Aortic Stenosis (AS)

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Stages of AS

Stage	Definition	Valve Anatomy	Valve Hemodynamics	Hemodynamic Consequences	Symptoms
A	At risk of AS	■ BAV (or other congenital valve anomaly) ■ Aortic valve sclerosis	Aortic V_{max} <2 m/s with normal leaflet motion	None	None
B	Progressive AS	■ Mild to moderate leaflet calcification/ fibrosis of a bicuspid or trileaflet valve with some reduction in systolic motion or ■ Rheumatic valve changes with commissural fusion	■ Mild AS: aortic V_{max} 2.0-2.9 m/s or mean ΔP <20 mm Hg ■ Moderate AS: aortic V_{max} 3.0-3.9 m/s or mean ΔP 20-39 mm Hg	■ Early LV diastolic dysfunction may be present ■ Normal LVEF	None
C: Asymptomatic severe AS					
C1	Asymptomatic severe AS	Severe leaflet calcification/fibrosis or congenital stenosis with severely reduced leaflet opening	■ Aortic V_{max} ≥4 m/s or mean ΔP ≥40 mm Hg ■ AVA typically is ≤1.0 cm^2 (or AVAi 0.6 cm^2/m^2) but not required to define severe AS ■ Very severe AS is an aortic V_{max} ≥5 m/s or mean P ≥60 mm Hg	■ LV diastolic dysfunction ■ Mild LV hypertrophy ■ Normal LVEF	■ None ■ Exercise testing is reasonable to confirm symptom status

C2	Asymptomatic severe AS with LV systolic dysfunction	Severe leaflet calcification/fibrosis or congenital stenosis with severely reduced leaflet opening	■ Aortic V_{max} ≥4 m/s or mean ΔP ≥40 mm Hg ■ AVA typically ≤1.0 cm^2 (or AVAi 0.6 cm^2/m^2) but not required to define severe AS	LVEF <50%	None
D: Symptomatic severe AS					
D1	Symptomatic severe high-gradient AS	Severe leaflet calcification/fibrosis or congenital stenosis with severely reduced leaflet opening	■ Aortic V_{max} ≥4 m/s or mean ΔP ≥40 mm Hg ■ AVA typically ≤1.0 cm^2 (or AVAi ≤0.6 cm^2/m^2) but may be larger with mixed AS/AR	■ LV diastolic dysfunction ■ LV hypertrophy ■ Pulmonary hypertension may be present	■ Exertional dyspnea, decreased exercise tolerance, or HF ■ Exertional angina ■ Exertional syncope or presyncope
D2	Symptomatic severe low-flow, low-gradient AS with reduced LVEF	Severe leaflet calcification/fibrosis with severely reduced leaflet motion	■ AVA ≤1.0 cm^2 with resting aortic V_{max} <4 m/s or mean ΔP <40 mm Hg ■ Dobutamine stress echocardiography shows AVA <1.0 cm^2 with V_{max} ≥4 m/s at any flow rate	■ LV diastolic dysfunction ■ LV hypertrophy ■ LVEF <50%	■ HF ■ Angina ■ Syncope or presyncope
D3	Symptomatic severe low-gradient AS with normal LVEF or paradoxical low-flow severe AS	Severe leaflet calcification/fibrosis with severely reduced leaflet motion	■ AVA ≤1.0 cm^2 (indexed AVA ≤0.6 cm^2/m^2) with an aortic V_{max} <4 m/s or mean ΔP <40 mm Hg **AND** ■ Stroke volume index <35 mL/m^2 ■ Measured when patient is normotensive (systolic blood pressure <140 mm Hg)	■ Increased LV relative wall thickness ■ Small LV chamber with low stroke volume ■ Restrictive diastolic filling ■ LVEF ≥50%	■ HF ■ Angina ■ Syncope or presyncope

The key measurements for clinical decision-making in patients with AS

	Severity of aortic stenosis		
Variables	Mild	Moderate	Severe
Peak aortic jet velocity (m.s-1)	< 3	3 - 4	> 4
Transvalvular mean gradient (mmHg)	< 20	20 - 40	> 40
Aortic valve area (cm^2)	> 1.5	1.0 - 1.5	< 1.0
Aortic valve area indexed to body surface area (cm^2/m^2)	>0.85	0.6-0.85	<0.6
Velocity ratio	-	>0.25	<0.25

DVI

- An additional measurement that may be useful when there are discrepancies in these measures or in other clinical or imaging data is the ratio of the velocity in the LV outflow tract proximal to the aortic valve and the velocity in the narrowed aortic orifice. The outflow tract–to–aortic velocity ratio is independent of body size and eliminates potential errors in calculated valve area related to measurement of LV outflow tract diameter or area. A normal ratio is close to 1.0, whereas a ratio of≤0.25 corresponds to a valve area 25% of normal for that patient, which is consistent with severe AS and is a predictor of symptom onset and adverse outcomes.

Diagnostic Testing: Initial Diagnosis

COR	LOE	RECOMMENDATIONS
1	A	1. In patients with signs or symptoms of AS or a BAV, TTE is indicated for accurate diagnosis of the cause of AS, assessment of hemodynamic severity, measurement of LV size and systolic function, and determination of prognosis and timing of valve intervention (1,2).
1	B-NR	2. In patients with suspected low-flow, low-gradient severe AS with normal LVEF (Stage D3), optimization of blood pressure control is recommended before measurement of AS severity by TTE, TEE, cardiac catheterization, or CMR (3–7).
2a	B-NR	3. In patients with suspected low-flow, low-gradient severe AS with reduced LVEF (Stage D2), low-dose dobutamine stress testing with echocardiographic or invasive hemodynamic measurements is reasonable to further define severity and assess contractile reserve (8–10).
2a	B-NR	4. In patients with suspected low-flow, low-gradient severe AS with normal or reduced LVEF (Stages D2 and D3), calculation of the ratio of the outflow tract to aortic velocity is reasonable to further define severity (1,11–13).
2a	B-NR	5. In patients with suspected low-flow, low-gradient severe AS with normal or reduced LVEF (Stages D2 and D3), measurement of aortic valve calcium score by CT imaging is reasonable to further define severity (14–18).

- **Class IIa in suspected low flow low gradient Severe AS**

1-DSE in low gradient low flow Severe AS with reduced EF

2-DVI (with normal or reduced EF)

3-Ca score of AV in CT (with normal or reduced EF)

HTN

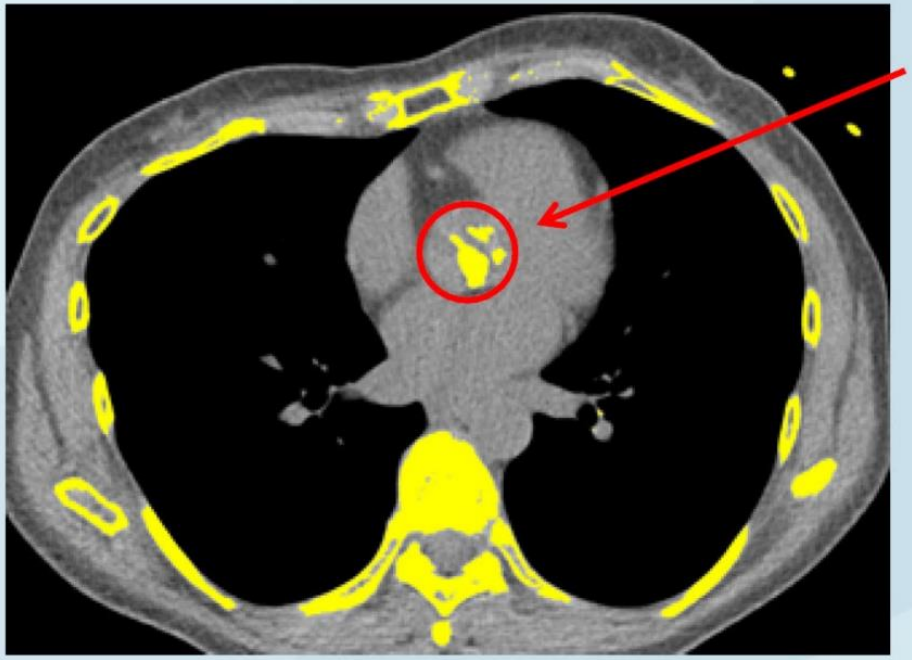
- Measurements of AS severity made when the patient is hypertensive may **underestimate** or, less often, overestimate stenosis severity. Systemic hypertension imposes a second pressure load on the LV, in addition to valve obstruction, which results in a lower forward stroke volume and lower transaortic pressure gradient than when the patient is normotensive.
- Thus, Doppler velocity data and invasive pressure measurements ideally **are recorded when the patient is normotensive (SBP<140 mmHg)**.
- If results indicate only moderate stenosis but were recorded when the patient was hypertensive, repeat measurements when the blood pressure is better controlled ensure that a diagnosis of severe AS is not missed.

