Cerebrovascular Disease

DR. Mohammadreza Sobhiyeh

Associate professore of vascular and Endovascular srgury of Kermanshah university of medical sciences

Background

1- The relationship between extracranial carotid disease and stroke had been surmised previously as early as 1875, when Gowers reported on a patient with carotid occlusion, left visual loss, and right hemiplegia.

2-Fisher's prediction of surgical treatment for carotid atherosclerosis was soon realized.(1954) .

3- Te frst successful carotid endarterectomy (CEA) was performed by DeBakey in 1953 and reported along with a 19-year symptom-free follow-up in 1975 .

Background

4- Te concept of using endovascular therapy for the treatmentof carotid artery stenosis was frst proposed in 1977 by Mathias, who reported successful results of carotid artery angioplasty using adapted peripheral arterial angioplasty technology.

5- In 1990 Teron et al. published their technique of performing carotid angioplasty using cerebral protection to prevent brain embolism.

CURRENT GUIDELINES

1- Because of the perceived higher periprocedural risk of stroke with CAS compared with CEA, based on results from the previously described RCTs, the current guidelines for use of CAS for treating carotid artery stenosis are limited .

2-A consensussupports the use of CAS in :

- symptomatic patients with high-grad stenoses who are deemed too high medical risk to undergo open surgery.

- the use of CAS in a lower medical risk population of patients with higher anatomic risk factors for CEA such as restenosis or neck radiation.

- Te use for CAS for patients who are low or moderate risk without prior neck radiation or surgery, or those who have asymptomatic carotid artery disease, is less well supported

Symptomatic Carotid Artery Stenosis

- CAS is indicated as an alternative to CEA for symptomatic patients at average or low risk of
complications associated with endovascular intervention when the diameter of the lumen
of the internal carotid artery is reduced by more than 70% as documented by noninvasive
imaging or more than 50% as documented by catheter angiography and the anticipated
rate of periprocedural stroke or mortality is less than 6%

ACC/AHA Recomendations

- CAS is indicated as an alternative to CEA for symptomatic patients at average or low risk of complications associated with endovascular intervention when the diameter of the lumen of the internal carotid artery is reduced by more than 70% as documented by noninvasive imaging or more than 50% as documented by catheter angiography and the anticipated rate of periprocedural stroke or mortality is less than 6%.

- Class I; level of evidence B

- Among patients with symptomatic severe stenosis (≥70%) in whom the stenosis is diffcult to access surgically, medical conditions are present that greatly increase the risk for surgery, or when other specifc circumstances exist, such as radiation-induced stenosis or restenosis after CEA, CAS may be considered.
Class IIb; level of evidence B

- CAS in the above setting is reasonable when performed by operators with established periprocedural morbidity and mortality rates of 4%–6%, similar to those observed in trials of CEA and CAS.

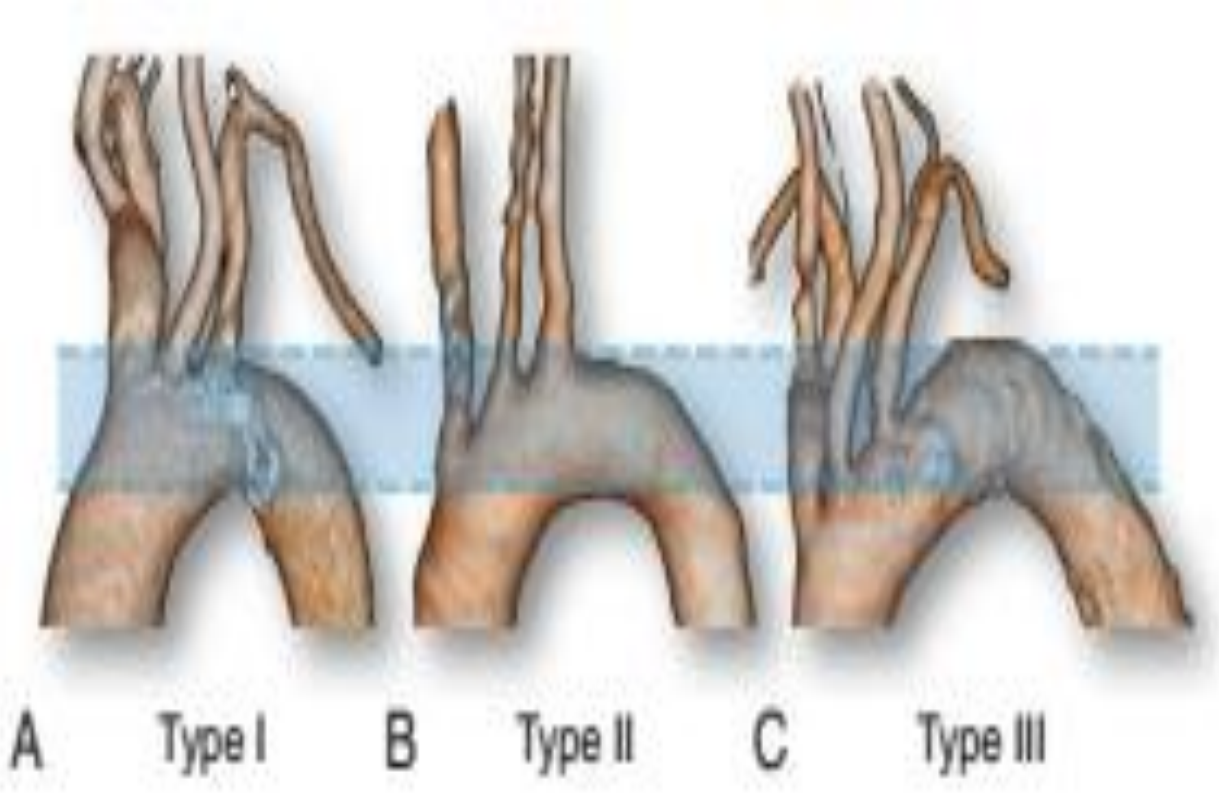
Aortic arch classification

Type I: the great vessels
arise above or in the same horizontal plane of the outer curvature of the arch.

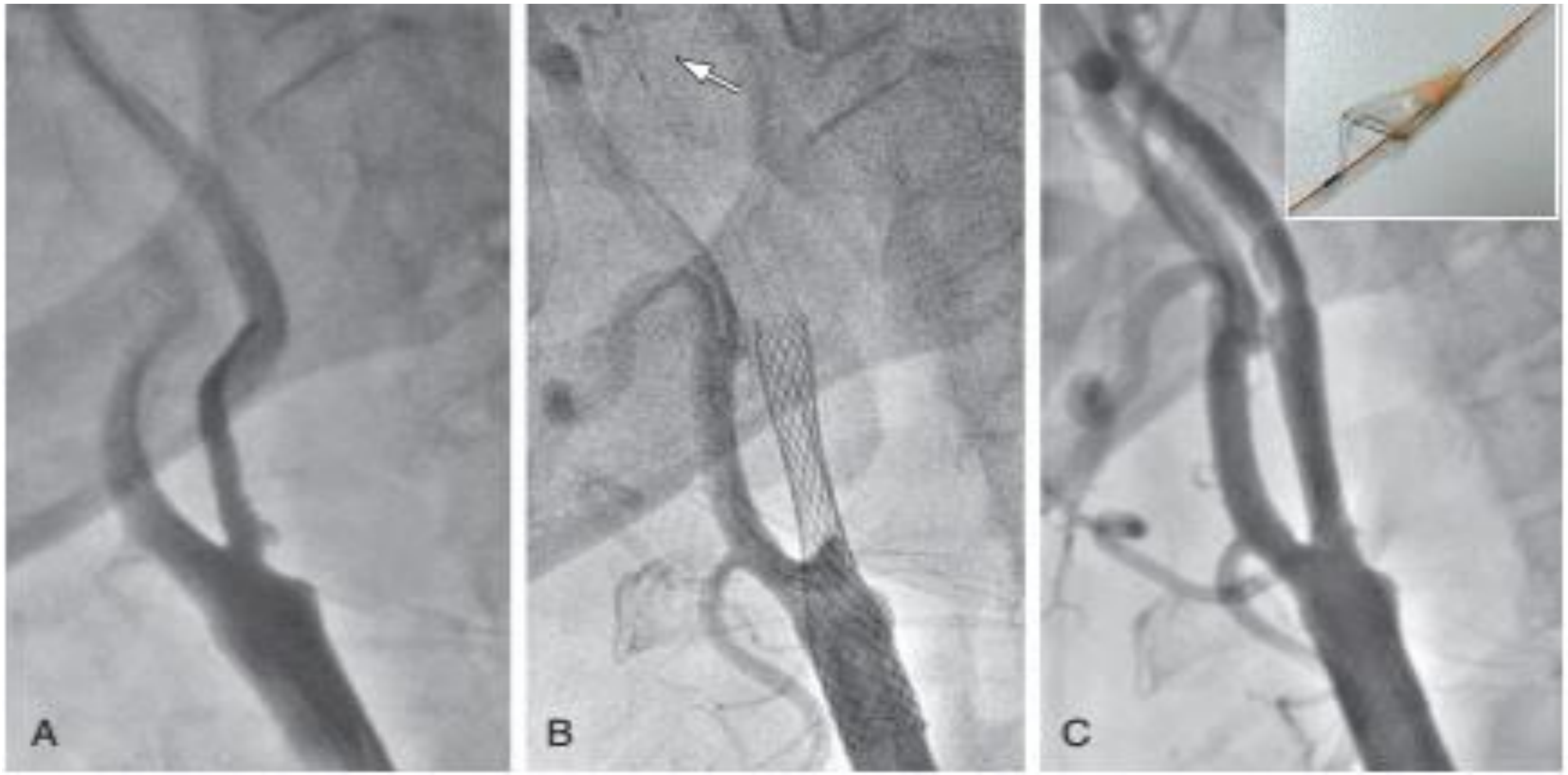
Type II: the origin of the innominate artery lies between
the horizontal planes of the outer and inner curvatures of the aortic arch.

Type III: the innominate artery lies below the horizontal plane
of the inner curvature of the arc

Normal variation of aortic arch



Ulcerated severe carotid stenosis in a symptomatic patient.



