Cigarette Smoking Effects

Cigarette smoke contains 4000 chemical agents (including Nicotine and CO) that exert a toxic effect on cardiorespiratory system. CO, Nicotine (Addictive), Free Radicals (Vessel wall damage), Radioactive cpds (Carcinogenic), Hydrogen Cyanide (Immobilises Ciliary blanket), Metals (Arsenic, Cadmium, Lead) (Carcinogenic)

Cigarette Smoking effects on CVS

Chemical agents in cigarette smoke cause an increase in oxidative stress, endothelial damage and dysfunction, and are associated with significantly higher serum concentrations of total cholesterol and triglycerides, and lower levels of the cardioprotective high-density lipoprotein.

1) By causing intravascular inflammation, smoking promotes the development of atherosclerosis and cardiovascular disease. (NB Free Radicals)

2) Nicotine deregulates cardiac autonomic function and boosts sympathetic activity. Promotes Ad and NA release from Adrenal Medulla

Increases heart rate (HR) at rest, while blunting HR elevation during progressive exercise and lowering the maximum HR that can be achieved.

BP is raised due to direct vasoconstriction

PHT due to Pulmonary Vasoconstriction

Increases Platelet stickiness ... and risk of clots.

Increased risk of Stroke

Vessel wall damage - Atherosclerosis

Cigarette Smoking effects on DO2

DO2 reduced
Smoking-generated CO binds with haemoglobin and myoglobin, reduces arterial O2 blood saturation, compromises the efficiency of respiratory enzymes, and causes dysfunction of the O2 production, transportation and delivery system, especially during exercise, substantially reducing the functional capacity and the performance of the circulatory system.

**Cigarette Smoking effects on RS that are relevant to conduct of anaesthesia**

- Increased /enhanced airway irritability to irritant volatile anaesthetics
- Increased bronchial secretions
- Reduced muco-ciliary activity
- Increases risk of Largospasm and Brobhospm Retained secretions causing collapse / consolidation
- Impairs tone of GOS ...increasing risk of reflux
- Chronic effects /pathophysiology:- COPD (Asthma/Bronchitis/Emphysema)

**Advice to 24 year old smoker 24 hrs preop under GA**

Stop at least 24 hours preoperatively (ideally should have stopped 8 weeks earlier). Reduces effects of Nicotine and CO on CVS and RS.

Consider Breathing Exercises.

Ensure any medications eg Bronchodilators / PPI /H2 Rec blockers are continued.

Offer Premedication if appropriate – to allay anxiety. Offer Nicotine patch to allay withdrawl?

Stop smoking permanently – Reduce risk of disease development
**Heparin and Protamine**

A 67 year old patient is to undergo CABG on CPB.

**a)** What dose of heparin is used to achieve full anticoagulation for CPB and how is it given. (2 marks)

Heparin 3-4mg/kg or 300-400 units /kg. Unfractionated. Intravenously slowly via central vein with appropriate timing and communication with surgeon prior to Aortic cannulation (allowing time for heparin to exert its anticoagulant action and be tested by POCT).

**b)** Which laboratory and point of care tests determine the effectiveness of heparin anticoagulation in CPB patients? Give the advantages and/or disadvantages of each test. (10 marks)

**Laboratory Tests**

Activate Partial Thomboplastin Time (APTT)

Normal Activate Partial Thomboplastin Time – test of intrinsic or Common Activation Pathway Normal value = 30secs. Systemic Heparinisation aimed at APTT ratio 2-3 :1

Less specific for Heparin Affected by deficiency of enzymes in Intrinsic pathway notably Factor 1,11,V,V111, 1X, X,X1 and X11 (not V11 and X111)

Prolonged APTT caused by....