DSE

- Patients with severe AS and LVEF <50% present wit an aortic valve area <1.0 cm2 but a low transvalvular velocity and pressure gradient (ie, velocity <4 m/s or mean gradient <40 mmHg) at rest. In these patients, severe AS with LV systolic dysfunction attributable to afterload mismatch must be distinguished from primary myocardial dysfunction with only moderate AS.
- DSE may be useful with measurement of aortic velocity (or mean pressure gradient) and valve area at baseline and at higher flow rates (maximum dose dobutamine 20 mcg/kg per minute) under appropriate clinical and hemodynamic monitoring.
- Severe AS is characterized by a fixed valve area, resulting in an increase in transaortic velocity to ≥4 m/s (mean gradient ≥40 mmHg) at any flow rate, but with valve area remaining ≤1.0 cm2.
- In contrast, in patients with moderate AS and primary LV dysfunction, there is an increase in valve area as volume flow rate increases, resulting in only a modest increase in transaortic velocity or gradient.
- Some patients **fail to show an increase in stroke volume ≥20% with dobutamine, referred to as "lack of contractile reserve" or "lack of flow reserve** ."

Use of Ca Scoring in LFLG AS

- ***Contractile/flow reserve: No***
- ***Stenosis severity: ???***



AVC: 2310AU

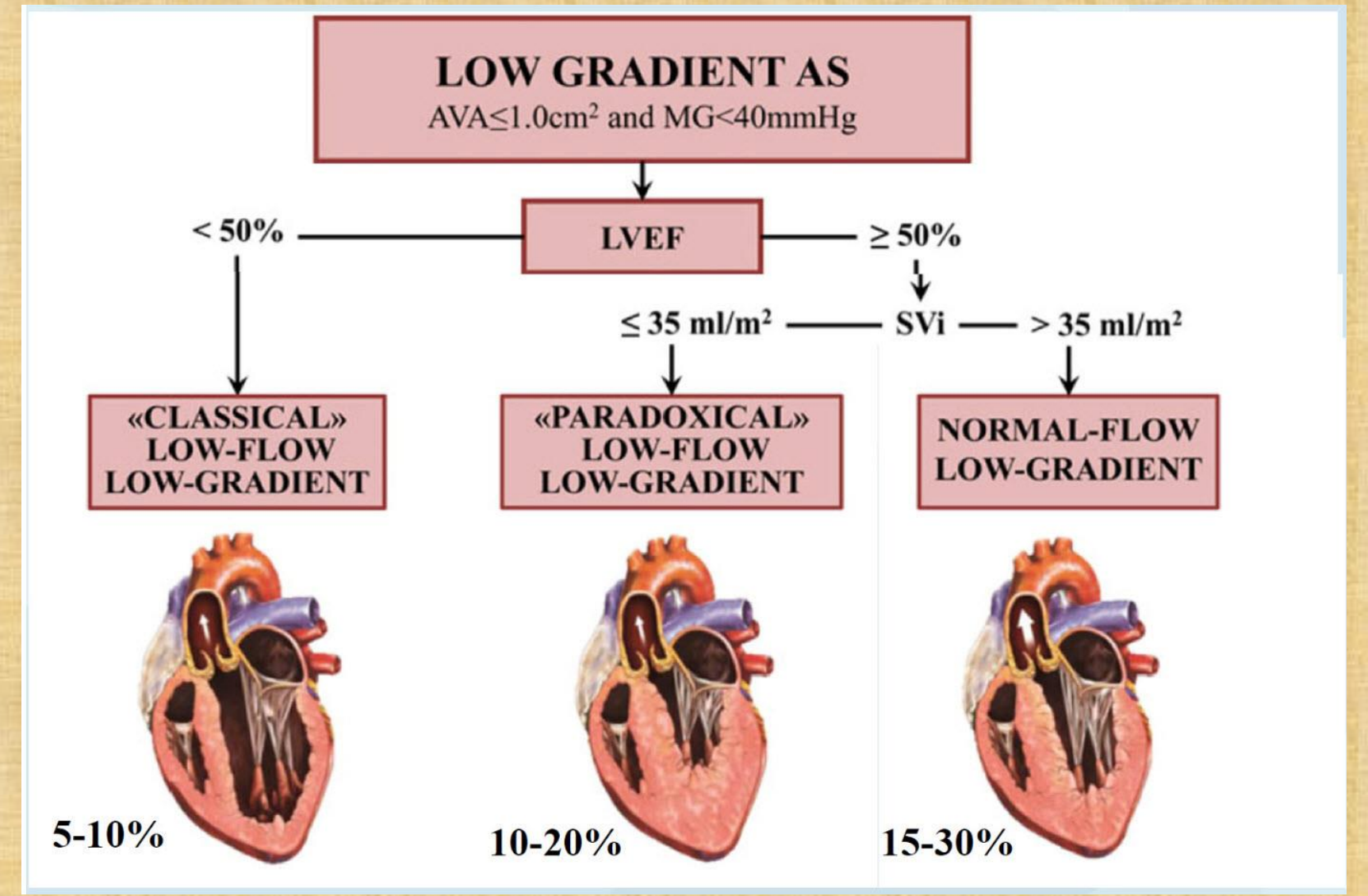
Sex	Threshold
Women	1274 AU
Men	2065 AU

- ***True severe AS***

High surgical risk

Low gradient Severe As

AVA≤ 1 cm2 and Mean gradient < 40 mmHg



Exercise Testing

COR	LOE	RECOMMENDATIONS
2a	B-NR	1. In asymptomatic patients with severe AS (Stage C1), exercise testing is reasonable to assess physiological changes with exercise and to confirm the absence of symptoms (1-4).
3: Harm	B-NR	2. In symptomatic patients with severe AS (Stage D1, aortic velocity ≥4.0 m/s or mean pressure gradient ≥40 mm Hg), exercise testing should not be performed because of the risk of severe hemodynamic compromise. (5)

