CHAPTER

7

Cardiac Anesthesia

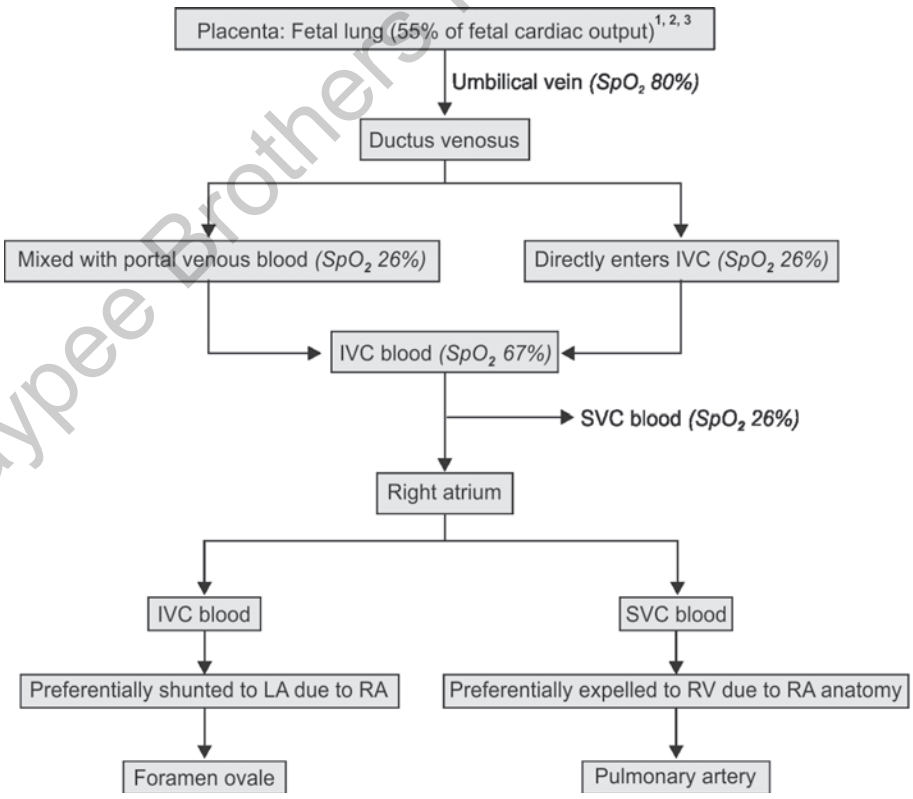
PHYSIOLOGY OF FETAL CIRCULATION[1,2,3,7]

Introduction

Fetal pulmonary and systemic circulation are essentially in parallel instead of in series as in adults due to two anatomical shunts, the ductus arteriosus and foramen ovale.

Fetal Circulation

- Fetal pulmonary circulation is essentially a high resistance circuit
- This is due to collapsed nature of the lungs in-utero
- Pulmonary capillaries are formed only after 24–25 weeks of gestation
- These later come to lie close to an immature alveolar epithelium.



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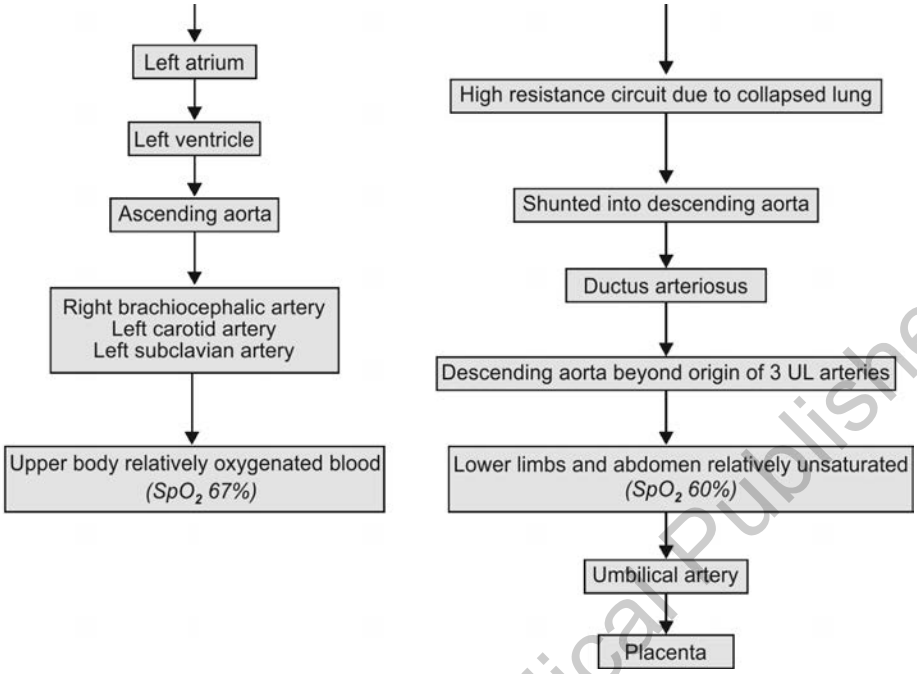

Changes Occurring at Birth

- The major cardiopulmonary adaptations at birth are:
  - Recovery from births asphyxia
  - Reflex initiation of breathing and expansion of lungs
  - Establishment and maintenance of adequate spontaneous ventilation
  - Conversion of fetal parallel circulation into series adult circulation.
- Closure of ductus arteriosus:
  - Occurs mainly due to reduction in pulmonary vascular resistance
  - This causes pulmonary vascular resistance to equalize SVR
  - Thus, blood flow decreases through ductus arteriosus
  - Eventually, ductus arteriosus closes
  - Physiological closure occurs at 10–15 hrs of life
  - Anatomical closure occurs at 2–4 weeks of life
  - Chemical mediators which aid in DA closure:
    - Prostaglandins
    - Bradykinin
    - Acetylcholine.
  - Factors causing reduced pulmonary vascular resistance:
    - Clamping of umbilical cord:
      - This separates fetus from placenta (fetal lung)
      - Thus, source of oxygenated blood is removed
      - This causes fetus to become increasingly asphyxia
      - Finally fetus gasps several times
      - Respiratory efforts initiated at 30 seconds
      - Efforts become sustained at 90 seconds.
    - ***Vaginal squeeze***:
      - Outward recoil of chest occurs as it passes through narrow vaginal vault
      - This makes intrapleural pressure negative (–30 to –50 mm Hg)
      - This causes expansion of lung
      - This factor is absent in cesarean section causing TTN.
- Closure of foramen ovale:
  - At birth, systemic vascular resistance increases
  - This causes an increase in LA pressure which causes closure of foramen ovale
  - Functional closure of foramen ovale occurs soon after birth
  - Anatomical closure occurs between 3 months to 1 year of life
  - Factors causing raised SVR:
    - Clamping of cord:
      - Causes increased umbilical artery pressure which raises SVR

    - Pushes around 100 ml of blood from placental circulation into systemic circulation (placental transfusion).
  - Increased LA pressure:
    - Increased pulmonary blood flow occurs due to reduction in PVR
    - This increases pulmonary venous return into left heart
    - Thus LA pressure increases, closing foramen ovale .
  - Vaginal squeeze:
    - Around 20–30 ml/kg of plasma ultrafilterate is present in fetal lungs
    - At birth, chest wall is squeezed as it passes through vaginal vault
    - Thus, ultrafilterate gets pushed into systemic circulation due to vaginal squeeze (90 ml).
- Fate of ductus venosus:
  - Ductus venosus atrophies as no blood flows from umbilical vein once it is clamped
  - It then forms *ligamentum venosum* of the liver.
- Thus the two sites of right to left shunting are closed at birth
- This converts fetal parallel circulation into adult circulation in series.

Applied Physiology

- Failure to establish adequate alveolar ventilation at birth will cause failure to recover from birth asphyxia.
- Conditions causing this are:
  - Congenital diaphragmatic hernia
  - Meconium aspiration
  - Congenital heart diseases
  - Polycythemia, infections
  - Hypoxia, hypercarbia, acidosis, prolonged stress
  - Raised PVR:
    - Truncus arteriosus
    - Pulmonary atresia.
- Persistent pulmonary HTN of newborn (PPHN):
  - Oxygen demand of fetus is 7 ml/kg/min
  - This increases to 18 ml/kg/min at birth before settling to 6 ml/kg/min
  - This is double the oxygen demand of adults (3 ml/kg/min)
  - This can cause rapid desaturation and further pulmonary vasoconstriction
  - This may lead to failure of closure of DA
  - Also reduced pulmonary venous return can cause persistent FO
  - Thus right to left shunting persists
  - This can worsen hypoxemia, leading to PPHN.

CLASSIFICATION OF CHD[1,2,3,5]

- Lesions causing outflow tract obstruction:
  - LVOT obstruction:
    - Coarctation of aorta
    - Aortic stenosis.
  - *RVOT obstruction:* Pulmonary valve stenosis
- Lesions causing left-right shunting:
  - Ventricular septal defects (VSD)
  - Atrial septal defects (ASD)
  - Patent ductus arteriosus (PDA)
  - Endocardial cushion defects
  - Partial anomalous pulmonary venous return (PAPVR).
- Lesions causing right-left shunting:
  - With decreased pulmonary blood flow
    - Tetralogy of Fallot (TOF)
    - Pulmonary atresia
    - Tricuspid atresia.
  - With increased pulmonary blood flow:
    - Transposition of great arteries (TGA)
    - Truncus arteriosus
    - Total anomalous pulmonary venous return (TAPVR)
    - Single ventricle
    - Double outlet right ventricle (DORV)
    - Hypoplastic left heart syndrome.

CORONARY CIRCULATION[1,2,3,5]

Introduction

- The coronary arteries encircle the heart like a crown and hence it is called coronary circulation.
- Averages around 250 ml/min at rest (5% of cardiac output).

Components

- Main components of coronary circulation are:
  - Right coronary artery
  - Left coronary artery
- Origin of coronary arteries:
  - Originate from the coronary sinuses present on the proximal aorta
  - Arise from just beyond the cusps of aortic semilunar valves
  - These are the first branches of proximal aorta
- Branches of right coronary artery:
  - Right marginal artery
  - Posterior descending artery

- Branches of left coronary artery:
  - Left circumflex artery
  - Left anterior descending artery.
- *Left coronary artery* is called ***widow artery*** as blockade of this artery results in sudden death.

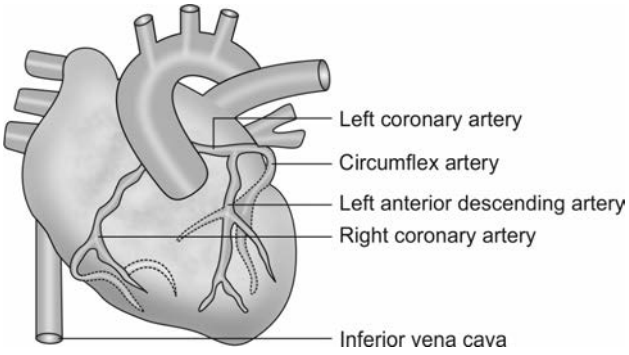

Fig. 7.1: Coronary circulation[2]

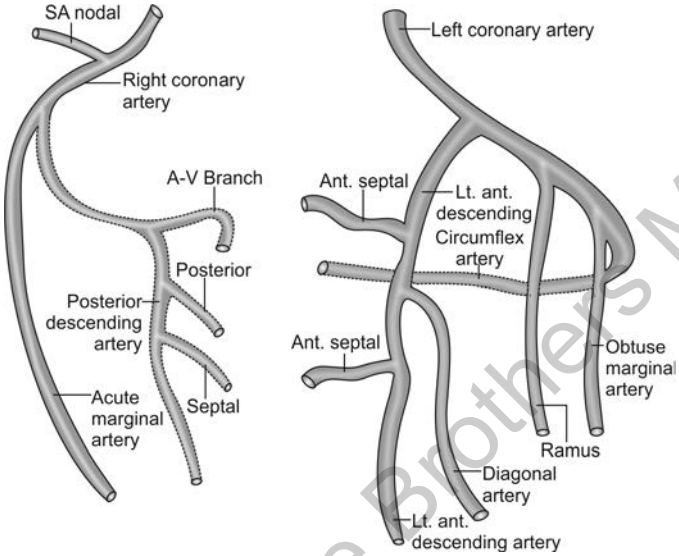

Fig. 7.2: Coronary circulation schematic[2]

Microcirculation

- The main coronary artery lies on the epicardial surface
- Branches of this penetrate the myocardium and supply the subendocardium with blood
- The diameter of these feeder branches is narrower and these are essentially end arteries.

Coronary artery	Part of myocardium supplied	Part of conduction system supplied
Right coronary artery		
Posterior descending artery	Right atrium	Sinoatrial node (60%)

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Coronary artery	Part of myocardium supplied	Part of conduction system supplied
Right marginal artery	Right ventricle	Atrioventricular node (85–90 %)
	Inferior LV surface (85%)	Proximal part of bundle of His
	Posterior LV surface (85%)	Part of posterior fascicle of LBB
Left coronary artery		
Left anterior descending artery	Anterior surface of LV	Most of RBB
	Part of lateral surface of LV	Anterior fascicle of LBB
	Most of interventricular septum	Part of posterior fascicle of LBB
Left circumflex artery	Left atrium	Sinoatrial node (40%)
	Part of lateral surface of LV	Atrioventricular node (10–15 %)
	Inferior surface of LV (15%)	
	Posterior surface of LV (15%)	

Cardiac Dominance

- The artery giving rise to posterior descending artery is called the dominant artery
- Most often, PDA arises from right coronary artery
- Therefore, most often coronary circulation is right dominant circulation
- In about 10% of hearts, RCA is small and does not give rise to PDA
- In these cases, LCX continues as PDA
- Such cases are called left dominant coronary circulation.

Coronary Veins

- Coronary veins travel alongside arteries
- They drain into coronary sinus or directly into right atrium via ***Thebasian veins***
- Coronary sinus:
  - This is largest vein draining the heart
  - Receives 85% of coronary blood flow
  - Opens into posterior wall of right atrium
  - Lies in the atrioventricular groove
  - Veins draining into coronary sinus:
    - *Great cardiac vein:* Lies along anterior interventricular groove
    - *Anterior cardiac vein:* Lies along RCA
    - *Middle cardiac vein:* Along posterior interventricular groove.

- Anterior cardiac veins also empty directly into RA via Thebasian veins which receive remaining 15% of coronary blood.

PHYSIOLOGY OF CORONARY CIRCULATION[1,2,3,5]

Introduction

- Unique as it is intermittent rather than continuous as in other organs
- During contraction, intramyocardial pressure in LV approaches systemic arterial pressure, almost completely occluding intramyocardial part of coronary artery.

Normal Perfusion Curves

- Left ventricle is perfused during diastole
- Right ventricle is perfused during systole and diastole
- Normal coronary flow = 250 ml/min at rest
- Coronary autoregulation occurs between perfusion pressures of 50–120 mm Hg.

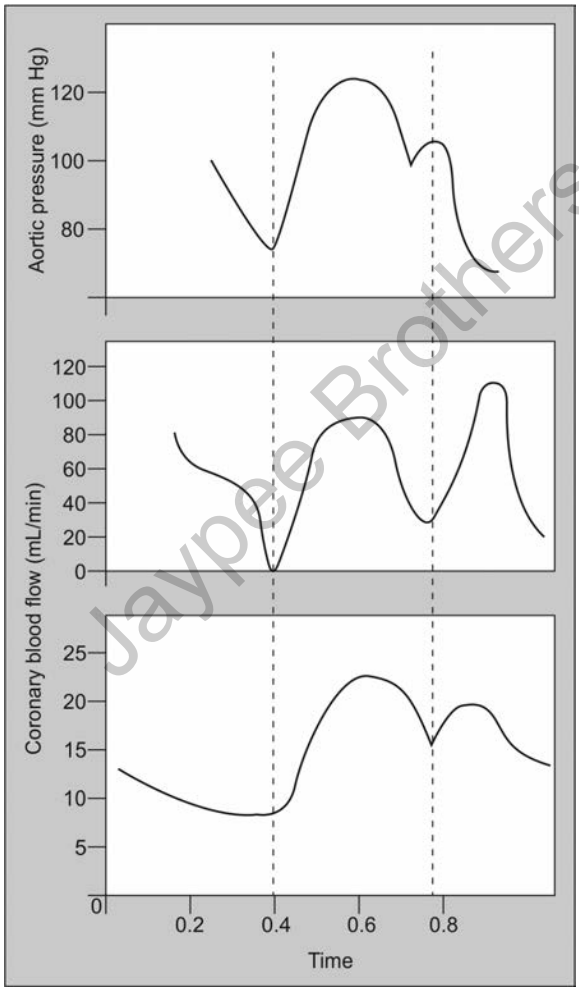


Fig. 7.3: Normal perfusion curves[2]

Determinants of Coronary Blood Flow

- Coronary perfusion pressure, coronary steal
- Myocardial metabolism, heart rate
- Myocardial extravascular compression
- Neurohumoral control:
  - Autonomic innervation
  - Coronary blood vessel diameter
  - Autoregulation.

Perfusion Pressure

- Coronary perfusion pressure = (Arterial diastolic BP) – (LVEDP)
- Arterial diastolic pressure is more important determinant of CBF than MAP.

Myocardial Metabolism

- Normal cardiac oxygen consumption = 80 – 100 ml/100 gm myocardium
- Flow-metabolism coupling
- Refers to link between myocardial metabolism and blood flow
- Factors which cause coupling:
  - Oxygen
  - Carbon dioxide
  - Nitric oxide
  - Adenosine
  - K_{ATP} channels.

Neural and Humoral Control

- Coronary innervation:
  - Sympathetic innervation:
    - From superior, middle and inferior cervical ganglia: Mainly stellate ganglion
    - Also from first four thoracic ganglia
  - Parasympathetic innervation by vagus nerve.
- β-adrenergic coronary dilation
- α-adrenergic coronary constriction
- Parasympathetic control of vagus.

Control of Coronary Blood Flow

- Autoregulation:
  - Refers to the tendency for organ blood flow to remain constant despite changes in arterial perfusion pressure
  - Occurs between MAP of 60–140 mm Hg
  - Occurs by metabolic coupling in blood vessels less than 150 μm
  - Critical mediator for autoregulation is oxygen, acting via adenosine.

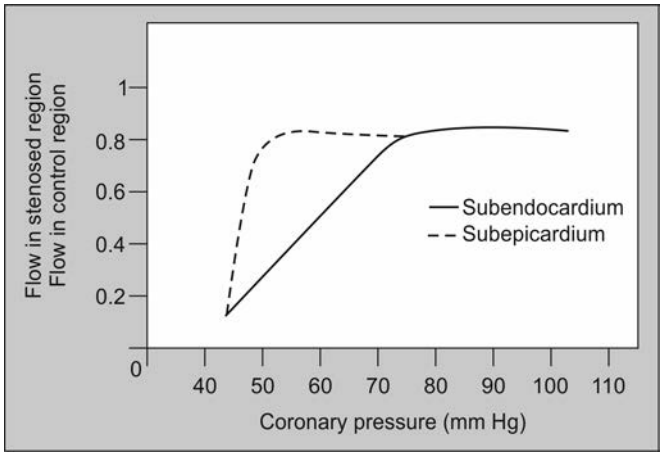


Fig. 7.4: Coronary autoregulation curve[2]

- Coronary reserve:
  - Intense vasodilatation occurs during MI, causing a large increase in blood flow
  - This is called reactive hyperemia
- Transmural blood flow.

Effects of Anesthetic Agents

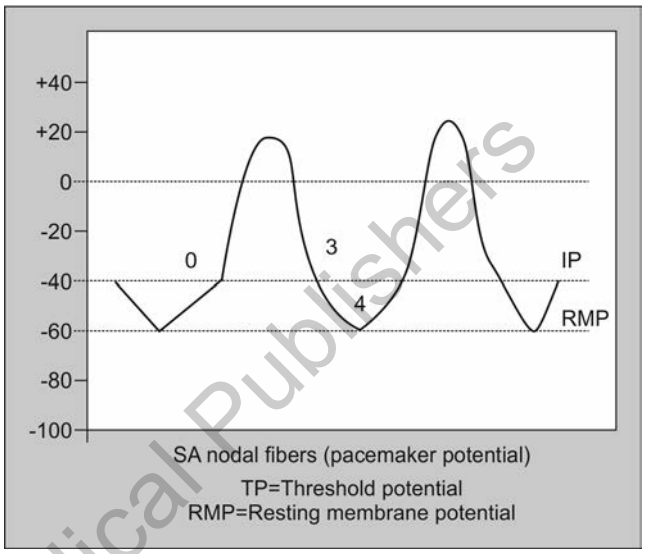
- Most volatile agents are coronary dilators through:
  - Action on ATP sensitive K^+ channels
  - Stimulation of adenosine A_1 receptors
- Halothane > isoflurane > desflurane > sevoflurane (negligible)
- Volatile agents protect against reperfusion injury
- They also reduce myocardial O_2 requirements during ischemia and infarction.

CARDIAC ACTION POTENTIAL[1,2,3,5]

Introduction

While action potential for skeletal muscle and nerve is due to abrupt opening of fast Na^+ channels, in cardiac muscle, it is due to opening of both fast Na^+ (spike) and slower Ca^{2+} channels (plateau).

Normal Action Potential



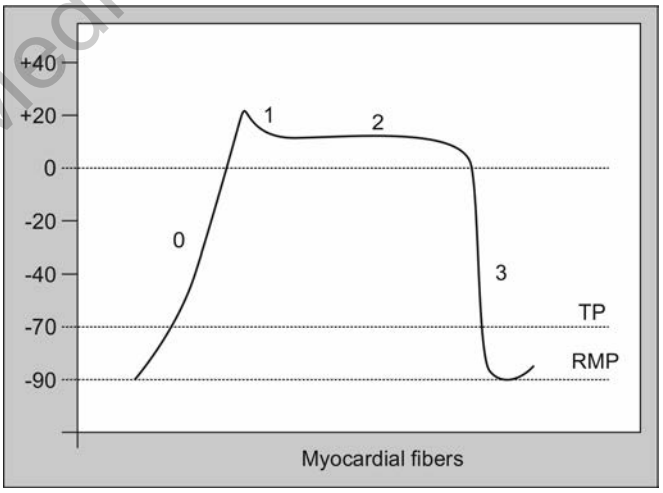


Fig. 7.5: Cardiac action potential[2]

Phase	Name	Event	Ion Movement
0	Upstroke	Opening of fast Na^+ channels	Na^+ comes into cell
1	Early rapid repolarization	Closure of Na^+ channels	K^+ goes out of cell
		Transient increase in K^+ permeability	
2	Plateau phase	Activation of slow Ca^{2+} channels	Ca^{2+} comes into cell
3	Final repolarization	Closure of Ca^{2+} channels	K^+ goes out of cell
		Increase permeability to K^+	
4	Resting potential	Normal permeability restored	K^+ goes out of cell
		Intrinsic slow leakage of Ca^{2+} into cells	Na^+ and Ca^{2+} enter cells

Events in Action Potential

Myocardial cells

↓

Normal ventricular resting membrane potential (–80 to –90 mV)

↓

Cell membrane potential is less negative

↓

Threshold potential reached (–70 mV)

↓

Action potential fired

↓

K^+ efflux once Ca^{2+} channels close

↓

Cell membrane potential becomes more negative

↓

Resting membrane potential

Pacemaker cells

↓

Normal sinoatrial resting membrane potential (–50 to –60 mV)

↓

Cell membrane potential less negative due to constant intracellular Na+ leakage

↓

Threshold potential reached (–40 mV)

↓

Action potential fired

↓

K^+ efflux, state of hyperpolarization

↓

Resting membrane potential

CARDIAC CYCLE[1,2,3,5]

Introduction

Sequence of electrical and mechanical events which occurs during course of a single heart beat.

Phases of Cardiac Cycle

- Atrial systole
- Ventricular systole:
  - Isovolumetric contraction
  - Rapid ejection phase
  - Slow ejection phase.

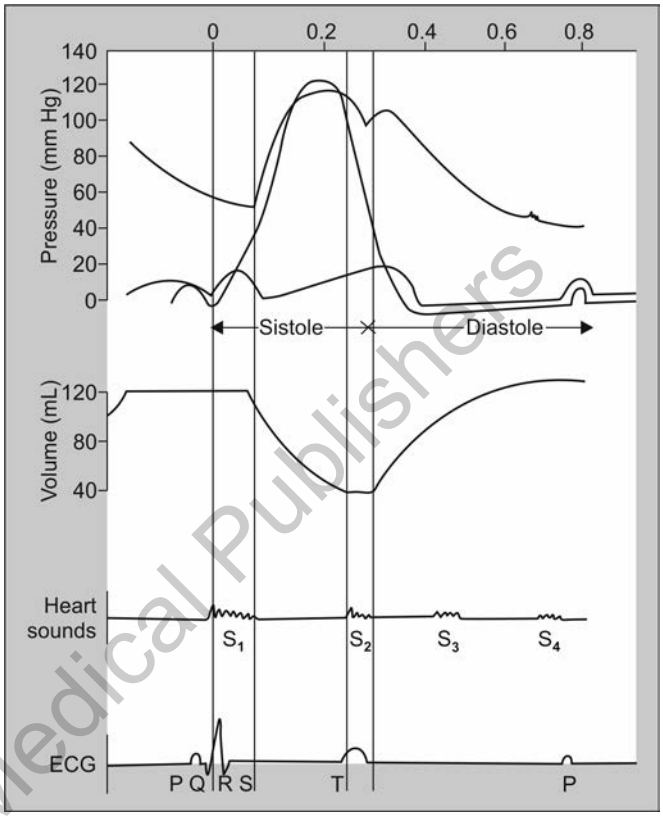


Fig. 7.6: Cardiac cycle[2]

- Ventricular diastole:
  - Isovolumetric relaxation
  - First rapid filling phase
  - Diastasis/slow filling phase
  - Second rapid filling phase.

Events in Cardiac Cycle

Atrial Systole

- Electrical event: P wave, PR interval
- Mechanical event:
  - Atrial systole/atrial kick
  - 25–30% cardiac output ejected
  - Fully completed before ventricle begins to contract.
- Pressure-volume event:
  - *a* waves in JVP
  - Atrial pressure increases.
- Heart sound event: S_4 produced, which is abnormal and seen in:
  - Massive pulmonary embolism
  - Cor pulmonale
  - Hypertrophic obstructive cardiomyopathy
  - Tricuspid regurgitation.

Isovolumetric Contraction

- Electrical event: QRS complex formed
- Mechanical event:
  - Atrioventricular valves close
  - Represents interval between closure of AV valves and opening of semilunar valves.
- Pressure volume event:
  - *c* wave in JVP
  - Contraction is isovolumetric, while intraventricular pressure increases.
- Heart sound event: S_1 produced due to closure of atrioventricular valves.

Rapid Ejection Phase

- Electrical event: Falls on QT segment
- Mechanical event: Semilunar valves open at the beginning of this phase
- Pressure volume event:
  - Opens up semilunar valve
  - Ejects blood, causing a sudden reduction in ventricular pressure
- *Heart sound event:* No event.

Slow Ventricular Ejection

- *Electrical event:* T wave due to repolarization
- *Mechanical event:* Aortic and pulmonary valves close at the end of this phase
- Pressure volume event:
  - Blood flow out of the ventricle reduces
  - Ventricular volume reduces more slowly
  - When ventricular pressure reduces below that in artery, semilunar valves open
- Heart sound event:
  - S_2 produced when semilunar valves close
  - Usually split S_2 as aortic valve closes before pulmonary valve.

Isovolumetric Relaxation

- *Electrical event:* No electrical deflection
- *Mechanical event:* Atrioventricular valves are closed, with closed semilunar valves
- Pressure volume event:
  - Atrial pressure increases, as blood collects passively
  - v waves occur in JVP
  - Ventricular pressure continues to fall
  - Ventricular volume is minimal.
- *Heart sound event:* S_2 occurs when semilunar valves close.

Rapid Filling Phase

- *Electrical event:* No events
- *Mechanical event:* Atrioventricular valves open
- Pressure volume event:
  - Blood from atria enters ventricles
  - Ventricular volume increases rapidly
- Heart sound event:
  - S_3 produced due to rapid passive filling of blood
  - Causes of S_3 gallop:
    - Myocardial infarction
    - Congestive cardiac failure
    - Hypertension.

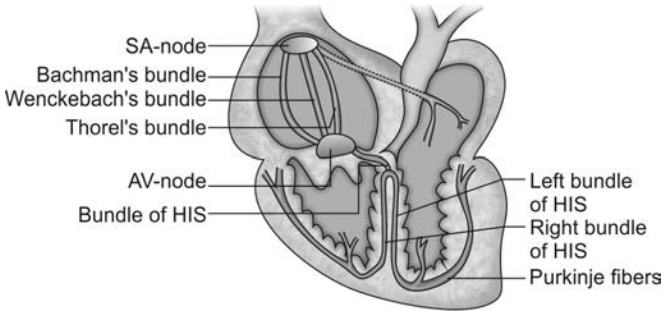
Diastasis

- *Electrical event:* No events
- *Mechanical event:* Rest of blood which has accumulated slowly flows into ventricles
- *Pressure volume event:* Ventricular volume increases more slowly
- *Heart sound event:* No events.

Second Rapid Filling Phase

- *Electrical event:* P wave, PR interval
- *Mechanical event:* Atrial systole/atrial kick
- Pressure-volume event:
  - *a* waves in JVP
  - Atrial pressure increases
- *Heart sound event:* S_4 produced.

CONDUCTION SYSTEM OF HEART[1,2,3,5]



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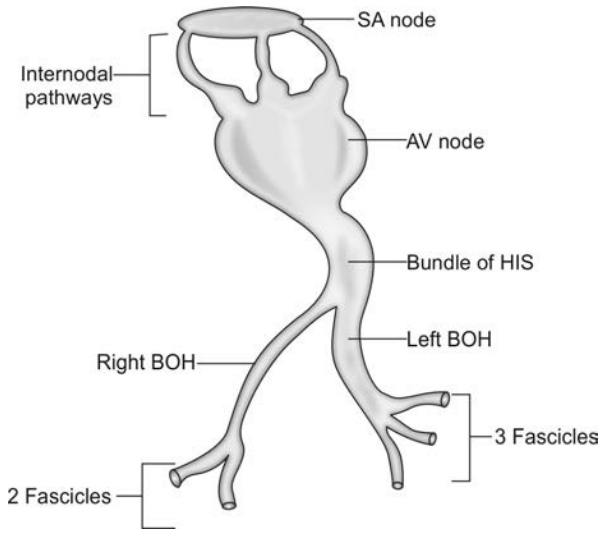


Fig. 7.7: Conduction system of heart[2]

Sinoatrial Node

- Location:
  - RA wall at SVC-RA junction
  - Located 1 mm below epicardial surface
  - ***Crista terminalis***
  - 50% cells have pacemakes activity
- Blood supply:
  - 60% RCA via SA nodal artery
  - 40% LCA
- Function:
  - Primary pacemaker of heart as it has fastest heart rate
  - Conduction to RA, interatrial septum, LA
- Intrinsic pacemaker activity:
  - 60–100 bpm
  - Called *sinus rhythm.*

Internodal Pathways

- Three pathways exist:
  - ***Bachmanns*** anterior bundle
  - ***Wenckebachs*** middle bundle
  - ***Thorels*** posterior bundle
- *Function:* Conducts impulses between SA node and AV node.

Atrioventricular Node

- Location:
  - Floor of RA
  - Situated just behind septal leaflet of tricuspid valve
  - Present near opening of coronary sinus.
- Anatomy:
  - Fibres are smaller with lesser gap junctions: Permits for AV nodal delay
  - Three functional regions:
    - Atrionodal region
    - Nodal region
    - Nodal-HIS regions.
  - AV delay occurs at Atrionodal and Nodal regions.
- Blood supply:
  - 90% RCA
  - 10% left circumflex artery.
- Function:
  - Receives impulses from SA node
  - Delays relay allowing atria to empty before ventricles contract.
- Intrinsic pacemaker activity:
  - 40–60 bpm
  - Called *junctional rhythm.*

Bundle of HIS

- *Location:* Upper part of interventricular septum
- Anatomy:
  - Divides into right and left Bundle of HIS
  - Right BOH has 3 fascicles
  - Left BOH has 3 fascicles:
    - Anterior fasciculus
    - Septal fasciculus
    - Posterior fasciculus
- *Blood supply:* Dual blood supply to left posterior fasciculus via LAD and PDA
- Function:
  - Receives impulses from AV node
  - Conducts them to Purkinje fibers
- *Intrinsic pacemaker activity:* 40–60 bpm.

Purkinje Fibers

- Location:
  - In ventricular myocardium
  - Penetrates one-third of ventricular myocardium.
- Function:
  - Fastest speed of conduction
  - Receives impulses from Bundle of HIS
  - Conducts them to ventricular myocardium.
- *Intrinsic pacemaker activity:* 20–40 bpm.

Variations

Pre-excitation Syndromes

- Terms used to describe rhythm that originate from above the ventricles but in which impulse travels via a pathway other than AVN and BOH.

- Types:
  - ***Wolff-Parkinson: White syndrome*** via ***bundle of Kent***
  - ***Lown-Ganong: Levine syndrome*** via ***James bundle***
  - ***Mahaim fibers.***

PRE-EXCITATION SYNDROME[1,2,3,5]

Introduction

Pre-excitation is a term used to describe rhythms that originate from above the ventricle but in which the impulse travels via a pathway other than the AV node and BOH.

Embryology

- During fetal life, strands of myocardial tissue form connection between the atria and ventricles, outside the normal conduction system
- These strands normally become nonfunctional soon after birth
- In patients with pre-excitation syndrome, these connections persist
- These strands form congenital malformations of working myocardial tissue.

Types of Malformations

- *Accessory pathways*: Bypass part or whole of the normal conduction system
- *Bypass tracts*: When one of the accessory pathway is attached to normal conductive tissue.

Examples: There are 3 major forms of pre-excitation syndromes:

- ***Wolff-Parkinson-White (WPW) syndrome***:

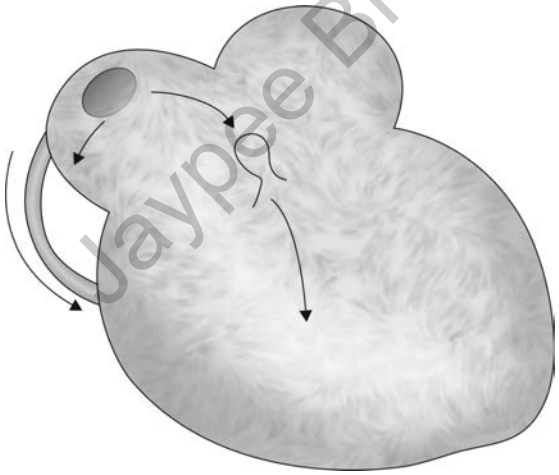


Fig. 7.8: WPW syndrome

  - The accessory pathway is called *Bundle of Kent*
  - This connects atria directly to ventricles, by passing the normal conduction system
  - Thus, the AV node is bypassed
  - Hence, AV nodal delay does not occur.
- ***Lown-Ganong–Levine syndrome:***
  - The accessory pathway is called *James bundle*
  - This connects atria directly to lower part of AV junction
  - Thus, it partially bypasses AV node.

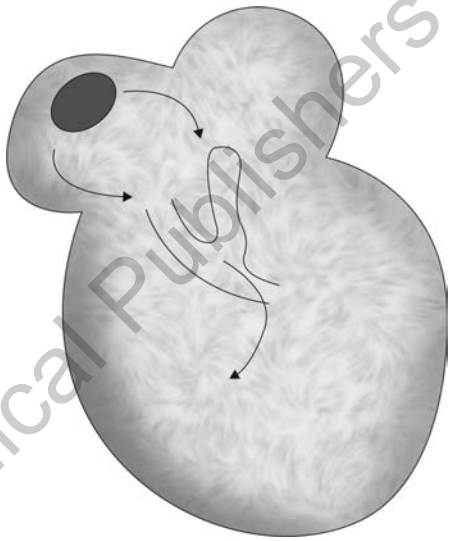


Fig. 7.9: LGL syndrome

- ***Mahaim fibers***:
  - These cause an unnamed pre-excitation syndrome
  - Fibers originate below AV node and insert into ventricular wall
  - This bypasses the ventricular conducting system.