- Heparin administration
- Antibodies vs coagulation factors (Inhibitors)
- Deficiency of coagulation Factors eg Haemophilia
- Antiphospholipid antibody (especially lupus anticoagulant which paradoxically predisposes to thrombosis)
- Sepsis – coagulation factor consumption

Thrombin Time (TT)
Normal = 12-14s Systemic Heparinisation aimed at ATTT ratio 2:1

Useful as normal in antiphospholipid syndrome.
Expensive

Anti-Factor Xa Assay

Therapeutic range with Heparin = 0.3-0.7iU/ml

Expensive

**Point of Care Tests**

1. **ACT**
   
   Aiming for ACT > 480 (Baseline pre Heparin ACT 100-130 secs)
   Rapid reliable POCT.
   Gives a figure which companies manufacturing CPB circuits recognise as appropriate for instituting CPB

   Inaccurate wrt Heparin levels in blood

2. **TEG**

   Dynamic test of viscoelastic properties of blood.

   Presence of Heparin r time in test is prolonged. By repeating test with Heparinase in tube one can calculate how much of the prolonged r time is due to the presence of Heparin.

   Useful for assessing any postop bleeding due to the release of heparin from fat stores where it gets sequestered during cpb.

**c) What are the causes of inadequate anticoagulation in a patient whom it is believed has already received heparin? (5 marks)**

1) **Heparin has not been given** (something else given ie drug error) or simple omission. In this case ACT will change little from baseline.

2) **Heparin Resistance.**
HR is defined as ‘failure of 500 units/kg heparin to prolong the ACT > 480’s.

Heparin requires Antithrombin for it’s action – as AT-Heparin has >1000 fold increased affinity for thrombin than AT without heparin. AT-heparin complex prevents thrombin mediated fibrinogen conversion to fibrin, platelet activation and release of tPA.

If AT levels are low then Heparin can exert its full effect on the clotting cascade and in these circumstances the ACT will usually rise a bit but not reach therapeutic values.

AT deficiency (and hence Heparin Resistance) can be Congenital or Acquired

**Congenital** 1:1000 may occur without prior heparin exposure.

**Acquired**

Occurs in patients who have previously had repeated exposure to heparin recently. The liver clears AT-Heparin complex and results in a low level of AT

Or

**Liver Failure (Cirrhosis)** – failure to synthesise AT

**Treatment**

FFP 2 units (as a source of AT) or synthetic AT +/- further Heparin dosing.

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**d) Describe the possible adverse reactions to Protamine (3 marks)**

**Common:**

Hypotension due to myocardial depression or vasodilation (mediated by Thromboxane) if Protamine given too rapidly. Usually transient and resolves – may need Intravenous fluid bolus or vasoconstriction.

Bradycardia
NB Caution with speed of injection

**Rare**

Anaphlactoid – more severe sustained hypotension secondary to histamine release
Anaphylaxis (IgE mediated and associated with an increased incidence of preexposure to Porcine insulin, fish allergy and vasectomy)

Pulmonary Hypertension, RV failure and systemic hypotension (mediated by Thromboxane). (Pulmonary Oedema)

Hypertension

Increased risk of bleeding

Miscellaneous (Nausea, Vomiting, Flushing, Temporary redness of face and neck)
**Cardioplegia**

a) Cardioplegia solutions

N.B. Plegia = paralysis

**Purposes:-**

Goal of myocardial protection with cardioplegia is to prevent myocardial injury during periods of intentional ischaemia that are required to perform heart operations.

Principles determinants of myocardial energy utilisation are LV End diastolic tension (LVEDP) and electrical activity.

Produce a still heart for the surgeon to operate on. There should be no electrical activity (Atrial or ventricle). Metabolic activity of heart should be reduced without losing high energy compounds (ATP).

Arrest the heart in Diastole (Low LVEDP, minimal myoc VO2 and energy substrate utilisation). The rapid onset of diastolic arrest at the onset of the ischaemic period following application of the Aortic Cross Clamp places heart in state of hibernation.

