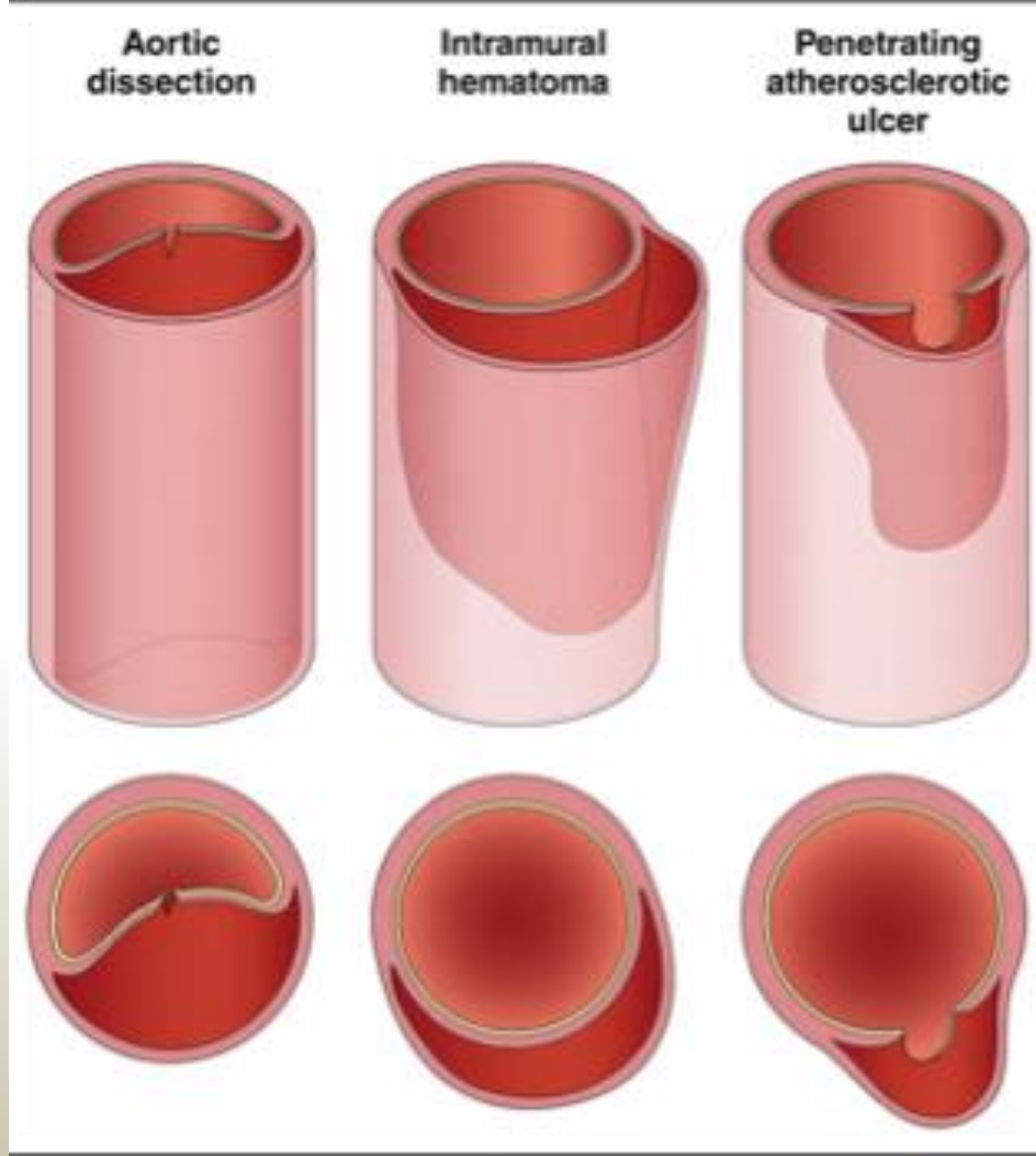
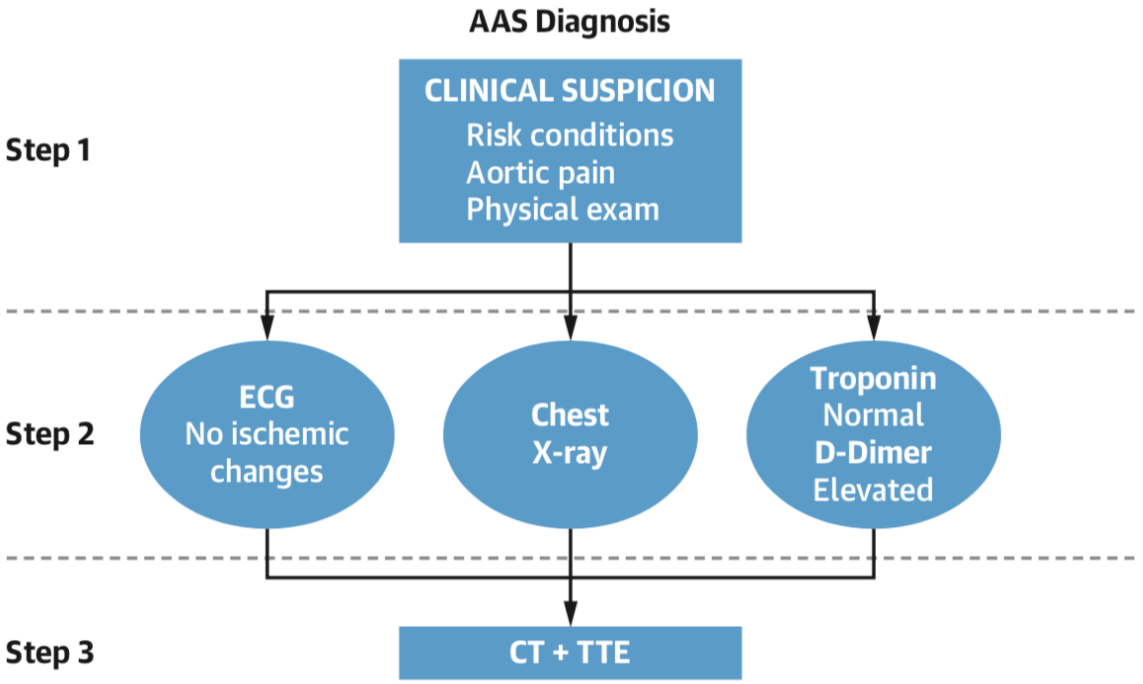
DISSEZIONE AORTICA: PROVE INCERTEZZE E TERAPIE FUTURE

