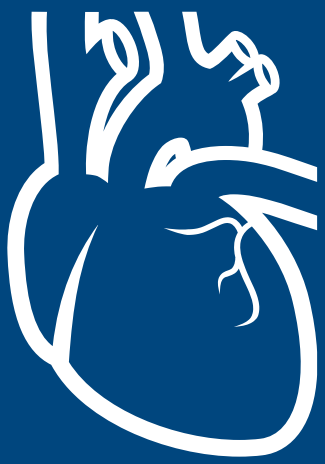
ISCAP Interventional Society for Cathlab Allied Professionals



Cardiac Catheterisation

Manual - Module 4

EDUCARE

ISCAP Interventional Society for Cathlab Allied Professionals

The ISCAP Catheterisation Manual

Endorsed by
The South African Society of Cardiovascular Intervention (SASCI)
The Society for Cardiovascular Angiography and Interventions Foundation (SCAI)

SASCI

South African Society of Cardiovascular Intervention

SCAI

The Society for Cardiovascular Angiography and Interventions Foundation

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Should one notice unintentional errors, or any information that could improve the content within, please connect with the ISCAP members within the editorials to suggest these improvements.

Foreword

Where once we were a murmur, we now have the opportunity to find our own rhythm and become the true heart of the cath lab.

Though the quality of South African Cardiology has always been on a par with the rest of the world, the training of professional nurses, technologists and radiographers (Allied Professionals), in the highly specialised field of cardiovascular intervention has been neglected. Our country has lacked guidelines that describe the requirements for a cardiovascular interventional laboratory to be managed successfully. There continues to be no official course to provide credentialing in the subject to the registered nurse.

The national Interventional Society of Cath Lab Allied Professionals (ISCAP) aims to uphold a high standard of cardiovascular interventional laboratory practice and improve the standing of the nursing and allied professional working within that environment. By these means our members will gain recognition as important participants in patient management within the cardiovascular interventional laboratory.

This second edition of the Cath Lab Manual is the continuation of this process. The Manual has been written for all those who work in the cardiovascular interventional laboratory, both as an introductory aid for the novice and as a reliable reference for the experienced practitioner.

By ensuring that educational material such as this is available on line and in hard copy , we are enabling ourselves to assume greater responsibility for our staff's development and our own job satisfaction. We also hope that the overall morale will also be enhanced.

CPD points will be attainable for those who wish to complete the questions at the end of each Chapter. There will also be a component whereby we can share information and experiences on line.

We need to equip staff with the knowledge and specific skills necessary for invasive physiology and anatomical assessment, also for the diagnosis and management of coronary and structural disease.

We trust you will find the Manual helpful. We look forward to hearing your comments and criticisms, so as to contribute to the greater value of this ongoing process of sharing information and thus learning from each other.

If you want to go quickly, go alone. If you want to go far go together

~ African proverb

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Editorial Panel

All new content is to be reviewed as it is created and thereafter approved by the panel below. This is for all digital and print versions.

Contents

4.1 High Risk Patients

Freed (1992) states that patients at increased risk of peri-procedural death-; MI, emergency CABG or a poor late outcome following PCI, are recognised as high risk cases. Procedural indications should be reviewed carefully to justify the increased risk associated with the procedure.

The impact of an adverse outcome can be decreased by identifying the high-risk patient, implementing preventative measures and recognising complications early. Some of the preventative measures include: correction of deranged electrolyte status before angiography, awareness of pre-existing AV-block, bundle branch block and bundle branch block with QT-time prolongation.

Freed (1992) continues by stating that the high risk groups include the following:

- Those with unprotected left main or left main equivalent disease
- A single vessel stenosis which supplies all remaining viable myocardium
- A proximal stenosis in a dominant vessel with high-risk lesion characteristics (thrombus, severe angulation, degenerated vein graft)
- Age > 70 years
- Ejection fraction < 40%
- Renal impairment with elevated creatinine
- Triple vessel disease
- Females
- Diabetic patient

Although angiography and intervention may represent the best form of therapy in these high risk groups, the risks and benefits of percutaneous revascularisation, bypass surgery and medical management should be discussed with the patient.

A higher level of surgical standby may be indicated in individual situations, minimising delays in transfer to the operating room in the event emergency bypass surgery is required (Freed: 1992).

STRATEGIES TO MINIMISE ADVERSE EFFECTS IN HIGH-RISK PATIENT

The diabetic patient

Askari (2011) suggests the following strategies:

- Arrange the procedure to be done early to minimise the risk of hypo- or hyperglycaemia
- A light breakfast (06h00) can be allowed, with half dose of insulin at the usual time
- Give a normal breakfast and insulin if an afternoon procedure is contemplated, with a mid-morning snack
- Monitor the haemoglycotest (HGT) pre- and post-procedure
- Allow patient to eat and drink normally after the procedure and administer insulin as per usual

A previous contrast reaction

Askari (2011) suggests the following strategies:

- Oral cortisone and anti-histamine therapy to start on the evening before the coronary angiogram
- Repeat dose on the morning of the coronary angiogram
- Continue post-procedural treatment for 48 hours
- Use an iso-osmolar contrast medium
- Administer a "test-dose" contrast medium of 5 ml, and wait for 3 – 5 minutes before the angiogram continues

The chronic renal patient

Butler (2007) suggests the following strategies:

- Patient can be admitted a day before coronary angiogram for pre-hydration therapy: 1L Saline/ 12 hours. Observe patient for pulmonary oedema
- Administer N-acetylcysteine 48 hours pre-/ and post-procedure (600 mg BD p.o) [although evidence of efficacy remains debatable] (Tepel: 2000)
- Keep contrast dose to the minimum
- Use an iso-ionic contrast medium
- Dialysis can be done post procedure to remove excess fluid and contrast medium. Renalguard.

The patient with PVD and previous femoral vascular surgery. Radial artery / dital radial artery

Chrisholm (1993) suggests the following strategies:

- Use alternative access routes: brachial/radial arteries
- Some of the problems that are associated with puncture of an old vascular femoral graft:
  - Uncontrollable bleeding
  - Haemtoma formation because of the non-vascular nature of the punctured graft
  - Disruption of the anastomotic suture line with subsequent false aneurysm formation
  - Infection of the graft site
  - Kinking and damage to the angiographic catheters due to scar tissue formation in the area and the firmness of the healed graft prosthesis
  - Inadvertent entry to the native arterial system may lead to the dead-end stump in the common femoral or iliac artery

Chrisholm (1993) advocates the following guidelines when a cardiologist is faced with this problem:

- Because the precise location of the suture line is not known and thus to avoid the anastomostic site, it is best to puncture the proximal end of the inguinal incision site as close to the inguinal ligament as possible
- To avoid kinking of the catheter at the puncture site, it is best to introduce the needle at an angle of 30° - 45° to the estimated long axis of the graft

- Sometimes, because of severe scarring, the entry site can be prepared by performing sequential dilatation with a small to progressively larger dilators (ending with a 1Fr size dilator) larger than the sheath selected for the procedure.

The patient with abdominal aorta pathology

Butler (2007) suggests the following strategies:

- Use a long exchange wire for "over-the-wire" technique to minimize plaque disruption in the aorta, but also the minimize injury to a aneurismal aorta
- Use a soft hydrophyllic wire if the femoral route is used
- Use gentle manoeuvres with guide wire and diagnostic catheter advancements to prevent perforation of the aorta
- During fluoroscopy, ensure that the dissection is not extended by cannulating a false lumen
- Be cautious not to displace thrombotic material from the aneurysm

Patients taking anticoagulant therapy

Di Mario (2011) suggests the following strategies:

- Usually patients with mechanical heart valve prosthesis
- It is safe to perform a coronary angiogram when the international normalised ratio (INR) (also known as clotting factors) is between 1.5 – 1.8
- Femoral sheath to be removed shortly after the procedure
- A vascular closure device can be used if the INR is higher than 2
- Anticoagulation can be started on the same day

The patient with acute coronary syndrome

Kern (2011) suggests the following strategies:

- The goal is to quickly and accurately assess all coronary vessels and rapidly identifying the culprit lesion
- This group of patients are at a high risk for developing life-threatening dysrythmias, cardiogenic shock and death
- Use of anticoagulation therapy places these patients at a higher risk for bleeding and vascular complications

- Catastrophic outcomes with associated diseased vessel:
- Griffiths (2010) and Askari (2011) describe the following catastrophic outcomes in association with the involved coronary artery:

1. Proximal Right coronary artery (RCA)
   - Large amount of myocardium in jeopardy
   - Closure of sinus node artery causing complete heart block
   - Closure of PDA causing mitral regurgitation
   - Persistent right ventricular infarction
2. Proximal Left anterior descending artery (LAD)
   - Very large amount of myocardium
   - Myocardium in jeopardy
   - Closure of diagonal artery causing acute mitral regurgitation
3. Sinus node artery
   - Causes complete heart block
4. First diagonal artery
   - Acute mitral regurgitation due to ischaemia or infarction of the postero-medial papillary muscle
5. First large Obtuse Marginal artery
   - Acute mitral regurgitation due to ischaemia or infarction of the antero-lateral papillary muscle
6. Right ventricular branch
   - Persistent hypotension due to right ventricular infarction
7. PDA of RCA/PDA or postero-lateral branch of CXA
   - Acute mitral regurgitation due to ischaemia or infarction of the postero-medial papillary muscle

The patient with left main artery disease

Kern (2011) suggests the following strategies:

- These patients have a twofold greater risk for complications from cardiac catheterisation
- Angiography in these patients can cause profound hypotension, and thus potentiating myocardial ischaemia
- Left main disease should be suspected in patients with a markedly poor stress test, ischaemic ECG changes in a large, anterior distribution, or in patients presenting with acute coronary syndrome with associated heart failure
- Often, the first sign suggesting left main stenosis is dampening of the arterial pressure with ostial engagement

The patient in Cardiogenic shock

Pepine (1994) and Griffiths (2010) suggests the following strategies:

- Cardiogenic shock is associated with a very high risk for mortality during coronary angiography
- The goal is to maintain coronary perfusion by inserting an intra-aortic balloon catheter
- Rapid identification of the culprit lesion is imperative

4.2 Minor and Major Complications

Emergency situations in the catheterisation laboratory can and do happen. The overall rate of major complications for cardiac catheterisation is between 1 and 2%.

It is crucial that the staff anticipate, recognise and immediately treat these emergencies. A fully equipped catheterisation laboratory will ensure better patient outcomes but competent staff plays an equally important role. All staff members need to be updated in basic as well as advanced cardiac life support skills. Most emergencies are minor with no long term sequelae, but the major emergencies may require immediate surgical intervention or may cause irreversible damage.

The risk of a major complication occurring during most procedures in current practice is generally less than 1% (Pepine: 1994). This prevalence indicates that the risk-benefit ratio still favours performing cardiac catheterisation as part of an investigation or treatment strategy for coronary artery disease. When a significant emergency/ complication occurs, the patient and the family members should be informed as soon as possible and the nature of the incident explained and an indication given whether any long term consequences are expected. An indication of all corrective interventions is also given.

MAJOR COMPLICATIONS:

- Cardiac arrest
- Cardiogenic shock
- Cardiovascular Accident (CVA)
- Congestive Heart Failure
- Contrast Recon - major (anaphylaxis)
- Death
- Mayocario infarction
- Resparetory arrest
- Vetricular tachycardia, fibrillation or serious arrythmia

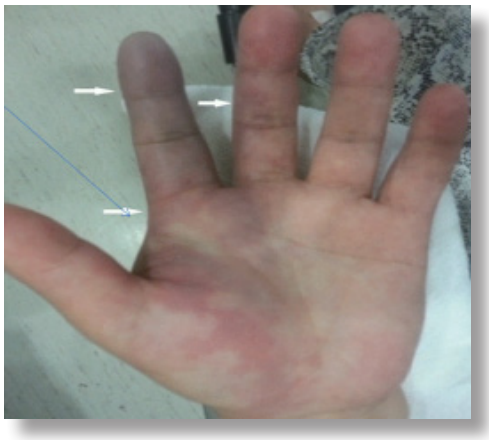

Figure 4.1 Ishaemia and necrosis of the hand if the pressure bandage was too tight.
This Patient will lose his thumb and index finger because the bandage was on for too long, and his hand did not get enough blood supply.

