MANAGEMENT OF COMMON CARDIOVASCULAR DISEASES

Prof. Dr Khin Mg Lwin

Prof. Dr Aung Zaw Myo

Dr Win Min Paing

Dr Linn Thaung Zin

Dr Shwe Pyi Kyaw

DEPARTMENT OF CARDIOVASCULAR SURGERY

YANGON GENERAL HOSPITAL

CONTENT

1. MANAGEMENT GUIDELINES OF CARDIAC TAMPONADE

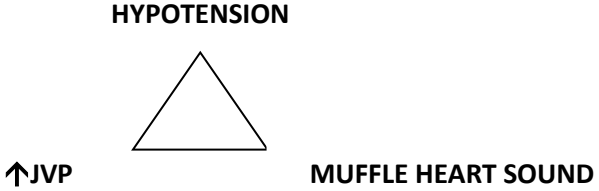
1.1. Clinical Evaluation

(a) History

- Symptoms – vary with cause and acuteness of tamponade
- Acute tamponade usually presents with dyspnoea, tachycardia, tachypnoea and cold and clammy extremities due to hypotension
- Comprehensive review of patient's history helps identification of possible cause of the probable aetiology of pericardial effusion.
- Symptoms associated with underlying causes
  - Systemic or malignant diseases → loss of weight, fatigue, or anorexia
  - Pericarditis or myocardial infarction → chest pain
  - Connective tissue disorders → musculoskeletal pain or fever
  - Uraemia → history of renal failure
  - Medications or drugs history → drug-related lupus leading to pericardial effusion
  - Recent cardiovascular surgery, coronary intervention or trauma → rapid accumulation of pericardial fluid and tamponade
  - Recent pacemaker implantation or CVP line insertion
  - HIV related pericardial effusion → IV drug abuser or opportunistic infections
  - Chest wall radiation → for CA lungs, mediastinal or oesophageal cancer

(b) Physical Examination

- Patient → dyspnoea, tachycardia, increased JVP
- Evidence of chest wall injury → blunt trauma or penetrating wound, esply around pericardial region
- >50% present with tachycardia, tachypnoea or hepatomegaly
- Approximately 1/3 present with diminished heart sound or pericardial friction rub
- May also present with dizziness, drowsiness or palpitation and cold and clammy skin and weak pulsation
- **Beck's Triad or Acute compression Triad**



- **Pulsus paradoxus**
- **Kussmaul sign** usually seen in constrictive effusive pericarditis
- **Ewart's sign** (Pins sign) – seen in large pericardial effusion → an area of dullness with bronchial breath sound and brochopony below the angle of left scapula
- ***'Y' descent of JVP wave*** – abolished
- Dysphoria → behavioural traits such as restless body movement, unusual facial expression, restlessness, sense of impending death → seen in 26% of cases
- Low pressure tamponade in severely hypovolaemic patients → classical signs (tachycardia, pulsus paradoxus or ↑JVP → infrequent in 10-20%

(c) Differential Diagnosis

- Cardiogenic shock of other causes
- Constrictive pericarditis or constrictive effusive pericarditis
- Pulmonary embolism
- Tension pneumothorax

1.2. Work-up / Investigations

(1) CXR

- Cardiomegaly, water-bottle shaped heart, pericardial calcification or evidence of chest wall trauma
- In children after CVP catherization → Bowed catheter sign

(2) Echocardiography → provides usually information

- Echo free space posterior and anterior to LV wall or behind LA
- After cardiac surgery, even localized collection of fluid without significant amount may compromise cardiac output
- Early diastolic collapse of right ventricular free wall

(3) Investigations according to possible underlying causes

1.3. Medical care

Cardiac tamponade is a ***MEDICAL EMERGENCY.***

Preferably, should be monitored in ICU

(a) Treatment

- O2
- Volume expansion with blood , plasma, dextran, or isotonic N/S solution as necessary to maintain adequate intravascular volume (if necessary)
- Bed rest with leg elevation => to improve venous return

- Inotrophic drugs eg. Dobutamine (increase CO but not systemic vascular resistance)

(b) Definitive treatment

- Removal of pericardial effusion by pericardiocentesis

- Subxiphoic approach, emergency lifesaving bedside procedure(16 or 18 G needle, 30 to 45 degree to sternum, left xiphocostal angle toward left shoulder), mortality rate ~ 4 % , complication approximately 17 %
- Echo-guided pericardiocentesis(usually through left intercostal space)
- Percutaneous balloon pericardiotomy

1.4. Surgical care

(a) Indications

- For haemodynamically unstable patients or one with recurrent tamponade

(b) Options

1) Pericardiocentesis

Indications

- *Emergency pericardiocentesis → in the presence of life threatening haemodynamic changes in a patient with suspected cardiac tamponade, Even aspiration of small amount of PE fluid may dramatically improve the patient haemodynamic status*
- *Non-emergent pericardiocentesis → aspiration of pericardial fluid in haemodymically stable patient – for diagnostic, palliative or prophylactic reason (performed under USG, CT, fluoroscopic visualization)*

Absolute contraindication

- *Uncorrected bleeding disorder*
- *Traumatic cardiac tamponade because arguments – present in treatment by emergency thorocotomy or pericardiocentesis*

Complications

Rate varies 4-40%

- Dysrhythmias
- Coronary artery puncture or aneurysm
- LIMA puncture or aneurysm
- Heamothorax
- Pneumothorax
- Pneumopericardium
- Hepatic injury
- False negative aspiration (clotted blood)
- False positive (intracardiac)
- Reaccumulation of pericardial fluid

2) Surgical creation of pericardial window (between pericardial space and intrapleural space)

Approach – subxiphoidian

-paraxiphoidian

-open thorocotomy and/or pericardiotomy

3) Sclerosing the pericardium (recurrent case) using corticosteroid, tetracycline or antineoplastic drugs (eg, anthracycline, bleomycin)

4) Pericardioperitoneal shunt

5) Pericardiectomy-resection of pericardium (through median sternotomy or left thoracotomy)

(c) Consultation

- Haemodymically stable => cardiologist
- Unstable => cardiologist , cardiothoracic surgeon

2. MANAGEMENT OF CARDIOVASCULAR INJURIES

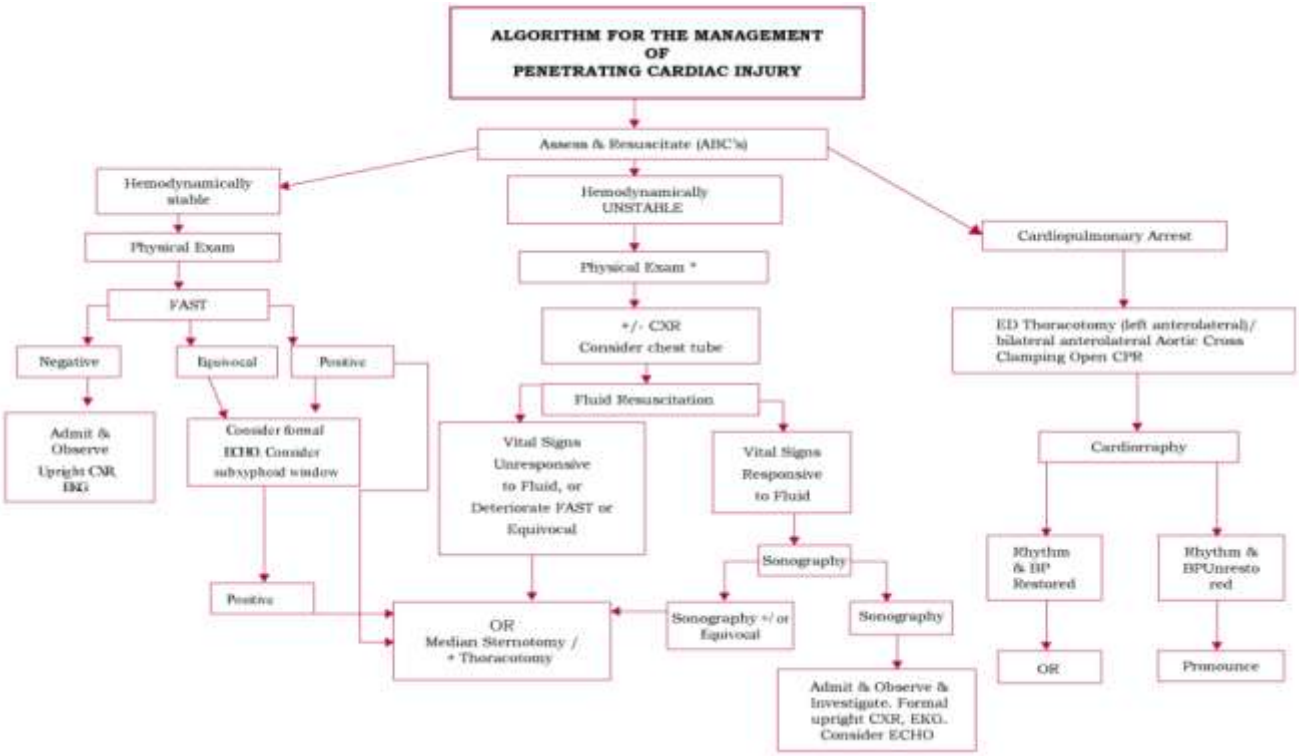
2.1. Management of cardiac injuries

(a) Initial assessment and general assessment

- The initial care of the trauma patient with cardiac injuries does not vary from standard Advanced Trauma Life Support (ATLS) protocols.
- The primary priority is ensuring the patency of the airway and establishing adequate oxygenation and ventilation.
- This may include tube thoracostomy for drainage of hemothorax from the pleural space to allow re-expansion of the lung.
- Subsequently, the circulatory system is assessed. Priority is given to establishing intravenous access for the administration of crystalloid and/or blood products.
- If cardiac tamponade is suspected, this should be confirmed with sonographic confirmation of hemopericardium and/or right ventricular collapse during diastole.
- If tamponade physiology is present, treatment for immediate drainage of the pericardial space should be initiated.
- This can be accomplished percutaneously by pericardiocentesis or via open pericardial window.
- The treatment algorithm for cardiac injured patients branches at this point depending on the mechanism of injury and hemodynamic status.
- As is the standard in all trauma care, cardiac injuries are categorized as either blunt or penetrating and we will explore their assessment and treatment separately.

