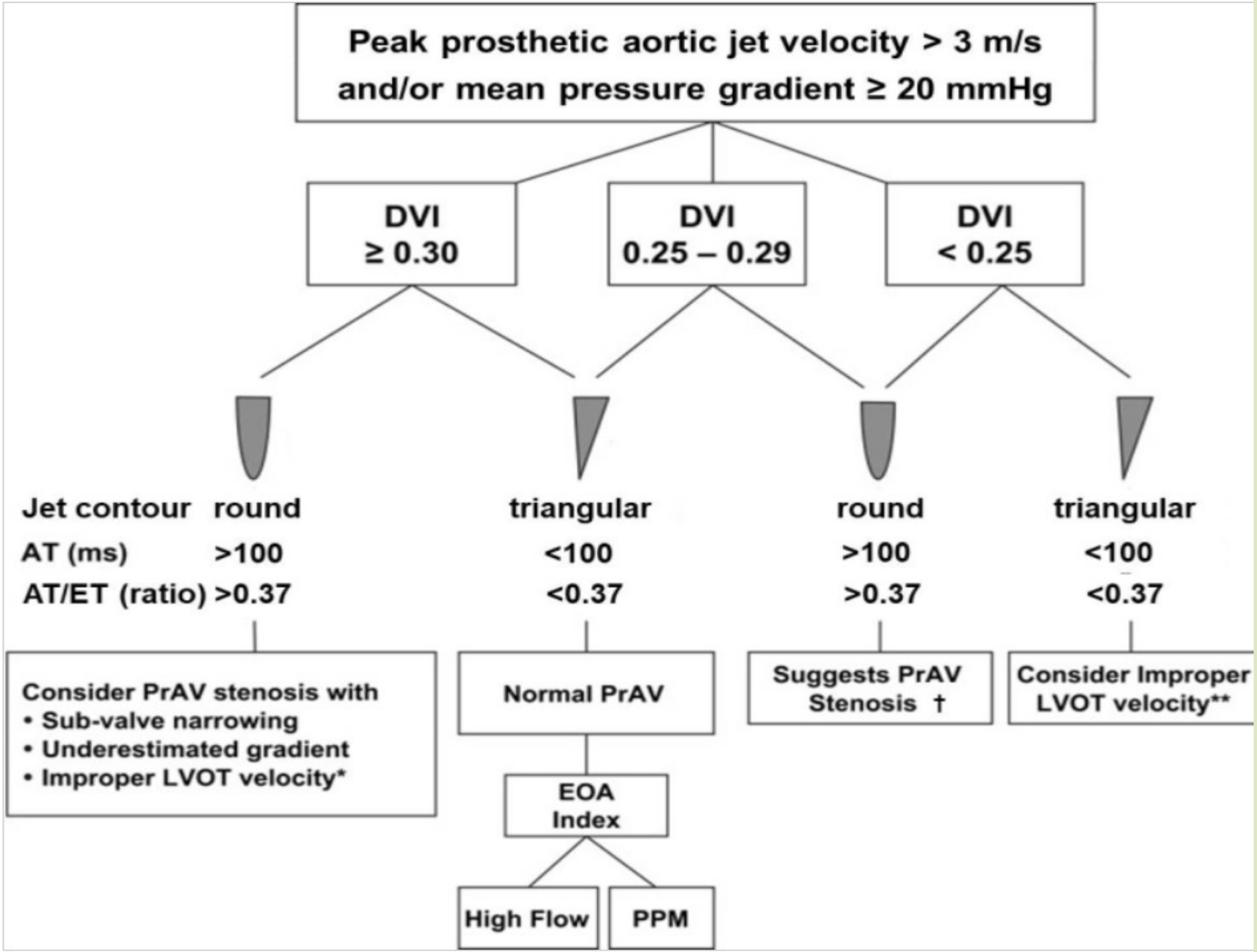


Valvulo-arterial impedance (Zva)