A. MONTALTO

OVERVIEW OF ACUTE AORTIC SYNDROME COMPONENTS AND THEIR MAIN MORPHOLOGIC CHARACTERISTICS



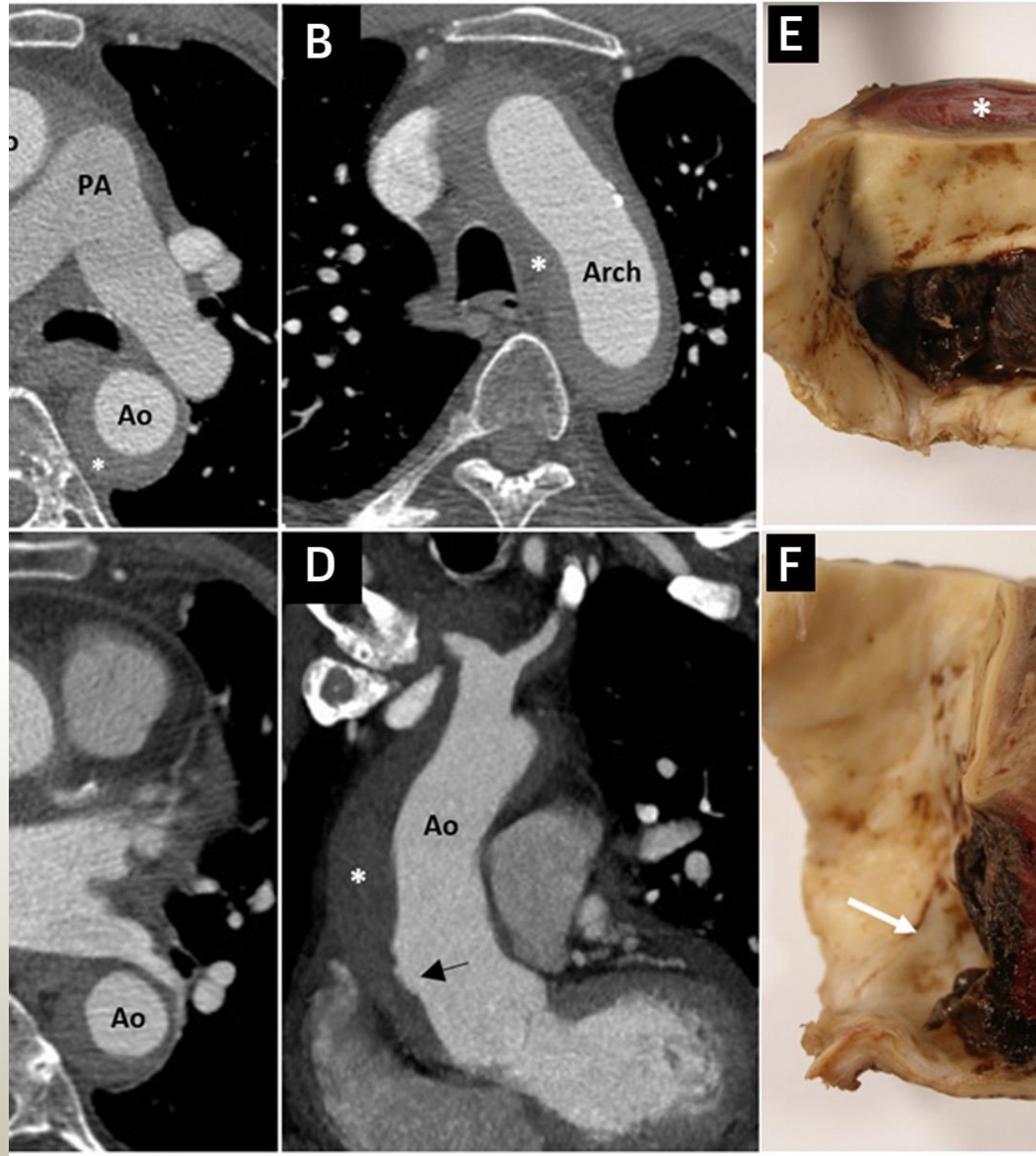


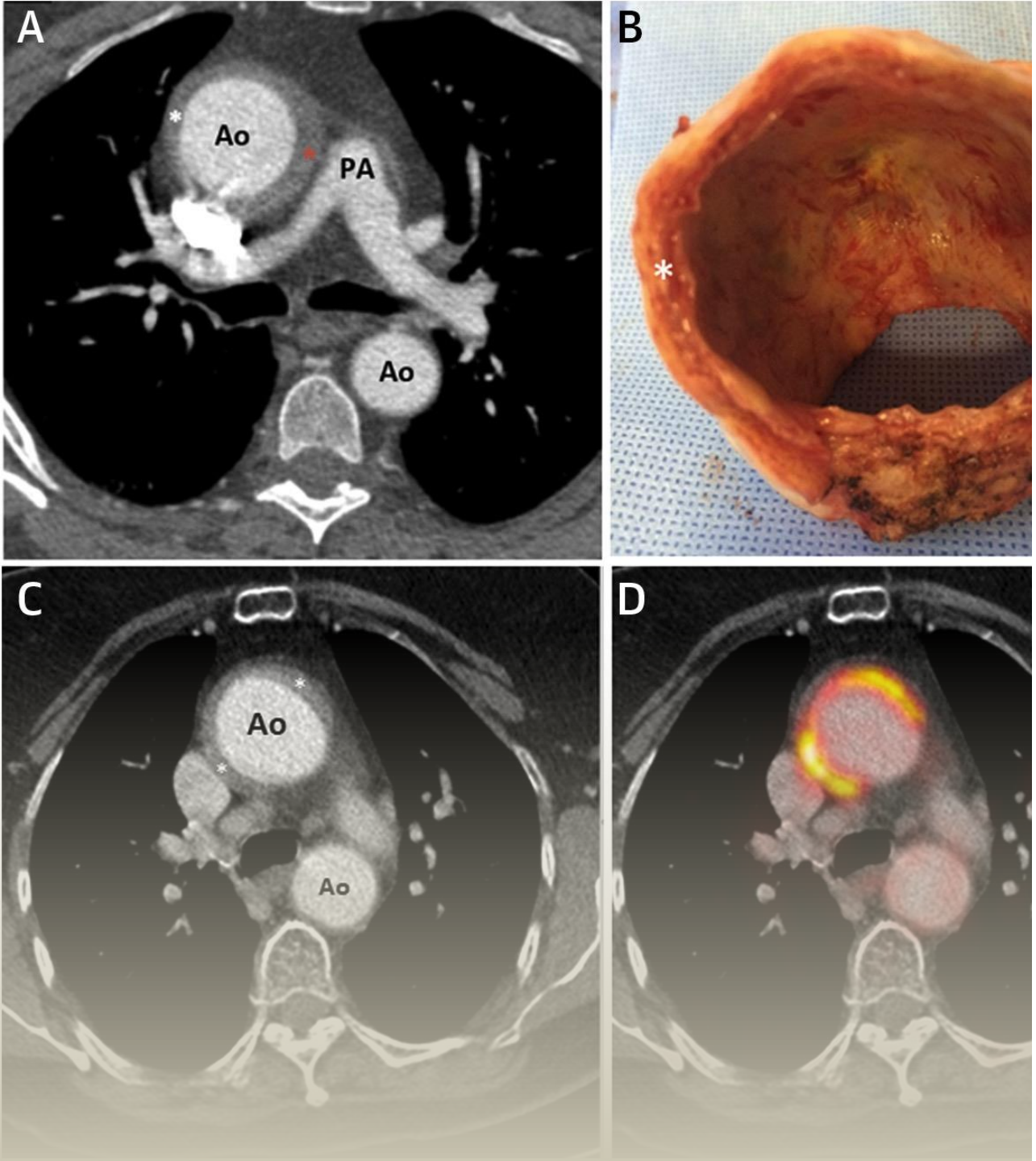
IMPROVED DIAGNOSIS OF PATIENTS WITH AAS: THE 3-STEP DIAGNOSTIC ALGORITHM

AAS-RELATED ENTITIES: GUIDELINES TO AVOID MISINTERPRETATION OF IMAGING FINDINGS

ACUTELY THROMBOSED CD VS IMH

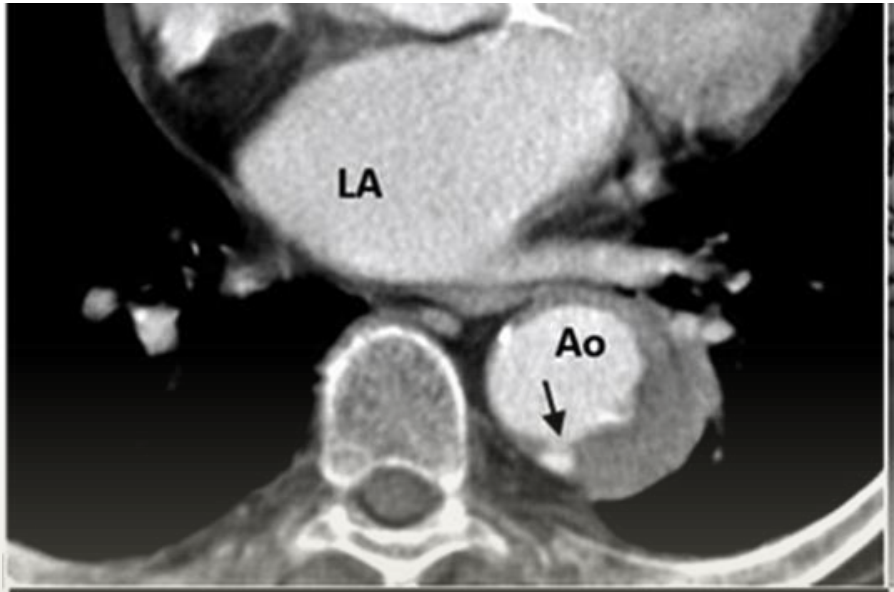
Combining axial and sagittal planes, a focal intimal contour alteration corresponding to a dissection tear will be well depicted in a thrombosed CD . IMH has no apparent entrance tear, and if it does, it is microscopic. Distinction of both entities by means of CT is not al ways possible.

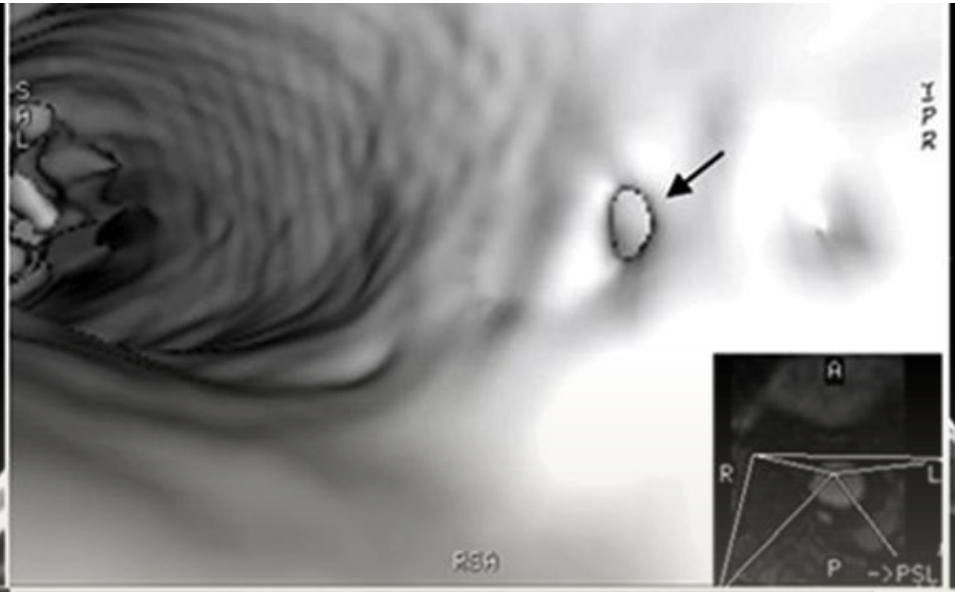




AORTITIS VS IMH

IMH appears as a crescentic (non-circumferential) aortic wall thickening with a smooth luminal surface. A hyper attenuated aortic wall contour is well appreciated on non contrast CT images. Patients with aortitis may unfrequently simulate an AAS at presentation . Circumferential arterial wall thickening, and homogeneous wall enhancement are typical features of aortitis on contrast-enhanced CT. However, this is not invariably the case. Characteristically, positron emission tomography/CT can depict the inflammatory process.





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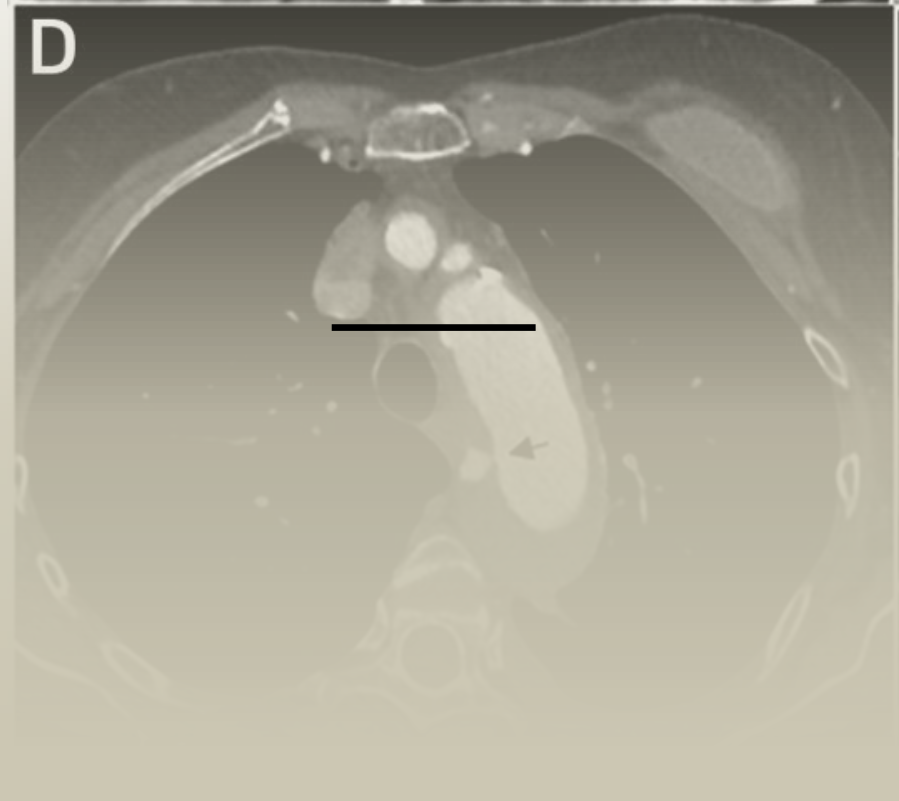

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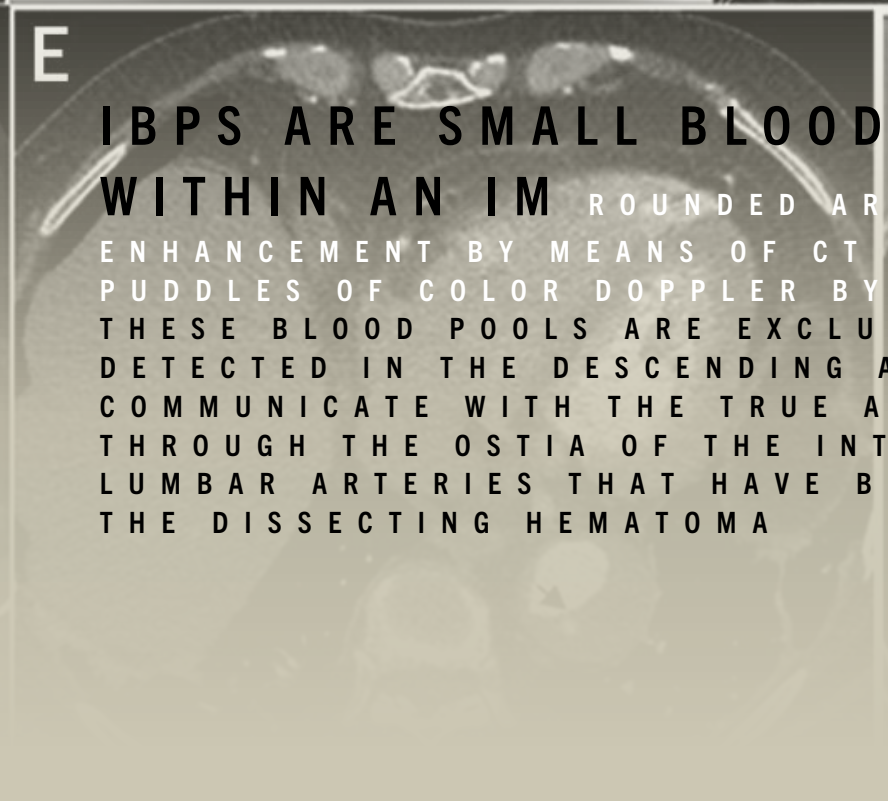