(b) Penetrating cardiac trauma

- Penetrating trauma to the heart most frequently occur with trauma to the anterior chest, but
- Should also be suspected with wounds to the upper abdomen, chest, back, and neck.
- When the pericardial wound is open and blood is able to flow freely into the pleural space, the patient can often be supported with fluid resuscitation and chest tube thoracostomy.
- Persistent drainage from the thoracostomy tube should warn of possible cardiac injury and surgical exploration is indicated.
- Conversely, if the blood is retained in the pericardial space, cardiac tamponade and physiology will ensue if not drained immediately.
- Management of the *stable patient* (systolic blood pressure greater than 90 mm Hg) allows for a more complete evaluation including chest x-ray and echocardiography.
- *Unstable patients* (systolic blood pressure less than 90 mm Hg) are taken directly to the operating room for exploration while patients with loss of vitals during transport or upon presentation to the hospital are treated with Emergency Department thoracotomy.



- If the diagnosis of penetrating cardiac injury is suspected but not confirmed, a subxiphoid pericardial window should be performed.
- Surgeons should be prepared to do a median sternotomy if an injury is identified in order to definitely address the wound.
- Upon opening the pericardial sac, any blood or fluid should be evacuated to allow the heart to properly fill and contract.
- Whatever injury is encountered and method of repair utilized, the operative principles are universal: relieve tamponade, stop the bleeding, and restore circulating volume.

(c) Blunt cardiac injury (BCI)

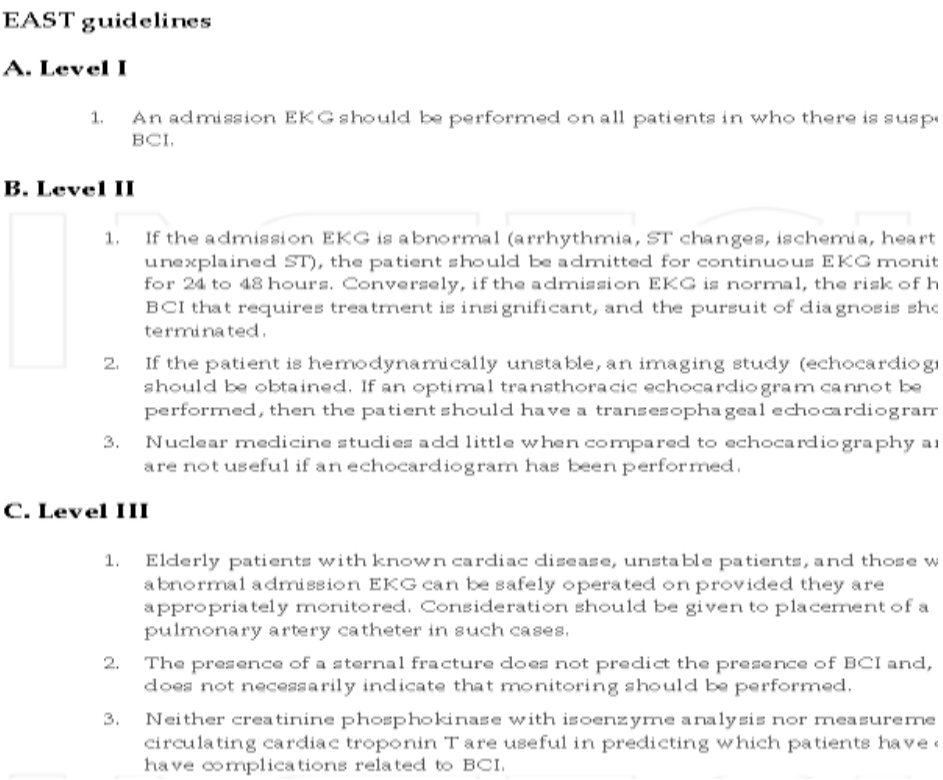
Background (mechanism, incidence, and pathophysiology)

- Blunt cardiac injury (BCI) is a spectrum of traumatic heart diseases with severity that can range from myocardial contusion and EKG changes to septal rupture and death.
- BCI is estimated to occur in 20% of motor vehicle collisions and in greater than 75% of thoracic blunt injuries independent of the mechanism.
- The primary mechanism of injury to the heart is from high-speed motor vehicle collision, but any injury that applies force in the form of kinetic energy to the chest wall and heart can result in a form of BCI.
- The following mechanisms of injury may result in BCI:
  - direct precordial impact,
  - a crush injury between the sternum and spine,
  - a deceleration injury causing injury from the fixation points of the aorta and vena cava,

  - a hydraulic effect from an intraabdominal injury that sends force to the great vessels and heart, or a crush injury.
- Injuries sustained with blunt cardiac injury (BCI) include contusion, ruptures, septal defects, valvular injuries, and coronary artery injuries.
- Contusion is the most common type of injury with left atrial chamber rupture being least common. Injuries can often occur concomitantly; approximately 20% of injuries with chamber rupture will have another chamber involved.
- The right heart is the most commonly injured as it is closest to the sternum which is impacted anteriorly by the steering wheel in motor vehicle collisions.
- Besides having concomitant cardiac injuries, the force needed to cause a BCI will often cause associated injuries such as chest pain, rib fractures, pulmonary contusions, and solid organ injuries.

Diagnosis

- The best test for diagnosing blunt cardiac injury has been debated for many years.
- Cardiac enzymes, radionuclide scans, EKG, cardiac ultrasound and continuous monitoring are some of the major methods that have been investigated.
- Eastern Association for the Surgery of Trauma (EAST) guidelines.



EAST guidelines

A. Level I

1. An admission EKG should be performed on all patients in who there is susp BCI.

B. Level II

1. If the admission EKG is abnormal (arrhythmia, ST changes, ischemia, heart unexplained ST), the patient should be admitted for continuous EKG monit for 24 to 48 hours. Conversely, if the admission EKG is normal, the risk of h BCI that requires treatment is insignificant, and the pursuit of diagnosis sh terminated.
2. If the patient is hemodynamically unstable, an imaging study (echocardiog should be obtained. If an optimal transthoracic echocardiogram cannot be performed, then the patient should have a transesophageal echocardiogram
3. Nuclear medicine studies add little when compared to echocardiography a are not useful if an echocardiogram has been performed.

C. Level III

1. Elderly patients with known cardiac disease, unstable patients, and those w abnormal admission EKG can be safely operated on provided they are appropriately monitored. Consideration should be given to placement of a pulmonary artery catheter in such cases.
2. The presence of a sternal fracture does not predict the presence of BCI and, does not necessarily indicate that monitoring should be performed.
3. Neither creatinine phosphokinase with isoenzyme analysis nor measureme circulating cardiac troponin T are useful in predicting which patients have have complications related to BCI.

Management

- Since blunt cardiac injury describes a spectrum of disease states, the treatment depends on the actual problem.
- Arrhythmia can be managed medically with the caveat that anticoagulation needs to be used cautiously in trauma patients.
- Hemopericardium can be seen with or without hypotension or tamponade.

    - If hemopericardium is suspected and the patient is stable a subxiphoid pericardial window can be used to verify the hemopericardium.
    - Once a pericardial window is performed, the surgeon must be prepared to proceed with a median sternotomy.
- If the patient is hypotensive and tamponade is expected then either a subxiphoid pericardial window or a thoracotomy can be performed.
- As a rule free wall rupture is more common in the atria than the ventricles and more common on the right than the left.
- This is thought to be due in part to the position of the heart in the chest.
- Septal rupture requires the patient to be placed on bypass.
- Coronary artery injury, valve injury and papillary muscle rupture are all very rare.
- These entities generally present with clinically significant acute congestive heart failure.
- Another rare entity is pericardial rupture with cardiac herniation.
- Whether you utilize a thoracotomy or sternotomy will depend on the details of the cardiac herniation.

2.2. Management of peripheral vascular injuries

(a) Causes

- Penetrating wounds → Gunshot, Stab or shotgun
- Blunt trauma → joint displacement, bone fracture, contusion → adjacent to major artery
- Invasive procedures → Arteiography, cardiac catheterization, ballon angioplasty

(b) Hard signs of arterial injury

- Immediate surgery
- External arterial bleeding
- Rapidly expanding hematoma
- Palpable thrill, audible bruit
- Obvious arterial occlusion → pulseless, pallor, paresthesia, pain, paralysis, poikilothermia, especially after reduction of dislocation or realignment of fracture)

(c) Soft signs of arterial injury

- Consider arteriogram, serial examination and duplex scan
- History of arterial bleeding at the scene
- Proximity of penetrating wound or blunt trauma to major artery
- Diminished unilateral distal pulsation
- Small nonpulsatile haematoma
- Abnormal ankle-brachial pressure index (<0.9)
- Abnormal flow-velocity waveform on Doppler ultrasound

(d) Importance of physical examination

- Palpable distal pulse, even if diminished, suggests that proximal arterial injury is limited
- Serial examinations are mandatory

(e) Role of diagnostic studies

- Prevent unnecessary operation
- Document presence of surgical lesion
- Localize surgical lesion to plan operative approach
  - *CT angiograph*
  - *Duplex scan*

(f) Nonoperative management

- Appropriate for non-occlusive wall or intimal lesions

(g) Surgery

Options for peripheral vascular repair

- Lateral arteriorrhaphy or venorrhaphy
- Patch angioplasty
- Resection with end-to-end anastomosis
- Resection with interposition graft
- Bypass graft
- Extraanatomical bypass
- Ligation

Adjuvant techniques for limb salvage

- Intramural shunts during orthopaedic stabilization
- Extraanatomic bypass around associated soft tissue injry
- Intraarterial vasodilators such as papaverine to reverse spasm
- Intrvenous low molecular weight dextran, 500 ml every 12 hours
- Specialized tissue coverage of exposed arterial repair using local myocutaneous or free flap

Special considerations for venous repair

- Popliteal vein is repaired rather than ligated
- Ligation of femoral or iliac vein, if necessary, is usually tolerated if elastic wraps are applied to extremity, which is elevated for 7-10 days
- Complex venous repairs

(h) Postoperative care

- Monitor distal arterial pulses by portable Doppler unit
- Continue intravenous antibiotics for 24 hours if sighificant contamination of wound or if interposition graft has been inserted for arterial or venous repair.
- Consider use of antiplatelet agent for 3 months whenever vein graft or synthetic graft has been.