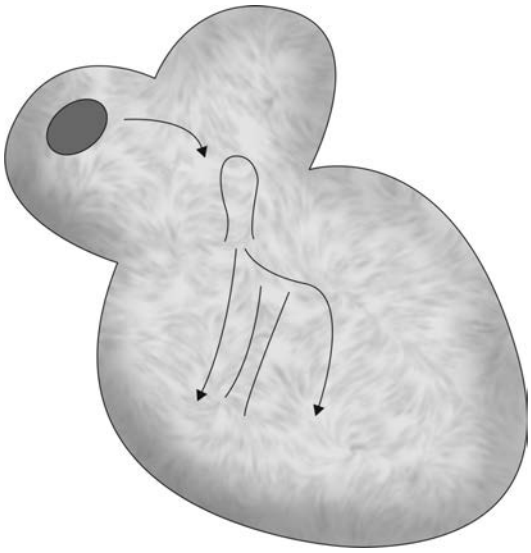


Fig. 7.10: Mahaim fibers

Incidence
- 1.5–3.1 per 1000 individuals
- More commonly occurs in men
- WPW syndrome is *most common* type of pre-excitation syndrome.

Description
- WPW syndrome
  - Delta waves
  - Short PR interval
  - Wide QRS duration (> 0.12 seconds)
- LGL syndrome:
  - No delta waves
  - Short PR interval
  - Normal QRS duration
- Mahaim fibers
  - Delta wave present
  - Normal PR interval
  - Widened QRS complexes.

Pathophysiology
- Patients with WPW are predisposed to tachyarrhythmias because of:
  - Accessory pathway bypassing the AV nodal delay
  - Pathway also provides a mechanism of re-entry
- Two types of re-entry are seen in WPW syndrome:
  - *Antidromic conduction*:
    - Rare variety
    - Occurs in an impulse generated from SAN
    - This travels via to ventricles via accessory pathway
    - Bypasses AV node on the way to ventricles
    - Impulse re-enters atria via AV node
  - *Orthodromic conduction*:
    - More common
    - Occurs in an impulse generated from SAN
    - This enters ventricles via AV node
    - Impulse re-enters atria via accessory pathway of Kent.

Clinical Features
- Palpitations, anxiety
- Light headedness, dizziness, weakness
- Chest discomfort, dyspnea, signs of shock
- *Complications:* Three main types of dysrrhythmias:
  - *AV nodal re-entrant tachycardia:* Most common
  - Atrial fibrillation
  - *Atrial flutter:* Least common.

Treatment
- Stable, symptomatic patient with orthodromic AVRT (narrow QRS):
  - Oxygen administration, IV access secured
  - Vagal maneuvers to convert rhythm
  - IV adenosine: May transiently increase risk of atrial fibrillation
  - IV calcium channel blockers/β blockers: Cause transient AV block
  - Avoid digoxin and verapamil as:
    - Conduction through AVN is slowed
    - This may speed up conduction through accessory pathway
- Stable, symptomatic patient with antidromic AVRT (wide QRS):
  - Oxygen administration
  - IV access secured
  - IV procainamide/amiodarone
  - Amiodarone preferred in CHF patients
- Unstable patients: Synchronized cardioversion.

CARDIAC OUTPUT[1,2,3,5]

Definition
Volume of blood ejected per minute from LV to aorta to support the metabolic demands of peripheral tissues.

Normal Values
- *Males:* 5–6 L/min
- *Females:* 10–20% less than males
- Children:
  - 350–400 ml/kg/min at birth
  - 150–200 ml/kg/min after first week of life
- *Cardiac index:* 3.2 L/min/m^2 (2.5–4.2 L/min/m^2)
- Body surface area calculated from nomogram based on weight and height.

Formula
- Cardiac output = stroke volume × heart rate
- Cardiac index = cardiac output/body surface area
- Stroke volume = (End diastolic volume) – (End systolic volume).

Factors Affecting Cardiac Output
- Factors increasing cardiac output:
  - Fever, raised BMR, exercise, pregnancy
  - Posture: Supine, lithotomy, trendelenburg
  - Anemia, beri-beri, hyperthyroidism
  - Arteriovenous fistula.

- Factors decreasing cardiac output:
  - Sleep, heart rate more than 120 bpm
  - Posture: Standing up, sitting
  - Reduced ventricular compliance due to:
    - Myocardial infarction
    - Valvular heart disease
    - Cardiac tamponade
    - Left ventricular hypertrophy
  - Reduced venous return due to:
    - Hemorrhage
    - Acute venodilatation, spinal anesthesia
    - Venous obstruction
    - Intermittent positive pressure ventilation.

Factors Affecting Stroke Volume

Preload

Definition

- Represents filling of heart chamber with blood during diastole
- Represents the muscle length prior to contraction or end diastolic fiber length
- Venous return is the amount of blood flowing from veins into RA each minute.

Formula

$$\text{Venous return} = \frac{\text{(Arterial pressure) – (RA pressure)}}{\text{Total peripheral resistance}}$$

- 55 to 60% of blood in body is in systemic veins
- Equivalent of EDV of LV in intact heart (normal around 120 ml).

Determinants of Preload

- Determinants of ventricular filling
- Venous return, blood volume
- Rhythm (artrial contraction)
- Heart rate
- Distribution of blood volume:
  - Posture
  - Intrathoracic pressure
  - Pericardial pressure
  - Venous tone (major determinant).
- Determinants of ventricular compliance:
  - Hypertrophy, asynchrony
  - Ischemia, fibrosis
  - Pericardial disease
  - Overdistension of contralateral ventricle
  - Increased pleural/airway pressure
  - Tumors, surgical compression.

Measurement of Preload

- Pulmonary artery pressure, PCWP
- CVP is poorest estimate of LV preload
- LVEDV via echocardiography
- LVEDP via cardiac catheterization.

Afterload

Definition

- Force which the heart must pump against, in ejecting blood from the heart
- SVR accounts for 95% of resistance to ejection.

Formula

$$\text{SVR} = \frac{\text{(MAP – RAP)}}{\text{CO}} \times 80$$

$$\text{PVR} = \frac{\text{(PAP – LAP)}}{\text{CO}} \times 80$$

- Normal SVR = 900–1500 dynes seconds/cm^{-5}
- Normal PVR = 150–250 dynes seconds/cm^{-5}
- SVR represents ratio of pressure to cardiac output.

Factors Affecting Afterload

- Viscosity and density of blood
- Aortic pressure, SVR
- Aortic valve area, vascular distensibility
- Volume and thickness of LV
- Systolic intraventricular pressure.

Contractility

Definition

- Myocardiums intrinsic ability to generate work from a given end diastolic fiber length
- Intrinsic force of myocardial contraction in inotropic state.

Factors Affecting

- Increased by:
  - Sympathetic activity
  - Calcium
  - Noradrenaline
  - Digoxin.
- Decreased by:
  - Parasympathetic activity:
  - Increased K^+ levels
  - Magnesium ions
  - Acidosis, hypoxia
  - β blockers, calcium channel blockers.

Measurement of Contractility

- Ejection fraction
- Isovolumetric contraction phase indices (d_p/d_t)
- Load dependent indices (slope of end systolic – pressure volume relation)
- Preload recruitable stroke work
- LV systolic wall thickening.

Heart Rate

- *Normal:* 60–80 bpm
- Formula:
  - Maximum heart rate = (220 – age in years)
  - Normal intrinsic heart rate = 118 bpm – (0.57 × age)
- *Factors increasing heart rate:* Exercise, fever, stress, high BMR
- *Factors decreasing heart rate:* Sleep, hypothermia, low BMR
- CO decreases progressively when heart rate ≥ 120 bpm, as diastolic filling time reduces.

Frank Starling's Law

Introduction

- Force of contraction of muscle is directly proportional to initial length of muscle fiber
- Represents the ability of heart to change its force of contraction and therefore stroke volume in response to changes in venous return.

Uses

- Illustrates relationship between CO and LVEDV
- Illustrates relationship between SV and RAP.

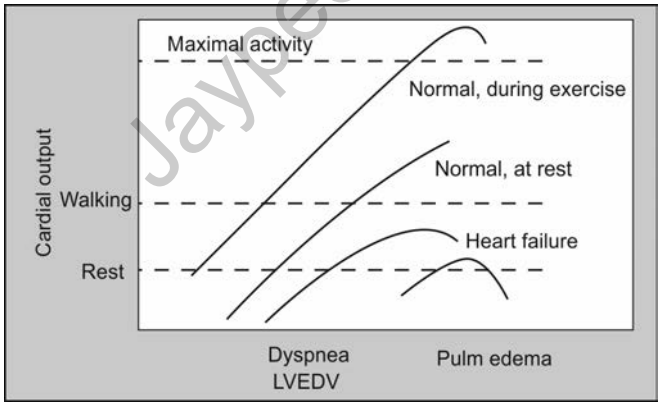


Fig. 7.11: Frank–Starlings law[3]

MYOCARDIAL OXYGEN SUPPLY DEMAND BALANCE[1,2,3,5]

Introduction

- Normal cardiac oxygen consumption = 80–100 ml O_2/100 g myocardium
- Normal oxygen supply = 250 ml/min
- Myocardium usually extracts 75–80% oxygen in arterial blood, compared with 25% in most other tissues
- Thus, it cannot compensate for a decrease in blood flow by extraction of more oxygen from hemoglobin
- This must be met with, by an increase in CBF.

Factors Affecting Supply

- Coronary blood vessel diameter
- Autonomic innervation
- Autoregulation
- Coronary steal
- Coronary perfusion pressure:
  - Aortic diastolic blood pressure
  - LVEDP/RVEDP
- Arterial oxygen content:
  - Arterial oxygen tension
  - Hemoglobin concentration
- *Heart rate:* Diastolic time
- Exogenous substances:
  - Oxygen, carbon dioxide
  - Nitric oxide
  - Adenosine
  - K^+, Ca^{2+}
  - pH, osmolarity.

Factors Affecting Demand

- Basal requirements (20%)
- Heart rate (most important)
- Wall tension:
  - Preload (ventricular radius)
  - Afterload
- Contractility.

Coronary Steal Phenomenon

- Occurs where perfusion pressure for a vasodilated vascular bed (in which flow is pressure dependent), is lowered by vasodilation in a parallel vascular bed; both beds being distal to a stenosis
- There is no evidence of coronary steal due to anesthetic agents in humans.

- Two types of coronary steal:
  a. Collateral steal
  b. Transmural steal.

Collateral Coronary Steal

- If arterioles of R_2 dilate, flow across R_1 to R_2 increases
- However, as R_3 arterioles are maximally dilated at rest, due to autoregulation, R_3 does not dilate anymore
- Hence flow across R_3 reduces.

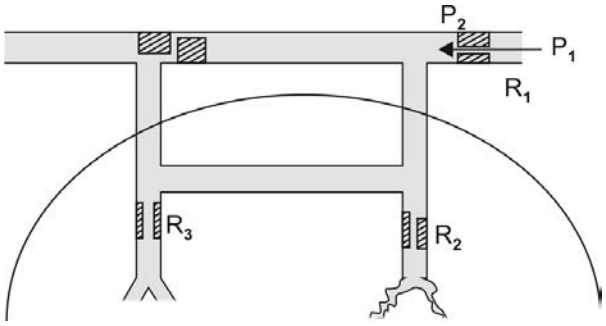


Fig. 7.12: Collateral coronary steal phenomenon

Transmural Coronary Steal

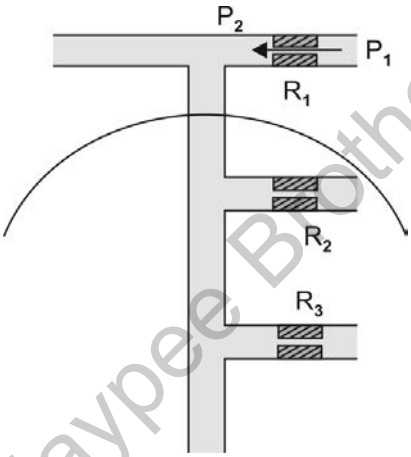


Fig. 7.13: Transmural coronary steal phenomenon

VENTRICULAR PRESSURE-VOLUME LOOPS[1,2,3,5]

Introduction

Aids to visualize changes in ventricular function in response to changes in preload, afterload and inotropy.

Method: LV pressure (LVP) is plotted against LV volume at multiple time points in the cardiac cycle.

Normal P-V Loop

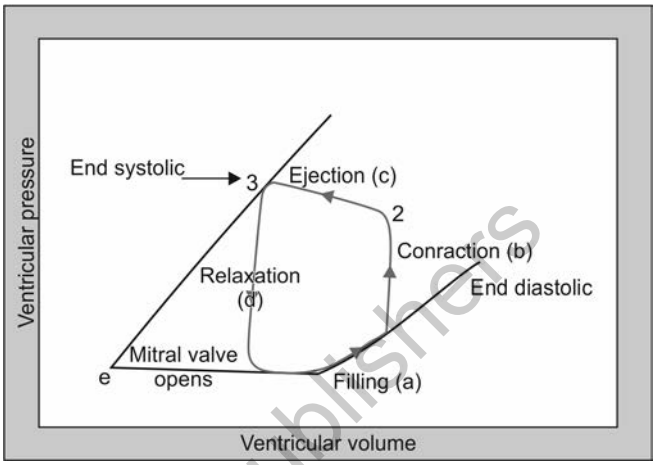


Fig. 7.14: Normal pressure-volume loop[2,3,5]

Point or phase	Event
Point 1	Mitral valve closes
Point 2	Aortic valve opens
Point 3	Aortic valve closes
Point 4	Mitral valve opens
Phase A	*Filling phase*
	Ventricles fill with blood
	LV volume increases
	LV pressure is constant at first and then slowly increases: Almost horizontal line
	Represents LVEDV and LVEDP
Phase B	*Isovolumetric contraction*
	Ventricular pressure increases
	Ventricular volume remains constant: Vertical line
Phase C	*Ejection phase*
	LV volume reduces
	LV pressure increases to a peak value called *peak systolic pressure*
Phase D	*Relaxation phase*
	LV volume remains constant initially due to isovolumetric relaxation
	When passive filling occurs, LV volume slowly increases
	LV pressure decreases during the entire process: Thus vertical line

Pressure Volume Loop in Mitral Stenosis

- Impaired LV filling occurs in mitral stenosis
- This reduces LVEDV which reduces stroke volume and CO
- This causes a low aortic pressure
- This results in a reduced width of curve (i.e. stroke volume) and low LVEDV.

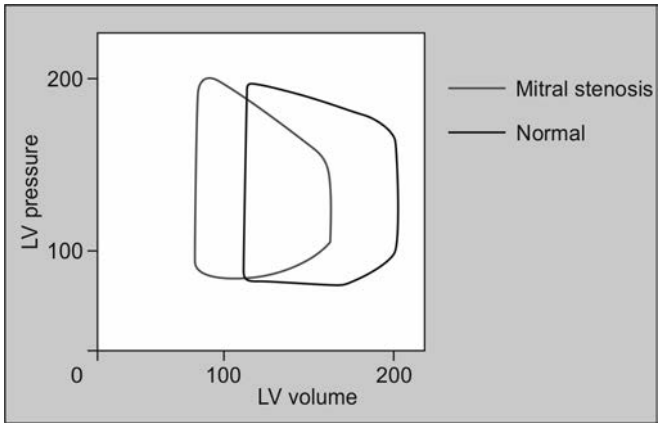


Fig. 7.15: Pressure volume loop in mitral stenosis[5]

Pressure Volume Loop in Mitral Regurgitation

- No true isovolumetric contraction phase present in MR, as blood flows back into LA during this phase
- Width of PV loop is increased (i.e. stroke volume is increased), as blood is ejected into aorta and also back into LA.

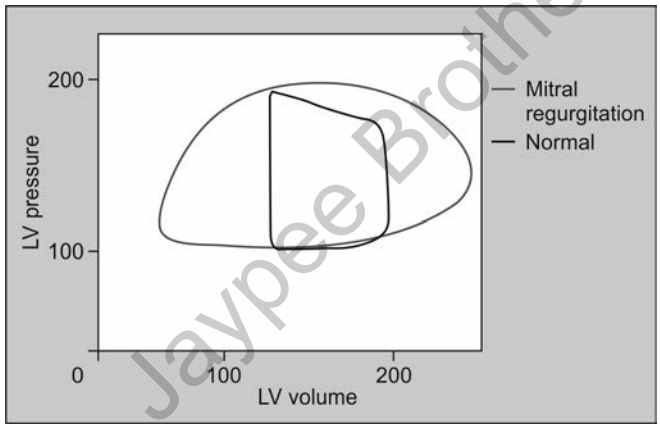


Fig. 7.16: Pressure volume loop in mitral regurgitation[5]

Pressure Volume Loop in Aortic Stenosis

- LV emptying is impaired causing a reduced stroke volume (small width of loop)
- High peak systolic pressures
- Raised end systolic volume and end diastolic volume.

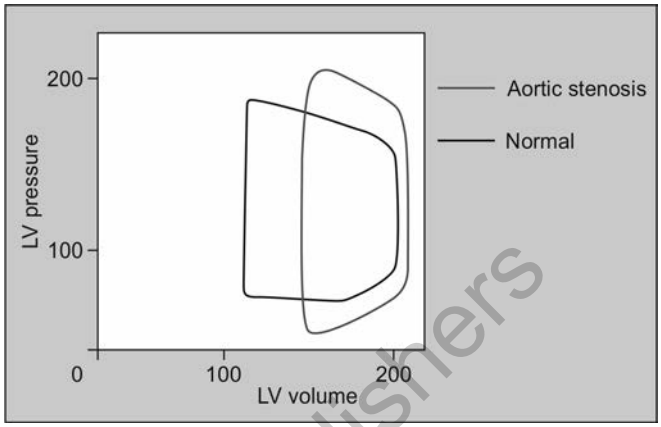


Fig. 7.17: Pressure volume loop in aortic stenosis[5]

Pressure Volume Loop in Aortic Regurgitation

- No true isovolumetric relaxation/contraction
- Greatly increased EDV, as blood flows from aorta and LA into LV
- Raised stroke volume, as volume from LA and aorta is ejected out.

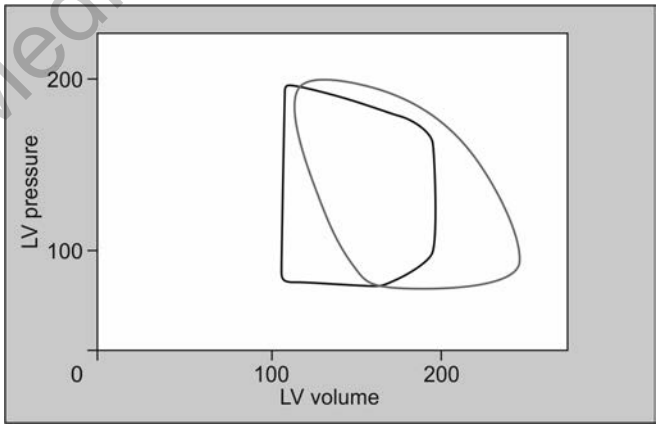


Fig. 7.18: Pressure volume loop in aortic regurgitation[5]

ASSESSMENT OF VENTRICULAR FUNCTION

Assessment of Systolic Function

- Initial rate of rise of arterial pressure tracing
- Change in ventricular pressure overtime during systole (d_p/d_t)
- Ejection fraction
- Ventricular pressure-volume loops
- Preload recruitable stroke work
- LV systolic wall thickening.

Assessment of diastolic function: Using Doppler ECHO via TTE/TEE.

Ejection Fraction

Introduction

- Fraction of end diastolic volume which is ejected from the ventricle
- This measures systolic function of heart.

Calculation

$$\text{Ejection fraction} = \frac{\text{Stroke volume}}{\text{End diastolic volume}} = \frac{(\text{EDV}-\text{ESV})}{\text{EDV}}$$

Normal value: 0.67 ± 0.08

Measurement

- Transthoracic ECHO
- Transesophageal ECHO
- Cardiac catheterization
- Radionucleotide studies.

Grading of LV Dysfunction

Ejection fraction	Grade
• 40–50	• Mild dysfunction
• 25–40	• Moderate dysfunction
• Less than 25	• Severe dysfunction

Factors Reducing Ejection Fraction

- MI/ischemia
- HOCM/DCM
- Myocarditis, amyloid infilterates
- Chronic pressure/volume overload
- HTN, LVF/CCF
- Congenital and valvular heart diseases
- Anemia
- Hypo/hyperthyroidism/pheochromocytoma.

Ventricular Function Curve

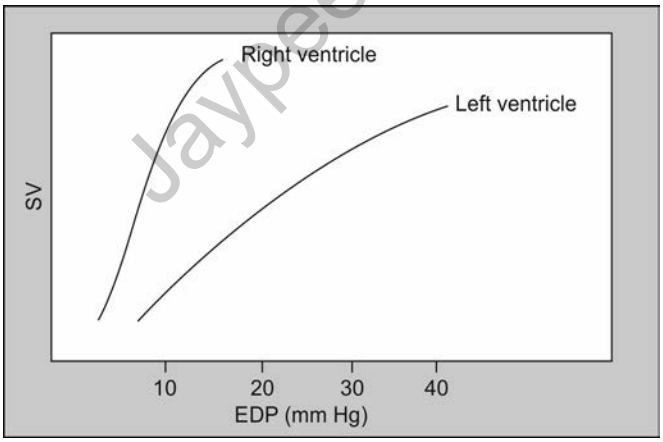
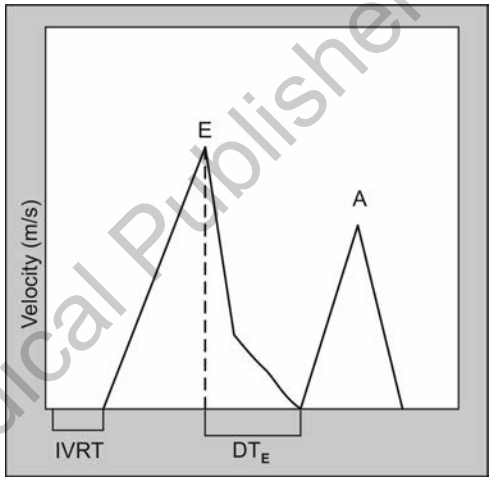
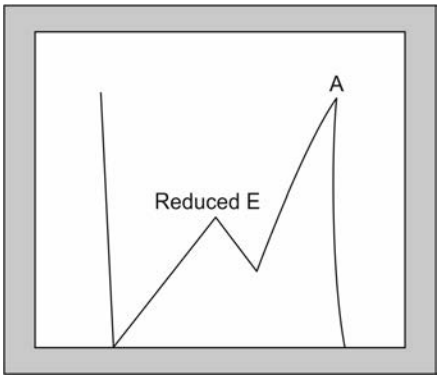


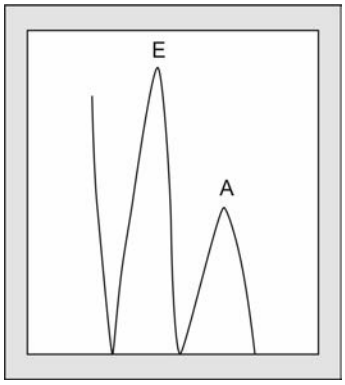
Fig. 7.19: Ventricular function curve

Assessment of Diastolic Function

- Flow velocities across mitral valve measure during diastole
- Three patterns of diastolic dysfunction based on isovolumetric relaxation time, which is the ratio of peak early diastolic flow (E) to peak atrial systolic flow (A)
- Deceleration time (DT) of E (DT_E) also measured and used to categorize diastolic dysfunction.







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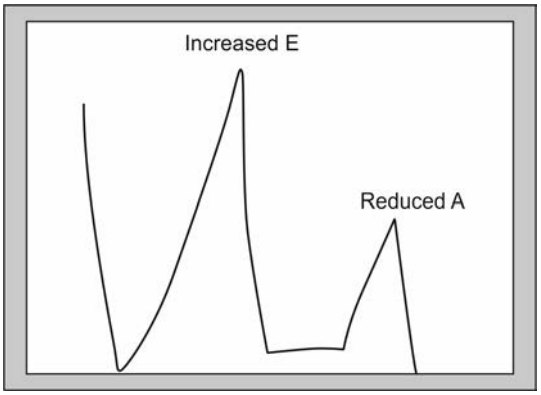


Fig. 7.20: Diastolic dysfunction curves

CAUSES OF INTRAOPERATIVE BRADYCARDIA[1,2,3,5]

Introduction

- Bradycardia is one of the most common arrhythmias that occur during anesthesia
- It may be hemodynamically significant particularly in patients with heart disease.

Causes

Intrinsic

- Systemic causes:
  - Hypoxia
  - Hypercarbia
  - Hypothermia < 34 °C
  - Raised ICT: ***Cushings reflex***
  - Drugs:
    - Opioids, volatile anesthetics, succinylcholine, vecuronium
    - Beta blockers, calcium channel blockers
    - Anticholinesterase inhibitors (neostigmine)
- Cardiac causes:
  - Sick sinus syndrome, sinus bradycardia
  - Junctional rhythm
  - AV nodal blocks
  - Myocardial infarction.

Vagal Response

- Traction on:
  - Extraocular muscles: ***Oculocardiac reflex***
  - Oropharynx (laryngoscopy and intubation, extubation)
  - Bronchus, peritoneum, bowel, rectum
- Right atrial distension
- Bladder distension.

Direct Effect

- Narcotics
- Volatile anesthesia
- Regional anesthesia
- High spinal/spinal shock.

Intrinsic Causes

- Hypoxemia:
  - *Most common* cause
  - Causes cardiovascular and respiratory depression: Bradycardia, hypotension, decreased minute ventilation.
- *Raised ICT:* Causes cushings reflex:
  - Consists of hypertension, bradycardia, irregular respiration.
  - Cushings reflex occurs due to medullary ischemia
- Hypothermia:
  - Causes tachycardia initially due to sympathetic stimulation especially in the presence of shivering
  - Proportionate reduction in heart rate as temperature reduces below 34 °C
  - Occurs due to direct effect of cold on SA node
  - Bradycardia is not affected by atropine or vagotomy.

Vagal Response

- Occurs due to stimulation of:
  - Oropharynx
  - Bronchus
  - Rectum
  - Peritoneum.
- Stimulation causes bronchospasm, bradycardia and hypotension in lightly anesthetized patient.
- Prevented by:
  - Using atropine IV
  - Topical anesthesia with local anesthetics
  - Adrenergic blocking agents
  - Deep anesthetic planes
  - Vasodilating agents.

Direct Anesthetic Effect

- Volatile anesthetics:
  - Mostly occurs with enflurane, isoflurane, halothane
  - Volatile agents directly depress SA node
  - Occurs after slope of phase II depolarization by affecting calcium flux across membrane.

- Regional block: Mild bradycardia on release of tourniquet may be seen.
- IV local anesthetic:
  - Fetal bradycardia seen when IV local anesthetic is given to mother
  - Paracervical block associated with 20–30% chance of bradycardia.
- Succinylcholine:
  - Bradycardia seen after first dose of SCH in children
  - In adults 80% cases present with bradycardia after second dose of SCH if given within 5 minute after first dose
  - This is due to:
    - Choline molecule from breakdown of SCH may sensitize patient to subsequent SCH
    - SCH may directly stimulate peripheral sensory receptors to produce bradycardia
    - SCH stimulation of parasympathetic NS.
- *Vecuronium:* Lacks vagolytic affect and thus may cause bradycardia.
- *Spinal anesthesia:* Bradycardia following SA is mediated by 3 reflexes:
  - ***Atrial/Bainbridge reflex***:
    - Reduction in venous return occurs following SAB
    - This causes reduced efferent output to cardioaccelerator fibers from the atria
    - This causes bradycardia.
  - ***SA node stretch reflex***:
    - Stretch receptors not stimulated in SAN due to reduced venous return following SAB
    - This causes bradycardia.
  - ***Bezold Jarisch reflex***:
    - Reduced venous return causes increased contractility of ventricle
    - This causes stretching of baroreceptors present in inferoposterior wall of left ventricle
    - This in turn increases vagal output from vasomotor center causing bradycardia.

DIAGNOSIS AND MANAGEMENT OF PERIOPERATIVE ARRHYTHMIAS[1,2,3,5,17]

Introduction

Arrhythmias are the most common perioperative cardiac abnormality in patients undergoing both cardiac and noncardiac surgery.

Etiology

- Patient related
  - *Geriatric age: Old age* increases chances of *atrial fibrillation*
  - *Thrombosis:* Coronary/pulmonary
  - History of coronary artery disease/recent cardiac ischemia
  - Subarachnoid hemorrhage
  - *Toxins:* Digoxin toxicity.
  - Trauma:
    - Cardiac tamponade
    - Tension pneumothorax
- Anesthesia related:
  - Light anesthetic plane/pain
  - Drugs:
    - *Halothane:* Enflurane
    - *Catecholamines:* Aminophylline
  - *Central neuraxial block:* Causes pharmacological sympathetectomy
  - Electrolyte imbalance:
    - Hypo/hyperkalemia
    - Hypo/hypermagnesemia: *Torsades de pointes.*
  - Mechanical irritation:
    - Central venous lines
    - Pulmonary artery catheter
    - Chest tubes
    - Tracheal intubation/extubation.
- Surgery related
  - Noncardiac surgery:
    - Traction on intestines
    - Carotid surgery—direct pressure on carotid body
    - Oculocardiac reflex
    - Neurosurgical causes.
  - Cardiac Surgery:
    - Cardiac compression on beating heart
    - Pericardial traction
    - Atrial sutures
    - Venous cannulation
    - Following release of aortic cross clamp.
- Miscellaneous:
  - Hypovolemia
  - Hypoxia
  - Hypercapnea
  - Hypoglycemia
  - Hypothermia/Fever
  - Acidosis
  - Thyrotoxic crisis.

Classification

- Bradyarrhythmias:
  - Sinus bradycardia
  - Heart block:
    - First degree
    - Second degree
    - Complete heart block.
- Tachyarrhythmias:
  - Narrow QRS (SVT) tachyarrhythmias:
    - Sinus tachycardia
    - Atrial premature complexes
    - Unifocal/multifocal atrial tachycardia
    - Atrial flutter
    - Atrial fibrillation
    - Paroxysmal supraventricular tachycardia (PSVT)
    - AV reciprocating tachycardia (AVRT)
    - AV nodal reentrant tachycardia (AVNRT)
  - Wide QRS tachyarrhythmias:
    - Ventricular premature extrasystole
    - Ventricular tachycardia
    - Ventricular fibrillation
    - Torsades de pointes.

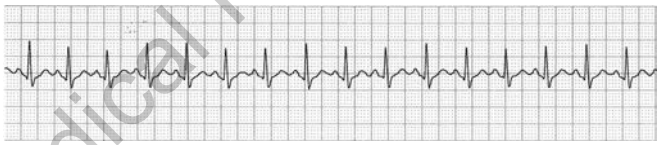
Mechanisms

- Automaticity enhanced:
  - *Normal:* Sinus tachycardia
  - Abnormal:
    - Unifocal atrial tachycardia
    - Accelerated idioventricular rhythms
    - VT post myocardial infarction.
- Triggerred activity:
  - Early after depolarization: Torsades de pointes
  - Late after depolarization:
    - Some VT
    - Digitalis induced arrhythmias.
- *Re-entry:* Sodium channel dependent:
  - Long excitable gap:
    - Atrial flutter
    - Circus movement tachycardia in WPW syndrome
    - Monomorphic VT.
  - Short excitable gap:
    - Atrial flutter
    - Atrial fibrillation
    - Circus movement tachycardia in WPW syndrome
    - Polymorphic VT
    - Monomorphic VT
    - Bundle branch re-entry
    - Ventricular fibrillation.
- *Re-entry:* Calcium channel dependent
  - AV nodal re-entrant tachycardia (AVNRT)
  - Circus movement tachycardia in WPW
  - Ventricular tachycardia.

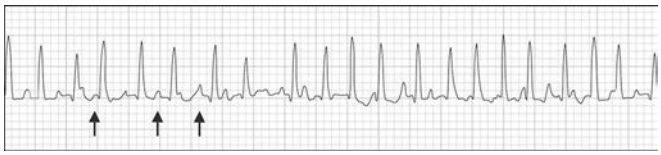
Diagnosis

Tachyarrhythmias

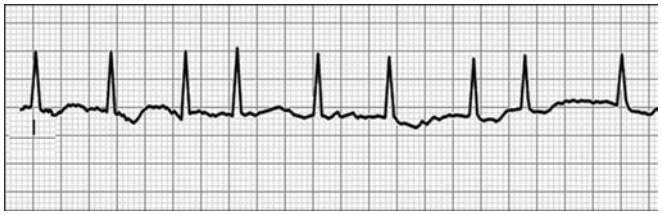
- Sinus tachycardia:
  - Rhythm—Regular
  - Rate—100–180 bpm
  - QRS duration—normal
  - P wave—visible before each QRS complex
  - PR interval—normal.



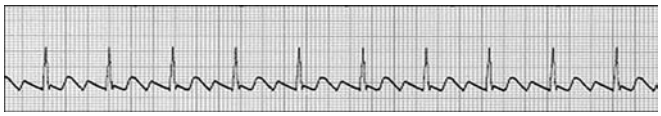
- Unifocal atrial tachycardia:
  - *Rhythm:* Regular
  - *Rate:* 75–200 bpm (ventricular), 120–250 bpm (atrial)
  - *QRS duration:* Normal
  - *P wave:* P wave with consistently abnormal morphology
  - *PR interval:* Usually normal.
- Multifocal atrial tachycardia:
  - *Rhythm:* Irregular
  - *Rate:* 75–200 bpm (ventricular), 120–250 bpm (atrial)
  - *QRS duration:* Normal
  - *P wave:* P waves of atleast 3 different morphologies
  - *PR interval:* Variable.



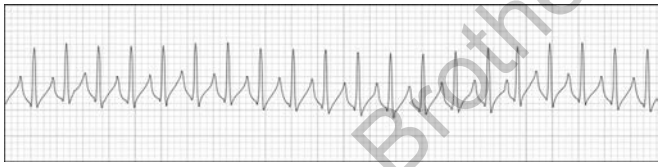
- Atrial Fibrillation:
  - *Rhythm:* Irregularly irregular
  - *Rate:* 100–160 bpm (ventricular), 400–600 bpm (atrial)
  - *QRS duration:* Usually normal
  - *P wave:* Indistinguishable, ***fibrillatory*** waves
  - *PR interval:* Not measurable.



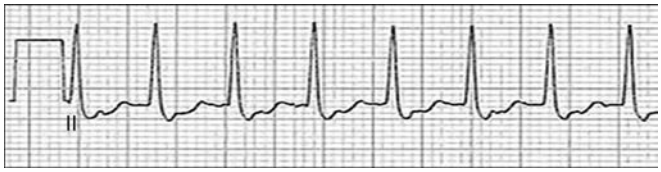
- Atrial flutter:
  - *Rhythm:* Regular
  - *Rate:* Around 110 bpm (ventricular), 250–350 bpm (atrial)
  - *QRS duration:* Usually normal
  - *P wave:* Replaced with multiple ***saw-tooth flutter waves***
  - *P wave rate:* 300 bpm, flutter wave: QRS complexes = 2:1.
  - *PR interval:* Not measurable.



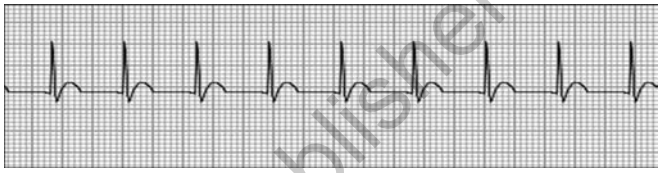
- AV nodal re-entrant tachycardia:
  - *Rhythm:* Regular
  - *Rate:* 180–250 bpm
  - *QRS duration:* Normal, < 120 msec
  - *P wave:* Absent (buried within QRS complex) or occurs immediately after QRS complex
  - *PR interval:* Unmeasurable.



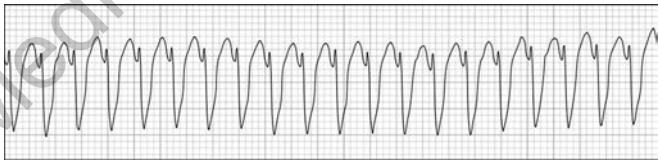
- AV reciprocating tachycardia:
  - *Rhythm:* Regular
  - *Rate:* 150–250 bpm
  - *QRS duration:* Normal in orthodromic, wider in antidromic with ***delta*** waves
  - *P wave:* Follows each narrow QRS complex in orthodromic AVRT
  - *PR interval:* Usually normal
  - *Orthodromic AVRT:* P wave follows each narrow regular QRS complex
  - *Antidromic AVRT:* Wider QRS complexes with ***delta*** waves.