OTHER COMPLICATIONS

- Air Embolus
- Aortic (Ao) dissection
- Cardiac dissection/perforation
- Contrast Reaction – minor (hives)
- Heart Block/asystole
- Hematoma
- Hemorrhage (local, retroperitoneal, pelvic)
- Hypotension
- Infection
- Loss of Distal Pulse – femoral
- Peripheral vascular dissection/perforation
- Pseudoaneurysm
- Renal Failure
- Retroperitoneal bleed
- Supraventricular tachyarrhythmia, atrial fibrillation

- Tamponade
- Vascular injury, pseudoaneurysm
- Vasovagal Reaction
- Loss of radial artery pulse
- Hypotension with cardiogenic/anaphylactic shock due to other major complications such as cardiac tamponade, lethal dysrhythmias and acute myocardial ischaemia (vessel occlusion or no reflow) and severe allergy to contrast media
- Pneumohaemothorax or a pneumothorax

MINOR EMERGENCIES ASSOCIATED WITH CORONARY ANGIOGRAPHY OR INTERVENTION

ALLERGIC REACTIONS

These include urticaria, bronchospasm, facial and laryngeal oedema.

Urticaria

Treatment includes:

- Phenergan 25 mg IMI (H1 receptor antagonist)

Bronchospasm

Treatment includes:

- Oxygen administration
- Berotec or Adrenaline nebulizer
- Adrenaline 0,3 mg subcutaneous
- Phenergan 25 mg IMI
- Solucortef 200 – 400 mg IVI
- Nexium (40mg Acute)

Facial oedema

Treatment includes:

- Phenergan 25 mg IMI (H1 receptor antagonist)
- Solucortef 200mg IVI

Laryngeal oedema

Treatment includes:

- Oxygen administration
- Adrenaline nebuliser
- Adrenaline IVI: 10 mcg/min
- Intubation and ventilation if above not effective

HYPOTENSION AND SHOCK

Hypotension is one of the most common problems during coronary angiography. The following conditions can be responsible for hypotension:

1. Hypovolemia: due to inadequate pre-hydration, blood loss or excessive contrast induced diuresis

2. Reduction in cardiac output: due to myocardial ischaemia, tamponade, dysrythmias or valvular regurgitation

3. Inappropriate systemic arteriolar vasodilatation: due to vasovagal response, excessive administra-tion of nitrates or opiate analgesia

Treatment options:

- Rapid intravenous fluid resuscitation
- Inotropic support if hypotension is unresponsive to fluid resuscitation. It is important to investigate other causes for hypotension if instituted measures fails

TABLE. **Summary of the Cases and General Characteristics***

Case	Age	Sex	CV Risk Factors	Indication	Time Until Diagnosis
Pseudoaneurysm					
1	62	M	Dyslipidemia, smoking	ACS	18 h
Arteriovenous fistula					
2	52	F	No	Stable angina	48 h
3	44	M	Systemic arterial hypertension	ACS	24 h
Perforation of the radial artery					
4	66	F	No	Atypical pain	Immediate
5	65	M	Systemic arterial hypertension	ACS	15 min
6	78	M	Systemic arterial hypertension	ACS	Immediate
7	65	M	Diabetes mellitus, dyslipidemia	ACS	Immediate
8	60	M	Smoking	ACS	Immediate

*CV indicates cardiovascular; M, male; F, female; ACS, acute coronary syndrome.

RETRO-PERITONEAL HAEMATOMA

A small haematoma is not likely to cause any haemodynamic instability or an increase in the retroperitoneal cavity pressure. An extensive retroperitoneal haematoma causes compression of the lumbar plexus and produce numbness and weakness of the muscles below the knee.

The clinical presentation includes: hypotension without apparent reason, blood loss without obvious source, supra-inguinal tenderness and fullness and flank discomfort. Usually, bleeding into the retroperitoneal site is self-limiting, unless the patient is on anticoagulation therapy.

A good practice is to visualise the urinary bladder under fluoroscopy. If the bladder appears "dented", a retroperitoneal haematoma can be expected (Freed: 1992).

Treatment options:

- Reverse the effects of heparin by administrating protamine sulphate
- Fluid resuscitation to maintain a systolic blood pressure of 100 mmHg
- Blood transfusion is indicated when the Hb level is lower than 8 g/dL
- A surgical consultation is needed should the patient's condition not improve

BRADYCARDIA

A vaso-vagal reaction is featured by a bradycardia, with hypotension, nausea, vomiting, yawning and sweating. It can be triggered by pain, anxiety and hypovolemia. It is also associated with the obtaining of vascular access or sheath removal. A vaso-vagal episode is usually benign, but supportive intervention is needed to maintain the patient's haemodynamic status (Kern: 2011).

Interventions include:

- Removal of the triggered stimulus
- Rapid volume resuscitation
- Atropine (0.5 mg IVI)
- Supplemental oxygen
- Monitoring of vital data until stable

Transient slowing of the heart rate occurs commonly during angiography, particular at the end of the right coronary artery injection using high osmolar contrast agents. Forceful coughing will aid in clearing contrast agents from the coronary artery.

Conduction disturbances, like bundle branch block can also occur and may predispose the patient to bradycardia. It may be precipitated when the angiographic catheter impacts the area of the right bundle during right-sided catheterisation. In case of right bundle branch block superimposed on pre-existing left bundle branch block, asystole and cardiovascular collapse may ensue unless an adequate escape rhythm takes over. The same scenario may be seen when left bundle branch block is produced as the aortic valve is crossed in a patient with pre-existing right bundle branch block (Di Mario: 2011).

CORONARY VESSEL SPASM

Severe generalised spasm of the left or right coronary artery may occur in some patients during or after injection of the contrast medium. This can lead to severe hypotension and myocardial ischaemia. It must be treated promptly with the injection of intracoronary nitrate (50 mcgms - 200 mcgms) bolus, which may be repeated if necessary. If hypotension occurs because of the nitrate administration and vasospasm persists, inotropic support with epinephrine and repeated

intracoronary bolus injection of nitrates may be needed. Persistent vasospasm can be fatal if not treated appropriately. Intracoranary verapamil (Isoptin) as a bolus injection of 50 mcgms - 100 mcgms can also be considered in refractory cases. Coronary spasm may also be related to the catheter itself, possibly caused by mechanical irritation and a myogenic reflex. It is most commonly seen when the right coronary artery is engaged. It is related to the catheter tip that enters the ostium at an angle, which produces tenting of the proximal vessel. It may occur in the LAD artery, but this is a rare occurrence.
Balloon dilatation results in coronary endothelial denudation and loss of endothelium-derived relaxing factor (EDRF). Depletion of this potent vasodilator results in increased sensitivity to local vasoconstrictors (serotonin released from aggregated platelets) and decreased sensitivity to the vasodilating effects of prostacyclin and prostaglandin E2.

Other potential mechanisms include an increased local production or impaired ability to degrade platelet-derived vasoconstrictors (thromboxane, serotonin, platelet-activating factor), alterations in arachidonic acid metabolism resulting in over production of vasoconstricting prostanoids and leucotrienes, release of endothelium-derived contractile factor, local adrenergic nerve dysfunction and stimulation of stretch dependent myogenic tone (Baim & Grossman: 2000).

Treatment options:

- Intracoronary (I/C) Nitroglycerine (200-300 mcg)
- Remove PTCA hardware
- Calcium antagonists (I/C Isoptin 1 – 1.5 mg over 10 minutes)

VASCULAR INJURY

Vascular complications are amongst the most common complications following cardiac catheterisation procedures. These problems include vessel thrombosis, distal embolisation, vessel dissection, haematoma formation, false aneurysmformation, and arteriovenous fistula formation. Vessel perforation or rupture may also occur.

Femoral artery thrombosis is a rare complication. It is mostly seen in patients with a small calibre common femoral artery lumen (as with peripheral vascular disease, diabetes and females) and the positioning of a large-diameter sheath with a long dwell-in time. These patients complain of leg pain and numbness and have diminished distal pulses. This obstructive limb ischaemia generally resolves when the sheath is removed. Patients who have ongoing pain and diminished pulses may have a flow-obstructing dissection or thrombus at the femoral artery puncture site or a distal arterial embolus. A surgical consultation may be needed to evaluate the need for surgical intervention or anticoagulation (Brueck: 2009).

Distal embolisation is a rare complication and is attributed to prolonged procedures (in the case of electrophysiological studies) or the compression of a vessel due to haematoma formation. Clots may form inside the sheath lumen during the procedure and then flushed into the circulation at the end of the procedure. Distal pulses may be diminished and a painful pale limb can be seen. A surgical consultation may be needed to assess the need for anticoagulation or surgical exploration (Brueck: 2009).

If the arterial puncture site continues to bleed,

even after an up-sized sheath has been placed, a **ruptured vessel** may be expected. Manual compression should be done with the sheath in place. Blood volume should be maintained by fluid resuscitation or the administration of blood products. A surgical consultation may be needed to evaluate the need for surgical repair (Nguyen: 2008).

Haematomas are a more frequently seen complication and are defined as a collection of blood within the soft tissue of the upper thigh that causes a tender palpable mass. If the bleeding stops with manual compression, the haematoma will resolve within 2 – 3 weeks. Large haematomas may require blood transfusion, though surgical evacuation is rarely required (Nguyen: 2008).

A **false (or pseudo) aneurysm** is when blood flow in and out of the arterial puncture, expanding the haematoma cavity during systole and decompressing back into the arterial lumen during diastole. The haematoma cavity contains no normal arterial wall structures (media or adventitia). It can be distinguished from a normal haematoma by the presence of a pulsation or audible bruit over the site. Surgical repair is needed due to the risk of enlargement and subsequent rupture of the aneurysm. Embolisation coils can also be used as an alternative to surgical repair. Thrombin injection into the false aneurysm at its origin may also seal the false aneurysm. The keys to preventing the development of a pseudo-aneurysm are accurate puncture of the common femoral artery and effective control of the bleeding (compression technique) post sheath removal. Puncture of the superficial femoral artery or the profunda artery is more likely to lead to false aneurysm formation because it is a smaller calibre vessel and the lack of a bony structure against which to compress after sheath removal (Pepine: 1994).

Ongoing bleeding from the femoral puncture site may decompress into the adjacent venous puncture site to form an arteriovenous fistula. This can be recognised by a to-and-fro continuous bruit over the puncture site.These fistulas enlarge with time. If they do not close within 2 – 4 weeks, surgical repair may be warranted (Sanmartin: 2004).

Please note:
The following contraindications exist for manual compression of an arteriotomy site:

a. Signs of local infection
b. Critical limb ischaemia
c. Large haematoma with overlying skin necrosis
d. Injuries above the inguinal ligament

PULMONARY OEDEMA

Pulmonary oedema is usually associated with compromised ventricular functioning. It is also present in patients with an ischaemic myocardium and left main coronary artery pathology. Fluid overload is noted with the administration of hypertonic contrast agents.
Treatment options:

- Supplemental oxygen
- Mechanical or non-invasive ventilation
- Administration of diuretics
- Nebulising the patient with beta stimulants
- Administration of morphine to decrease filling pressures
- Instituting inotropic support in the de¬compensated patient is imperative to en¬sure good patient outcomes

ENTRAPPED EQUIPMENT

Although angiographic catheters have a high degree of reliability, devices can knot and become entrapped in anatomical structures or deposit fragments into the circulation.

When devices fail, it is imperative to have a contingency plan to recover them. Vascular snares, bioptomes and vascular baskets are available as bail-out devices.

To ensure that the stent is not displaced use a balloon when there is a trapped wire.

INFECTION

Coronary angiography is a sterile procedure, and thus the infection incidence is low: 0.06% is reported. Refer to the chapter on asepsis in the cardiac catheterisation laboratory for more details.

Exposure to blood through splashes, glove punctures and needle stick injuries are real contamination and infection risks. The staff should employ all protective equipment to safeguard them against these occupational hazards.

It is not acceptable to re-sterilise disposable (non-reusable) stock. It is also recommended not to use multi-dose medication vials in the laboratory.

CONTRAST INDUCED NEPHROPATHY

Contrast induced nephropathy (CIN) can occur in 15% of patients undergoing invasive cardiovascular procedures (Mehran: 2006).

CIN can occur in 50% of patients with pre-existing renal insufficiency, low cardiac output failure, nephrotic syndrome, exposure to other nephrotoxic agents (Non steroidal anti-inflammatory drugs (NSAIDS), Aminoglycosides), Diabetes, dehydration, hypertension and old age (>70 years). CIN is defined as a 25% rise in the baseline serum-creatinine level 48-72 hours after the administration of contrast media (Nguyen: 2008).

The two most important measures to prevent CIN are:

a. a proper hydration of the patient pre and post procedure
b. limiting the volume of contrast medium. As a general rule, if the volume of contrast used is < 4X the creatinine clearance/glomerular filtration rate (GFR) the risk of CIN is very low. 4-8X creatinine clearance: increasing risk of CIN, > 8X creatinine clearance: risk of CIN is almost 100%.