3. MANAGEMENT OF ACUTE AORTIC SYNDROME

(a) Definition

- Acute aortic syndrome (AAS) is a modern term and consists of interrelated emergency conditions with similar clinical characteristics and challenges.
    - These conditions include aortic dissection, intramural haematoma (IMH), and penetrating atherosclerotic ulcer (PAU and aortic rupture); trauma to the aorta with intimal laceration may also be considered.

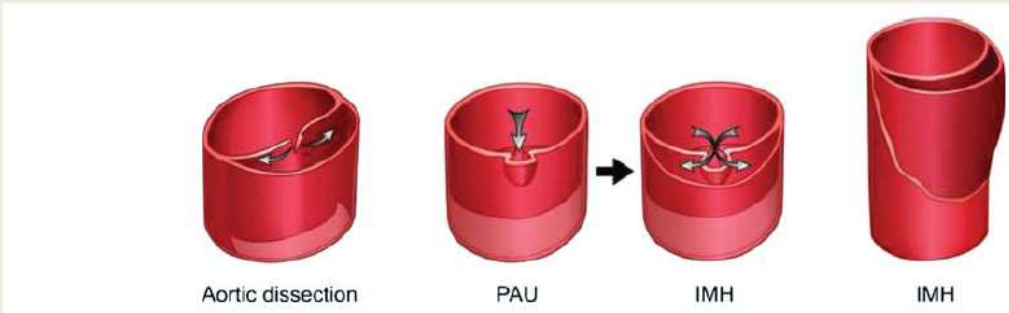


Figure 2 Schematic of aortic dissection (left), penetrating ulcer (middle), and intramural haematoma (right) all causing acute aortic syndrome.

(b) Clinical signs and symptoms

- Pain is the most commonly presenting symptom of acute aortic dissection (AAD), independent of age, sex, or other associated clinical complaint.
- Although classically described as tearing or ripping, patients are more likely to describe the pain of acute dissection as sharp or stabbing, and fluctuating.
- Pain location and associated symptoms reflect the site of initial intimal disruption and may change as the dissection extends along the aorta or involves other arteries or organs.
- Pain radiating to the neck, throat, and/or jaw may indicate the involvement of the ascending aorta, particularly when associated with murmur of aortic regurgitation, pulse differentials, or signs of tamponade; conversely, pain in the back or abdomen may herald dissection of the descending aorta.
- Pain of aortic origin may often be confused with acute coronary syndromes.
- Cardiac enzymes, troponin, and ECG changes may be instrumental in the diagnostic work-up, but only the absence of both D-dimer elevation and ECG changes is considered specific to rule out AASs.
- D-dimers when elevated above 500 mg/L appear to correlate with the extent and severity of AAD, but fail to distinguish AAS from pulmonary embolism; critically elevated D-dimer should, however, prompt undelayed computed tomography (CT) or transoesophageal echocardiogram (TEE) for confirmation of either life-threatening entity.

(c) Classification systems

- Regarding time from the onset of initial symptoms to the time of presentation, acute dissection is defined as occurring within 2 weeks of onset of pain; subacute, between 2 and 6 weeks from the onset of pain; and chronic, more than 6 weeks fromthe onset of pain.

- Anatomically, acute thoracic aortic dissection can be classified according to either the origin of the intimal tear or whether the dissection involves the ascending aorta (regardless of the site of origin). Accurate classification is important as it drives decisions regarding surgical vs. non-surgical management.

The two most commonly used classification schemes are the **DeBakey** and the **Stanford** systems.

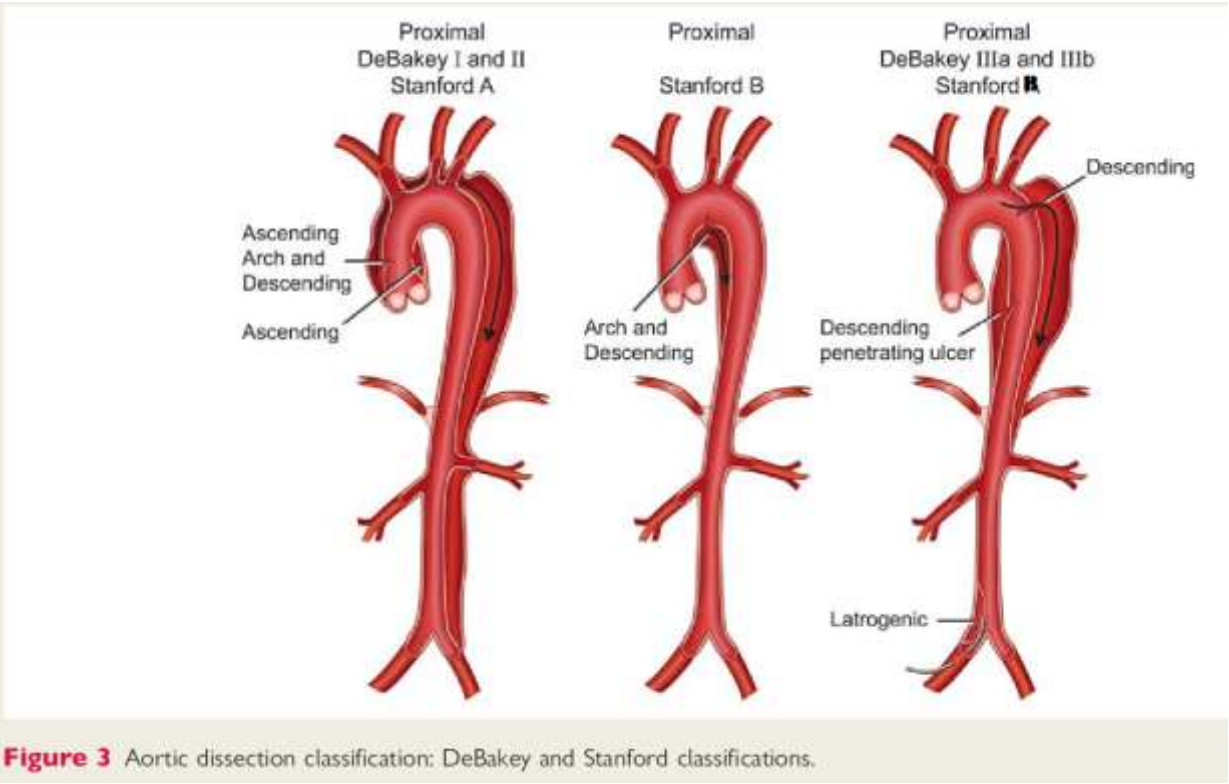


Figure 3 Aortic dissection classification: DeBakey and Stanford classifications.

(d) Intramural haematoma (IMH)

- Aortic IMH is considered a precursor of dissection, originating from ruptured vasa vasorum in medial wall layers (aortic wall apoplexy) potentially provoking secondary tear and classic aortic dissection
- IMH may, progress, dissect, regress, or resorb; two-thirds of cases are located in the descending aorta and are typically associated with hypertension.
- Similar to dissection, chest pain is more common with ascending (proximal) IMH, whereas back pain is more common with descending (distal) IMH.
- Nonetheless, the diagnosis of IMH cannot be made on clinically grounds, but by tomographic imaging in the appropriate clinical setting.
- Acute IMH accounts for 5–20% of all AAS, with regression in 10%, progression to classic aortic dissection in 28–47%, and a risk of rupture in 20–45%.

(e) Penetrating aortic ulcer

- Deep ulceration of atherosclerotic aortic plaques can lead to IMH and present as acute pain syndrome with aortic dissection or perforation.

- Symptomatic ulcers with signs of deep erosion are more prone to develop dissection or rupture.
- PAUs originate from atherosclerotic aortic segments and are localized in the descending thoracic aorta in over 90%.
- The typical patient is elderly (usually over 65 years of age), hypertensive with atherosclerosis, presenting with chest or back pain but no signs of aortic regurgitation or malperfusion; asymptomatic patients may also be found with aortic lesions indistinguishable from PAU by imaging criteria.

(f) Traumatic aortic rupture (TAR)

- With evidence of polytrauma, examination usually reveals signs similar to coarctation of the aorta with arm blood pressure higher than leg pressure, delay between radial vs. femoral artery pulsation, and a harsh interscapular murmur.
- Although the best method for diagnosing TAR is debated, chest X-ray with a nasogastric tube in position has 80% sensitivity for TAR by showing displacement of the tube by haematoma.
- A biplane contrast aortogram may fail to detect the tear until the development of a pseudoaneurysm.
- Both TEE and CT are often used to establish the diagnosis, but suffer both from limitation.

(g) Diagnostic algorithm in acute aortic syndrome

Confirmatory imaging

- In the setting of suspected aortic dissection, biomarkers (such as myocardial markers, D-dimers elevated 0.500 mg/L, may be used strategically in combination with swift imaging.

Table Diagnostic evaluation by imaging modalities

Clinical suspicion of AAS		
Non-imaging exams	**Unstable/critical conditions**	**Follow-up evaluation**
Compulsory: ECG, chest radiography, biomarkers (TNT, TNI, D-dimer >500 μg/L)	1. TEE with colour Doppler 2. MD-CT with CTA	1. MRI with Gd enhancement MRA (with or without Gd), 3D reconstruction, virtual angioscopy
Non-imaging exams	**Stable clinical condition**	**Follow-up evaluation**
Optional: ECG, chest radiography, biomarkers (TNT, TNI, D-dimer)	1. TEE with colour Doppler flow 2. MD-CT with CTA or MRI with MRA 3. Angiography rarely required	1. MRI with Gd enhancement MRA (with or without Gd), 3D reconstruction, virtual angioscopy

AAS, acute aortic syndrome; numbers denote the suggested order of diagnostic testing under given conditions. TNT, troponin I; TNI, troponin I.