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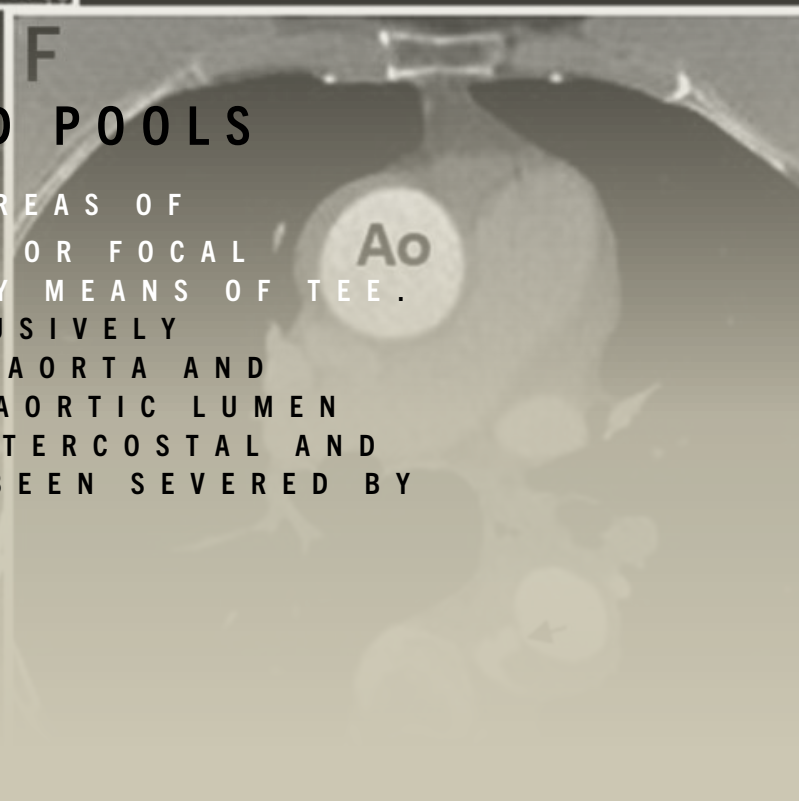


IBPS ARE SMALL BLOOD POOLS WITHIN AN IM ROUNDED AREAS OF ENHANCEMENT BY MEANS OF CT OR FOCAL PUDDLES OF COLOR DOPPLER BY MEANS OF TEE. THESE BLOOD POOLS ARE EXCLUSIVELY DETECTED IN THE DESCENDING AORTA AND COMMUNICATE WITH THE TRUE AORTIC LUMEN THROUGH THE OSTIA OF THE INTERCOSTAL AND LUMBAR ARTERIES THAT HAVE BEEN SEVERED BY THE DISSECTING HEMATOMA

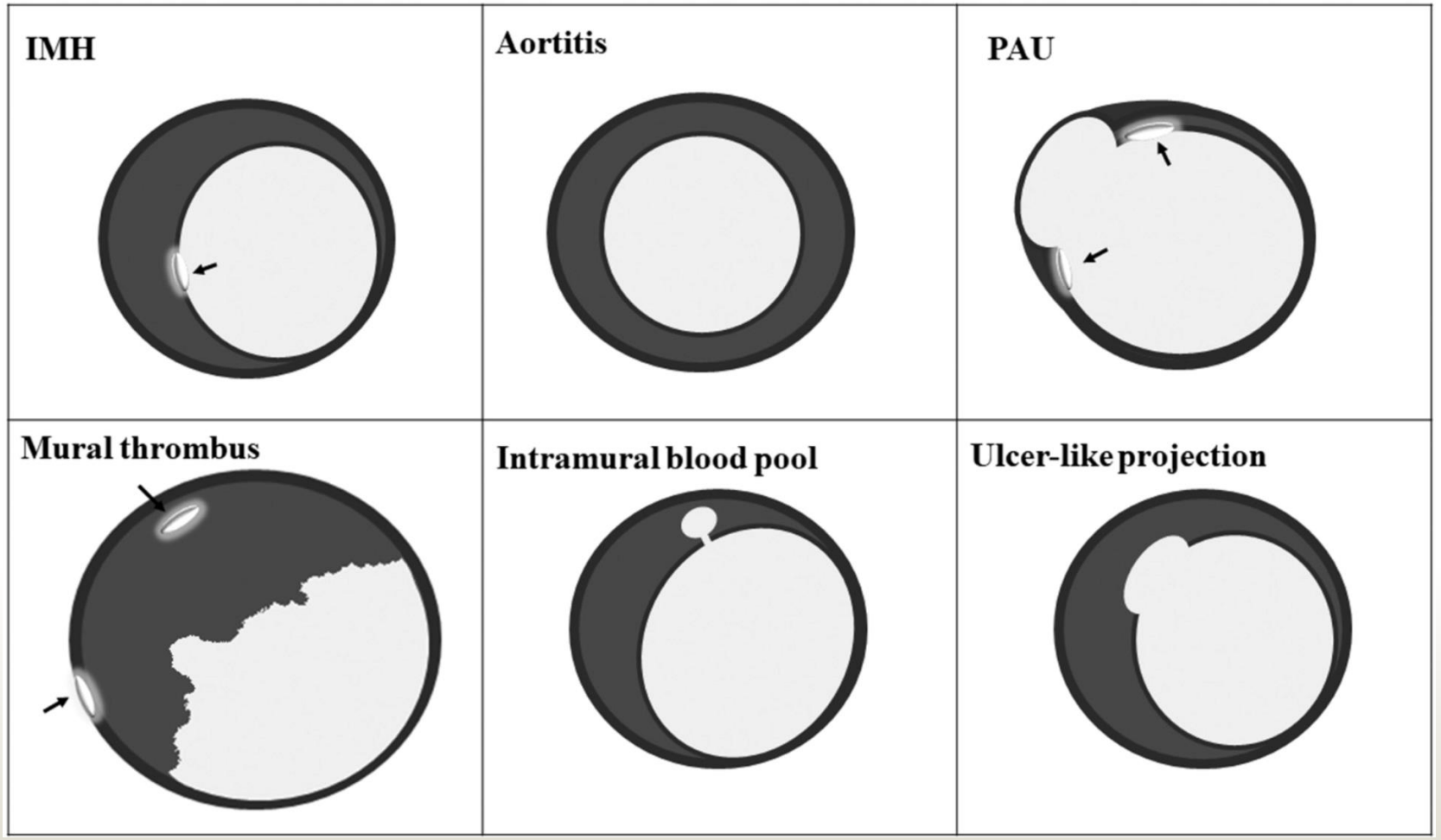
MURAL THROMBUS VS IMH - PAU VS ULPS VS IBPS

Mural thrombus = a crescentic wall thickening, usually with an irregular luminal surface.

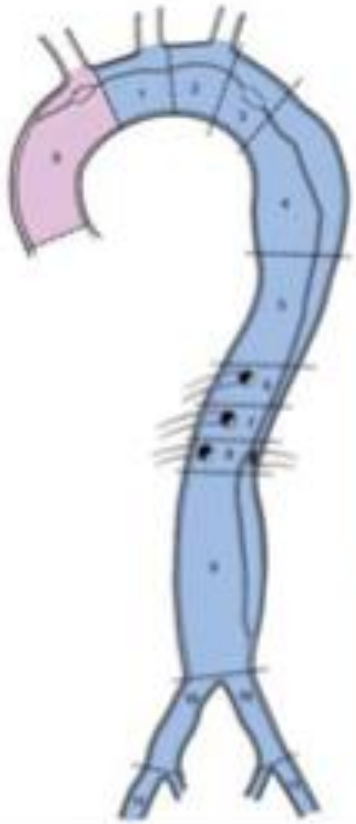
IMH displaces intimal calcifica-tions inward, whereas wall calcifications are located along the outer border of the aorta in mural thrombosis.

PAU VS ULPs VS IBPs. PAU and ULPs are not equivalent terms.

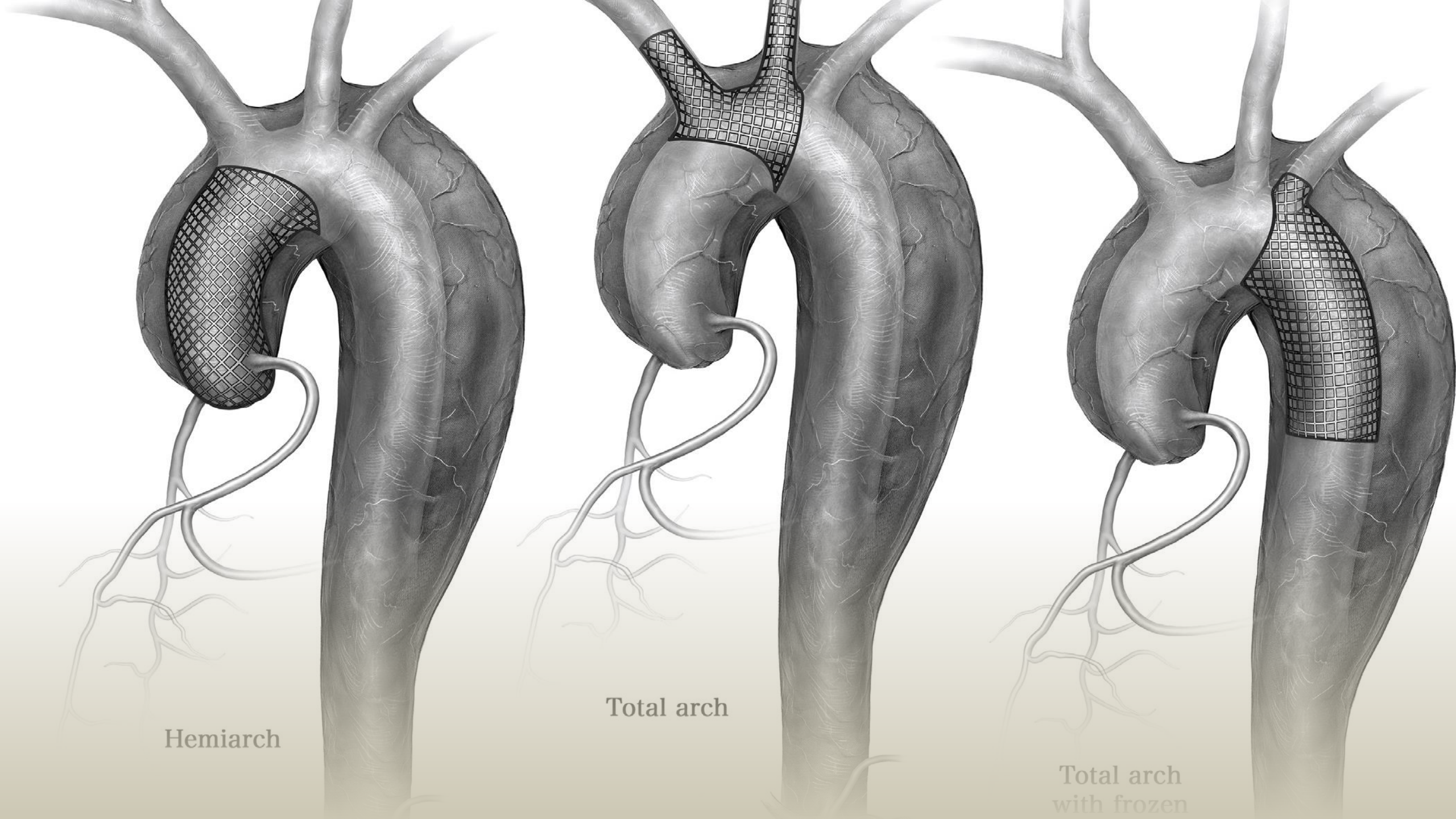
ULPs result from an intimal disruption in a segment with a dissecting hematoma and appear as small saccular areas of enhancement with wide mouths that protrude from the aortic lumen into the aortic wall . Frequently, they are not accompanied by atherosclerotic lesions (calcified plaques) and represent true entrance tears of acutely thrombosed CD. PAUs are wide-mouth saccular areas classically associated with atheromatous plaques. They are usually accompanied by some degree of IMH and typically produce a remodeling of the aortic wall contour.