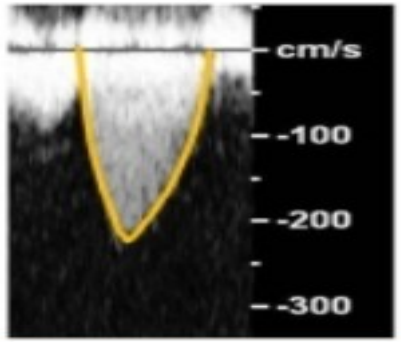
- European Association for Cardiovascular Imaging (EACVI)
  - Increase in mean gradient at follow-up
    - Possible obstruction with an increase in mean gradient of 10-19 mmHg
    - Significant obstruction with an increase ≥20 mmHg




Peak prosthetic aortic jet velocity > 3 m/s
and/or mean pressure gradient ≥ 20 mmHg
DVI
≥ 0.30
DVI
0.25 – 0.29
DVI
< 0.25
Jet contour
AT (ms)
AT/ET (ratio)
round
>100
>0.37
triangular
<100
<0.37
round
>100
>0.37
triangular
<100
<0.37
Consider PrAV stenosis with
• Sub-valve narrowing
• Underestimated gradient
• Improper LVOT velocity*
Normal PrAV
EOA
Index
High Flow
PPM
Suggests PrAV
Stenosis †
Consider Improper
LVOT velocity**

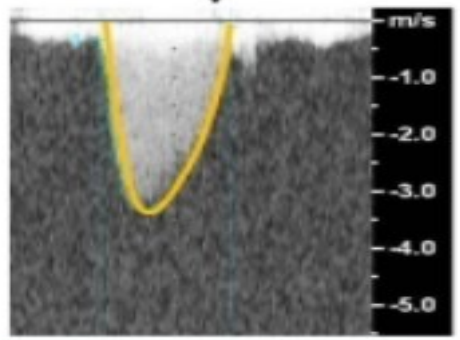


Signs (high gradient) or symptoms suggesting aortic prosthetic valve stenosis



- Normal valve structure and motion
- V_{max} < 3 m/s
- Mean ΔP < 20 mmHg
- Change in mean ΔP during FU < 10 mmHg
- EOA > 1.2 cm^2
- Indexed EOA > 0.85 cm^2/m^2
- DVI ≥ 0.35
- EOA similar to normal reference EOA (±0.3 cm^2)
- AT < 80 ms
- AT/LVET < 0.32

Normal Prosthetic Valve Function



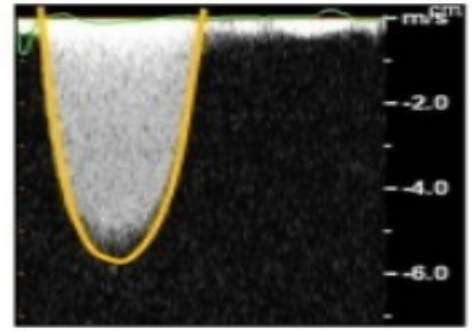
- V_{max} 3-4 m/s
- Mean ΔP 20-34 mmHg
- EOA 0.8-1.2 cm^2
- Indexed EOA ≤ 0.85 cm^2/m^2
- DVI 0.25-0.34

- Normal valve structure and motion
- Change in mean ΔP during FU< 10 mmHg
- EOA similar to normal reference EOA (±0.3 cm^2)
- AT/LVET < 0.32

Prosthesis-Patient Mismatch or High Flow States

- Abnormal valve structure and motion
- Increase in during FU ≥ 10 mmHg
- Decrease in EOA during FU ≥ 0.3 cm^2 (or ≥ 25%)
- EOA < normal EOA (-0.3 cm^2)
- Decrease in DVI during FU ≥ 0.10 (or ≥ 20%)
- AT/LVET > 0.32