- Although screening transthoracic echocardiography (TTE) provides vital information (e.g. new-onset aortic insufficiency, pericardial effusion, or even visualization of proximal dissection), additional TEE interrogation of the thoracic aorta is the logical next step, or multidetector-CT (MD-CT) scanning of the entire aorta if considered safe.
- Both imaging modalities provide further detail both in Type A and B (or distal) dissection and are useful for strategic planning. Magnetic resonance imaging has no place in urgent diagnostic work-up of acutely symptomatic patients.
- Additional information not crucial in immediate management includes arch vessel and side branch involvement usually seen on CT angiography (CTA) without the need for invasive coronary angiography even in the presence of ST-changes.

(h) Treatment concepts

- Acute aortic syndromes (dissection or IMH) involving the ascending aorta are surgical emergencies; in selected cases, hybrid approaches of an endovascular and open combination may be considered.
- Conversely, acute aortic pathology confined to the descending aorta is subject to medical treatment unless complicated by organ or limb malperfusion, progressive dissection, extraaortic blood collection (impending rupture), intractable pain, or uncontrolled hypertension.

Initial medical therapy

- Initial management of AAS, particularly dissection, is directed at limiting propagation of dissected wall components by control of blood pressure
- Reduction in pulse pressure to just maintain sufficient end-organ perfusion is a priority.
- First-line therapy is intravenous b-blockade. Labetalol, with both a- and b-blockade, is useful for lowering both blood pressure with a target systolic pressure of 100–120 mmHg and a heart rate of 60–80 b.p.m.
- Patients ideally managed in an intensive care setting.
- Opiate analgesia should be prescribed to attenuate the sympathetic release of catecholamines to pain with resultant tachycardia and hypertension.
- Further management including endovascular intervention is dictated by the site of the lesion and evidence of complications (persisting pain, organ malperfusion), as well as evidence of disease progression on serial imaging.

Care for pathology of the ascending aorta

- Acute Type A dissection has a mortality of 1–2% per hour during the first 24–48 h of presentation, and if left untreated, up to 50% of the patients will be dead in 1 week.
- Death is caused by proximal or distal extension of the dissection leading to valvular dysfunction, pericardial tamponade, arch vessel occlusion, or rupture.
- Medical management alone is associated with a mortality of 20% by 24 h and 30% by 48 h;
- In the case of pericardial effusion, pericardiocentesis is discouraged.
- Swift surgical treatment aims to treat or prevent the common and lethal by excision of the intimal tear, obliteration of entry into the false lumen, and reconstitution of the aorta with interposition of a synthetic graft with or without reimplantation of the coronary arteries.
- In addition, restoration of aortic valve competence is paramount in patients who develop aortic insufficiency. This can be achieved by resuspension of the native aortic valve or by aortic valve replacement and is dependent upon the size of the aortic root and the condition of the aortic valve.
- Operative mortality for ascending aortic dissections at experienced centres varies widely between 10 and 35%, but below the 50% mortality with medical therapy.
- Adjunctive measures such as profound hypothermic circulatory arrest and selective retrograde perfusion of the head vessels have been used in the surgical management of arch repair of an open distal anastomosis with good outcomes.

- Early open surgery is advocated, and in those surviving, it has been shown to be a durable solution.

Care for pathology of the descending aorta

- Open surgical replacement of the diseased aorta has been traditionally performed through a left posterolateral thoracotomy with prosthetic graft replacement of the descending thoracic aorta, in conjunction with single-lung ventilation, full heparinization, cardiopulmonary bypass, profound hypothermia, cerebrospinal fluid drainage, and circulatory arrest in an attempt to minimize morbidity, particularly in reference to stroke and paraplegia rates
- Given the reasonable results with medical management for uncomplicated Type B dissections, medical therapy constitutes a gold .
- Endovascular repair is developing as a strong alternative to surgery.
- Intuitive advantages include the ability to obliterate the false lumen by sealing the aortic tear with an aortic endograft.

Care for intramural haematoma

- Similar to Type A and B aortic dissection, surgery is advocated in patients with Type A IMH and initial medical therapy in patients with T B IMH.

Long-term follow-up

- Medical therapy including b-blockers is needed to minimize aortic wall stress, and serial imaging to detect signs of progression, redissection, or aneurysm formation.
- Regular assessment of the aorta should be performed after discharge and annually thereafter. Important imaging findings are progressive diameter, signs of aneurysm formation and haemorrhage at surgical anastomoses, or stent-grafted sites.
- The observation that both hypertension and aortic expansion/dissection are common and not difficult to predict early after discharge, it seems to justify such aggressive follow-up strategy.

4. MANAGEMENT OF ABDOMINAL AORTIC ANEURYSM

Abdominal aortic aneurysm (AAA) means a dilatation or widening of the abdominal aorta.

- AAA can be defined as an abdominal aortic diameter of 3.0 cm or more in either anterior-posterior or transverse planes.
- It is also defining abdominal aortic aneurysm as the maximum infrarenal aortic diameter being at least 1.5 times larger than the expected normal infra-renal aortic diameter.

(a) Clinical evaluation

- Risk for abdominal aortic aneurysms (AAAs) - > 65 years, peripheral atherosclerotic vascular disease, smoking, chronic obstructive pulmonary disease (COPD), and hypertension
- Less frequent causes include Marfan and Ehlers-Danlos syndromes, collagen vascular diseases, and mycotic aneurysm.
- Patients who have a first-degree relative with AAA are at increased risk.

(i) Clinical features

Asymptomatic

- Unimpressive back, flank, abdominal, or groin pain for some time before rupture.
- Pressure symptoms from local compression, including early satiety, nausea, vomiting, urinary symptoms, or venous thrombosis from venous compression.
- Back pain can be caused by erosion of the AAA into adjacent vertebrae.
- Other symptoms include abdominal pain, groin pain, embolic phenomena affecting the toes and fever.
- Occasionally, small AAAs thrombose, producing acute claudication.
- Patients may describe a pulse in the abdomen and may actually feel a pulsatile mass.
- An expanding AAA commonly causes sudden, severe, and constant low back, flank, abdominal, or groin pain.
- Syncope may be the chief complaint, with pain less prominent.

Symptoms of ruptured AAA

- The most typical manifestation of rupture is abdominal or back pain with a pulsatile abdominal mass.
- However, the symptoms may be vague, and the abdominal mass may be missed.
- Symptoms may include groin pain, syncope, paralysis, and flank mass.
- The diagnosis may be confused with renal calculus, diverticulitis, incarcerated hernia, or lumbar spine disease.
- Transient hypotension should prompt consideration of rupture because this finding can progress to frank shock over a period of hours.
- Temporary loss of consciousness is also a potential symptom of rupture.
- Patients with a ruptured AAA may present in frank shock, as evidenced by cyanosis, mottling, altered mental status, tachycardia, and hypotension.

- As many as 65% of patients with ruptured AAAs die of sudden cardiovascular collapse before arriving at a hospital.

(ii) Physical examination

- Most clinically significant AAAs are palpable upon routine physical examination; however, the sensitivity of palpation depends on the experience of the examiner, the size of the aneurysm, and the size of the patient.
- Patients may have normal vital signs in the presence of a ruptured AAA as a consequence of retroperitoneal containment of hematoma.
- The presence of a pulsatile abdominal mass is virtually diagnostic of an AAA but is found in fewer than 50% of cases.

The following are potential complications of AAAs:

- Death (1.8-5% mortality for elective open repair, <1% for endovascular repair, and 50% if the AAA has ruptured.
- Pneumonia (5%)
- Myocardial infarction (2-5%)
- Groin infection (<5%)
- Graft infection (<1%)
- Colon ischemia (<1% for elective repair, 15-20% if the AAA has ruptured)
- Renal failure related to preoperative creatinine level, intraoperative cholesterol embolization, and hypotension
- Incisional hernia (10-20%)
- Bowel obstruction
- Amputation from major arterial occlusion
- Blue toe syndrome and cholesterol embolization to feet
- Impotence in males - Erectile dysfunction and retrograde ejaculation (>30%)
- Lymphocele in groin (~2%)
- Late graft enteric fistula

(c) Management of Unruptured Aneurysms

- The decision to treat an unruptured AAA is based on operative risk, the risk of rupture, and the patient's estimated life expectancy.
- Operative risk is based on patients' comorbidities and hospital factors. Patient characteristics, including age, sex, renal function, and cardiopulmonary disease are perhaps the most important factors.

Table Operative Mortality Risk of Open Repair of Abdominal Aortic Aneurysm

Lowest Risk	Moderate Risk	High Risk
Age < 70 y	Age 70-80 y	Age 80 y
Physically active	Active	Inactive, poor stamina
No clinically overt cardiac disease	Stable coronary disease; remote MI; LVEF >35%	Significant coronary disease; recent MI; frequent angina; CHF; LVEF < 25%
No significant comorbidities	Mild COPD	Limiting COPD; dyspnea at rest; O_2 dependency; FEV_1 < 1 L/sec
...	Creatinine 2.0-3.0 mg/dL	...
Normal anatomy	Adverse anatomy or AAA characteristics	Creatinine >3 mg/dL
No adverse AAA characteristics	...	Liver disease (↑ PT; albumin < 2 g/dL)
Anticipated operative mortality, 1%-3%	Anticipated operative mortality, 3%-7%	Anticipated operative mortality, at least 5%-10%; each comorbid condition adds ~3%-5% mortality risk
AAA—abdominal aortic aneurysm; CHF—chronic heart failure; COPD—chronic obstructive pulmonary disease; FEV_1 —forced expiratory volume in 1 second; LVEF—left ventricular ejection fraction; MI—myocardial infarction; PT—prothrombin time.		