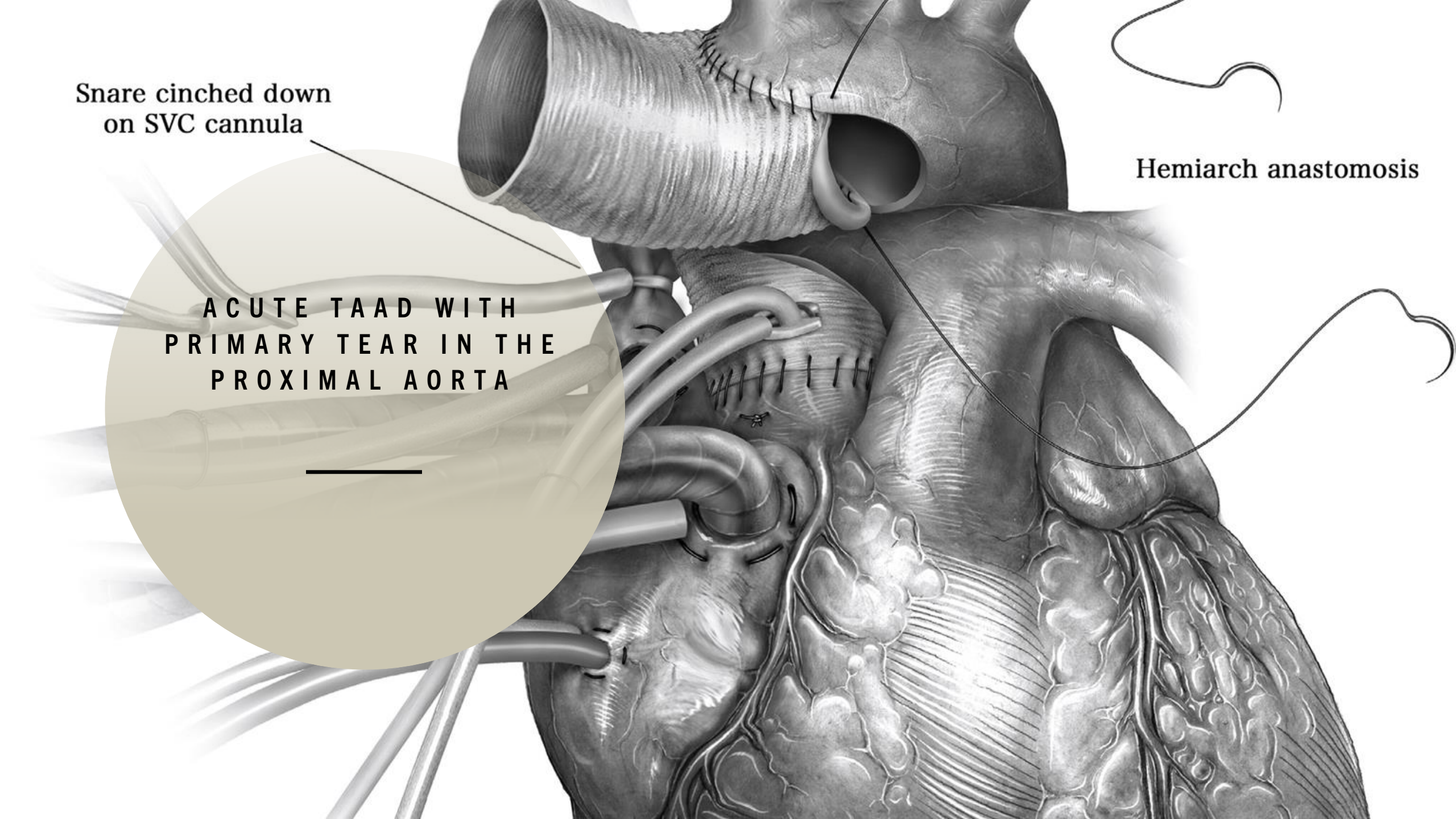


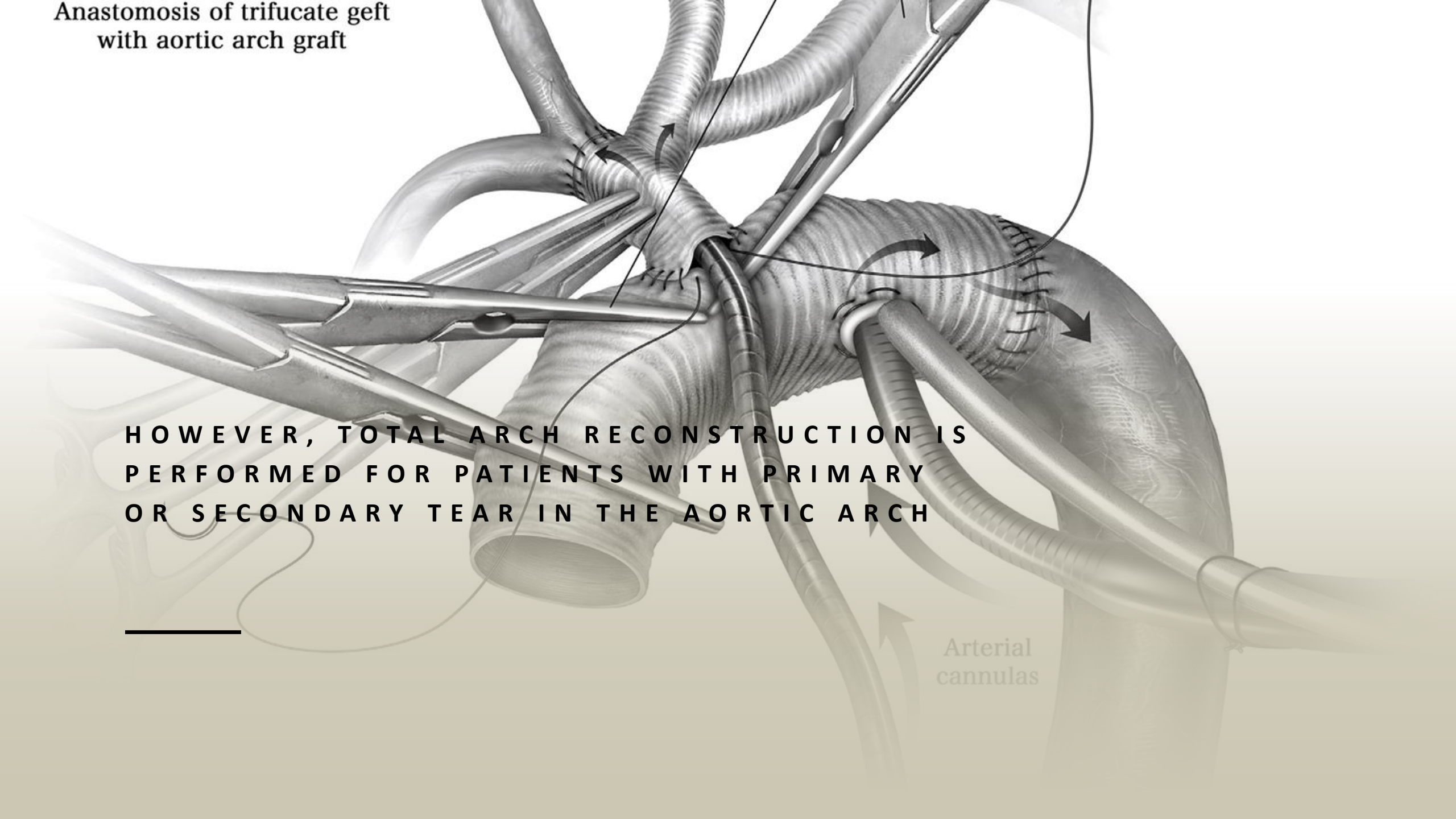
DISSECTIONS ARE DEFINED ANATOMICALLY ACCORDING TO THE LOCATION OF INTIMAL TEARS AND THE PROXIMAL AND DISTAL EXTENT OF THE DISSECTION PROCESS



Type	Proximal Extent	Distal Extent
A_D Entry tear: **Zone 0**	0	0
	1	1
	2	2
	3	3
	4	4
B_{PD} Entry tear: **≥Zone 1**	5	5
	6	6
	7	7
	8	8
	9	9
	10	10
I_D Unidentified entry tear involving **Zone 0**	11	11
	12	12