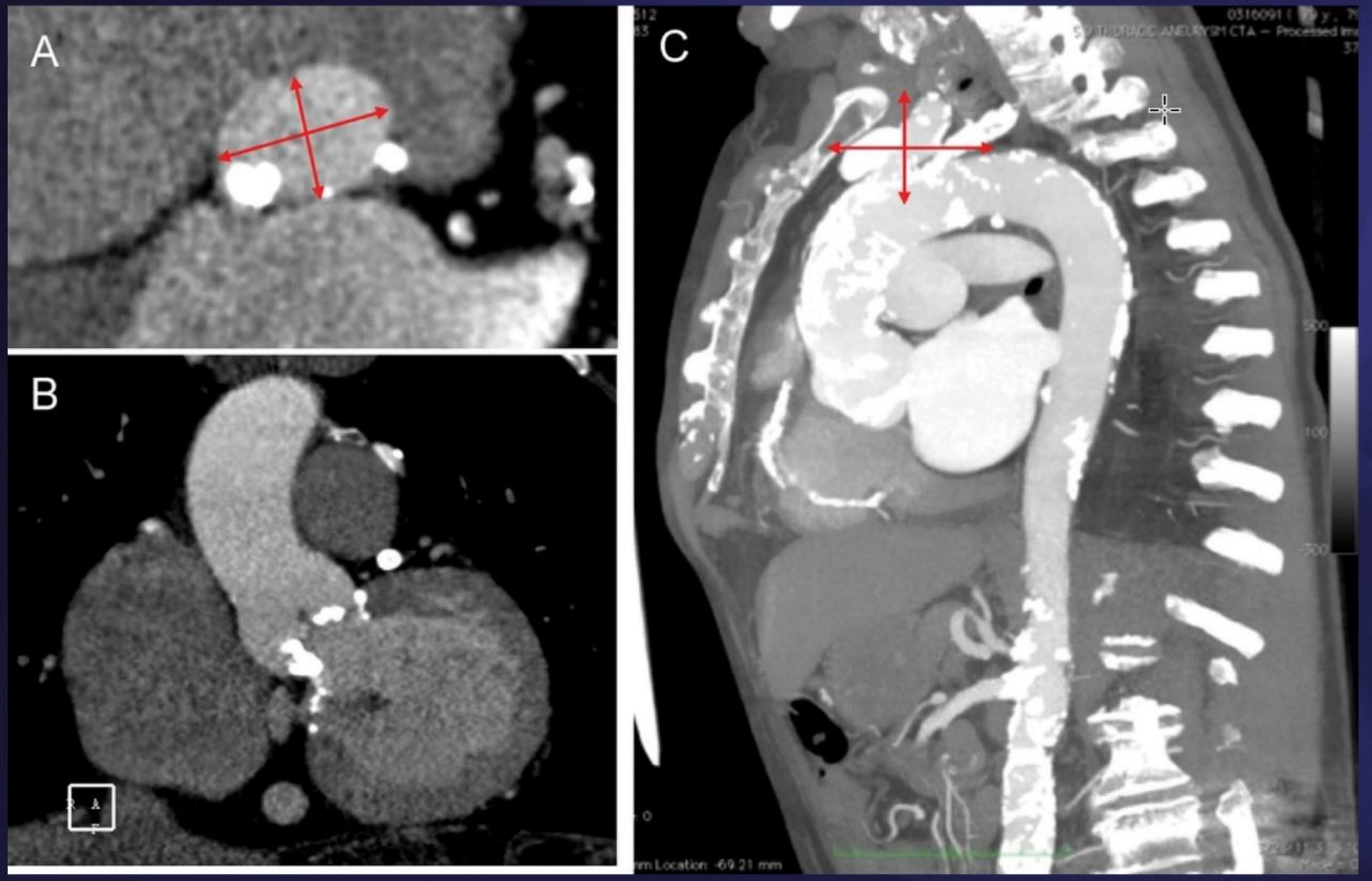
Exercise testing is avoided in symptomatic patients with AS because of a high risk of complications, including syncope, ventricular tachycardia, and death.

CT-scan

- The degree of aortic **valve calcification** is a strong predictor of clinical outcome, even when evaluated qualitatively by echocardiography .
- Quantitation of aortic valve calcium by CT imaging is especially useful in patients with low-flow, low-gradient AS of unclear severity with either a normal or reduced LVEF.
- Sex specific Agaston unit thresholds for diagnosis of severe AS are **1300** in women and **2000** in men.
- CT imaging also is used for procedural planning in patients undergoing TAVI, for measurement of annulus area, leaflet length, and the annular–to–coronary ostial distance.

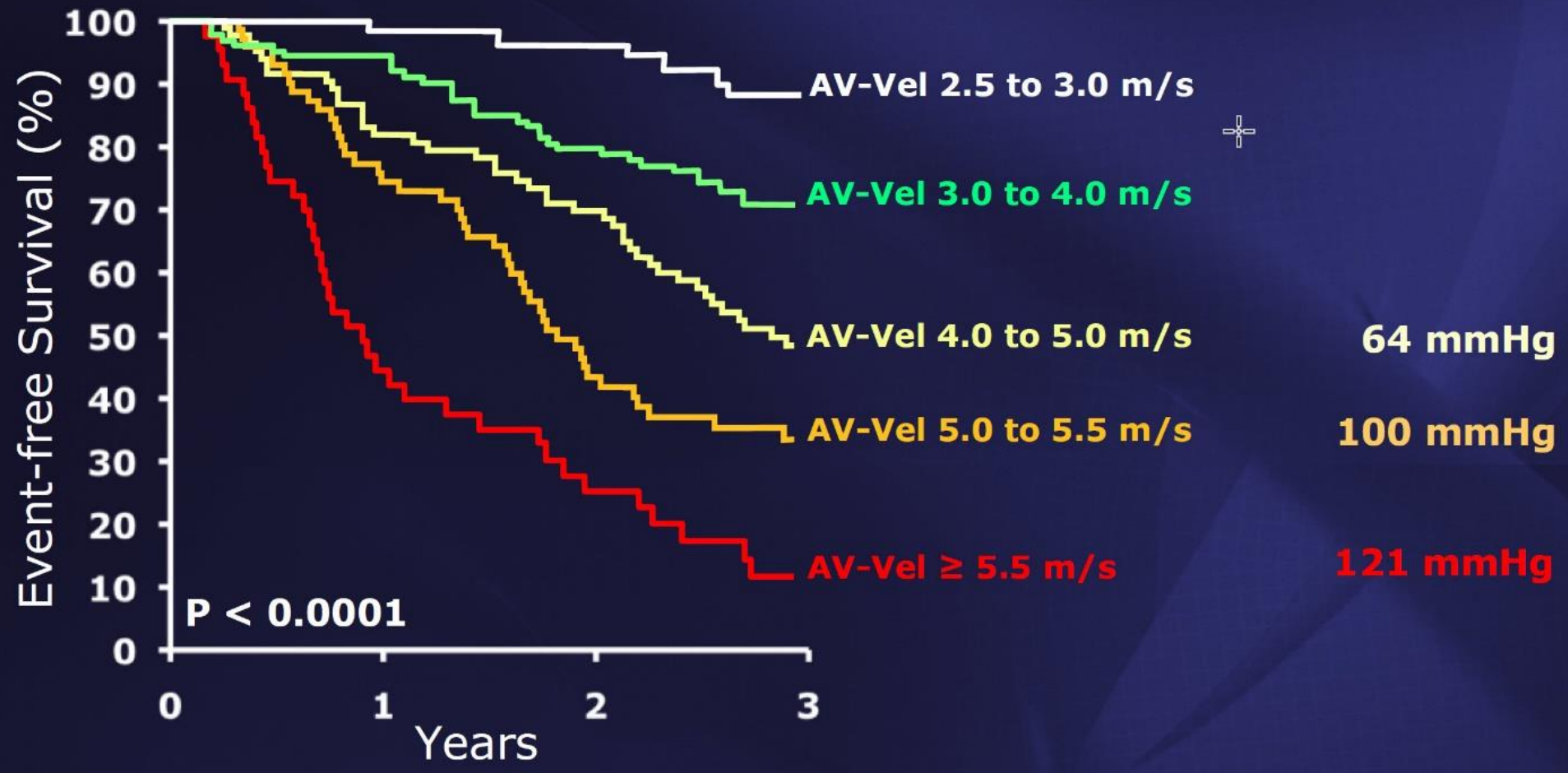
Preinterventional MSCT

Aortic Calcification, Root, Ascending Aorta



The Spectrum of Aortic Stenosis

Natural History



Rosenhek R et al. Eur Heart J 2004;25:199-205
Rosenhek R et al. N Engl J Med 2000;343:611-617
Rosenhek R et al. Circulation 2010;121:151-156