Table Factors Affecting Risk of Abdominal Aortic Aneurysm Rupture

	Low Risk	Average Risk	High Risk
Diameter	< 5 cm	5-6 cm	>6 cm
Expansion	< 0.3 cm/y	0.3-0.6 cm/y	>0.6 cm/y
Smoking/COPD	None, mild	Moderate	Severe/steroids
Family history	No relatives	One relative	Numerous relatives
Hypertension	Normal blood pressure	Controlled	Poorly controlled
Shape	Fusiform	Saccular	Very eccentric
Wall stress	Low (35 N/cm^2	Medium (40 N/cm^2	High (45 N/cm^2)
Sex	...	Male	Female
COPD—chronic obstructive pulmonary disease.			

- Abdominal ultrasonography can provide a preliminary determination of the aneurysm's presence, size, and extent.
- Rupture risk is in part indicated by the size of the aneurysm.
- In patients with small AAAs, attempts should be made to reduce the expansion rate and rupture risk.
- Smoking cessation is of paramount importance.
- Hypertension should be aggressively controlled.
- Beta-blocker therapy should be instituted to lower blood pressure and reduce stress on the artery wall.
- These agents can be administered safely unless the patient has a contraindication to their use (eg, COPD, allergy to the drug, bradycardia, or severe chronic heart failure).

(i) Initial Management

Prehospital care

- Ensuring adequate breathing
- Maintaining oxygenation
- Treating shock

Emergency department care

- The presence of a pulsatile abdominal mass in a patient suspected of having an AAA mandates immediate surgical intervention.
- Hemorrhagic shock is managed by means of fluid resuscitation, blood transfusion, and immediate surgical consultation.
- The concept of permissive hypotension, whereby aggressive fluid resuscitation is avoided so as not to aggravate bleeding by raising the blood pressure too much, should be taken into consideration.
- Treatment for coagulopathy may be initiated in the ED for patients who are receiving warfarin or heparin.
- Patients with leaking AAAs, if normotensive, do not require pharmacotherapy. In the setting of hypotension, reduction of blood pressure may be contraindicated.
- Initial therapeutic goals include elimination of pain and reduction of systolic blood pressure to 100-120 mm Hg or to the lowest level consistent with adequate vital organ (cardiac, cerebral, or renal) perfusion.
- Whenever systolic hypertension is present, beta blockers can be used to reduce the rate of rise of the aortic pressure.
- To prevent exacerbations in tachycardia and hypertension, patients should be treated with IV morphine sulfate.
- Patients should be admitted when they are unstable or symptomatic, when they have significant comorbid conditions, or when the diagnosis is uncertain.
- Elderly patients or those with preexisting conditions (eg, emphysema, hypertension, congestive heart failure, coronary artery disease, cerebrovascular disease, or renal insufficiency) may require stabilization before elective surgery.
- Asymptomatic patients with inflammatory AAA or AAA that is associated with distal emboli, pain, or bowel obstruction require emergency repair regardless of the size of the aneurysm.

(ii) Surgical Intervention

- There are two primary methods of AAA repair, open repair and EVAR.
- Open AAA repair requires direct access to the aorta via an abdominal or retroperitoneal approach. It is well established as definitive treatment, having been in use for over 50 years.
- EVAR is advocated for patients who are at increased risk with open repair, but until results from randomized controlled trials are available, patient preference is the strongest determinant in deciding between endovascular and open approaches.

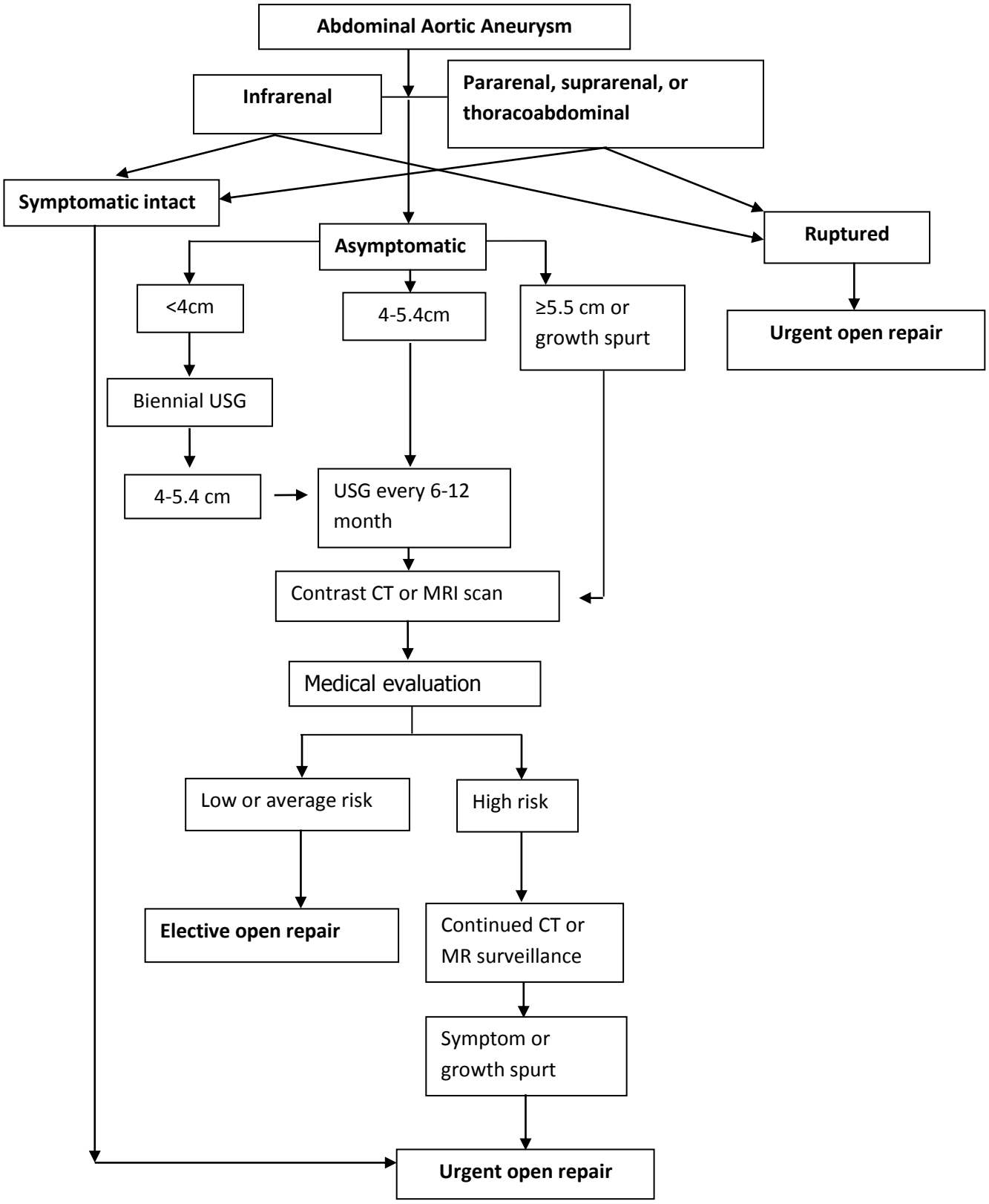
Open repair

- Open repair of thoracic and abdominal aortic aneurysms has a mortality of about 4%, with myocardial infarction being a frequent cause of death.
- Preoperative reduction of cardiac risk by means of cardiac investigations and beta blockade may lower mortality.
- Autologous transfusion techniques (eg, acute normovolemic hemodilution and intraoperative cell salvage) reduce the need for allogeneic blood and the complications associated with open surgery.

(iii) Postoperative care

- Fluid shifts are common after aortic surgery. Fluid requirements may be high in the first 12 hours, depending on the amount of blood loss and fluid resuscitation in the operating room.
- The patient should be monitored in the surgical intensive care unit for hemodynamic stability, bleeding, urine output, and peripheral pulses.
- Postoperative ECG and chest radiography are indicated.
- Prophylactic antibiotics are administered for 24 hours.
- The patient is seen in 1-2 weeks for suture or skin staple removal, then yearly thereafter.

Treatment Algorithm for Management of Abdominal Aortic Aneurysm



5. MANAGEMENT OF ACUTE LIMB ISCHAEMIA

(a) Definition

Acute limb ischaemia results from sudden interruption of the limb blood supply by thrombus, embolus or trauma. It carries a high morbidity, including loss of limb.

There are two major categories:

- Acute limb ischaemia (Embolic occlusion)
- Acute-on-chronic limb ischaemia

(b) Risk Factors

- Diabetes
- Hypertension
- Heart disease
- Hypercholesterolaemia
- Smoking
- Pre-existing peripheral vascular disease
- Abdominal aortic aneurysm, popliteal aneurysm
- Atrial fibrillation
- Haematological disorders such as polycythemia
- Malignancy

(c) Clinical Features

Symptoms and Signs of Acute Limb Ischaemia

Symptoms/signs	Comment
Pain	May be absent in complete acute ischaemia; severe pain is also a feature of chronic ischaemia
Pallor	Also a feature of chronic ischaemia
Pulseless	Also a feature of chronic ischaemia
Perishing cold	Unreliable, as the ischaemic limb takes on the ambient temperature
Paraesthesia and paralysis	Loss of function is the most important feature of acute limb ischaemia and denotes a threatened limb that is likely to be lost unless it is re-vascularized within a few hours