Treatment options for CIN:

- Pre-hydration therapy with NaCℓ 0.9% (Morcos:1999)
- NSA (N-Acetylcysteine-solmucol) prior to intervention
- Ascorbic acid (Spargias et al: 2004)
- NaHCO3- (Merten: 2004)
- Stop use of Metformin (Glucophage) at start of study and to be recommenced 48 hours after study (Uretsky: 1997)
- Use of Renalguard

MAJOR COMPLICATIONS ASSOCIATED WITH CORONARY ANGIOGRAPHY AND INTERVENTIONS

DEATH

The mortality incidence associated with coronary angiography and intervention is 0.1 – 1% (Freed:1992).

The following patient characteristics are associated with an increased mortality from coronary angiography and intervention:

a. **Age:** Infants (<1 year) and the elderly (>60 years)
b. **Functional class:** Mortality in class IV NYHA patients is 10 times greater than in class I – II patients. Class IV patients are patients with a left ventricular ejection fraction <30% and the presence of left main disease.
c. **Severity and location of coronary obstruction:** mortality is 10 times higher in patients with left main disease than in patients with single vessel disease
d. **Valvular heart disease:** In combination with coronary disease, is associated with a higher mortality rate
e. **Left ventricular dysfunction:** mortality in patients with LV function <30% is more than 10 times greater than if the ejection fraction is >50%
f. **Severe non-cardiac diseases:** patients with renal insufficiency, insulin dependent diabetics, advanced vasculopathy and severe pulmonary insufficiency appear to have an increased incidence of procedural mortality

(Adapted from Baim Donald S and Grossman William (eds). Grossman's Cardiac Catheterisation, Angiography and Intervention, 6th edn. Philadelphia: Lippincott Williams & Wilkins, 2000, p.37)

CARDIAC TAMPONADE

Pericardial tamponade is usually associated with the perforation of the cardiac chambers, coronary arteries and intra-thoracic great vessels. This complication ascribes to a low incidence of 0.8% of cases (Kern: 2011).

The right sided chambers are more commonly perforated due to their thinner walls and the use of stiffer catheters (as in the cases of trans-septal catheterisation, endomyocardial biopsy, balloon valvuloplasty, placing of temporary pacing leads and needle pericardiocentesis).
When cardiac perforation occurs, it is usually heralded by bradycardia and hypotension due to vagal stimulation. As blood accumulates in the pericardium, the cardiac silhouette may enlarge and the normal pulsation of the heart borders on fluoroscopy may become blunted. Arterial paradoxus and elevation of the right atrium pressure (with loss of y-wave) can be seen. It is essential to have a Pericardiocentesis Kit available.

Perforation of the great vessels is extremely rare. The aorta is elastic and can resist perforation, except in the case of a weakened aorta.

Pulmonary artery perforation is also very rare. The occurrence is noted by massive haemoptysis.

The incidence of perforation of the coronary vessels is 1% (Kern: 2011), and is more related to atherectomy interventions. Guide wire perforations are caused by distal positioning of stiff or hydrophylic-coated guide wires and are limited to deep injury to the vessel wall. Aggressive or overdilatation of coronary artery lesions, especially calcified lesions can lead to rupture of the artery with a false aneurysm or cardiac tamponade.

Treatment options:

- If the patient is haemodynamically stable, a

trans-thoracic echocardiogram may help to assess the extent of the tamponade

- If the patient is unstable, an emergency pericardiocentesis must be performed.
- After aspirating free blood from the pericardium, systemic heparinisation can be reversed by the administration of protamine sulphate (early protamine sulphate administration may cause blood to set in the pericardium, making aspiration impossible).
- Consult a cardio-thoracic surgeon to evaluate the need for corrective surgery. Most chamber perforations will seal spontaneously.
- Volume replacement with crystalloids, colloids or blood products
- Instituting inotropic support to maintain blood pressure
- Placing of an artificial airway to maintain the patient's airway (to avoid tracheal compression)
- Coronary vessel perforations:
- Seal the site of the leakage by inflating a balloon in the perforated segment at a low pressure
  - Deployment of a covered stent
  - Ongoing leakage may result in surgical repair
  - Extracting some fat globules from the patient's thigh/abdomen and injecting it down a microcatheter to occlude the leak

ACUTE MI

Transient myocardial ischaemia occurs commonly during coronary angiography, but responds quickly to intracoronary nitroglycerine or when the balloon is deflated.

Myocardial infarction is uncommon and ascribe to 1 - 4.8% (Butler:2007) of intra-procedural cases. Common causes that may precipitate myocardial infarction are:

a. Vessel dissection
b. Abrupt vessel closure, as seen in "snow-plow" phenomenon of side branches
c. Spasm of epicardial vessels (no-reflow phenomenon)
d. Intracoronary thrombosis
e. Distal embolisation

Chest pain post-procedure is common and relates to the stimulation of adventitial pain receptors by local stretching at the treatment site. Careful examination of the cine-angiograms should be done to evaluate loss of side branches and dissections of the coronary arteries.

LETHAL DYSRHYTHMIAS

A variety of dysrythmias or conduction disturbances may occur during coronary angiography. These dysrythmias are short-lived and rarely cause circulatory collapse. The staff in the cardiac catheterisation laboratory must be able to distinguish between the non-fatal and the fatal rhythms.

The life threatening dysrythmias are: ventricular fibrillation, pulseless ventricular tachycardia and asystole. All the ECG images were obtained from: www.ECGpedia.org.

Ventricular Fibrillation

Ventricular fibrillation occurs in 0.7% of cases (Baim & Grossman: 2000). It is usually associated with excessive catheter manipulation and intra-coronary contrast medium injection. Ventricular fibrillation may also be more prominent in patients with prolonged QT interval syndromes.

The major cause of ventricular fibrillation is due to profound myocardial ischaemia. Other causes of ventricular fibrillation include: premature

ventricular complexes with R-on-T phenomenon; electrocution; hypoxia and severe acid-base abnormalities.

Treatment options:

- Immediate defibrillation
- Immediate CPR interventions
- Emergency drugs: Adrenaline 1mg every 3-5 minutes; Cordarone X (amiodarone) 300mg bolus, and treat reversible causes (H's and T's refer page 113).

Ventricular Tachycardia

Ventricular tachycardia is seen in 30% of cases (Baim & Grossman: 2000). It is noted with passage of angiography catheters through the ventricular outflow tract and positioning of ventriculography catheters.

Other causes of ventricular tachycardia include: acute ischaemia with areas of ventricular irritability, low ejection fraction due to chronic systolic heart failure, all QT-prolongation drugs (tricyclic antidepressants, antipsychotics etc) and R-on-T phenomenon.

Treatment options:

- Synchronised cardioversion, but if the patient is pulseless, defibrillate the patient immediately
- Correct metabolic and electrolyte disturbances
- Cordorone X (Amiodarone) 300 mg IVI bolus indicated for refractory ventricular tachycardia
- If Cordorone is not available, Lignocaine can be administered: 1 – 1.5 mg/kg IVI as the first dose, followed by 0.5 – 0.75 mg/kg at 5-10 minute intervals
- Magnesium sulphate 1-2 g diluted in 10 ml saline administered over 5-20 minutes, if torsades de pointes are present

Asystole

Asystole is featured by circulatory collapse (no detectable blood pressure or pulse), unresponsiveness, agonal gasps and death! Common causes for asystole include: a dying heart, massive myocardial ischaemia, hypoxia, asphyxiation and massive electrocution (including lightning strikes).

Treatment options:

a. Immediate CPR

b. Emergency drugs: As above

Complete Heart Block (3rd Degree Heart Block)

This atrio-ventricular block is a condition in which no atrial impulses are conducted to the ventricles. Independent ventricular escape beats of a rate of 20– 60 per minute is initiated. There is no synchrony between the atria and the ventricles. The patient can complain of chest pain or shortness of breath. Decreased level of consciousness, hypotension, pulmonary congestion, shock and congestive cardiac failure may develop.

Common causes include: acute myocardial infarction, particularly the LAD and branches that supply the inter-ventricular septum, digitalis toxicity and overdose of other rate controlling medications.

Treatment options:

- Temporary pacing
- Institute inotropic support if the patient is haemodynamic unstable

The above treatment strategies are prescribed by the American Heart Association's ACLS guidelines for 2010 (Available from: www.heart.org/cpr. AccessedMay 2012).

The H's and T's

This approach is used to treat all the reversible causes for cardiac arrest.

H – Hypovolemia	T – Tension pneumothorax
H – Hypoxia	T – Tamponade (cardiac)
H – Hydrogen ions (acidosis)	T – Toxins
H – Hypo hyper¬kalemia	T – Thrombosis
H – Hypothermia	T – Thrombosis (coronary)

CVA: CEREBROVASULAR ACCIDENT

The Society for Cardiac Angiography reported the incidence of embolic incidents at 0.07%. This is a devastating complication, and although a rare complication, the incidence should be discussed with the high risk patient.

Possible embolic aetiologies include:

- Emboli released by disruption of unrecognised plaques on the walls of the aorta
- Liberating cholesterol crystals