Medical therapy

COR	LOE	RECOMMENDATIONS
1	B-NR	1. In patients at risk of developing AS (Stage A) and in patients with asymptomatic AS (Stages B and C), hypertension should be treated according to standard GDMT, started at a low dose, and gradually titrated upward as needed, with appropriate clinical monitoring (1-3).
1	A	2. In all patients with calcific AS, statin therapy is indicated for primary and secondary prevention of atherosclerosis on the basis of standard risk scores (4-6).
2b	B-R	3. In patients who have undergone TAVI, renin-angiotensin system blocker therapy (ACE inhibitor or ARB) may be considered to reduce the long-term risk of all-cause mortality (7,8).
3: No Benefit	A	4. In patients with calcific AS (Stages B and C), statin therapy is not indicated for prevention of hemodynamic progression of AS (4-6).

- Medical treatment of hypertension and hyperlipidemia according to GDMT is appropriate for patients with AS.
- ACE inhibitor or ARB treatment may reduce the mortality rate in patients with AS who underwent TAVI.
- Hypertension is common in patients with AS, may be a risk factor for AS, and adds to the total pressure overload on the LV in combination with valve obstruction.
- Concern that antihypertensive medications might result in a decrease in cardiac output has not been corroborated in studies of medical therapy, likely because AS does not result in “fixed” valve obstruction until late in the disease process.

- Diuretics may reduce stroke volume, particularly if the LV chamber is small at baseline. In theory, ACE inhibitors may be advantageous because of the potential beneficial effects on LV fibrosis, in addition to control of hypertension.
-

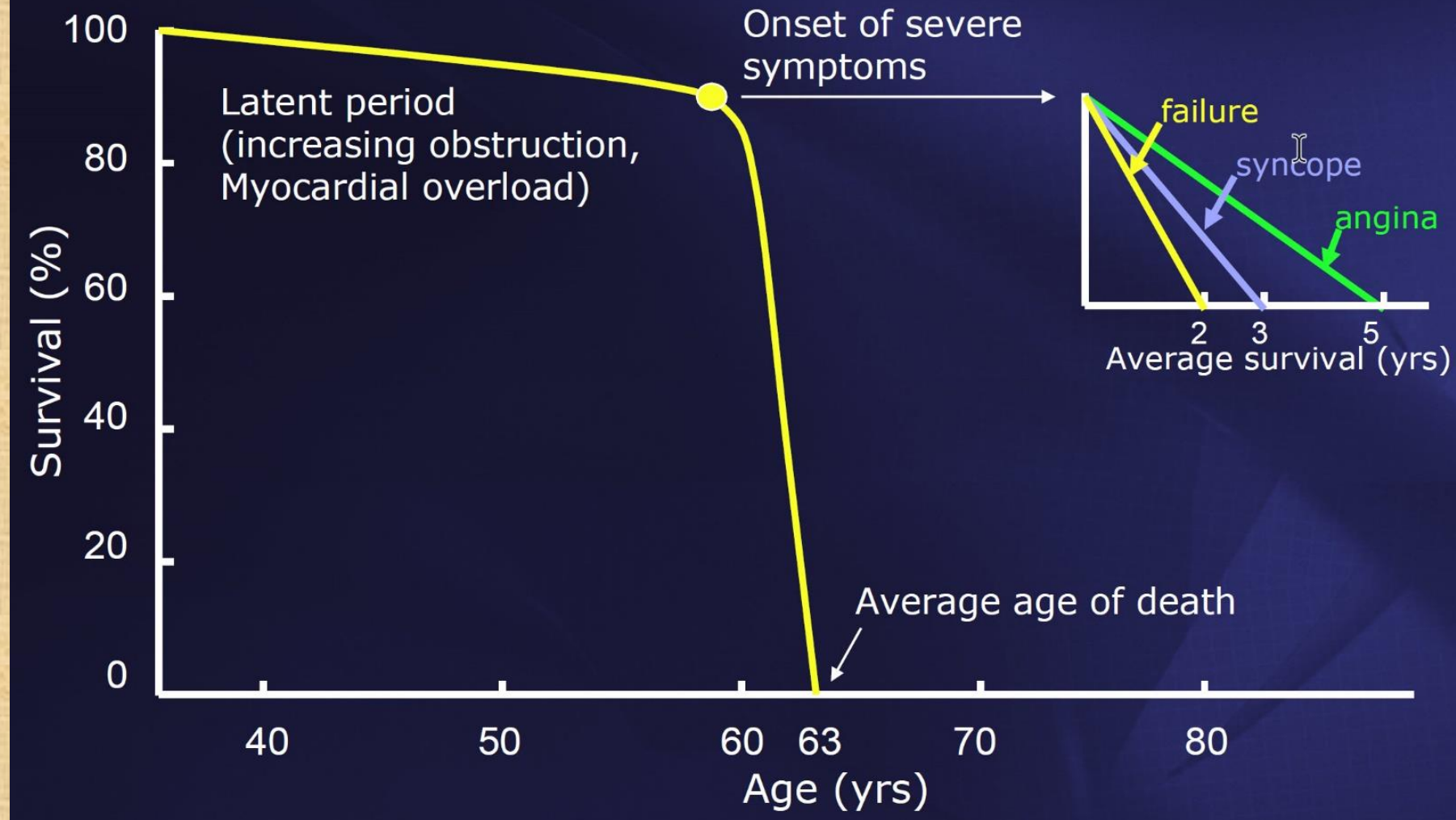
Timing of Interventio n

COR	LOE	RECOMMENDATIONS
1	A	1. In adults with severe high-gradient AS (Stage D1) and symptoms of exertional dyspnea, HF, angina, syncope, or presyncope by history or on exercise testing, AVR is indicated (1-7).
1	B-NR	2. In asymptomatic patients with severe AS and an LVEF <50% (Stage C2), AVR is indicated (8-11).
1	B-NR	3. In asymptomatic patients with severe AS (Stage C1) who are undergoing cardiac surgery for other indications, AVR is indicated (12-16).
1	B-NR	4. In symptomatic patients with low-flow, low-gradient severe AS with reduced LVEF (Stage D2), AVR is recommended (17-24).
1	B-NR	5. In symptomatic patients with low-flow, low-gradient severe AS with normal LVEF (Stage D3), AVR is recommended if AS is the most likely cause of symptoms (25-27).
2a	B-NR	6. In apparently asymptomatic patients with severe AS (Stage C1) and low surgical risk, AVR is reasonable when an exercise test demonstrates decreased exercise tolerance (normalized for age and sex) or a fall in systolic blood pressure of ≥10 mm Hg from baseline to peak exercise (13,28-30).
2a	B-R	7. In asymptomatic patients with very severe AS (defined as an aortic velocity of ≥5 m/s) and low surgical risk, AVR is reasonable (15,31-35).
2a	B-NR	8. In apparently asymptomatic patients with severe AS (Stage C1) and low surgical risk, AVR is reasonable when the serum B-type natriuretic peptide (BNP) level is >3 times normal (32,36-38).
2a	B-NR	9. In asymptomatic patients with high-gradient severe AS (Stage C1) and low surgical risk, AVR is reasonable when serial testing shows an increase in aortic velocity ≥0.3 m/s per year (39,40).
2b	B-NR	10. In asymptomatic patients with severe high-gradient AS (Stage C1) and a progressive decrease in LVEF on at least 3 serial imaging studies to <60%, AVR may be considered (8-11,33).
2b	C-EO	11. In patients with moderate AS (Stage B) who are undergoing cardiac surgery for other indications, AVR may be considered.