Mild-to-Moderate Stenosis



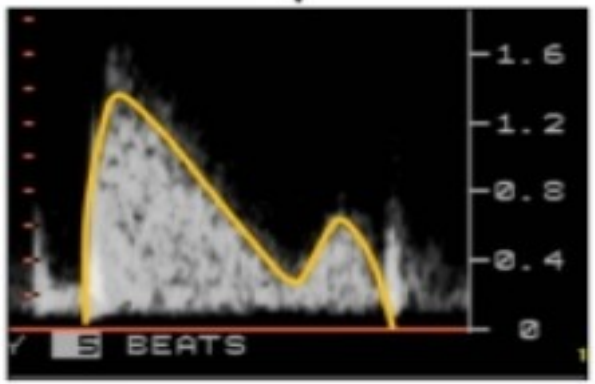
- Abnormal valve structure and motion
- V_{max} ≥ 4 m/s
- Mean ΔP ≥ 35 mmHg
- Increase in mean gradient during FU ≥ 20 mmHg
- EOA < 0.8 cm^2
- Decrease in EOA during FU ≥ 0.6 cm^2 (or ≥ 50%)
- EOA < normal EOA (-0.6 cm^2)
- Indexed EOA ≤ 0.65 cm^2/m^2
- DVI < 0.25
- Decrease in DVI during FU ≥ 0.20 (or ≥ 40%)
- AT > 100 ms
- AT/LVET > 0.37

Significant Stenosis

- For mitral prosthetic valvess, a peak transvalvular velocity >2.5 m/s, mean gradient >10 mmHg or pressure half-time (PHT) >200 ms provides a strong argument for significant obstruction
  - The proposed mean pressure gradient is only valid for normal ventricular rates (between 60 and 80 bpm) because an increase in heart rate is accompanied by a disproportionate decrease in the diastolic filling period and a consecutive increase in the pressure gradient across the mitral valve
  - PHT cannot be used to calculate EOA since it has only been validated for native mitral valve stenosis.
    - EOA should be calculated using continuity equation

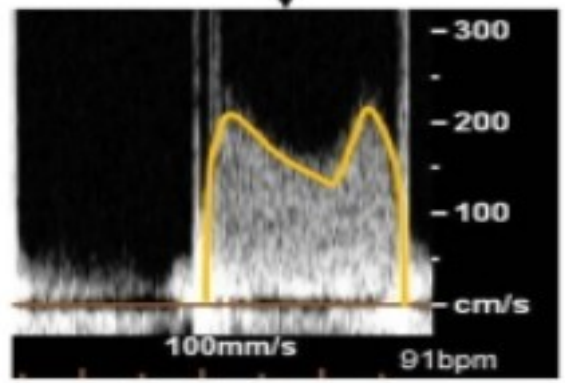


Signs (high gradient) or symptoms suggesting mitral prosthetic valve stenosis



- Normal valve structure and motion
- V_{max} < 1.9 m/s
- Mean ΔP ≤ 5 mmHg
- Increase in mean ΔP during FU < 3 mmHg
- EOA ≥ 2 cm^2
- Indexed EOA > 1.2 cm^2/m^2
- DVI < 2.2
- PHT < 130 ms

Normal Prosthetic Valve Function



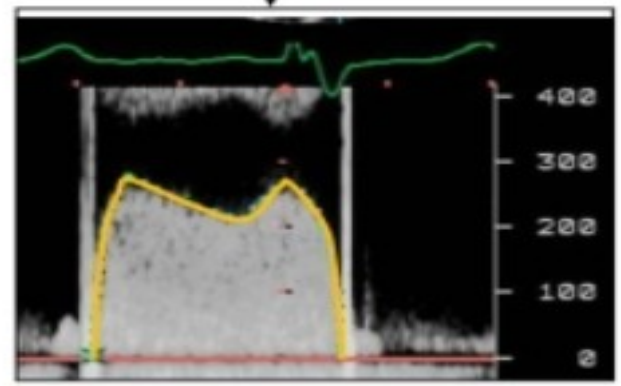
- V_{max} 1.9-2.5 m/s
- Mean ΔP 6-10 mmHg
- EOA 1.0-1.9 cm^2
- Indexed EOA ≤ 1.2 cm^2/m^2
- DVI 2.2-2.5
- PHT 130-200 ms

- Normal valve structure and motion
- Increase in mean ΔP during FU < 3 mmHg
- EOA similar to normal reference EOA (±0.3 cm^2)

Prosthesis-Patient Mismatch or High Flow States

- Abnormal valve structure and motion
- Increase in mean ΔP during FU ≥ 3 mmHg
- EOA < normal EOA (-0.3 cm^2)