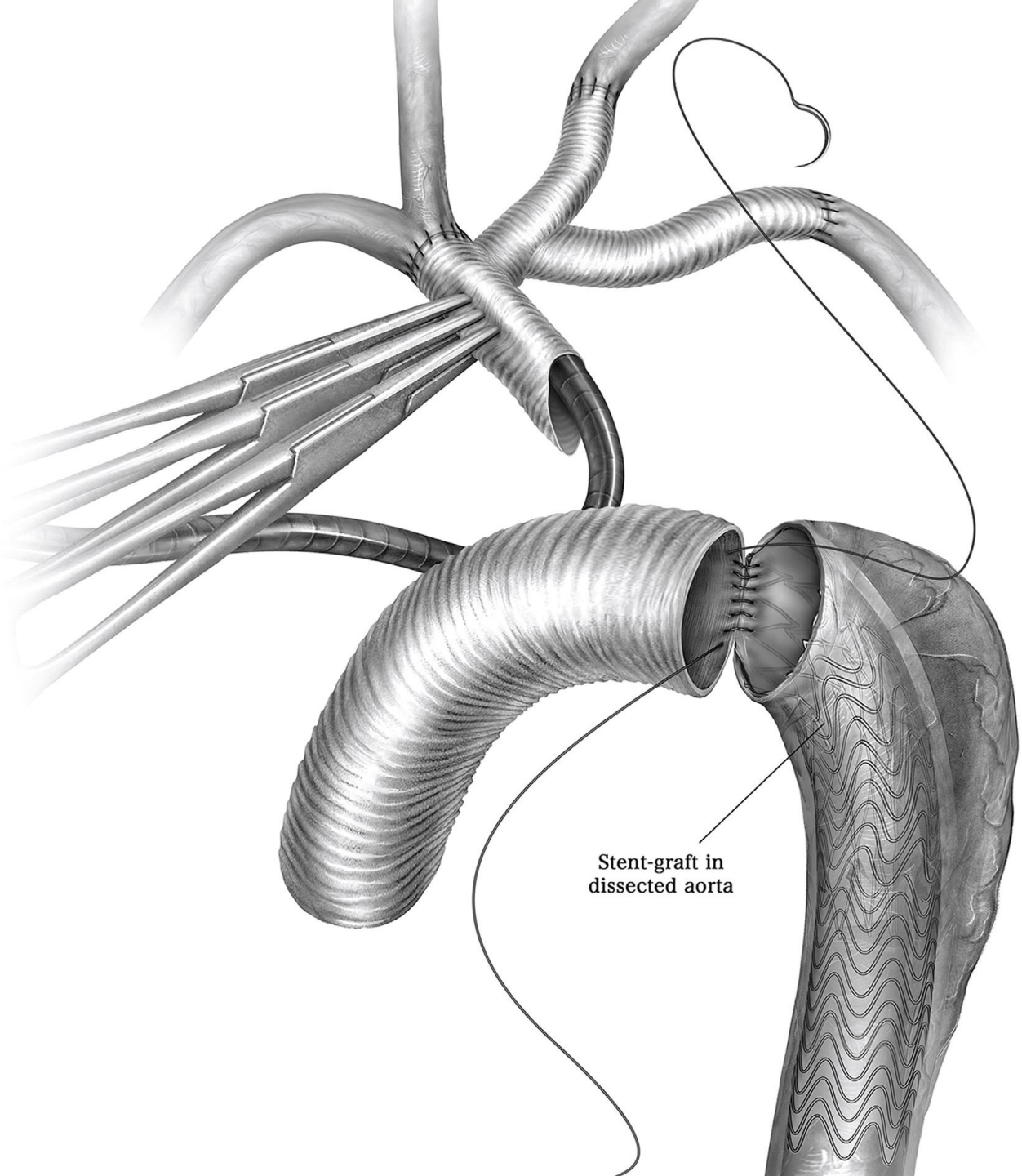

Hemiarch
Total arch
Total arch
with frozen


Snare cinched down
on SVC cannula
Hemiarch anastomosis
ACUTE TAAD WITH
PRIMARY TEAR IN THE
PROXIMAL AORTA

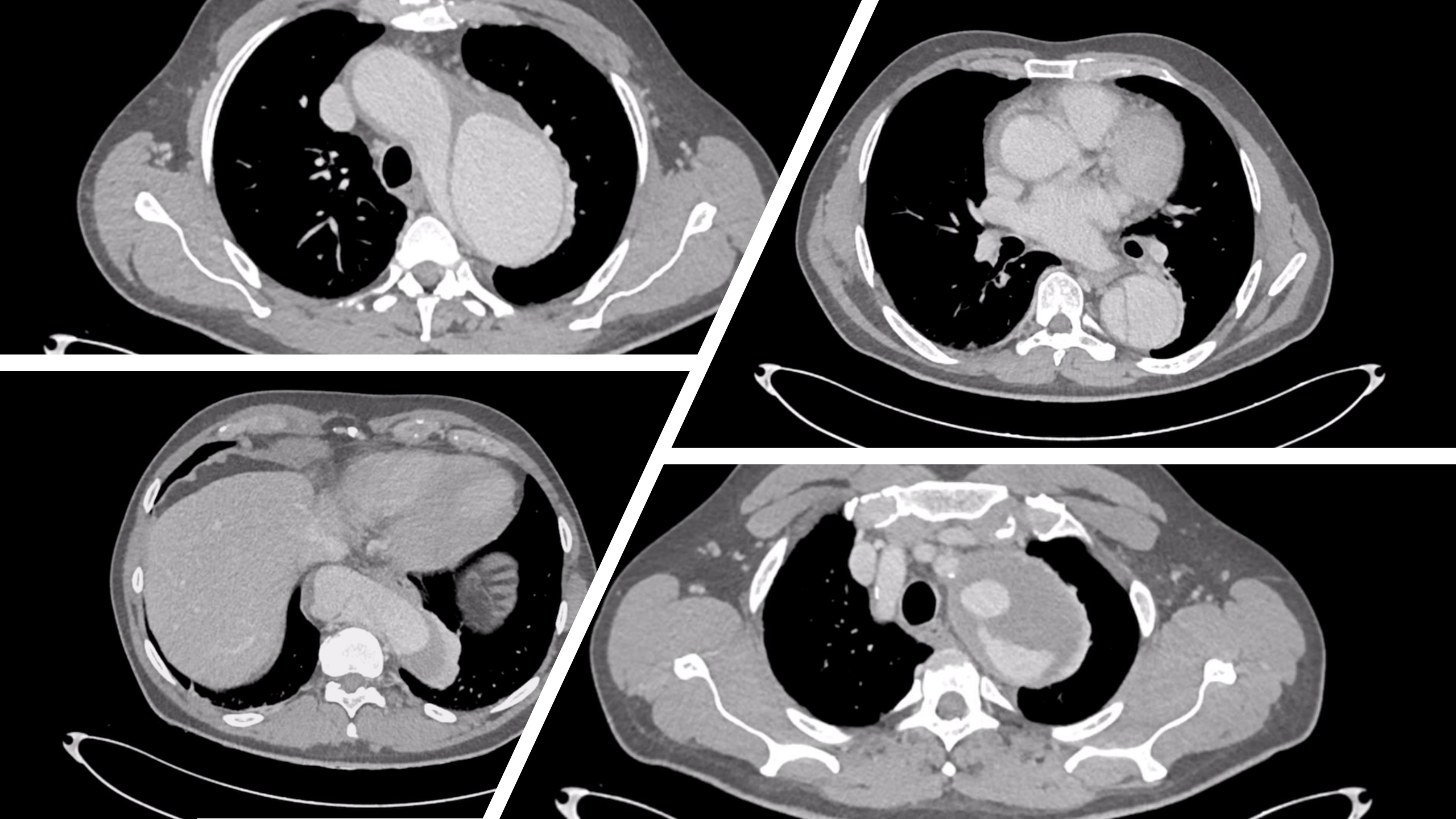


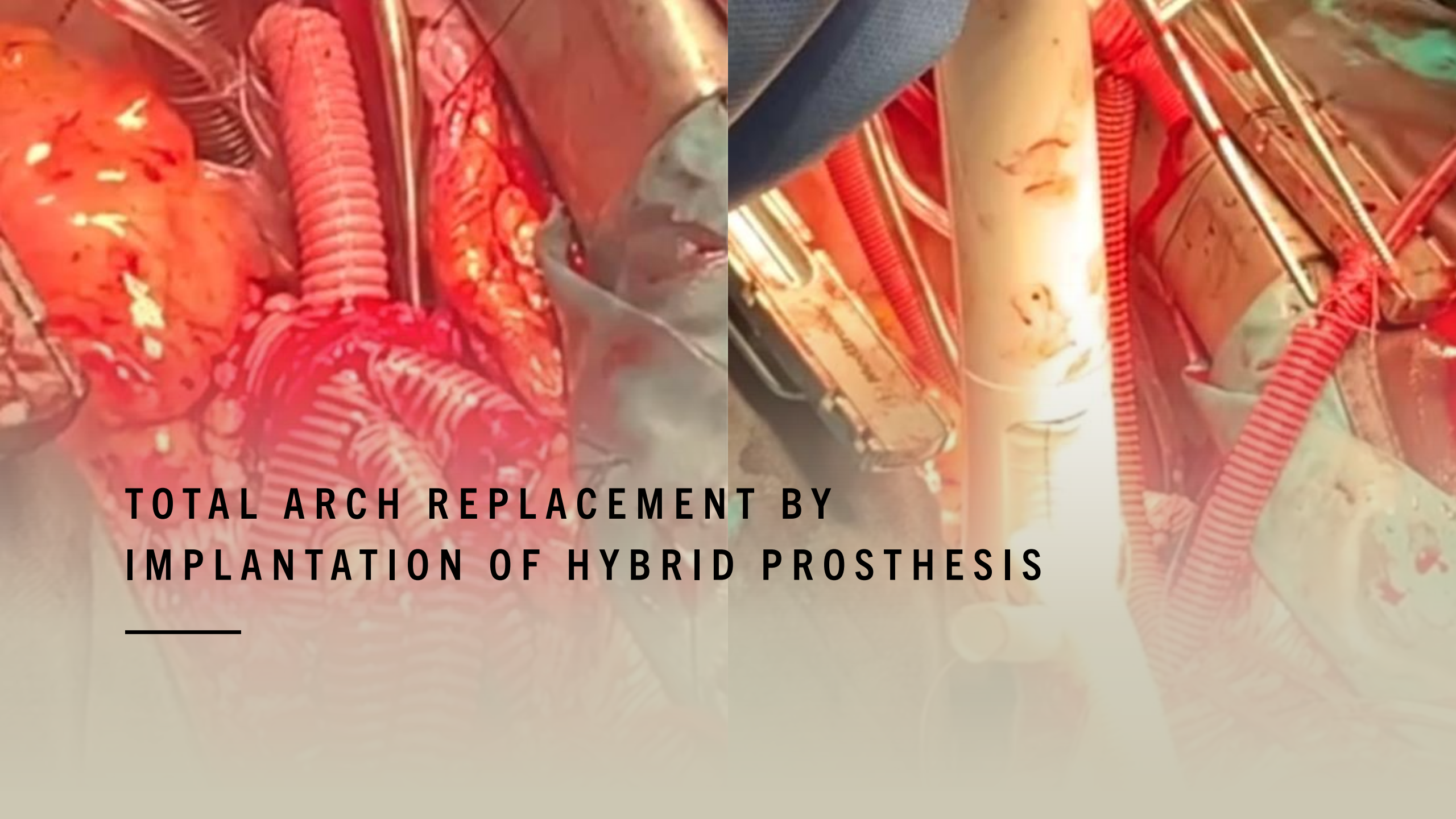
HOWEVER, TOTAL ARCH RECONSTRUCTION IS PERFORMED FOR PATIENTS WITH PRIMARY OR SECONDARY TEAR IN THE AORTIC ARCH