Provide a source of nutrients

Stabilise myocardial cell membrane

Prompt arrest of cardiac electromechanical activity

Combats intracellular ion losses

Buffers ischaemic acidosis

NB Lowest Myocardial VO2 = Arrested and Decompressed LV (via Venting Ao/ PA or Apex LV)
Typical Composition

K > 20 mM/l / Slightly hyperosmolar (reduce oedema), Alkaline (address pH changes) / Low Ca / Complimented by substrate / additives.

Crystalloid /Colloid /Blood based solutions (Blood cardioplegia Bl:Crystalloid 4:1)

Na 100-200mM/l

High concentration of Potassium 20-40mM/l – responsible for arresting the heart in diastole.

St Thomas Solution:-

Low Ca++ important in integrity of cell membrane and prevent Ca influx in reperfusion.

Magnesium – stabilises myocardium


Citrate

THAM (as buffer to prevent acidaemia)

Bicarbonate

Essential amino acids – Arginine and Glycine

Glutramate and Aspartate – maximise high energy phospahe production intend to be depleted – substrate enhanced cardioplegia

Lignocaine – as membrane stabiliser

FFA – as nutrients

Cardioplegia can be warm (body temperature known as a hot shot – enhances cellular assimilation of substrates and hastens myocardial contractility recovery) or cold (reduces myocardial temperature but reduces activity of Na\K pump)

Blood – prevents haemodilution from rpt crystalloid cardioplegia / maintains oncotic pressure / buffer / rheological benefits / free radical scavenging.

Other means of stilling heart ...

Inducing VF (Cross Clamp Fibrillation technique).

Myocardial Hypothermia - directly cooling myocardium reduces BMR

Hypothermia - Central hypothermia – reduces BMR of body and BMR of myocardium – reduces myocardial electrical activity.

b) Routes of administration

Infusion
- Injection into Aortic Root (and thence down coronary aa) – fails if AR (Antegrade)
- Injection directly into coronary aa – requires opening Aorta first – essential if severe AR (Antegrade)
- Injection into coronary sinus – achieves myocardial arrest distal to coronary aa occlusion. (Retrograde)

Fluid Blood / Crystalloid

Temperature: Warm or Cold

Administration: Continuous or Intermittent

c) Complications of cardioplegia solutions

- Failure if patient has significant AR – cardioplegia leaks back from Ao Root – into LV leading to LV dilation (and potential damage)
- Failure if coronary aa disease very severe – aided by retrograde cardioplegia
- Requires regular attention to repeat myocardial protection / cardioplegia application by surgeon. Poor myocardial protection is a cause of myocardial failure post CPB
- Damage to Aorta (dissection) coronary aa (eg dissection) or coronary sinus if cannula positioned poorly – detected by high line pressure and poor flow.
- Hyperkalaemia (Blood Potassium rises post injection of cardioplegia) – MI cardiac arrhythmias
- Ischaemic Injury / Reperfusion Injury.
Cardiac Tamponade

65 year old man underwent CABG earlier in day.

a) Which clinical signs suggest the development of acute cardiac tamponade? (40%)

Postoperative cardiac tamponade is cardiogenic shock due to compression of the heart by haematoma.

- Hypotension and Pulsus Paradoxus (> 10 fall in BP during Inspiration) (Respiratory swing on a-line, SBP decreases on inspiration if patient breathing spontaneously decreased on expiration if IPPV) – decreased SV and compression of heart. (During Inspiration Negative ITP > Increased RV pressure > IVS bulges to Left > Decreased filling of LV. Compression of RV > collapse of RV)
- Shock (Peripheral vasoconstriction cool and clammy, Tachycardia, SOB and decreasing conscious level)
- Raised JVP/ CVP – due to impaired venous return to heart.
- Muffled Heart Sounds – Heart compression
- ECG – low voltage / electrical alterans / EMD