Complications of CAS

- Local complications:
- Hemorrhage ,Arterial occlusion, Retroperitoneal hemorrhage, Infection, Pseudoaneurysm, Arteriovenous Fistula, Vessel thrombosis , Dissection with limb ischemia
- Cardiovascular complications:
- Severe bradycardia, Hypotension from stretching of carotid bulb
- MI , Deterioration of CHF due to osmotic load associated with administration of contrast agent.
- Neurologic complications:
- Spasm , Thrombosis, Embolization, Intracranial hemorrhage, Neurocognitive complications.

Clinical factors associated with increased stroke risk

Patients characteristics:

Neurologic symptoms, Contralateral stroke, Contralateral carotid occlusion, Renal insufficiency, Smoking, Clinical silently emboli by TCD or MRI/CT

Plaque Characteristics:

Diameter stenosis, Plaque ulceration, Plaque progression, Echolucent Plaque, Disrupted fibrous cap, Active inflammation by MRI, Discrete white areas within plaque

Brachiocephalic Artery

The innominate, left common carotid, and left subclavian arteries constitute the branches of the transverse aortic arch. Known as the brachiocephalic arteries, together these vessels supply blood to the upper extremities, head, and neck.

Atherosclerosis of these vessels can lead to flow-limiting stenosis or distal embolization, which can result in transient ischemic attacks, stroke, upper extremity ischemia, and vertebrobasilar insuffciency.

In addition to atherosclerosis, aneurysmal degeneration, dissection, vasculitides (including Takayasu disease and giant cell arteritis), infection, and fbrosis, such as from radiation therapy, can develop in the brachiocephalic arteries.

Clinical findings

The clinical manifestations of lesions involving the brachiocephalic arteries depend on the etiology of the disease, the presence of single-vessel or multivessel disease, and anatomic location.

Atherosclerosis is the most common disease affecting the brachiocephalic arteries.

Severe disease is defned as stenosis greater than 75% of the vessel's diameter.

Single-artery occlusion involving the origin of the subclavian artery can cause subclavian-vertebral steal and lead to vertebrobasilar insuffciency, myocardial ischemia (in the case of prior LIMA coronary artery bypass grafting), and hemiparesis or aphasia.

Indications of Revascularization

- Symptomatic lesions:
- Stroke or TIA due to atheroembolism,low flow states , leading to vertebrobasilar insufficiency or less common , cortical symptoms.
- Steal syndrome from proximal innominate or subclavian artery occlusion , leading to vertebrobasilar insufficiency, myocardial ischemia after internal mammary artery bypass or Anterior cerebral TIA /stroke.

- Asymptomatic lesions:
- Some authors suggest that asymptomatic patients with sever stenosis(>75%) of the innominate or common carotid artery should undergo revascularization if they have reasonable surgical risk

Asymptomatic lesions

- There is agreement , that revascularization of severe (>75%) asymptomatic stenosis of subclavian artery should be performed if coronary artery bypass with the ipsilateral internal mamery artery is planned.

Severe stenosis of LSA

Anatomic Determinations of procedural risk with arch vessel Endovascular Therapy

- Anatomical lesions favorable to intervention:
- Stenosis
- Concentric lesions
- Nonostial lesions
- Lesions with vessel origin distal in the arch
- Noncalcified lesions
- Nonulcerated lesion

Anatomical Determinations of…….

- Anatomical lesions unfavorable to intervention:
- Occlusion
- Eccentric lesions
- Lesions with proximal rotation of the vessel origin
- Calcification
- Ulceration
- Symptomatic lesions
- Lesions bear the vertebral artery origin

Lower extremity

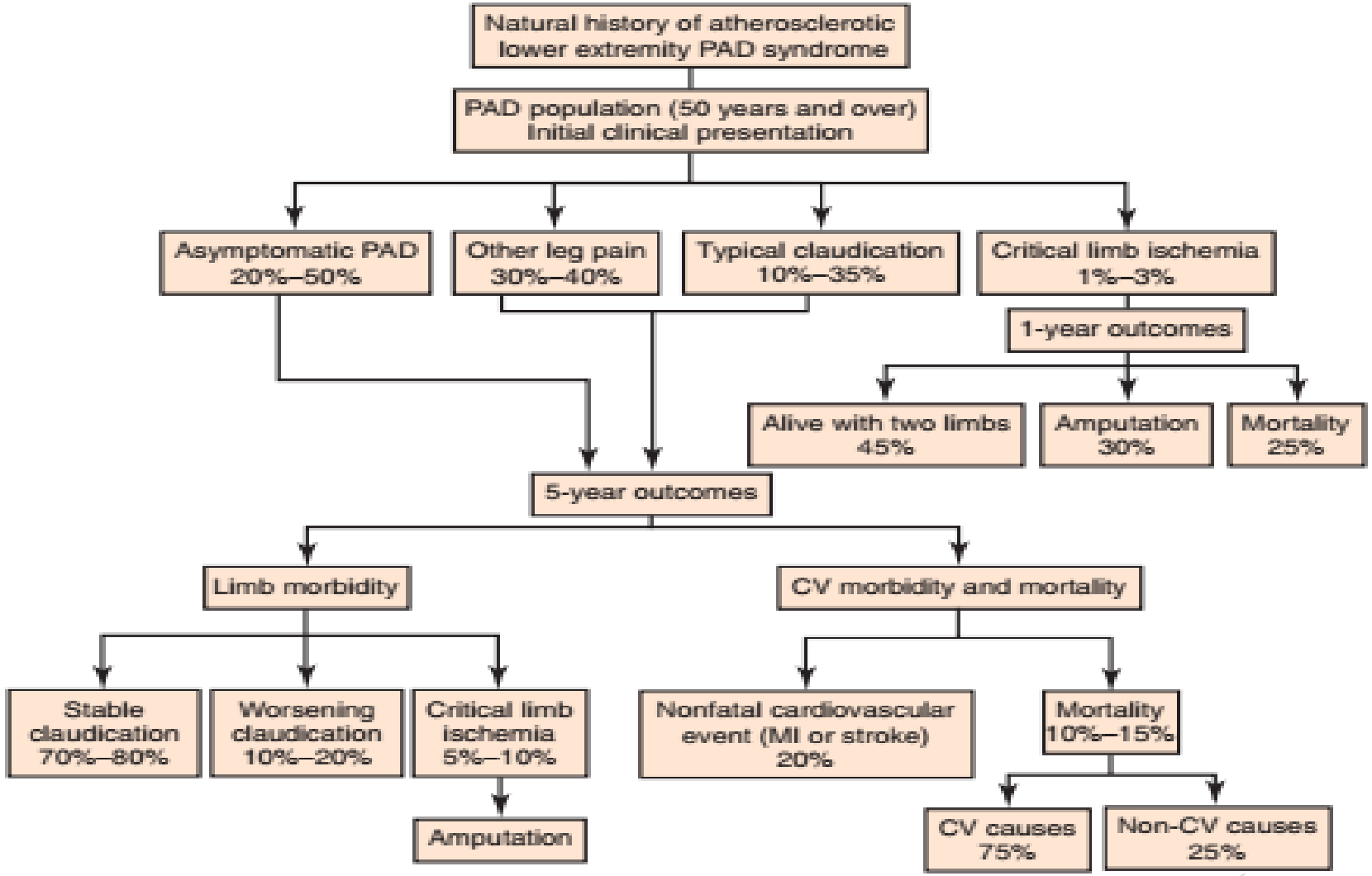
- Chronic lower extremity ischemia due to peripheral arterial disease (PAD) is the most common cause of walking disability seen by vascular specialists.

- The manifestation of chronic lower extremity ischemia often include pain produced by varying degrees of ischemia (intermittent claudication) , to ischemia rest pain, ulcer , gangrene.

Nonatherosclerotic causes of intermittent claudication

- Thromboangiitis obligerans
- Popliteal aneurysm
- Aortic coarctation
- Fibromuscular dysplasia
- Takamatsu's disease
- Popliteal entrapment
- Primary vascular tumor
- Adventitial cyst of popliteal artery
- Endocibrosis of external Iliac artery(iliac artery syndrome in cyclists)

Natural History of PAD



Stages of chronic limb ischemia

Fontaine Grade	Rutherford Category	Clinical Description	Objective Criteria
I	0	Asymptomatic	Normal treadmill or reactive hyperemia test
IIa[a]	1	Mild claudication	Completes treadmill exercise[b]; AP after exercise >50 mm Hg but at least 20 mm Hg lower than resting value
IIb[a]	2	Moderate claudication	Between categories 1 and 3
	3	Severe claudication	Cannot complete standard treadmill exercise[b]; AP after exercise <50 mm Hg
III[a]	4	Ischemic rest pain	Resting AP <30–50 mm Hg; ankle or metatarsal PVR flat or barely pulsatile; TP <30 mm Hg
IV	5	Minor tissue loss	Resting AP <50–70 mm Hg; ankle or metatarsal PVR flat or barely pulsatile; TP <40 mm Hg in nondiabetics, <50 mm Hg in diabetics; $tcPO_2$ <30 mm Hg
	6	Major tissue loss[c,d]	Same as Rutherford 5 (Fontaine IV)

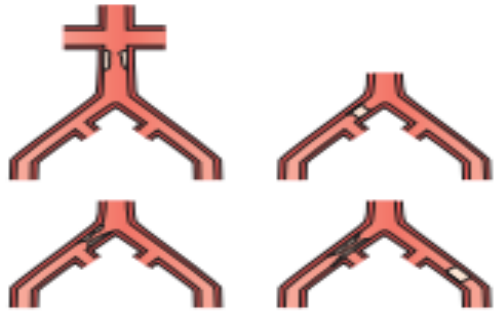
Trans Atlantic Inter Society Consensus (TASC) Classification of Aortoiliac lesions

TYPE A LESIONS

- Unilateral or bilateral stenoses of CIA
- Unilateral or bilateral single short (≤3 cm) stenosis of EIA

TYPE B LESIONS

- Short (≤3 cm) stenosis of infrarenal aorta
- Unilateral CIA occlusion
- Single or multiple stenoses totaling 3-10 cm involving the EIA not extending into the CFA
- Unilateral EIA occlusion not involving the origins of internal iliac or CFA