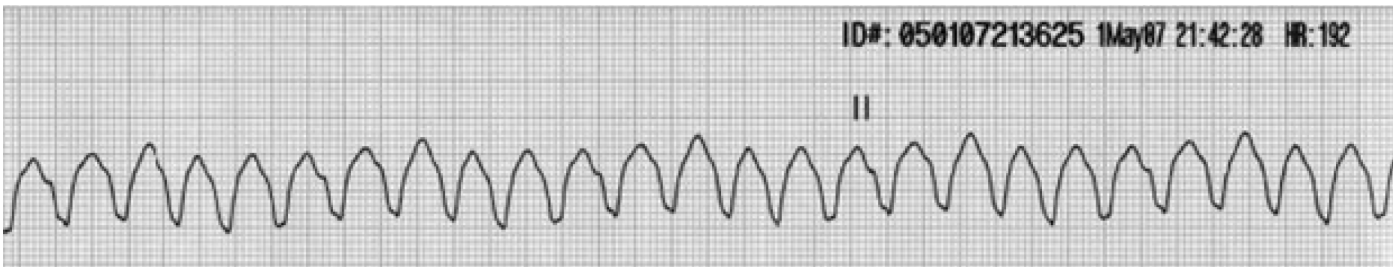

Figure 4.2: Ventricular tachycardia

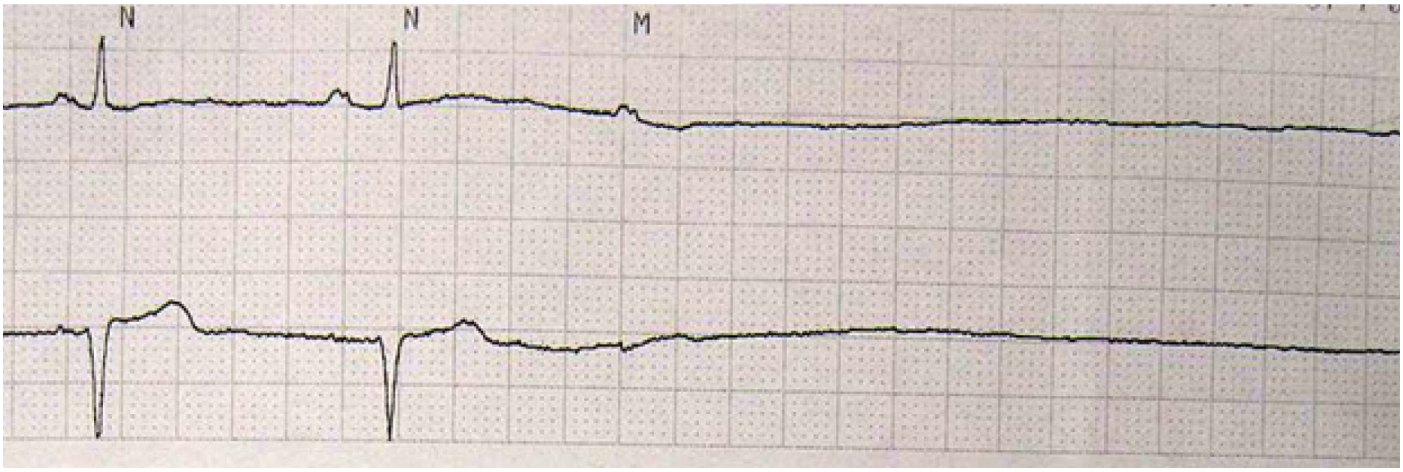

Figure 4.3: Ventricular asystole

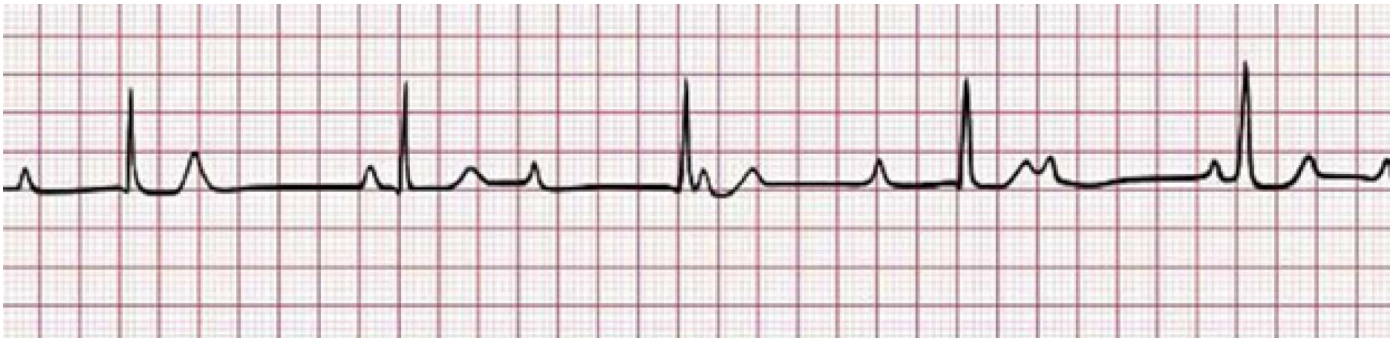

Figure 4.4: 3rd degree heart block

- Calcified material
- Platelet-fibrin thrombus
- Introduction of air bubbles during contrast injections "wire dwell times" of more than 3 minutes during attempts to cross the aortic valve
- Failure to wipe and immerse guide wires in heparinised saline before re-introduction during the catheterisation procedure
- Embolic material within the cardiac chambers (due to transmural infarctions, ventricular aneurysm or chronic atrial fibrillation)
- Embolic material on the surface of the cardiac valves (such as vegetations due to endocarditic diseases)
- Thrombus in side-arm of femoral sheath, which can be dislodged by wire or catheter

Intracerebral bleeding

Intracerebral bleeding can also occur as a complication due to aggressive anti-coagulation and thrombolytic therapy.

Di Mario (2011) suggests the following strategies to prevent embolic incidents:

- Identify high risk patients and employ anti-coagulation strategies
- Consider the use of embolic protection devices
- Angiography catheters must be advanced in a smooth manner without excessive "catching". "Catching" may indicate friable plaques.
- All catheters and sheaths must be flushed priorto use and after removal from the vessel
- If the procedure is interrupted whilst the cardiologist views the pictures - the sheath must be flushed frequently
- Ensure that blood is withdrawn from the sheath and discarded then the sheath is flushed with heparinised saline.
- Timing of the inserted guide wire and catheter when attempting to cross the aortic valve
- The use of pre-procedural heparin
- Always wipe the guide wires with heparinised saline before balloon catheters or balloon mounted stents are railed over the guide wires
- Take extra care when dealing with fresh thrombus in the coronary vessel to prevent embolisation of the clot
- Administer anti-coagulation judiciously to prevent extreme bleeding tendencies, hence precipitating intracerebral bleeding
- Treat hypertension immediately

PULMONARY INFARCTION

A rare complication seen in the cardiac catheterisation laboratory, and is mostly related to prolonged balloon inflations of the pulmonary artery catheter (Watson: 2005).

Treatment options:

- Cardio-respiratory supportive care

NO RE-FLOW PHENOMENON

Occurs immediately after balloon deflation or stent deployment, and is characterised by an acute reduction in coronary blood flow in the presence of a widely patent coronary artery (Baim & Grossman: 2000).

There is a stagnant column of contrast despite the absence of dissection, thrombus, severe spasm or high-grade residual stenosis at the PTCA/stent site.

This phenomenon is more common following angioplasty or stenting of thrombus containing lesions, degenerated saphenous vein grafts or following rotational atherectomy of vessel containing large plaque burden.

Mechanisms and mediators responsible for this micro-vascular phenomenon remains speculative: capillary obstruction consequent to distal embolisation of thrombus or atheroma, thromboxane-induced capillary spasm, oxygen free radical mediated injury manifesting as endothelial protrusions, erythrocyte rouleaux formation and neutrophil plugging (Uretsky: 1997).

Treatment strategies:

- **EXCLUDE** coronary dissection, thrombus or spasm
- Forceful injection of blood through the guide catheter in order to raise driving pressure across the capillary bed
- I/C Nitroglycerine: 100 mcg – 200 mcg
- I/C Verapamil (Isoptin): 100 mcg – 200 mcg
- I/C Adenosine (Adenocor): 12 mcg – 18 mcg
- I/C Nitroprusside (Hypoten): 40 mcg – 200 mcg
- I/C Glycoprotein IIb/IIIa bolus dose and infusion
- I/C Papaverine: 8 mg
- Increase intra-coronary perfusion (IABP)

CORONARY VESSEL DISSECTION

Coronary artery dissection is defined as the presence of either an intra-luminal filling defect, extra-luminal collection of contrast material (cap) or linear luminal density or luminal staining.

After balloon dilatation, varying degrees of plaque fracture and intimal splitting occur, creating new channels for coronary flow which give the angiographic appearance of minor coronary dissections (like intra-coronary radiolucencies or parallel tracts during contrast injection which disappear after contrast clearance).

When balloon-induced injury extends beyond the tunica intima and into the media, an extra-luminal collection of contrast is frequently evident which persists after contrast clearance from the coronary lumen (Pepine: 1994)

Treatment strategies:

- Small intimal disruptions heal spontaneously without causing massive haemodynamic instability
- A dissection that does not result in acute ischaemia will heal with time.
- Dissections which impair coronary flow or induce ischaemia require dilatation with prolonged low pressure balloon inflations
- Long dissections with more than 50% residual stenosis and impaired flow, should be stented immediately. The vessel diameter must be >2.5mm
- Securing and maintaining wire access across the occluded artery is the single most important consideration.
- When facing a spiral dissection, the distal end is stented to stop further propagation of the dissection.
- If the guide wire position is lost, the lesion can be re-crossed with a soft wire rather than a stiff wire. Review the angiograms to establish

How to identify a coronary dissection:

- An intra-luminal flap, extra-luminal linear or spiral extravasation of contrast agent, would suggest a dissection
- An intra-luminal lucency with a smooth contour in an oval shape or an area ofhaziness or a flat, rounded cut-off would indicate an intra-coronary thrombus
- No distal flow in the coronary artery

the plane of dissection and the most likely location of entry into the true lumen.

- Hypotension due to cardiogenic/anaphylactic shock resulting from other complications
- Pneumothorax occurs when the needle punctures the lung whilst accessing the subclavian vein during a permanent pacemaker or temporary pacing procedure. O2 saturating will drop and the patient becomes restless and breathless. A small pneumothorax (< 10%) can be left alone but the patient must be re x-rayed 24 hours later to ensure no progression. A mild to moderate pneumothorax (10-25%) can be aspirated by needle and syringe from the anterior chest wall. A moderate to large pneumo-thorax will need a chest drain to be placed. It is important to have a Pneumothorax set and bottle available in the Cath Lab. (Reference: Essential Cardiac Catheterisation. Butter, Gunning, Nolan. (Hadder Arnold)).

How to confirm the true lumen:

- Contrast agent will be present in the true lumen
- There is no contrast flow in a false lumen
- Many side branches can be identified coming out from a true lumen
- There are no side branches in a false lumen

- Brockenborough needle - Septum - RA to LA
  If the Brockenborough needle goes through the aorta by mistake, use an amplatzer device - PFO size to close the hole

4.3 Cardiomyopathies

Fuster et al (2011) defined cardiomyopathies as a heterogeneous group of diseases of the myocardium associated with mechanical and/or electrical dysfunction, which usually (but not invariably) exhibit inappropriate ventricular hypertrophy or dilatation, and are due to a variety of aetiologies that are frequently genetic. Cardiomyopathies are either confined to the heart or are part of generalised systemic disorders, often leading to cardiovascular death or progressive heart failure-related disability.

Cardiomyopathies are thus usually associated with failure of myocardial performance, which may be mechanical (diastolic or systolic dysfunction) or as a primary electrical dysfunction prone to life- threatening dysrhythmias.

CLASSIFICATION OF CARDIOMYOPATHIES

Fuster et al (2011) state the typical classification of cardiomyopathies as:

- Primary-genetic(hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardio-myopathy/dysplasia, left ventricular non-compaction, conduction system disease and ion channelopathies)
- Primary-mixed (genetic and non-genetic: dilated cardiomyopathy and primary restrictive non-hypertrophied cardiomyopathy)
- Primary-acquired (inflammatory cardiomyopathy and stress cardiomyopathy)
- Secondary (infiltrative, storage, toxicity, endomyocardial, granulomatous and endocrine)

PRIMARY GENETIC CARDIOMYOPATHY

Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy (HCMO) is a genetic heart disease transmitted as an autosomal dominant trait. It is caused by a variety of mutations encoding protein components of the cardiac sarcomere. It is the most common cause of sudden cardiac death in the young as well as in trained athletes. It is characterised by a hypertrophied non-dilated left ventricle in the absence of other cardiac or systemic disease capable of producing such ventricular wall thickening (like hypertension or aortic valve stenosis), independent of whether obstruction to left ventricular outflow is present.

Histologically, the myocardium is characterised by monocyte disarray, small vessel disease producing micro-vascular dysfunction, replacement scarring and increased interstitial fibrosis.

Most HCMO patients have the propensity to develop dynamic obstruction to the left ventricular outflow (hypertrophic obstructive cardiomyopathy) under resting or physiologically provocable (exercise) conditions, produced by

systolic anterior motion (SAM) of the mitral valve and ventricular septal contact.

Arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D)

ARVC/D is an uncommon form of inherited autosomal dominant heart disease. It is characterised by electrical instability and the risk of life-threatening dysrhythmias. It predominantly involves the right ventricle with progressive loss of myocytes and fibro-fatty tissue replacement, resulting in regional (segmental) or global abnormalities. Aneurysmal dilatation or bulging of the right ventricle in the triangle of dysplasia (inflow, apex and outflow) is a specific feature.

ARVC/D has a broad clinical spectrum, usually presenting with ventricular dysrhythmias and left bundle branch block morphology. Ventricular arrhythmias are due to a re-entrant mechanism with slow conduction within the myocytes embedded in fibro-fatty tissue.

Left ventricular non-compaction (LVNC)

Left ventricular non-compaction predominantly involves the apical portion of the left ventricular chamber with deep inter-/intra-trabecular recesses in communication with the ventricular cavity, resulting from an arrest in the normal embryogenesis. It can also be associated with complex cyanotic congenital heart conditions.

LVNC is characterised by left ventricular systolic dysfunction, heart failure, thromboembolism, dysrhythmias, sudden death and ventricular remodelling.

Conduction system disease

Lenègre disease, also known as progressive cardiac conduction defect, is characterised by progressive development of cardiac conduction defects in the His-Purkinje system leading to widening of the QRS-complex and AV block with long pauses and bradycardia that may trigger syncope.

Ion channelopathies

This disorder is caused by mutations in genes encoding defective ionic channel proteins governing cell and sarcoplasmic reticulum membrane transit of sodium, potassium and calcium ions.

These ion channel disorders include long QT syndrome, short QT syndrome, Brugada syndrome and catecholaminergic polymorphic ventricular tachycardia.

Brugada syndrome (first described in 1989) is identified by a distinctive ECG pattern consisting of right bundle branch block and coved ST-segment elevation followed by a negative T-wave in the anterior precordial leads (V1 – V3).

Catecholaminergic polymorphic ventricular tachycardia is characterised by syncope, sudden death and polymorphic ventricular tachycardia triggered by vigorous physical exertion or acute emotion, a normal resting ECG, and the absence of structural cardiac disease.

PRIMARY MIXED GENETIC AND NON-GENETIC CARDIOMYOPATHIES

Dilated cardiomyopathy (DCMO)

The primary feature is ventricular chamber enlargement and systolic dysfunction with normal left ventricle wall thickness. DCMO leads to heart failure and a decline in left ventricular

contractile function, ventricular and supra-ventricular dysrhythmias and thromboembolism. Other cardiomyopathies include: ischemic cardiomyopathy, hypertensive cardiomyopathy and valvular cardiomyopathy.