FINALLY, A FROZEN ELEPHANT TRUNK IS PERFORMED WHEN THERE IS A DISTAL ARCH TEAR

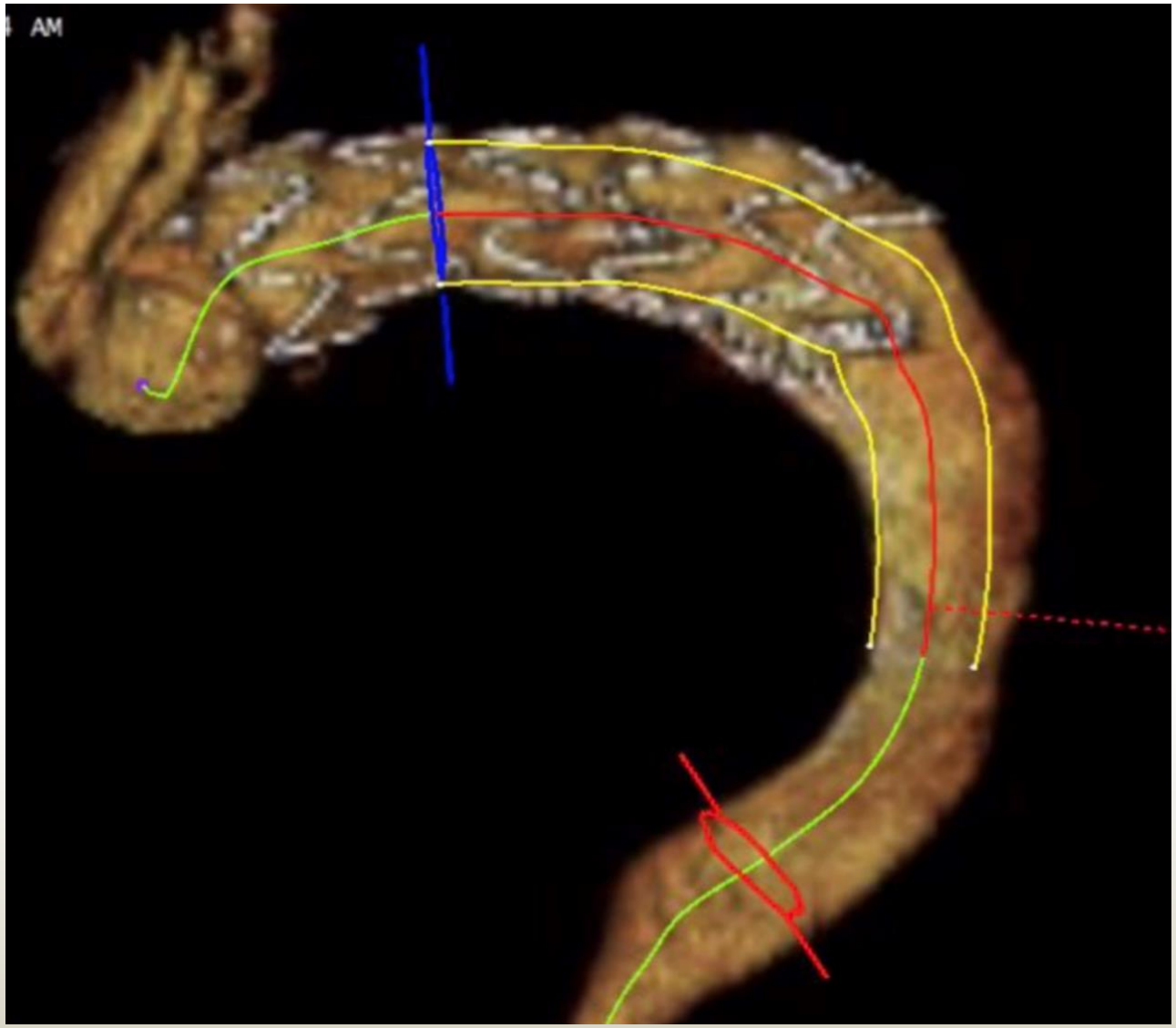


HYBRID PROSTHESIS

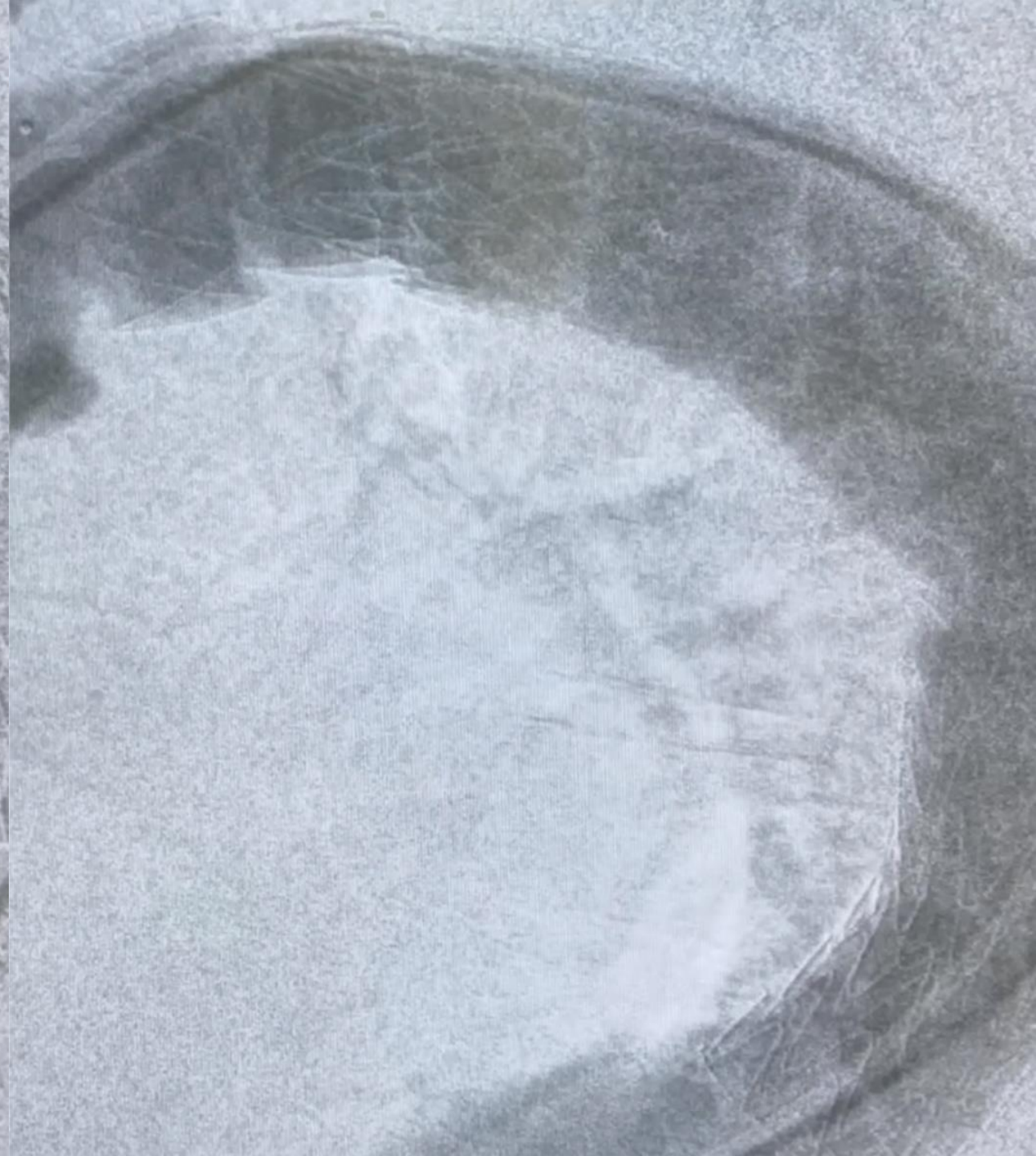




TOTAL ARCH REPLACEMENT BY IMPLANTATION OF HYBRID PROSTHESIS

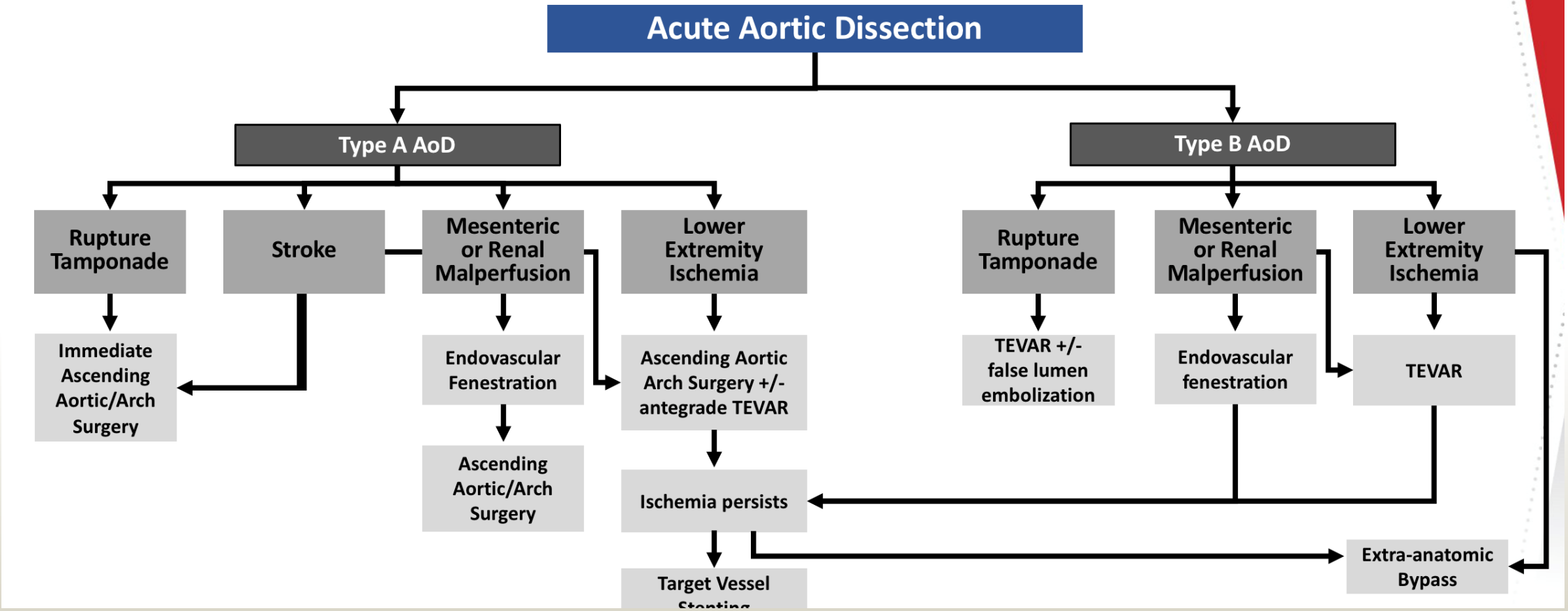

AM

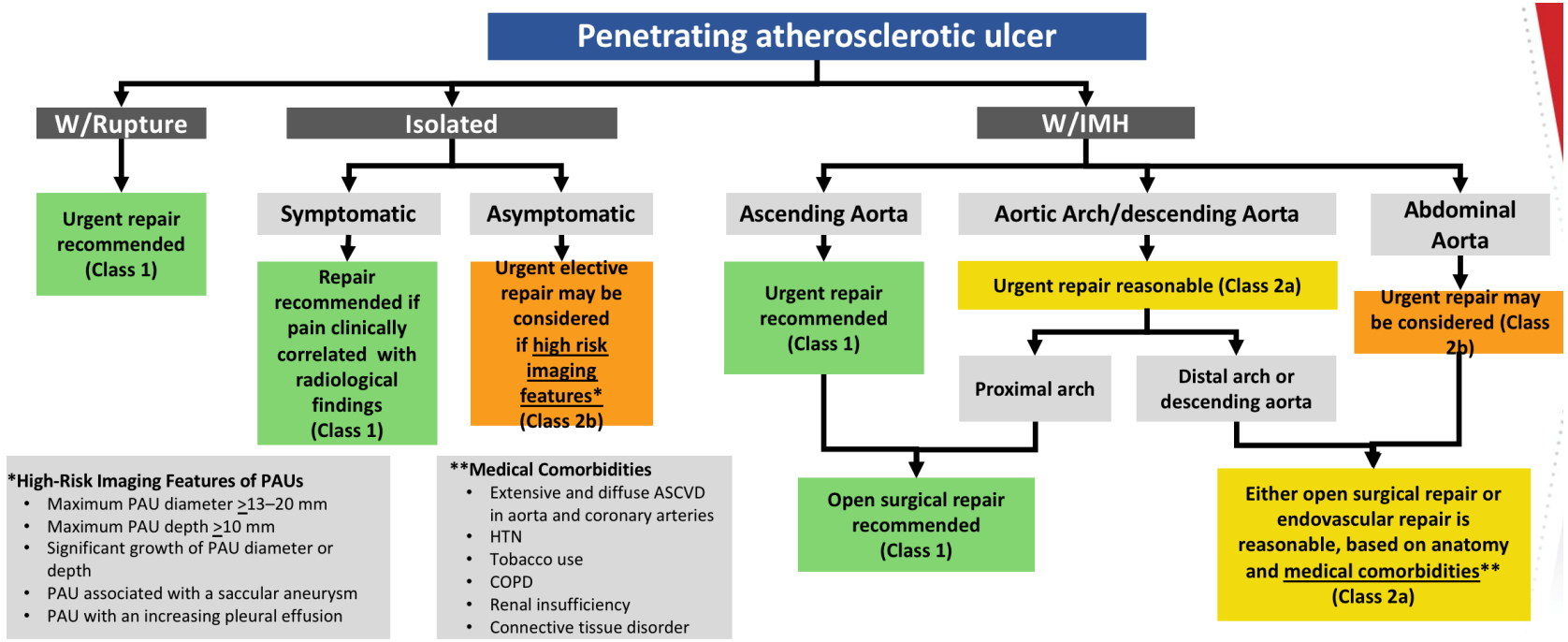
STAGED ENDOPROSTHESIS IMPLANT



MANAGEMENT OF PATIENTS WITH AAS: CURRENT STATUS

Acute Aortic Dissection: Malperfusion Treatment Options



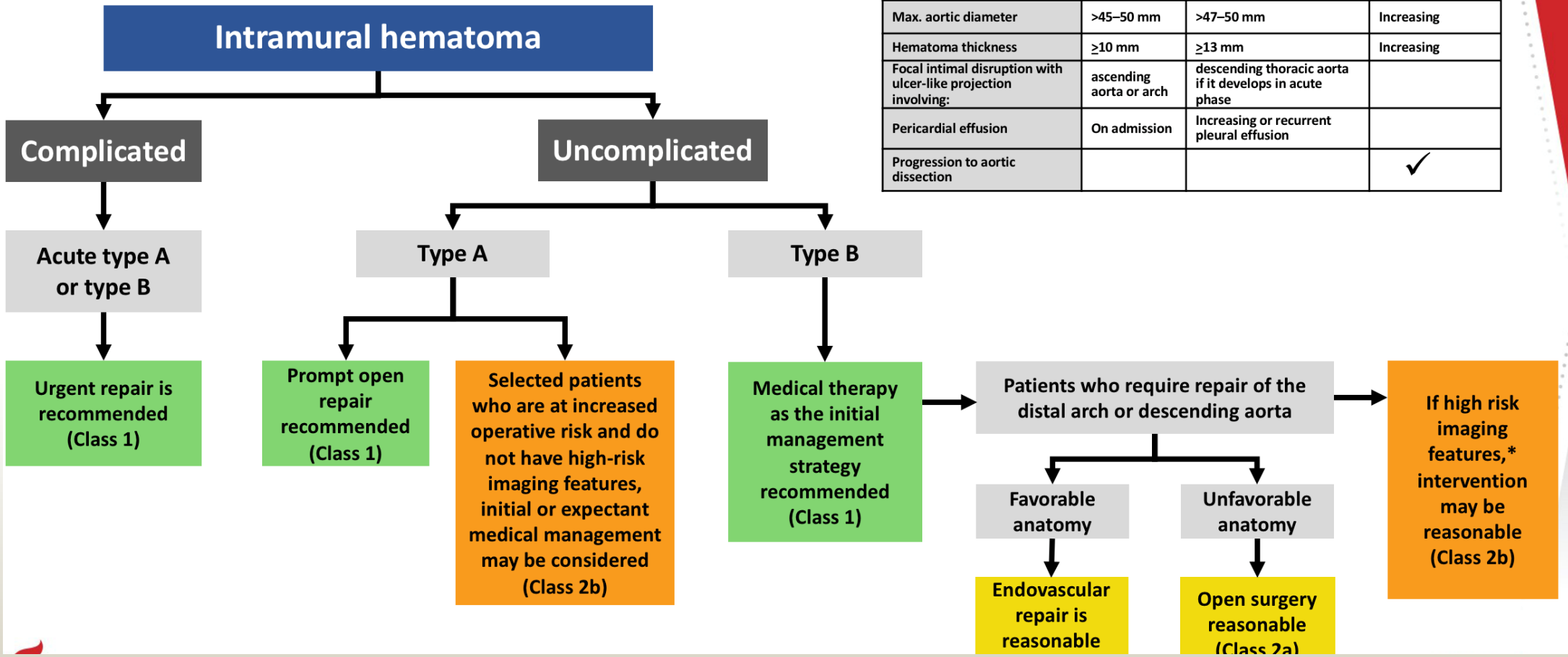


RECOMMENDATION FOR PENETRTING ATHEROSCLEROTIC ULCER AND TYPE OF REPAIR

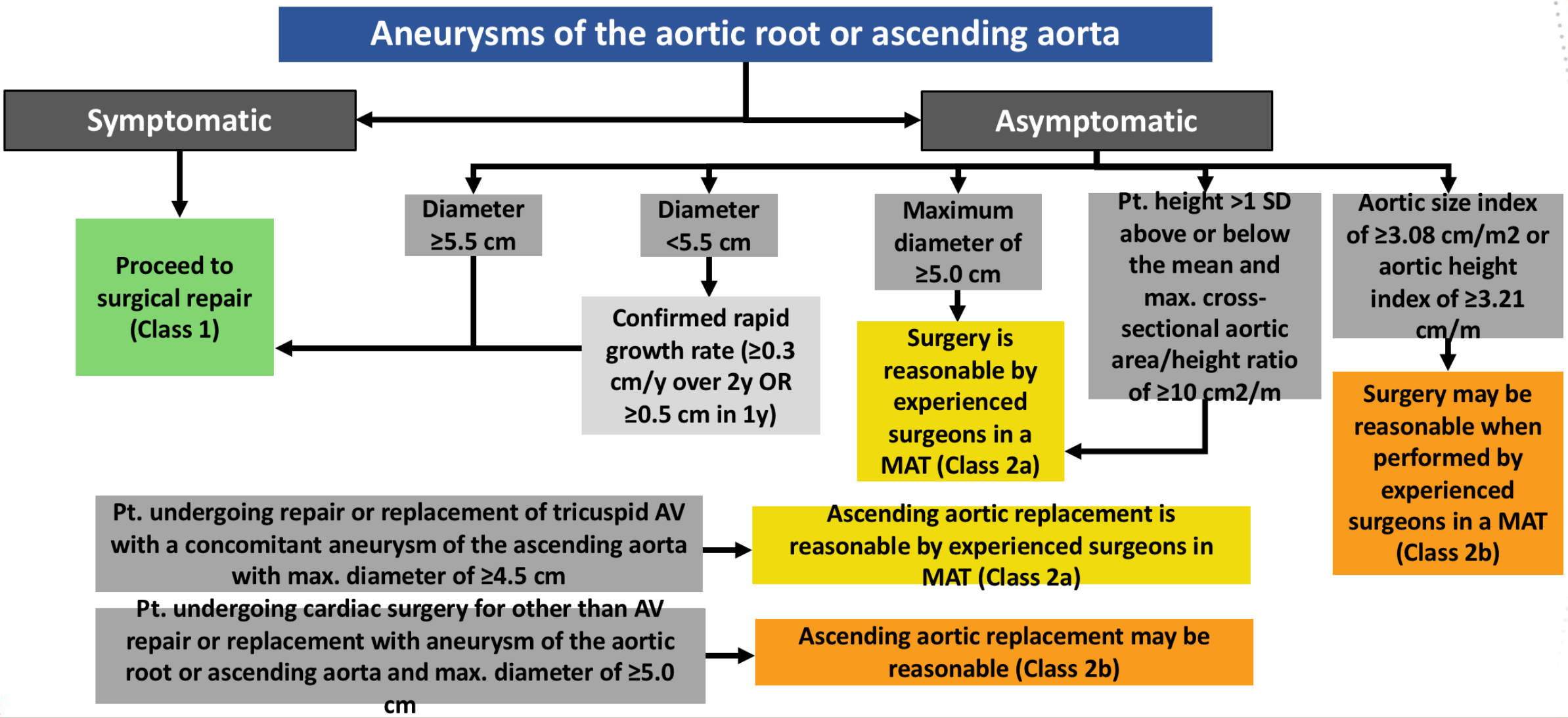