- **Class I**: Severe AS(high gradient or Low flow-low gradient) with either 1-symtoms or 2-LVEF<50% or need for noncardiac surgery even without symtoms.
- **Class IIa**: Asymptomatic Severe AS with abnormal ETT/very severe AS with Peak velocity ≥ 5 m/sec/BNP level >3xtimes normal/Rapid progressive severe AS,Velocity ≥0.3 m/sec/year.
- **Class IIb**: Severe asymptomatic AS with progressive decrease in EF<60% on at least 3 serial imaging studies/Moderate AS need to another cardiac surgery.

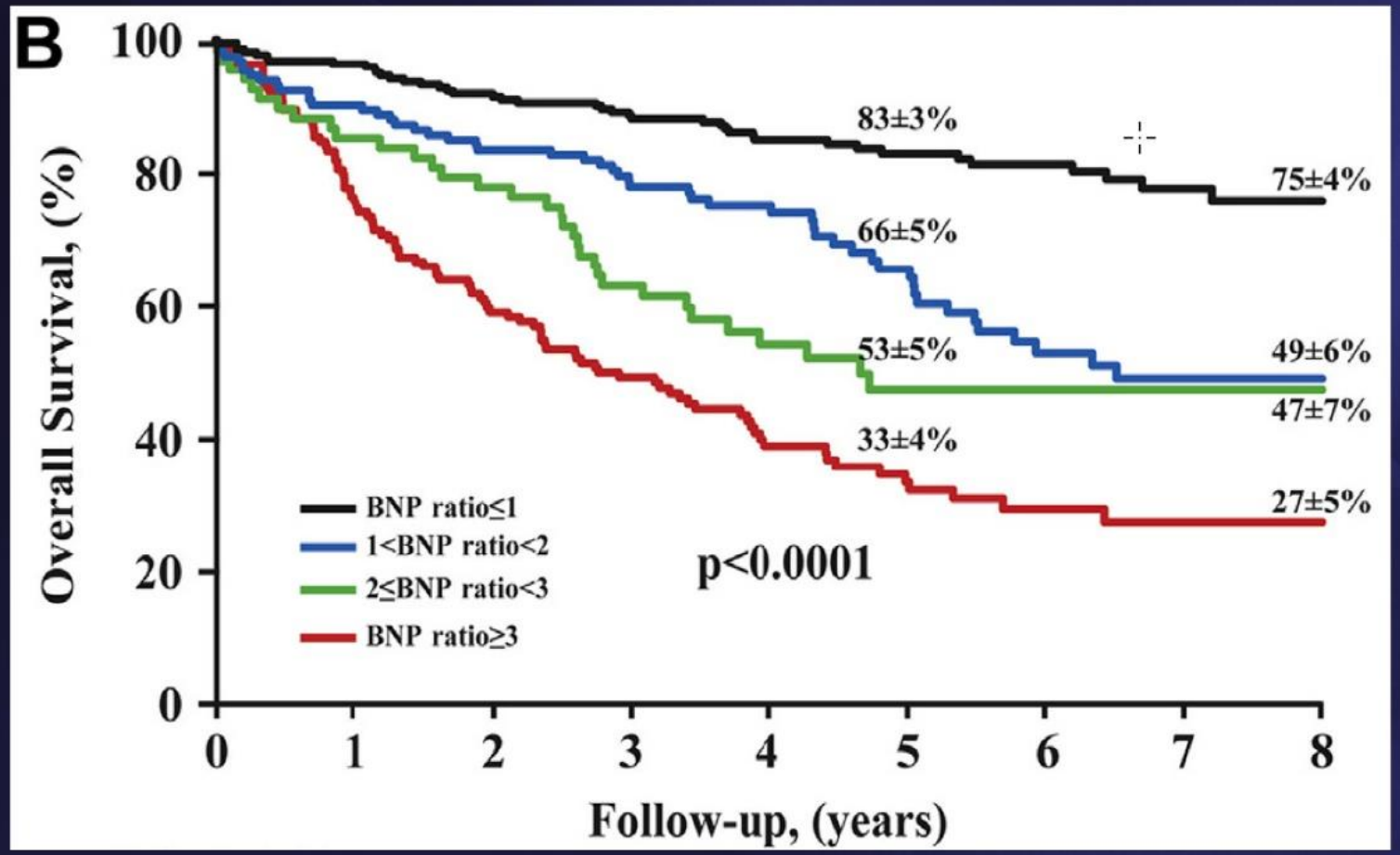
- The most common initial symptom of AS is exertional dyspnea or decreased exercise tolerance.
- Clinical vigilance is needed to recognize these early symptoms and proceed promptly to AVR.
- More severe “classical” symptoms of AS, including HF, syncope, or angina, can be avoided by appropriate treatment at the onset of even mild symptoms.