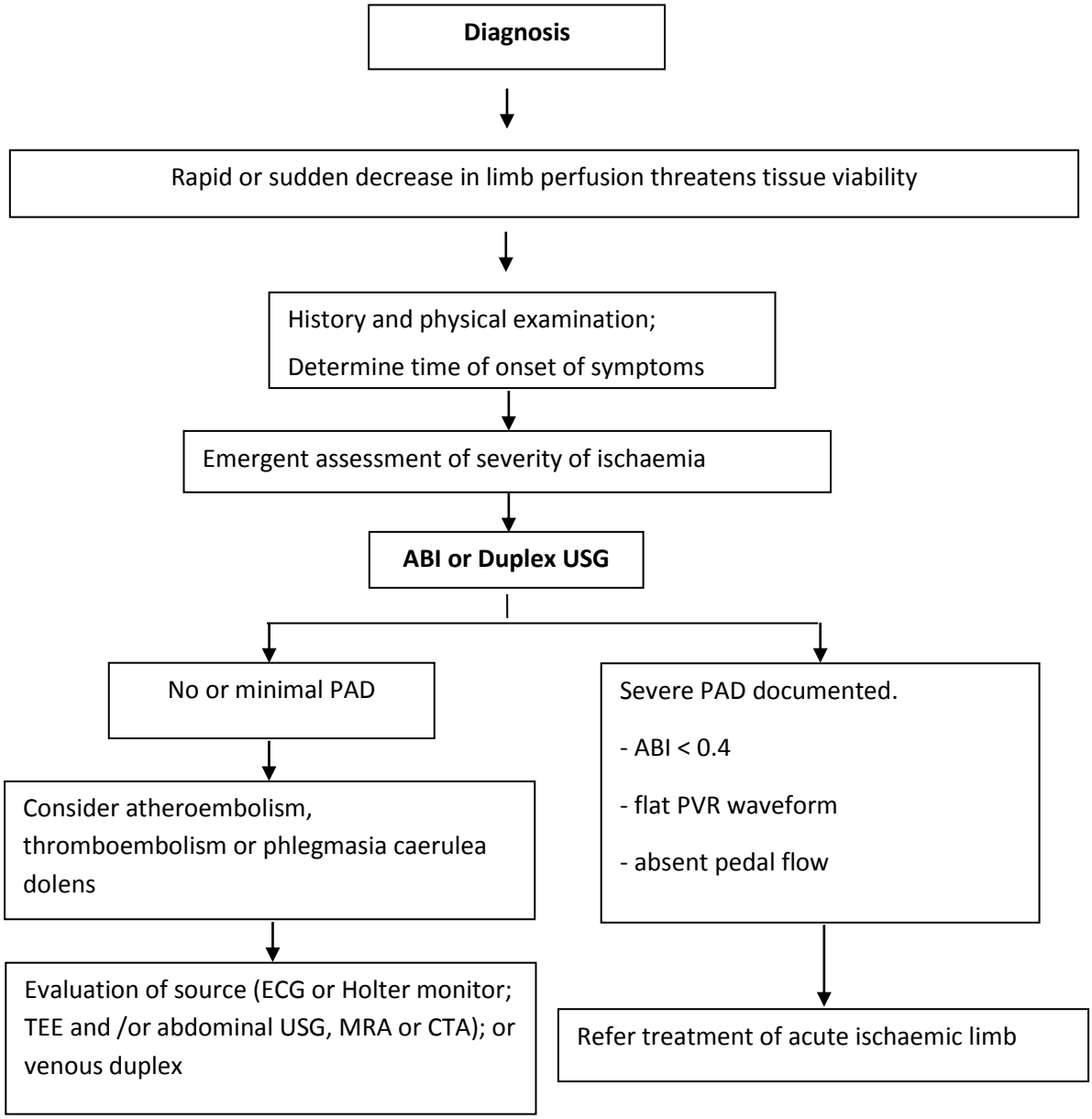
Clinical Categories of Acute Limb Ischaemia

	Clinical findings		**Doppler**		**Prognosis**
Categories	***Sensory loss***	***Motor weakness***	***Arterial signals***	***Venous signals***	
I. Viable	None	None	Audible (>30mmHg)	Audible	Not immediately threatened
II.a Marginal threat	Minimal sensory loss (toes) or none	None	Inaudible	Audible	Salvageable if prompt treatment (there is time for angiography)
II.b Immediate threat	Rest pain with sensory loss more than toes	Mild, moderate	Inaudible	Audible	Salvageable with immediate treatment (no time for angiography)
III. Irreversible	Profound anesthesia	Profound, paralysis with muscle rigor	Inaudible	Inaudible	Not salvageable, permanent N. & muscle damage , needs amputation

(d) Investigations

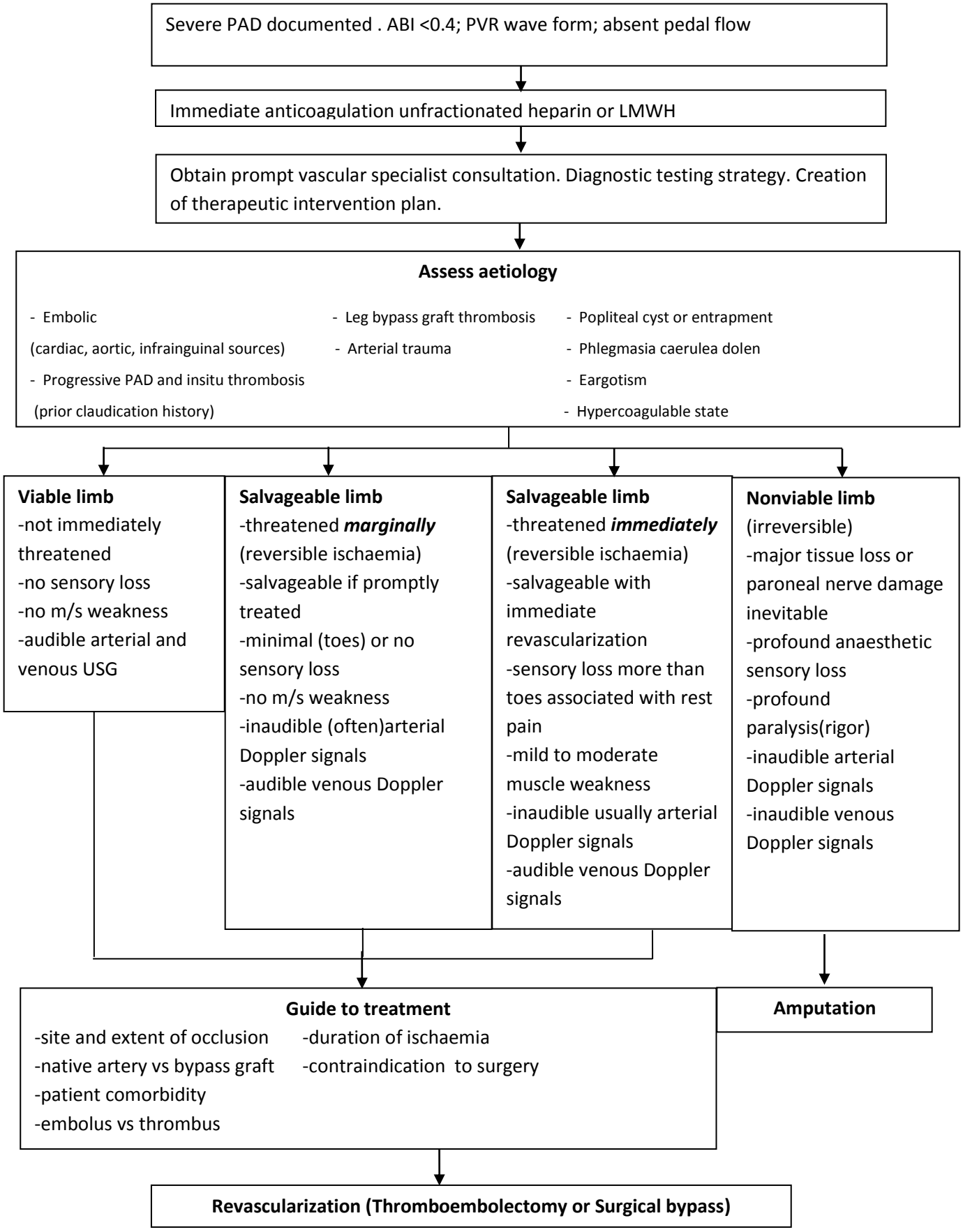
- The urgency of the situation and the availability of local resources will determine which investigations are appropriate and indeed likely to be helpful.
- In acute limb ischaemia, the aims are to evaluate underlying casues, risk factors, severity of disease and to support the treatment such as vascular surgical interventions.
- Routine or baseline laboratory & imaging studies should be obtained for treatment plans, disease progression, evaluation of causes and risk factors; such as full blood count, urea, electrolytes & creatinine, lipid profiles, serum uric acid, blood sugar, PT(INR), aPTT, infection screenings, ECG and chest X-ray.
- USG (abdomen) and CT (chest/abdomen) should be done for suspected aneurysm.

- Duplex scan or colour doppler would be helpful for treatment plans, even in acute situation, for arterial assessment and detection of aneurysm (example - Popliteal aneurysm).



- TEE = Transoesophageal echocardiography
- MRA = Magnetic resonance angiography
- CTA = CT angiography

(e) Treatment of Acute Limb Ischaemia



6. MANAGEMENT OF CHRONIC ISHAEMIC LIMB

Peripheral vascular disease commonly affects the arteries supplying the leg and is mostly caused by atherosclerosis.

- Restriction of blood flow due to arterial stenosis or occlusion often leads patients to complain of muscle pain on walking (intermittent claudication).
- Any further reduction in blood flow causes ischemic pain at rest, which affects the foot.
- Ulceration and gangrene may then supervene and can result in loss of the limb if not treated.
- The Fontaine score is useful when classifying the severity of ischemia.

Fontaine classification of chronic leg ischemia	
Stage I	Asymptomatic
Stage II	Intermittent claudication
Stage III	Ischemic rest pain
Stage IV	Ulceration or gangrene, or both

- Many patients with critical limb ischemia can undergo revascularization, which has a reasonable chance of saving the limb.
- Many patients, however, still require major amputation.
- Rehabilitation of elderly patients after amputation can prove difficult and costly.

(a) Intermittent claudication

(i) History and examination

- A history of muscular, cramp-like pain on walking that is rapidly relieved by resting, together with absent pulses, strongly supports the diagnosis of intermittent claudication.
- Disease of the superficial femoral artery in the thigh results in absent popliteal and foot pulses and often causes calf claudication.
- Disease of the aorta or iliac artery results in a weak or absent femoral pulse, often associated with a femoral bruit.
- Disease at this level may cause calf, thigh, or buttock claudication.
- The dorsalis pedis artery lies superficially on the dorsum of the foot, although its position varies considerably.
- The posterior tibial artery lies deeper behind the medial malleolus.
- Many healthy people have only one foot pulse.
- The popliteal pulse can be difficult to palpate in muscular patients. A prominent popliteal pulse suggests the possibility of a popliteal aneurysm.