Beck’s Triad = Low BP, Distended neck vv and distant muffled HS

- Cool peripheries (due to vasoconstriction and low cardiac output)
  Capillary refill >2 ses
- Oliguria
- Chest drainage – suggesting ongoing bleeding or if drains obstructed drainage may stop and there is then a real risk of blood accumulating within the chest and causing tamponade.

b) List the investigations and their associated derangements that could confirm the diagnosis of acute cardiac tamponade (15%)

Differential Diagnosis : Respiratory Tamponade / Tension Pneumothorax / Acute Heart Failure or Cardiogenic Shock

Arterial blood gas results – metabolic acidaemia
CXR – widening mediastinum / heart shadow +/- pleural effusion (haemothorax)

ECG – low voltage complexes, electrical alterans, EMD with end stage tamponade

FBC – falling Haemoglobin (WBC raised)

Chest Drainage – may be increasing blood loss but also drainage may discontinue if drains block and blood will then accumulate in the chest and compress heart.

Tranoesophageal or Transthoracic Echocardiography: Features suggest compression of heart chambers rather than diagnose tamponade ie should be interpreted in the light of clinical findings.

Evidence of Systolic compression of Atria (RA or LA) Diastolic collapse of Ventricle (RV or LV)

Echogenic mass compressing chamber suggests clot.

PWD study across TV or MV 20% variation in height of E wave suggests tamponade physiology

   c) What is the management of acute cardiac tamponade in this patient? (45%)

Check haemoglobin, platelet count and coagulation studies. Group and ensure cross match and blood available

TEG

Biochemistry (U&E’s)

Pericardiocentesis – unlikely to be effective as in this case of due to clot compression. Pericardiocentesis needle Xiphisternum towards Left shoulder. ECG control. ? Drain insertion

Pericardial Window – unlikely to be effective as in this case of due to clot compression. Window cut in Pericardium via mini-sternotomy or mini-thoracotomy. Drain blood through window.

Emergency Resternotomy – preferably in theatre (Occasionally necessary in ITU if end stage tamponade).

Theatre staff surgeon and anaesthetist need contacting. Anasethesia provided suitable for the management of a patient with cardiogenic shock (Fentanyl
Midazolam /Etomidate Suxamethonium / Atrarium) and patient induced in theatre with cardiac surgeon ready to perform emergency reopening.

Aline /CVC transduced ECG SO2 EtCO2 TOE aids diagnosis and confirms release of cardiac compression and assess need and response to any inotropic rugs.

Blood products administered in relation to blood results and TEG findings

Inotropic agents given via CVC – determined by BP/CVP/Echo findings

Complications if late detection.... Shock, MI, arrhythmia, rupture, SIRS and MOF, Wd infection)
Heart Transplant

A 56-year old man is listed for elective surgery. He received an orthotopic heart transplant 12 years earlier.

a) What alterations in cardiac physiology and function must be considered when planning general anaesthesia? (50%)

Transplanted heart is initially denervated (though with time some reinervation may occur)

Cardiac function is dependent on the Frank Starling mechanism, levels of circulating catecholamines and metabolites (lactate and H+)

Resting HR is usually slightly higher than normal and rises to a lesser degree on exercise or when stressed

Ischaemic injury to the SA Node or AV Node may produce severe bradycardia. 20% patients need a pacemaker.

Cardiac Allograft Vasculopathy is present in 30-50% patients at 5 years (CAV involves all vessels of transplanted heart (arteries and veins) leading to occlusions.

Planning Anaesthesia

1) Only direct acting agents eg Isoprenaline /Adrenaline may increase heart rate.
2) Anaesthesia should try to avoid agents that cause bradycardia.
3) Usual caution with presence of Pacemakers  (Pre anaesthetic review of function, report programme to Paced rate 90, use bipolar diathermy if operating near pacemaker, recheck pacemaker function postop)
4) Importance of maintaining Preload (Frank Starling mechanism) IV fluid load may be useful
5) Myocardial function may be depressed (due to CAV)
6) Monitor for ischaemia development (a risk due to CAV)

b) What are the implications of the patient’s immunosuppressive therapy in the perioperative period? (30%)
Cyclosporin, Azathioprine, Mycophenalate, Antilymphocyte globulin and Corticosteroids are most widely used immunosuppressants.