Severe Aortic Stenosis
Prognosis of Symptomatic Patients

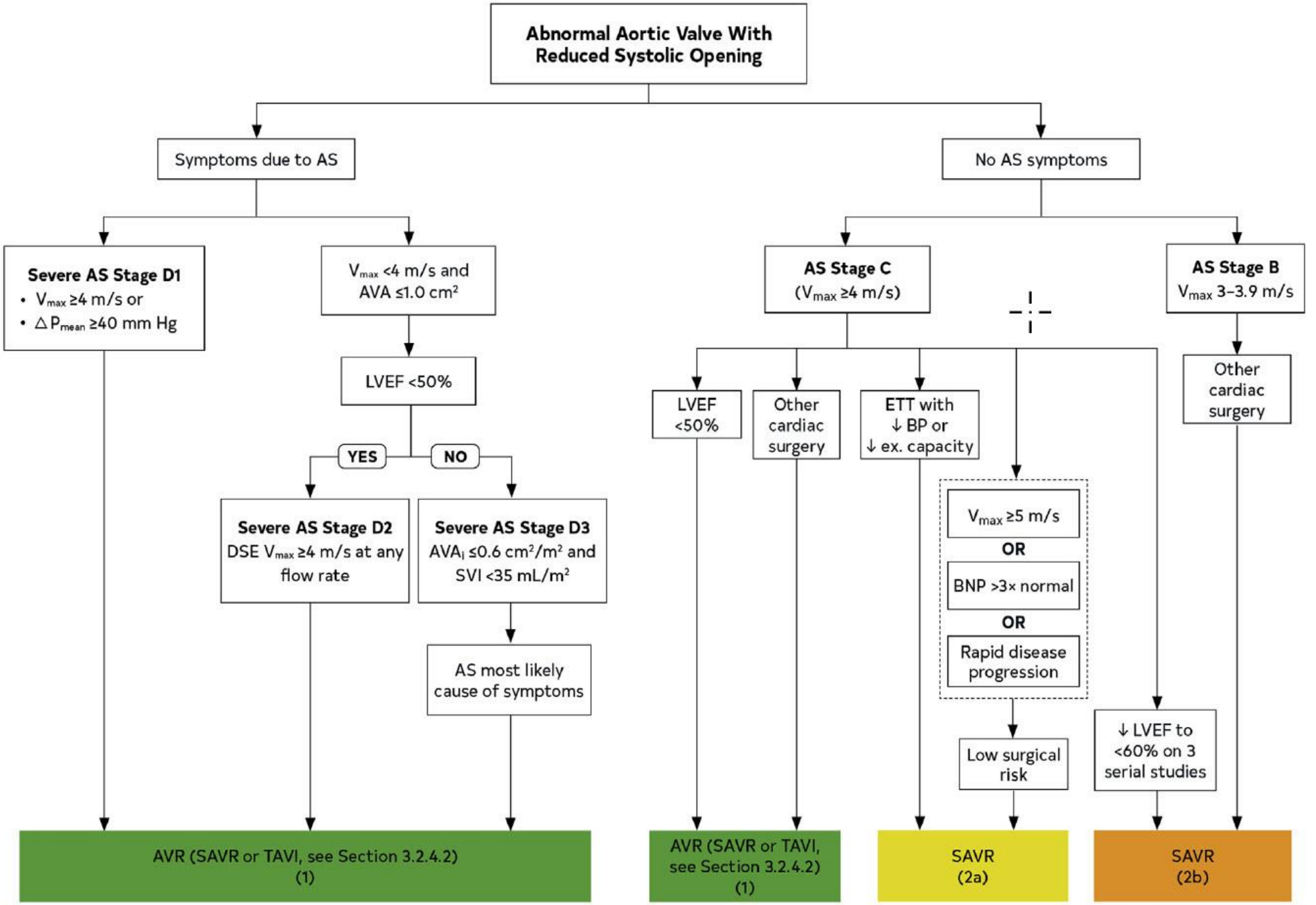


Ross, Braunwald. Circulation 1968

Aortic Stenosis
Survival: **BNPratio**



Clavel MA et al. J Am Col Cardiol 2014;63:2016-25


Abnormal Aortic Valve With Reduced Systolic Opening
Symptoms due to AS
No AS symptoms
Severe AS Stage D1
• Vmax ≥4 m/s or
• ΔPmean ≥40 mm Hg
Vmax <4 m/s and AVA ≤1.0 cm²
LVEF <50%
YES
NO
Severe AS Stage D2
DSE Vmax ≥4 m/s at any flow rate
Severe AS Stage D3
AVAi ≤0.6 cm²/m² and SVI <35 mL/m²
AS most likely cause of symptoms
AVR (SAVR or TAVI, see Section 3.2.4.2) (1)
AS Stage C
(Vmax ≥4 m/s)
AS Stage B
Vmax 3–3.9 m/s
LVEF <50%
Other cardiac surgery
ETT with ↓ BP or ↓ ex. capacity
Vmax ≥5 m/s
OR
BNP >3× normal
OR
Rapid disease progression
Low surgical risk
↓ LVEF to <60% on 3 serial studies
Other cardiac surgery
AVR (SAVR or TAVI, see Section 3.2.4.2) (1)
SAVR (2a)
SAVR (2b)

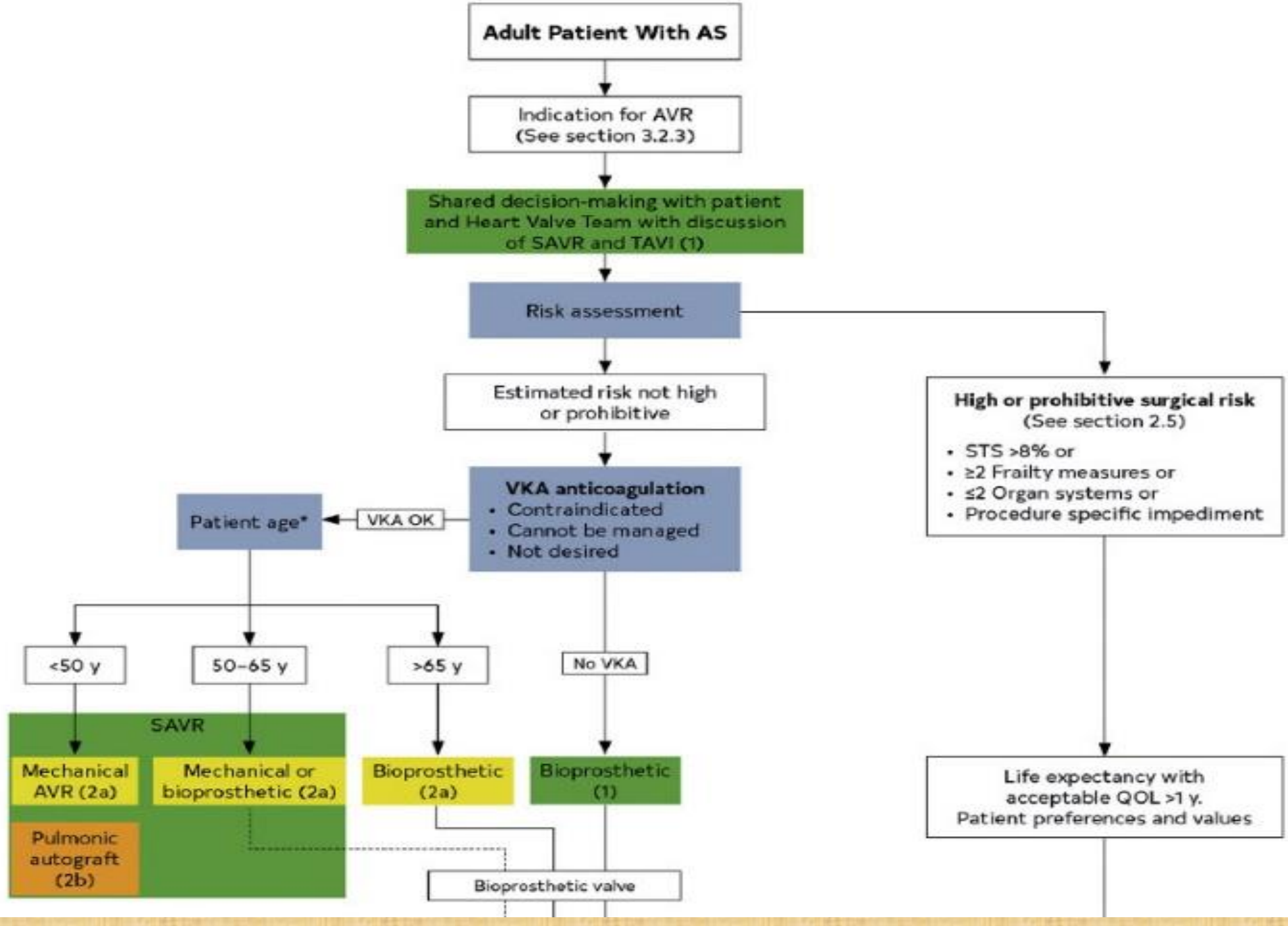
Choice of Mechanical Versus Bioprosthetic AVR