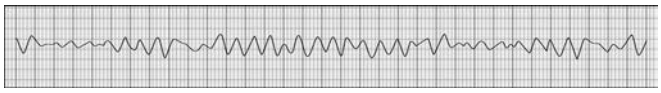
- AV junctional tachycardia:
  - Rhythm: Regular
  - *Rate:* 70–130 bpm (ventricular), 60–100 bpm (atrial)
  - *QRS duration:* Usually normal or widened with aberrant ventricular conduction
  - *P wave:* Inverted/abnormal morphology, irregular relationship with QRS complexes
  - *PR interval:* < 120 msec if present.



- Ventricular tachycardia:
  - *Rhythm:* Regular
  - *Rate:* 180–190 bpm
  - *QRS duration:* Prolonged
  - *P wave:* Not seen
  - *PR interval:* No measurable.

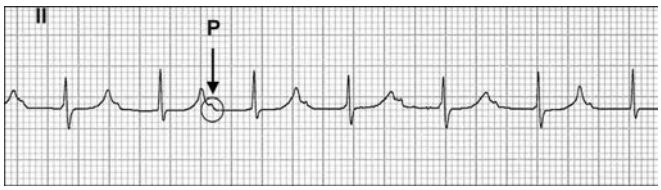


- Ventricular fibrillation:
  - *Rhythm:* Irregular
  - *Rate:* > 300, disorganized
  - *QRS duration:* Not recognizable
  - *P wave:* Not seen
  - *PR interval:* Not measurable.



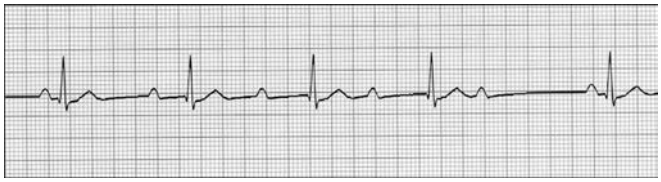
Bradycardia

- First degree AV block
  - *Rhythm:* Regular
  - *Rate:* Normal
  - *QRS duration:* Normal
  - *P wave:* Ratio 1:1
  - *PR interval:* Prolonged > 20 msec.

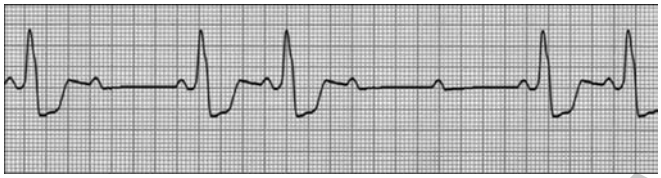


- Second degree AV block type I (Wenckebach)
  - *Rhythm:* Regularly irregular

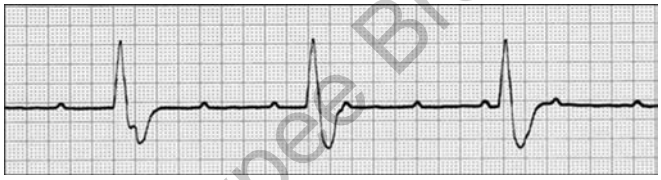
- *Rate:* Normal or slow
- *QRS duration:* Normal
- *P wave:* Ratio 1:1 for 2–4 cycles and then 1:0
- *P wave rate:* Normal but faster QRS rate
- *PR interval:* Progressive lengthening of PR interval until a QRS complex is dropped.



- Second degree AV block type II (Mobitz)
  - *Rhythm:* Regular
  - *Rate:* Normal or slow
  - *QRS duration:* Prolonged
  - *P wave:* Ratio 2:1, 3:1
  - *PR interval:* Normal or prolonged but constant.



- Third degree AV block:
  - *Rhythm:* Regular
  - *Rate:* Slow
  - *QRS duration:* Prolonged
  - *P wave:* Unrelated
  - *P wave rate:* Normal but faster than QRS rate
  - *PR interval:* Variation.



Management

General

- Ensure adequate oxygenation and ventilation
- Deepen plane of anesthesia
- Ensure optimum PaO_2, $PaCO_2$
- Correct acid base imbalance and dyselectrolytemias
- Re-evaluate for cardiac pathology
- Prepare:
  - Anti-arrhythmic drugs
  - Anti-ischemic drugs
  - Pacing equipment
  - Defibrillator
- Vagal maneuvers:
  - Carotid massage
  - Ocular massage
  - Valsalva maneuver.

Specific Arrhythmia Treatment

- Supraventricular Tachycardia:
  - Adenosine—***drug of choice*** – 0.1 mg/kg bolus followed by 0.2 mg/kg after 2 minutes if refractory
  - Verapamil—***second drug of choice*** – 0.1–0.2 mg/kg at 1 mg/min
  - Diltiazem—0.25 mg/kg at 2.5 mg/min followed by 0.35 mg/kg if refractory
  - Metoprolol—0.1 mg/kg given every 5 mins to a maximum of 0.3 mg/kg
  - Rapid overdrive pacing
  - Radiofrequency ablation:
    - For AVNRT, focal atrial tachycardia, atrial flutter, atrial fibrillation
    - Ablation around pulmonary veins, coronary sinus, right atrium, SVC
    - Reduces frequency of recurrent atrial fibrillation in 60% patients.
- Atrial fibrillation:
  - Rate control:
    - Diltiazem: (***rule of 15*** in adults)
      - 0.25 mg/kg at 2.5 mg/min
      - Repeat the dose after 15 min if refractory
      - Infusion at 2.5 mg/kg/hr titrated to control ventricular rate.
    - Verapamil:
      - 0.1 mg/kg at 1 mg/min
      - Repeated in 30 mins
      - Watch for prolonged hypotension.
    - Beta blockers:
      - Esmolol 0.25–0.5 mg/kg followed by 0.05 mg/kg/min titrated to a maximum of 0.2 mg/kg/min
      - Propranolol 0.15 mg/kg given over 2 mins.
    - Digoxin:
      - 5–10 µg/kg IV rapid digitalizing dose
      - Half the total calculated dose administered first
      - Remaining dose administered in fractions at 6–8 hour intervals.
  - Anticoagulation:
    - Unfractionated heparin 80 IU/kg followed by 18 IU/kg infusion
    - Maintain aPTT 1.5 to 2 times reference value

- Gradually shift to warfarin therapy
- Maintain INR of 2–3 to prevent thromboembolic sequelae.

– Restore sinus rhythm:
  - DC cardioversion:
    - 200 J with monophasic defibrillator
    - 120–200 J with biphasic defibrillator
    - 0.5–1 J/kg increased to 2 J/kg if ineffective.
  - Chemical cardioversion:
    - Procainamide:
      - 10–15 mg/kg over 30–60 min until one of the four situations arise:
        - Arrhythmia suppression
        - Hypotension develops
        - QRS prolongation > 50% from baseline.
        - Maximum of 17–20 mg/kg has been administered
      - Maintenance dose 1-4 mg/min.
    - Ibutilide:
      - < 60 kg 0.01 mg/kg over 10 min
      - >60 kg 1 mg over 10 min.
    - Dofetilide:
      - Dose adjusted according to creatinine clearance
      - 250–500 mics PO Q12H.
    - Propafenone:
      - 150–300 mg PO Q8H immediate release
      - 225–325 mg PO Q12H extended release.
    - Amiodarone:
      - 5 mg/kg bolus dose over 20 mins
      - 1 mg/min infusion for 6 hrs
      - 0.5 mg/min maintenance for 18 hrs.

- Ventricular tachycardia/ventricular fibrillation:
  – Unsynchronized DC defibrillation
    - 200 J unsynchronized defibrillation with biphasic defibrillator
    - 360 J unsynchronized defibrillation with monophasic defibrillator
    - In children:
      - First shock 2J/kg, second shock 4 J/kg
      - Subsequent shocks > 4 J/kg up to maximum of 10 J/kg.
  – Resistant VT/VF:
    - Lidocaine:
      - 50–100 mg IV over 5 mins
      - Followed by 2–4 mg/min infusion
    - Amiodarone
      - 5 mg/kg bolus dose over 20 mins
      - May repeat twice for refractory cases
    - Bretilium:
      - 5–10 mg/min over 2–5 min
      - Followed by 1–2 mg/min.

MYOCARDIAL PROTECTION[1,2,3,5,17]

Introduction

Myocardial protection refers to the set of pharmacological and physiological strategies aimed at attenuating the intensity of myocardial ischemia-reperfusion injury during cardiac surgery and its consequences on myocardial function.

History

- Early efforts with topical hypothermia:
  – Caused postoperative myocardial depression
  – Also lead to myocardial contracture ***'stone heart'***
- This late led to potassium cardioplegia done by ***Melrose*** in 1955 with:
  – 2.5% solution of potassium citrate and blood
  – High levels of potassium used, produced direct myocardial damage.
- This was replaced in 1970s by cold hyperkalemic blood cardioplegia using lower potassium levels.

Goals of Myocardial Protection

- To provide a quite bloodless field
- Limitation of myocardial damage by reduction of:
  – Intracellular acidosis
  – Edema
  – Depletion of high energy phosphate stores (ATP)
- Preservation of coronary endothelial functions and myocardial flow
- Reduced reperfusion injury.

Principles

- Myocardial vulnerability to injury is a product of:
  – Preoperative functional class of patient
  – Presence of ventricular hypertrophy
  – Coronary artery disease.
- Myocardial protection should begin with preparation of heart prior to surgery
- Metabolic requirements should be reduced during arrest interval
- Favorable metabolic mileu during arrest provides a margin of safety with reduced metabolism
- Measures taken must represent a balance between requirement during aortic cross clamping and reperfusion.

- Reperfusion modification after an ischemic insult can minimize structural and functional damage to myocardium.

Determinants of Oxygen Supply

- Oxygen content of blood
- Aortic root pressure
- Autonomic innervation
- Ventricular pressure
- Local vascular resistance, autoregulation, coronary steal
- Diastolic perfusion time (Heart rate)
- Exogenous drugs.

Determinants of Oxygen Consumption: MvO_2

- Myocardial contractility (inotropy) = velocity of pressure development (dp/dt)
- Wall tension = $\frac{\text{Pressure} \times \text{Radius}}{2 \times \text{Wall thickness}}$
- Heart rate
- Basal oxygen consumption
- Work = Area within systolic pressure – volume loop.

Phases of Myocardial Injury

- Focal ischemia causes irreversible injury, manifested as myocellular necrosis after as little as 30 minutes following coronary occlusion in the intact working heart
- Similar period of global ischemia causes moderate to severe functional depression without obvious necrosis
- Myocardial injury sustained in the surgical setting is divided into 3 phases:
  - Antecedent ischemia:
    - Also called ***Unprotected ischemia***
    - Occurs prior to institution of CPB or delivery of cardioplegia solution.
  - *Protected ischemia:* Occurs during electively initiated chemical cardioplegia
  - Reperfusion injury: Occurs:
    - During intermittent infusion of cardioplegia solution
    - After removal of cross clamping
    - After discontinuing CPB.

Methods of Myocardial Protection

Measures Against Antecedent Ischemia

- Continue all preoperative cardiovascular drugs:
  - Antiarrhythmics
  - β blockers
  - Calcium channel blockers
  - Nitrates, aspirin
  - Digoxin.
- *Premedication:* Sedation for CABG patients
- Conversation to reduce level of anxiety
- Intubation response suppression:
  - *Lignocaine:* 1.5–2 mg/kg IV 90 seconds before intubation
  - *$MgSO_4$:* 20–60 mg/kg IV
  - *Nitroglycerine drip:* 0.5–10 µg/kg/min
  - *Labetolol/esmolol:* 20 mg IV or 300 µg/kg IV respectively
  - Deepen planes of anesthesia: Inhalational or fentanyl 1 µg/kg
  - Brief duration of laryngoscopy: ≤ 10 seconds
- Administration of glucose to increase hearts ability to tolerate ischemic arrest
- Avoid ventricular distension and fibrillatory arrest
- Control of heart rate to < 80 bpm in patients with coronary artery disease
- Adequate hydration to enable heart to tolerate hypotension prior to initiation of CPB
- Anesthetic preconditioning with volatile anesthetics.

Measures Against Protected Ischemia

- These measures are taken during the period of CPB:
  - Asystole with chemical cardioplegia
  - Hypothermia
  - Hypocalcemia
  - Buffering
  - Intermittent cross clamping with brief periods of reperfusion
  - Hypothermic ventricular fibrillation
  - Left ventricular venting.

Measures Against Reperfusion Injury

- Oxygen free radical scavenging
- IV sedation with propofol
- Anti-ischemic agents:
  - ATP sensitive potassium channel openers:
    - Cromokalim
    - Pinacidil
    - Nicorandil
  - Agents which increase intravascular adenosine:
    - 5-amino-4-imidazole carboxamide riboside deaminase inhibitors
    - Transport inhibitors.
- Monoclonal antibodies directed against neutrophil/endothelial components of adhesive glycoprotein complexes.

Asystole

Rationale of Use

- Continued electromechanical activity after cross clamping increases the rate of high energy phosphate depletion
- This causes a more rapid fall in ATP reserves before anoxic arrest ensues
- Intentional chemical induction of diastolic arrest avoids depletion of ATPs
- This conserves myocardial energy reserves
- Reserves can be used during ischemia to maintain ionic and metabolic homeostasis.
- Total electromechanical silence reduces oxygen demand by about 80–90% of which:
  - 30–40% of this reduction is contributed by ventricular decompression
  - The remainder is contributed by asystole accomplished by chemical cardioplegia.

Methods of Inducing Rapid Asystole

- Hyperkalemia
- Hypermagnesemia
- Hypocalcemia
- Calcium channel blockers
- Infusion of local anesthetic agents.

Hypothermia

Benefits

- Hypothermia with sustained myocardial activity reduces myocardial oxygen demand
- Hypothermia in combination with cardiac arrest reduces MVO_2 by 97% if cooling is done to < 22 °C
- Hypothermic cardioplegia increases the duration for which aortic clamping may be safely imposed from < 30 minutes to as much as 4 hours
- Hypothermia enables tissues to withstand complete interruption of blood flow for periods of 20–40 minutes.

Rationale

- Major methods of myocardial protection by hypothermia is:
  - Reduction in myocardial metabolic rate
  - Reduction in oxygen demand of myocardium.
- ***Vant Hoff's law/Q_{10} effect***:
  - MVO_2 in the quiescent heart reduces by 50% for every 10 °C reduction in temperature.
  - Greatest reduction in MVO_2 occurs between 37 °C and 25 °C
  - At 28 °C, a reduction in metabolic rate by 50% occurs
  - Q_{10} is the ratio of metabolic rates at two different temperatures
  - Normally, Q_{10} = 2 to 2.2.
- Greatest protection at 28 °C with little additional protection at temperature < 28 °C.
- Hypothermia is achieved on CPB by two methods:
  - *Passive cooling:* Patients core temperature is allowed to equalize with ambient temperature
  - *Active cooling:* Using heat exchanger.

Advantages of Hypothermia

S. No.	Advantages
1	Reduces myocardial metabolism
2	Reduces oxygen requirement
3	Reduced rate of degradative reactions
4	Reduced progression of ischemic injury
5	Reduced tolerable period of ischemia
6	Reduced potassium needed to accomplish arrest
7	Prolongs cardiac arrest
8	Inhibits intracellular Ca^{2+} gain
9	Reduces excitatory neurotransmitters

Disadvantages of Hypothermia

S. No.	Disadvantages
1	Reduces rate of reparative process
2	Increased myocellular swelling
3	Increases inotropic state and MVO_2 per beat
4	Induces fibrillation
5	Impairs oxygen dissociation
6	Impairs autoregulation
7	Poses threat of phrenic nerve ***freeze injury*** on local application of ice
8	Increases rouleaux formation in RBCs

Hypocalcemia

- Calcium plays a central role in excitation—contraction coupling
- Calcium is needed to transduce electrical signals into a mechanical event
- Removal of calcium from extracellular environment produces a negative inotropic effect
- This causes cessation of contractile activity at very low levels of extracellular ionic calcium.
- ***Calcium paradox***:
  - Can be precipitated in ischemic myocardium by moderate hypocalcemia

  - Hypocalcemic reperfusion causes extended infarct size after reversible coronary occlusion.
- Reduction of extracellular calcium done by direct chelation using:
  - Sodium citrate which may chelate other ions like magnesium
  - Alternative is EGTA.

Oxygen Radical Scavenging

- Hydrogen peroxide is an important transitional molecule in the oxygen radical cascade
- It is involved in ischemic reperfusion injury
- Infusion of cardioplegia is also a form of reperfusion during which injury can occur
- Enzymes and scavengers which inhibit or dismutate free radicals should be administered as a pretreatment therapy
- Free radical scavengers which can be used:
  - Superoxide dismutase
  - Catalase
  - Ibuprofen
  - Indomethacin
  - Nitric oxide.

Anesthetic Preconditioning

- Halothane:
  - Attenuates ST segment changes caused by brief coronary artery occlusion
  - Reduces ST elevation more than sodium nitroprusside and propranolol
- Isoflurane and sevoflurane:
  - Reduce myocardial reperfusion injury
  - Improve functional recovery after global ischemia
- Volatile anesthetics reduce MvO_2 due to:
  - Direct negative inotropic, lusitropic and chronotropic effects
  - Reduction in LV afterload (reduces SVR)
- Volatile anesthetics reduce oxygen free radicals and also cause coronary vasodilatation.

Propofol

- IV sedation with propofol after myocardial revascularization resulted in 17% less frequent incidence of tachycardia and 28% less frequent HTN.
- Infusion of propofol results in significant reduction in myocardial blood flow and myocardial oxygen consumption.
- High dose propofol (120 µg/kg/min) administered for CABG or OPCAB showed improved troponin levels and better hemodynamic function.

CARDIOPLEGIA[1,2,3,5,17]

Introduction

This is the intentional, temporary cessation of the heart for cardiac surgery, by which cardiac standstill is achieved using high potassium concentrations of 12–30 mEq/L within 1–2 minutes.

Mechanism

- Potassium containing cardioplegia solution arrests the heart in diastole
- This causes a diastolic arrest which allows for a longer ischemic time
- This reduces myocardial oxygen demand by ≥ 80%
- Exogenous potassium causes depolarization of myocyte membrane
- This renders the myocardium unexcitable during the time that potassium remains within the tissue
- Wash out of hyperkalemic cardioplegia solution by noncoronary collateral flow causes resumption of electrical and mechanical activity
- This is countered by intermittent (every 20–30 min) replenishment of cardioplegia solution.

Goals

- To provide a quiet and bloodless field.
- Limitations of myocardial damage by reduction of:
  - Intracellular acidosis
  - Edema
  - Depletion of ATP stores
- Preservation of coronary endothelial function and myocardial flow
- Reduce reperfusion injury.

Types

Based on Temperature

- Warm cardioplegia
  - Delivered at 37 ºC
  - Mechanisms of action:
    - Reduces reperfusion injury
    - Preserves coronary endothelial function
    - Rapid establishment of myocardial energy stores
    - Reduces calcium influx
    - Causes less activation of lymphocytes
    - Up-regulates protective heat shock proteins
  - Single hot shot warm cardioplegia used for initiation and termination of arrest.
- Cold cardioplegia:
  - Delivered at 10 ºC
  - Heat exchanger used
  - Reduces oxygen demand and ischemic damage
  - Ice is used for surface cooling.

Based on Contents

- *Crystalloid cardioplegia:* Using lactated ringers
- Blood cardioplegia:
  - *Using 4:1 or 8:1 concentration of blood:* Crystalloid
  - Blood useful as:
    - Acts as source of oxygen to heart
    - Buffering and antioxidant properties
    - Preferred in anemic and pediatric patients.

Methods of Delivery

- Anterograde:
  - Delivered into coronary arteries via aortic root through a needle placed between aortic cross clamp and aortic valve.
  - Can be delivered through individual grafts in CABG once distal anastomoses has been made
  - Most physiological
- Retrograde:
  - Delivered into coronary sinus through a balloon tipped cannula
  - Better distribution of cardioplegia in:
    - Significant coronary artery disease
    - Aortic regurgitation
    - Aortic valve surgeries
  - Adequate delivery to right ventricular myocardium and posterior one-third of septum is difficult
  - Should be delivered at < 40 mm Hg pressure to limit perivascular edema and hemorrhage.
- *Combined:* Thought to be superior to either of the above.

Timing of Delivery

- Intermittent cardioplegia:
  - Initially 1–1.5 L of *high potassium* cardioplegia (CP) delivered
  - Then 200–500 ml of *low potassium* CP infused at periodic intervals of 20–40 minutes
  - High potassium CP solution contains 20–30 mEq/L of potassium
  - Low potassium CP solution contains 10–20 mEq/L of potassium.
- Continuous cardioplegia:
  - Delivered at low flows
  - Continuous warm cardioplegia advocated for long surgeries
  - Concern about protection of right ventricle and posterior septum in this method.

Composition

- Potassium:
  - *High potassium CP:* 20–30 mEq/L
  - *Low potassium CP:* 10 mEq/L
  - Higher levels cause increased energy requirement and increased potassium load
- Sodium:
  - Concentration lesser than plasma sodium (140 mEq/L)
  - Ischemia increases intracellular sodium.
- Calcium:
  - 0.7–1.2 mmol/L of calcium in CP solution
  - Maintains cellular integrity
  - Hypocalcemic solutions cause massive calcium influx: ***Calcium paradox.***
- Magnesium:
  - 1.5–15 mmol/L of magnesium in CP solution
  - Controls increased influx of calcium.
- Buffers:
  - Bicarbonate prevents increased build up of acid metabolites.
  - *Alternate buffers:* Histidine, THAM (trometh-amine)
- Mannitol: To control cellular edema
- Membrane stabilizing agents:
  - Procaine
  - Lidocaine
  - Glucocorticoids.
- Energy substrates:
  - Glucose
  - Glutamate
  - Aspartate
  - Blood/crystalloid.

Strategies to Reduce Ischemic Injury with Cardioplegia[5,17]

No	Principle	Mechanism	Component
1	Reduce O_2 demand	Hypothermia	Blood/crystalloid
			Ice slush/lavage
		Asystole	Potassium chloride, adenosine
			Hyperpolarizing agents
2	Substrate supply and use	Oxygen	Blood, PFC
		Glucose	Blood, glucose, citrate-phosfate-dextrose
		Amino acids	Glutamate, aspartate
		Buffers	Blood, bicarbonate, phos-phate, THAM, histidine

Contd…

Contd…

No	Principle	Mechanism	Component
3	Control calcium influx	Hypocalcemia	Citrate
			Calcium channel blockers
			Potassium channel openers
4	Reduce edema	Hyperosmolar solutions	Glucose
			Potassium chloride
			Mannitol
		Moderate infusion pressure	Less than 50 mm Hg

Adverse Effects of Cardioplegia

- Excessive cardioplegia:
  - Absence of electrical activity
  - AV conduction block
- Poorly contractile heart at termination of bypass
- Persistent systemic hyperkalemia.

Cardioplegia Circuits

Two types of circuits:

- Recirculating circuit:
  - For asanguineous delivery
  - Crystalloid CP solution is kept running constantly in the circuit
  - It is delivered to a patient on removing a clamp thus directing flow away from recirculation circuit and to patient
- Non-recirculating circuit:
  - For sanguinous delivery
  - Involves shunting of arterialized blood from oxygenator into the CP circuit
  - Here it is mixed with a crystalloid base solution and delivered
  - Makes only a single pass through heat exchanger.

CARDIOPULMONARY BYPASS-BASIC CIRCUITRY

Introduction

- CPB is a form of extracorporeal circulation in which patients blood is rerouted outside the vascular system and the functions of the heart and lungs is temporarily assumed by surrogate technology
- Almost 25–30% of patients circulating volume is outside the body during CPB.

Goals

- To provide a quiet and bloodless field.
- Limitations of myocardial damage by reduction of:
  - Intracellular acidosis
  - Edema
  - Depletion of ATP stores
- Preservation of coronary endothelial function and myocardial flow
- Reduce reperfusion injury.

Types of Extra Corporeal Circuitry

- Cardiopulmonary bypass (CPB)
- Left heart bypass
- Cardiopulmonary support
- Extra Corporeal Membrane Oxygenation (ECMO).

Components

- Venous line
- Venous reservoir
- Arterial pump
- Heat exchanger
- Oxygenator
- Arterial line filter
- Arterial line
- Accessory pumps:
  - Cardiotomy suction
  - LV vent
  - Cardioplegia pump.
- Accessory devices:
  - Ultrafilter
  - Volatile agent vaporizers
  - In line blood gas monitor.
- Prime.

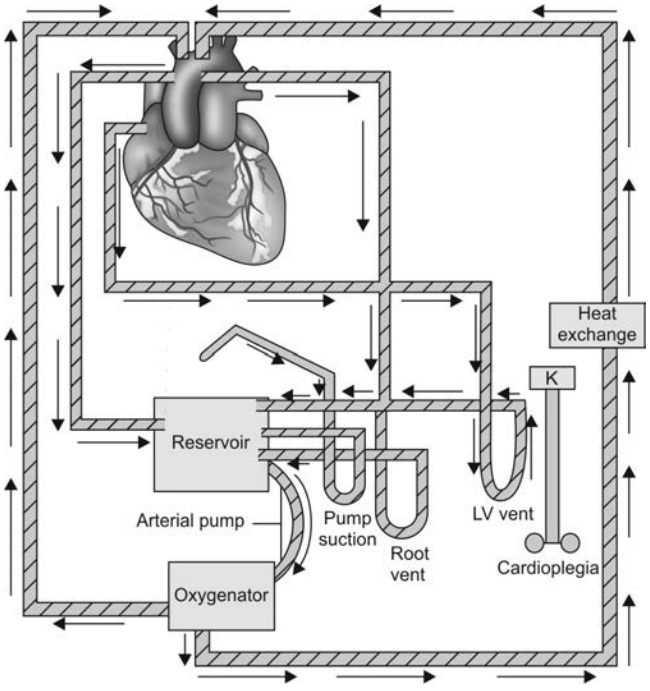


Fig. 7.21: CPB basic circuitry[1]

Venous Line

Description

- Made of medical grade polyvinyl chloride with surface coatings which:
  - Alter bioactivity of surface to reduce markers of subclinical coagulation
  - Attenuate cytokine and other inflammatory markers.

Types of Venous Cannulation

- Single atrial cannulation:
  - Cannula present in RA which drains blood from both RA and IVC
  - It is simpler, faster, less traumatic with one less incision
  - Interferes least with caval return when off bypass.
- Bicaval cannulation/single stage cannula:
  - Placed in SVC and IVC
  - Cannula is snared to prevent systemic venous blood from entering the heart
  - Used anytime when right heart entry is anticipated.
- Cavoatrial cannulation/dual stage cannulation:
  - Superior decompression of right heart
  - Reduced incidence of myocardial ischemia and better protection
  - One cannula is in RA which extends into IVC.
- Femoral venous cannulation done in:
  - In emergent situation
  - Aortic surgery
  - Thoracic surgery.

Venous Reservoir

Description

- Act as buffer for imbalances between venous return and arterial flow
- Postured between venous line and arterial pump
- May be collapsible plastic bags/clear hard shelled plastic containers
- Blood flows in via gravitational drainage
- The amount of venous drainage is proportional to:
  - The pressure in central veins (directly proportional)
  - Height difference between reservoir and patient (directly proportional)
  - Resistance in venous cannula and line (inversely proportional).
- Reservoir has integrated positive and negative pressure release valves which are important for application of suction to the reservoir to augment venous drainage
- If venous reservoir is allowed to empty, air can enter the main pump and cause fatal air embolism
- *Arterial reservoir:* When reservoir is placed beyond oxygenator as in the case of a bubble oxygenator.

Clinical Uses

- Provides time for perfusionist to act if venous drainage is sharply reduced or stopped, in order to avoid pumping air
- Acts as buffer for imbalances between venous return and arterial flow
- Facilitates displacement of large volume of blood out of circulation at strategic times during the operation
- Serves as gross trap for air which enters the venous line
- Serves as a site where blood, fluids and drugs can be added.

Arterial Pumps

- Three types of arterial pumps:
  a. Roller pumps
  b. Centrifugal pumps
  c. Pulsatile assist devices.

Roller Pumps

- Positive displacement pumps which function by occluding a point in a piece of tubing and then rolling the occlusive point of contact along a length of the tubing.
- Can generate extremely high positive and negative pressures and can pump massive quantities of air
- Regulated to automatically reduce their speed when high pressure/air is detected in the path of blood
- If pump inflow is occluded negative pressure will develop in rollerhead causing cavitation
- If pump outflow is occluded, increased pressure may develop proximal to occlusion causing tubing connection to separate or tube to burst
- Subtotal occlusion prevents RBC trauma
- All roller pumps have cranks to allow manual pumping
- Pulsatile flow is possible with some roller pumps
- Pulsations can be produced by instantaneous variations in rate of rotation of roller heads

- Can cause *spallation* which is development of plastic microemboli due to tube compression.
- Advantages of pulsatile flow:
  - *Increased tissue perfusion:* Increases renal and cerebral blood flow
  - Increased O_2 extraction
  - Reduces release of stress hormones
  - Reduces SVR during CPB.

Centrifugal Pumps

- These are non-volatile kinetic energy pumps
- They generate flow by magnetically coupling the high speed revolution of a reusable motor cone to plastic plates inside a disposable cone
- This produces a constrained vortex which propels fluid through the opening on side of cone while drawing fluid into the point of the cone
- In contrast to roller pumps which are placed after the oxygenator, centrifugal pumps are normally between venous reservoir and oxygenator.
- Disadvantages:
  - Unable to generate extremely high/low pressures
  - If bolus of air is introduced, pump will deprime, rendering it unable to pump large volumes of air
  - Lack of point of tubing occlusion allows retrograde flow from patients high pressure arterial system backwards through arterial line, oxygenator, and venous reservoir when revolution of pump goes below a critical threshold
  - Cannot produce pulsatile flow.
- Advantages:
  - Reduced risk of RBC trauma as they are non-occlusive
  - Reduced cavitation
  - Reduces risk of massive air emboli
  - Elimination of tubing wear off and spallation.

Pulsatile Assist Devices

- Examples:
  - Extracorporeal balloon
  - Intra-aortic balloon pump (IABP)
  - Ventricular type pneumatic/hydraulic pumps
  - Modified roller pumps
  - Modified centrifugal pumps
- These pumps may increase the risk of air embolism.

Heat Exchanger

- Facilitates management of patients blood temperature
- Hypothermia common on CPB as up to 25–30% of patients blood is outside the body
- Either cooling and warming of the patient can be achieved by altering temperature of water flowing through the exchanger (4–42 ºC)
- Heat transfer occurs by conduction
- They usually have an inbuilt filter to filter air bubbles which may form during rewarming.
- Counter current device where either heated/cooled water is circulated around a conducting material with good thermal properties which is in contact with patients blood
- The blood is subsequently warmed/cooled and maintained at the desired temperature
- It is located proximal to gas exchange section of circuit to minimize the risk of releasing gas bubbles from blood which can happen if blood is warmed after being saturated with gas.

Oxygenator

- Substitutes for patients lung and performs gas exchange through a blood gas interface
- *Two types of oxygenators:* Membrane/bubble oxygenators
- Membrane oxygenator:
  - Made of microporous polypropylene extruded into thin straws with:
    - Outer diameter of 200–400 µm
    - Wall thickness of 20–50 µm
    - Total surface area of 2–4 m^2
    - Microscopic pores on the sides of fibers (0.5–1 µm)
  - Usually placed after the pump as resistance in most membrane oxygenators requires blood to be pumped through them
  - Has separate gas inlet and outlet ports
  - Ports are small enough to prevent plasma and formed elements from leaving out but still are large enough to allow gas to pass through
  - The venous blood entering the oxygenator is directed across the outside of the fibers: *Blood space.*
  - The gas is concurrently circulated through the inside of the fibers: *gas space*
  - Can arterialize up to 7 L/min of venous blood
  - Arterial oxygenation is inversely proportional to thickness of blood film in contact with the membrane.
  - Arterial CO_2 tension is directly proportional to total gas flow.

- Bubble oxygenators:
  - *Work on principle of direct blood:* Gas contact
  - Positioned proximal to pumps
  - Blood gas interface may cause hemolysis, platelet destruction and microemboli
  - Divided into two sections:
    - Mixing chamber:
      - Fresh gas flows through a perforated plate/screen which forms gas bubbles
      - Diffusion takes place on the bubble surface
      - By using small bubbles a large surface area develops
      - The driving force behind the diffusion of gases is the difference in partial pressures between gases in bubbles and dissolved gases in the blood
      - The oxygenated blood then enters a reservoir/heat exchanger.
    - Reservoir chamber:
      - Blood is allowed to settle and is passed through a defoaming matrix
      - This causes bubbles to destabilize and break down prior to returning to the patient
      - This may cause time dependent destruction of blood elements if CPB time > 90 min.

Arterial Line Filter

- Placed in arterial line as the last component through which blood passes before it returns to patient
- Can be depth filters/screen filters
- Pore sizes of 20–40 μm which removes:
  - Gaseous microemboli
  - Particulate emboli:
    - Thrombi
    - Fat globules
    - Calcium
    - Tissue debris.
- Always has a bypass limb (normally clamped) in case if becomes clogged/develops high resistance.

Arterial Line

- Can be placed in:
  - Ascending aorta
  - Femoral artery
  - Axillary artery
  - Iliac artery which is exposed via retroperitoneal suprainguinal approach.
- Location of cannula determined by:
  - Patient anatomy
  - Surgeon preference
  - Surgical procedure.
- Iliac/femoral artery used when:
  - Aneurysm of ascending aorta
  - When surgery involves multiple procedures involving ascending aorta
  - For peripheral cannulation under local anesthesia in unstable patients
  - When antegrade dissection complicates aortic cannulation.
- Optimal arterial BP required during arterial cannulation as:
  - If too high, increased chances of aortic dissection and tears
  - If too low aorta may collapse
  - Mean blood pressure of 80–100 mm Hg preferred
  - Systolic blood pressure of 100–120 mm Hg preferred.

Cardiotomy Suction

- Aspirates blood from surgical field during CPB and returns it to main pump reservoir
- Excessive suction pressure used may cause RBC trauma due to high negative pressure
- This damage to RBCs precludes blood salvage from cardiotomy
- Disadvantages of cardiotomy suction:
  - Causes:
    - Hemolysis
    - Particulate and gaseous microemboli
    - Fat globule formation
    - Platelet injury
  - This occurs due to amount of air which is aspirated with blood
  - The air blood mixture causes turbulence and high shear stresses which injure both RBCs and platelets.
- Cell saver suction devices:
  - Aspirates blood with controlled vacuum
  - RBCs are automatically washed with saline, separated from the fluid by centrifugation and returned to extracorporeal circuit
  - It removes microaggregates fat, air and tissue debris.

Left Ventricular Vent

- Blood may accumulate in LV due to:
  - Residual pulmonary blood flow

  - From PDA and bronchial arteries
  - Thebesian veins
  - From major aortopulmonary collaterals
  - Due to aortic regurgitation
  - This compromises myocardial protection and thus LV requires regular venting.
- Blood which accumulates is suctioned out using LV vent.
- This is done via catheter passed into:
  - Left ventricular apex directly
  - Left ventricle via right superior pulmonary vein and left atrium
  - Into main pulmonary artery.
- Vented blood passes through a filter and returns to venous reservoir.
- Uses of LV venting:
  - Prevents myocardial distension
  - Reduces myocardial rewarming
  - *Prevents ejection of air:* Used to aspirate air from heart during de-airing procedure
  - Facilitates surgical exposure, e.g. when working on aortic valve.
- Importance:
  - Distension of ventricle causes mechanical damage to muscle from excessive stretching
  - Venting improves preservation by reducing myocardial oxygen demand
  - Venting reduces wall tension due to a reduction in radius of ventricle
  - It facilitates subendocardial perfusion
  - It prevents pulmonary venous hypertension with pulmonary edema.
- *Disadvantage:* May cause systemic air embolism by introducing air into left heart.

Cardioplegia Pump

- Allows optimal control over infusion pressure, rate and temperature of cardioplegia
- Separate heat exchanger ensures accurate control of temperature of cardioplegia solution
- This solution may be infused from a cold IV fluid bag given under pressure or by gravity.