Primary restrictive non-hypertrophied cardiomyopathy

A rare condition where the left ventricle is not hypertrophied or dilated. Heart failure is characterised by normal or decreased volume of both ventricles associated with bi-atrial enlargement, normal left ventricle wall thickness and AV-valves, impaired ventricular filling with restrictive physiology and normal systolic function.

ACQUIRED CARDIOMYOPATHY

Inflammatory cardiomyopathy

Myocarditis may be an acute or chronic inflammatory process affecting the myocardium caused by toxins, drugs and infectious agents. There is an inflammatory cell that infiltrates leading to interstitial oedema and myocytes necrosis and ultimately, fibrosis. These processes lead to an electrically unstable substrate, predisposing ventricular dysrhythmias and sudden cardiac death.

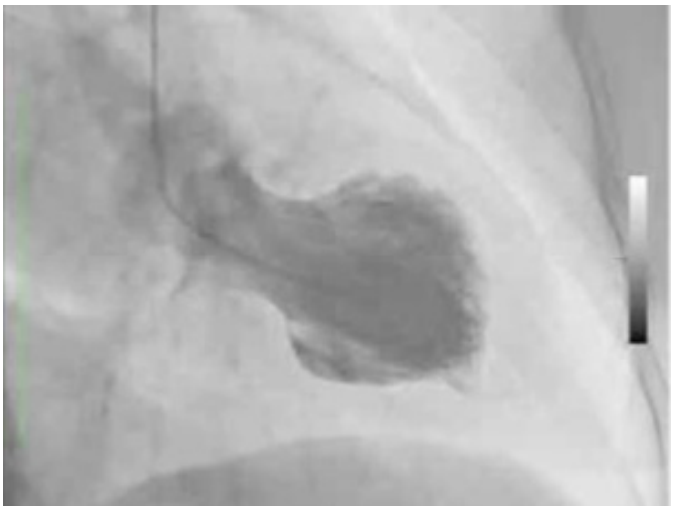

Figure 4.5: A radiographic image of Tako-Tsubo's cardiomyopathy

Stress cardiomyopathy

Also known as Tako-Tsubo's cardiomyopathy, this is an acute but rapidly reversible left ventricular dysfunction in the absence of atherosclerotic coronary artery disease, and is in most cases triggered by profound psychological stress. This form of ventricular stunning typically affects older women. There is apical ballooning and basal hypercontractility of the left ventricle. The outcome of this cardiomyopathy is favourable with appropriate medical therapy. This condition mimics an acute myocardial infarction, because with hyper-acute ST-segment elevation on ECG.

Other cardiomyopathies

Peri-partum cardiomyopathy

It is a rare, dilated cardiomyopathy associated with left ventricular systolic dysfunction and heart failure. The cause is unknown. It manifests in the third trimester, usually in obese, multi-parous women with preeclampsia. This cardiomyopathy resolves within 6 months in 50% of patients.

Tachycardia-induced cardiomyopathy

Left ventricular contractile dysfunction occurs as a result of prolonged periods of supraventricular or ventricular tachycardia. Dysfunction is attributed to a stunned myocardium. Systolic function normalises upon cessation of the tachycardia.

Alcoholic cardiomyopathy

This form of dilated cardiomyopathy is directly due to the excessive consumption of alcohol. This condition is potentially reversible upon cessation of alcohol intake.

D. SECONDARY CARDIOMYOPATHIES

Secondary cardiomyopathies show pathologic myocardial involvement as part of a vast number and variety of generalised systemic (multi-organ) disorders.

A Summary of the Causes of Secondary Cardiomyopathy

Cause	Example
Infiltrative causes	Amyloidosis
Storage aetiologies	Gaucher disease, Hurler disease, Hunter disease, haemachromatosis, Fabry disease & Nieman-Pick disease
Toxins	Heavy metals and chemicals & drugs
Endomyocardial diseases	Endomyocardial fibrosis & Löffler endocarditis
Granulomatous disease	Sarcoidosis
Endocrine disease	Diabetes, hyperthyroidism, hypothyroidism, acromegaly, pheochromocytoma, hypoparathyroidism & hyperparathyroidism
Nutritional deficiencies	Beri-beri, pellagra, scurvy, kwashiorkor, carnitine & selenium
Autoimmune/collagen diseases	SLE, dermatomyocitis, rheumatoid arthiritis & scleroderma
Electrolyte imbalances	Due to cancer therapy: radiation and the use of cyclophosphamide & anthracycline (doxorubicin)
Neuromuscular or neurological diseases	Friedrich's ataxia, muscle dystrophy, neurofibromatosis and tuberous sclerosis

4.4 Cardiac Pharmacology

Pharmacology is defined as the study of the effects of chemical substances on living tissue.

The administration of medication is a basic activity of nursing practice. Therefore, it is important that nurses are knowledgeable about the specific drugs, their administration, response and drug interactions. Nurses are accountable for the safe administration of drugs and should question those orders that are incomplete, unclear, outside a dosing range or contra-indicated in a specific situation or condition.

A broad landscape of medications is now used in the cath labs to decrease both acute and long term complications.

The use of medication in cardiology is a very dynamic field. This chapter is an overview of the medications administered to patient in the cath lab and CCU scenario.

The "Five-Rights" of drug administration:

The five "rights" of drug administration are: the right patient, the right drug, the right dose, the right time and the right route. This traditional model has been practiced to ensure safe drug administration, because nurses are legally liable when a drug is administered and these concepts are contravened.

A drug taken orally goes through 3 phases: pharmaceutic, pharmacokinetic and pharmacodynamic phase. In the pharmaceutic phase, the drug becomes a solution crossing the biologic membrane. The pharmacokinetic phase is composed of 4 processes: absorption, distribution, metabolism and excretion. In the pharmacodynamic phase, a biologic or physiologic response will ensue. When drugs are administered via the subcutaneous route, intramuscular or intravenous routes, there is no pharmaceutic phase (LeFever: 2009).

During the pharmaceutic phase, the drug needs to be in a solution so that it can be absorbed. A drug in a solid form must disintegrate into smaller particles to dissolve into a solution. Drugs in a liquid form are already in a solution. Enteric-coated drugs resist disintegration in the gastric acid, allowing disintegration within the alkaline environment of the duodenum. Enteric-coated tablets can remain in the stomach for a long time, therefore, their effect may be delayed in onset (LeFever: 2009).

Pharmacokinetics is the process of drug movement to achieve drug action. Pharmacodynamics is the study of drug concentration and its effects on the body. The primary effect of the drug is its desirable effect. The secondary effect of a drug may be desirable or undesirable, e.g.: the primary effect of Phenergan is to treat the symptoms of an allergy.

The secondary effect is that it suppresses the central nervous system leading to drowsiness. The secondary effect is undesirable if the patient has to drive his car, but when taken at bed time, the sedative effect is desirable.
(Chernow: 1995).

Drugs bind to receptors. Most receptors, protein in structure, are found on cell membranes. Drug-binding sites are primarily on proteins, glycoproteins, proteolipids and enzymes (Gahart: 2000).

This chapter is only an introduction and overview of pharmacology and medicine administration in the catheterisation laboratory, and it is not all inclusive.

This chapter will focus on the following drugs: analgesics and sedatives, vasodilators, anti- dysrhythmic agents, inotropic agents, vasopressor agents, platelet inhibitors and thrombolytics.

ANALGESIA AND SEDATIVES

Benzodiazepines are a class of medications that can cause anxiolysis, hypnosis, amnesia and sedation. Benzodiazepines also have muscle relaxant and anti-convulsant properties

.

MIDAZOLAM (Dormicum)

Midazolam (Dormicum) is a sleep-inducing agent characterized by a rapid onset and short duration of action. It also exerts anxiolytic, hypnotic, anticonvulsant and muscle-relaxant effects. Midazolam impairs psychomotor function. As with other benzodiazepines, it is believed that the effects of Midazolam are mainly mediated via agonistic binding to gamma-amino butyric acid receptors (GABAA) in the central nervous system (CNS). The hypothesis is that benzodiazepines do not directly activate GABAA receptors, but require the endogenous ligand (GABA), to exert the effects.

Table 4.1: Specific receptor actions

Receptor	Location	Response to stimulation
Alpha 1 (α-1)	Vessels of the skin, muscles, kidneys and intestines	Vasoconstriction of peripheral arterioles
Alpha 2 (α-2)	Pre-synaptic	Inhibit nor-epinephrine release
Beta 1 (β-1)	Cardiac tissue	Increased heart rate; increase conduction; increase contractility
Beta 2 (β-2)	Vascular and bronchial smooth muscle	Vasodilatation of peripheral arterioles; bron-chodilatation
Dopamine (DA1)	Renal, mesenteric, splenic, and cardiovascular smooth muscle	Vasodilatation (especially in the renal arteries); natriuresis
Dopamine (DA2)	Pre-synaptic	Inhibit nor-epinephrine and acetylcholine release

Dose: 2 – 5 mg IVI as a bolus dose
Side-effects:

- Drowsiness and over-sedation
- Disorientation, confusion and hallucinations
- Anterograde amnesia
- Hypotension
- Apnoea
- Withdrawal phenomenon
- Nausea, vomiting and hiccups

Antagonist:
Anexate (Flumazenil)
(Available from: www.drugs.com)

LORAZEPAM (Ativan)

Exerts the same effect as Midazolam. The pharmacological action appears to be in the limbic system.

Dose: 4 mg IVI or 1 mg sublingually
Side-effects:

- Drowsiness
- Over-sedation
- Disorientation
- Confusion
- Nausea/vomiting
- Vertigo
- Ataxia

DIAZEPAM (Valium)

Exerts the same effect as Midazolam. Acts as a central nervous system depressant and also depresses the duration of electrical discharges in muscle cells.
Dose: 5 – 10 mg IVI
Side-effects:

- As for midozolam
- May have paradoxal effects: restlessness, agitation, irritability, aggressiveness, delusions, hallucinations and inappropriate behaviour

PROPOFOL (Diprivan)

Used because of its rapid onset and short duration. Exerts the same effects as the other Benzodiazepines.

Dose: 1, 5 – 2, 5 mg/kg or 0, 15 – 0, 25 ml/kg
Side-effects:

- Pain at IV site
- Hypotension
- Apnoea
- Nausea/vomiting
- Hiccups
- Headache

(Available from: www.drugs.com)

ANALGESIA

Morphine Sulphate

Morphine acts as an agonist, particularly at ќ (kappa), μ (mu), and Λ (lambda) receptors in the Central Nervous System. Morphine also decreases sympathetic discharge and catecholamine release (www.healthhype.com).

Contra-indications:

- Respiratory depression, chronic obstructive airways disease (COAD) and excessive bronchial secretions, bronchial asthma
- Acute alcoholism, convulsive disorders, head injuries, raised intracranial pressure (ICP) and comatose patients
- Pregnancy and lactation
- Uncorrected shock states
- Biliary and renal colic as sphincter constriction occurs

Dose: 0.05 – 0.1 mg/kg

Side-effects:

- Drowsiness and over-sedation
- Histamine release with subsequent

anaphylactic reactions
- Pruritis, urticaria
- Disorientation, confusion and hallucinations
- Miosis
- Hypotension
- Apnea or respiratory depression
- Nausea, vomiting constipation and decreased motility
- Ureteric spasm and urinary retention
- Euphoria, that may lead to abuse and dependence

Antagonist:
Narcan (Naloxone hydrochloride)

VASODILATORS

LeFever (2009) states that vasodilating drugs cause venous and arterial dilatation, with preload and afterload reduction. This mechanism conserves cardiac energy and allows the heart to focus on contracting and moving blood through vessels with an increased capacitance.

NITRATES

Nitrates act as a vasodilator, both systemic and the coronary arteries. They produce decreased left and right ventricular end-diastolic pressures to a greater extent than the decrease in systemic arterial pressure, thereby reducing afterload and especially the after load of the heart.

Nitrates influence the oxygen supply to ischaemic myocardium by causing the redistribution of blood flow along collateral channels and from epicardial to endocardial regions by selective dilatation of large epicardial vessels.
They reduce the requirement of the myocardium for oxygen by increasing venous capacitance, causing a pooling of blood in peripheral veins, thereby reducing ventricular volume and heart wall distension (AHFS Drug information: 2000).