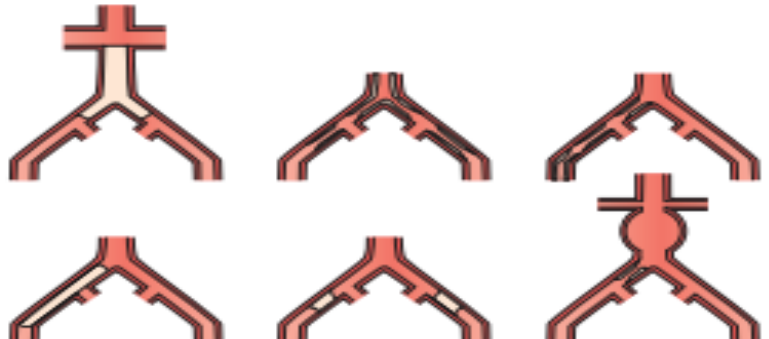


TYPE C LESIONS

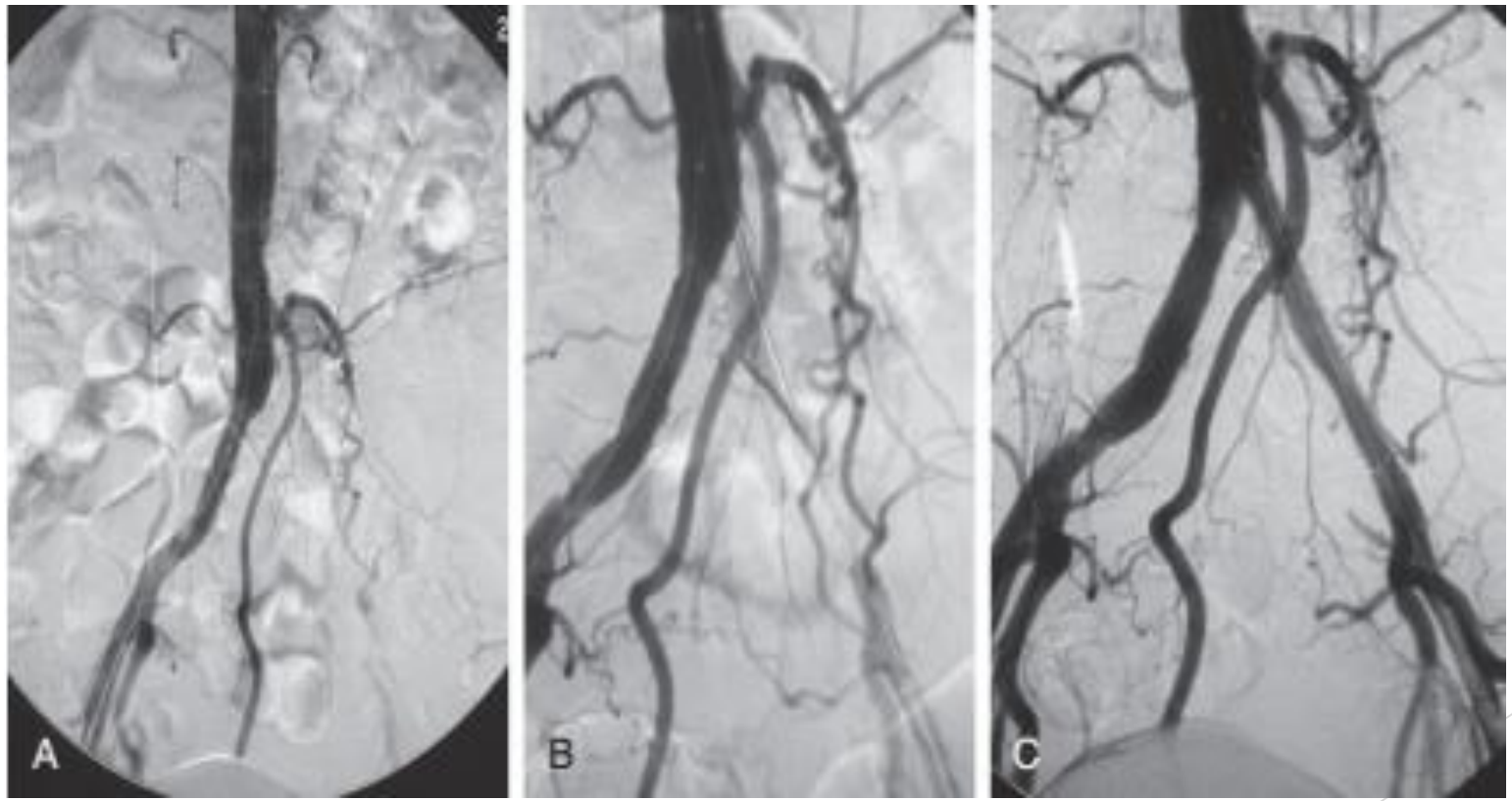
- Bilateral CIA occlusions
- Bilateral EIA stenoses 3-10 cm long not extending into the CFA
- Unilateral EIA stenosis extending into the CFA
- Unilateral EIA occlusion that involves the origins of internal iliac and/or CFA
- Heavily calcified unilateral EIA occlusion with or without involvement of origins of internal iliac and/or CFA

TYPE D LESIONS

- Infrarenal aortoiliac occlusion
- Diffuse disease involving the aorta and both iliac arteries requiring treatment
- Diffuse multiple stenoses involving the unilateral CIA, EIA, and CFA
- Unilateral occlusions of both CIA and EIA
- Bilateral occlusions of EIA
- Iliac stenoses in patients with AAA requiring treatment and not amenable to endograft placement or other lesions requiring open aortic or iliac surgery



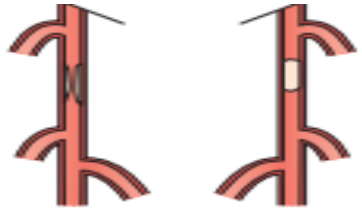
Balloon angioplasty and stenting of complete occlusion of the left common and external iliac arteries



Trans Atlantic Inter Society Consensus (TASC) of femoral-popliteal lesions

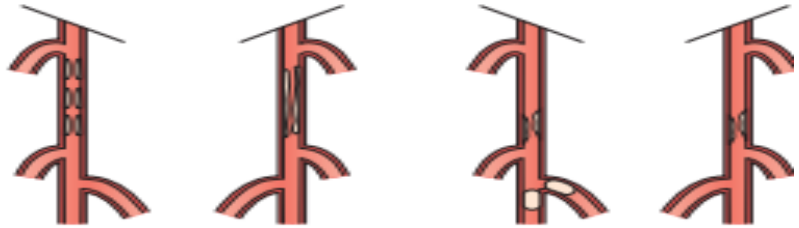
TYPE A LESIONS

- Single stenosis ≤10 cm in length
- Single occlusion ≤5 cm in length



TYPE B LESIONS

- Multiple lesions (stenoses or occlusions), each ≤5 cm
- Single stenosis or occlusion ≤15 cm not involving the infrageniculate popliteal artery
- Single or multiple lesions in the absence of continuous tibial vessels to improve inflow for a distal bypass
- Heavily calcified occlusion ≤5 cm in length
- Single popliteal stenosis

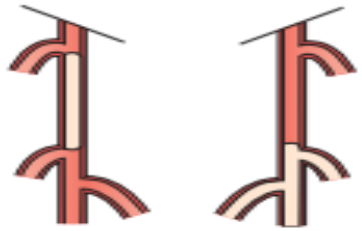


TYPE C LESIONS

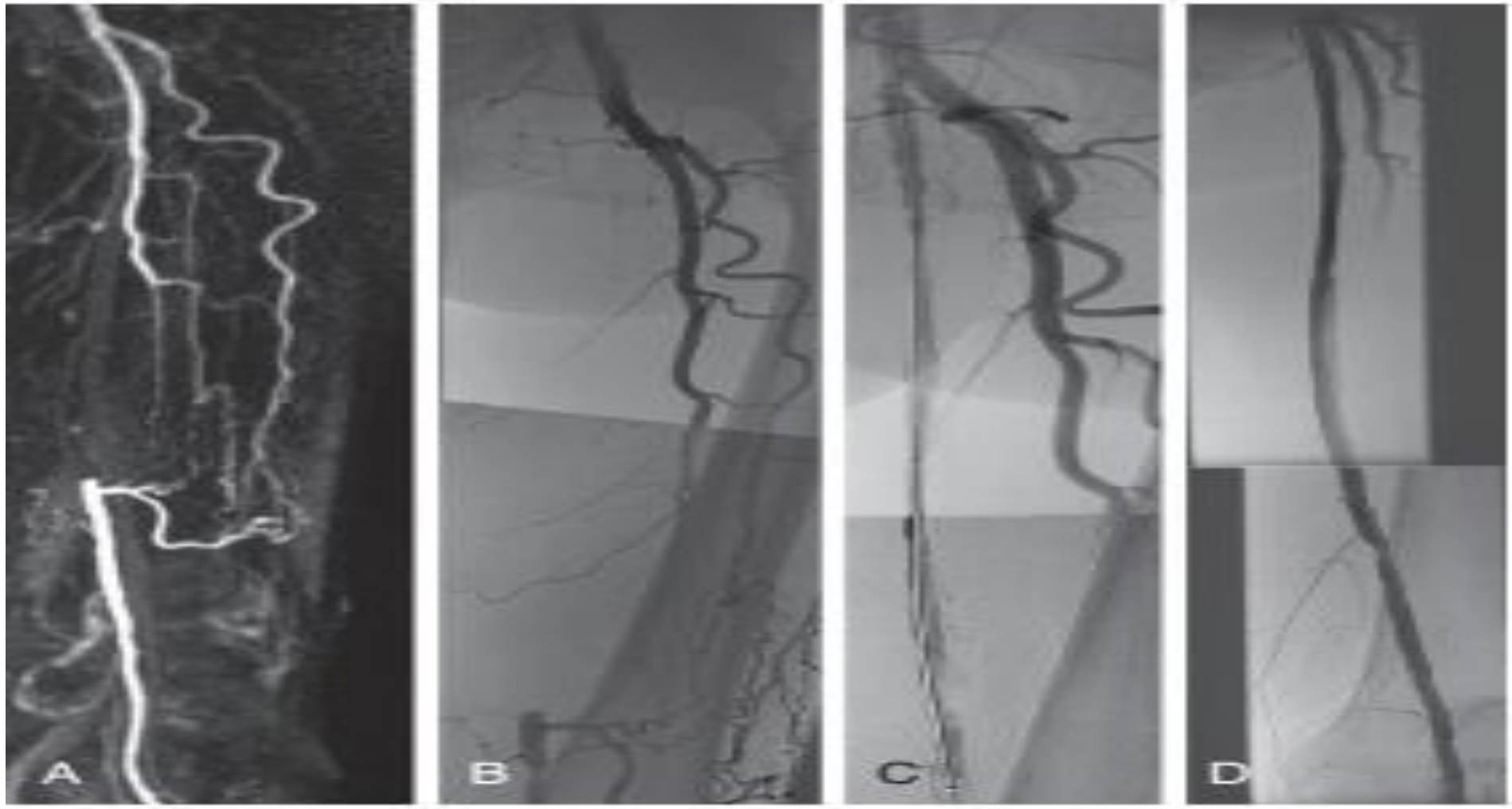
- Multiple stenoses or occlusions totaling >15 cm with or without heavy calcification
- Recurrent stenoses or occlusions that need treatment after two endovascular interventions