Ultrafilter/Hemofilter/Hemoconcentrator

- Used to increase patients hematocrit without transfusion
- Contains hollow semipermeable fibers which can function as membrane
- Blood is passed through fibers either from arterial side/venous reservoir using an accessory pump
- Hydrostatic pressure forces water and electrolytes across the fiber membrane
- This allows separation of aqueous phase of blood from its cellular and proteinaceous elements
- Effluents of up to 40 ml/min can be removed
- Can be used post bypass to concentrate pump blood before it is given back to patient
- Conserves coagulation factors, platelets and albumins.

In Line Blood Gas Monitors

- Non-invasive flow-through devices to measure blood gases in arterial and venons lines
- Arterial monitor provides continuous assessment of arterial oxygenation like a pulse oximeter
- Venous oximetry provides rapid assessment of balance of oxygen supply and demand.

Automatic Data Collection Systems

- To assist preoperative calculation, processing and storing data during bypass
- This allows perfusionist to attend to more important tasks.

Prime

- Fluid contained within CPB tubing is called prime
- Initially blood was used but practice was stopped due to:
  - Increased demand for blood
  - Scarcity of supply
  - ***Capillary leak syndrome***: Postoperative pulmonary dysfunction due to histamine release
- Crystalloid solutions like RL used now due to similar osmolarity and electrolyte composition
- Average prime volume is 1500–2500 ml
- Minimal volume used based on body weight/BSA
- Other components which are added to make up prime:
  - Mannitol for diuresis
  - Albumin to reduce postoperative edema
  - Electrolytes like calcium to prevent hypocalcemia due to citrate in transfused blood
  - Corticosteroids for anti-inflammatory action
  - Heparin for safe level of anticoagulation
- Blood may be added in:
  - Children

- Small adults
- Patients with preoperative anemia to prevent dilutional anemia

- *Lowest safe hematocrit on CPB is 20%.*

VENTRICULAR SEPTAL DEFECT[1,2,3,5,17]

Introduction

It refers to an abnormal communication between the two ventricles.

Incidence

- Isolated VSDs account for around 25% of all congenital heart defects
- Incidence is higher in premature infants and also slightly higher in males.

Embryology

- Single ventricle divided into two by the union of:
  - Membranous portion of ventricular septum
  - Bulbus cordis
  - Endocardial cushions
- Formation of septum occurs in fifth week of intrauterine life
- Development of VSD due to:
  - *Perimembranous VSD:* Defects in development of membranous septum
  - *Outlet VSD:* Failure of fusion of conus septum
  - *Inlet VSD:* Failure of fusion of endocardial cushion with muscular septum
  - *Muscular VSD:* Inadequate fusion of medial walls of right and left ventricles.

Classification

- *Perimembranous VSD (70%):* May extend into adjacent muscular septum and can be classified based on the direction of extension into:
  - Trabecular extension (typical type)
  - Inlet extension (AV canal type)
  - Outlet extension (TOF type).
- Muscular VSDs (25%):
  - Trabecular extension
  - Inlet extension
  - Outlet extension.
- Subarterial infundibular VSD (5%):
  - Are located just beneath the semilunar valves
  - Thus, aortic regurgitation is commonly associated as aortic valve cusps often protrude into the infundibular VSD.

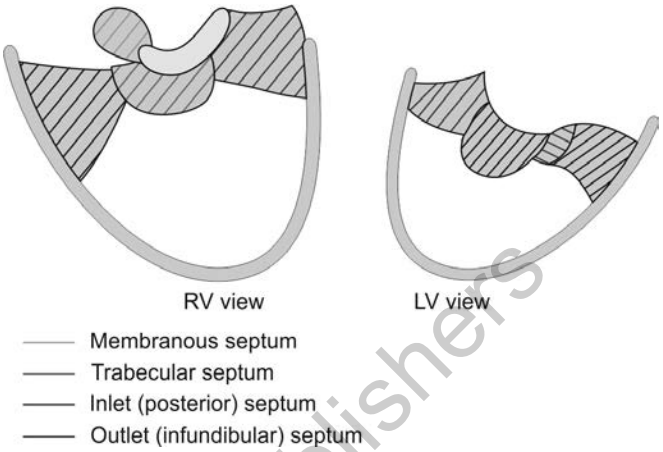
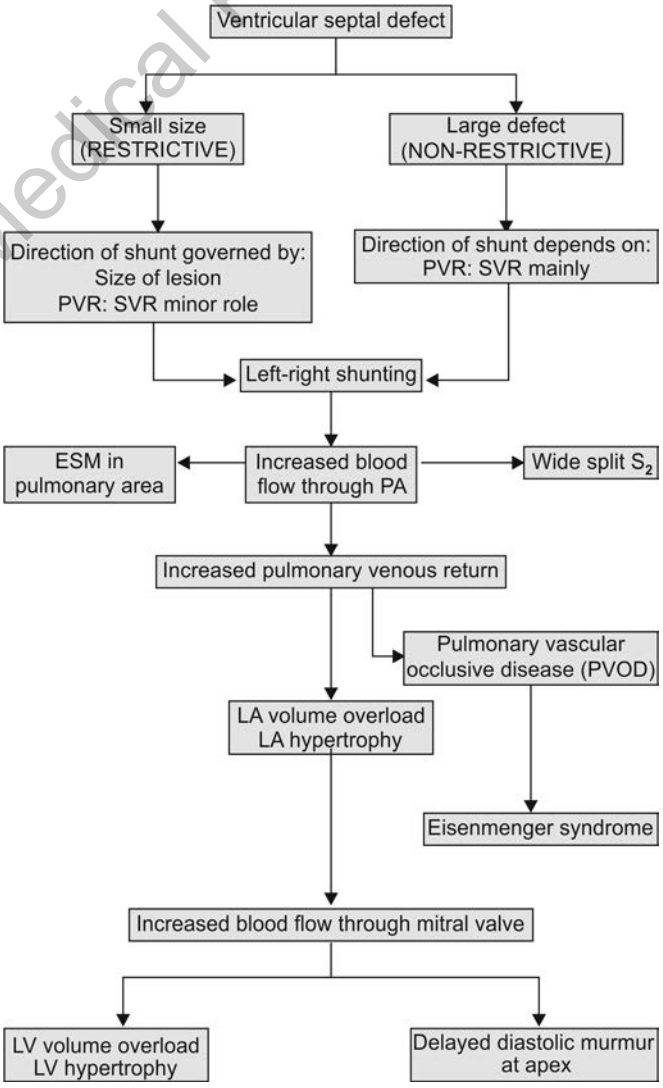


Fig. 7.22: Ventricular septal defects

Pathophysiology



- Only if a large VSD exists, direct transmission of LV pressure to RV causes biventricular hypertrophy
- In small VSDs, since shunting occurs when both ventricles are contracting simultaneously, the shunted blood directly enters pulmonary artery
- Thus, only LV changes are seen and RV is spared in small VSDs.

Clinical Features

- Spontaneous closure:
  - Occurs in 30–40% within first year of life
  - Smaller VSDs have higher chances of spontaneous closure
- Large VSDs:
  - Most patients develop CCF at around 2 months age when PVR reduces to a critical level
  - PVOD may begin at 6–12 months of age
  - The resulting right-left shunt occurs only in teenage years.
- History:
  - Large VSD with or without CCF:
    - Reduced exercise tolerance
    - Recurrent URTI/LRTI
    - Impaired growth and development.
  - Eisenmangers syndrome:
    - Dyspnea on exertion
    - Chest pain, syncope, hemoptysis
    - Cyanosis, clubbing, erythrocytosis.
- Examination:
  - Palpation:
    - Hyperkinetic precordium
    - Systolic thrill at left sternal border
    - LV type of apex.
  - Auscultation:
    - Heart sounds:
      - S_1: Masked by pansystolic murmur
      - S_2: Widely split, increased intensity of P_2
      - S_3: In small left-right shunts.
    - Murmur:
      - *Shunt murmur:* Pansystolic murmur in left third/fourth ICS
      - Flow murmur
      - Pulmonary ejection systolic murmur
      - Mitral delayed diastolic murmur.

Assessment of Severity

- Small VSD:
  - Pansystolic murmur
  - S_2 normally split with normal P_2
  - No delayed diastolic murmur.
- Large VSD:
  - Pansystolic murmur becomes shorter, softer and appears like ESM
  - S_2 widely split, increased intensity of P_2
  - Delayed diastolic murmur present.

Investigations

- Complete blood count:
  - Polycythemia
  - Thrombocytopenia.
- Baseline ABG:
  - For PaO_2 and electrolytes
  - Check K^+ levels if on digoxin therapy
  - Blood glucose for hypoglycemia.
- Renal function tests
- Coagulation profiles:
  - Associated coagulatopathy may be present
  - PT, INR, platelet count, APTT to be checked.
- ECG:
  - *Small VSD:* Normal ECG
  - *Moderate VSD:* LAH, LVH
  - *Large VSD:* LAH, biventricular hypertrophy
  - *Pulmonary vascular occlusive disease:* RVH without LAH/LVH.
- Chest X-ray:
  - *Small VSD:* Normal
  - *Large VSD:* Biventricular enlargement
  - Pulmonary plethora
  - *Moderate VSD:* LA and LV enlargement with pulmonary plethora.
- Echocardiography:
  - Provides definite diagnosis
  - Size and site of VSD identified
  - Direction and magnitude of shunt
  - Color Doppler studies.
- Cardiac catheterization:
  - Location and size of VSD
  - Magnitude of shunt
  - Qp:Qs ratio
  - Pulmonary and systemic vascular resistance
  - Ventricular function.

Treatment

- Medical management:
  - Close follow-up
  - Treat CCF, anemia and recurrent chest infections
  - Congestive cardiac failure (CCF):
    - *Diuretics:* Furosemide 1 mg/kg or 2–3 mg/kg IV or oral.

- *Digoxin:* 20–50 µg/kg PO followed by 8–10 µg/kg maintenance dose
- Reduce by 25% when giving IV therapy.

– Anemia:
- Maintenance of hematocrit is important
- Anemia causes increase in left-right shunt, thereby worsening CCF
- Frequent feeding with high calorie formulas by NGT or oral feeds
- Oral iron therapy can be supplemented.

• Transcatheter closure:
– Indications:
- Mid-muscular/apical VSDs which are difficult to close surgically
- Those which require left ventriculotomy, which is associated with increased chances of LV dysfunction postoperatively.

– Done with *umbrella device*
– Associated with:
- Heart block
- Blood loss
- Hemodynamic instability.

• Surgical correction:
– Usually done between 1–2 years of age
– Increased mortality if done below 6 months of age.
– Indications:
- Significant growth retardation
- CCF unresponsive to medical therapy
- Large VSD with signs of PVOD
- Qp : Qs ≥ 2
- *If associated with CoA or PDA:* Closure of PDA or CoA done first.

– Procedure:
- Approach through RA or RV approach
- Surgical closure done with dacron or pericardial patch repair
- *LV approach seldom used:* LV dysfunction may persist for several years postoperatively
- Distortion of tricuspid valve occurs frequently.

– Contraindications:
- Development of PVOD with right-left shunt
- PVR: SVR ≥ 0.75
- If PVR ≤ one-third of SVR, progression of PVOD in postoperative period is rare.

– PA banding:
- Done very rarely as palliative surgery
- Done if multiple VSDs present (*Swiss cheese* appearance).

Anesthesia for VSD

Anesthetic Goals

- *Rhythm:* Sinus rhythm
- *Rate:* Maintain age appropriate rate
- *Contractility:* Maintain contractility
- *Preload:* Avoid fluid overload, especially if signs of CCF
- *Afterload:* Maintain afterload
- Ventilation:
  – Avoid hypoxia
  – Avoid hypercarbia
  – Avoid hyperventilation and hyperoxemia
  – Avoid hyperinflation and atelectasis
- *Temperature:* Maintain, prevent hypothermia
- *Hematocrit:* Maintain, prevent anemia or polycythemia.

Factors Causing Left-Right Shunt

- Low hematocrit
- Increased SVR, reduced PVR
- Hyperoxemia
- Hyperventilation
- Negative airway pressure.

Factors Causing Right-Left Shunt

- Polycythemia
- Reduced SVR, high PVR
- Hypoxia
- Hypoventilation, hypercarbia
- PEEP.

Preoperative Preparation and Premedication

- Preoperative assessment includes:
  – History of:
    - Symptoms
    - Palliative surgery (PDA/COA)
    - Current medications
    - URTI, immunization
  – Examination should include airway for anticipated difficult intubation
  – Investigation including coagulation profile, ABG and electrolytes.
- *Fasting guidelines:* Avoid hypoglycemia and dehydration
  – 6 hours for solids
  – 4 hours for breast milk
  – 2 hours for clear fluids and water.
- Informed consent
- Treat preoperative URTI/LRTI adequately

- Premedication (in children ≥ 6 months age):
  - *Oral:* Midazolam 0.5 mg/kg with atropine 10 µg/kg
  - *Intramuscular:* Morphine 0.1 mg/kg with atropine 10 µg/kg
  - Avoid premedication in:
    - Children less than 6 months age
    - Those with righ-left shunts.
- Continue all medications except diuretics and anticoagulants
- Infective endocarditis prophylaxis as indicated.

Regional Anesthesia

- Caudal anesthesia:
  - 2 mg/kg bupivacaine + 4 mg/kg lidocaine used
  - Used in noncardiac surgery where applicable
  - Care to prevent reduction in SVR at the time of induction
  - Reduction in SVR causes acute right-left shunting and rapid desaturation
- Contraindicated if coagulation abnormalities present.

OT Preparation

- Anesthesia machine with circuit
- *Airway equipment:* ETT, laryngoscope airways, suction apparatus
- *Anesthetic drugs:* Morphine, fentanyl, NDMR
- Defibrillator, temporary pacemaker
- Equipment for thermal homeostasis
- *Emergency drugs:* Atropine, adrenaline, phenylephrine, dopamine, dobutamine, NTG, isoprenaline.

Monitors

- Pulse oximeter, E_TCO_2
- ECG, NIBP
- Airway pressure monitor
- IBP, CVP, PAC and PCWP as demanded by the procedure
- Left upper extremity not to be used for NIBP/IBP in patients who underwent surgery for CoA
- Foleys catheter, urine output
- Tranesophageal echocardiography
- ABG, electrolytes, blood glucose and hematocrit
- Temperature, precordial stethoscope if noncardiac surgery.

Induction

- Adequate preoxygenation
- Patients with IV line in place:
  - *Good LV function:* Thiopentone 3–4 mg/kg + vecuronium 0.1 mg/kg + fentanyl 2 µg/kg.
  - *Poor LV function:* Ketamine 1–2 mg/kg + vecuronium 0.1 mg/kg.
- Patients with no IV line:
  - Inhalational induction with less than 2% halothane if good LV function
  - IM ketamine 5 mg/kg if poor LV function.
- IV induction is slow in patients with left-right shunt
- Avoid air bubbles in IV line as they will cause paradoxical systemic air embolism in the presence of a right-left shunt.

Maintenance

- If good LV function:
  - O_2 + air + 1% isoflurane
  - Fentanyl 1 µg/kg and vecuronium 0.1 mg/kg intermittent boluses.
- If poor LV function:
  - O_2 + fentanyl 2 µg/kg/hour infusion
  - Vecuronium 0.1 mg/kg and midazolam 0.05 mg/kg intermittent boluses.
- Avoid nitrous oxide as it increases pulmonary HTN and size of air bubbles.

Ventilation

- ET intubation and CMV preferred
- Use low FiO_2 as hyperoxemia increases left-right shunt
- Maintain $PaCO_2$ between 30–40 mm Hg
- Small PEEP preferred as patients have increased pulmonary blood flow
- Avoid:
  - *Hyperventilation and hyperoxemia:* Increases left-right shunt
  - *Hypoxia, hypercarbia and acidosis:* Increases right-left shunt.

Hemodynamics

- Judicious fluids as required if poor LV function
- Avoid anemia as left-right shunt increases (maintain hematocrit ≥ 10 g%)
- Maintain normothermia as hypothermia increases left-right shunt
- Isoprenaline (0.01–0.05 µg/kg/min) or dobutamine (3–5 µg/kg/min) in patients with pulmonary HTN, while coming off bypass
- Pacemaker support if complete heart block post-VSD correction.

Extubation

- Postoperative ventilation required if:
  - Poor preoperative cardiovascular reserve
  - Congestive cardiac failure

- Reversal with neostigmine 0.05 mg/kg and glycopyrollate 0.02 mg/kg
- Extubate when:
  - Patient fully awake
  - Hemodynamically stable
  - Adequately rewarmed
  - Normal acid base status and ABG.

Postoperative Care

Management

- Adequate analgesia to prevent crying
- Avoid factors which precipitate pulmonary hypertension:
  - Crying, stress
  - Hypoxia, hypercarbia
  - Acidosis.

Analgesia

- Titrated opioids to avoid undue sedation
- Patient controlled analgesia
- Ketorolac 0.5 mg/kg or paracetamol 15 mg/kg IV as adjuvants
- Caudal/epidural analgesia where applicable
- Multimodal analgesia preferable.

Monitors

- Pulse oximetry, E_TCO_2
- ECG, NIBP/IBP
- Temperature, urine output
- Echocardiography for LV function and residual VSDs
- CVP, PA pressure.

Complications

- TV regurgitation common, AR rarely
- RVOT obstruction from pericardial patch
- RV/LV dysfunction which persists for years later
- Residual pulmonary HTN/VSD
- Rarely, cerebrovascular accidents and stroke
- Infective endocarditis
- Arrhythmias:
  - Right bundle branch block
  - Right bundle branch block with left anterior hemiblock
  - Complete heart block.

ATRIAL SEPTAL DEFECT[1,2,3,5,17]

Introduction

It is an abnormal communication between the two atria.

Incidence

- OS-ASD accounts for 7–11% of all congenital heart defects
- More commonly seen in girls.

Embryology

- Interatrial septum develops between 4th and 6th weeks of gestation
- A depression is formed in roof of common atrium, which enlarges as a crest
- This forms the *septum primum* (SP)
- SP extends downwards to meet the endocardial (EC) cushions formed in atrioventricular canal
- The opening between the atria formed before the SP meets EC is called ostium primum (OP)
- This is closed when the endocardial cushion fuses with septum primum
- As the septum primum extends downwards, fenestration appear superiorly which ultimately unite together and form the *ostium secundum* (OS)
- At the same time, a thin septum called the septum secundum develops to right of septum primum
- This grows and covers the OS in an incomplete fashion, resulting in the formation of foramen ovale
- After birth, with the increase in SVR and decrease in PVR, foramen ovale usually closes but may persist in 25% causing PFO.

Classification

Sinus Venosus

- ASD situated high in interatrial septum, at the junction of SVC with RA
- Frequently associated with anomalous connection of right pulmonary veins to SVC.

Ostium Secundum Defect

- ASD situated at the center of interatrial septum at the site of foramen ovale
- Situated either at the fossa ovalis, or superior/inferior to fossa ovalis
- OS defect is ten times as common as ostium primum defect
- OS-ASD is the *most common* type of ASD
- When multiple fenestrations of IAS are present in the centered IAS, it is called *Chiari network*
- Occurs due to:
  - Resorption of septum primum
  - Short septum primum
  - Defective septum secundum development.

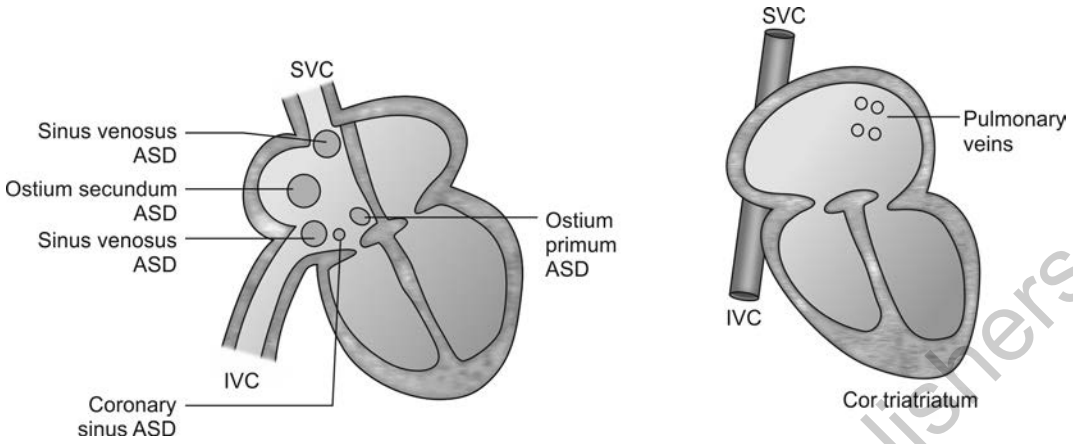


Fig. 7.23: Atrial septal defects

Ostium Primum Defect

- Located at lower end of IAS immediately adjacent to atrioventricular valves
- Due to endocardial cushion defect.
- More common in Down's syndrome
- Associated with cleft in anterior mitral leaflet and mitral incompetence
- Also associated with tricuspid valve incompetence and VSD
- Defect is usually large and manifests early
- Classified as incomplete atrioventricular canal/ partial endocardial cushion defect.

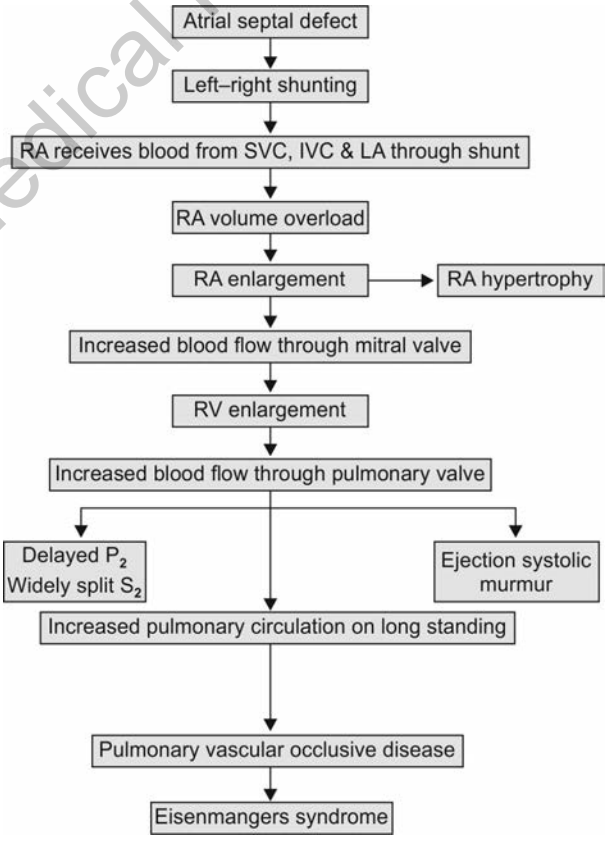
Coronary Sinus Defect

- Defect lies posterior to fossa ovalis, which normally houses the ostium of coronary sinus
- Frequently associated with anomalous connection of right pulmonary veins.

Cor Triloculare Biventriculare

- Called *Common atrium*
- Complete absence of interatrial septum
- Right sided portion of common atrium receives blood from SVC, IVC and coronary sinus
- Left sided portion receives blood from pulmonary veins.
- Commonly associated with:
  - Asplenia and polydactyly
  - ***Ellis-Van Creveld*** syndrome
  - Systemic anomalous venous return
  - Complete or partial anomalous pulmonary venous return.

Pathophysiology



Clinical Features

- History:
  - Majority of patients lead a normal life until adulthood
  - Symptoms of CCF and failure to thrive in 3rd to 4th decades.

- Recurrent chest infections due to increased pulmonary blood flow
- Cyanosis, clubbing, fatigue, dyspnea on exertion
- Precordial pain if pulmonary hypertension and right-left shunt develops
- Mild dyspnea, fatigue are most common early symptoms.

- Examination:
  - Inspection:
    - Skeletal anomalies *(Holt-Oram syndrome)*
    - Facial anomalies (*Down's syndrome*)
    - Visceral anomalies (asplenia)
    - Prominent left costal cartilage from cardiomegaly
    - *Gracile habitus:* Long and narrow bones with poorly developed skeletal muscles.
  - Palpation:
    - Parasternal impulse
    - Systolic thrill in left second ICS
    - Palpable P2.
  - Auscultation:
    - Sounds:
      - S_1: Loud, accentuated
      - S_2: Wide and fixed split, increased P_2 intensity
      - S_3: Inconstant.
    - Murmurs:
      - Shunt murmur absent
      - Flow murmur
      - Delayed diastolic murmur of TR
      - Ejection systolic murmur of PV.
    - S_2 is widely split as increased blood flow through pulmonary valve causes delayed pulmonary valve closure
    - Also, S_2 splitting is fixed and does not vary with inspiration
    - This is because the RV is already fully loaded with shunted blood
    - Thus further inspiration cannot draw more blood into RV.

Complications

- Usually in 3rd to 4th decade
- Congestive cardiac failure
- Eisenmanger syndrome
- Pulmonary vascular occlusive disease.
- Arrhythmias:
  - Atrial flutter
  - Atrial fibrillation, PSVT
  - Atrioventricular conduction abnormalities.

Assessment of severity: If ASD is large:

- Cardiomegaly is more
- Increased intensity of ESM and delayed diastolic murmur.

Investigations

- Complete blood count, renal function tests, coagulation profile
- Baseline ABG and blood glucose
- Chest X-ray:
  - Cardiomegaly: Enlarged RA and RV
  - Pulmonary plethora
  - Localized dilation of SVC at the entrance of anomalous pulmonary veins
- Electrocardiogram:
  - RAD with RVH and RBBB in ostium secundum defects, RVH
  - LAD with RVH in ostium primum defects
- Echocardiogram:
  - Paradoxical anterior ventricular septal motion which contacts with right ventricle rather than LV
  - Location and size of ASD
  - Estimation of left-right shunt
- Catheterization:
  - Not routinely indicated
  - Used only if:
    - Inconsistency in clinical data
    - Significant PAH
    - Associated anomalies
  - Transesophageal echocardiography done if transthoracic echo is ambiguous.

Treatment

- Medical treatment:
  - Consists of treatment of:
    - Recurrent chest infections
    - *Congestive cardiac failure:* Diuretics and digoxin
    - Arrhythmias
  - Spontaneous closure occurs within 25–50% cases within 1st year.
- Surgical treatment:
  - Indications are:
    - Qp:Qs ≥ 2 (ratio of pulmonary to systemic flow)
    - Marked cardiomegaly
    - Marked CCF
    - Retarded growth and development
    - Usually done between 4–6 years of age.

  - Contraindications:
    - Small defect
    - Trivial left-right shunt
    - Severe PAH (as acute RV failure occurs post-operatively).
- Transcatheter closure:
  - Indications:
    - Defects less than 25 mm (8–20 mm)
    - Appropriate sized patients
    - Good interatrial septum length.
  - Devices used are:
    - Clamshell device (Rashkinds double disc device)
    - Angel-Wings ASD device
    - Rashkind umbrella device
    - Sideris buttoned device.
  - Contraindications:
    - Defects more than 25 mm
    - Ostium primum/sinus venous defects
    - Defects associated with anomalous pulmonary venous connection
    - Pulmonary vascular occlusive disease.
  - Complications:
    - Heart block
    - Cardiac erosions
    - Thrombus.

Natural History

- *Small ASDs:* Hemodynamically insignificant, usually symptomless
- Moderate ASDs:
  - May manifest in 3rd–4th decade
  - 14% develop CCF, 20% develop arrhythmias
- *Large ASDs:* Develop PVOD, have reduced life expectancy.

Anesthesia for ASD

Premedication

- Treat preoperative URTI/LRTI adequately
- Informed consent
- Fasting guidelines:
  - *6 hours:* Solids
  - *2 hours:* Clear fluids, water.
- *Premedication:* Morphine 0.1 mg/kg and glycopyrrolate 20 µg/kg IV
- Continue all preoperative medication except diuretics
- Antibiotic prophylaxis as indicated.

Anesthetic Goals

- *Rhythm:* Sinus rhythm
- *Rate:* Maintain age appropriate rate
- *Contractility:* Maintain contractility
- *Preload:* Avoid fluid overload, especially if signs of CCF
- *Afterload:* Maintain afterload
- Ventilation:
  - Avoid hypoxia
  - Avoid hypercarbia
  - Avoid hyperventilation and hyperoxemia
  - Avoid hyperinflation and atelectasis
- *Temperature:* Maintain, prevent hypothermia
- *Hematocrit:* Maintain, prevent anemia or polycythemia.

Factors Causing Left-Right Shunt

- Low hematocrit
- Increased SVR, reduced PVR
- Hyperoxemia
- Hyperventilation
- Negative airway pressure.

Factors Causing Right-Left Shunt

- Polycythemia
- Reduced SVR, high PVR
- Hypoxia
- Hypoventilation, hypercarbia
- PEEP.

OT Preparation

- Anesthesia machine with circuit
- *Airway equipment:* ETT, laryngoscope airways, suction apparatus
- *Anesthetic drugs:* Morphine, fentanyl, NDMR
- Defibrillator, temporary pacemaker
- Equipment for thermal homeostasis
- *Emergency drugs:* Atropine, adrenaline, phenylephrine, dopamine, dobutamine, NTG, isoprenaline.

Monitors

- Pulse oximeter, E_TCO_2
- ECG, NIBP
- Airway pressure monitor
- IBP, CVP, PAC and PCWP as demanded by the procedure
- Foleys catheter, urine output
- Transesophageal echocardiography
- ABG, electrolytes, blood glucose and hematocrit
- Temperature, precordial stethoscope if noncardiac surgery.

Induction

- Adequate preoxygenation
- Patients with IV line in place:
  - *Good LV function:* Thiopentone 3–4 mg/kg + vecuronium 0.1 mg/kg + fentanyl 2 µg/kg
  - *Poor LV function:* Ketamine 1–2 mg/kg + vecuronium 0.1 mg/kg
- Patients with no IV line:
  - Inhalational induction with less than 2% halothane if good LV function
  - IM ketamine 5 mg/kg if poor LV function
- IV induction is slow in patients with left-right shunt
- Avoid air bubbles in IV line as they will cause paradoxical systemic air embolism in the presence of a right-left shunt.

Maintenance

- If good LV function:
  - O_2 + air + 1% isoflurane
  - Fentanyl 1 µg/kg and vecuronium 0.1 mg/kg intermittent boluses
- If poor LV function:
  - O_2 + fentanyl 2 µg/kg/hour infusion
  - Vecuronium 0.1 mg/kg and midazolam 0.05 mg/kg intermittent boluses
- Avoid nitrous oxide as it increases pulmonary HTN and size of air bubbles.

Ventilation

- ET intubation and CMV preferred
- Use low FiO_2 as hyperoxemia increases left-right shunt
- Maintain $PaCO_2$ between 30–40 mmHg
- Small PEEP preferred as patients have increased pulmonary blood flow
- Avoid:
  - *Hyperventilation and hyperoxemia:* Increases left-right shunt
  - *Hypoxia, hypercarbia and acidosis:* Increases right-left shunt.

Hemodynamics

- Judicious fluids as required if poor LV function
- Avoid anemia as left-right shunt increases (maintain hematocrit ≥ 10 g%)
- Maintain normothermia as hypothermia increases left-right shunt.
- Isoprenaline (0.01–0.05 µg/kg/min) or dobutamine (3–5 µg/kg/min) in patients with pulmonary HTN, while coming off bypass
- Pacemaker support especially for ostium primum defects (rare in OS-ASD and SV-ASD).

Extubation

- Early extubation in OT or early postoperative period possible
- Neostigmine 0.05 mg/kg and glycopyrrolate 0.02 mg/kg used for reversal
- Extubate when:
  - Fully awake
  - Hemodynamically stable
  - Adequate rewarmed
  - Normal ABG.

Postoperative Care

Analgesia

- Cautious use of opioids to avoid undue sedation
- Patient controlled analgesia
- Ketorolac 0.5 mg/kg or paracetamol 15 mg/kg IV as adjuvants
- Caudal/epidural analgesia used where applicable
- Multimodal analgesia.

Monitors

- Pulse oximetry, E_TCO_2
- Urine output, NIBP/IBP
- CVP/PAC
- ECG, ABG
- Echocardiography
- Temperature.

Complications

- Postpericardiotomy syndrome (rare)
- Air embolism, cerebrovascular accidents
- SVC obstruction (due to SV-ASD closure)
- Arrhythmias:
  - Transient atrial arrhythmias (Atrila fibrillation and flutter) common
  - Complete heart block occurs in OP-ASD
- Severe brady/tachyarrhythmias rare
- Mitral regurgitation especially in ostium primum.

PATENT DUCTUS ARTERIOSUS[1,2,3,5,17]

Introduction

- The persistence of ductus arteriosus (which is the fetal communication between descending aorta

to pulmonary artery) beyond 3 months after a full term birth is called patent ductus arteriosus.
- Also called *Botallos duct*.

Incidence
- 1 in 2500 full-term normal births
- Premature infants more common, female:male = 3:1
- Incidence increases in congenital rubella syndrome.

Embryology
- Aortic arch develops between 5–6 weeks of gestation from trucus arteriosus as six paired arches
- Pulmonary artery arises from 6th arch
- When pulmonary vascularization is established, the communication with right dorsal aorta completely regresses, while that with left dorsal aorta persists until after birth as ductus arteriosus
- Persistance of this communication beyond 3 months after birth causes PDA.

Anatomy
- PDA has diameter of 5–15 mm and length of 2–15 mm
- Arises from left of PA bifurcation near origin of LPA
- Inserted into lesser curvature of aorta, slightly distal to and opposite to origin of left subclavian artery
- Relations of PDA:
  - *Left main bronclus:* Posteriorly
  - *Vagus nerve:* Anteriorly
  - Recurrent laryngeal nerve encircles ductus and ascends into neck.
- In small children, wall of DA is thick and strong
- In older children, wall is thin and friable especially in those with pulmonary HTN
- Anatomical types of PDA:
  - Cylindrical
  - Window
  - Funnel shaped
  - Aneurysmal which can be spontaneous/acquired from:
    - Mycotic infections
    - Trauma
    - Hypertension.
- Closure of ductus arteriosus:
  - Functional closure of DA begins within 12 hrs after birth
  - Usually is physiologically closed by the second day of life
  - Permanent anatomical closure occurs normally within 1st 2 months of life.

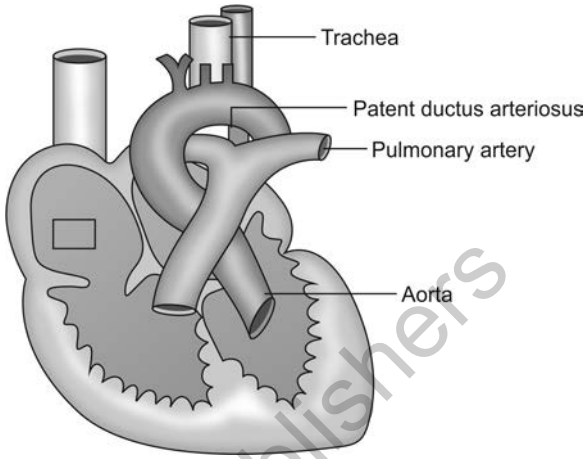
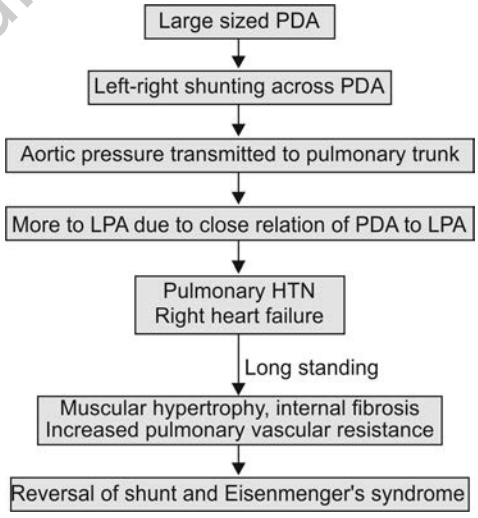


Fig. 7.24: Patent ductus arteriosus

Pathophysiology
- Amount of blood flow through the PDA depends on:
  - Size and shape of communication
  - Difference between SVR and PVR.



Ductus Dependent Cardiac Malformations
- Lesions with restricted pulmonary blood flow:
  - Pulmonary atresia/stenosis associated with:
    - VSD
    - ASD
    - TGA.
  - Tricuspid atresia associated with:
    - ASD
    - VSD.
- Lesions with restricted systemic blood flow:
  - Mitral stenosis with ASD
  - Aortic stenosis with ASD/VSD

- Preductal CoA
- Interrupted aortic arch
- Hypoplastic left heart syndrome.