Dose:
- Sublingual: 0.4 mg and may be repeated up to
- 3 doses
- IV infusion: 10 mcg / minute

Side-effects:
- Facial flushing
- headache
- palpitations
- tachycardia
- nausea
- dizziness
- orthostatic hypotension and rapid fall in systemic arterial pressure that can cause decreased cerebral blood blow and decreased coronary perfusion

ANTI-DYSRHYTHMIC AGENTS

Carlson (2009) states that anti-dysrhythmic drugs comprise a diverse category of pharmacological agents used to terminate or prevent an array of abnormal cardiac rhythms. These drugs are commonly classified according to their primary effect on the action potential of cardiac cells (see table below). Some agents are more difficult to categorise, because they have characteristics of more than one class and others have no characteristics of the current classification system.
Anti-dysrhythmic drugs have the following modes of action:

- They block adrenergic stimulation of the heart
- They depress myocardial excitability and contractility
- They decrease the conduction velocity in cardiac tissue

- They increase the recovery time (repolarization) of the myocardium
- They suppress automaticity (spontaneous depolarisation to initiate heart beats)

THE CLASSIFICATION OF ANTI-DYSRHYTHMIC DRUGS

CLASS I agents decrease the influx of sodium ions through the "fast" channels during phase 0 depolarization. This prolongs the absolute (effective) refractory period, thus decreasing the risk of premature impulses from ectopic foci. In addition, these drugs depress automaticity by slowing the rate of spontaneous depolarizations of pace maker cells during the resting phase (phase 4) (Shell & Puntillo: 2001).

Shell & Puntillo (2001) state that Class 1 drugs can be further subdivided into three groups according to their potency as sodium channel inhibitors and their effect on phase 3 repolarization. Class I A agents block not only the fast sodium channels, but also phase 3 repolarisation and thereby prolong the action's potential duration. This class agents may depress myocardial contractility. Examples: Quinidine, Rythmodan (Disopyramide).

Table 4.2: The Vaughn-Williams classification of anti-dysrhythmic drugs

Classification of drug	Mechanism of action	Comment
1A	Na+ channel blocker	Slows Phase 0 depolarisation
1B	Na+ channel blocker	Shortens Phase 3 repolarisation
1C	Na+ channel blocker	Markedly slows Phase 0 depolarisation
II	β-Adrenoreceptor blocker	Suppresses Phase 4 depolarisation
III	K+ channel blocker	Prolongs Phase 3 repolarisation
IV	Ca2+ channel blocker	Shortens action potential

Class I B drugs only have a moderate effect on sodium channels and actually accelerate phase 3 repolarization to shorten the duration of the action potential. Example: Lignocaine.

Class I C agents are the most potent sodium channel blockers, with little effect on repolarization. These agents increase both the PR and QRS intervals. Examples: Tambocor

(Flecanide), Rythmol (Propafenone).

CLASS II agents are beta-adrenergic blockers. These drugs inhibit dysrythmias mediated by the sympathetic nervous system by competing with endogenous catecholamines for available receptor sites. Spontaneous depolarization during the resting phase (phase 4) is depressed and atrioventricular conduction is slowed. Drugs in this class are further divided into cardio-selective (those that only block β1 receptors) and non-cardioselective (those that block β1 and β2 receptors). Beta-blockers are negative inotropes and should be cautiously used in patients with left ventricular dysfunction. Beta-blockers are used in the treatment of supra- ventricular tachycardia's, such as atrial fibrillation and atrial flutter (Chernow: 1995). Examples: Breviblock (Esmolol), Lopresor (Metroprolol), Inderal (Propranalol). These drugs are mentioned because they can be administered intravenously.

CLASS III drugs slow the rate of phase 3 repolarization, increasing the effective refractory period and the action potential duration (Chernow: 1995). Examples: Cordarone-X (Amiodarone), Sotalol.

CLASS IV drugs are calcium channel blockers that inhibit the influx of calcium through the slow channels during the plateau phase. This effect occurs primarily in tissue where slow calcium channels predominate, mostly the SA node and AV node (Chernow: 1995). Examples: Isoptin (Verapamil), Adalat (Nifidepine), Amloc (Amlodipine), Zildem (Diltiazem).

UNCLASSIFIED agents include Adenosine and magnesium sulphate.

COMMONLY USED ANTI-DYSRHYTMIC DRUGS

A. SODIUM CHANNEL BLOCKERS LIGNOCAINE

Lignocaine is a Class 1B antiarrythmic drug. It can rapidly associate and dissociate from the sodium channels. It shortens phase 3 of the action potential. Lignocaine suppresses arrhythmias caused by abnormal automaticity and abolishes ventricular re-entry.

Indications:

- Alternative to Amiodarone in cardiac arrest from ventricular tachycardia/ventricular fibrillation (VT/VF)
- Stable monomorphic VT with preserved ventricular function
- Stable polymorphic VT with normal baseline QT-interval and preserved LV function when ischaemia is treated and electrolyte balance is corrected
- Can be used for stable polymorphic VT with baseline QT-interval prolongation if torsades suspected

Dosage:

- Initial dose: 1 – 1.5 mg/kg slowly IVI
- For endotracheal administration: 2 – 4 mg/kg
- For an infusion: Mix 1 gram into 50 ml normal saline (NS) 9% (1 mg = 20 mg)
- Infusion to be started at 12 ml/hr and weaned as soon as possible. (12 ml/hr = 240 mg/hr). This dose equates to 4 mg/minute, and is deemed as the maximum dose, due to toxicity of this drug
- The following guideline can be used for infusion:
  - 4 mg/min - 12 ml/hr
  - 3 mg/min - 9 ml/hr
  - 2 mg/min - 6 ml/hr
  - 1 mg/min - 3 ml/hr
- Discontinue infusion immediately when signs

of toxicity develop

Side-effects:

- Drowsiness
- Muscle tremors
- Slurred speech
- Convulsions
- Paresthesia
- Confusion
- Agitation

(Available from: dailymed.nlm.nih.gov/druginfo)

FLECANIDE (tambocor)

Tambocor produces a decrease in intracardiac conduction in all parts of the heart with the greatest effect on the His-Purkinje system (H-V conduction). Effects upon atrioventricular (AV) nodal conduction time and intra-atrial conduction times, although present, are less pronounced than those on ventricular conduction velocity. Significant effects on refractory periods were observed only inthe ventricle.

- **Indications:** In patients without structural heart disease, flecanide is indicated for the prevention of paroxysmal supra-ventricular tachycardia (PSVT), including atrioventricular nodal re-entry tachycardia, atrioventricular re-entry tachycardia
- Flecanide is also indicated for the prevention of ventricular arrhythmias, such as sustained ventricular tachycardia (sustained VT)

Contra-indications:

- Patients with pre-existing second- or third-degree AV block, or with right bundle branchblock when associated with a left hemiblock (bifascicular block), unless a pacemaker is present to sustain the cardiac rhythm should complete heart block occur.
- Flecanide is also contraindicated in the presence of cardiogenic shock or known hypersensitivity to the drug.

Note: Flecanide, like other antiarrhythmic agents, may cause new or worsened supraventricular or ventricular arrhythmias.

Precautions:

- Not to be used in patients with heart failure
- Tambocor should be used only with extreme caution in patients with sick sinus syndrome because it may cause sinus bradycardia, sinus pause, or sinus arrest.

Dose:

A bolus dose of 2 mg/kg over 2 – 3 minutes

Side-effects:

- Dizziness, faintness, unsteadiness
- Visual disturbance
- Cardiovascular: tachycardia, sinus pause or arrest
- Vomiting, diarrhoea, dyspepsia, anorexia
- Facial flushing
- Increased sweating
- Bronchospasm

(Available from: home.intekom.com/pharm)

B. BETA-BLOCKERS METOPROLOL (Lopressor)

Dose:

5 mg IVI slowly, up to a total dose of 15 mg

Contra-indications:

See above

(Available from: home.intekom.com/pharm)

PROPRANOLOL (Inderal)

Dose:

0.5 – 1 mg over 1 minute IVI. May repeat dose to a total dose of 0.1 mg/kg.

Contra-indications:

Cocaine induced ACS

Side-effects:

- Hypoglycaemia
- Dizziness
- Fatigue
- Thrombocytopenia
- Bradycardia
- CCF
- Cold extremities
- Hypotension
- Bronchospasm (in Asthmatic patients)
- Impotence

(Available from: home.intekom.com/pharm)

C. DRUGS WHICH PROLONG REPOLARISATION

AMIODARONE (Cordarone)

Amiodarone prolongs the duration of the action potential, particularly in the nodal and Purkunje tissue. It depresses membrane responsiveness, and prolongs the refractory period in the:

- Atria
- AV – node
- His - Purkinje System
- Ventricles
- Accessory atrioventricular conduction pathways.

It also reduces conduction rate in the:

- Atria
- AV-node
- Accessory pathways

Indications:

Where rapid response is required in the control of tachy-arrhythmias:

- Wolf-Parkinson-White
- Paroxysmal tachy-arrhythmias:
- Supraventricular dysrhythmias
- Nodal dysrhythmias
- Ventricular dysrhythmias
- Atrial flutter
- Atrial fibrillation
- Ventricular fibrillation

Contra-indications:

- Sinus bradycardia
- Sino-atrial heart block
- Sinus node disease
- Severe respiratory failure
- Circulatory collapse
- Severe arterial hypotension
- History of thyroid dysfunction
- Patients with known hypersensitivity to iodine

Amiodarone intravenous infusion:

- Amiodarone IV is INCOMPATIBLE with Saline 0.9%
- Should be administered in Dextrose 5%

Loading dose:

- 5mg/kg in 200ml Dextrose 5% over 20 min
- ±300mg in 200ml Dextrose 5% continuous infusion

Side-effects:

- May cause moderate and transient reduction in blood pressure
- Too rapid administration or overdose:
  - Severe hypotension followed by circulatory collapse
  - Anaphylactic shock
  - Temporary hot flushes
  - Sweating

- Nausea

- In asthmatics:
  - Bronchospasm
  - Apnoea

Cardiac:

- Bradycardia
- Conduction disturbances:
  - Sino-atrial block
  - Various degrees of AV-block
- Intravenous amiodarone infusion induces ECG changes.
  - QT interval lengthening corresponding to prolonged repolarisation
  - U-waves and deformed T-waves may occur because of the fixing of amiodarone in myocardial tissues
  - These are not signs of intoxication and administration may continue.

- Excessive a dosage may lead to severe brady-cardia and to conduction disturbances with the appearance of an idio-ventricular rhythm, par-ticularly in the elderly patients or during digitalis therapy.
  - Treatment should be withdrawn.
  - Atropine has been used successfully to treat bradycardia.

Hepatic:

Elevations of liver enzymes may occur during IV therapy and are usually transient or respond to reduction in dosage.

Neurological:

- Benign raised intracranial pressure.
- Headaches
- Sleeplessness

(Available from: home.intekom.com)

SOTALOL (Sotacor) is a beta-blocker with additional Type III anti-arrhythmic properties.

Indications:

Sotalol is indicated for the treatment of supra-ventricular arrhythmias and ventricular arrhythmias in patients without structural heart disease.

Dose:

40 mg IVI over 5 minutes

Precautions:

- Should be avoided in patients with poor perfusion because of its significant negative inotropic effect
- Use with caution with other drugs that prolong QT-interval
- Do not use in patients with impaired renal function (due to toxicity)

Side-effects:

- Bradycardia, AV-block
- Hypotension
- Torsades de pointes

(Available from: home.intekom.com)

LABETALOL (Trandate)

Labetalol is a reversible β-blocker with concurrent α1-blocking actions that produce peripheral vasodilatation, thereby reducing blood pressure. It is used in hypertensive patients with angina, acute MI, hypertensive crisis and to produce controlled hypotension during surgery.

Dose:

1. Mix 200mg in 200ml NS
2. Administer a bolus dose of 10 mg IVI
3. Commence infusion at 1 – 2 mg/min, until normotension is achieved. Maximum dose is

160 mg/hr.

4. Reduce dose to 0.5 mg/min or stop infusion. Maximum cumulative dose within 24 hrs is 300 mg. Long duration of action facilitates conversion to oral therapy. Maximum effect is seen 5 minutes after administration.

Precautions:

- Caution should be taken in administering trandate to patients with hepatic disease, or patients treated for heart failure
- Use with caution in patients already treated with β-blockers

Side-effects:

- Hypotension
- Dizziness
- Head ache
- Nausea
- Tiredness
- Cramps
- Bradycardia

(Available from: www.accessmedicine.com)

D. CALCIUM CHANNEL BLOCKERS

LeFever (2009) states that calcium channel blockers (CCB) inhibit calcium ion movement from plasma into cells through calcium channels, thereby limiting vascular smooth muscle contraction.

Gahart (2000) categorises calcium channel blockers into 3 types: phenylalkylamine, benzothiazepine and dihydropyridine.