Possible Stenosis

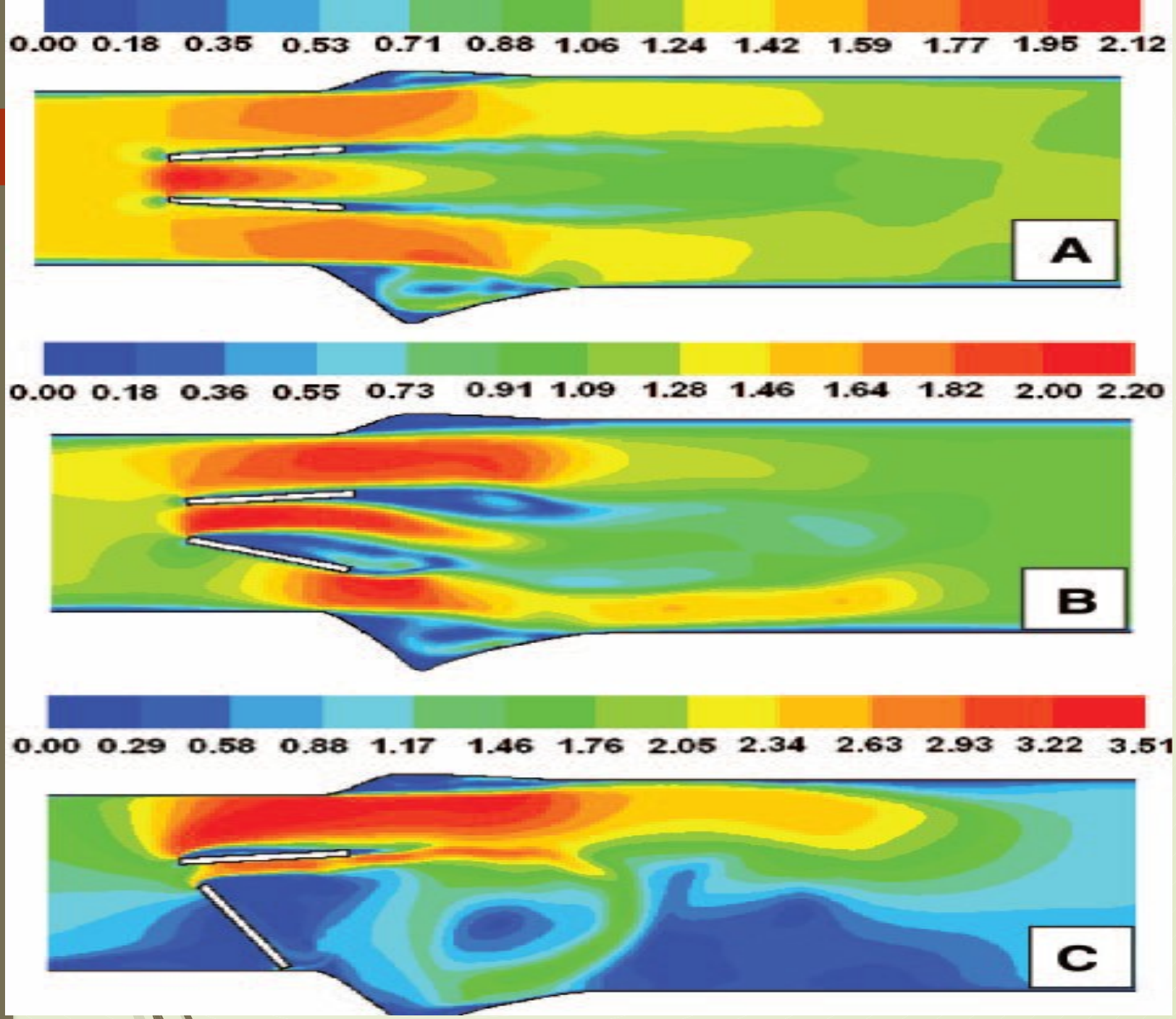


- Abnormal valve structure and motion
- V_{max} ≥ 2.5 m/s
- Mean ΔP ≥ 10 mmHg
- Increase in mean ΔP during FU ≥ 5 mmHg
- EOA < 1 cm^2
- EOA < normal EOA (-0.6 cm^2)
- Indexed EOA ≤ 0.9 cm^2/m^2
- DVI > 2.5
- PHT > 200 ms

Significant Stenosis

- Occasionally, an abnormally high jet gradient is recorded in bileaflet mechanical prostheses regardless of the position (mitral or aortic) because the smaller central orifice jet has a higher velocity than the jets in both larger outside orifices.
  - This phenomenon may lead to underestimation of the EOA and a false suspicion of prosthesis obstruction.