Importance of ctd immunosuppressive regimen to prevent rejection. Eg Cyclosporn blood levels must be maintained. Bioavailability oral vs iv is different. Expert help should be sought so as to avoid risk of low levels and increased risk of rejection, high levels and risk of renal damage.

SE of Imunosuppresants – Infection risk / Risk of Lymphoproliferative disorder / Melosuppression – anaemia, thrombocytopenia, leukopenia / GIT – haemorrhage / Renal – impaired tubular function, hypertension / Obesity

c) What long term health issues may occur in this type of patient?(20%)

Hypertension
CAV – impaired systolic function and IHD
Renal impairment in function
Increased risk of other tumours notably lymphoproliferative disorders.
SE of Immunosuppressants cf above
**Off Pump Surgery**

a) **What are the theoretical advantages of ‘off pump’ coronary artery bypass grafting (OPCAB) compared to ‘on bypass’ technique? (35%)**

**General:**
- Avoid CPB and Cannulation – avoid air and particulate emboli.
- Avoid Cannulation – and risk of Aortic damage (eg Ao Dissection)
- Avoid sternotomy if single vessel CABG (Avoid thoracotomy / LAST Limited short anterior thoracotomy feasible for single graft MIDCAB Minimally invasive direct coronary artery bypass graft)

**Myocardial:**
- Avoid cardioplegia (and events assoc with cardioplegisa)
  - Reduced release of markers of myocardial injury (ie troponins)
  - Reduced incidence of myocardial infarction and myocardial reperfusion Injury.
  (Potential for incomplete revascularisation / long term graft patency ?)

**Neurological:**
- No requirement for Ao cannulation. Hence reduce microemboli and evidence of reduced CVA in large retrospective studies.
  (NB Cognitive improvement not proven!)

**Renal:**
- Reduce risk of dialysis dependent renal failure in patients with pre-existing renal dysfunction.

**Pulmonary:**
- Ventilation and Perfusion maintained throughout (No significant improvement in postop pulmonary function)

**Haematological:**
- Less heparin administered (10,000 units versus 300-400units/kg)
  - Less bleeding postop and less requirement for blood products.
Immunological: Reduce SIR avoids increase in vascular permeability and TBW assoc with CPB

Cost: Cost saving – Reduce LOS in ITU, reduce disposable costs, reduce costs of blood products

b) What causes haemodynamic instability during OPCAB? (20%)

Most causes are due to the abnormal positioning of the heart – elevation and rotation plus surgical retraction

Reduction of venous return and impede LV and RV outflow (heart elevation and distortion impairs venous drainage into heart and outflow and ejection) RV and LV distortion (heart positioning) Ventricle septal displacement due to chamber compression Valve distortion and regurgitation (NB MR) Partial Ao clamping (side-biting clamp for proximal anastomosis) Conduction system ischaemia partic during RCA grafting → heart block

c) What strategies help to minimise this haemodynamic instability? (25%)

Careful monitoring: ECG for ischemia and HR Invasive arterial pressure and Et CO2 to detect fall in CO (? Some anaesthetists use continuous PAFC or ODM to give continuous CO measurements) TOE to detect subtle RWMA (NB ME views only as TG unobtainagle due to heart positioning) INVOS – cerebral perfusion???? Maintain preload (Fill the heart pre positioning) – avoid hypovolaemia Avoid excessive rises in CVP (Reduces CPP) Patient position Trendelenberg and lateral tilt (facilitates heart positioning) Beta block to maintain HR =80 (?Remifentanil / Esmolol infusion) Maintain perfusion pressure (Metaraminol . Inotropes) Surgery efficient and timely Careful positioning of heart by surgeon – check CVS effects when heart positioned Pharmacology – in addition to above Antidysrhythmics, Potassium, Magnesium, GTN , SNP. Use of IABP
Epicardial pacing if required for heart block
Conversion to CPB

d)  Outline the measures that help minimise perioperative hypothermia during OPCAB? (20%)