Clinical Features

Presentation

- Signs of congestive cardiac failure:
  - Failure to thrive
  - Cough, dyspnea, tachypnea
  - Tachycardia
  - Hepatosplenomegaly
- Wide pulse pressure with bounding pulse
- Hyperdynamic precordium
- Systolic and systolic-diastolic murmurs
- *Hallmark:* continuous machinery murmur present in first/second left intercostal space
- *Graham-Steele* murmur:
  - If Eisenmanger syndrome present
  - High pitched diastolic decrescendo murmur
  - Characteristic of pulmonary valvular insufficiency.

Associated Anomalies

- Extracardiac anomalies:
  - Mental retardation
  - Eye defects
  - Deafness
  - Sternal deformities
  - Scoliosis
  - Clubfoot.
- Cardiac anomalies:
  - Lesions with restricted pulmonary blood flow:
    - Pulmonary atresia/stenosis associated with:
      - VSD
      - ASD
      - TGA.
    - Tricuspid atresia associated with:
      - ASD
      - VSD.
    - Lesions with restricted systemic blood flow:
      - Mitral stenosis with ASD
      - Aortic stenosis with ASD/VSD
      - Preductal CoA
      - Interrupted aortic arch
      - Hypoplastic left heart syndrome.
- Syndromes:
  - *Charge syndrome:* Iris coloboma, heart, choanal atresia, micrognathia, difficult airway
  - *Edwards syndrome:* Trisomy 18, micrognathia, small mouth, difficult airway
  - *Goldenhaar syndrome:* CoA, maxillary/mandibular hypoplasia, spine abnormalities
  - *Vater syndrome*
  - *Pataus syndrome:* Trisomy 13.

Differential Diagnosis

- Venous hum
- VSD with aortic regurgitation
- Systemic arterio-venous fistula
- Pulmonary arteriovenous fistula
- Coronary arterial fistula
- Ruptured sinus of Valsalva.

Investigations

- Complete blood count, electrolytes
- Coagulation profile including PT, APTT, platelet count
- Serum protiens, calcium levels
- Arterial blood gas, urine analysis, urine specific gravity
- X-ray chest:
  - Normal or biventricular hypertrophy
  - Enlargement of LA, LV, ascending aorta and aortic arch
  - Enlarged pulmonary vasculature
- *ECG:* Normal or LVH/biventricular hypertrophy
- ECHO:
  - Dilatation of left heart structures
  - Increased LVEF due to arteriovenous shunting
- *Catheterization*: Step up in oxygen saturation at PA level
- Color doppler can viaualize jet of abnormal blood flow.

Medical Management

- Patients with congestive cardiac failure:
- Fluid restriction
- *Furosemide*: May promote patency of DA due to its effects on prostaglandin metabolism
- Digoxin:
  - No benefit in children < 1250g as it does not increase SVR
  - But it may reduce heart rate and thus decrease cardiac output
- Dopamine
- Role of prostaglandins:
  - PGE_1, $PGF_{2\alpha}$ and PGI_2 are used to keep the ductus patent in ductus dependent lesions

- Done in order to promote flow through pulmonary artery
- PGE_1 infused with starting dose of 50–100 ng/kg/min
- Dose adjusted to 10–20 ng/kg/min after favorable effect is seen.
- Side effects of prostaglandins:
  - Fever
  - Hypotension
  - Cutaneous flushing
  - Tachycardia
  - Apneic spells
  - Seizures.

Closure of Ductus Arteriosus

- Indomethacin:
  - Reduces synthesis of prostaglandins by inhibiting cyclooxygenase
  - Indomethacin 0.2 mg/kg IV three times over 24 hours or daily for 72 hours
  - Associated side effects like:
    - Increased bleeding time
    - Renal and hepatic insufficiency
    - Sepsis
    - Necrotizing enterocolitis.
- *Ibuprofen:* Useful and has less effects on organs also
- *Transcatheter closure:* 3 types of occlusion devices:
  - *Porstmann's Ivalon plug*
  - *Rashkinds double disk*
  - *Sideris button device*
  - Close to 90% success rate.
- Surgical closure:
  - Either by division/ligation of PDA
  - *Thoracoscopic technique:* Less pain, can be discharged on same day.

Effects of Various Drugs on Ductus Patency

- Drugs causing PDA contraction:
  - *Increased* PaO_2*:* Hyperoxia
  - $PGF_{2\alpha}$, Indomethacin, aspirin
  - Acetylcholine, Succinylcholine, histamine
  - NSAIDs
  - Norepinephrine.
- Drugs causing PDA relaxation:
  - PGE_1, PGE_2
  - Hypoxemia, acidosis
  - High altitude
  - Hypothermia.

Anesthetic Considerations

- *Avoid hypothermia*: Causes PDA relaxation and increased left-right shunting
- Avoid hemodilution
- Cross matched blood must be available as blood vessel may be damaged during ligation
- Postoperative ventilation may be required if heart failure is present
- Left lateral thoracotomy approach
- Avoid hypoxia/hyperoxia:
  - *Tidal volume:* Adjusted to keep peak inspiratory pressure between 15–25 cm H_2O
  - E_TCO_2*:* Maintain between 30–35 mm Hg
  - SpO_2*:* Maintained between 87–92%
  - PaO_2*:* Maintained between 50–70 mm Hg.

Preoperative Evaluation

History

- Birth trauma
- Respiratory and cardiovascular status
- History of maternal drug intake
- Fluid status.

Examination

- Airway examination
- Fluid status:
  - Skin turgor
  - Mucous membrane
  - Anterior fontanelle
- Cardiovascular and abdominal examination.

Investigations

- Complete blood count, arterial blood gas
- Electrolytes, coagulation profile
- Urine analysis, urine specific gravity
- X-ray chest and abdomen.

Preoperative Medications

- Informed consent
- Orally hydrated till 2 hrs before surgery
- If this is not possible, IV hydration to be started
- Judicious fluid administration to prevent overload
- Continue inotropic support if present
- Children under 6 months may not require any preoperative sedation
- Midazolam 0.5–0.6 mg/kg PO 30 minutes before surgery.

Induction

- Adequate preoxygenation as apnea and bradycardia can occur
- Prolonged onset time of IV agents with no change if inhalational induction
- *If IV line present:* Ketamine 1 mg/kg + glycopyrrolate 20 µg/kg + vecuronium 0.1 mg/kg IV
- If no IV line present:
  - Halothane/sevoflurane induction
  - Start IV line (largest possible)
  - Fentanyl 1µg/kg+vecuronium 0.1 mg/kg + glycopyrrolate 20 µg/kg
- Avoid succinylcholine as it causes contracture of ductus.

Position

Right lateral decubitus as left posterolateral thoracotomy has to be done

Monitoring

- ECG
- Pulse oximeter in right hand
- Invasive BP monitoring right hand
- End tidal CO_2, inspiratory pressure gauge
- Urinary bag, precordial/esophageal stethoscope
- Esophageal/axillary temperature probe
- Doppler transducer, arterial blood gas monitoring.

Maintenance

- Halothane + NDMR + fentanyl 10 µg/kg or sufentanil 1.5 µg/kg
- N_2O not to be used if pulmonary hypertension is present.

Hemodynamics

- Fluid therapy:
  - Maintenance with 5% dextrose in 1/4th strength NS
  - Maintenance fluids at 4 ml/kg/hr with added 3rd space and blood loss
  - IV tubing to be bubble free to prevent embolization
  - *Maintain hematocrit:* Hemodilution associated with an increase in left-right shunt
  - Replace blood loss with PRBCs or 1:3 crystalloid if hematocrit > 30%.
- Watch for:
  - Bradycardia while handling the ductus
  - Systolic hypotension at the time of ligation of the ductus.
  - Abrupt increase in DBP and mean BP with interruption of DA
  - Increased DBP due to the elimination of low resistance pulmonary circulation.

Ventilation

- Continuous mandatory ventilation (CMV) done with pediatric bellows
- Ventilatory goals:
  - Tidal volume adjusted to keep peak inspiratory pressure between 15–25 cm H_2O
  - FiO_2 adjusted to keep PaO_2 between 50–70 mm Hg
  - Maintain SpO_2 between 87–92%
  - High FiO_2 avoided to prevent retinopathy of prematurity
  - E_TCO_2 to be maintained between 30–35 cm H_2O
- Hypoventilation:
  - Causes hypoxic pulmonary vasoconstriction
  - Reverses shunt to right-left shunt
- Hyperventilation:
  - Reduces pulmonary vascular resistance
  - Causes an increase in left-right shunt
- Lung compliance is reduced by lung retraction and packing during the procedure
- May be hand ventilated with *Jackson-Rees* circuit to:
  - Compensate for changes in pulmonary compliance during thoracotomy
  - Allow adequate field exposure during surgery
  - Deairing pleural cavity before closure.

Intraoperative Complications

- Tear/avulsion of ductus arteriosus with profuse bleeding
- Bradycardia while handling ductus arteriosus
- Inadvertent left pulmonary artery/aortic ligation
- Phrenic nerve/recurrent laryngeal nerve injury
- Trauma to thoracic duct
- Left lung injury from pressure and retraction of lung
- Bacterial endocarditis
- Systemic embolization from calcification of aorta
- Residual shunt with ligation
- Postoperative aneurysm
- *Postoperative hypertension is very common.*

Extubation

- Fully awake extubation in lateral decubitus position
- Majority extubated on table/soon after surgery
- Only sick children require postoperative ventilation
- Done after full reversal with neostigmine and atropine.

Postoperative

Management

- Maybe considered to have normal cardiovascular system postoperatively
- Do not require routine infective endocarditis prophylaxis for subsequent surgeries.

Analgesia

- Local anesthetic infilteration
- Intrapleural block
- Thoracic epidural
- Opioid analgesia
- Paracetamol rectal suppository 30–60 mg/kg.

Complications

- Hypertension related to baroreceptor dysfunction
- Recurrent laryngeal nerve injury
- Phrenic nerve injury
- Systemic thromboembolism
- Reduced systemic blood flow causing:
  - Renal dysfunction
  - Necrotizing enterocolitis due to gastrointestinal hypoperfusion.

COARCTATION OF AORTA[1,2,3,5,17]

Introduction

Congenital constriction of the aorta which impedes the flow of blood below the constriction and increases the blood pressure above the constriction.

Incidence

- Occurs in approximately 7–9% of patients with congenital heart disease
- Most common near ductus arteriosus but also occurs in lower thorax and abdominal aorta.

Embryology

- Aortic arch normally arises from left 4th arch of primitive arches of truncus arteriosus between 5th and 7th weeks of gestation
- Aorta proximal to site of ductal insertion, arising from left 4th arch is the *most common* site of CoA (called preductal CoA)
- Anomalous development occurs from 6th primitive aortic arch to the point of fusion of the two dorsal aortae resulting in post ductal CoA
- Aortic luminal diameter maybe reduced to as little as 1–3 mm.

Etiology

- Intrusion of ductus arteriosus closure into adjacent normal aorta
- Extension of ductal muscle into aortic wall
- Primary aortic malformation/hypoplasia.

Classification

- Thoracic aortic CoA:
  - Preductal/infantile CoA
  - Ductal/juxtaductal CoA
  - Postductal/adult CoA
- Lower thoracic CoA
- Abdominal aortic CoA.

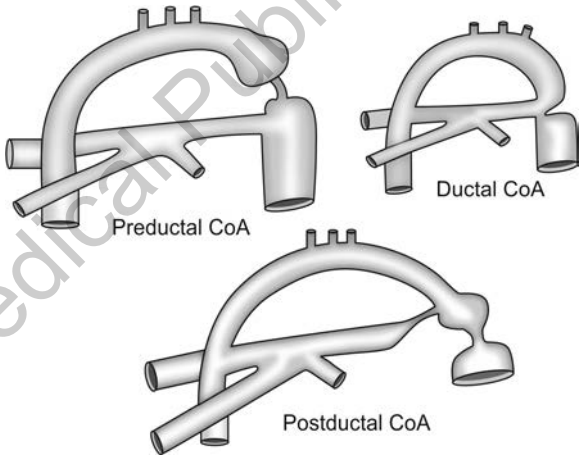


Fig. 7.25: Types of coarctation of aorta

Pathophysiology

Preductal

- Little or no collateral circulation as perfusion of lower body occurs via ductus arteriosus
- Systemic pressures in RV ensures perfusion of ascending aorta and lower body of neonate with preductal CoA, so long as ductus arteriosus remains patent
- Thus, no pressure gradient, upon which development of collateral circulation depends ever occurs
- When the DA closes, these neonates develop CCF, shock, oliguria and CRF.

Postductal

- Pressure gradients between 30–40 mm Hg develop which stimulates collateral circulation development
- Thus, closure of DA has lesser effect on patients with postductal CoA than those with preductal CoA
- Often remains asymptomatic into their teenage/later, in contrast to those with preductal CoA.

Collateral Circulation

- Intercostal artery
- Internal mammary artery
- Internal thoracic artery
- Subdivision of subclavian artery
- Superior intercostal except for first 2 pairs
- Superior epigastric artery
- Perhaps anterior spinal and vertebral artery
- Patients with collaterals are less likely to develop paraplegia following aortic cross clamping.

Clinical Presentation

Symptoms

- Present early if preductal CoA
- Most common cause of CCF between 1–3 weeks age (later in teens if postductal CoA)
- Tachycardia, tachypnea, dyspnea, poor feeding, weight gain, signs of CCF within first 3 months of life.

Signs

Vitals

- Pulses in lower limb is delayed/diminished/absent
- Pulse in upper limb is bounding
- BP decreased in lower limb
- BP abnormally increased in upper limb which may exceed lower limb by ≥ 20 mm Hg
- Localization of lesion:
  - CoA located at aortic isthmus when pulse felt in:
    - Right arm
    - Right carotid artery
    - Left arm but absent on left side
  - CoA is proximal to or adjacent to its subclavian artery take off when pulse is:
    - Present in right arm
    - Absent in left arm and legs
    - Or right arm BP is more than left arm BP
  - If pulse is palpable in left arm but not right upper arm/leg, CoA is distal to left subclavian artery in association with rare anomalous right subclavian artery with original distal to CoA.

Associated Lesions

- More common with preductal CoA
- VSD, PDA, TGA in 40% patients with CoA
- MS, MR, ASD, endocardial fibroelastosis and bicupid aortic valve (very common) may coexist.

Others

- Differential cyanosis of lower half of body:
  - Upper half receives well oxygenated blood from vessels originating from aortic arch above level of CoA
  - But lower half of body is poorly perfused with lower saturation blood from RV via PA and PDA
  - Tissue extraction is high relative to local tissue perfusion and blood oxygen content, from blood in lower half of body, causing cyanosis
- Systolic thrill may be palpable in suprasternal notch area
- Auscultation:
  - S_3 gallop, diffuse ejection systolic murmur
  - Systolic ejection murmur grade 2–3 radiating over right upper sternal border, left sternal border/occasionally posteriorly in interscapular area left of midline
  - Ejection click of bicuspid aortic valve
  - Diastolic murmur of aortic regurgitation
- Metabolic acidosis and oliguria/anuria due to CCF
- *Suzman's sign*:
  - Pulsation in the back due to collateral
  - Most prominent over scapula
  - Best visualized with the patient bending forwards.

Investigations

- ECG:
  - Left Ventricular Hypertrophy with Left Axis Deviation
  - Normal ECG in some patients with postductal CoA
  - Right Ventricular Hypertrophy with Right Bundle Branch Block.
- Chest X-ray:
  - Heart size normal or slightly enlarged with or without ascending aortic dilation
  - In overpenetrated chest films, *3 sign*, representing pre and poststenotic dilation
  - Reverse 3/E sign:
    - On left anterior oblique projection of barium swallow
    - Shows anterior displacement of esophagus
    - Poststenotic aortic dilation will cause an indentation, making it look like E.
  - Pulmonary edema and pulmonary venous congestion

  - Inferior rib notching:
    - Occurs on ribs 4 to 9
    - *Pathognomonic* of collateral circulation
    - Is not seen in infancy and patients < 5 yrs age.
- ECHO:
  - Shelf like membrane in decending aorta from suprasternal notch view
  - Associated defects like VSD/bicuspid aortic valve
- Cardiac catheterization:
  - Indicated when:
    - Information from ECHO is incomplete
    - Concern over collateral flow that may affect the planning of operative repair.
  - Studies pressure differential between ascending and decending aorta with significant pressure drop distal to the site of CoA
  - Confirms VSD, TGA, bicuspid aortic valve.

Treatment

Medical Therapy

- Oxygen therapy, digitalization, diuretics and intropic support for children with CCF
- Prostaglandin E_1 to maintain ductal patency if systemic perfusion depends on right-to-left shunt.

Surgical Therapy

- Usually planned between 4–8 yrs of age
- Resection and anastomosis:
  - *Surgery of choice* when feasible
  - Up to 5 cm can be excised in neonates and infants
  - Only 2.5 cm can be excised in older children and adults as progressive fibrosis reduces elasticity.
- Alternatives:
  - Subclavian artery flap
  - Angioplasty
  - Dacron patch repair
- Prosthetic graft *mandatory* for adult CoA repair.

Anesthetic Management

Preoperative Preparation and Premedication

- Evaluate:
  - Patients physical status
  - Associated cardiac and other medical problems
  - Whether patient has been optimally treated to put patient in best physical condition for surgical stress.
- NPO orders
- Informed consent
- Sedation:
  - Inappropriate as a rule for child < 6–9 months age
  - Sedation for other patients individualized as per needs
  - 0.5–0.7 mg/kg midazolam orally within 15–30 min of induction
  - Better to give sedation in holding area of operating room.
- Temperature:
  - Maintain thermally neutral environment in transfer of patient from hospital room to OR
  - Done so that there is no additional stress on heart due to change in SVR from vasoconstriction/shivering
  - Passive measures like a clear plastic wrap/active methods like warming blankets may be used.
- *Antisialagogue*: Anticholinergic premedication like atropine useful
- Antibiotic chemoprophylaxis:
  - Against gram-positive organisms and common gram-negative organisms
  - Done for:
    - All patients with bicuspid aortic valve
    - Other lesions predisposing to bacterial endocarditis.

Monitoring

- Blood pressure:
  - Left arm cuff not used as left subclavian artery may be clamped/used for surgical repair of CoA
  - Right arm BP cuff can provide valuable information regarding cerebral circulation during periods of left subclavian artery occlusion
  - BP cuff on patients thigh/calf to measure BP gradient between upper and lower limb before and after repair
  - Invasive BP if aorta will be cross clamped at any level - *Right radial artery only*
  - Umbilical artery catheterization may be helpful but it may give very low or no readings at all during aortic cross clamping.
- Pulse oximeter probe on right arm
- ECG lead placement to take into account left lateral thoracotomy approach and right lateral decubitus position
- Central Venous Pressure monitoring.

Induction and Intubation

- Techniques of induction:
- In hemodynamically stable patients with good LV reserve:
  - Thiopentone 3–5 mg/kg with succinylcholine 1.5–2 mg/kg after atropine 5–10 µg/kg
  - Defasciculating dose of NMBA preferred
- In patients with suspected/known reduction in LV function:
  - Rapid IV induction with etomidate 0.15–0.3 mg/kg or ketamine 1–2 mg/kg and vecuronium 0.1 mg/kg
  - Combined benzodiazepine with narcotic induction with midazolam 0.1–0.3 mg/kg and fentanyl 5–20 µg/kg or sufentanil 0.5–1.5 µg/kg with vecuronium 0.1 mg/kg.
- In patients with no intravenous access inhalational induction with halothane to <1.5 MAC as risk of:
  - Bradycardia
  - Severe myocardial depression
  - Complete heart block.
- High arterial oxygen pressures and succinylcholine avoided as if patient has ductus dependent physiology as it may reduce flow through PDA.
- Debubbling is done scrupulously to avoid possibility of right-to-left shunting of air bubbles and peripheral embolization
- Ketamine and other agents which increase SVR to be avoided in patients with bicuspid aortic valve and significant AS/AR
- Left-to-right shunting may cause slight decrease in rate of IV induction
- Right-to-left shunting may increase rate of inhalational induction.

Maintenance

- Primarily narcotic based in patients with depressed myocardial function with:
  - Fentanyl 10–25 µg/kg or sufentanil 1–2.5 µg/kg total dose
  - Low dose inhalational agent used to maintain balanced anesthesia.
- Avoid N_2O due to:
  - Potential for myocardial depression in those getting narcotics
  - Also causes enlargement of air bubbles.
- Neuromuscular blockade with vecuronium/pancuromium.

Hemodynamics

- Ductal manipulation may cause bradycardia:
  - Atropine should be immediately available
  - Manipulation of aorta should be suspended temporarily.
- Avoid left-to-right shunting by preventing:
  - Respiratory alkalosis
  - Hyperoxia
  - Excessive reduction in hematocrit
  - Reduction in pulmonary vascular resistance
  - Increases in systemic vascular resistance.
- In patients with VSD:
  - Avoid hypervolemia to avoid increase in left-to-right shunting
  - Avoid increase in pulmonary artery pressure and pulmonary vascular congestion.
- Dextrose free fluids used except in neonates
- Blood:
  - Given judiciously to avoid reduction in oxygen delivery
  - Administered at rates not likely to provoke MI/cardiac failure.
- Coagulation products generally not required in non-CPB cases.

Postoperative

Postoperative Ventilation

- Healthy older individuals can be extubated early in postoperative period
- Delayed weaning and extubation for:
  - Sicker patients with limited cardiopulmonary reserve
  - Preoperative heart failure.
- Prior to weaning, check:
  - Patient must be rewarmed to a normal body temperature
  - Fluid, electrolytes and coagulation status
  - Hemoglobin levels
  - Recovery of muscle strength and CNS status.

Complications

- Paraplegia:
  - Due to:
    - Ischemia of thoracic spinal cord from prolonged cross clamping
    - Spinal cord HTN below the level of aortic cross clamping causing cord infarction

- Hyperthermia to 38–40 °C during cross clamping.
  - Maintain distal aortic press > 60 mm Hg to reduce incidence of neurological injury
  - Spinal cord infarction is rare if aortic cross clamp time is < 20 mins
  - If cross clamp time > 20 mins, consider placement of a temporary shunt
  - Hypothermia may offer cord protection by reducing spiral cord oxygen requirements and consumption.
- Hypertension:
  - Most patients develop HTN in immediate postoperative period (within 24 hrs)
  - Occurs due to:
    - Increased catecholamine levels, especially norepinephrine
    - Increased plasma renin acitivity
  - β blockers and ACE inhibitors may be used to treat HTN
- Transient renal failure
- Postcoartectomy syndrome:
  - Necrotizing mesenteric arteritis presenting with:
    - Ileus
    - Abdominal pain and distension
    - Melena, vomiting
    - Fecal incontinence and bowel necrosis
  - Less common now due to better control of postoperative HTN.

Natural History

- 50% mortality in first month of life
- ≥ 80% mortality in first 3 months of life
- With surgical correction, mortality reduces to 5%
- For post ductal CoA, average prognosis for survival is about 35 yrs
- Fatality of 60–70% by age 40 yrs due to:
  - Rupture of aorta
  - Congestive cardiac failure
  - Bacterial endocarditis
  - Intracerebral bleed.

MITRAL REGURGITATION[1,2,3,5,17]

Introduction

Mitral regurgitation results from several mechanisms permitting systolic blood flow from left ventricle, back into left atrium.

Causes

No	Cause	Characteristics	Location
Functional mitral regurgitation			
1	Annular dilatation	Dilated cardiomyopathy	Annulus
2	LV ischemia	Ischemic Heart Disease	Tensor apparatus
Organic mitral regurgitation			
Chronic Mitral Regurgitation			
1	Congenital	Cleft mitral valve	Leaflet
		Double orifice mitral valve	
2	Mitral prolase	Myxomatous degeneration	Leaflet
		Redundant tissue	
		Ruptured chordae	
3	Rheumatic	Thickened leaflets	Leaflet
		Calcified leaflets	
		Commissural fusion	
4	Miscellaneous	Fenfluramine	Drug related
Acute mitral regurgitation			
1	Endocarditis	Perforated leaflets	Leaflet
		Vegetations	
2	Post MI	Papillary muscle rupture	Tensor apparatus
3	Post chest trauma	Papillary muscle rupture	Tensor apparatus

Pathophysiology

- Chronic mitral regurgitation:
  - Regurgitation of blood into LA occurs during systole
  - On long standing, the LA dilates, making it more compliant
  - The massively dilated LA, protects the pulmonary capillaries from experiencing significant pressure elevation
  - Thus, pulmonary edema is an uncommon initial presentation in chronic MR.
- Left ventricular ejection fraction in chronic MR:
  - Most often normal or supranormal
  - This is because the LA serves as a low pressure pathway during systolic ejection
  - Thus ejection fraction overestimates the ventricular function
  - Thus, ventricular dysfunction may be unmasked after valve repair/replacement.
  - LVEF becomes low when:
    - LV has decompensated with chronic MR
    - Acute LV ischemia.

- Acute onset mitral regurgitation:
  - LA pressure increased as there is no time for LA compensatory changes to occur
  - *v wave* may be present on LAP/PAP/PCWP recordings
  - In contrast, in chronic MR, the increase in LAP is less dramatic
  - This is due to compliance changes in LA as a function of chamber dilation.
- Determinants of volume of mitral regurgitation:
  - LA–LV pressure differential depends on:
    - Systemic vascular resistance
    - LA compliance
  - Regurgirant volume reduced with:
    - Decrease in systemic vascular resistance
    - Increase in LA pressure.

Clinical Features

- May remain asymptomatic for 20–40 yrs
- If acute MR:
  - Have normal or reduced LA compliance
  - Usually have pulmonary vascular congestion and severe pulmonary edema
  - Congestive heart failure.
- If chronic MR:
  - Have increased LA compliance
  - Usually show signs of low cardiac output and forward LV failure
  - Dyspnea on exertion
  - Paroxysmal nocturnal dyspnea
  - Atrial fibrillation.
- Features of right heart failure:
  - Hepatic congestion and ascites
  - Pedal edema and anasarca
  - Raised JVP (if acute MR).
- Examination:
  - Normal BP, arterial pulse with sharp upstroke
  - Raised JVP with:
    - Prominent *a waves* in patients with sinus rhythm
    - Prominent *v waves* if severe tricuspid regurgitation.
  - Systolic thrill at apex
  - Hyperdynamic LV with brisk systolic impulse
  - Apex beat displaced laterally
  - S_1 absent, wide splitting of S_2.
  - Mitral regurgitation murmur:
    - Holosystolic murmur at apex, radiating to axilla
    - Increased intensity with squatting/hand grip
    - Reduced intensity with valsalva maneuver
    - If acute MR, seagull /cooing quality of murmur.
- Complications:
  - Chest pain due to low diastolic pressure
  - Infective endocarditis
  - Arrhythmias
  - Embolic events.

Investigations

- Complete blood count, BUN, SC, LFT
- Chest X-ray:
  - LA and LV are dominant chambers
  - LA might form right border of cardiac silhouette if massively enlarged
  - Calcification of mitral valve
  - *Kerley A, Kerley B lines, Stag antlers signs* if pulmonary edema.
- ECG:
  - Normal in some
  - Inverted or biphasic T waves
  - ST segment changes in inferior leads
  - Arrhythmias:
    - *Supraventricular tachyarrhythmias most common*
    - Bradyarrhythmias
    - AV bypass tract especially with mitral valve prolapse (*Barlows syndrome*)
  - Biatrial enlargement features.
- Echocardiography:
  - Regurgitant fraction < 30% of total stroke volume: Mild symptoms
  - Regurgitant fraction 30–60% of total stroke volume: Moderate symptoms
  - Regurgitant fraction > 60% of total stroke volume: Severe symptoms.

Carpentier Classification of Mitral Regurgitation

Carpentier type	Leaflet motion	Jet direction
1	Normal	Central
2	Excessive (prolapsed, flail)	Away from lesion
3a	Restricted, structure is abnormal	Variable
3b	Restricted, structure is normal	

- Flow volume curve
- *Transesophageal echocardiography*: Classification of MV leaflet motion.

Treatment

Medical Therapy

- Digoxin
- Diuretics
- Vasodilators which cause reduction in SVR resulting in:
  - Increased forward stroke volume
  - Reduced regurgitant volume
- Afterload reduction maybe life saving in acute MR.

Surgical Therapy

- Considered for patients with moderate to severe symptoms
- Surgical valvuloplasty is preferred, to avoid problems associated with valve replacement like:
  - Thromboembolism
  - Hemorrhage
  - Prosthetic failure.

Anesthetic Goals

- Faster, fuller and vasodilated to maintain forward flow
- *Rate*: maintain slightly higher range (80–100 bpm)
- Rhythm:
  - Maintain sinus rhythm
  - If atrial fibrillation present, control ventricular rate.
- Preload:
  - Maintain preload
  - Increase in preload worsens MR.
- *Afterload*: reduced with anesthetics and vasodilators
- Contractility:
  - May be depressed
  - Titrate myocardial depressants carefully
- M_vO_2: Compromised if MR coexists with IHD.

Rationale for Anesthetic Goals

Heart Rate and Rhythm

- Maintain sinus rhythm in high normal range (80–100 bpm)
- Bradycardia has dual detrimental effects:
  - *Increased duration of systole:* Prolongs regurgitation
  - *Increased diastolic filling interval:* More LV distension.

Preload

- Maintain preload
- Mitral regurgitation is a dynamic condition
- Ventricular distension may cause expansion of an already dilated MV annulus
- This may worsen MR.

Afterload

- Lower SVR so that forward cardiac output is maximum
- Afterload reduction strategies:
  - Maintain adequate anesthetic depth
  - Systemic vasodilators and inodilators
  - Mechanical reduction of afterload with IABP
- Temporary use of ephedrine after which inotropic support needed to augment pressures
- Arteriolar dilators are effective as they reduce LA-LV gradient.

Contractility

- EF indices poorly correlate with LV systolic function
- Manage hypotension by manipulating heart rate and preload
- Persistent hemodynamic instability common inspite of adjusting heart rate and preload
- Consider inotropes for hemodynamic instability:
  - Dobutamine
  - Low dose epinephrine
  - Milrinone
- EF correlates poorly with LV function as it includes both forward systolic flow and regurgitant volume.

Pulmonary Hypertension

- Avoid increases in pulmonary HTN
- Pulmonary artery pressure and pulmonary vascular resistance is already raised
- Avoid hypoxia, hypercarbia and acidosis.

Choice of Anesthetic Technique

- Spinal/epidural anesthesia well tolerated as it reduces the SVR causing reduction in regurgitant fraction
- Bradycardia associated with spinal anesthesia should be avoided as it increases the regurgitant volume.

Preoperative Preparation and Premedication

- Informed consent
- NPO orders

- Premedicate:
  - Premedicate with caution
  - Light premedication advised to prevent airway obstruction
  - Hypoventilation and asphyxia may exacerbate pulmonary HTN
  - Done in high dependency room under supervision
  - 0.5 mg midazolam IV titrated and given in increments
  - Supplemental oxygen can be given at this time
- *Blood loss is usually moderate*: Arrange for 2 units of blood
- Continue antiarrhythmics until day of surgery
- Change warfarin to heparin preoperatively.

Anesthetic Management

Induction

- Opioid based induction:
  - Oxygen + fentanyl 5 µg/kg
  - Pancuronium may be used as muscle relaxant to avoid opiod induced bradycardia
- Acute increases in LV afterload, as after intubation and surgical stimulation to be avoided
- Deep planes during intubation advised
- NTG may be useful to control increases in afterload.

Position

Supine position.

Monitoring

- Pulse oximetry, capnography
- Invasive BP, ECG
- Temperature, urine output
- Transesophageal echocardiography
- Pulmonary artery catheter recording:
  - Very useful, especially for those on vasodilator therapy
  - MR recognized on PA wedge waveform as large *v wave with rapid y descent*
  - Especially prominent in acute MR or chronic MR with acute deterioration
  - Used for:
    - Assessment of intravascular filling
    - Measurement of cardiac output
    - Evaluation of effect of treatment
  - Suspect functional TR if raised PAP occurs.

Maintenance

- Patients with moderate to severe LV dysfunction are very sensitive to the depressant actions of volatile anesthetics
- Opioids + oxygen + isoflurane used
- Isoflurane used though it causes myocardial depression as it also reduces systemic vascular resistance
- Avoid N_2O as it:
  - Increases pulmonary arterial pressure
  - Causes LV dysfunction.

Ventilation

- Avoid factors causing increase in pulmonary arterial pressure:
  - Hypoxia
  - Hypercarbia
  - Acidosis
  - *High airway pressures:* Might constrict pulmonary capillaries and increase PAP
  - Positive end expiratory pressure.

Hemodynamics

Maintain Preload

- Judicious fluid administration
- Avoid fluid overload
- In case of hypotension: (especially acute MR)
  - Administer fluids
  - Inotropic support is rarely required prebypass
  - Avoid vasoconstrictors
  - IABP useful to reduce afterload and increase cardiac output.

Maintain SVR

- NTG especially useful if any increase in SVR/PAP occurs
- Select agents which promote vasodilatation and tachycardia
- Replacement of mitral valve in acute MR is difficult as LA would be small
- Amrinone and milrinone useful as inodilators if RV failure occurs
- *Paradoxical deterioration:*
  - Occurs postmitral valve repair soon after starting inotropes and vasodilators
  - This is due to coexistent HOCM and SAM of mitral valve
  - If this is suspected a trial of volume expansion and vasoconstrictors is given.

Reduce Pulmonary Vascular Resistance

- Avoid hypoxia and hypercarbia
- Check ABGs regularly
- Avoid acidosis
- Pulmonary vasodilatation if PAP > 2/3rd the systemic pressures with:
  - Nitric oxide 40 ppm is the agent of choice
  - Milrinone, amrinone, sildenafil, exoximone are alternatives
  - Inhaled PGE_1:
    - Dilates pulmonary smooth muscle
    - Almost complete first pass metabolism occurs in pulmonary endothelium
    - Therefore it is pulmonary selective vasodilator.

Extubation

- Early extubation avoided to prevent hypoxia and hypercarbia
- Postoperative ventilation may be required
- Extubate if:
  - Spontaneous ventilation with normal ABG
  - No bleeding and hemodynamically stable patient
  - Normothermia.

Postoperative Period

Complications

- Postoperative LV dysfunction, RV dysfunction and biventricular failure
- Paravalvular leaks
- Transverse midventricular disruption
- Posterior wall MI
- Coagulopathy and postoperative bleeding
- Hypothermia from incomplete and non-uniform rewarming
- Pulmonary HTN.

Monitors

- ECG, pulse oximetry
- Invasive BP
- Urine output, CVP
- PA catheter
- ABG, temperature.

Analgesia

- Severe pain common
- *Opioids*: Fentanyl or morphine
- Intercostal nerve block
- Patient controlled analgesia.

HYPERTROPHIC CARDIOMYOPATHY[1,2,3,5,17]

Introduction

- HCM is a genetically determined disease characterized by histologically abnormal myocytes and myocardial hypertrophy developing in the absence of a pressure/volume overload
- Also called:
  - *Idiopathic hypertrophic subaortic stenosis*
  - *Asymmetrical septal hypertrophy*
  - *Hypertrophic obstructive cardiomyopathy*
  - *Muscular subaortic stenosis*.

Etiology

- Autosomal dominant inheritance with variable penetrance
- Mutation of cardiac β myosin chain gene on chromosome 14
- This gene codes for cardiac sarcomere proteins
- Mutation results in hypertrophy of segments.

Pathology

- Bizarre and disorganized arrangement of cardiac muscle cells
- Disorganized myofibrillar architecture
- Myocardial fibrosis and thickening of small intramural coronary arteries
- Systolic compression of intramyocardial segment of coronary artery.

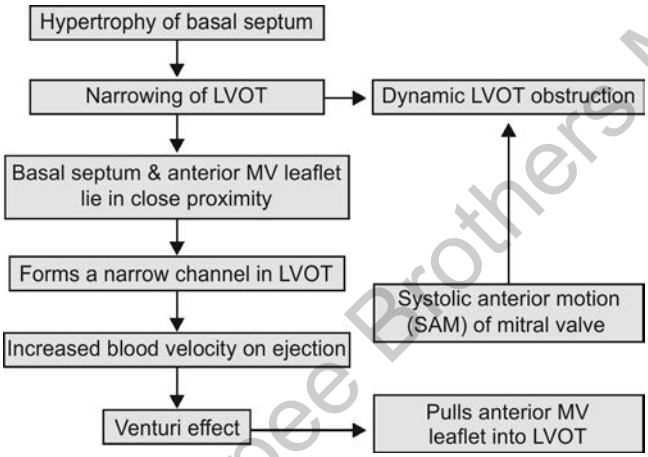
Regions of Hypertrophy

- Septal involvement *most common* (upper interventricular septum just below aortic valve)
- Apex
- Free wall of left ventricle
- *Concentric LVH*: < 10% patients
- Striking regional variations in hypertrophy in contrast to the secondary hypertrophy as in hypertension where it is more uniform.