Numerical simulation showing the flow velocity distribution in bileaflet mechanical valves at a cardiac output of 5 L/min.



Bioprosthetic Valve Thrombosis

- More frequently in transcatheter than in surgical valves
- large BPV and sinus of Valsalva, possibly with resultant slower blood flow and stasis in the valve periphery that could contribute to thrombosis
- Hypo-attenuated leaflet thickening (HALT) of aortic bioprosthetic valves associated with reduced leaflet motion (RELM) is the hallmark finding of subclinical leaflet thrombosis using four-dimensional, volume-rendered CT
    - TEE has 100% concordance
- Paucity of colour Doppler flow by TTE within a region of the BPV in the absence of artefacts may be a clue to underlying HALT in patients with unremarkable transprosthetic gradients.
- Initiation of anticoagulation with either VKA or NOACs usually results in thrombus resolution and restoration of valve function



- The incidence of pannus formation causing prosthetic obstruction is similar in biological and mechanical prosthetic valves
- If the diagnosis of valve dysfunction is still in question
  - TEE, cinefluoroscopy and MDCT



Treatment of Mechanical Prosthetic Valve Obstruction

- Heart valve team
  - Intensified antithrombotic therapy
  - Thrombolytics or
  - Repeat surgery



Thromboembolic and Bleeding Complications

- Estimated incidence of clinical events ranging from 0.6% to 2.3% per patient-year.
- The risk of thromboembolic complications is similar for patients with mechanical valves on warfarin therapy and bioprosthetic valves without warfarin therapy.
- Risk factors
  - prosthesis type
  - valve position
  - thrombogenicity,
  - patient risk factors, and
  - antithrombotic treatment.



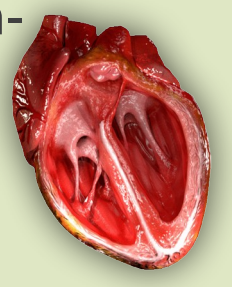
Treatment of prosthetic valve regurgitation

- Heart failure symptoms or haemolysis with refractory anaemia
  - Redo open heart surgery with either suture repair or repeat valve replacement
- High or prohibitive surgical risk, a transcatheter closure of paravalvular leak
- Degenerated BPVs and transprosthetic valvular regurgitation are candidates for transcatheter valve-in-valve (VIV) implantation



Infective endocarditis

- The incidence of prosthetic valve endocarditis is 0.5% per patient-year, even with appropriate antibiotic prophylaxis
- High mortality rates (30% to 50%).
- Blood cultures and echocardiographic evidence of prosthetic infection, including vegetations, paraprosthetic abscesses, or a new paravalvular regurgitation.
- Despite prompt and appropriate antibiotic treatment, many patients with prosthetic valve endocarditis will eventually require surgery.
- Medical treatment alone is more likely to succeed in late prosthetic valve endocarditis (occurring 6 months after surgery) and in non-staphylococcal infections



- Surgery is indicated:
  - failure of medical treatment;
  - hemodynamically significant prosthesis regurgitation, especially if associated with deterioration of LV function;
  - large vegetations; and
  - development of intracardiac fistulas.