VERAPAMIL (Isoptin)

Dose:

2.5 – 5 mg IVI over 2 minutes. May repeat a second dose of 5 – 10 mg up to a total dose of 20 mg. An alternative dosing regimen: 5 mg bolus every 15 minutes to a total dose of 30 mg.

Precautions:

- Only to be administered for narrow-complex re-entry SVT
- Do not use in case of Wolf-Parkinson-White syndrome, atrial fibrillation, AV-block and sick sinus syndrome
- Concurrent use with β-blockers may cause severe hypotension

Side-effects:

- May decrease myocardial contractility
- Can produce peripheral vasodilatation
- AV-block
- Headache
- Fatigue
- Palpitations
- Gastrointestinal disturbances

Antagonist:

Calcium channel blocker overdose can be managed by the administration of Calcium gluconate or Calcium Chloride

DILTIAZEM (TILAZEM)

Dose:

15 – 20 mg IVI over 2 minutes. May repeat dose in 15 minutes @ 20 – 25 mg over 2 minutes

Indication:

- To control ventricular rate in atrial fibrillation and atrial flutter
- May terminate re-entry arrhythmias that require AV nodal conduction for their propagation

Precautions:

- Do not use in wide complex tachycardia
- Do not use in Wolf-Parkinson-White syndrome
- Concurrent use with β-blockers may cause severe hypotension

Side-effects:

- Gastrointestinal disturbances
- Confusion
- Headache
- Hypotension
- Dizziness
- Bradycardia
- AV-block
- Facial flushing

(Available from: AHFS Drug information: 2000)

E. UNCLASSIFIED AGENTS ADENOCOR

Adenosine

Administered by rapid IV injection slows conduction through the AV node. This action can interrupt re-entry circuits involving the AV node and restore normal sinus rhythm in patients with paroxysmal supra ventricular tachycardia. Once the circuit has been interrupted the tachycardia stops and normal sinus rhythm is re-established. Neither the kidney nor the liver is involved in the degradation of exogenous adenosine (Sinz & Navarro: 2001).

FIRST DOSE:

- 6 mg
- As a rapid IVI bolus over 1 - 3 seconds
- Followed by saline flush of 20 ml
- If Cardioversion does not occur within 1-2 minutes proceed to 2nd dose

SECOND DOSE:

- 12 mg
- As a rapid IVI bolus over 1 - 3 seconds
- If Cardioversion does not occur within 1 -2 minutes proceed to 3rd dose

Table 4.3: The different types of calcium channel blockers (CCB)

Type	CCB	Heart rate	Vasodilatation	Contractility
Phenylalkylamine	Isoptin	Slow	Some	Decrease
Benzothiazepine	Tilazem	Slow	Some	Decrease
Dihydroperidine	Adalat	May increase	Yes	Promote

THIRD DOSE:

- 12 mg
- As a rapid IVI bolus over 1 - 3 seconds
- Cardioversion should occur within 1 - 2 minutes

Injection technique:

- Place patient in mild reverse Trendelenburg position before administration of drug
- Record rhythm strip during administration
- IV saline flush and Adenocor dose in 2 separate syringes
- Attach both syringes in the IVI injection part closest to patient (3 way stopcock)
- Administer IVI Adenocor, in 1 - 3 seconds
- While maintaining pressure on Adenocor plunger, push saline flush as rapidly as possible, and elevate the legs

MAGNESIUM SULPHATE

Sinz & Navarro (2001) stipulate:

Dose:

1 – 2 g in 50 ml Dextrose over 20 minutes

Precautions:

- May cause hypotension with rapid administration
- Use with caution in the presence of renal failure

INOTROPIC AGENTS

Carlson (2009) states that inotropes amplify the contractile force of the heart. Inotropes increase the influx of calcium into the cardiac muscle cells to aid in myofibril shortening. Catecholamine drugs mimic central nervous system effects and increase contractility by increasing the production of cyclic adenosine monophosphate (cAMP).

ADRENALINE

Adrenaline is a catecholamine, which activates both α– and β-adrenergic receptors. At low doses, beta effects results in vasodilatation, and in high doses, vasoconstriction. Adrenaline strengthens myocardial contractility (positive inotropy) and increases the rate of contraction (positive chronotropy), and thus increasing cardiac output. Adrenaline also acts as a powerful bronchodilator (β2 effect).

Dosing:

Titrate to effect

DOBUTAMINE (Dobutrex)

Dobutamine directly stimulates β-adrenergic receptors and is generally considered a selective β1-adrenergic agonist. It is believed that the β-adrenergic effects result from stimulation of adenyl-cyclase activity. In therapeutic doses, Dobutamine also has mild β2 - and α1 - adrenergic receptor agonist effects, which are relatively balanced and result in minimal net direct effect on systemic vasculature. The main effect of therapeutic doses of Dobutamine is cardiac stimulation. The positive inotropic effect of the drug on the myocardium appears to be mediated principally via β1-adrenergic stimulation.

The β1-adrenergic effects of Dobutamine exert a positive inotropic effect on the myocardium and result in an increase in cardiac output due to increased myocardial contractility and stroke volume. In therapeutic doses, Dobutamine causes a decrease in peripheral resistance; however, systolic blood pressure and pulse pressure may remain unchanged or be increased because of augmented cardiac output. With usual doses, heart rate is usually not substantially changed. Coronary blood flow and myocardial oxygen consumption are usually increased because of increased myocardial contractility.

Dobutamine facilitates atrio-ventricular conduction. The tendency of Dobutamine to induce cardiac arrhythmias may be slightly less than that of dopamine. Dobutamine does not seem to affect dopaminergic receptors and causes no renal or mesenteric vasodilatation; however, urine flow may increase because of increased cardiac output.

Indication:

Dobutamine is indicated in adults who require inotropic support in the treatment of low output cardiac failure associated with myocardial infarction, open heart surgery, cardiomyopathies, septic shock and cardiogenic shock.

Dose:

3 – 10 mcg/kg/min infusion

(Available from: www.intekom.com)

DOPAMINE (Intropin)

Dopamine (3.4-dihydroxyphenylethylamine) is the third naturally occurring catecholamine and is a metabolic precursor of nor-adrenaline and adrenaline. Dopamine is used therapeutically as the hydrochloride and its main effects are seen in the cardiovascular system and the kidneys.

Heart:

Dopamine exerts positive inotropic and chronotropic effects on the myocardium, acting as an agonist at beta-adrenergic receptors. In addition to its direct action on beta-adrenergic receptors, dopamine acts indirectly by releasing nor-adrenaline from sympathetic storage sites.

Blood Vessels:

Depending on the vascular bed being studied and the dose administered, Dopamine can cause relaxation or contraction of vascular smooth muscle.

Dopamine Receptors:

Unlike other endogenous catecholamines or sympathomimetic amines, Dopamine caused vasodilatation in renal, coronary, mesenteric and intracerebral arterial vascular beds. This vasodilator effect is not antagonised by beta-adrenergic blockers, atropine or antihistamines. However, butyrophenones, phenothiazines, apomorphine and bulbocapnine selectively attenuate dopamine-induced vasodilatation, thus suggesting the existence of specific dopamine vascular receptors similar to those in the basal ganglia and other areas in the central nervous system.

Alpha-adrenergic Receptors:

Dose response studies indicate that with a sufficiently large dose, the vasoconstrictor effect of dopamine predominates over its vasodilator effect.

Kidneys:

Intravenous infusions of dopamine (3.0 - 5.0 µg/kg/min) increases the average renal flow. Although the diuretic and natriuretic effects of dopamine may result from vasodilatation in renal vascular bed (vide supra), disassociation between natriuresis and increments in renal blood flow has been observed, suggesting other mechanisms such as redistribution of intra-renal blood flow.

- Other common side effects: Ectopic heart beats, tachycardia, anginal pain, palpitations, hypotension, vasoconstriction, nausea, vomiting, headache, dyspnoea.
- Gangrene of the feet has occurred following doses of 10 - 14 microgram/kg/min and higher in a few patients with pre-existing vascular disease

(Available from: www.accessmedicine.com)

VASOPRESSORS

Schell & Puntillo (2001) define a vasopressor as an agent that constricts vascular beds, thus increasing both preload and after load. By increasing systemic vascular resistance, blood pressure will be improved.

PHENYLEPHRINE

Phenylephrine is an alpha1 adrenergic receptor agonist which binds primarily to α-receptors and favours α1 receptors over α2 receptors. It is not a catechol derivative and therefore not a substrate for catechol O-methyltransferase (COMT). Phenylephrine is a vasoconstrictor which raises

both systolic and diastolic blood pressure, because it increases the vascular resistance (after load). It has no effect on the heart rate.

Note: By increasing systemic vascular resistance, phenyephrine may create enough afterload to decrease cardiac output and even induce pump failure. In addition, excessive vasoconstriction may intensify hydrostatic pressure and force fluid out of the vessels, creating a relative hypovolemia. Vasopressors can also divert blood flow away from organ beds, to further aggravate cellular hypoxia and perpetuate shock.

Dose:
40 – 100 mcg as a bolus dose
(Available from: www.healthhype.com)

ANTI-PLATELET DRUGS

Various drugs are used to maintain or restore circulation. There are 3 major groups: anti-coagulants, anti-platelet aggregators and thrombolytics.

Anticoagulants prevent the formation of blood clots.

Anti-platelet aggregators prevent the clumping of platelets, and thus preventing clot formation.

Thrombolytics attack and dissolve blood clots which have already formed.

Platelets do not usually stick together unless there is a break in the endothelial lining of a blood vessel. When platelets adhere to the broken surface of an endothelial lining, they synthesise thromboxane A2 which is a product of prostaglandins and a potent stimulus for platelet aggregation. The platelet receptor protein that binds fibrinogen, known as glycoprotein IIb/IIIa, also promotes platelet aggregation. Thromboxane A2 and adenosine diphosphate (ADP) increase the activation of this receptor (LeFever: 2009).

ANTI-COAGULANTS

HEPARIN

Unfractionated Heparin (UFH) combines with anti-thrombin III, which accelerates the anticoagulant cascade of reactions that prevents thrombus formation. By inhibiting the action of thrombin, conversion of fibrinogen to fibrin does not occur and the formation of a fibrin clot is prevented. Heparin is usually given as a bolus dose before commencement of any percutaneous intervention.

Standard heparin doses: 2500 - 10 000 IU, or weight adjusted heparin doses: 50 - 100 IU/kg. Continuous infusion is sometimes used if longer anticoagulation is needed.

Antagonist:
Protamine sulphate: 1 mg/100 U of Heparin

Low-Molecular-Weight Heparin (LMWH) is a derivative of standard heparin. The low-molecular-weight fractions are extracted through depolymerisation, enabling production of an equivalent of anticoagulation with a lower risk of bleeding. LMWH inactivates factor Xa, but it is less able to inactivate thrombin. Commonly used LMWH are: **Clexane** (Enoxaparin) and **Fragmin** (Dalteparin), Fraxiparine (Nadroparin). The half-life of LMWH is 2 – 4 times longer than that of heparin.

Common side-effects:

- Heparin induced thrombocytopenia
- Bleeding tendencies: Retroperitoneal bleeding,haematomas, epistaxis, haematuria
- Intra-spinal haematoma if used with the presence of an epidural catheter in situ

Antagonist:

None available. It is not effective to reverse the effects of LMWH.

Other Parenteral Anticoagulants:

Fondaparinux (Arixtra): It is a synthetic and specific inhibitor of activated Factor X (Xa). Fondaparinux can be used for the management of acute coronary syndromes. A single, once- daily 2.5 mg subcutaneous dose is used in unstable angina and NSTEMI patients. In patients with STEMI, 2.5 mg IVI is used for the first day followed by 2.5 mg s.c (subcutaneously) daily. There is an increased risk for catheter thrombosis in patients with ACS undergoing PCI soley treated with fondaparinux. It should, therefore, not be used if primary PCI is the planned reperfusion strategy in patients with STEMI. If patients should undergo subsequent PCI while treated with fondaparinux, UFH (Heparin) at standard dosage of 60-80 IU/kg must be given. Maintain the activated clotting time (ACT) at 250-300seconds.

Antagonist:

none available

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ORAL ANTI-COAGULANTS:

Warfarin inhibits the hepatic synthesis of vitamin K derived clotting factors: II, VII, IX & X. It needs regular monitoring with INR testing to adjust the dosage for the desired level of anticoagulation required.