Maintain high ambient theatre temperature
Use of heated mattresses
Use of Sterile FCH blanket (after harvesting saphenous vein and skin closure) – with NP temperature monitoring
Humidification assoc with ventilation (HME filters)
Warm intravenous fluids
Use of insulating material to reduce heat loss from the patients head.
Hypotension and Cardiac Tamponade

List the causes of hypotension in the first 24 hours post cardiac surgery (6 marks)

What investigations help in the diagnosis and management of a patient with hypotension post cardiac surgery? (6 marks)

What are the features of Cardiac Tamponade (3 marks) Briefly outline ‘Tamponade physiology (4 marks)

Causes of Hypotension

Hypovolaemia due to Bleeding
Cardiac Tamponade
Myocardial Failure (LV or RV Failure or CABG failure)
Vasodilation due to Sepsis Syndrome

Tension Pneumothorax
Pumonary Embolus
Anaphylaxis

Investigations

Bloods – FBC Coagulation TEG U&E Cross match / Platelets / Clotting Factors ABG and s Lactate

Urine Output
CXR – widened mediastinum / pleural effusion / collection /pneumothorax

TOE –

Tamponade - = clinical diagnosis TOE show compression of chamber eg RV and RA most commonly (RV Diastolic collapse RA Systolic collapse) 20% respiratory variation in TransTricuspid or Transmitral doppler velocities

Myocardial Failures RWMA – hypokinesia / akinesia /dyskinesia Global LV or RV dilation or impairment in systolic function

Hypovoalemia FAC reduced LV (kissing papillary muscles) on TG SAX view appears empty

Sepsis Empty LV ESA and EDA reduced Good systolic contractility

TOE used to monitor therapy... Drainage of Cardiac Tamonade / Transfuion in Hypovolaemia / Inotropes in Myocardial Failure or IABP or redo CABG / PCI in Cath Lab

Features of Tamponade
Bleeding – Chest Drainage slows or stops (CVP increases BP decreases)
Clinical Diagnosis Hypotension, Tachycardia Raised JVP (CVP) + Muffled HS = Beck’s Triad
Pulsus Paradoxus
(Equailsation of RAP, PAD and PAWP)
Low voltage ECG (or Electrical Alterans)

Associated with .... Oligo-anuria
Increasing acidaemia (Lactate and Base excess decrease)
Respiratory variation in SBP (Respiratory swing on trace) and CVP

Tamponade Physiology

In patient breathing spontaneously

SBP reduced on Inspiration (Pulsus Paradoxus) – Inspiration associated with an increase in Intrathoracic pressure reduces venous return and SV This is exaggerated in heart compressed by encasement with blood.

SBP increases on expiration

This variation is seen as a marked >20% variation in SBP with respiration

(These changes can be seen in the Doppler signals measured across the Mitral Valve ie E and A decrease on inspiration and increase on expiration. Opposite changes occur across the Tricuspid Valve)

If patient is on a ventilator the opposite occurs ie E and A increase in inspiration and decrease in expiration across the Mitral Valve and the opposite occurs across the TV)
Aortic Dissection

What is the Stanford Classification of Aortic Dissections. What is the clinical management of a patient presenting with Aortic Dissection? (8 marks)

How does Transoesophageal echocardiography help in the diagnosis and management of a patient with an Aortic Dissection? (6 marks)

What are the complications of operative repair of Aortic Dissection? (6 marks)

**Stanford Classification**

Type A Dissection entrance (dissection flap) is proximal to origin of Left Sublavian a from Aorta. Hence Type A always involves Ascending Aorta

Type B Dissection entrance (dissection flap) is distal to the origin of Left Subclavian a from Aorta. Type B involves only Descending Aorta.