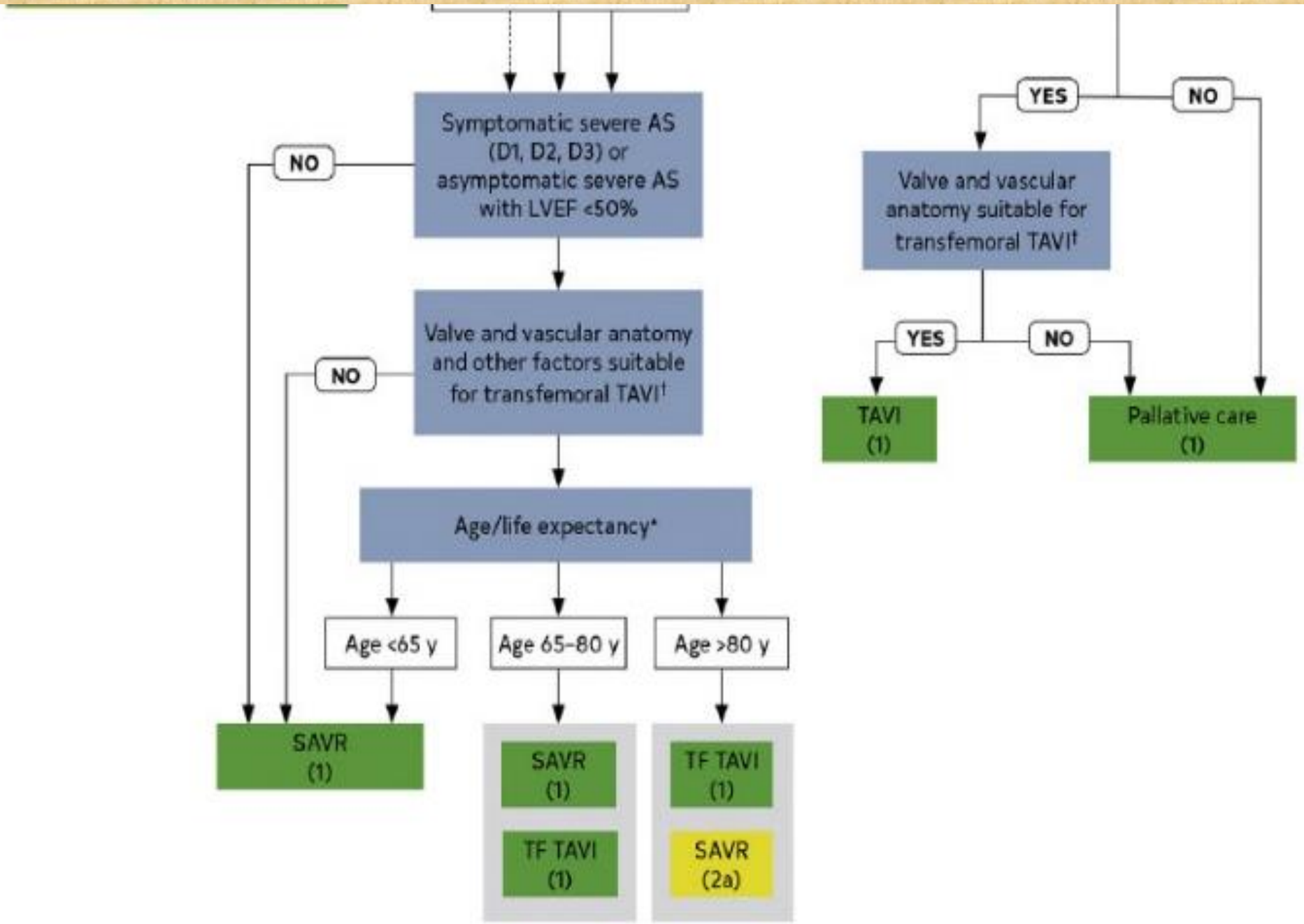
COR	LOE	RECOMMENDATIONS
1	C-EO	1. In patients with an indication for AVR, the choice of prosthetic valve should be based on a shared decision-making process that accounts for the patient's values and preferences and includes discussion of the indications for and risks of anticoagulant therapy and the potential need for and risks associated with valve reintervention.
1	C-EO	2. For patients of any age requiring AVR for whom VKA anticoagulant therapy is contraindicated, cannot be managed appropriately, or is not desired, a bioprosthetic AVR is recommended.
2a	B-R	3. For patients <50 years of age who do not have a contraindication to anticoagulation and require AVR, it is reasonable to choose a mechanical aortic prosthesis over a bioprosthetic valve. (1)
2a	B-NR	4. For patients 50 to 65 years of age who require AVR and who do not have a contraindication to anti-coagulation, it is reasonable to individualize the choice of either a mechanical or bioprosthetic AVR with consideration of individual patient factors and after informed shared decision-making. (1–10)
2a	B-R	5. In patients >65 years of age who require AVR, it is reasonable to choose a bioprosthesis over a mechanical valve. (1)
2b	B-NR	6. In patients <50 years of age who prefer a bioprosthetic AVR and have appropriate anatomy, replacement of the aortic valve by a pulmonic autograft (the Ross procedure) may be considered at a Comprehensive Valve Center (11–13).

Choice of SAVR Versus TAVI for Patients for Whom a Bioprosthetic AVR Is Appropriate

COR	LOE	RECOMMENDATIONS
1	A	1. For symptomatic and asymptomatic patients with severe AS and any indication for AVR who are <65 years of age or have a life expectancy >20 years, SAVR is recommended (1–3).
1	A	2. For symptomatic patients with severe AS who are 65 to 80 years of age and have no anatomic contraindication to transfemoral TAVI, either SAVR or transfemoral TAVI is recommended after shared decision-making about the balance between expected patient longevity and valve durability (1,4–8).
1	A	3. For symptomatic patients with severe AS who are >80 years of age or for younger patients with a life expectancy <10 years and no anatomic contraindication to transfemoral TAVI, transfemoral TAVI is recommended in preference to SAVR (1,4–10).
1	B-NR	4. In asymptomatic patients with severe AS and an LVEF <50% who are ≤80 years of age and have no anatomic contraindication to transfemoral TAVI, the decision between TAVI and SAVR should follow the same recommendations as for symptomatic patients in Recommendations 1, 2, and 3 above (1,2,4–10).
1	B-NR	5. For asymptomatic patients with severe AS and an abnormal exercise test, very severe AS, rapid progression, or an elevated BNP (COR 2a indications for AVR), SAVR is recommended in preference to TAVI (1–3,11).
1	A	6. For patients with an indication for AVR for whom a bioprosthetic valve is preferred but valve or vascular anatomy or other factors are not suitable for transfemoral TAVI, SAVR is recommended (1–3,11).
1	A	7. For symptomatic patients of any age with severe AS and a high or prohibitive surgical risk, TAVI is recommended if predicted post-TAVI survival is >12 months with an acceptable quality of life (12,13,14,15).
1	C-EO	8. For symptomatic patients with severe AS for whom predicted post-TAVI or post-SAVR survival is <12 months or for whom minimal improvement in quality of life is expected, palliative care is recommended after shared decision-making, including discussion of patient preferences and values.
2b	C-EO	9. In critically ill patients with severe AS, percutaneous aortic balloon dilation may be considered as a bridge to SAVR or TAVI.

- **Choice of SAVR Versus TAVI When AVR is Indicated for Valvular AS**


Adult Patient With AS
Indication for AVR
(See section 3.2.3)
Shared decision-making with patient and Heart Valve Team with discussion of SAVR and TAVI (1)
Risk assessment
Estimated risk not high or prohibitive
High or prohibitive surgical risk
(See section 2.5)
• STS >8% or
• ≥2 Frailty measures or
• ≤2 Organ systems or
• Procedure specific impediment
VKA anticoagulation
• Contraindicated
• Cannot be managed
• Not desired
VKA OK
Patient age*
<50 y
50–65 y
>65 y
No VKA
SAVR
Mechanical AVR (2a)
Mechanical or bioprosthetic (2a)
Pulmonic autograft (2b)
Bioprosthetic (2a)
Bioprosthetic (1)
Bioprosthetic valve
Life expectancy with acceptable QOL >1 y.
Patient preferences and values


Symptomatic severe AS (D1, D2, D3) or asymptomatic severe AS with LVEF <50%
NO
Valve and vascular anatomy and other factors suitable for transfemoral TAVI†
NO
Age/life expectancy*
Age <65 y
Age 65–80 y
Age >80 y
SAVR (1)
SAVR (1)
TF TAVI (1)
TF TAVI (1)
SAVR (2a)
YES
NO
Valve and vascular anatomy suitable for transfemoral TAVI†
YES
NO
TAVI (1)
Pallative care (1)

TAVI/ SAVR

- TAVI has a slightly lower mortality risk and is associated with a shorter hospital length of stay, more rapid return to normal activities, **lower risk of transient or permanent AF**, less bleeding and less pain than SAVR.
-
- Compared with SAVR, **TAVI results in higher rates of vascular complications, paravalvular regurgitation, permanent pacemaker implantation, and valve intervention**

- The specific choice of a balloon expandable valve or self-expanding valve depends on patient anatomy and other considerations.
- The mortality rate has been higher with TAVI by nonfemoral access routes than with SAVR, possibly because of the access approach itself, but more likely because of the higher comorbidity burden and risk in patients with vascular disease severe enough to preclude transfemoral access.