TYPE D LESIONS

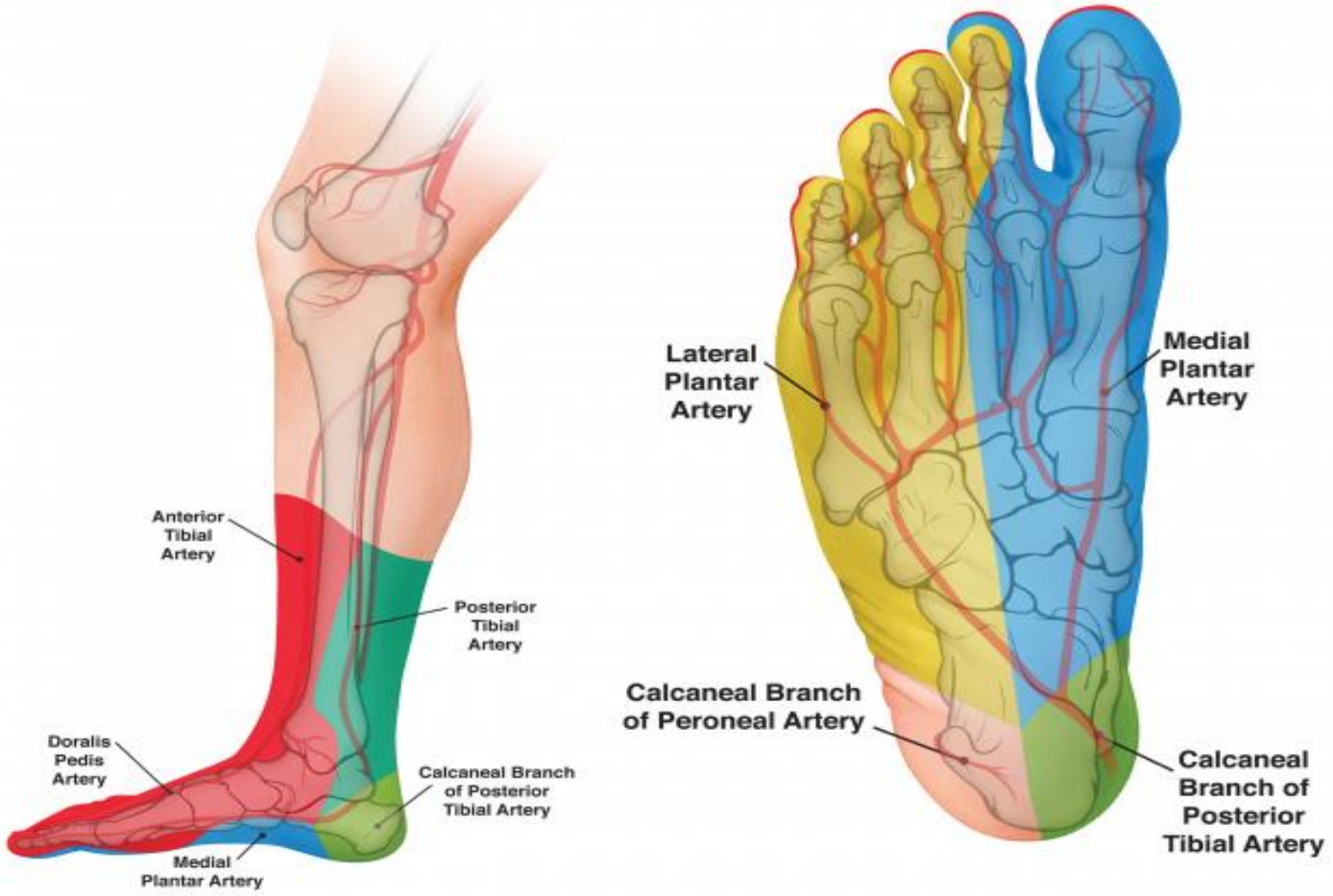
- Chronic total occlusions of CFA or SFA (>20 cm, involving the popliteal artery)
- Chronic total occlusion of popliteal artery and proximal trifurcation vessels



Balloon angioplasty and stenting of SFA



Angiozome concept



Determinants of Outcome of Endovascular Treatment

FAVORABLE STATUS FOR ENDOVASCULAR THERAPY	UNFAVORABLE STATUS FOR ENDOVASCULAR THERAPY
LESION CHARACTERISTICS	**LESION CHARACTERISTICS**
• Proximal lesion location	• Distal lesion location
• Stenosis	• Occlusion
• Short lesion length	• Long lesion length
• Focal stenoses	• Multiple same-segment stenoses
PATTERN OF VASCULAR DISEASE	**PATTERN OF VASCULAR DISEASE**
• Single-level disease	• Multilevel disease
• Normal, 3-vessel runoff	• Poor, <3-vessel runoff
PATIENT DEMOGRAPHICS	**PATIENT DEMOGRAPHICS**
• Male gender	• Female gender
• Low comorbid disease burden	• High comorbid disease burden (e.g., diabetes, ESRD)
CLINICAL INDICATION	**CLINICAL INDICATION**
• Claudication	• Critical limb ischemia
• Primary stenoses	• Recurrent stenoses
INTRAPROCEDURAL FACTORS	**INTRAPROCEDURAL FACTORS**
• No residual stenoses or dissection	• Presence of residual stenoses >30% or flow limiting dissection
• Robust hemodynamic response	• Minimal hemodynamic response

Balloon angioplasty of Tibialis artery

Arterial Aneurysm

The term *aneurysm* describes dilatation of any blood vessel. Arterial aneurysms occur throughout the body but are most prevalent in the infrarenal aorta.

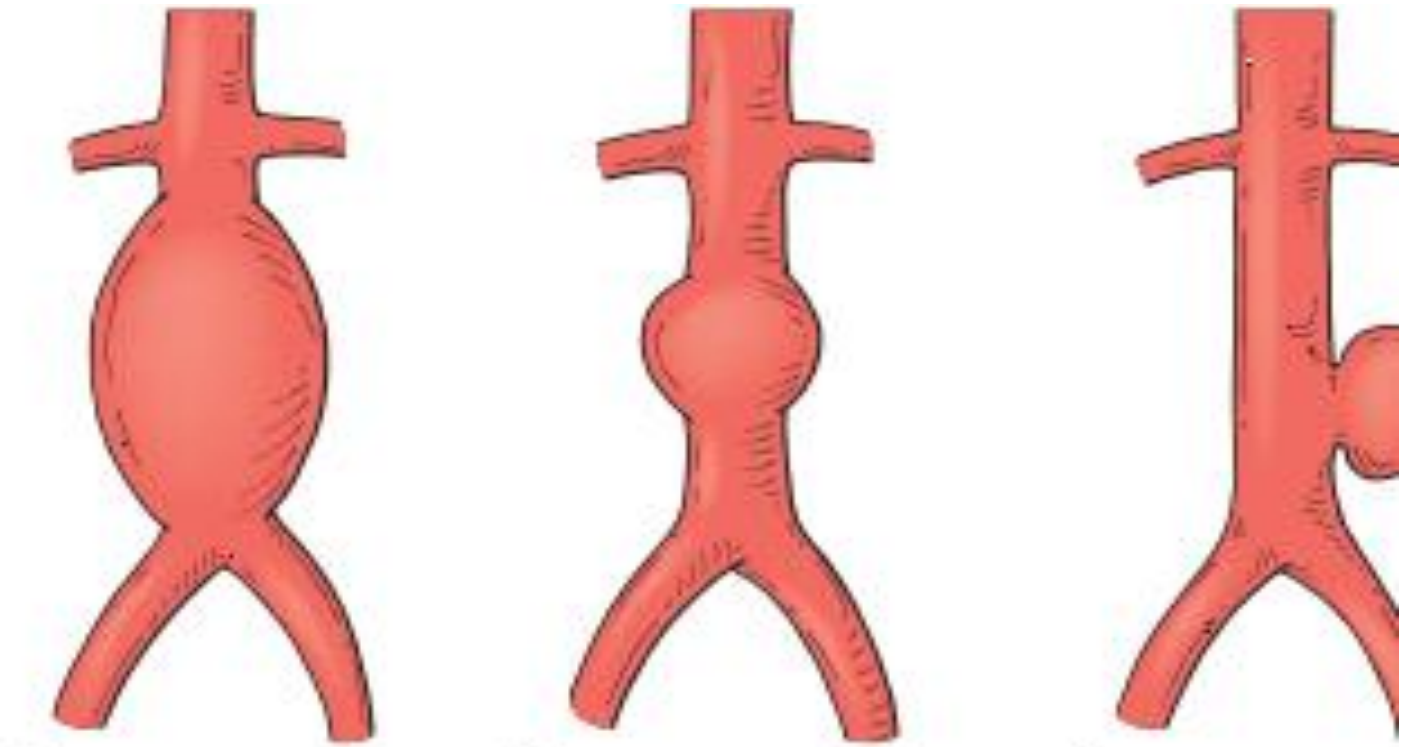
These aneurysms represent the primary cause of the signifcant death and disability
attributed to arterial aneurysm disease.