Type A Dissection

90% mortality when managed conservatively 50% mortality when managed surgically. Hence Type A Ao Dissections represent a surgical emergency.

**Initial management...**

1) Clinical Diagnosis Pain which is often sudden in onset tearing in nature and located retrosternally and radiating to the back interscapular region. Hemiparesis may occur if CA involved in dissecting flap. Shock from Tamponade Iscahemia if coronary aa involved LVF/Shock if severe AR (Syncope, Dyspnoea, Pulse deficits, Murmur of AR)

2) Diagnosis and conformation of complications CT Scan + TOE

CT demonstrate the extent of the Dissection and involvement of other organs.
Not so god at detecting entry site (Dissection Flap). 90% sensitivity 85% specificity
TOE more invasive in a patient who might be unstable cardiovascularly but good for detecting flap and complications.
(MRI highest sens and spec but diff in availability and monitoring sick patients.
Angiography less often used as less invasive procedures popular)

3) Manage Pain which is often a severe tearing nature going through to the back – Potent Opiates. Important differential from Acute Ischaemic Pain (ACS) NB Former requires Dual antiplatelets and Heparin which could be disastrous for a patient with an Ao Dissection

4) Control BP Labetalol + GTN infusions monitor with invasive arterial monitoring

5) Preparation FBC Coag Gp and X match = 6 units U&E ECG (Ischaemia or low voltage ECG) CXR (wide mediastinum or effusion)

6) Discuss with CT Centre and Transfer for intervention.

7) Consent
8) Theatre – Surgical Repair involves repairing the entry point of the Dissection Flap (and prevent death by proximal migration of Dissection) which usually means inserting an As Ao Interposition Dacron graft. The native AoV may be resuspended in the graft. If AR is present a new AVR may need inserting and also the coronary aa may need re-implanting if the Sinuses are damaged or CABG. The Distal anastomosis of the graft to the Aorta may require DHCA to facilitate.

9) Anaesthesia – in OT. 14g cannula + A line + CVC prior to induction. Cardiostable induction consider RSI need. A line both Radial aa. SLT. (If Arch of Aorta transgressed then Femoral aa monitored). Nasopharyngeal and Bladder Temp monitor if DHCA

10) Operation major bleeding transfusion blood products. TEG monitoring is useful

11) Postop ITU care

Type B Ao Dissection Initial Survival 90% with conservative Tx and Operative Tx is assoc with an increase in serious morbidity (Spinal Cord Ischaemia)

Clinical diagnosis / Invx CT Scan / Control BP / Refer to Interventional Radiology fopr consideration of Thoracic stent.

How does TOE help in diagnosis and management of Ao Dissection

1. Diagnosis – Excellent sensitivity (95%) and specificity (94%) for detecting Dissection Flap in Asc Ao most often at STJ.
2. Identify False and True lumens
3. Detect Aortic Valve Dissuption AR and assess severity and mechanism
4. Detect Cardiac compression – Tamponade physiology RV RA compression Resp variation in TM Doppler
5. Detect RWMA if coronary aa involvement
6. Track Ao Dissection in Arch and Desc Ao
7. Postop assess AoV resuspension / New AVR / Assess LV function / Filling of LV and RV

What are the complications of Aortic Dissection

1. Tamponade
2. Ischaemia
3. Heart Failure assoc with AR
4. Cardiogenic Shock and Cardiac arrest
5. CVA
6. Major Haemorrhage
7. Renal Failure
8. Mysenteric Infarction
9. Global Cerebral Hypoxia with DHCA (Incidence reduced due to DHCA management and in particular use of Antegrade Cerebral Pefusion
11. RV dysfunction postop due to poor myoc preservation and warmth under lights
12. Prolonged ITU stay with associated complications
13. Postoperative chest infection
14. Organ Dysfunction due to malperfusion from false lumen