- The survival and symptom reduction benefit of TAVI is seen only in appropriately selected patients.
- Baseline clinical factors associated with a poor outcome after TAVI include advanced age, frailty, smoking or COPD , pulmonary hypertension, liver disease, prior stroke, anemia, and other systemic conditions.
- Patients with a mechanical impediment to SAVR, such as a porcelain aorta or prior chest radiation damage, may have better outcomes after TAVI.
- TAVI is not recommended in patients with 1) a life expectancy of <1 year even with a successful procedure or 2) those with a chance of “survival with benefit” of <25% at 2 years.

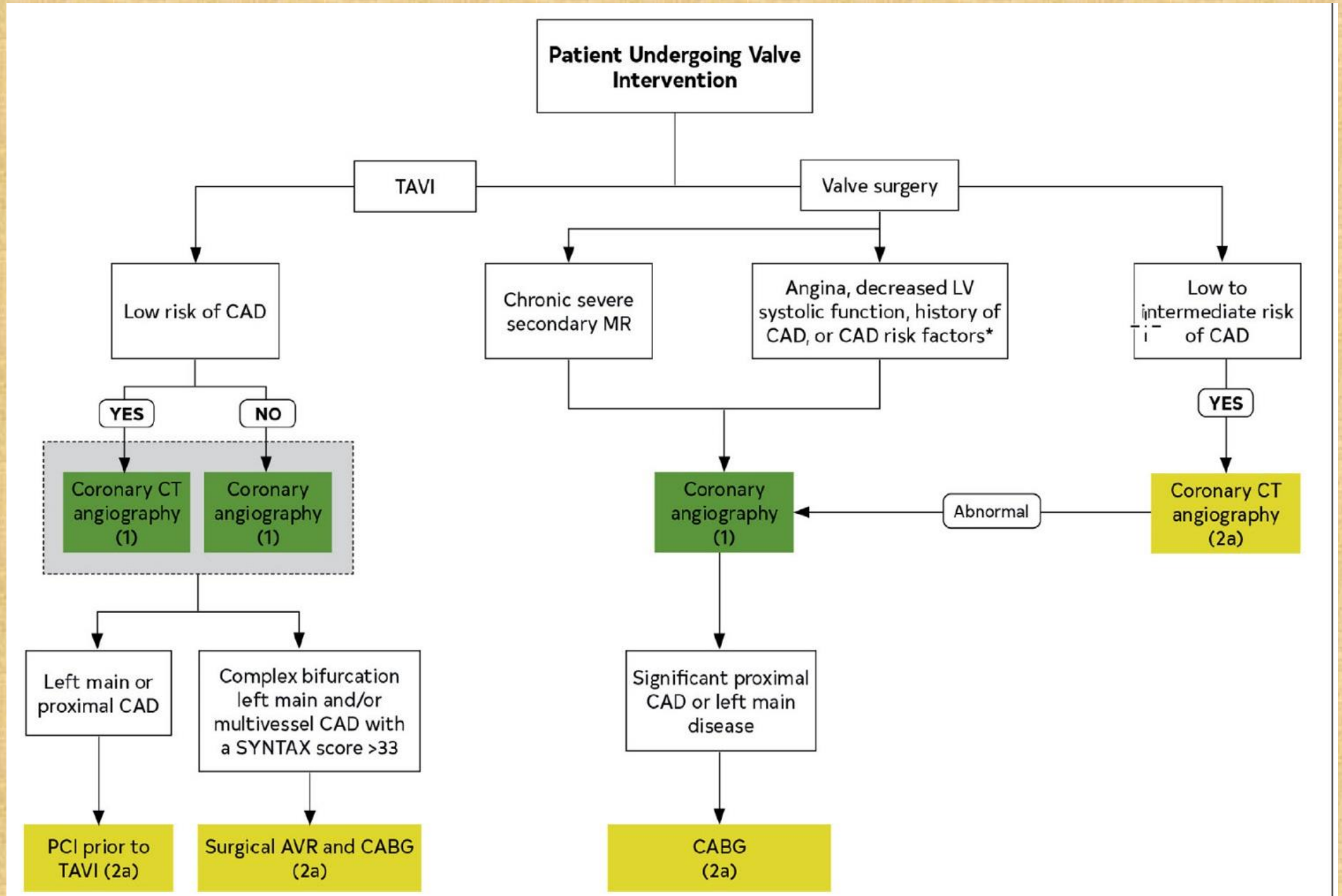
balloon dilation

- Percutaneous aortic balloon dilation has a role in treating **children**, **adolescents**, and **young** adults with AS, but its role in treating older patients is very limited.
- The mechanism by which balloon dilation modestly reduces the severity of stenosis in older patients is fracture of calcific deposits within the valve leaflets and, to a minor degree, stretching of the annulus and separation of the calcified or fused commissures. Immediate hemodynamic results include a moderate reduction in the transvalvular pressure gradient, but the postdilation valve area rarely exceeds 1.0 cm2.
- Despite the modest change in valve area, an early symptomatic improvement usually occurs. However, serious acute complications, including acute severe AR, restenosis, and clinical deterioration, occur within 6 to 12 months in most patients. Therefore, in patients with AS, percutaneous aortic balloon dilation is not a substitute for AVR.

- Percutaneous aortic balloon dilation can have a temporary role in the management of some symptomatic patients, such as those patients with severe AS and refractory pulmonary edema or cardiogenic shock, who might benefit from percutaneous aortic balloon dilation as a "bridge" to TAVI or SAVR.
- However, this approach is used less frequently given the availability and success of immediate TAVI even in very high-risk patients.

Management of CAD in Patients Undergoing TAVI

COR	LOE	RECOMMENDATIONS
1	C-EO	1. In patients undergoing TAVI, 1) contrast-enhanced coronary CT angiography (in patients with a low pretest probability for CAD) or 2) an invasive coronary angiogram is recommended to assess coronary anatomy and guide revascularization.
2a	C-LD	2. In patients undergoing TAVI with significant left main or proximal CAD with or without angina, revascularization by PCI before TAVI is reasonable (1,2).
2a	C-LD	3. In patients with significant AS and significant CAD (luminal reduction >70% diameter, fractional flow reserve <0.8, instantaneous wave-free ratio <0.89) consisting of complex bifurcation left main and/or multivessel CAD with a SYNTAX (Synergy Between Percutaneous Coronary Intervention With Taxus and Cardiac Surgery) score >33, SAVR and CABG are reasonable and preferred over TAVI and PCI (3,4).


Patient Undergoing Valve Intervention
TAVI
Valve surgery
Low risk of CAD
Chronic severe secondary MR
Angina, decreased LV systolic function, history of CAD, or CAD risk factors*
Low to intermediate risk of CAD
YES
NO
YES
Coronary CT angiography (1)
Coronary angiography (1)
Coronary angiography (1)
Abnormal
Coronary CT angiography (2a)
Left main or proximal CAD
Complex bifurcation left main and/or multivessel CAD with a SYNTAX score >33
Significant proximal CAD or left main disease
PCI prior to TAVI (2a)
Surgical AVR and CABG (2a)
CABG (2a)

- Overall, nonrandomized studies suggest that PCI before TAVI is safe and feasible , even patients with left main disease .
- Conceptually, pre-TAVI PCI also allows a safer procedure and circumvents future post-TAVI PCI, which can be occasionally challenging.
- Staged PCI before TAVI is a common strategy in clinical practice and is associated with lower contrast volume and renal failure than is the strategy of TAVI with concomitant PCI , although the timing of pre-TAVI PCI remains controversial.