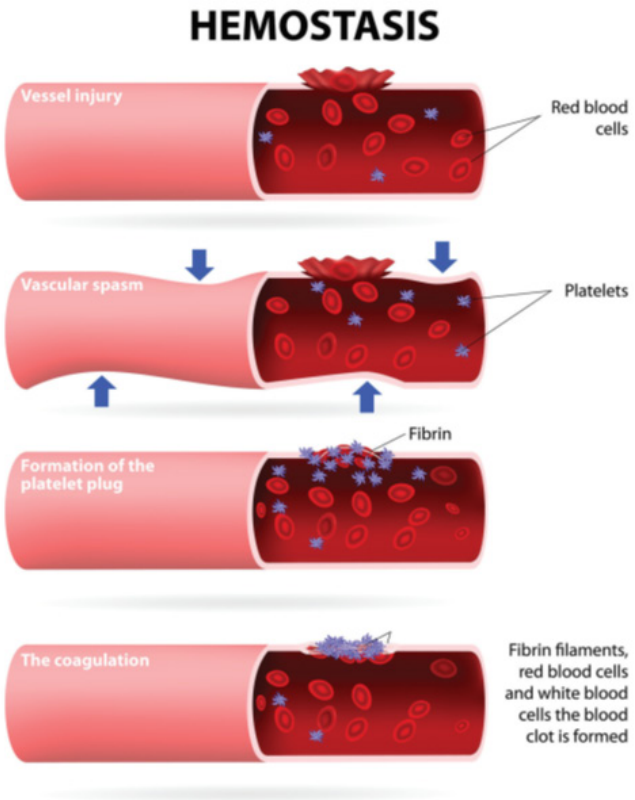

Figure 4.6: Primary and secondary hemostasis

Antagonist:
Vitamin K1 (Phytonadione)

Drug interactions with oral preparates:
Aspirin, NSAIDS, phenytoin, cimetidine, allopurinol and oral hypoglycaemic drugs enhance the effect of oralanti-coagulants.

New or Novel oral anti-coagulants (NOAC)

- Dibigitran dexilate (Pradaxa): an oral potent, competitive, reversible direct thrombin inhibitor.
- Rivaroxaban (Xarelto): an oral synthetic specific inhibitor of activated Factor X (Xa).

Dibigitranran dexilate is currently available in RSA and registration of Rivaroxaban and apixaban are imminent.

ANTI-PLATELET AGGREGATORS ASA (Acetylsalicylic acid)

Aspirin prevents platelet aggregation by inhibiting cyclo-oxygenase, an enzyme needed by platelets to synthesise thromboxane A2. The inhibition of platelet function lasts for the life time of the platelet(5 – 7 days).

Dosing of Aspirin:
81 mg, 100 mg, 150 mg and 300 mg

Common side-effects:

- GI disturbances: gastritis, haematemesis, abdominal pain
- Bleeding tendencies: ecchymosis, petechiae, purpura
- Suppression of red bone marrow
- Toxicity

Examples:
Disprin, Ecotrin, Cardio-Asprin, Disprin CV.

ADP-INHIBITORS

Adenosine diphosphate is a potent aggregating agent released by platelets to cause aggregation when in the process of forming a thrombus. If ADP inhibitors are administered, aggregation is thus inhibited for the life time of the platelet, by blocking the binding of ADP to the platelet ADP-receptor.

Examples: **Clopidogrel** (Plavix/Clopiwin, Prsugrel Effient)

Common side-effects:

- Bleeding
- Thrombocytopenia
- Gastrointestinal disturbances
- Skin reactions

GP IIb/IIIa INHIBITORS

Glycoprotein is a platelet surface receptor that is responsible for the attraction and clumping of platelets. By inhibiting the surface glycoprotein, clumping of platelets are inhibited.

TIROFOBAN HYDROCHLORIDE (AGGRASTET)

Tirofiban hydrochloride (Aggrastat) is a non-peptidal antagonist of the GP IIb/IIIa receptor, an important platelet surface receptor involved in platelet aggregation. Tirofiban hydrochloride prevents fibrinogen from binding to the GP IIb/IIIa receptor, thus blocking platelet aggregation. Tirofiban hydrochloride leads to inhibition of platelet function, by its ability to inhibit ADP-induced platelet aggregation and to prolong bleeding time (BT). Platelet function returns to baseline within eight hours after discontinuation. (Contra-indications and side-effects similar for Aggrastet and Integrilin)

EPTIFIBATIDE (Integrilin)

Eptifibatide, a synthetic cyclic heptapeptide, is an inhibitor of platelet aggregation and belongs to the class of RGD (arginine-glycine-aspartate)-mimetics.

Eptifibatide reversibly inhibits platelet aggregation by preventing the binding of fibrinogen, von Willebrand factor and other adhesive ligands to the glycoprotein (GP) IIb/IIIa receptors.

Contra-indications:

- Evidence of gastrointestinal bleeding, gross genito-urinary bleeding or other active abnormal bleeding within the previous 30 days of treatment
- History of stroke within 30 days or any history of haemorrhagic stroke
- Known history of intracranial disease (neoplasm, arteriovenous malformation, aneurysm)
- Major surgery or severe trauma within the past 6 weeks
- A history of bleeding diathesis
- Thrombocytopenia
- Severe hypertension (SBP > 200 mmHg and DBP > 100 mmHg)
- Severe renal impairment or patients on dialysis
- Clinically significant hepatic impairment

Side-effects:

- Retroperitoneal bleeding
- ICH
- Epistaxis
- Pulmonary bleeding
- Gum bleeds
- Allergic reactions
- Nausea
- Haematuria
- Fever
- Puncture site bleeding
- Headache

REOPRO

ReoPro is the Fab fragment of the chimeric monoclonal antibody 7E3. It is directed against the glycoprotein (GP) IIb/IIIa (αIIbß3) receptor located on the surface of human platelets. ReoPro inhibits platelet aggregation by preventing the binding of fibrinogen, von Willebrand factor, and other adhesive molecules to GPIIb/IIIa receptor sites on activated platelets. ReoPro also binds to the vitronectin receptor (αvß3) found on platelets and endothelial cells.

The vitronectin receptor mediates the pro-coagulant properties of platelets and proliferative properties of vessel wall endothelial and smooth muscle cells. Because of its dual specificity, ReoPro more effectively blocks the burst of thrombin generation that follows platelet activation than agents which inhibit GPIIb/IIIa alone.

Contra-indications:

- Active internal bleeding
- Recent (within 6 weeks) gastrointestinal (GI) or genitourinary (GU) bleeding of clinical significance
- History of cerebrovascular accident (CVA) within 2 years, or CVA with a significant residual neurological deficit
- Bleeding diathesis
- Administration of oral anticoagulants within 7 days unless prothrombin time ≤1.2 times control
- Thrombocytopenia (<100,000 cells/μℓ)
- Recent (within 6 weeks) major surgery or trauma

- Intracranial neoplasm, arteriovenous malformation, or aneurysm
- Severe uncontrolled hypertension
- Presumed or documented history of vasculitis
- Use of intravenous dextran before percutaneous coronary intervention, or intent to use it during intervention
- Known hypersensitivity to any component of this product or to murine proteins

Dose:

- IV bolus 0.25 mg/kg 10 to 60 minutes before PCI followed by IV infusion 0.125 mcg/kg/minute (max of 10 mcg/minute) for 12 hours

Reversibility Upon discontinuation of therapy with ReoPro, bleeding time returns to less than or equal to 12 minutes within 12 hours. Inhibitory effects of ReoPro can be rapidly reversed, at least in part, with platelet transfusions.

Preparation of bolus injection:

Withdraw the necessary amount of ReoPro for the bolus injection into a syringe. Filter the bolus injection using a sterile, non-pyrogenic, low protein-binding 0.2 µm/0.22 µm or 5.0 µm syringe filter. The bolus should be administered over one (1) minute.

Preparation of IV infusion:

Withdraw the necessary amount of ReoPro for the continuous infusion into a syringe. Inject into an appropriate container of sterile sodium chloride 9 mg/ml (0.9%) solution for injection or 5% glucose solution and infuse at the calculated rate via a continuous infusion pump. The continuous infusion should be filtered either upon admixture, using a sterile, non-pyrogenic, low protein-binding 0.2 µm/0.22 µm or 5.0 µm syringe filter, or upon administration, using an in-line, sterile, non-pyrogenic, low protein-binding 0.2 µm or 0.22 µm filter. Discard the unused portion at the end of the infusion period.

Side-effects:

- Bleeding
- Back pain
- Hypotension
- Nausea
- Chest pain
- Vomiting
- Headache
- Bradycardia
- Fever
- Puncture site pain
- Thrombocytopenia

THROMBOLYTICS

Thrombolytics are clot dissolution drugs, and act primarily by converting plasminogen to plasmin, a proteolytic enzyme which has the ability to break down fibrin, fibrinogen and other coagulating factors. All thrombolytics activate plasminogen, but they differ in terms of mechanisms of plasminogen activation and their fibrin specificity.

TENECTAPLASE (Metalase)

Tenecteplase is a recombinant fibrin-specific plasminogen activator that is derived from native t-PA. It binds to the fibrin component of the thrombus and selectively converts thrombus-bound plasminogen to plasmin, which degrades the fibrin matrix of the thrombus.

Administration:

Tenecteplase metalyse is given as a single, weight dependant bolus, via a peripheral line or a central line, followed by a 10 ml saline or water for injection flush.

ALTEPLASE (Actilyse)

Is known as tissue-type plasminogen activator. It rapidly activates plasminogen bound to fibrin. It has the advantage of lysing only fibrin, without unwanted degradation of other proteins, like fibrinogen. Once bound to fibrin, it is activated, inducing the conversion of plasminogen to plasmin and thus leading to dissolution of the fibrin clot.

Note:

At dose levels of Actilyse, circulating plasminogen may be activated, resulting in haemorrhage.

Patient weight	Dosage
< 60	30 mg (8000 U)
60 – 69	35 mg (8000 U)
70 – 79	40 mg (8000 U)
80 – 89	45 mg (10000 U)
≥ 90	50 mg (10 000 U)

Table 4.4: Dosing regimen for the administration of Metalyse

Administration:

3. 15 mg = 15 ml - over 1 – 2 minutes stat
4. 50 mg = 50 ml - over 30 minutes
5. 35 mg = 35 ml - over 60 minutes

Contra-indications and precautions:

The American Heart Association (2010: 38 highlights the following precautions:

- Active internal bleeding (except menses) within 21 days
- History of cerebrovascular, intracranial, or intraspinal event within 3 months (stroke, AV malformations, neoplasm, aneurysm, recent trauma, recent surgery)
- Major surgery or serious trauma within 14 days
- Aortic dissection
- Severe, uncontrolled hypertension
- Known bleeding disorders
- Prolonged CPR with evidence of thoracic trauma
- Lumbar puncture within 7 days

STREPTOKINASE

Streptokinase is an extracellular protein purified from culture broths of Group C β-haemolytic streptococci.

Streptokinase has no enzymic activity; instead it forms an active 1:1 complex with plasminogen, which then converts uncomplexed plasminogen to the active enzyme, plasmin. In addition to the hydrolysis of fibrin plugs, the complex also catalyzes the degradation of fibrinogen and clotting Factors V and VIII.

Take note:

Streptokinase is a foreign protein and is antigenic. Rashes, fever and anaphylaxis can occur. Since most individuals have had streptococcal infection sometime in their lives, circulating antibodies against streptokinase are likely to be present in most patients. These antibodies can combine with streptokinase and neutralize its fibrinolytic properties. Therefore, sufficient quantities of streptokinase must be administered to overwhelm the antibodies and provide a therapeutic concentration of plasmin. Streptokinase should not be administered between 5 days and a minimum of 2 years following initial treatment, as these antibodies may persist for up to 2 years.

Administration:

1. Reconstitute the whole vial of 1.5 MU in 200 ml Saline or Dextrose 5%

2. Infuse over one hour

Considerations:

- Perform a baseline prothrombin time (PTT) and INR.
- No IMI injections and try to keep venipuncture to a minimum for at least 48 hours post administration
- Monitor patient for any signs of haemorrhage (visible and occult)
- Monitor re-perfusion arrhythmias.
- Do an ECG: pre-, intra- and post-administration.
- In case of hypotension:
  - Stop infusion
  - Position head of bed down (supine)
  - Administer IV fluid challenge and increase IV-therapy
  - Once normotensive, continue streptokinase administration

(Available from: www. Home.intekom.com)

Weight (kg)	Total dose (mg)
40	60
45	67.5
50	75
55	82.5
60	90
64	96

Table 4.5: Weight adjusted dosage for patients <65 kg: 1.5 mg/kg

This Module is Linked to a CPD Accredited Online Questionaire at
www.sasci.co.za

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