Aortic aneurysms can cause a variety of symptomatic clinical conditions (e.g., embolism,
obstruction of adjacent hollow viscera), the primary danger is from rupture and uncontrolled hemorrhage leading to death.

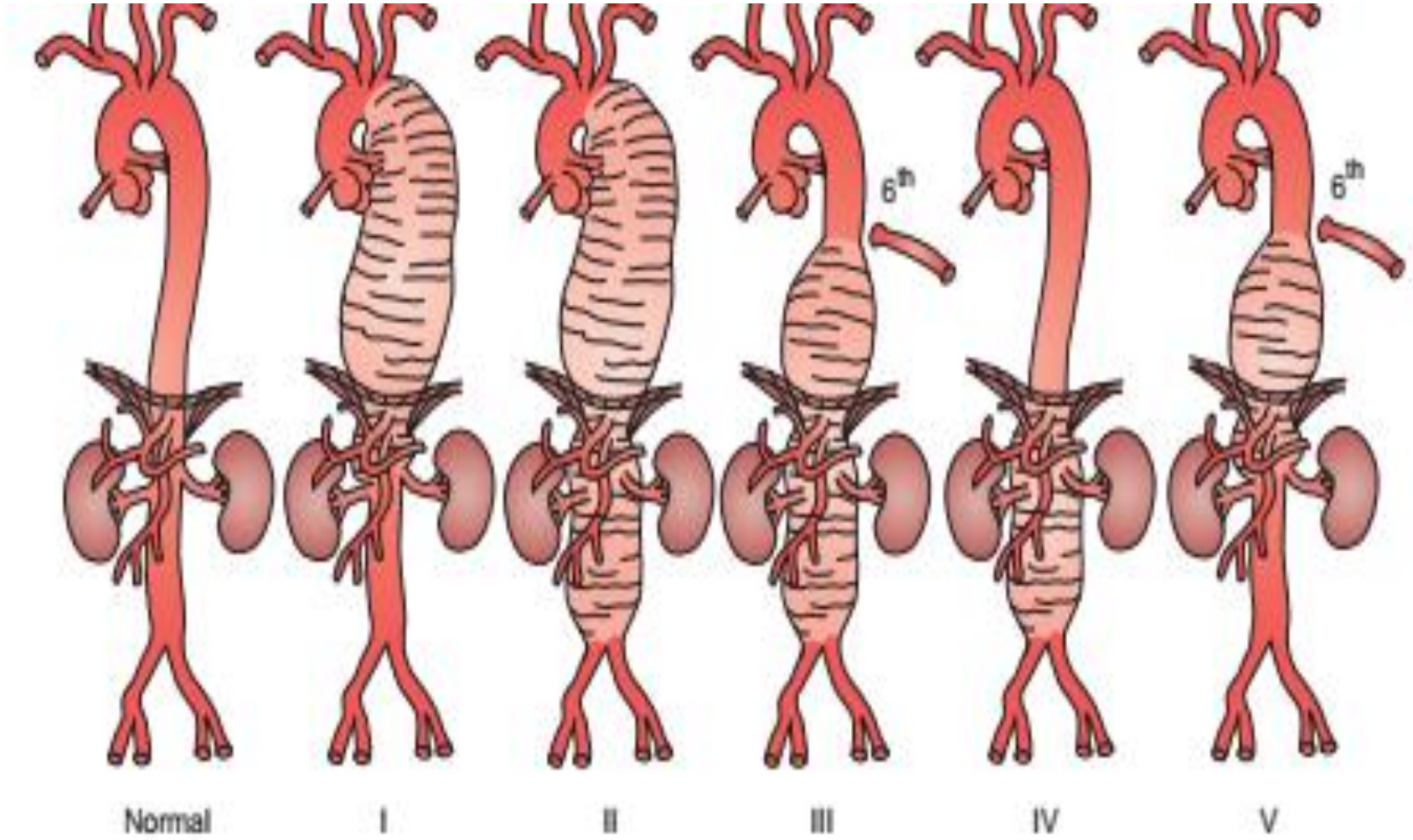
The risk of rupture is related to both the absolute size of the aneurysm and its size relative to normal diameters on the basis of location, body size, and gender.

A variety of other factors, including etiology, growth rate, and aneurysm morphology (i.e., fusiform vs saccular), can also be critical in assessing aneurysm risk in certain circumstances.