Differential diagnosis

- The pain of nerve root compression can be mistaken for vascular claudication.
- A careful history can usually distinguish nerve root compression, especially sciatica due to compression of the lumbosacral root.

- Compression of the cauda equina due to spinal stenosis, however, can be more difficult to diagnose. This condition usually causes pain that radiates down both legs. Although the pain is made worse by walking, it also comes on after prolonged standing and is not rapidly relieved by rest, unlike vascular claudication.

(ii) Investigation

- Many causes of leg pain can occur in the presence of asymptomatic peripheral vascular disease.
- Therefore, the absence of pulses does not necessarily imply a causal link.
- Furthermore, the presence of pulses at rest does not exclude symptomatic peripheral vascular dis ease.
- A good history together with an ankle brachial systolic pressure index of less than 0.9 confirms the diagnosis.
- Exercise testing provides an objective measurement of walking distance and highlights other exercise limiting conditions, such as arthritis and breathlessness
- Only those with a good history of claudication and normal resting ankle brachial systolic pressure indices require an exercise test.
- Duplex ultrasound scanning is useful for delineating the anatomic site of disease in the lower limb.
- CT angiography for this purpose or when the results of duplex scanning are equivocal. This expensive investigation should not be requested unless there is a plan to proceed with revascularization, if possible.

(iii) Principles of treatment

- Intermittent claudication seems a relatively benign condition, although severe claudication may preclude patients from manual work.
- The risk of generalized vascular disease is probably more important.
- Patients with claudication have three times higher risk of death compared with age matched controls.
- Modification of risk factors is therefore vital to reduce death from myocardial infarction and stroke. All patients should be advised to stop smoking and participate in regular exercise.
- They should also be screened for hyperlipidemia and diabetes.
- Obesity reduces exercise capacity, and losing weight will improve the walking distance.

General Management of Claudication

- Patients with symptoms of intermittent claudication should undergo a vascular physical examination; including measurement of the ABI
- In patients with symptoms of intermittent claudication, the ABI should be measured after exercise if the resting index is normal
- Patients with intermittent claudication should have significant functional improvement with a reasonable likelihood of symptomatic improvement and absence of other disease that would comparably limit exercise even if the claudication was improved (e.g., Angina, Heart failure, Chronic respiratory disease or Orthopaedic limitations) before an evaluation for revascularization.

- Cilostazol (100mg PO BD) is indicated as an effective treatment to improve symptoms and increase walking distance in patients with lower extremity PAD and intermittent claudication in the absence of heart failure.
- A therapeutic trial of Cilostazol should be considered in all patients with lifestyle limiting claudication in the absence of heart failure.
- Pentoxifyline (400mg tds) may be considered as second line alternative to Cilostazol to improve walking distance in patients with intermittent claudication.

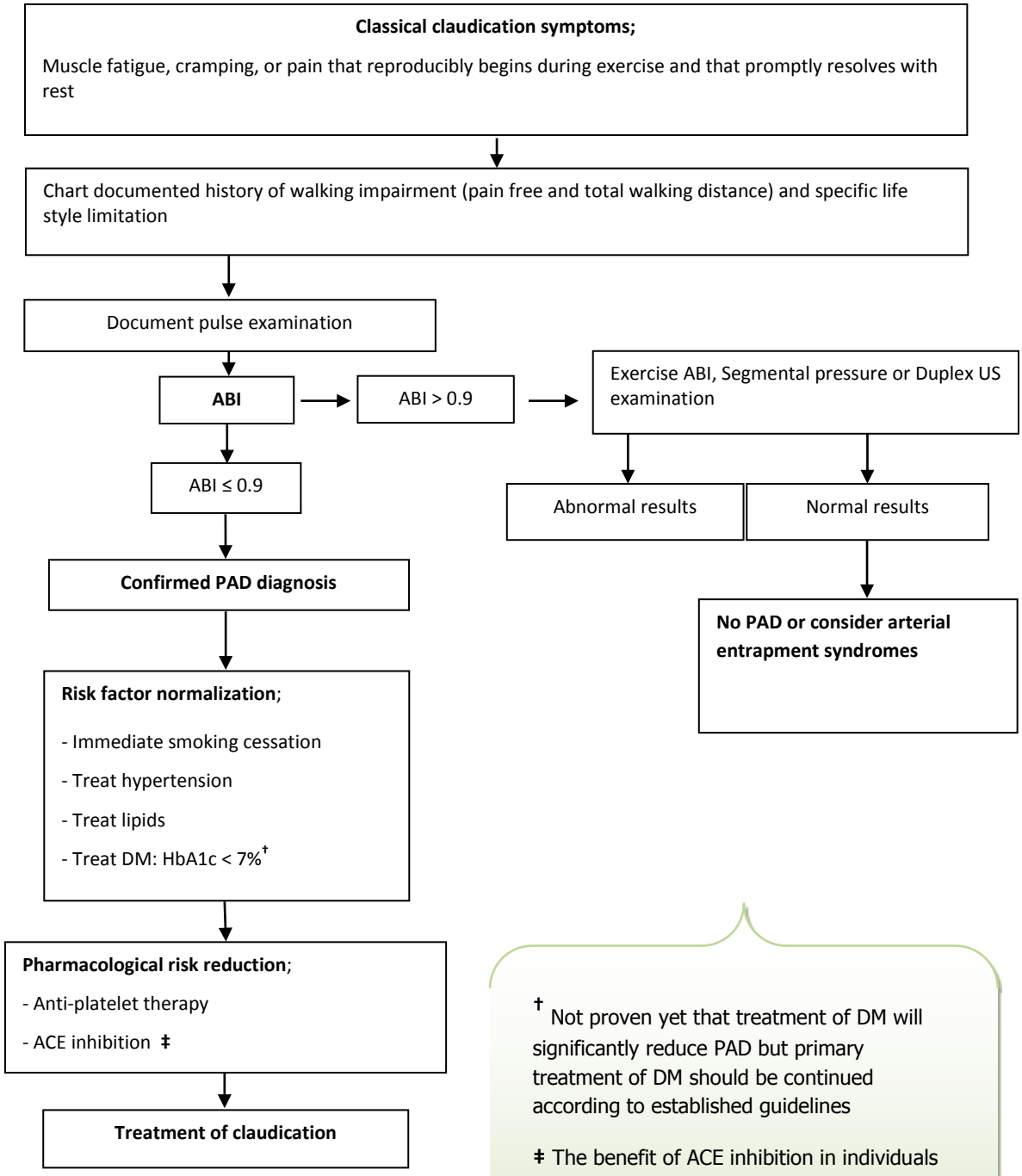
(iv) Endovascular techniques

- A number of percutaneous transluminal angioplasties performed for claudication has risen steeply in recent years.
- In some situations, endovascular techniques have virtually replaced conventional surgery.
- Percutaneous transluminal angioplasty seems best suited for stenoses or short occlusions of the iliac and superficial femoral vessels, with 1-year patency rates of 90% and 80%, respectively.
- Angioplasty carries a small but definite risk of losing the limb because of thrombosis or embolization, and patients should be informed of this risk.
- Metallic stents push back the atheroma and improve on the initial lumen gain after angioplasty alone.
- The indications for iliac stents include a residual stenosis or dissection after angioplasty and long occlusions, but there seems little evidence to justify their routine use.

(v) Surgery

- Polyester (Dacron) aortobifemoral bypass grafts have 5-year patency rates of over 90% but are associated with a mortality of up to 5%.
- Complications include graft infection and postoperative impotence.
- Femoropopliteal bypass grafting, using autologous long saphenous vein, polyester, or polytetrafluoroethylene (Gore-Tex) yields patency rates of less than 70% at 5 years.
- The early patency of prosthetic grafts seems similar to that of vein grafts, although the long-term results seem less good.
- Femoropopliteal bypass grafts should rarely be used for patients with claudication.