Recommendations for Management of Intramural Hematoma

*High-Risk Imaging Features of IMH

	Type A IMH	Type B IMH	Both Type A and Type B IMH
Max. aortic diameter	>45–50 mm	>47–50 mm	Increasing
Hematoma thickness	≥10 mm	≥13 mm	Increasing
Focal intimal disruption with ulcer-like projection involving:	ascending aorta or arch	descending thoracic aorta if it develops in acute phase	
Pericardial effusion	On admission	Increasing or recurrent pleural effusion	
Progression to aortic dissection			✓

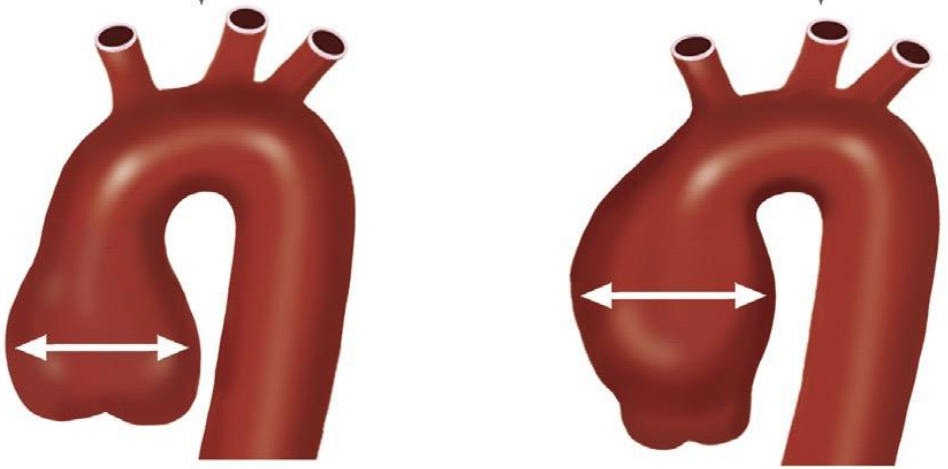


Recommendations for Surgery for Sporadic Aneurysms of the Aortic Root and Ascending Aorta