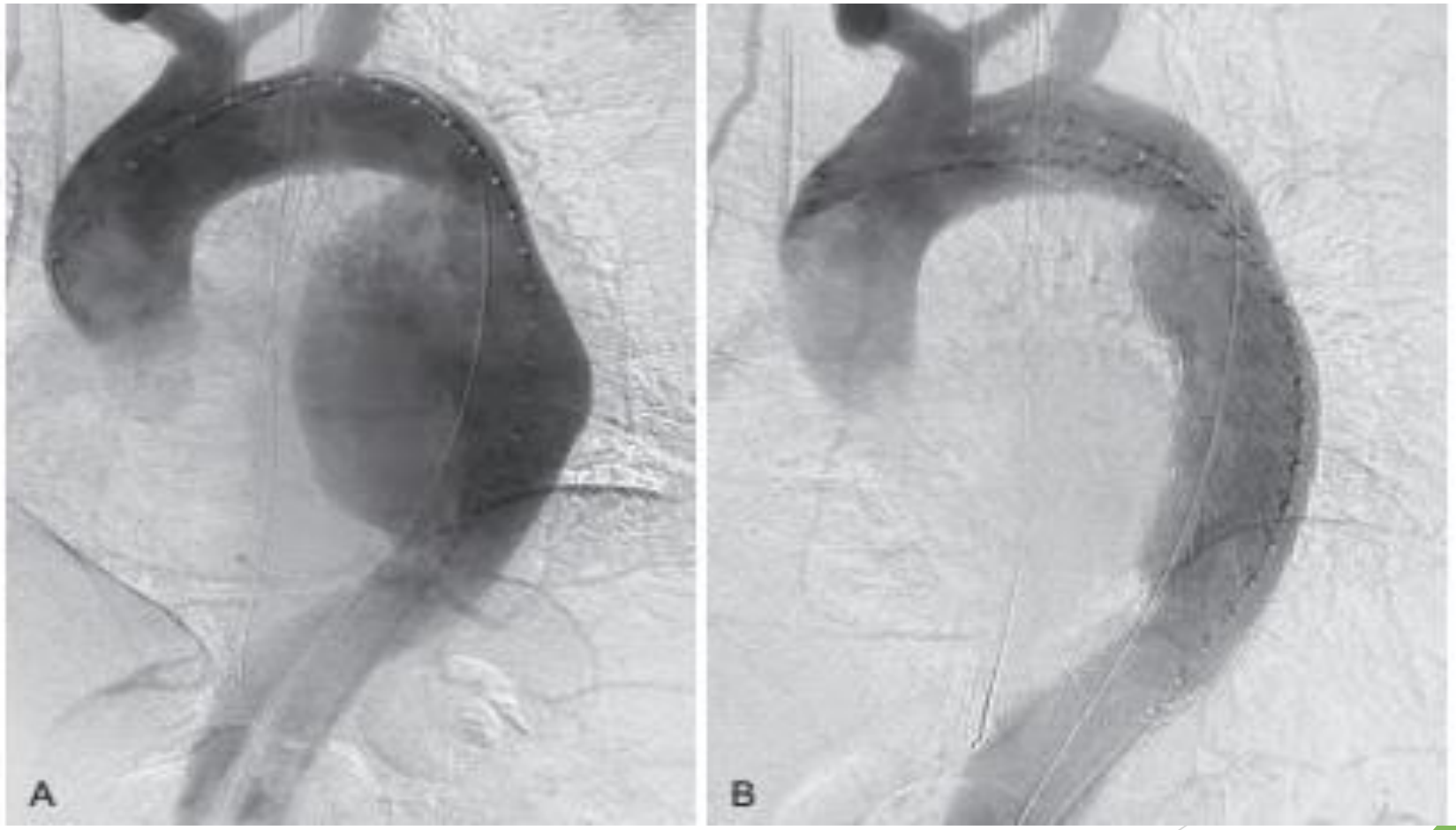
Morphology of Aneurysm



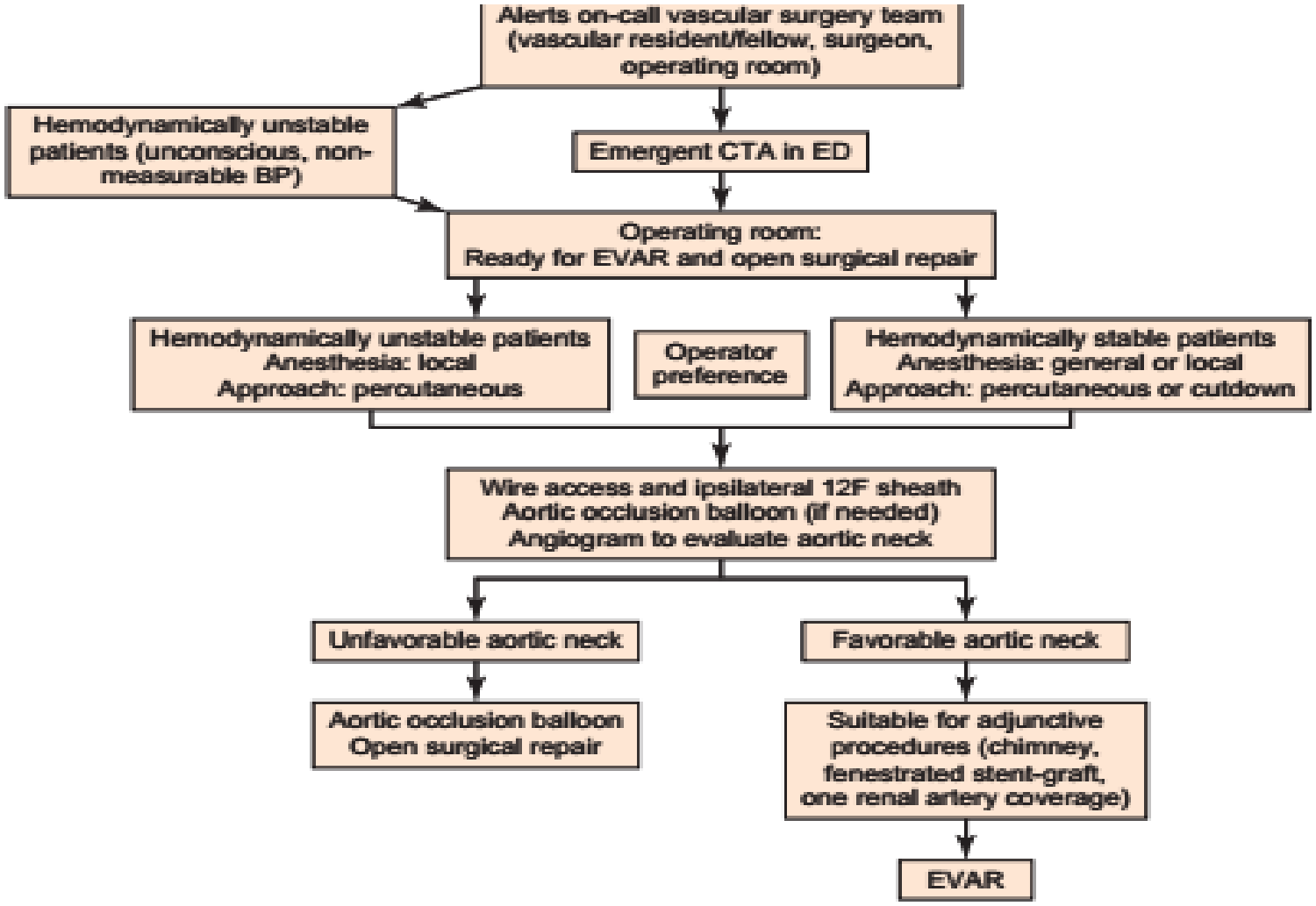
Crawford classification of thoracoabdominal aortic aneurysms



large fusiform descending thoracic arotic aneurysm. TAG stent grafts are deployed



Protocol for endovascular aneurysm repair of ruptured abdominal aortic aneurysm



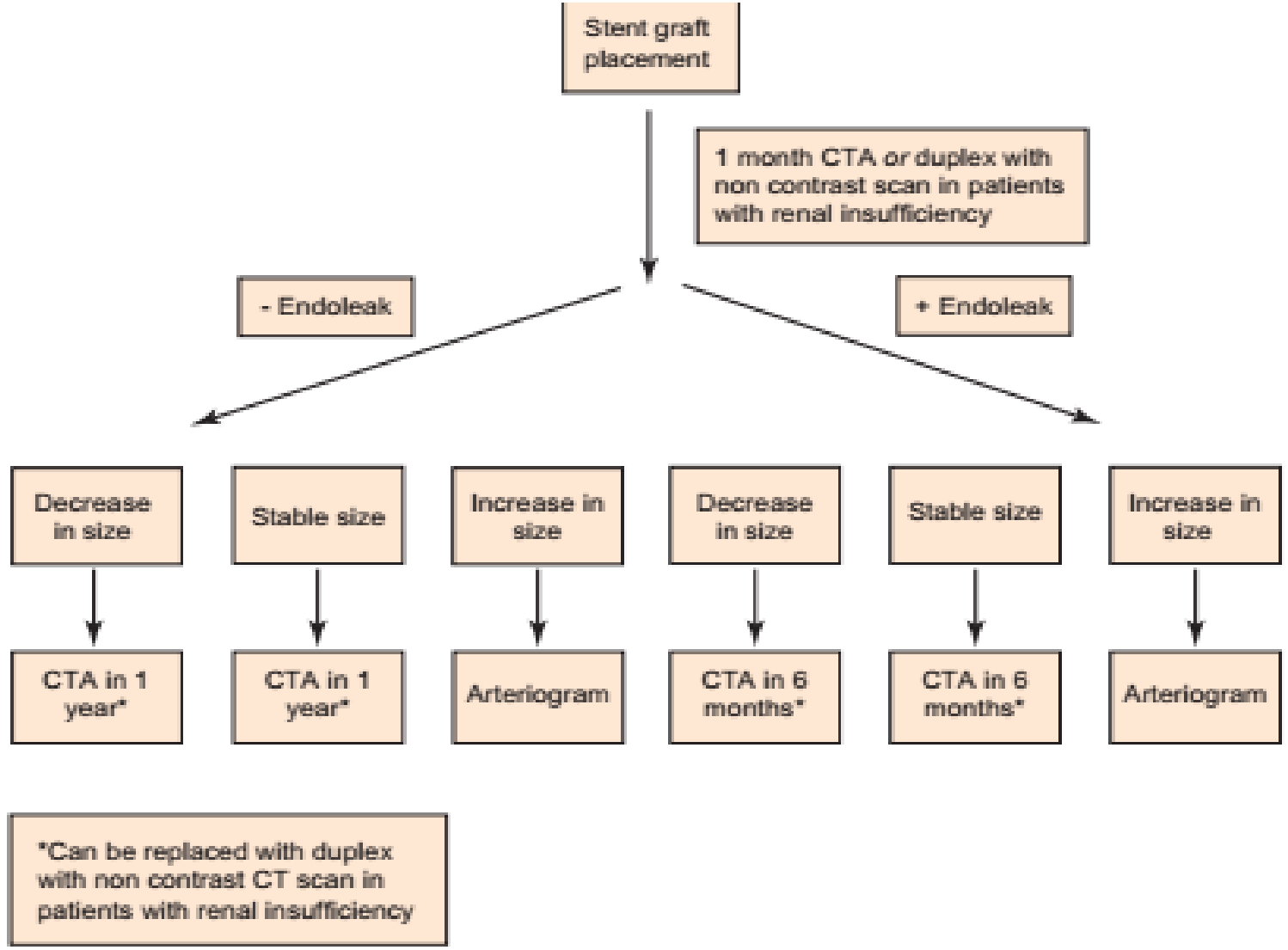
Relative contraindications to percutaneous Access for EVAR

- Severely scarred groin
- High femoral bifurcation
- Need for frequent introducer sheath changes
- Significant proximal iliac occlusive disease
- Small iliofemoral arteries
- Anterior calcific femoral disease

Relative indications for Alrto-uni-iliac Endograft configuration

- Very small (<15 mm) terminal aorta (which would not accommodate a bifurcated device)
- Severe unilateral iliac occlusive disease
- Secondary treatment of migration of a short-body endograft