Patient-Prosthesis Mismatch

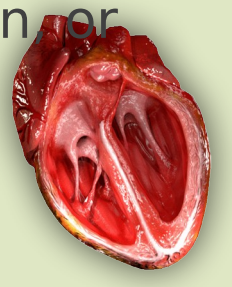
- Functional area of the prosthetic valve is too small for the cardiac demands in that patient

Table 3. Threshold Values of Indexed Prosthetic Valve EOA for the Identification and Quantification of PPM

	Mild or Not Clinically Significant, cm^2/m^2	Moderate, cm^2/m^2	Severe, cm^2/m^2
Aortic position	>0.85 (0.8–0.9)	≤0.85 (0.8–0.9)	≤0.65 (0.6–0.7)
Mitral position	>1.2 (1.2–1.3)	≤1.2 (1.2–1.3)	≤0.9 (0.9)

Hemolytic Anemia

- 50% to 95% of patients with mechanical valves, there is some evidence of intravascular hemolysis.
- Hemolysis may occur with either central or paravalvular regurgitation due to rapid acceleration and deceleration or high-peak shear rates
- Most patients with mild anemia can be treated conservatively with iron and folate replacement therapy.
- Repeat valve surgery or surgical repair of a paravalvular leak may be needed when prosthetic valve hemolysis is associated with severe refractory anemia, significant paravalvular regurgitation, or high-output failure.



Postperfusion and Postpericardiotomy Syndromes



Postperfusion Syndrome

- /t occurs in **3** to 10 percent of patients undergoing cardiopulmonary **bypass.**
- **CMV IS IMPLICATED**
- Fever, splenomegaly and atypical lymphocytosis
- Occurs 1 to 8 weeks after operation, most commonly in the 3rd or 4th week.
- Fever usually appears first, followed by splenomegaly in about 1 week and the appearance of atypical lymphocytes after another 1 or 2 weeks.
- Patients are never very ill and commonly feel quite well.
- Received substantial amounts of fresh blood, usually with cardiopulmonary bypass but occasionally without.



- Temperature is normal or near normal each morning and rises to a late afternoon peak of 100’ to 103^0 F
- Atypical lymphocytes with fenestrated, eccentric or indented nuclei, perinuclear pallor and plasmacytoid cytoplasm eventually comprise 3 to 60 percent of the lymphocytes in the peripheral blood.
- Fever gradually subsides in 1 to 4 weeks and is rarely associated with malaise, anorexia or toxicity after the first few days.
- Splenomegaly and atypical lymphocytes may disappear with the fever but commonly persist for several months.



Postpericardiotomy Syndrome

- fever and pleuropericarditis
- Immunologic response to damaged autologous tissue within the pericardium
- Most commonly appear in the 2nd or 3rd week after operation but may appear as early as 1 week or as late as 5 to 10 months
- Fever and chest pain
  - Sharp or knifelike, precordial or substernal, aggravated by recumbency and deep breathing and relieved by sitting upright.
- Neutrophilic leukocytosis,
- S-T segment elevation and T wave inversion



- No long term sequelae, and neither chronic adhesive nor constrictive pericarditis has been observed
- Anti-inflammatory drugs
- Drainage