Diagnosis of Claudication and Systemic Risk Treatment

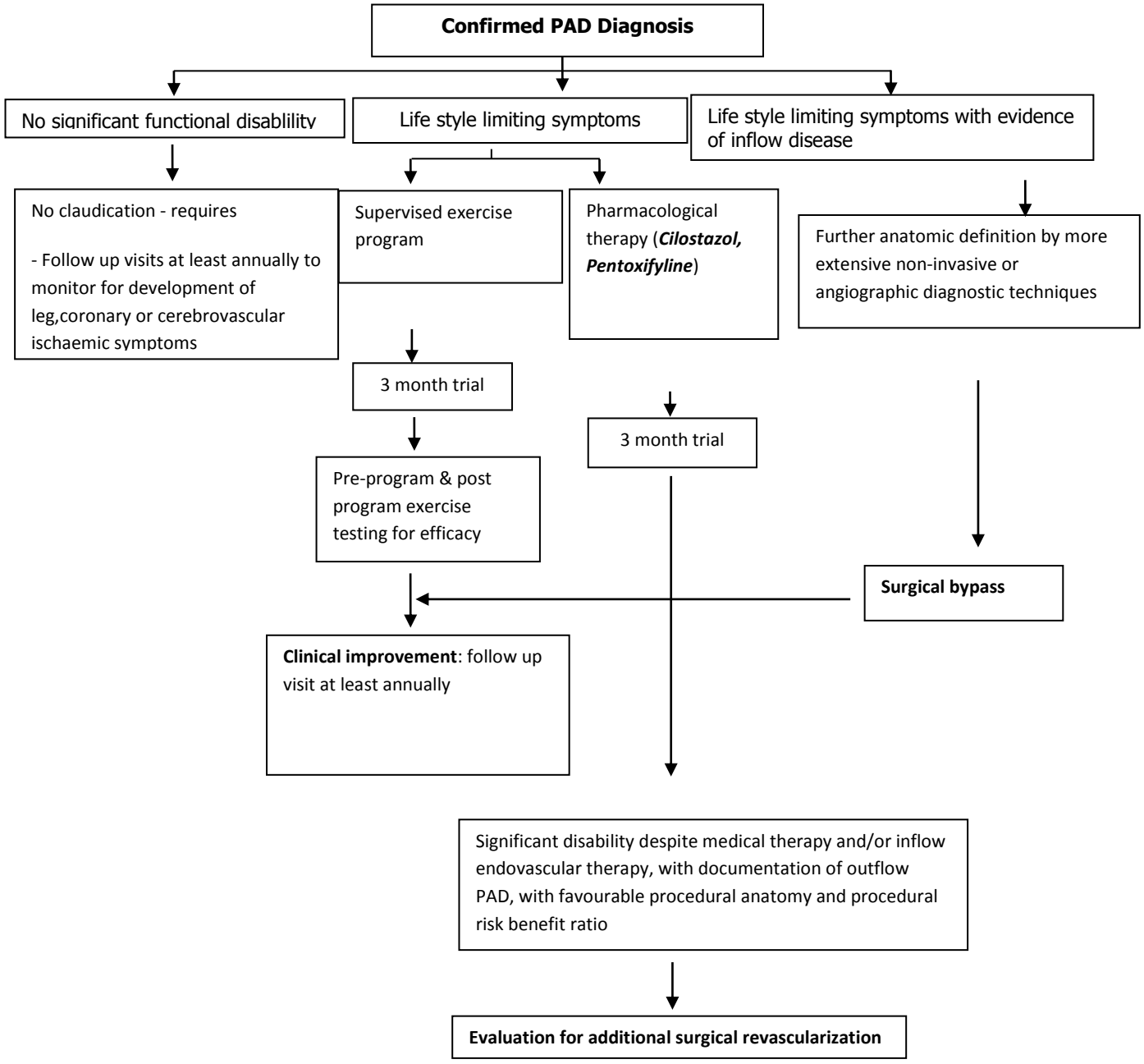


† Not proven yet that treatment of DM will significantly reduce PAD but primary treatment of DM should be continued according to established guidelines

‡ The benefit of ACE inhibition in individuals without claudication has not been specifically documented in prospective clinical trials but has been extrapolated at risk population

ABI = Ankle-Brachial Index

Treatment of Claudication



(b) Critical limb ishaemia

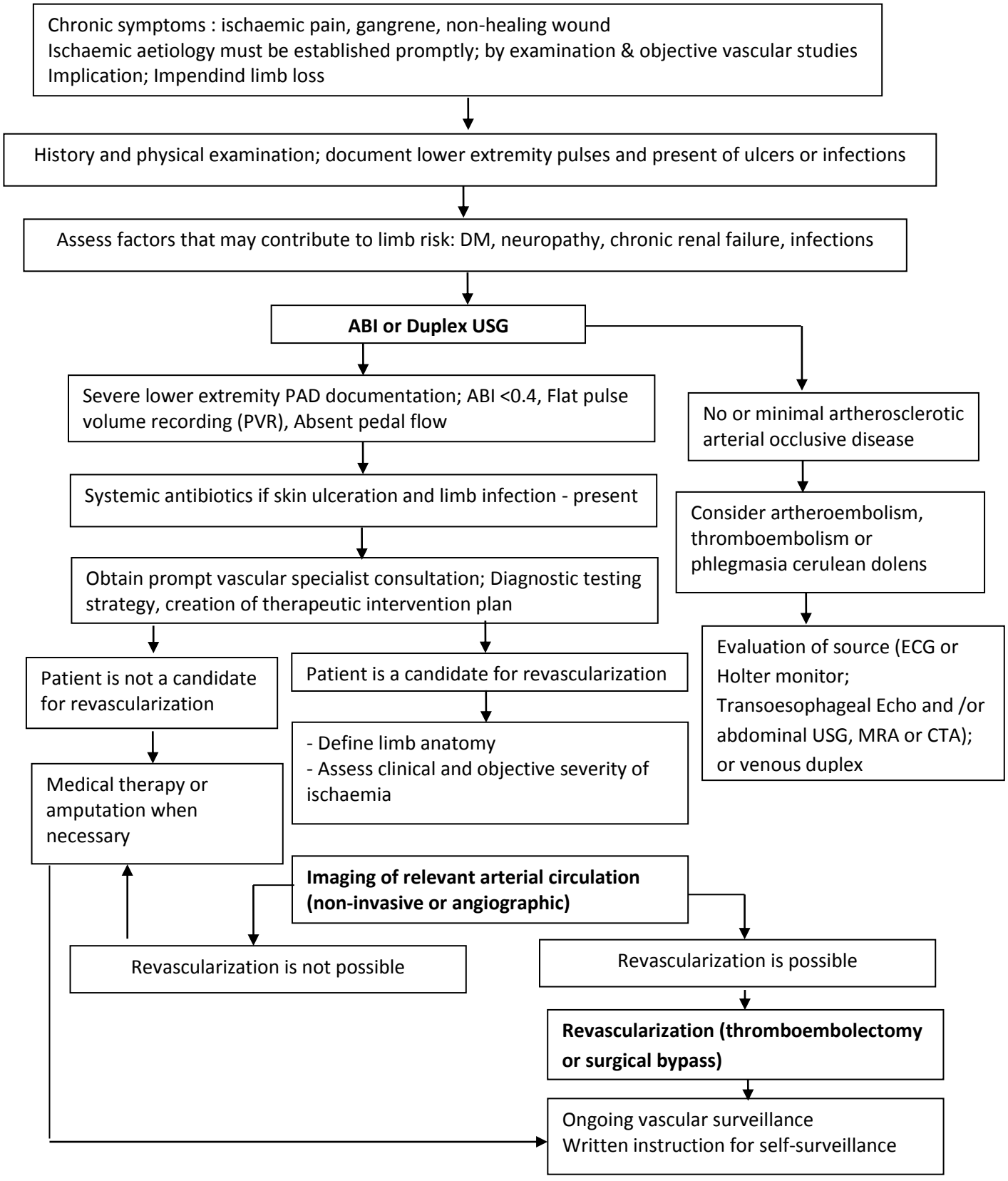
(i) History and examination

- Patients with critical limb ischemia often describe a history of deteriorating claudication, progressing to nocturnal rest pain.
- Ulceration or gangrene commonly results from minor trauma.
- Nocturnal rest pain often occurs just after the patient has fallen asleep, when the systemic blood pressure falls, further reducing perfusion to the foot.
- Hanging the foot out of bed increases perfusion and produces the typical dusky red hue due to loss of capillary tone.
- Elevation causes pallor and venous guttering. Inspect the foot carefully for ulceration under the heel and between the toes.
- Swelling suggests deep infection.
- If you can palpate foot pulses, consider an alternative cause of pain, such as gout.
- Patients with critical limb ischemia require urgent referral to a vascular surgeon.

(ii) Investigation

- The ankle brachial systolic pressure index is usually less than 0.5.
- Arterial calcification may result in falsely increased pressures, and caution is needed when relying on Doppler pressures alone, especially in patients with diabetes.
- All patients with critical limb ischemia should ideally have arteriography with a view to endovascular treatment, if feasible.
- Duplex scanning may be used instead of angiography and for mapping of the long saphenous vein before distal bypass surgery.

Diagnosis and Treatment of Critical Limb Ischaemia



7. MANAGEMENT GUIDELINES OF DEEP VEIN THROMBOSIS

(a) Clinical Assessment

- Swelling – start from distal part – extend proximally
- Tenderness, pain
- Stiffness
- Firmness and pitting oedema
- Fever may be present
- All pulsations (+)
- Associate diseases like malignancy (lungs, GIT, breast, uterus)

Risks

- Hyperlipidaemia
- Oral contraception
- Smoking
- Obesity
- Trauma
- Sepsis
- Old age
- Prolonged bed ridden patients like patients with fractures, chronic illnesses
- Non 'O' blood groups
- Pregnancy

(b) Confirmed by

- Doppler USG (Duplex scan)

(c) Treatment

(1) Aim

- Facilitating resolution of existing thrombi
- Limiting further thrombosis
- Minimizing the risks of pulmonary embolism

(2) Actions

- Bed rest (foot of the bed – elevated)
- Crepe bandaging
- Standing still and sitting with hanging legs – prohibited (usually – pain, tenderness, swelling resolve over 5-7 days)
- Ambulation started when pain, tenderness and swelling reduce (with continue elastic stocking)
- Adequate anticoagulation should be started simultaneously with above procedures

➢ initially with Heparin (SC 5000 IU 6 hourly usual dosage or low molecular weight Heparin 0.4 - 0.6ml BD) for 3-4 days

➢ then with oral Wafarin

Control - injection – Heparin (activated partial thromboplastin time-aPTT)

- Oral Warfarin (prothrobin time-PT(INR) – Range 1.3 – 1.5 times the control value

After acute episode of DVT, anticoagulation should maintained for a minimum of 3 months (may need 5 months for larger veins)

In Pregnancy

- Injection Heparin is drug of choice
- SC injection
- Allow normal delivery
- Can be continued in post-partum
- Oral Warfarin – CONTRAINDICATED

(d) Prophylaxis

(1) Aim - reducing venous stasis - altering blood coagulability

(2) Physiotherapy - early ambulation

- electrical stimulation of calf muscle

(3) Drugs - anticoagulants; inj Heaprin or Oral Warfarin

- antiplatelet – Aspirin, Dipyridamole

(should balance the risks and benefits of these drugs)

MANAGEMENT GUIDELINES FOR WAFARIN OVER ANTICOAGULATION

1. Recommendation For Managing Elevated INR Or Bleeding In Patient Receiving Vit K Antagonist
2. INR > therapeutic < 5, no bleeding => lower dose or skip dose
3. INR 5-9 no bleeding => omit next 1-2 doses => if bleeding risk , omit dose and give oral Vit K (1-2.5mg PO)
4. INR > 9 no bleeding => hold warfarin => give high dose per oral Vit K 2.5-5 mg
5. Serious bleeding with elevated INR => give high dose IV Vit K (10mg IV slow infusion), can repeat 9-12hour => consider supplementation with FFP, PCC or r VII a
6. Life threatening bleeding => hold warfarin => give FFP, PCC, or r VII a supplementation with IV Vit K (10mg IV slow infusion)
7. All patients with elevated INR should be monitored frequently
8. Resume doses should be 10-15% lower