Surveillance strategy following EVAR

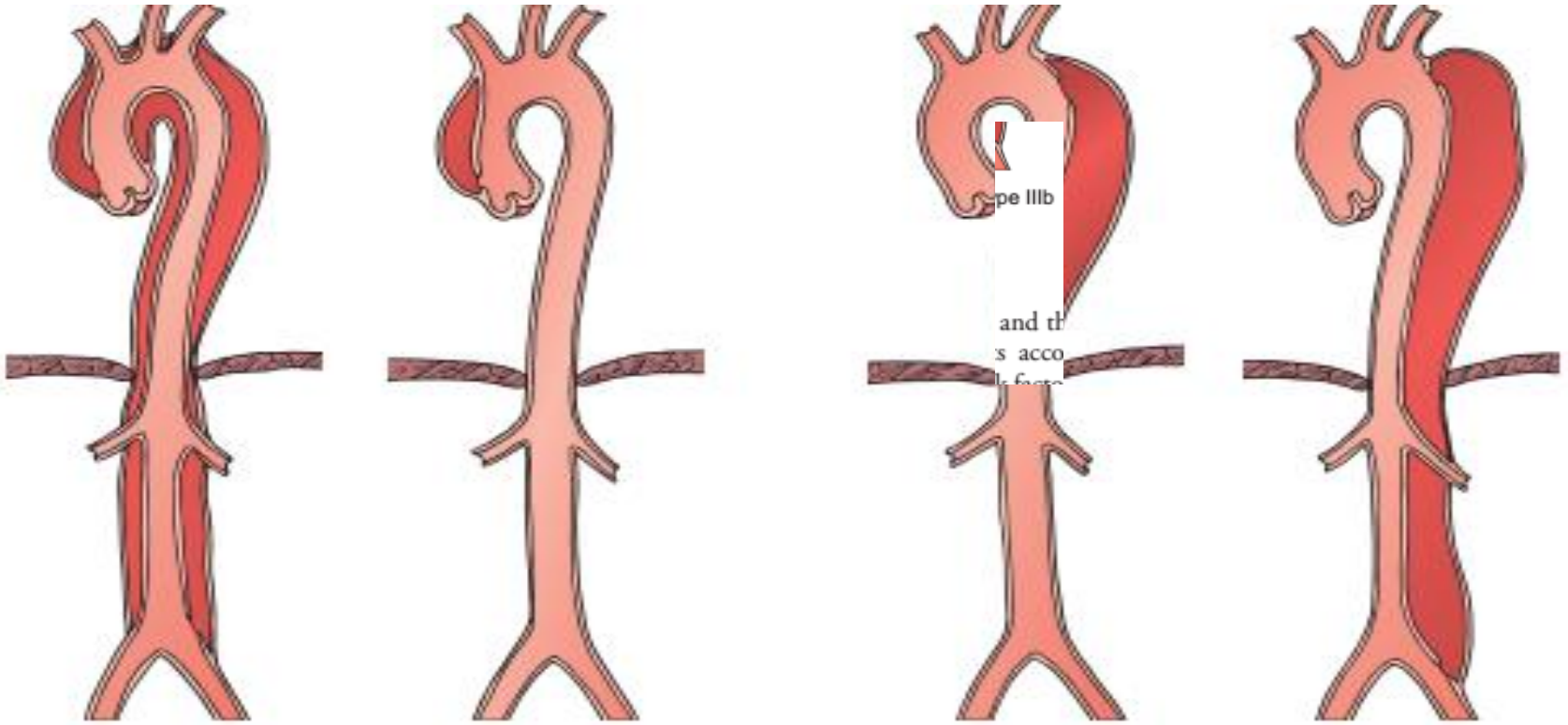


Aortic Dissection

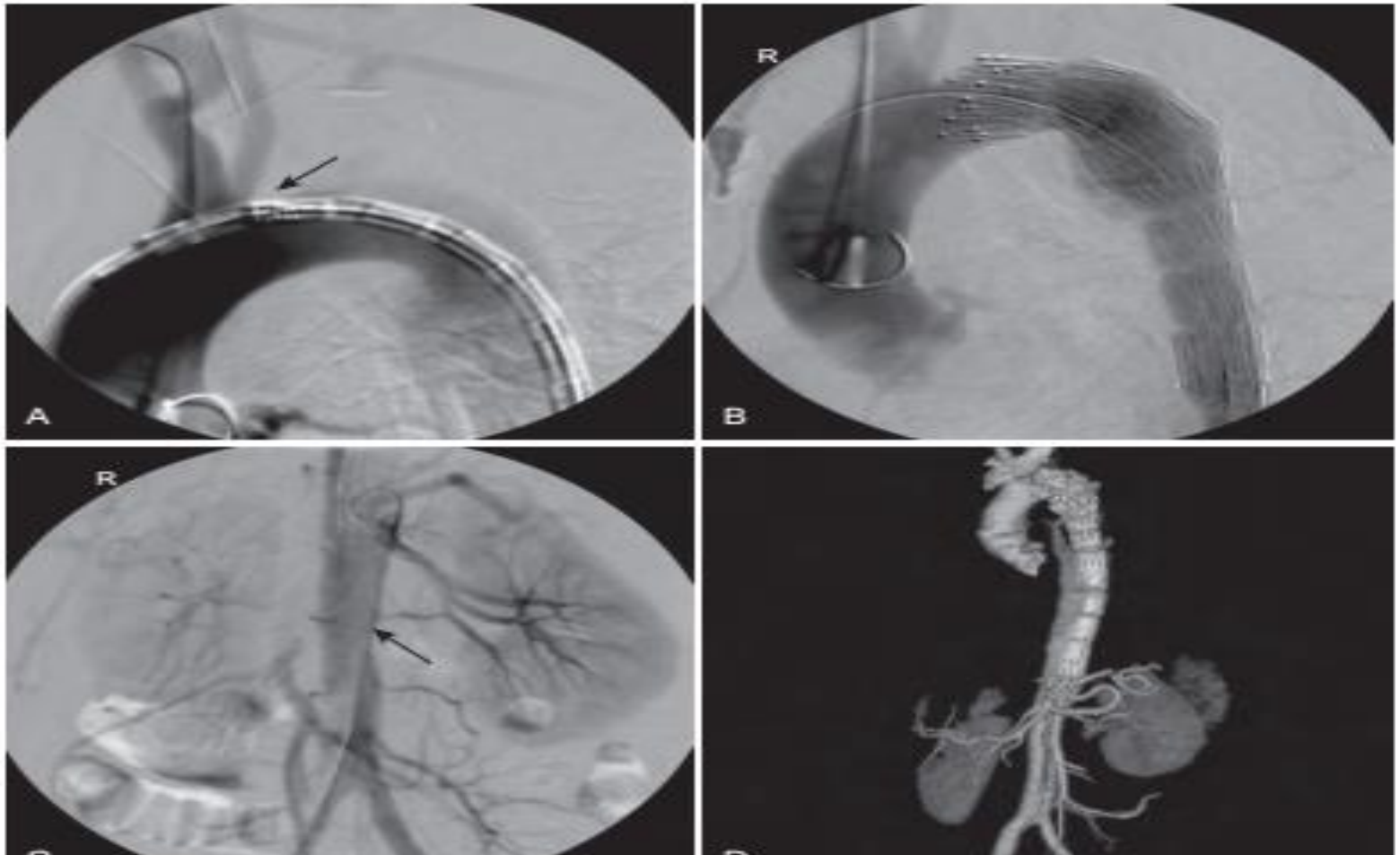
- Acute aortic dissection is the most common catastrophic event affecting the aorta, with an incidence exceeding that of ruptured abdominal aortic aneurysm.

Without treatment, the majority of atients with aortic dissections will die within 3 months of presentation, and few survive the chronic phase more than 5 years due to aneurysmal degeneration and rupture of the outer wall of the false lumen.

Debaky & Stanford systems for Acute aortic dissection



TEVAR for type B Dissection



Isolated Iliac artery aneurysm

- Iliac artery aneurysms (IAAs) commonly occur concurrently with other more proximal arterial aneurysms.
- IAAs were initially described by Valentine Mott in 1827, and later by Halstead in 1912.
- In contrast to generalized aortoiliac aneurysms, isolated iliac aneurysms are much less freqruently recognized, and are prevalent in ≤ 2% of the general population.
- Aneurysmal degeneration typically involves the common iliac (70%-90%) and internal iliac (10%-30%) arteries, or both of these segments contiguously; external iliac artery (EIA) involvement is extremely rare.
- Bilateral common IAAs are present in at least 50% of affected patients.7

Management

- Iliac aneurysms are targeted for repair as they enlarge or become symptomatic.

current recommendations favor elective repair for asymptomatic IAAs ≥ 3.5 cm in diameter in healthy patient.

Asymptomatic IAAs less than 3 cm in diameter are best managed with serial surveillance imaging studies.

In fact, general abdominal ultrasound screening for aortoiliac aneurysms is recommended by the US Preventive Services Task Force (USPSTF) for men aged 65 to 74 years who have ever smoked.

Classification Scheme for Endovascular Treatment of Isolated Iliac artery aneurysm

Type	Proximal	Distal	Configuration	Endovascular Techniques
A	CIA	CIA		Direct aneurysm exclusion with covered stents within proximal and distal fixation zones (balloon-expandable or self-expandable)
B1	CIA	IIA	IIA diameter < 15 mm	**Anatomical with IIA preservation** Iliac side-branch device **Extraanatomical with IIA preservation** 1. Placement of AUI in contralateral nonaneurysmal CIA with a femorofemoral bypass and endograft deployment from ipsilateral EIA to ipsilateral IIA 2. Placement of bifurcated endograft with ipsilateral extension to EIA, ipsilateral retroperitoneal open surgical ligation of IIA, bypass grafting from ipsilateral distal EIA to the ipsilateral IIA 3. Placement of bifurcated endograft with an ipsilateral iliac limb extension toward the ipsilateral IIA and adjunctive femorofemoral bypass and distal EIA ligation **Sacrificing IIA** Exclusion with coverage or embolization of IIA and placement of covered stent across CIA aneurysm to EIA
	CIA	IIA	At least 20 mm of nonaneurysmal tissue within IIA	
B2			Aneurysm extends into IIA IIA diameter < 15 mm Insufficient length of IIA (< 20 mm)	
C	Aorta	CIA		**Anatomic with IIA preservation** Placement of bifurcated endograft with the ipsilateral iliac limb extending to the distal CIA landing zone **Extraanatomic with IIA preservation** 1. Placement of AUI into contralateral nonaneurysmal CIA with femorofemoral bypass and endograft extending from the ipsilateral EIA to the ipsilateral IIA 2. Placement of AUI into contralateral nonaneurysmal CIA with femorofemoral bypass and occlusion of distal CIA before bifurcation
D1	Aorta	IIA	IIA diameter < 15 mm	**Anatomic with IIA preservation** 1. Iliac side-branch device 2. Double barrel graft technique using a bifurcated device **Extraanatomical with IIA preservation** 1. Placement of AUI into contralateral nonaneurysmal CIA with femorofemoral bypass and endograft extending from ipsilateral EIA to the ipsilateral IIA 2. Placement of bifurcated endograft with an ipsilateral iliac limb extension toward the ipsilateral IIA and adjunctive femorofemoral bypass and distal EIA ligation **Sacrificing IIA** 1. Exclusion of aneurysm with placement of bifurcated endograft with ipsilateral extension to the ipsilateral EIA and ipsilateral IIA embolization
D2	Aorta	IIA	At least 20 mm of nonaneurysmal tissue within IIA Aneurysm extends into IIA IIA diameter < 15 mm Insufficient length of IIA (< 20 mm)	

Renovascular Disease

- Renovascular disease (RVD) encompasses a range of disorders that affect renal artery structure and kidney blood flow.

The primary importance of RVD relates to associated hypertension (HTN) and loss of renal excretory function, which in turn contribute to cardiovascular morbidity, dialysis dependence, and death.

.

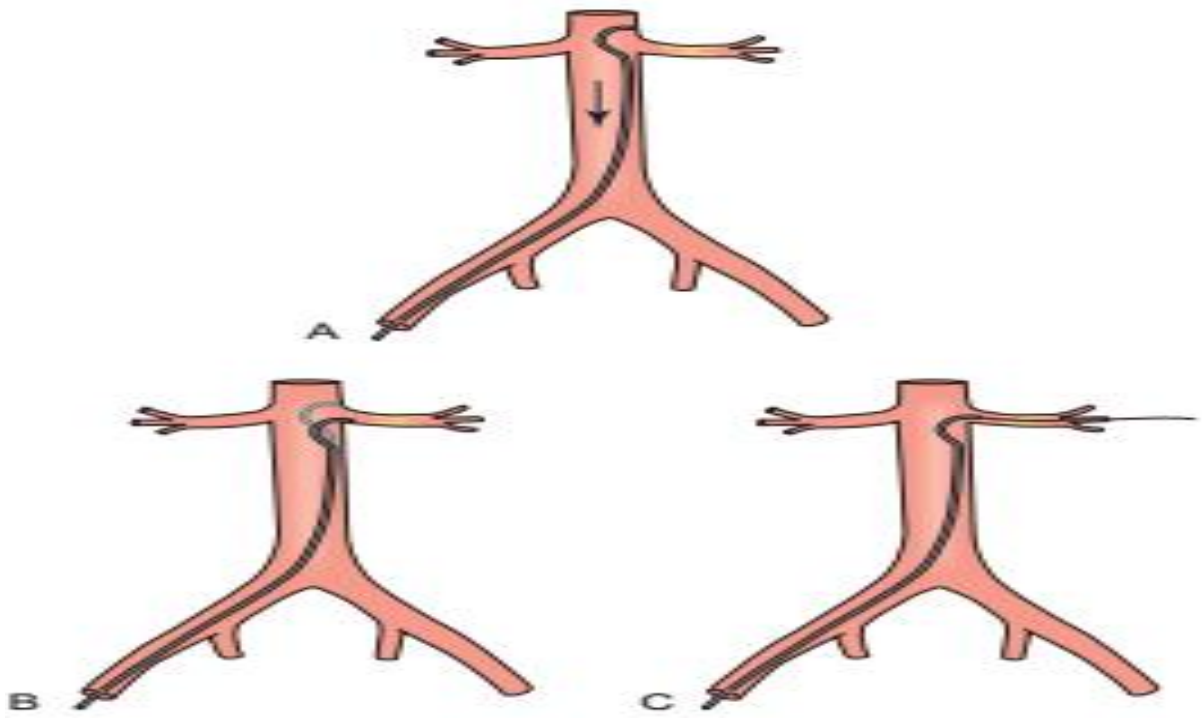
Te most common pathology is atherosclerotic renal artery stenosis (RAS), which can be found in up to 7% of elderly individuals in the Uenited States.