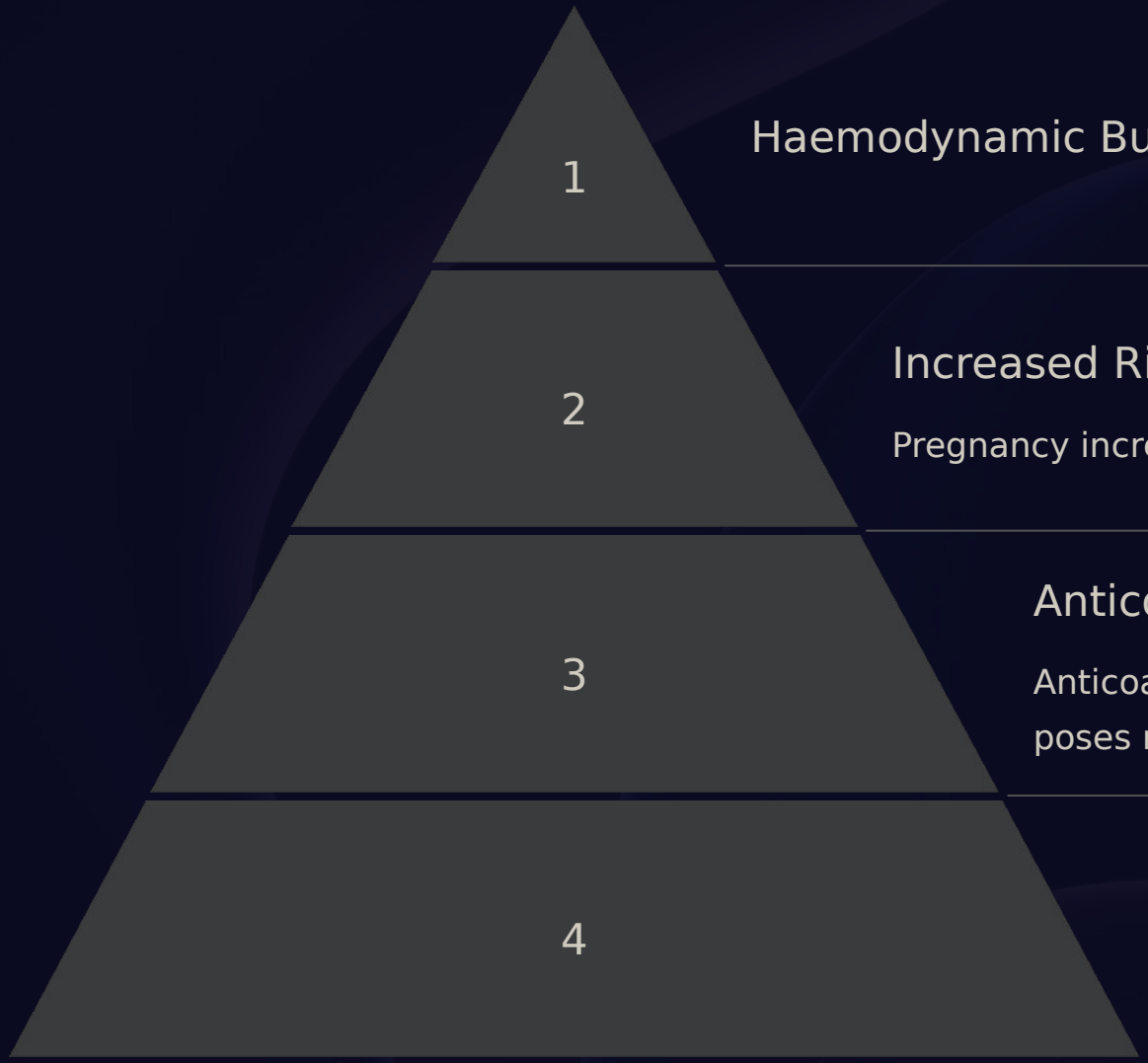


LV Pseudoaneurysm

- 0.5% to 2.0% of cases
- Types
  - Posterior annular suture line, and consists of a disruption in the integrity of the basal LV wall with escape of blood outside the heart contained by pericardial adhesions.
  - Mitral aortic intervalvular fibrosa.
    - Fibrous tissue between the posterior aortic root and base of the anterior mitral leaflet becomes dilated, creating a blind pouch that lies between the aorta and left atrium, which communicates with the LV.
    - Rupture can lead to aortic or mitral regurgitation, or rupture into the pericardial space.



Prosthetic Valves in Pregnancy



Haemodynamic Burden

Increased Risk

Pregnancy increases the risk of heart failure, thrombosis, and other complications.

Anticoagulation

Anticoagulation is essential for women with mechanical valves, but poses risks for both mother and fetus.

SVD

Bioprosthetic valves in young women carry a higher risk of SVD, potentially requiring re-operation.

Made with Gamma

Future Directions in Prosthetic Valve Development

1

Improved Hemodynamics

Ongoing research focuses on developing valves that more closely mimic the natural hemodynamics of native valves.

2

Enhanced Durability

Efforts are underway to create valves with longer lifespans, reducing the need for repeat surgeries.

3

Minimized Biological Response

Researchers are exploring materials and coatings that minimize the body's immune response to prosthetic valves.

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Key Takeaways and Next Steps

1

Patient-Centered Approach

The choice of prosthetic valve should be individualized based on patient factors, valve durability, and anticoagulation risks.

2

Ongoing Research

Continued research and development are crucial for improving the safety, efficacy, and longevity of prosthetic heart valves.

3

Multidisciplinary Care

Effective management of prosthetic heart valve patients requires a multidisciplinary approach involving cardiologists, surgeons, and other healthcare professionals.




Thank You
TTHANKS