Pathophysiology

- Characteristically display *diastolic dysfunction* which is reflected by an elevated LVEDP in spite of hyperdynamic ventricular function
- Hypertrophic Obstructive Cardiomyopathy (HOCM):
  - Occurs in 20–30% patients of HCM
  - Called HOCM when HCM patients have subvalvular obstruction

  - In these patients LVOT gradient is formed due to:
    - Systolic septal bulging into LVOT
    - Malposition of anterior papillary muscle
    - Drag forces
    - Hyperdynamic LV contraction causing *Venturi effect*.
  - Obstruction:
    - The obstruction is dynamic and peaks in mid-late systole
    - As opposed to the obstruction in AS which is fixed
    - Beat to beat variability of the degree of obstruction occurs.
- Obstruction is accentuated by any intervention which reduces ventricular size as it facilitates septal-leaflet contact.
- The interventions which increase obstruction are:
  - Increased contractility
  - Increased heart rate
  - Reduced preload/ventricular volume
  - Reduced afterload.



Clinical Features

Symptoms

- Mostly asymptomatic and found as incidental finding on echocardiogram
- Sudden cardiac death (SCD):
  - Most common cause SCD in young people (< 30 yrs)
  - This usually occurs during physical exertion
  - SCD may even be the 1st clinical manifestation.
  - Most common autopsy finding in previously healthy athletes who die suddenly.
- Angina during exercise:
  - Occurs even in the absence of CAD
  - Owing to increased systolic LV pressure, coronary microcirculation is unable to supply hypertrophied myocardium
  - Angina reduces in recumbent posture is very classical as LV size reduces on sitting.
- Others:
  - Dyspnea on exertion
  - Fatigue, syncope
  - Palpitations due to arrhythmias.

Examination

- *Pulses bisferiens*
- Double/triple apical precordial impulse
- Rapidly rising carotid pulse
- SVT/ventricular arrhythmias are common
- 4th heart sound usually audible
- Murmur:
  - Harsh systolic murmur
  - Begins well after 1st heart sound
  - May have holosystolic and blowing quality if associated MR present
  - Best heard at left lower sternal border and apex
  - Reduced by squatting and hand grip
  - Increased by Valsalva maneuver and standing.

Investigations

- Routine blood, BUN, SC, LFT
- ECG:
  - Left ventricular hypertrophy pattern
  - Broad Q waves suggestive of:
    - Old MI
    - Accentuation of normal septal depolarization
- Holter monitoring: For SVT, AF and VT
- Chest X-ray:
  - Maybe normal
  - Mild to moderate increase in cardiac silhouette
- Echocardiogram:
  - Confirmatory
  - LVH with septum ≥ 1.3 times thickness of posterior free wall of LV
  - *Ground glass appearance* of septum
  - LVEF > 80%
- *Doppler echocardiogram:* For peak pressure gradient by determining peak velocity in LVOT
- Thallium 201 scans: Show myocardial perfusion defects even in asymptomatic patients.

- Catheterization studies show:
  - Reduced ventricular compliance
  - Systolic gradient between LV body and subaortic region.

Treatment

Medical Therapy

- β blockers:
  - Reduce inotropy
  - Prevent increases in subaortic pressure gradient.
- Calcium Channel Blockers:
  - Reduce inotropy
  - Improve diastolic compliance by relaxing the heart.
- *Amiodarone*: Used for SVT and ventricular arrhythmias
- Avoid nitrates, digoxin and diuretics
- HOCM with pulmonary edema:
  - Avoid diuretics and digoxin
  - Give volume overload
  - Give β blockers..

Surgical Therapy

- Alcohol ablation of IV Septum, if septal thickness at site of injection ≤ 15 mm
- Pacing:
  - DDD (dual mode, dual chamber, dual sensing)
  - DDD pacing with short AV delay can be useful
  - Cannot be used in patients with atrial fibrillation.
- Septal myectomy (complete):
  - For patients who are symptomatic despite maximal pharmacological therapy
  - For resting gradient ≥ 30 mm Hg and exercise gradient ≥ 50 mm Hg.
- Partial myectomy via aortic approach
- Mitral valve replacement.

Anesthetic Technique

- Regional anesthesia is relatively contraindicated as it reduces SVR and preload
- Continuous spinal/epidural anesthesia may be preferable to bolus dose of LA as it allows better control of LA level
- Ropivacaine is *agent of choice* for Cesarean sections
- Ephedrine and epinephrine contraindicated for hypotension.

Anesthetic Goals

- Rate:
  - Maintain in normal range
  - Avoid tachycardia
  - β blockers used to reduce LVOT gradient and increase LVEDP.
- Rhythm:
  - Maintaining sinus rhythm important
  - Atrial pacing via pulmonary artery catheter maybe useful.
- Preload:
  - Maintain high preload
  - Saturated intravascular space preferred
  - One of the first steps to treat hypotension.
- Afterload:
  - Maintain high systemic vascular resistance
  - Treat hypotension aggressively with α-agonist.
- Contractility:
  - Prefer myocardial depression
  - Avoid inotropic agents.

Rationale for Anesthetic Goals

- Maintain high preload:
  - LV is thick and hypertrophied with a reduced compliance
  - So outflow tract obstruction increases in hypovolemic conditions.
- *Afterload:* Maintain high afterload:
  - Afterload reduction increases the LVOT obstruction
  - Increasing the afterload reduces the outflow gradient
  - This leads to a reduction in SAM and outflow tract obstruction
  - Thus, vasoconstrictors like phenylephrine and vasopressin are used.

Preoperative Evaluation and Premedication

- Evaluate the patient for:
  - Dynamic obstruction
  - Malignant arrhythmias
  - Myocardial infarction.
- NPO orders
- Maintain adequate preload
- Informed consent
- Heavy premedication advised to avoid anxiety induced tachycardia
- Avoid atropine (glycopyrrolate/scopolamine preferred)
- Chronic β blockade and calcium channel blockers continued up to and on the day of surgery
- These are restarted immediately after surgery especially in those undergoing cardiac surgery.

Monitoring
- Pulse oximetry, capnography
- ECG:
  - Leads V5 and lead II used for monitoring
  - Lead II for diagnosing SVT/junctional tachycardia
  - Lead V5 shows abnormal Q waves at times due to accentuation of normal septal depolarization
  - Abnormal Q waves not to be confused for old MI
  - Some patients may have short PR interval.
- Invasive BP monitoring:
  - Arterial waveform shows pulsus bisferiens
  - Initial rapid peak represents early unobstructed ventricular ejection
  - Subsequent downslope and second peak are a result of dynamic obstruction.
- CVP line:
  - Inaccurate guide to changes in LV volume
  - Useful to administer vasoactive drugs.
- PA catheter:
  - Useful for gauging intravascular volume
  - Maintain PCWP in high normal to elevated range
  - PCWP may overestimate patients true volume status
  - PA catheter with pacing potential preferred.
- Urine output
- Temperature.

Position
- Supine position
- Abrupt positioning changes can cause hemodynamic compromise due to changes in preload.

Induction
- *Avoid histamine releasing agents*
- O_2+N_2O+ opioid induction
- Deep planes of intubation preferred to avoid tachycardia
- Thiopentone given in graded doses.

Maintenance
- O_2 + N_2O + volatile anesthetic (halothane/enflurane)
- Opioids and NDMR given as intermittent boluses
- Vecuronium preferred to avoid tachycardia associated with pancuronium
- Halothane and enflurane especially useful as myocardial depression desirable
- Deep level of GA maintained to avoid tachycardia, especially to surgical incision.

Hemodynamics
- Control of atrial rate and rhythm may be beneficial during prebypass period
- Treat hypotension with volume and vasoconstrictors rather than inotropes and vasodilators to preserve adequate cerebral perfusion pressures
- β blockers used as increased heart rate and contractility will increase LVOT obstruction.
- Avoid agents causing increased contractility:
  - Inotropes
  - Calcium
  - β agonists.

Considerations for CPB
- Surgeon should cannulate aorta before atrial manipulation as insertion of venous cannula may precipitate arrhythmias and hypotension
- If junctional tachycardia/SVT while coming off bypass cardioversion to be done
- In cases of junctional rhythm:
  - Patient preload is dependant on atrial contraction
  - Patients will benefit from atrial pacing which is done by:
    - Transesophageal pacing
    - Use of PA catheter with pacing capability.

Ventilation
- Postoperative ventilation may be required
- Extubate if:
  - Spontaneous ventilation with normal ABG
  - No bleeding and hemodynamically stable patient
  - Normothermia.

Postoperative
Monitors
- BP, ECG
- Pulse oximetry
- Temperature
- Urine output
- Arterial blood gas
- Echocardiography.

Analgesia
Opioids/PCA

Complications
- Arrhythmias:
  - Junctional rhythm
  - Supraventricular tachycardia
- Bleeding.

VENOUS AIR EMBOLISM[1,2,3,5,10,17]

Introduction

- Venous air embolism can occur:
  - During any surgical procedure in which operative site is ≥ 5 cms above RA
  - When pressure within an open vein is subatmospheric.

Pathophysiology

- Two preconditions must exist:
  - Direct communication between a source of air and vasculature
  - Pressure gradient to favor passage of air into circulation.
- Small amounts of air:
  - Broken up in capillary bed and absorbed
  - Does not produce symptoms.
- Large amounts of air:
  - 20 ml of air intravenously can produce complications
  - 5 ml/kg of air in intravenous space required for significant injury: Shock/cardiac arrest
  - 2–3 ml of air in cerebral circulation can be fatal
  - 0.5 ml of air is left anterior descending artery can produce VF.
- Transpulmonary passage of air:
  - Air can traverse pulmonary vascular bed to reach systemic circulation when a large volume of air is presented to pulmonary vascular filter
  - Pulmonary vasodilators including volatile anesthetics, reduce the threshold for transpulmonary passage.

Pathogenesis

Can be caused by any open vein/sinus above the level of heart due to:

- Inability of vein to collapse:
  - Intracranial venous sinuses:
    - Transverse sinus
    - Sigmoid sinus
    - Posterior half of sagittal sinus.
  - Diploic spaces
  - Emissary veins
  - Cervical epidural veins
  - Self-retaining surgical retractors
  - Tracks formed around indwelling catheters (like central venous catheter).
- Level of venous pressure at the site:
  - *Posture*: Sitting > supine > prone > lateral
  - Reverse Trendelenburg position:
    - At + 25° intracranial venous sinus pressure 0 cm H_2O
    - At + 90° produces a negative pressure of -12 ± 3 cm H_2O.
  - Spontaneous ventilation:
    - Causes negative intrathoracic pressure
    - This potentiates negative pressure at operative site.
  - In prone position, suction effect of pendulous abdomen can potentiate negative venous pressure within epidural veins.

Etiology

Surgical Procedures

- Neurosurgery and otolaryngological surgery:
  - Most common cause
  - Especially common in *Fowlers sitting position*
  - Also common in posterior fossa surgeries.
- Craniofacial surgery
- Dental implant surgeries
- Liver transplantation
- Genitourinary surgery:
  - Those done in Trendelenburg position
  - Cesarean section
  - Compromised delivery
  - Surgeries involving tumors with high degree of vascularity.
- Orthopedic surgeries:
  - Hip replacement
  - Spine surgery
  - Arthroscopy.
- Vascular surgery:
  - End arterectomies
  - Lateral decubitus thoracotomy.
- *Laparoscopic procedures:* Incidence of VAE > 50%.

Mechanical Insufflation

- Arthroscopic procedures
- Carbon dioxide hysteroscopy
- Laparoscopic surgeries
- Urethral procedures
- Orogenital sexual activity during pregnancy by entering myometrial veins
- Inadvertent infusion of air during IV contrast agent injection for:
  - CT angiography
  - Cardiac catheterization
  - Cardiac ablation procedures.

- Positive pressure ventilation due to barotrauma (VILI) common in:
  - ARDS
  - Hyaline membrane disease
- Ingestion of hydrogen peroxide (rarely).

Iatrogenic Creation of Pressure Gradient

- Procedures causing pressure gradient:
  - During lumbar puncture
  - Insertion of CVP catheters
  - Thoracocentesis
  - Insertion of hemodialysis catheters/Hickmann's catheters.
- Mechanisms of pressure differential:
  - Fracture/detachment of catheter connection
  - Failure to occlude needle hub
  - Presence of persistent catheter tract following removal of CVP catheter
  - Deep inspiration during insertion/removal which causes negative pressure
  - Hypovolemia which reduces CVP
  - Upright position which reduces CVP.

Diving

- Due to pulmonary barotrauma from voluntary breatholding
- *Decompression sickness*: Air bubbles precipitate out into bloodstream if gas dissolved in blood at pressure, is not allowed sufficient time to outgas as ascent
- Rapid rewarming of following CPB.

Clinical Features

Symptoms

In awake patient:
- Dyspnea, nausea, dizziness
- Continuous cough, substernal chest pain
- Gasp reflex:
  - Classic gasp at times
  - Reported when a bolus of air enters pulmonary circulation
- Agitation, disorientation
- Circulatory arrest if large amount of air blocks RVOT/TV/PV or PA (*Saddle embolus*).

Signs

Central Nervous System:
- Altered mental status
- Loss of consciousness
- Seizures, collapse, coma
- Transient focal neurodeficits
- Air bubbles in retinal blood vessels.

Cardiovascular System:
- Raised JVP
- Nonspecific ST and T changes
- Hypotension
- Dysrrhythmias, MI, cardiovascular collapse
- Pulmonary arterial HTN
- Mill wheel murmur:
  - Loud, machinery like churning murmur
  - Occurs due to blood mixing with air in right ventricle
  - Best heard over precordium.

Respiratory System:
- Tachypnea, rales, wheeze
- Cyanosis, hemoptysis
- Reduced E_TCO_2, Low SpO_2
- Raised airway pressure
- Pulmonary edema
- Raised pulmonary vascular resistance.

Skin:
- Crepitus over superficial blood vessels
- *Livedo reticularis*.

Diagnosis

Arterial Blood Gas

- Hypoxia, hypercapnea
- Metabolic acidosis.

Chest X-ray

- May be normal
- Gas in pulmonary arterial system
- Pulmonary arterial dilatation
- *Focal oligemia*: Westermark sign
- Pulmonary edema.

ECG

- Low sensitivity
- Tachycardia, RV strain pattern
- ST depression, transient MI.

CT Scan

- For embolism in central venous system:
  - Axillary and subclavian vein
  - Right ventricle
  - Pulmonary artery.
- CT head for:
  - Intracerebral air
  - Cerebral edema/infarction.

MRI

- Increased water concentration in affected tissues
- Not so reliable.

Transesophageal ECHO

- *Highest sensitivity*, but invasive modality
- More sensitive than precordial Doppler
- Can detect air bubbles as small as 0.25 ml
- Can detect as less as 0.02 ml/kg of air
- Also evaluates cardiac function, right-left shunting of air.

Precordial Dopper

- *Most sensitive non-invasive modality*
- Placed in right parasternal location between 2nd and 3rd or 3rd and 6th ribs
- Interruption of regular swishing of Doppler signal by sporadic roaring sounds indicates VAE
- Precordial Doppler along with E_TCO_2 is the current standard of care
- Transcranial Doppler useful for cerebral microemboli.

End Tidal CO_2

- Sudden reduction in E_TCO_2 occurs
- This is due to sudden increase in pulmonary dead space causing V-Q mismatch
- Reduction of E_TCO_2 by 2 mm Hg is indicative of VAE.

End Tidal Nitrogen

- Most sensitive gas sensing VAE
- Increased ETN_2 occurs if VAE is present
- *Response time is much faster than E_TCO_2*: Changes occur 30–90 seconds earlier.

Pulse Oximetry

Changes in SPO_2 occur very late

Pulmonary Artery Catheter

- Increase in PA pressure can occur
- Relatively insensitive indicator.

Mean Airway Pressure

Increase in MAP is in direct proportion to amount of air entrained.

Prevention of Venous Air Embolism

- Measures to reduce risk during surgery:
  - Nitrous oxide:
    - N_2O can be used provided it is eliminated when VAE occurs
    - If used, better to use 50% N_2O rather than 70%.
  - Positive end expiratory pressure:
    - PEEP no longer used as even 10 cm H_2O PEEP is unlikely to increase CVP
    - PEEP increases chances of pulmonary artery embolism due to barotrauma
    - It also reduces preload, adding to hypotension.
  - Head end elevation:
    - Elevate head end only as much as necessary
    - + 25° Trendelenburg causes 0 cm H_2O pressure in cerebral circulation
    - + 90° Trendelenburg causes +12 cm H_2O pressure in cerebral circulation.
  - Reduce pressure gradient between site of potential entry and RA
  - Surgeons to be meticulous about:
    - Cauterizing
    - Tying blood vessels
    - Applying bone wax.
  - Screening contrast ECHO to detect ASD before sitting position surgeries
- Measures to reduce risk during CVP placement:
  - Avoid and treat hypovolemia before catheter placement
  - During catheter insertion/removal:
    - Patient should be in supine position with head lowered
    - Insertion site should be 5 cms below RA
    - If patient is awake, he can hold breath to increase CVP
    - Doing Valsalva maneuver also increases CVP.
  - Maintain all connections to central line, line closed/docked when not in use
- Measures to reduce risk during IPPV:
  - Prevent barotrauma by minimizing airway pressure during IPPV
  - Avoid PEEP.

Treatment

- Prevent further air entry:
  - Surgeon should flood or pack surgical field with saline

- Bone wax is applied to skull edges until entry site is identified
- Intravascular volume infusion to increase CVP to 10–12 cm H_2O
- Jugular vein compression:
  - Increases cranial venous pressure
  - Also slows air entrainment and cause back bleeding
  - This may help surgeon identify the source of embolus.
- Lower the head end to prevent cerebral embolization
- Close wound quickly if none of measures help
- Inflatable neck tourniquet available in case of VAE

- Treatment of intravascular air:
  - *Aspirate right heart catheter*: RA catheter mandatory in sitting craniotomies
  - Discontinue N_2O immediately
  - FiO_2 100% with inhalational anesthetic
  - Pressors and inotropes
  - *Durant's position*:
    - Lateral position with right side up
    - Allows air to remain in RA, where it will not contribute to airlock in RV
    - Air can then be aspirated from RA.
  - Chest compressions:
    - Maintains cardiac output
    - Helps to break large air bubbles into smaller ones
    - *Forces air out of RV into pulmonary blood vessels*: Improves cardiac output.
  - Hyperbaric oxygen therapy:
    - In case of neurological manifestations/cardiovascular instability
    - Helps by compression of existing bubbles
    - Establishes high diffusion gradient to speed up resolution of existing bubbles
    - Improves oxygenation of ischemic tissue and also reduces intracranial pressure
    - Good prognosis if initiated within 6 hrs.

Paradoxical Air Embolism

- Passage of air across patent foramen ovale which is present in 25% adults
- Necessary gradient to open foramen ovale may be 5 mm Hg
- Significant increase in right heart pressure must occur for PAE to occur
- Even when mean LAP > mean RAP, PAE can occur due to transient reversal of interatrial pressure gradient which occurs during each cardiac cycle
- PEEP and hypovolemia increase risk of PAE
- Avoid even small air bubbles in IV infusion
- *Major cerebral and coronary morbidity*: Causes MI/stroke postoperatively.

TETRALOGY OF FALLOT[1,2,3,5,17]

Introduction

- Tetralogy of Fallots includes:
  - Large subaortic outlet VSD
  - Right ventricular outflow tract obstruction
  - Overriding of aorta
  - Right ventricular hypertrophy.
- Trilogy of Fallots includes:
  - Atrial septal defect
  - Pulmonary stenosis
  - Right ventricular hypertrophy.
- Pentalogy of Fallots includes:
  - Large subaortic outlet VSD
  - Right ventricular outflow tract obstruction
  - Overriding of aorta
  - Right ventricular hypertrophy
  - Atrial septal defect.

Incidence

2 per 10,000 live births

Embryology

- Due to anterior displacement of the conal septum
- This causes large VSD due to malalignment and narrows the RVOT
- Minor anomalies of the anterior leaflet of tricuspid valve also exist as this leaflet is formed by the coral septum.

Anatomy

- VSD is large and unrestrictive and perimembranous
- RVOT obstruction can be at the level of:
  - Infundibulum of RV
  - Pulmonary valve/annulus
  - Main pulmonary artery
  - Peripheral pulmonary arteries
- Infundibular stenosis due to hypertrophy of subpulmonic muscle: *Crista ventricularis*.

Associated Anomalies

- *Patent foramen ovale*: 50–60%
- *Right aortic arch*: 20–25%

- *Multiple muscular VSDs*: 3–15%
- *Left superior venacava*: 8%
- *Abnormalities of origin of LAD*: Arises from RCA and crosses RVOT
- *PDA*: 4%
- *PA stenosis*: 15–30%
- Unilateral absence of PA, usually LPA.

Pathophysiology

- RVOT obstruction:
  - Is dynamic obstruction
  - Can be increased by increases in heart rate and contractility
  - Degree of RVOT obstruction determines the pathophysiological changes.
- Moderately severe RVOT obstruction
  - Predominant shunt is left-right
  - *Acyanotic patient*: Called *Pink tet*:
    - Patient with TOF with source for adequate PA blood flow from:
      - PDA
      - Major aorto-pulmonary collateral arteries: MAPCAs
      - Naturally occurring PA collaterals:
        - Intercostal artery
        - Bronchial arteries
        - Coronary arteries
    - Patient with TOF and insignificant RVOT obstruction.
- Severe RVOT obstruction:
  - Significant right-left shunt
  - Cyanotic patient.
- Acute increase in right-left shunt is due to:
  - *Reduced SVR*: Hypotension
  - *Increased PVR*: Hypoxia, hypercarbia, acidosis
  - *Increased RVOT obstruction*: Tachycardia, increased contractility.
- Congestive cardiac failure in TOF:
  - Since the VSD decompresses RV, CCF is rare in TOF
  - CCF occurs only if TOF is associated with:
    - Anemia
    - Infective endocarditis, myocarditis
    - Systemic HTN
    - Aortic regurgitation/pulmonary regurgitation.

Clinical Features

- Symptoms:
  - Cyanosis
  - Clubbing
  - *Hypercyanotic Tet spells*
  - Failure to thrive, malnourishment.
- Signs:
  - Cyanosis
  - Clubbing, prominent a waves of JVP
  - RV parasternal heave
  - Single audible A_2 as aorta is anterior displaced
  - P_2 is absent as hardly any blood flow occurs through PA
  - Single S_2 helps to differentiate TOF from isolated VSD or PS
  - Systolic murmur along upper left sternal border.
- If VSD with pulmonary atresia, continuous murmur occurs over anterior and posterior chest bilaterally due to aortopulmonary collateral circulation
- Complications:
  - Paradoxical air embolism
  - Polycythemia
  - Coagulation defects
  - *Pulmonary, renal/cerebral thrombosis*: Especially if hematocrit > 65%
  - Cerebral abscess
  - Infective endocarditis
  - *Cardiomyopathy, AR:* Occur late and cause death.
- Without surgical correction:
  - 25% die in 1st year of life
  - 40% die by 4 years
  - 70% die by 10 years
  - 90% die by 40 years.
- *Assessment of severity*: More severe if:
  - Malnourished, inactive
  - Cyanosis is severe due to increased right-left shunt
  - *Shorter and less intense ESM*: Implies PV is more tight.

Hypercyanotic Tet Spells

Introduction

Paroxysmal episodes in which cyanosis acutely worsens.

Precipitating Factors

Increased sympathetic activity due to:

- Crying, fear, anxiety
- Feeding
- Defecation
- Exercise.

Presentation

- Usually does not begin until after 6 months age
- Most common in acyanotic/mildly cyanotic patients
- Occur most commonly in morning and begins with irritability and crying
- Progresses with increasing cyanosis
- May cause seizures if untreated
- Each attack is potentially fatal
- Frequency varies from once in few days to numerous attacks per day.

Mechanism

Due to acute increase in right-left shunt due to:

- Increase in PVR:
  - Hypoxia
  - Negative intrapleural pressure
  - Hypercarbia
  - Acidosis
  - Intermittent positive airway pressure, PEEP.
- Dynamic outflow obstruction:
  - Increased heart rate
  - Increased contractility:
    - Surgical stimulus
    - Inotropes
  - Hypovolemia.
- Decrease in SVR:
  - SBP < 60 mm Hg triggers episodes
  - Drugs:
    - Volatile anesthetics
    - Histamine releasers
    - Ganglionic blockade.

Treatment of TET Spells

- Reduce PVR:
  - Hyperventilate with 100% oxygen
  - Correct acidosis with IV sodium bicarbonate
- Reduce RVOT obstruction:
  - Deepen anesthetic plane with morphine 0.1–0.2 mg/kg IV
  - β blockers:
    - Propranolol 0.1 mg/kg IV
    - Esmolol
  - Volume administration.
- Increase SVR:
  - *Knee chest position*
  - Correct anemia
  - Phenylephrine: 0.25–0.5 µg/kg bolus or 0.025–0.05 µg/kg/min infusion
  - Norepinephrine 0.05 µg/kg/min infusion
  - Intubation and emergent surgery if unresponsive
  - Abdominal aorta/ascending aortic compression.

Investigations

- *Complete blood count*: Polycythemia
- ECG:
  - Right axis deviation
  - Right ventricular hypertrophy.
- Chest X-ray:
  - May be normal
  - Prominent RV, upturned RV apex
  - Concavity of pulmonary conus
  - Oligemic pulmonary fields
  - Right sided aortic arch
  - Boot shaped heart or *Coeur-en-sabot*.
- Echocardiogram:
  - Perimembranous VSD
  - Aortic override
  - Delineates VSD, atrioventricular valve anatomy
  - Used to determine whether there is continuity between MPA and RPA/LPA.
- Doppler ECHO:
  - To estimate gradient between RV and MPA
  - Also diagnose PDA or LSVC.
- Cardiac catheterization studies:
  - Provides information on PVR
  - Ratio of pulmonary to systemic blood flow (Qp: Qs)
  - Degree of valvular PS.
- *Angiocardiography*: Demonstrates:
  - RVOT obstruction
  - Degree of narrowing of pulmonary annulus and MPA
  - *Major Aortopulmonary Collateral Arteries*: MAPCAS.
- *Fundoscopy:* Recognition of papilledema is difficult due to polycythemia with congested retina.

Management

Medical Management

- Treat hypercyanotic spells
- Prevent factors which causes spells like prolonged examination or venipunctures
- Patients with questionable hypercyanotic spells are treated prophylactically with propranolol 0.5–1 mg/kg TID PO.

Surgical Management

Types of Surgeries

- Palliative procedures:
  - Balloon dilation of pulmonary valve
  - Systemic-pulmonary shunts:
    - *Classic BT shunt:* Right subclavian to right pulmonary artery
    - *Modified BT shunt:* Left subclavian to left pulmonary artery with Goretex material
    - *Glenn shunt:* Superior vena cava to right pulmonary artery
    - *Waterstons shunt:* Ascending aorta to right pulmonary artery
    - *Potts shunt:* Descending aorta to left pulmonary artery.
- Definite correction:
  - Right ventriculotomy
  - VSD closure with dacron patch
  - Excision of obstructing RVOT bundles
  - Pulmonary valvotomy
  - Graft for RVOT.
- Rastellis procedure:
  - External conduit placed from body of RV to PA beyond the stenosis
  - This is done if a major coronary artery crosses RVOT
  - Ventriculotomy avoided in this region in such a situation
  - This is the only contraindication to definitive correction after 2 months of age.
- Unifocalization:
  - If TOF with pulmonary atresia, reconstruction of PA with multiple surgeries followed by VSD closure is done
  - Initially RVOT obstruction is relieved and VSD is left open
  - PA is reconstructed via unifocalization procedure
  - The largest of MAPCAS is used to construct RPA and LPA
  - These are than connected to the RV with a homograft
  - This allows flow from RV into PA, thus helping them to grow
  - Eventually VSD is closed and any residual RVOT obstruction is relieved
  - When necessary, a valved homograft is placed between RV and PA.

Indications for Surgery

- Palliative shunts done if:
  - Very small babies (< 2 mths age)
  - Small PA, as it allows PA to enlarge before total repair
- Definitive surgeries:
  - In infants > 2 months age
  - Severe cyanosis/progressive cyanosis.

Timing of Surgery

Elective surgeries usually done between 6–18 months of age.

Anesthetic Management for Definitive Correction

Preoperative Assessment

- *History and clinical examination*: Check for:
  - Upper respiratory infections
  - Ear discharge
  - Loose teeth.
- Investigations:
  - Serum electrolytes, hematocrit
  - Coagulation profile, blood glucose, acid base status
  - Degree of RVOT obstruction and LSVC presence noted.

Preoperative Preparation and Premedication

- NPO guidelines:
  - 6 hours solids, 4 hours breast milk, 2 hours clear fluids
  - Maintain oral feeds/administer IV fluids to avoid dehydration
- Premedicate to avoid sympathetic stimulation:
  - Midazolam 0.5–1 mg/kg 20 minutes before surgery PO
  - Morphine 0.05–0.2 mg/kg IV
  - Avoid sedation in infants < 6 months age
- Anticholinergic to avoid bradycardia associated with anesthetic induction:
  - Atropine 20 µg/kg
  - Glycopyrrolate 10 µg/kg IV as alternative
  - Atropine 10–15 µg/kg PO can be used
  - *Important as patient is already β blocked*: Profound bradycardia can occur.
- Antibiotics:
  - Class IIa recommendations for infective endocarditis prophylaxis
  - Cefazoline 25 mg/kg IV

- Vancomycin 20 mg/kg IV if penicillin allergy, given over 1 hour
- Continue propranolol up to and on day of surgery
- Continue PGE_1 infusion if already present to keep PDA patent.

OT Preparation

- Suction equipment
- Oxygen source
- *Airway:* ET tube, laryngoscope, blade, airways
- Anesthetic drugs
- Emergency drugs:
  - Epinephrine
  - Atropine
  - Phenylephrine
  - Calcium gluconate
  - Propranolol.

Monitoring

- Pulse oximetry
- Blood pressure:
  - NIBP and IBP used
  - *Location of IBP:* Avoid arteries affected by previous shunts
  - Allows continuous BP assessment and ABG sampling
- ECG, E_TCO_2
- Rectal and esophageal temperature probe
- Urine output
- Central venous catheter:
  - Right IJV/femoral vein
  - Left IJV cannulated if left sided SVC is present.
- Transesophageal echocardiography:
  - For LV and RV function
  - Presence of residual VSD/intracardiac air post correction.
- PA catheter:
  - Not necessary
  - Used to assess PA and LA pressures.

Anesthetic Considerations

- Maintain adequate preoperative hydration and sedation
- Maintain SVR and intravascular volume
- Minimize PVR
- Provide mild myocardial depression
- Relatively slow heart rate desirable
- Deep planes of anesthesia preferable
- Avoid:
  - Histamine releasing agents:
    - Atracurium
    - Morphine
    - Pethidine
  - Excessive PEEP
  - Air bubbles in IV line.

Induction

- Atleast one, but preferably two large bore IV cannulas secured
- Adequate preoxygenation for 5 mins
- *If IV line present:* Induction with ketamine 2 mg/kg and vecuronium 0.1 mg/kg
- If IV line not present:
  - *Inhalational induction:* With oxygen and halothane/sevoflurane
  - IM induction:
    - With ketamine 5 mg/kg IM
    - Obtain IV access
    - Administer IV vecuronium 0.1 mg/kg or pancuronium 0.1 mg/kg.
- IV induction is faster than inhalational induction
- Intubate in deeper planes to prevent intubation response
- Leak above 22–25 cm H_2O preferred if uncuffed ET tube is used
- Fentanyl infusion at 0.1 µg/kg/min causes less hemodynamic changes at the time of intubation and sternotomy
- Correct any acidosis if present
- Cross matched blood to be readily available
- Avoid:
  - Air bubbles in tubing to prevent PAE
  - Histamine releasing drugs as it reduces SVR
  - Excitement/agitation at the time of induction.

Maintenance

- O_2 with halothane/isoflurane
- N_2O avoided
- If used, 50% N_2O with 50% oxygen preferred
- Fentanyl 5–10 µg/kg or sufentanyl 1–2 µg/kg with vecuronium or pancuronium
- Manage tet spells as mentioned earlier.

Ventilation

- Avoid increased airway pressure
- Avoid excessive PEEP
- *Mild hyperventilation*: Maintain $PaCO_2$ around 30 mm Hg

- N_2O does not cause much increase in PVR, but 100% O_2 preferred to avoid hypoxia.

Hemodynamics

- Maintain SVR, and euvolemia
- Avoid increases in PVR
- Slow heart rate preferable
- Provide mild myocardial depression
- Initiation of CPB causes hemodilution
- Hematocrit as low as 20% may be allowed on CPB
- Plasma and platelets may be required in post-bypass period to reduce bleeding secondary to dilutional thrombocytopenia and coagulopathy
- Whole blood may be useful.

Considerations for CPB

- After institution of CPB, administer:
  - Opioids, benzodiazepines and muscle relaxants
  - Repeat antibiotic on CPB.
- Any pre-existing shunt (systemic-pulmonary) is clamped to avoid pulmonary hyperperfusion
- Moderate hypothermia (25 ºC–28 ºC) used for CPB
- Deep hypothermic arrest:
  - 18–20 ºC for deep hypothermic arrest
  - Administer 10 ml/kg dextran 40 in 5% dextrose before cooling is initiated.
- Blood cardioplegia with high dose potassium (30 mEq/L) for cardiac arrest
- Monitor:
  - Blood gases every 20–30 minutes to ensure adequate gas exchange and perfusion
  - Mixed venous O_2 saturation to assess tissue perfusion
  - ACT every 20–30 minutes to ensure adequate anticoagulation
  - ACT maintained ≥480 sec
  - *Urine output*: Maintain urine output at 1–2 ml/kg/hr.
- Heparin reversed with protamine after removal of aortic cannula
- After heart is closed, Trendelenburg position given and deairing to remove air bubbles
- Inotropic support with epinephrine/dopamine/dobutamine/milrinone after declamping
- Weaning from CPB after temperature reaches 36 °C and heart has stable hemodynamics.

Extubation

- Usually mechanically ventilated postoperatively, for 24–72 hours
- Once patient is fully awake, hemodynamically stable and peripherally warm, patient is extubated.

Postoperative

Ventilation

- Avoid increased airway pressure, high FiO_2
- Avoid excessive PEEP
- *Mild hyperventilation*: Maintain $PaCO_2$ around 30 mm Hg.

Complications

- Immediate complications:
  - Left anterior hemiblock/complete heart block/RBBB
  - Residual VSD, residual RVOT obstruction
  - Residual PS
  - Pulmonary regurgitation as valve is frequently excised
  - Low output state
  - Coagulopathies
  - Renal failure
  - Stroke
  - Infection.
- Late complications:
  - RVOT obstruction
  - RVOT aneurysm
  - Residual VSD
  - Valvular insufficiency.

Monitoring

- SpO_2, IBP, ECG
- CVP, ABG
- Urine output, blood glucose
- Peripheral temperature, airway pressure
- Echocardiography.

Pain

- Adequate analgesia important
- Morphine 0.1 mg/kg IV or fentanyl 0.5 µg/kg IV boluses.

Anesthetic Management for Shunt Procedures

Preoperative Assessment

- *History and clinical examination*: Check for:
  - Upper respiratory infections
  - Ear discharge
  - Loose teeth.
- Investigations:
  - Serum electrolytes, hematocrit

- Coagulation profile, blood glucose, acid base status
- Degree of RVOT obstruction and LSVC presence noted.

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  - Avoid sedation in infants < 6 months age
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  - Atropine 10–15 µg/kg PO can be used
  - Important as patient is already β blocked: Profound bradycardia can occur.
- Antibiotics:
  - Class IIa recommendations for *infective endocarditis prophylaxis*
  - Cefazoline 25 mg/kg IV
  - Vancomycin 20 mg/kg IV if penicillin allergy, given over 1 hour
- Continue propranolol up to and on day of surgery
- Continue PGE_1 infusion if already present to keep PDA patent.