Less common etiologies includerenal artery fbromuscular dysplasia (FMD) , Dissection, aneurysms, and trauma , arteritis , and developmental abnormalities of the middle aorta and its branches.

Accuracy of DUS , CTA , MRA for evaluation of RA

Series	Year	Modality	Number of Kidneys	Sensitivity (%)	Specificity (%)
Hansen et al.[116]	1990	Duplex	122	93	98
Hua et al.[101]	2000	Duplex	58	91	75
Fraioli et al.[186]	2006	CTA	50	100	98.6
Rountas et al.[189]	2007	Duplex, CTA, MRA, DSA	129	75 (duplex) 94 (CTA) 90 (MRA)	89.6 (duplex) 93 (CTA) 94.1 (MRA)

Contralateral femoral access often facilitates selective renal catheterization



Mesenteric arterial Disease

- . Te most common cause is progressive occlusive disease of the visceral arteries, usually related to atherosclerosis.
- Because CMI is relatively uncommon, it is frequently misdiagnosed as a gastrointestinal disorder. Patients tpically have undergone an extensive workup for other potential etiologies before being diagnosed with CMI.
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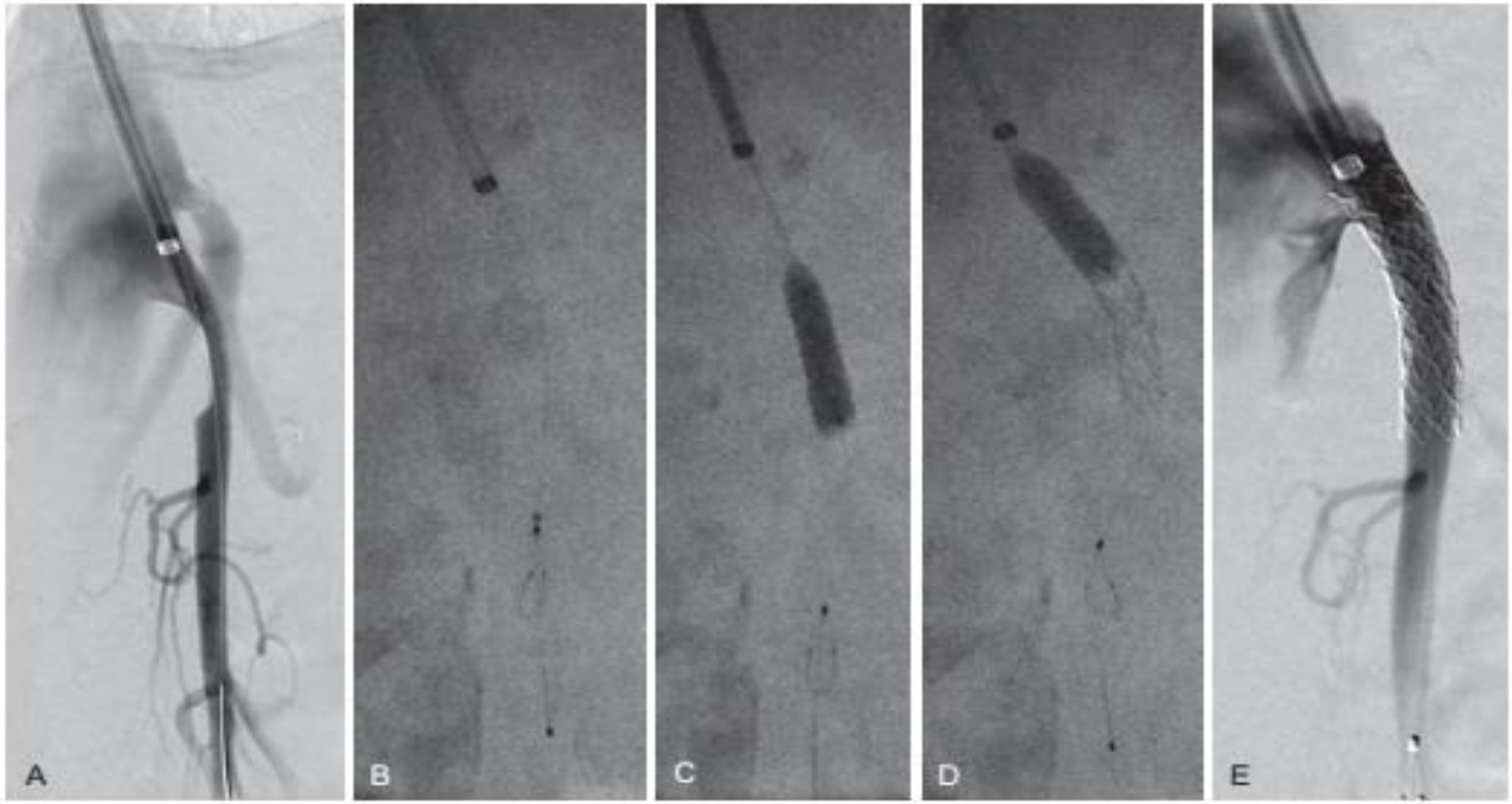
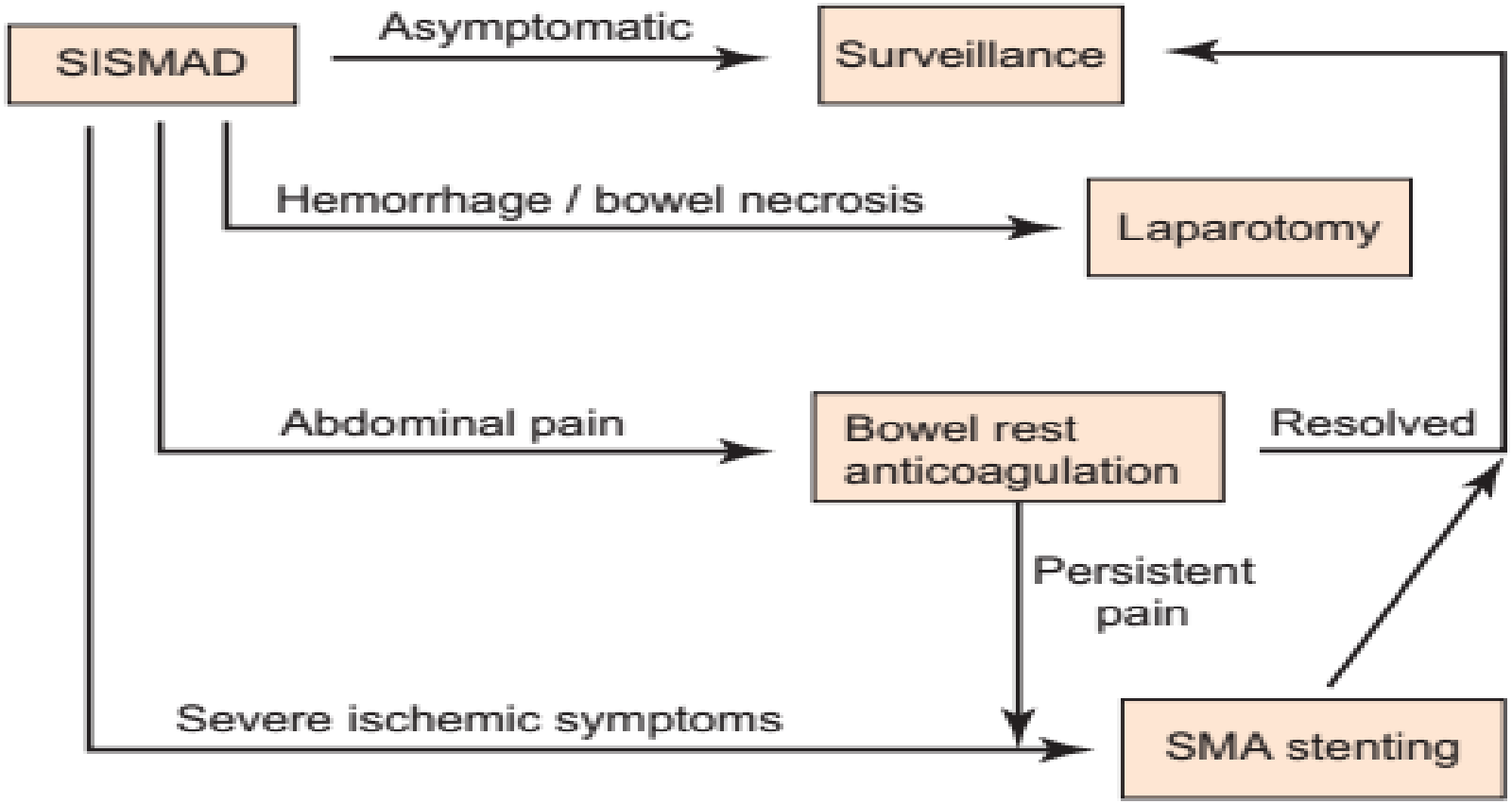
Angioplasty and stenting of focal stenosis of SMA



Treatment for spontaneous isolated superior mesenteric artery dissection

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