Aortic Dilation <5.5 cm with Acute Ascending Aortic Dissection

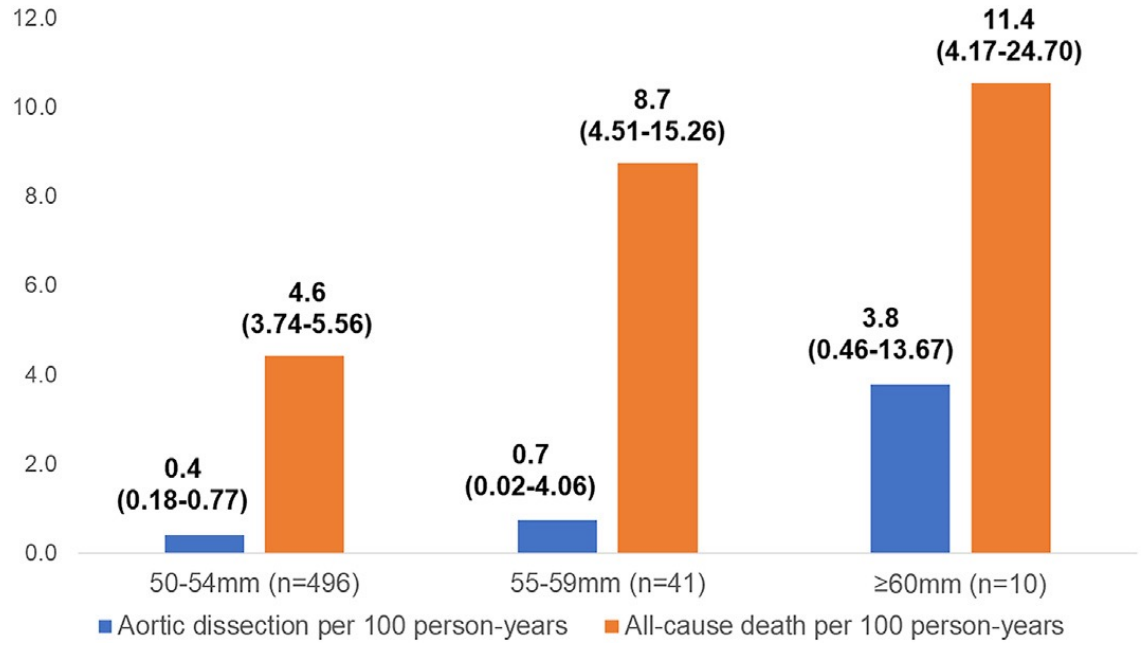
Location of Aortic Dilation Matters

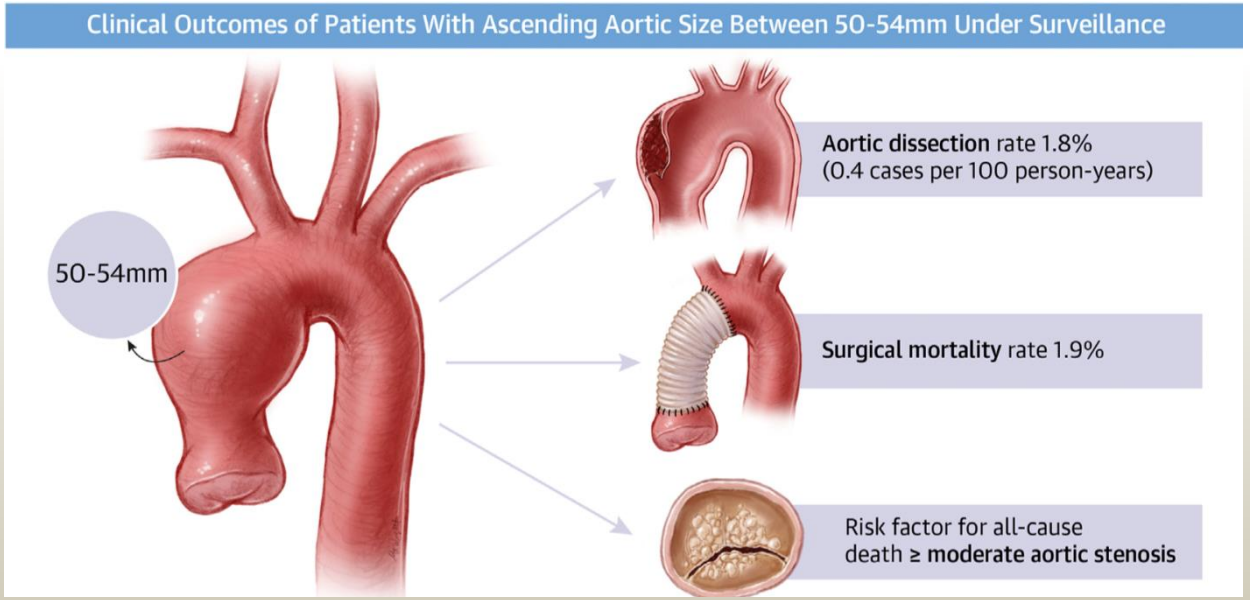


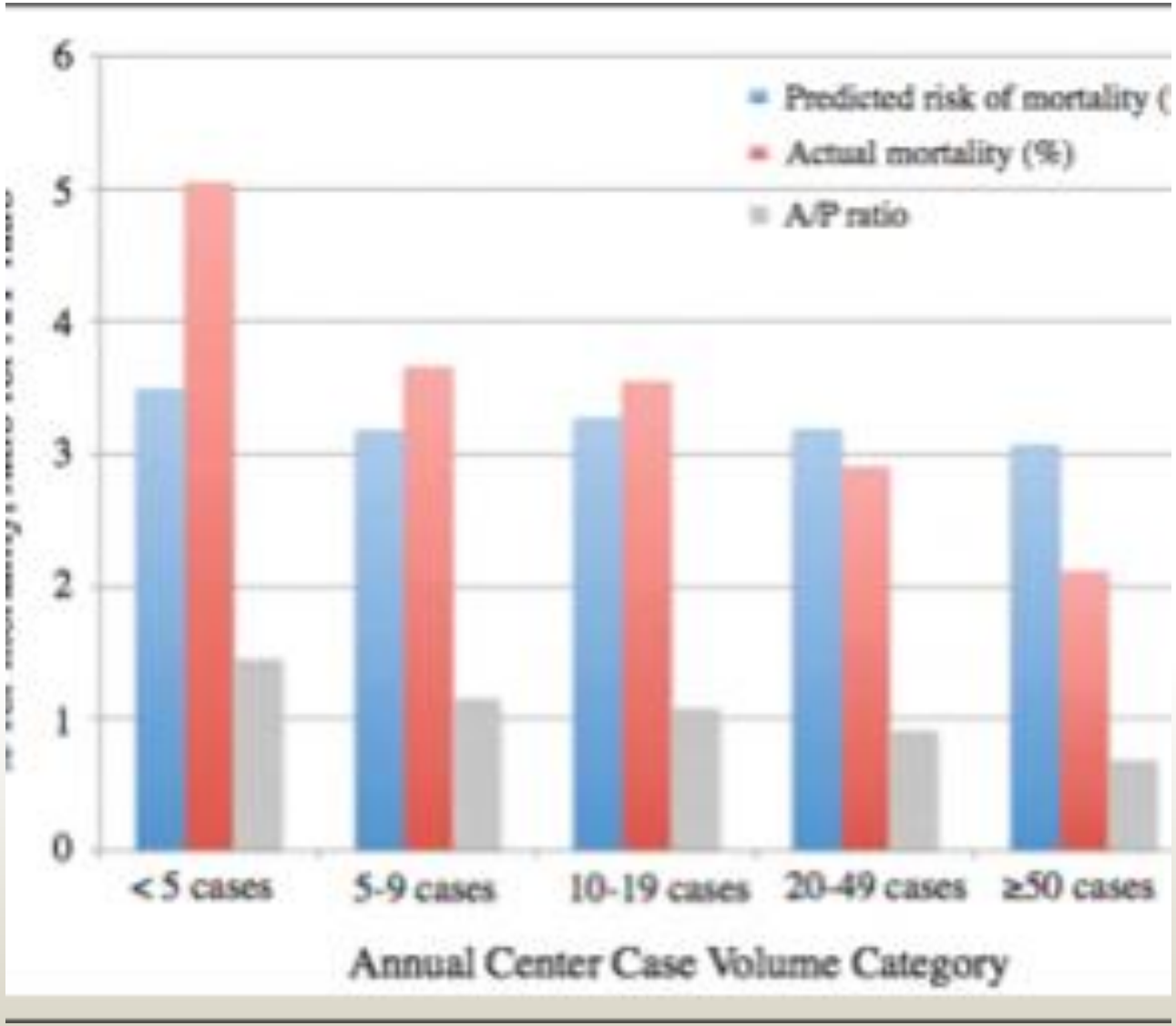
Mean Diameter of Dissection for Root: 4.6cm

Mean Diameter of Dissection for Ascending Aorta: 4.8cm

Take Home Message: Patients with a maximal dilation of the aortic root dissect at a smaller diameter than the ascending aorta. Further research into the mechanisms of this finding is warranted.


12.0
10.0
8.0
6.0
4.0
2.0
0.0
0.4
(0.18-0.77)
4.6
(3.74-5.56)
0.7
(0.02-4.06)
8.7
(4.51-15.26)
3.8
(0.46-13.67)
11.4
(4.17-24.70)
50-54mm (n=496)
55-59mm (n=41)
≥60mm (n=10)
Aortic dissection per 100 person-years
All-cause death per 100 person-years


Clinical Outcomes of Patients With Ascending Aortic Size Between 50-54mm Under Surveillance
50-54mm
Aortic dissection rate 1.8%
(0.4 cases per 100 person-years)
Surgical mortality rate 1.9%
Risk factor for all-cause
death ≥ moderate aortic stenosis


6
5
4
3
2
1
0
Predicted risk of mortality (
Actual mortality (%)
A/P ratio
< 5 cases
5-9 cases
10-19 cases
20-49 cases
≥50 cases
Annual Center Case Volume Category

CONCLUSIONS

Since the publication of the Stanford classification of aortic dissection in 1970.

- Diagnostic tools and management of ATAAD have undergone substantial evolution.
- Technical complexity has increased with more extensive repair involving the proximal and distal extent of the aorta.
- Long-term survival after ATAAD repair has improved over time.
- Short-term proximal aortic reoperation incidence has decreased over the decades.

Operative Details After Weighting

	2000-2009 (n = 282.3)	2010-2019 (n = 523.7)	p Value
Perfusion details			
Cross-clamp time, min	120.2 ± 54.0 (108.0 [77.0-154.0])	131.5 ± 60.0 (122.0 [85.0-168.0])	0.010
Cardiopulmonary bypass time, min	203.9 ± 69.9 (190.0 [156.0-244.0])	228.4 ± 164.3 (200.0 [158.0-258.0])	0.0042
Circulatory arrest time, min	31.9 ± 14.0 (30.0 [23.0-37.0])	38.1 ± 104.1 (27.0 [21.0-35.0])	0.22
Cerebral protection approach			<0.0001
Antegrade cerebral protection	122 (57.3)	340 (75.1)	
Retrograde cerebral protection	18 (8.9)	83 (18.3)	
Antegrade and retrograde cerebral protection	0 (0)	4 (1.0)	
Hypothermia alone	72 (33.8)	23 (5.3)	
Arterial cannulation strategy			
Direct aortic	12 (4.4)	109 (20.8)	<0.0001
Femoral	152 (54.0)	75 (14.4)	<0.0001
Axillary	129 (46.0)	297 (56.8)	0.0032
Innominate	5 (1.8)	48 (9.3)	<0.0001
Other	9 (3.3)	5 (1.0)	0.018
Lowest bladder temperature, °C	21.7 ± 4.5 (21.0 [18.4-23.5])	25.1 ± 3.4 (25.6 [22.4-28.0])	<0.0001
Intraoperative transfusion	135 (54.7)	411 (85.0)	<0.0001
Packed red cells, U	4.8 ± 5.0 (4.0 [2.0-7.0])	2.2 ± 3.9 [1.0 [0-3.0])	<0.0001
Platelets, U	2.8 ± 1.9 (2.0 [2.0-4.0])	2.1 ± 1.6 [2.0 [1.0-3.0])	<0.0001
Fresh frozen plasma, U	8.3 ± 5.2 (8.0 [5.0-11.0])	4.3 ± 4.3 [3.0 [1.0-6.0])	<0.0001
Cryoprecipitate, U	8.0 ± 8.1 (10.0 [0-10.0])	2.7 ± 4.7 [2.0 [1.0-3.0])	<0.0001

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THANKS FOR ATTENTION