OT Preparation

- Suction equipment
- Oxygen source
- *Airway*: ET tube, laryngoscope, blade, airways
- Anesthetic drugs
- Emergency drugs:
  - Epinephrine
  - Atropine
  - Phenylephrine
  - Calcium gluconate
  - Propranolol.

Monitoring

- Pulse oximetry:
  - Placed on hand opposite to the side of proposed shunt
  - Occasionally placed on both upper and lower extremity.
- ECG, $ETCO_2$
- BP:
  - NIBP cuff during induction
  - IBP if required after induction
  - Site of IBP should avoid arteries affected by current procedure
  - Same side subclavian artery will be clamped, obliterating radial pulse
- Rectal and esophageal temperature probes
- Precordial stethoscope
- Urine output.

Induction

- Atleast one, but preferably two large bore IV cannulas secured
- Adequate preoxygenation for 5 mins
- *If IV line present*: Induction with ketamine 2 mg/kg and vecuronium 0.1 mg/kg
- If IV line not present:
  - Inhalational induction: With oxygen and halothane/sevoflurane
  - IM induction:
    - With ketamine 5 mg/kg IM
    - Obtain IV access
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- Intubate in deeper planes to prevent intubation response
- Leak above 22–25 cm H_2O preferred if uncuffed ET tube is used
- Fentanyl infusion at 0.1 µg/kg/min causes less hemodynamic changes at the time of intubation and sternotomy
- Correct any acidosis if present
- Cross matched blood to be readily available
- Avoid:
  - Air bubbles in tubing to prevent PAE
  - Histamine releasing drugs as it reduces SVR
  - Excitement/agitation at the time of induction.

Position

- *Lateral position:* If thoracotomy
- *Supine position:* If midline sternotomy.

Maintenance

- O_2 with halothane/isoflurane
- N_2O avoided

- Fentanyl 5–10 µg/kg or sufentanyl 1–2 µg/kg with vecuronium
- Manage tet spells as mentioned earlier.

Ventilation

- Avoid increased airway pressure
- Avoid excessive PEEP
- *Mild hyperventilation:* Maintain $PaCO_2$ around 30 mm Hg
- Maintain mild alkalosis
- 100% O_2 preferred to avoid hypoxia as:
  - SpO_2 may decrease with one lung ventilation
  - SpO_2 may further decrease with clamping of PA to facilitate anastomosis.

Hemodynamics

- Maintain SVR:
  - Avoid drugs which reduce SVR
  - Promptly treat hypotension with vasoconstrictors
  - Whole blood or PRBCs avoided for volume replacement as preoperative HCT is usually high.
- Decrease PVR:
  - High FiO_2
  - Hypocarbia, mild alkalosis
  - Deep planes of anesthesia
  - NTG, SNP, phentolamine, PGE_1, inhaled nitric oxide if required.
- Favor myocardial depression:
  - Halothane
  - β blockers
  - Fentanyl 0.1 µg/kg/min infusion.
- Careful application of cross-clamp to prevent PA distortion helps to maintain pulmonary blood flow
- Severe desaturation and bradycardia at the time of cross-clamp may necessitate institution of CPB
- *Persistent hemodynamic instability*: Dopamine 5–10 µg/kg/min as it also increases flow through the new shunt
- Once shunt is completed and graft is in place, systemic BP may reduce due to runoff into PA
- Some surgeons may use heparin to maintain graft patency.

Intraoperative Complications

- Hypercyanotic tet spells
- Bleeding
- Severe desaturation during chest closure due to:
  - Change in relation of intrathoracic contents
  - Distortion of PA
  - Kinking of the shunt.

Extubation

- Can be done soon after surgery
- Criteria for extubation:
  - Spontaneous ventilation
  - Normal ABG
  - Normothermia
  - Stable hemodynamics
  - Controlled bleeding.
- Prolonged intubation not advised.

Postoperative

Ventilation

- Extubated soon after surgery
- Prolonged intubation not advised

PAIN: Morphine 0.1 mg/kg or fentanyl 0.1 µg/kg IV.

Monitoring

- SpO_2, BP
- ECG, ABG
- Temperature, urine output
- Echo for RV/LV function.

Complications

- Bleeding
- Pneumothorax
- Pulmonary hyperperfusion and pulmonary edema
- Desaturation due to small shunt size
- Shunt thrombosis
- PA hypoplasia.

Role of RA in Shunting/Definitive Surgery

- More effective in inhibiting stress response associated with surgery than IV narcotics
- Spinal anesthesia:
  - Provides excellent pain relief and allows early extubation
  - Spinal given soon after intubation to maximize time before anticoagulation
  - Given for procedures not involving CPB.
- Epidural (continuous) and caudal anesthesia:
  - Useful and can be used for procedures involving CPB
  - In children, epidural catheter can be threaded through sacrococcygeal membrane for 16–18 cms to reach thoracic epidural space .
  - Complications:
    - Hypotension is very uncommon in children

- Risk of epidural hematoma is small
- Epidural catheter removed after return of normal coagulation parameters postoperatively.

– Continuous epidural doses:
- Initial bolus 0.25% bupivacaine 0.5 ml/kg with hydromorphone 7–8 µg/kg
- *Supplemental doses*: 0.25% bupivacaine 0.3 ml/kg
- *Postoperative period*: 0.1% bupivacaine with hydromorphone 3 µg/kg at 0.3 ml/kg/hr.

INFECTIVE ENDOCARDITIS[1,2,3,5,8]

Introduction

- Proliferation of microorganisms on endothelium of the heart
- Proliferation of microorganisms on arteriovenous shunts, arterioarterial shunts (PDA) or coarctation of aorta is called infective endarteritis.

Pathology

- Vegetations occur at the site of infection
- Vegetation consists of:
  – Platelets
  – Fibrin
  – Microcolonies of microorganisms
  – Scant inflammatory cells.
- Sites of infection:
  – Heart valves (native/prosthetic)
  – On low pressure side of septum in VSD
  – Mural endocardium at sites of damage by aberrant jets of blood or foreign bodies
  – Intracardiac devices.

Classification

- Acute endocarditis:
  – Usually caused by:
    - β hemolytic streptococci
    - Staphylococci
    - Pneumococci
  – *Hectic fever*
  – Rapidly damages cardiac structures
  – Hematogenously seeds extracardiac sites
  – Progresses to death within weeks.
- Subacute endocarditis:
  – Usually caused by:
    - Streptococcus viridians
    - Enterococci
    - Coagulase negative staphylococci
    - HACEK organisms.
  – Indolent course
  – Duration of illness > 3 weeks
  – Structural damage occurs slowly
  – Rarely causes metastatic infection
  – Gradually progressive unless complicated by major embolic event or ruptured mycotic aneurysm.

Etiology

Causative Organisms

- Bacteria:
  – Streptococci, HACEK
  – Pneumococci, enterococci
  – Staphylococcus aureus
  – Coagulase negative staphylococcus
  – HACEK group of organisms
  – Polymicrobial.
- Fungal:
  – Candida
  – Histoplasma
  – Aspergillus
  – Blastomyces
  – Mucormyces.

Predisposing Factors

- Congenital heart disease:
  – Ventricular septal defect
  – Tetralogy of fallot
  – Aortic stenosis
  – Bicuspid aortic valve
  – Tricuspid atresia.
- Surgical/natural shunts:
  – Blalock-Taussig shunt
  – Coarctation of aorta.
- Biprosthetic valves and intracardiac devices:
  – Intercostal drain
  – Transvenous pacemaker.
- Drug addicts: Most commonly, right sided IE involving TV/PV.

Precipitating Factors

- Dental extraction
- Brushing of teeth
- Nasotracheal intubation
- Orotracheal intubation
- Use of oral irrigation devices
- Upper GI endoscopy
- TURP
- Barium enema.

Clinical Features

Features Indicating Infection

- Fever:
- 103–104 ºF, acute
- Low grade in subacute infective endocarditis
- Chills, rigors, night sweats
- Arthralgia and diffuse myalgia
- Weakness, malaise, loss of appetite, weight loss
- Amenorrhea in females.

Cardiac Manifestations

- New regurgitant murmur suggests infective endocarditis
- Change in quality of pre-existing murmur
- Perivalvular absesses and fistula
- Embolic events:
  - Stroke
  - Splenic infarct
  - Mesenteric embolism.
- CCF in 30–40% due to:
  - Valvular dysfunction
  - IE associated myocarditis.
- Pericarditis from burrowing of abscess into pericardium
- Heart block due to extension of infection into upper interventricular septum
- MI due to emboli into coronary artery.

Immunological Manifestations

- Arthralgia, myalgia
- Petechiae over skin, mucous membrane and conjunctiva
- *Roth spots:* Petechiac in retina
- *Oslers nodes:* Tender erythematous nodules on pulp of fingertips
- *Janeway lesions:* Non-tender erythematous patches on palms and soles
- *Splinter hemorrhages:* Hemorrhagic spots under nails
- Clubbing, splenomegaly, hematuria.

Other Noncardiac Manifestations

- *CNS:* Stroke, aseptic/purulent meningitis, intracranial hemorrhage due to hemorrhagic infarcts or ruptured mycotic aneurysms, seizures, encephalopathy
- *Mycotic aneurysms:* Focal dilatation of arteries at points in arterial wall weakened by infection in vasa-vasorum where septic emboli have lodged
- Hematogenously seeded infection of skin, spleen, kidney, skeletal system and meninges
- *Embolic events:* Extremities, spleen, kidney, bowel, brain
- Diffuse glomerulonephritis, renal failure/embolic renal infarcts.

Investigations

Identification of Organism

- Blood culture:
  - For diagnosis and detecting antibiotic susceptibility
  - *Sampling*: In the absence of prior antibiotic therapy:
    - Total of three blood culture sets
    - Ideally with first sample separated from the last by atleast 1 hour
    - Obtained from different venipuncture sites over 24 hrs
  - If culture remains negative after 45–72 hrs, two additional cultures obtained to detect fastidious organisms
  - Mainstay of diagnosis.
- Serological tests:
  - Brucella
  - Bartonella
  - Legionella
  - Coxiella burnetti.
- Special stains like periodic acid schiff for tropheryma whippelii
- PCR to recover microbial DNA/RNA.

Echocardiography

- TEE safe and more sensitive compared to transthoracic echocardiography
- Uses:
  - Confirms IE
  - Size of vegetation
  - Detection of intracardiac complications
  - To assess cardiac function.

Other Studies

- Complete blood counts:
  - Normocytic normochromic anemia
  - Leukocytosis
  - Thrombocytopenia.

- Serum creatinine, chest X-ray, ECG
- ESR, CRP levels
- Circulating immune complex titres, RF concentration
- Low complement levels, hypergammaglobulinemia
- *Urine*: Microscopic hematuria and albuminuria
- Cardiac catheterization to check patency of coronary arteries in elderly patients undergoing surgery for endocarditis.

Diagnosis

Dukes criteria

Major Criteria

- Positive blood culture:
  - Recovery of microorganisms consistent with IE from:
    - Blood culture drawn ≥ 12 hrs apart or
    - All of 3 or a majority of 4 or more separate cultures with 1st and last drawn at least 1 hour apart.
  - Single positive culture for C. Burnetti
- Evidence of endocardial involvement:
  - Positive echocardiogram:
    - Oscillating intracardiac mass on valve or
    - Abscess or
    - New partial dehiscence of prosthetic valve.
  - New valvular regurgitation.

Minor Criteria

- Predisposing heart conditions or injection drug abuse
- Fever ≥ 38 °C (≥ 100.4 °F)
- Vascular phenomena:
  - Major arterial emboli
  - Stroke
  - Septic pulmonary infarcts
  - Mycotic aneurysms
  - Janeway lesions.
- Immunological phenomenon:
  - Glomerulonephritis
  - Oslers nodes
  - Roths spots
  - RF factor.
- *Microbiology:* Positive blood culture not meeting major criteria/serology tests.

Interpretation

Diagnosed as infective endocarditis if:

- 2 major criteria or
- 5 minor criteria or
- 1 major with 3 minor criteria.

Treatment

Antimicrobial Therapy

Bacterial endocarditis:

No	Organism	Drug	Duration
1	Streptococcus		
	Penicillin sensitive	Penicillin G	4 weeks
		Penicillin G and gentamicin	2 weeks
		Ceftriaxone	4 weeks
		Vancomycin	4 weeks
	Moderately penicillin resistant	Penicillin G and gentamicin	4–6 weeks
2	Enterococci	Penicillin G and gentamicin	4–6 weeks
		Ampicillin and gentamicin	4–6 weeks
		Vancomycin and gentamicin	4–6 weeks
3	Staphylococcus		
	MSSA with native valve	Nafcillin and gentamicin	4–6 weeks and 3–5 days
		Cefazolin and gentamicin	4–6 weeks and 3–5 days
		Vancomycin	4–6 weeks
	MRSA with native valve	Vancomycin	4–6 weeks
	MSSA with prosthetic valve	Nafcillin and gentamicin and rifampicin	6–8 weeks and 2 weeks and 6–8 weeks
	MRSA with prosthetic valve	Vancomycin and gentamicin and rifampicin	6–8 weeks and 2 weeks and 6–8 weeks
4	HACEK	Ceftriaxone	4 weeks
		Ampicillin and gentamicin	4 weeks

Fungal Endocarditis

- Amphotericin B:
  - Started at 0.25 mg/kg/day
  - Increased by 0.25 mg/kg/day till maximum of 1 mg/kg/day × 2 wks IV.
- 5 fluorocysteine 50–150 mg/kg/day in four divided doses PO
- Both antifungals continued for 2–3 wks
- After this, patient is taken up for surgical removal of vegetation

Drug Doses

No	Drug	Dosage	Frequency
1	Penicillin G	10–20 million units/day	4 doses/day
2	Cefazoline	60 mg/kg/day	3 divided doses
3	Gentamicin	7.5 mg/kg/day	2 divided doses
4	Ampicillin	400 mg/kg/day	4 divided doses
5	Cloxacillin	200 mg/kg/day	3 divided doses
6	Vancomycin	60 mg/kg/day	2–3 divided doses
7	Oxacillin/nafcillin	2 gms	Q4H

Surgical Treatment

- Indications for cardiac surgery:
  - Surgery required for optimal outcome:
    - Moderate to severe CCF
    - Partially dehisced prosthetic valve
    - *Ineffective antimicrobial therapy*: Brucella/fungal infections
    - S. aureus prosthetic valve endocarditis with intracardiac complications.
  - Surgery strongly considered for improved outcome:
    - Perivalvular extension of infection
    - Large (≥ 10 mm diameter), mobile vegetation
    - Persistent unexplained fever (≥ 10 days)
    - Antibiotic resistant enterococci/gram negative bacilli.
- Timing of cardiac surgery:

No	Timing	Indication
1	Emergent (same day)	Acute AR with preclosure of mitral valve
		RSOV into right heart
		Rupture into pericardial sac
2	Urgent (within 1–2 days)	Valve destruction by vegetation
		Unstable (dehisced prosthesis)
		Acute AR/MR with heart failure
		Septal perforation
		Perivalvular extension of infection
3	Elective (usually earlier preferred)	Progressive paravalvular prosthetic regurgitation
		Fungal endocarditis
		Persistent infection after 7–10 days of antibiotic therapy

Infective Endocarditis Prophylaxis[1]

ACC-AHA 2008 guidelines

IE Prophylaxis Recommended for Following Procedures

- Dental procedures involving:
  - Manipulation of gingival tissues
  - Periapical region of teeth
  - Perforation of oral mucosa.
- Respiratory procedures involving incision or biopsy of respiratory muscles
- Infected skin, skin structures or musculoskeletal tissue
- Patients with joint replacement undergoing high risk procedures if:
  - During first 2 yrs after joint replacement
  - Immunocompromised patients
  - Patients with inflammatory arthropathies:
    - Systemic lupus erythematosus
    - Rheumatoid arthritis
  - Comorbid conditions:
    - Previous prosthetic joint infection
    - Malnutrition
    - Hemophilia
    - Insulin dependent diabetes mellitus
    - Malignancy.
- For elective procedures, coexisting enterococcal UTIs should be treated before genitourinary/gastrointestinal procedures
- For emergent procedures, IE prophylaxis considered for patients at highest risk for IE.

IE Prophylaxis no Longer Recommended for Following Procedures

- Gastrointestinal or genitourinary procedures
- For bronchoscopy, unless the procedure involves incision of respiratory mucosa
- For non-dental procedures like:
  - Transesophageal echocardiography
  - Esophagogastroduodenoscopy
  - Colonoscopy
- In patients with screws/pins/plates
- In patients with previous bypass surgeries/stents.

IE Prophylaxis Recommended for Following Cardiac Conditions

- There are no class I recommendations for IE prophylaxis.

- Class IIa recommendations:
  - Prosthetic cardiac valves
  - Patients with unrepaired cyanotic CHD including palliative shunts and conduits
  - Completely repaired CHD with prosthetic material during first 6 months after procedure when endothelialization is taking place
  - Cardiac transplant recipient with valve regurgitation
  - Repaired CHD with residual defects
  - Previous infective endocarditis
  - Patients with congenital heart disease.

IE Prophylaxis Not Recommended for Following Cardiac Conditions

Class III recommendations:

- Patients with isolated secundum ASD
- 6 mths or more after ASD/VSD/PDA repair
- Patients with physiological, functional/innocent heart murmurs, including aortic sclerosis
- Physiological MR without murmur and structurally normal valves
- Physiological TR/PR without murmur and structurally normal valves.

Drugs for Prophylaxis

Single dose given 30–60 mins before surgery

No	Situation	Drug	Adults	Children
1	Oral	Amoxicillin	2 gms PO	50 mg/kg
2	Unable to take orally	Ampicillin	2 gms IV/IM	50 mg/kg
		Cefazoline	1 gms IV/IM	50 mg/kg
		Ceftriaxone	1 gm IV/IM	50 mg/kg
3	Allergic to penicillin, oral	Cephalexin	2 gms PO	50 mg/kg
		Clindamycin	600 mg PO	20 mg/kg
		Azithromycin	500 mg PO	15 mg/kg
		Clarithromycin	500 mg PO	15 mg/kg
4	Allergic to penicillin	Cefazoline	1 gm IV/IM	50 mg/kg
	Unable to take oral	Ceftriaxone	1 gm IV/IM	50 mg/kg
		Clindamycin	600 mg	20 mg/kg
5	Allergic to penicillin, MRSA	Vancomycin	1 gm IV	20 mg/kg

PACEMAKER[1,2,3,5,17]

Classification of Pacemakers

NASPE/BPE 2002 Code

Position I Pacing chamber	Position II Sensing chamber	Position III Response to sensing	Position IV Programmability	Position V Antibradycardia pacing chamber
O = None	O = None	O = None	O = None	O = none
A = Atrium	A = Atrium	I = Inhibited	R = Rate modulation	A = Atrium
V = Ventricle	V = Ventricle	T = Triggerred		V = Ventricle
D = Dual (A+V)	D = Dual (A+V)	D = Dual (T+I)		D= Dual (A+V)

Generic Defibrillator Code

NASPE/BPEG

Position I Shock chamber	Position II Antitachycardia pacing chamber	Position III Tachycardia detection	Position IV Antibradycardia pacing chamber
O = None	O = None	E = Electrogram	O = None
A = Atrium	A = Atrium	H = Hemodynamic	A = Atrium
V = Ventricle	V = Ventricle		V = Ventricle
D = Dual (A+V)	D = Dual (A+V)		D = Dual (A+V)

Indications for AICD

- *Brugadas syndrome*
- Arrhythmogenic RV dysfunction
- Long QT syndrome
- Ventricular fibrillation
- Hypertrophic Obstructive Cardiomyopathy (HOCM).

Preoperative Evaluation

- Focussed history:
  - History of:
    - HTN, DM, MI
    - Previous cardiac surgery
    - Cardiomyopathy
    - Peripheral vascular disease
    - Valvular heart disease
    - Congenital heart disease.
  - *History suggestive of pacemaker dysfunction*: Battery failure:
    - Vertigo
    - Syncopal attacks.
- Focussed examination:
  - Assess previous cardiac disease
  - Assess pacemaker function:
    - Reason for pacemaker insertion
    - Type of pacemaker:
      - *Demand:* Synchronous
      - *Fixed:* Asynchronous
      - AICD.
    - Time of insertion:
      - Electrode displacement possible within 4–6 weeks of insertion
      - Battery failure if long-term.
    - *Irregular heart rate*: Possible competition of pacemaker with intrinsic heart rate
    - Check if pacing pulses are present and create paced beats
- Cardiologists opinion of pacemaker function
- Investigations:
  - ECG:
    - For ischemia/previous MI
    - *Confirm pacing capture:* Pacing rate > intrinsic rate
    - *No intrinsic rhythm:* Patient is pacemaker dependent
    - *Only intrinsic rhythm:* Test pacemaker function by converting to fixed mode with magnet.
  - Chest X-ray:
    - Heart size, lung fields
    - Continuity of pacing leads, distal tip to be within cardiac cavity
    - Location of generator
    - Dual/single chamber
  - *Serum potassium:* Hyperkalemia increases pacing threshold
  - *Acid base balance:* May affect pacing threshold.

Factors Affecting Pacing Function Perioperatively

- *Electrolyte:* Hypo/hyperkalemia
- *Acid base balance:* Acidosis/alkalosis
- *Myocardial:* Ischemia/acute infarction
- *Drugs:* Digoxin toxicity, catecholamines, antiarrhythmics
- *Metabolic:* Hypothermia, thyroid disturbances, hypoxia, hypoglycemia
- Factors which increase the pacing threshold:
  - Hyperkalemia
  - Acidosis, alkalosis
  - Hypoxia, hypoglycemia
  - Hypothermia
  - Antiarrhythmics
  - Thyroid disturbances.

Preoperative Preparation

- Informed consent
- NPO orders
- Premedication with benzodiazepines/opioids
- *Correct associated imbalances*: Potassium and acid base imbalances
- If electromechanical interference (EMI) unlikely, no special precaution needed
- If EMI likely and Cardiac Rhythm Management Device (CRMD) is pacemaker:
  - Reprogram to asynchronous mode when indicated
  - Suspend rate adaptive functions
  - Magnet kept ready to convert to asynchronous mode.
- If EMI likely and CRMD is AICD:
  - Suspend antitachyarrhythmia function
  - Switch off AICD before surgery and attach external pads.

OT Preparation

- Suction apparatus
- Oxygen delivery apparatus
- Anesthetic drugs

- Monitors
- Chronotropic drugs kept ready
  - Atropine: 0.5–3 mg
  - Isoprenaline: 10–100 µg bolus or 1–10 µg/min
  - Ephedrine: 3–30 mg
  - Adrenaline
- Temporary pacing made available
- External cardioverter-defibrillator kept ready.

Monitoring

- Pulse oximetry
- BP, urine output
- *ECG:* Turn off artifact filter
- Peripheral pulse to check cardiac output and pacemaker function
- Invasive monitoring if indicated for surgery
- ABG for electrolytes
- *Nerve stimulator*: May interfere with pacing
- Beat to beat continuous CO monitor in HOCM.

Choice of Anesthetic Technique

- Neuraxial blocks/regional anesthesia:
  - Vasodilation may be poorly tolerated with fixed heart rate
  - Avoid underhydration
  - Caution during nerve blocks as nerve stimulator interferes with pacing
- General anesthesia:
  - *Better preferred*
  - TIVA is preferable.

Considerations for General Anesthesia

Intraoperative

- Ketamine and etomidate cause myofasciculations which may interfere with pacing
- TIVA preferable as volatile anesthetics increase AV delay and pacing threshold
- Halothane is avoided
- N_2O accumulates in pacemaker generator pockets
- Caution with succinylcholine as:
  - Acute increase in potassium causes increase in pacing threshold
  - Myopotentials during fasciculations may be abnormally sensed
- Avoid underhydration
- Considerations for diathermy/electrocautery interference:
  - Use bipolar cantery/ultrasonic scalpel
  - Position of return plate:
    - ICD or pacemaker should not be between the return plate and active electrode
    - Plane described by return plate and active electrode of electrocautery is perpendicular to plane described by pacemaker and pacemaker electrode
  - Use smallest current required to cut/coagulate
  - *Use in short bursts*: One second for every 10 seconds
  - Avoid using electrocautery within 6 inches of the device/leads
  - Activating electrocautery within 15 cms of pacemaker will cause interference, even if not touching the patient.
- Considerations for pacemaker/AICD:
  - Signals from electrocautery maybe misinterpreted as dysrhythmias
  - Convert AICD to no-response either by programming or using magnet
  - Convert pacemaker to asynchronous mode so that it is not inhibited by cautery
  - Once converted, device will not deliver therapy secondary to misinterpretation
  - Do not use electrocautery when AICD is programmed to sense and deliver therapy
  - AICDs must be programmed to respond to a magnet
  - Magnet will not change bradycardia related pacing parameters in the ICD
  - Problems due to electromagnetic interference caused by electrocautery:
    - Reprogramming/inhibition of pacemaker
    - Noise reversion mode/electrical reset
    - Increase in pacemaker threshold
    - Myocardial burns
    - Inappropriate sensing and charging in ICDs.

Postoperative Care

- Immediate postoperative period:
  - Check pacemaker functions, if procedure involved cardioversion and diathermy
  - Monitor cardiac rate and rhythm continuously
  - Back up pacing and cardioversion/defibrillation capability.
- Postoperative restoration of CRMD function:
  - Use cardiologist help
  - Interrogate to assess function
  - Reprogram appropriate setting
  - If CRMD is AICD, restore all antitachycardia therapy.

Treatment of Pacemaker Failure[1,3]

Rate	Response
adequate to maintain BP	Oxygen, airway control
	Place magnet over pacemaker
	Atropine, if sinus bradycardia
Severe bradycardia and hypotension	Oxygen, airway control
	Place magnet over pacemaker
	Transcutaneous/transvenous pacing if magnet fails
	Atropine if sinus bradycardia
	Isoproterenol to increase ventricular rate
No escape rhythm	Cardiopulmonary resuscitation
	Place magnet over pacemaker
	Transcutaneous/transvenous pacing if magnet fails
	Isoproterenol to increase ventricular rate

Emergency Cardioversion/Defibrillation

- Terminate all EMI sources
- Remove magnet to enable defibrillation pads
- Minimize current flow through pulse generator/leads
- Defibrillation pads placed as far away from pulse generator as possible
- Defibrillation pads placed perpendicular to major axis pulse generator/leads
- To extent possible, pads placed in anterior-posterior location
- Use clinically appropriate energies.

Complications

- Failure to fire
- Failure to capture
- *Pacemaker syndrome* seen with VVI mode
- Pacemaker tachycardia.

Patients with AICD

- All ICDs have pacemakers incorporated into circuitry
- Preoperative assessment of cardiac condition
- Cardiology opinion for:
  - ICD interrogation
  - Programming device to no-response mode
- No clear preferences of anesthetic technique between regional/general anesthesia
- Apply patches for external defibrillation when ICD is programmed to no response
- Ensure these pads are as far away as possible from device
- Pads not to be in same plane as device and electrodes
- If PA catheter monitoring is required:
  - Discuss with the cardiologist
  - Document the need for PA catheter and discussion with cardiologist
  - Discuss possibility of dislodgement of ICD electrodes with patient
  - Maintain sterile technique, consider antibiotics before inserting lines.
- Continue antiarrhythmic drugs until time of surgery
- If intraoperative arrhythmias occur:
  - Treat intraoperative causes to prevent recurrence
  - If dysrhythmia continues and *magnet has been used* to create no response mode
  - Remove magnet from ICD
  - Allow ICD to charge and deliver a response
  - If dysrhythmia continues and ICD has been *programmed* to no response mode
  - Reprogram ICD to deliver a response
  - Alternatively, use external defibrillation directly
  - Place external defibrillation paddles in antero-posterior location
  - Deliver sufficient shock
  - External pacing may be required if pacemaker/ICD is damaged with the shock.
- While transporting the patient from OR:
  - Monitor patients ECG
  - Be prepared to deliver external defibrillation
  - Interrogate and reprogram ICD when patient has entered PACU.

Special Considerations

- TURP:
  - Use harmonic scalpel
  - Cutting mode may cause electromagnetic interference
  - Use cautery in short bursts
  - Coagulation mode does not interfere
  - May have to convert to fixed mode if diathermy plate placed around chest
  - Possibility of random reprogramming due to EMI.
- Elective cardioversion:
  - Use lowest possible energy
  - Paddles placed as far away from generator as possible (>10 cm)

  - Paddles placed perpendicular to line between generator and lead tip
  - Follow-up with formal pacemaker interrogation.
- MRI:
  - Contraindicated in pacing dependent patients
  - If required, consult:
    - Physician
    - Cardiologist
    - Radiologist
    - Manufacturer.
- Radiotherapy:
  - ≥ 1000 rads causes pacemakers damage
  - Pulse generator and leads should be outside radiotherapy field
  - Possible surgical relocation of pulse generator
  - Verify pulse generator function before and after therapy.
- Lithotripsy:
  - < 16 KV shock energy to be used
  - Do not focus lithotripsy beam near generator
  - Disable atrial pacing if R waves trigger lithotripsy.
- Radiofrequency catheter ablation:
  - Avoid contact of radiofrequency catheter with generator/leads
  - Radiofrequency current path to be far away from generator or leads
  - Discuss with the surgeon.

PERIOPERATIVE BETA-BLOCKADE[1,2,3,5,17]

Introduction

Beta blockers are amongst the most commonly prescribed drugs and are frequently taken by patients about to undergo surgery.

Classification

- *Nonselective beta blockers*: Act at β_1 and β_2 receptors
  - Propranolol, sotalol, timolol
  - Nadolol, oxprenolol
  - Pindolol, penbutalol
- Cardioselective β_1 blockers:
  - Atenolol, esmolol
  - Betaxolol, metaprolol
  - Bevantolol
- Selective β_2 blockers:
  - Butoxamine
  - ICI-118551.

Intrinsic Sympathomimetic Activity

- Exert partial agonist effect at the receptor while blocking access to more potent agonist
- Thus, may behave like conventional beta blockers when sympathetic activity is high
- This property may protect against harmful effects of β-blockers withdrawal
- β-blockers with Intrinsic Sympathomimetic Activity (ISA):
  - Acebutalol, dilevalol, pindolol
  - Carteolol, oxprenolol
  - Celiprolol, penbutalol.

Reasons for Cardioprotective Effects

- Reduced myocardial oxygen demand and consumption demand in supply ratio
- Less stress on ischemic myocardium due to reduction in heart rate and contractility
- Redistribution of coronary blood flow to ischemic areas
- Increased coronary flow due to increased diastolic time
- Plaque stability due to reduction in shear forces
- Antiarrhythmic effects
- Possible anti-inflammatory effects
- Antiapoptotic.

Indications for Beta Blockade

ACC/AHA 2007 Guidelines

Surgery	No clinical risk factors	≥ 1 clinical risk factor	CHD High risk patients	Currently on β blockade
Vascular	Class IIb, LOE:B	Class IIa, LOE:B	Those found to have ischemia on preop test: Class I, LOE:B	Class I, LOE: B
			Those without ischemia or no previous test: Class IIa, LOE:B	
Intermediate	-	Class IIb, LOE:C	Class IIa, LOE:B	Class I, LOE:C
Low risk	-	-	-	Class I, LOE:C

Current Class I Indications

ACC-AHA 2007 guidelines:

- Should be used in patients previously taking β blockers for all types of surgeries

- Should be used in those with positive stress test undergoing major vascular surgery, although acute administration without titration is associated with harm.

Uses of Beta Blockers

- Treatment of essential HTN
- Treatment of angina pectoris and acute coronary syndromes
- Suppression of cardiac arrhythmias
- Treatment of congestive cardiac failure
- *Prevention of excessive sympathetic nervous system activity*: Intubation, incision
- Preoperative preparation of hyperthyroid and pheochromocytoma patients
- Treatment of migraine, closed angle glaucoma
- Control of *tet spells*.

Beta Blocker Dosages[5]

No	Drug	Oral dose	IV dose
1	Atenolol	50–100 mg QID	
2	Metoprolol	50–100 mg QID	5 mg Q5 min (upto 3 times)
3	Propranolol	60 mg QID	0.1 mg/kg (maximum)
4	Labetolol	100–600 mg QID	1–2 mg/kg
5	Esmolol		50–300 µg/kg/min
6	Carvedilol	25–50 mg BD	15 mg

Adverse Effects of Beta Blockade

- Life threatening bradycardia/asystole
- Congestive cardiac failure
- Glucose intolerance/insulin resistance in diabetes
- Raynauds phenomenon precipitated if pre-existing peripheral vascular disease
- MI on sudden withdrawal
- Uncontrolled HTN if used without prior α-blockade in pheochromocytoma
- Bronchospasm.

Controversy of Perioperative Beta-Blockade

- *Poise trial*:
  - Significant reduction in outcome of cardiovascular events with 30% reduction in MI rate
  - Increased risk of 30-day all cause mortality and stroke
- *MSPIRG trial:*
  - Multicenter Study of Perioperative Ischemic Research Group
  - No improvement in inhospitable outcomes
  - But lower incidence of cardiac events at 6–8 months follow-up
  - Reduction in all-cause mortality at 2 years
- *Pobble study:* No reduction in 30-day cardiovascular mortality in patients undergoing vascular surgery
- *Dipom study:* No benefits in diabetics undergoing major noncardiac surgery
- Conclusion:
  - Thus, early reports showed improved perioperative outcomes
  - Later studies showed no improvement or even worse outcomes in:
    - Low risk patients
    - Those in whom heart rate is not controlled.

Guidelines for Perioperative Beta Blockade

- Uses:
  - Beta blockers should be continued in those already taking them for HTN, angina or arrhythmias
  - β blockers like labetolol and esmolol can be used during intubation and preventing surgical stress response
  - Attempts to discontinue preoperative β blocker therapy causes:
    - Increased risk of rebound tachycardia, with or without AF
    - Myocardial infarction in patients with CAD.
- Dosage:
  - Dosages for all individuals are not the same
  - Dose should be individualized according to patient and surgery
  - *Heart rate control*: Heart rate should be non-ischemia inducing heart rate, 70–80 bpm.
- Administration:
  - Beta blockers should be continued up to time of surgery
  - Should be continued in IV form when GI absorption is in question
  - If β blocker has been omitted in preoperative regimen, use esmolol and labetolol
  - For perioperative MI prophylaxis:
    - Start atenolol 50–100 mg OD 30 days preoperatively
    - Continue in IV form intraoperatively.

ELECTIVE CARDIOVERSION[1,2,3,5]

Introduction

It is the process of conversion of ventricular or supraventricular arrhythmias into normal sinus rhythm using shock delivery which is synchronized with QRS complexes on the ECG.

Indications

- Atrial flutter
- Atrial fibrillation:
  - *Recent onset:* < 12 hours
  - No history of thrombus
- Unstable paroxysmal SVT
- Wolff Parkinson white syndrome
- Unstable monomorphic VT.

Mechanism

- Depolarize entire myocardium
- Prolong refractoriness
- Interrupt re-entrant circuits
- Discharge ectopic foci
- Establishing electrical homogenecity.

Contraindication

- Digitalis induced tachyarrhythmias due to risk of inducing ventricular fibrillation
- Stable atrial flutter with well controlled ventricular rate
- Stable atrial fibrillation with atrial clot.

Complications

- Post shock arrhythmias:
  - Bradycardia, asystole
  - Atrial/ventricular ectopics
  - AV block
  - Ventricular tachyarrhythmias.
- Transient ST elevation with normal cardiac enzymes usually
- Unsuccessful cardioversion due to:
  - Metabolic imbalance
  - Hypovolemia
  - Hypothermia
  - Tension pneumothorax
  - Hemothorax
  - Tamponade
- Myocardial damage
- Systemic/pulmonary embolism in AF.

Preoperative Assessment

- Patient's associated conditions should be stabilized
- Patient should be hemodynamically stable
- Digoxin not to be stopped unless some complication is present
- Serum electrolytes, acid base balance done prior to cardioversion
- ECG taken for detecting new arrhythmias
- Echocardiography to exclude LA clots especially if atrial fibrillation
- Prior anticoagulation for 2–3 weeks duration for AF/flutter ≥ 2–3 days duration
- Anticoagulation continued for 4 weeks after cardioversion
- Antiarrhythmic drugs started prior to procedure reduce incidence of post shock arrhythmias
- Antiarrhythmic drugs used are:
  - Procainamide
  - Amiodarone
  - Quinidine.

Preoperative Preparation

- NPO guidelines:
  - Solids 6 hrs
  - Clear fluids 2 hrs
  - Nonhuman milk 6 hrs
  - Breast milk 4 hrs.
- Sedatives not required
- Antiaspiration prophylaxis:
  - Metaclopranide 0.2 mg/kg IV
  - Ranitidine 1–2 mg/kg IV
- Continue digoxin unless some complications
- *Antiarrhythemic drugs started:* Procainamide, amiodarone
- Continue warfarin therapy.

Dosage

Use only lowest possible energies to avoid myocardial damage:

- *1–3 J/kg or 10–15 J/yr of life*: Pediatric dose
- *Atrial fibrillation:* 100 to 200 J increased in stepwise fashion
- *Atrial flutter and PSVT*: 50–100 J
- *Monomorphic VT*: 100 J → 200 J → 300 J → 360 J (increased in stepwise fashion).

Intraoperative Management

OT Preparation

- All equipment, defibrillator
- *Pacing wires*: Transvenous and transcutaneous

- All intubation equipment
- All emergency drugs.

Monitoring

- Pulse oximetry
- BP
- ECG:
  - Lead which produces large R/S wave and small T wave chosen
  - Obtain 10 lead ECG before and after shocks.

Procedure

- Activate synchronization mode of cardioverter
- Check for proper R-S wave synchronization
- Induction:
  - Preoxygenation for 3–5 minutes
  - Propofol 1–2 mg/kg with fentanyl 10–20 µg IV
  - Etomidate 0.3 mg/kg better choice as induction agent.
- When eyelash reflex lost, apply paddles
- Position of paddles:
  - Anteroposterior position:
    - *Anterior:* Apical area
    - *Posterior:* Interscapular area
  - Anterolateral position:
  - *Lateral:* 5th intercostals space along midclavicular line
  - *Anterior:* Second intercostals space right of sternum.
- Adequate gel applied on paddles to prevent fire hazard
- Remove mask and confirm that no one is touching patient or cart
- Synchronized shock given
- Ventilate patient with 100% O_2 until fully awake/able to maintain patient airway
- Nasopharyngeal airway may be used to maintain airway patency.

Postoperative Management

Monitors

- Pulse oximetry
- *ECG:* Obtain 12 lead ECG
- Noninvasive blood pressure

Complications

- Post-shock arrhythmias
- VF and cardiac arrest
- Thromboembolism to cerebral emboli
- Pulmonary edema
- Aspiration pneumonia.

NEGATIVE PRESSURE PULMONARY EDEMA[1,2,3,5,10,17]

Introduction

- Phenomenon of alveolar capillary membrane injury after exposure of alveoli to subatmospheric pressures leading to abnormal accumulation of fluid in extravascular compartment of lung
- Also called:
  - Postobstructive pulmonary edema
  - Laryngospasm induced pulmonary edema.

Incidence

- 0.05–0.1% incidence
- Mortality as high as 40%
- Occurrence is under-recognized and often misdiagnosed.

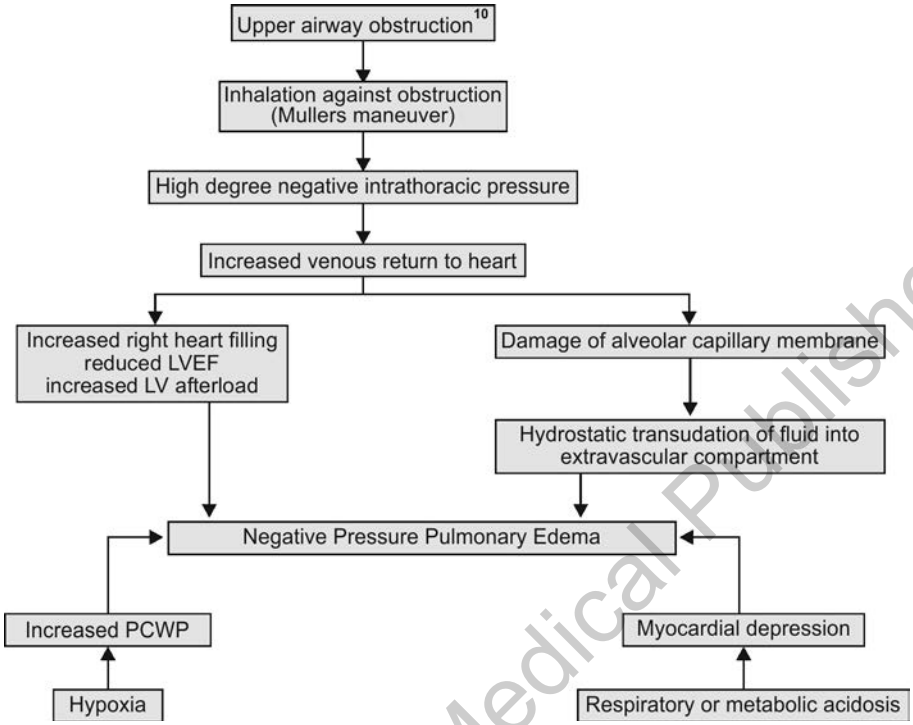
Risk Factors

- Patient related:
  - Male sex, young age
  - Athletes who can generate negative intrathoracic pressures up to –140 cm H_2O
  - Pediatric patients due to complaint chest wall
  - Obstructure sleep apnea, upper airway obstruction, obesity
  - Pharyngeal tumor mass, mediastinal tumor
  - Enlarged tonsils, epiglottitis, croup, Ludwigs angina
  - Vocal cord paralysis
  - Foreign body aspiration
- Surgery related:
  - Nasal, oral or pharyngeal surgeries
  - Chest tube suction
  - Temporomandibular joint arthroscopy
- Anesthesia related:
  - Anatomically difficult intubation
  - Laryngospasm
  - ET tube obstruction
  - ET tube and LMA biting
  - Tongue fall.

Precipating Factors

- Upper airway obstruction
- Laryngospasm
- ***Mullers maneuver:*** Inspiration against closed glottis
- Sudden release of obstruction.

Pathophysiology



Clinical Features

- Usually presents immediately but may occur several hours later
- Rapid onset respiratory distress following occurrence of airway obstruction on emergence from general anesthesia
- Dyspnea and hemoptysis with pink frothy sputum is *hallmark*
- Tachycardia, hypertension and diaphoresis due to sympathetic activation
- Rales and wheeze (due to fluid compressed airway)
- Clinical and X-ray features usually resolve in 24-hours.

Investigations

- Hypoxemia on pulse oximetry
- Hypoxia, metabolic, respiratory or combined acidosis on ABG
- Chest X-ray shows two phases of edema:
  - Intestitial edema
    - *Kerley lines*
    - Peribronchial cuffing
    - Subpleural effusion
    - Occurs with increase in transmural arterial pressure of 15–25 mm Hg .
  - Alveolar flooding:
    - *White-out areas*: Diffuse alveolar and interstitial infiltration
    - Occurs when transmural pressure > 25 mm Hg
    - Nodular opacities which coalesce into frank consolidation
    - Cardiac silhouette usually normal.

Differential Diagnosis

- Aspiration pneumonia
- ALI/ARDS
- Cardiogenic pulmonary edema
- Fluid overload pulmonary edema
- Drug induced noncardiogenic pulmonary edema
- Anaphylaxis
- Pulmonary embolism
- Myocardial infarction
- Pneumothorax.

Prevention

- Prevent laryngeal stimulation and postoperative stridor
- Laryngotracheal topical anesthesia (LTA) before intubation with:

  - 4 % lidocaine solution
  - 10 % lidocaine spray
- IV lidocaine 1–1.5 mg/kg 90 seconds before intubation.

Treatment

- Early diagnosis
- Oxygen supplementation:
  - 100 % oxygen via face mask
  - CPAP/nasal bilevel positive airway pressure (BIPAP)
  - Apply early PEEP to promote alveolar expansion
  - Endotracheal intubation with positive pressure ventilation.
- Diuretics:
  - Role is uncertain yet
  - Furosemide 1 mg/kg IV can be tried.
- Break laryngospasm early:
  - Larsons maneuver
  - Racemic epinephrine nebulization: 1 mg of 1: 1000 solution in 5 ml normal saline
  - Succinylcholine 0.5 mg/kg IV or 0.1–0.2 mg/kg if patient is biting ET tube
  - Dexamethasone/hydrocortisone.

Prognosis

Self limited, with X-ray clearing and normalization of ABG occurring within 24–72 hrs.

ANESTHESIA FOR OPCAB[1,3,5]

Introduction

Bypassing a coronary artery stenosis with an arterial or venous graft without using cardiopulmonary bypass is called Off Pump Coronary Artery Bypass surgery.

Key Surgical Features

- Operation on beating heart
- Absence of cardiopulmonary bypass
- Use of epicardial stabilizer (octopus/vortex stabilizer)
- Multiple vessel grafting
- Temporary interruption of coronary blood flow during anastomosis of distal vessels and intracoronary shunts
- Fast-track extubation either in OT/shortly thereafter.

Advantages of OPCAB[5]

Feature	OPCAB	CABG
Intraoperative		
Ischemia	Transient, coronary	Global myocardial ischemia
Heparin dose	Less	More
Postoperative		
Arrythmias	Rare	Common
Use of antiarrhythmics	Less used	More used
Post-procedure cardiac pacing	Less used	More used
Inotropic support	Fewer required	More required
Perioperative bleeding	Less	More
Perioperative transfusion	Less	More
Metabolic disturbances	Less	More
Cost	Less	More
Side effects of CPB		
Drug pharmacokinetics	More predictable	Less predictable (on CPB)
Intraoperative awareness	Less	More
Hemodilution	Less	More
Complement activation, SIRS	Avoided	Present
Neuropsychiatric impairment	Reduced (< 1%)	More (2–3 %)
Lung injury	Less	More
Troponin I	Less released	More released
Renal function	Better preserved	Impaired
Platelet dysfunction	Less	More

Patient Selection

- Multiple vessel bypass
- Patients at risk for injury with cardiopulmonary bypass like:
  - Advanced age
  - History of CVA/TIA/neuropsychiatric patient
  - Respiratory disease (COPD)
  - Impaired renal function/dialyzed patients
  - Immunosuppression
  - Severe myocardial dysfunction
  - Calcified aorta, aortic disease with increased risk of dissection, rupture
  - Atherosclerotic aorta.

High-risk Patients for OPCAB

- High grade LMCA (left main coronary artery) lesions
- Proximal LAD (left anterior descending artery) lesions
- Triple vessel disease.

Indications to Convert OPCAB To CABG

- *Hemodynamic instability:* Persistence for more than 15 minutes of:
  - Mean arterial pressure less than 50 mm Hg
  - Cardiac index less than 1.5 L/min/m^2
  - ST segment elevation more than 2 μV
  - Malignant arrhythmias
  - Severe LV dysfunction on TEE
  - $MvO_2 \leq 60\%$
- New regional wall motion abnormalities
- Inability to access areas which require revascularization
- Intramyocardial vessels
- Vessel endarterectomy.

Preoperative Assessment

- History:
  - Angina/recent MI
  - Cerebrovascular accident
  - *LV dysfunction:* Orthopnea, paroxysmal nocturnal dyspnea
  - Diabetes mellitus, hypertension, smoking, hyperlipidemia.
- Physical examination:
  - Raised JVP, basal crepitations
  - Pitting edema, S_3 gallop
  - *Airway examination:* Difficult airway anticipated due to DM/obesity.
- Investigations:
  - Routine blood investigations, clotting time, urine, electrolytes, renal function tests
  - ECG, Chest X-ray
  - Echocardiography:
    - 40–50 % EF: Mild LV dysfunction
    - 25–40 % EF: Moderate LV dysfunction
    - Less than 25 % EF: severe LV dysfunction.
  - Angiogram done within last 12 months
  - Depending on patient requirements:
    - Dobutamine stress test
    - Technetium 99 m scan
    - Pulmonary function tests
    - CT brain and chest
    - Endoscopic GI examination.

Preoperative Preparation and Premedication

- Optimize patient's preoperative DM, BP and lipid profile and heart rate
- Good patient communication necessary to avoid anxiety induced tachycardia
- NPO orders
- Informed consent
- Premedication:
  - Diazepam 0.1 mg/kg IV
  - Midazolam 0.03 mg/kg IV
  - Morphine 0.05–0.1 mg/kg IV
  - Ranitidine 1 mg/kg IV or pantoprazole 20 mg IV.
- *Antibiotic prophylaxis:* Cefotaxime 1–2 gms + gentamicin 1.5 mg/kg IV
- Continue morning dose of β-blockers, CCB, and nitrates till day of surgery
- Stop ACE inhibitors to prevent precipitous hypotension intraoperatively
- Oral hypoglycemic to be withheld on the morning of surgery.

OT Preparation

- Anesthetic drugs
- Machine check
- *Emergency medications*: Atropine, adrenaline, phenylephrine, calcium
- *Airway equipment*: ET tube, laryngoscope, airway, LMA, gum elastic bougie
- *CPB machine*: Primed and on stand by
- IABP, if required, in LMCA lesions
- Suction equipment
- External defibrillation pads before preparation and draping, defibrillator.

Procedure

- Sternotomy
- Saphenous vein/radial artery dissection
- Internal mammary artery (IMA) harvesting
- Heparin 2 mg/kg before IMA isolation
- ACT done after 3–5 minutes
- LIMA-LAD anastomosis
- Distal anastomosis with induced bradycardia
- Intracoronary stent applied during anastomosis
- Proximal anastomosis by partial cross clamping
- Heparin reversal with protamine
- Hemostasis
- Closure.

Position

Supine

Monitoring

- ECG:
  - Lead II for rhythm disturbances
  - Lead V_5 with ST segment analysis for ischemia
  - V_4R and V_5R used if right ventricular ischemia

  - May show flatline during circumflex artery anastomosis due retraction of heart.
- Pulse oximetry, capnography
- Invasive blood pressure monitoring
- Bispectral index or entropy monitoring, neuromuscular monitoring
- CVP/PA catheter
- Temperature, urine output
- Periodic ABGs
- Continuous cardiac output monitoring, transcranial *Doppler:* Optional
- Transesophageal echocardiography:
  - Especially in patients with raised LVEDP, to assess LV preload
  - Also for LV function and detecting new RWMA during anastomosis.

Anesthetic Goals

- Cardiac protective anesthetic technique used
- *Rate:* Slow, 70–80 bpm (non-ischemia producing heart rate)
- Rhythm: Sinus rhythm
- *Contractility:* Myocardial depression preferred (if LV function is normal)
- Preload:
  - Judicious preload
  - Reduce wall tension and LVEDP.
- *Afterload:* Maintain afterload (hypertension preferred to hypotension)
- *Temperature:* Keep patient warm
- MvO_2:
  - Maintain myocardial oxygen demand-supply ratio
  - Treat myocardial oxygen supply related disturbances
- *Severe LMCA disease:* Maintain diastolic pressure and heart rate at preoperative values.

Induction

- Adequate preoxygenation for 3–5 minutes
- Induction in calm and relaxed manner
- Two methods of induction:
  - Patients with good LV function:
    - Normal doses of anesthetic agents as sympathetic response usually intact
    - Thiopentone 3–5 mg/kg + vecuronium 0.1 mg/kg + fentanyl 5 µg/kg IV for induction
    - Propofol 2 mg/kg maybe used as alternative
    - Induction agents in titrated doses to prevent hypotension at induction
    - β blockers and vasodilators kept ready to negate intubation response.
  - Patients with poor LV function:
    - Reduced doses of anesthetic as they are unable to produce significant response to stimulation
    - Pancuronium 0.1 mg/kg + fentanyl 10 µg/kg + midazolam 0.15 mg/kg IV for induction
    - Pancuronium is used as muscle relaxant to counter opioid induced bradycardia
    - Etomidate maybe used for induction to maintain hemodynamic stability
    - Vasopressors and inotropes kept ready.
- Deep planes of anesthesia maintained at the time of intubation
- Prevention of intubation response:
  - IV lignocaine 1.5 mg/kg 60–90 seconds prior to intubation
  - Use of NTG/esmolol to blunt intubation response
  - Fentanyl 4–5 µg/kg + vecuronium 0.1 mg/kg IV prior to laryngoscopy.
- Two large (18 G) bore IV cannula inserted
- Ryles tube inserted to deflate stomach and left open
- NTG infusion started and continued throughout procedure.

Maintenance

- Opioid based balanced anesthetic technique used
- Propofol infusion less preferred as inotropic requirements and troponin I increase
- Pure opioid based anesthesia technique preferred if preoperative LV dysfunction is present
- O_2 + N_2O + isoflurane 1 MAC used for balanced anesthesia
- Vecuronium and fentanyl given as intermittent boluses
- Fentanyl bolus given before sternotomy to avoid hypertensive response
- Anticoagulation:
  - Heparin 2 mg/kg IV given before commencing distal anastomosis
  - ACT maintained above 250 seconds.
- Protamine:
  - Given to reverse anticoagulation
  - Dose given once the heart is reperfused with multiple grafts, after proximal anastomosis.

Hemodynamics

- Rigid control of heart rate and blood pressure required
- Heart rate:
  - Maintain heart rate of around 70 bpm (non-ischemia producing heart rate)
  - Esmolol infusion/metaprolol boluses useful.
- Dysrhythmias:
  - Occurs due to manipulation of heart, ischemia or reperfusion injury
  - Keep electrolytes at normal level
  - Infusion of magnesium sulfate or xylocard before manipulation of heart, is useful in preventing arrhythmias
  - Pacemaker to be readily available to treat bradyarrhythmias.
- Hypotension:
  - During distal anastomoses, especially obtuse marginal anastomosis
  - Treatment:
    - Give IV fluids rapidly, 20 Trendelenburg position
    - Phenylephrine 100–200 µg IV bolus or dobutamine infusion
    - Adjust stabilizer and reposition the heart.
- Hypertension:
  - Nitroglycerine 0.5–3 µg/kg/min IV
  - β blockers: Labetalol/esmolol
  - Calcium channel blockers.
- Cardiac output:
  - Maintaining cardiac output more important than BP
  - Maybe reduced due to reduced contractility from ischemia/displacement of heart
  - Dobutamine/dopamine used to maintain cardiac output
  - Insertion of IABP may be necessary, especially in LMCA lesions.
- *Temperature:* Prevent hypothermia:
  - Use warm IV fluids
  - Humidification of air
  - Maintain warm OT temperature
  - Position patient on heating mattress to prevent hypothermia.

Ventilation

- Lungs to be deflated at the time of sternotomy
- Hand ventilation may be required during:
  - Dissection of left internal mammary artery (LIMA)
  - Distal anastomosis to aid surgical exposure, especially for marginal branches of left circumflex artery.
- Hyperventilate (to prevent atelectasis and hypercapnea) subsequently.

Conduct of Anastomosis

Distal Anastomoses

- Most demanding phase of surgery
- Aggressive maintenance of hemodynamic stability required
- LAD-LIMA anastomosis:
  - Intracoronary shunts used to improve coronary blood flow during grafting
  - Prevent hypotension:
    - IV fluids to prevent fall in preload, which occurs when heart is retracted
    - 20° Trendelenburg position helpful to improve preload
    - Avoid steep Trendelenburg to prevent reduction in pulmonary compliance
    - Infusion of dobutamine/dopamine used to maintain BP and CO:
      - If preoperative cardiac function is impaired
      - If cardiac output reduces after application of epicardial stabilizer.
  - Cardiac output improves once anastomosis is complete and stabilizer removed
  - Watch for reperfusion injury when snugger is removed
  - BP and cardiac output should be returned to normal before next anastomosis.
- Obtuse marginal and posterior descending artery anastomosis:
  - Deep pericardial retractors or sling may be used to retract heart
  - This may displace heart with apex pointing anteriorly
  - This may lead to RV dysfunction and biventricular failure
  - This will cause severe reduction in stroke volume as RV cannot expand during diastole
  - Boluses of epinephrine or ephedrine if cardiac index continues to fall during anastomosis (CI < 1.5) to avoid progressive cardiac failure
  - Monitor PA pressure in view of biventricular dysfunction
  - Monitor ABG and maintain pH above 7.3.

Proximal Anastomosis

- Partial cross clamping of aorta done prior to proximal anastomosis
- This requires rapid reduction of blood pressure:
  - Reverse Trendelenburg position beneficial
  - Volatile anesthetics/nitroglycerine useful
- Watch for reperfusion injury on release of snuggers.

Postoperative Management

Ventilation

- *Early extubation*: When extubated less than 10 hours postoperatively
- Extubation if:
  - Awake, normothermic
  - Hemodynamically stable
  - Not bleeding/acidotic
- Early institution of chest physiotherapy.

Postoperative Analgesia

- IV opioids, patient controlled analgesia
- Thoracic epidural
- Ketorolac + small dose opioids
- Multimodal approach.

Monitors

- Pulse oximetry, temperature
- Blood pressure:
  - Maintained at less than 130 mm Hg to protect graft top-ends and reduce bleeding
  - Maintain nitroglycerine infusion to prevent graft spasm.
- *Drains*: Should be less than 100 ml/hr
- ACT, repeated ABG and blood sugar
- Echocardiography for:
  - LV ejection fraction
  - Regional wall motion abnormalities
  - Pericardial effusion and tamponade.

Complications

- Pain, hypertension, tachycardia
- Bleeding, arrhythmias
- Cognitive dysfunction
- LV dysfunction
- Low cardiac output syndrome (LCOS).

Anticipation and Management of Ischemia

- Communication between surgeon and anesthetist
- Observe surgical field, heart size, position and function
- Pharmacological preconditioning
- Maintain adequate coronary perfusion pressures
- Avoid hemodynamic alterations associated with ischemia
- Avoid tachycardia (especially if hypothermia present): Use β-blockers
- Volume loading, trendelenburg, reduction in depth of anesthesia and use of α-agonists may be indicated
- Epicardial pacing may be required for ischemia induced bradycardia.

Regional Anesthesia

- Epidural anesthesia advantages:
  - Reduced stress response
  - Better analgesia
  - Better respiratory function postoperatively
  - *Antianginal*: Reduces dynamic occlusion
  - Better distribution of coronary blood flow
  - Reduced oxygen demand.
- Epidural anesthesia disadvantages:
  - More hypotension
  - Epidural hematoma, infection
  - Dural puncture with post-dural puncture headache
  - Spinal cord injury.

Epicardial Stabilizers

- Allows better access to inferior and posterior surfaces of heart
- Examples:
  - Vortex stabilizer system
  - Octopus stabilizer
  - Cohn stabilizers.

ANESTHESIA FOR ON-PUMP CABG[1,2,3,5]

Introduction

Most commonly performed cardiac surgical procedure where a coronary artery stenosis is bypassed with an arterial/venous graft using cardiopulmonary bypass.

Preoperative Assessment

- History:
  - Angina/recent MI
  - Cerebrovascular accident
  - *LV dysfunction:* Orthopnea, paroxysmal nocturnal dyspnea
  - Diabetes mellitus, hypertension, smoking, hyperlipidemia.

- Physical examination:
  - Raised JVP, basal crepitations
  - Pitting edema, S_3 gallop
  - *Airway examination:* Difficult airway anticipated due to DM/obesity.
- Investigations:
  - Routine blood investigations, clotting time, urine, electrolytes, renal function tests
  - ECG, Chest X-ray
  - Echocardiography:
    - 40–50% EF: Mild LV dysfunction
    - 25–40% EF: Moderate LV dysfunction
    - Less than 25% EF: Severe LV dysfunction
    - Check for MR/AS/AR/VSD/ventricular aneurysm.
  - Angiogram done within last 12 months
  - Depending on patient requirements:
    - Dobutamine stress test
    - Technetium 99 m scan
    - Pulmonary function tests
    - CT brain and chest
    - Endoscopic GI examination.

Preoperative Preparation and Premedication

- Optimize patient's preoperative DM, BP and lipid profile and heart rate
- Good patient communication necessary to avoid anxiety induced tachycardia
- NPO orders
- Informed consent
- Premedication:
  - Diazepam 0.1 mg/kg IV
  - Midazolam 0.03 mg/kg IV
  - Morphine 0.05–0.1 mg/kg IV
  - Ranitidine 1 mg/kg IV or pantoprazole 20 mg IV
- *Antibiotic prophylaxis:* Cefotaxime 1–2 gms + gentamicin 1.5 mg/kg IV
- Continue morning dose of β-blockers, CCB, and nitrates till day of surgery
- Stop ACE inhibitors to prevent precipitous hypotension intraoperatively
- Oral hypoglycemic to be withheld on the morning of surgery.

OT Preparation

- Anesthetic drugs
- Machine check
- *Emergency medications:* Atropine, adrenaline, phenylephrine, calcium
- Airway equipment: ET tube, laryngoscope, airway, LMA, gum elastic bougie
- *CPB machine:* Primed and on stand by
- IABP, if required, in LMCA lesions
- Suction equipment
- External defibrillation pads before preparation and draping, defibrillator.

Position

Supine.

Monitoring

- ECG:
  - Lead II for rhythm disturbances
  - Lead V_5 with ST segment trending for ischemia
  - V_4R and V_5R used if right ventricular ischemia.
- Pulse oximetry, capnography
- Invasive blood pressure monitoring:
  - Preferred on side opposite to IMA dissection
  - This is because sternum will be retracted during dissection of IMA
  - This may cause tenting of subclavian artery and reduce flow through subclavian artery.
- Bispectral index or entropy monitoring, neuromuscular monitoring
- CVP/PA catheter if:
  - Ejection fraction less than 45 %
  - Significant left ventricular RWMA
  - LVEDP more than 18 mm Hg
  - Recent MI or unstable angina
  - Post-MI complications.
- Nasopharyngeal temperature, urine output
- Periodic ABGs
- Continuous cardiac output monitoring, transcranial Doppler: Optional
- Transesophageal echocardiography:
  - Especially useful in patients with raised LVEDP
  - To assess LV preload and LV function
  - Earliest detector of intraoperative MI
  - Also helps in detection of air.

Anesthetic Goals

- Cardiac protective anesthetic technique used
- *Rate:* Slow, 70–80 bpm (nonischemia producing heart rate)
- *Rhythm:* Sinus rhythm
- *Contractility:* Myocardial depression preferred (if LV function is normal).

- Preload:
  - Judicious preload
  - Reduce wall tension and LVEDP
- *Afterload:* Maintain afterload (hypertension preferred to hypotension)
- *Temperature:* Keep patient warm
- MvO_2:
  - Maintain myocardial oxygen demand-supply ratio
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- *Severe LMCA disease:* Maintain diastolic pressure and heart rate at preoperative values.

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- Adequate preoxygenation for 3–5 minutes
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    - Induction agents in titrated doses to prevent hypotension at induction
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    - Etomidate maybe used for induction to maintain hemodynamic stability
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- Prevention of intubation response:
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  - Use of NTG/esmolol to blunt intubation response
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- Two large (18 G) bore IV cannula inserted
- Ryles tube inserted to deflate stomach and left open
- NTG infusion started and continued throughout procedure.

Ventilation

- Lungs to be deflated at the time of sternotomy to avoid pleural/lung injury
- Hand ventilation may be required during IMA dissection
- Hyperventilate thereafter, if low tidal volume used during dissection, to prevent atelectasis
- Controlled ventilation during de-airing maneuvers while coming off CPB, where positive airway pressure of 30 cm H_2O is given, to empty the heart of air, with Trendelenburg position.

Hemodynamics

- Maintain heart rate around 70 bpm (esmolol/metaprolol useful)
- Maintain systolic BP around 100–120 mm Hg during the procedure
- Maintain systolic BP at around 80–100 mm Hg at the time of aortic cannulation
- Manage hypotension with IV fluids, Trendelenburg position, vasopressors or inotropes
- Hypertension managed with nitroglycerine/β blockers
- Two units of cross matched and grouped PRBCs kept ready in OT
- Blood is especially important in redo cases as RV perforation may occur at the time of sternotomy.

Maintenance

Prebypass

- Isoflurane is safe in patients with CAD, in the absence of hypotension
- Halothane and enflurane also may be used
- Heparin 400 IU/kg given prior to IMA clamping
- Ensure that ACT is more than 480 seconds prebypass
- O_2 + air + isoflurane 0.6–2 MAC used to maintain balanced anesthesia
- Fentanyl 1 µg/kg + pancuronium 0.05 mg/kg intermittent boluses
- Add vecuronium 0.1 mg/kg + morphine 0.1–0.2 mg/kg (fentanyl 250–500 µg) + midaolam 0.05 mg/kg to pump prime
- Make note of urine output once CPB ensues.

During Bypass

- Heparin:
  - Repeated at half the initial dose at 1 hour
  - One-fourth the initial dose at 2 hours and every hour thereafter
- Vecuronium 0.05 mg/kg + morphine 0.1 mg/kg + midazolam 0.03 mg/kg every hour
- Check ABG and ACT every half hour on CPB
- Record temperature, mean arterial pressure, hematocrit, serum potassium and urine output at termination of bypass.

Postbypass

- Deairing maneuvers once heart is being closed
- Defibrillate/cardiovert if VF/SVT
- Inotropes started once aortic cross clamp is removed
- Ventilation is started after:
  - PA perfusion is adequate
  - Last proximal graft is being sutured
  - Prominent pulsations are present on arterial line tracing.
- Discontinue N_2O at this stage to prevent expansion of air bubbles
- Increase inotropes if low BP with rising LA, PCWP and RA pressure at the time of weaning from bypass.
- Protamine (1:1 with heparin) given after venous decannulation
- Last internal suction done after half the protamine dose has been given
- ABG taken 5–10 minutes after protamine completed (for ACT and ABG).

Checklist Prior to CPB Initiation[1,5]

Item	Anesthetic checklist
Anesthetic level	• Narcotics, BZD, additional dose muscle relaxant • Volatile anesthetic availability on pump • Narcotics, benzodiazepines, relaxants added to pump prime
Monitors	• To be visible to anesthetist and perfusionist • Retrograde pressure monitored • Swan-Ganz catheter pulled back 5–10 cms before CPB initiation
Anticoagulation	• Heparin 400 IU/kg given • ACT maintained above 480 seconds
Arterial line	• Check for air
Venous line	• Minimize air to prevent air-lock
Urine output	• Maintained more than 0.5 ml/kg/hour
Retrograde cardioplegia line	• Pressure displayed (usually RV trace) • Position confirmed by TEE if available

Check List Post CPB Initiation[1,5]

Item	Objective	Problems	Solution
Arterial blood flow	Adequate pump flow	Low flow	Check line for kinks
	Normal pump pressure	High pump pressure	Check for aortic dissection
	MAP > 50 mm Hg	Low MAP	Check for innominate A cannulation
Arterial blood color	Bright	Dark color	Check O_2 supply to oxygenator
Venous return	Good return	Poor return	Check line for kinks
	Low venous pressure	High venous pressure	Check line for air Check table height is adequate
Heart filling	Heart to be empty	Distended ventricle	Check venous return Vent the heart If AR present, reduce flow
IV Fluids	Should be off	Still running	Stop IV fluids
Ventilation	Stopped	Ventilation on	Turn off unless partial CPB
	Deflated lungs	Lungs inflated	Check PEEP valve
Anesthesia	Adequate depth	Anesthesia too light	Add vapor to pump gas Repeat IV drugs every hour
Anticoagulation	ACT > 480 sec	Inadequate	Heparin half dose at 1 hour Heparin quarter dose at 2 hours Repeated every hour thereafter
Urine output	More than 2 ml/kg/hr	Inadequate	Diuretics

Check List for Weaning from CPB[1,5]

Item	Objective	Problem	Solution
acid base status	Normal	Base deficit > 5 mmol/L ECF	Correct with bicarbonate
Electrolytes	Normal potassium	Hypo/hyperkalemia	Correct appropriately
	Normal magnesium	Hypo/hypermagnesemia	Use insulin if K^+ very high
	Normal calcium	Hypo/hypercalcemia	
Hematocrit	≥ 7 g/dl	< 6.5 g/dl	Transfusion
			Hemoconcentration
Glucose	< 15 mmol/L	Hyperglycemia	Insulin
Anticoagulation	≤ 120 seconds	≥ 121 seconds	Additional protamine
			FFP/platelet concentrates
Transducer	Zeros correct	Zero drift	Zero again at rewarming
Temperature	36–37 °C	< 36°C	Inadequate rewarming
Blood pressure	between 95–125 mm Hg	Less than 95 mm Hg	Inotropes
Heart rate	≥ 70 bpm	Bradycardia	Treat with anticholinergics
		Tachycardia	Treat hypovolemia/ pain
Rhythm	Sinus rhythm	Heart block	Pacing (DDD is best)
		Atrial fibrillation	Cardioversion
		Ventricular fibrillation	Defibrillation, amiodarone
		Multiple VPCs	Check electrolytes
Cardiac output	Normal	Low	Inotropes ready
SVR	Normal	Inappropriate vasodilation	Vasopressors
		Inappropriate vasoconstriction	Vasodilators
Anesthesia	Adequate depth	Too light	Check infusion
			On vaporizer
IV fluids	Ready to run	Not ready	Prepare IV fluids
Ventilation	Ventilation on	Ventilation off	Switch on ventilation
	FiO_2 > 50%	FiO_2 < 50%	Increase FiO_2 > 50%
	No IMA stretching		
	Check pneumothorax		

FAST TRACK MANAGEMENT OF CABG[1,5]

Introduction

Consists of rapid postoperative extubation (within 4–6 hours), postoperative rehabilitation and discharge from hospital (within postop day 5).

Components of Fast Track CABG

- Patient education
- Same day admission
- Altered anesthetic and surgical management
- Special recovery areas
- Early extubation early mobilization
- Prophylactic/aggressive treatment of complication
- Discharge from ICU on postoperative day number 1
- Discharge from hospital on postoperative day number 5
- Out of hospital follow-up.

Advantages of Early Extubation

- Earlier discharge
- Respiratory benefits:
  - Lesser endotracheal tube related complications
  - Improves mucus transport.
- Cardiovascular benefits:
  - Improved cardiac output
  - Ventricular function returns to baseline within 24 hours after CABG in 90% patients.

Anesthetic Management

- Lower dose of opioids:
  - Fentanyl less than 30 µg/kg
  - Sufentanyl less than 7 µg/kg
  - Remifentanyl less than 3 µg/kg/min.
- Supplementation with:
  - Inhalational agents: Isoflurane 0.6–2 MAC
  - Propofol infusion 25–100 µg/kg/min
  - Midazolam
  - Muscle relaxants
- Postoperative ICU sedation:
  - Propofol infusion continued
  - Midazolam boluses.
- Postoperative analgesia:
  - Morphine, NSAIDs
  - Patient controlled analgesia
  - Neuraxial techniques.

Surgical Approaches to Fast Track Extubation

- Appropriate patient selection
- Rehearsed, efficient surgical team
- Well-planned, expeditious and technically superior surgery
- Meticulous myocardial protection
- Avoid intraoperative complications and residual structural defects
- Appropriate early use of inotropic support
- Management of bleeding
- Avoidance of stroke.

Management in ICU

- Return to ICU:
  - Reverse muscle relaxant as soon as stable to assess level of sedation
  - Start fentanyl infusion at 0.01–0.03 µg/kg/min
  - Continue propofol infusion at 10–30 µg/kg/min
- Awakening:
  - If temperature < 35 °C, maintain propofol infusion
  - If temperature 35–35.5 °C, reduce propofol infusion
  - If core temperature > 35.5 °C:
    - Stop propofol infusion
    - Reduce fentanyl infusion by half
    - Give IV ketorolac 10–20 mg (0.5mg/kg).
- Criteria for weaning:
  - Minimal bleeding (less than 50 ml/hr)
  - Stable hemodynamics
  - No significant arrhythmias
  - Adequate urine output > 0.5 ml/kg/hr
  - Acceptable SpO_2 on 50 % FiO_2
  - Awake enough to follow commands
- Weaning:
  - Wait for 10 minutes after discontinuing propofol infusion to start weaning
  - 10–30 minutes taken to wean ventilatory support
  - Place patient on CPAP when adequate spontaneous efforts
  - Use E_TCO_2 and SpO_2 to assess adequacy of ventilation
  - Maintain:
    - E_TCO_2 < 50 mm Hg
    - SpO_2 > 94%.
- Extubation:
  - Leave fentanyl infusion on as needed
  - Continue monitoring O_2 saturation, ABG, BP, ECG and urine output.

PERIOPERATIVE MYOCARDIAL INFARCTION[1,3,5]

Introduction

- Rise or fall in troponin I levels (215 IU/ml), or other biomarkers with:
  - Cardiac symptoms
  - ECG changes:
    - ST depression more than 1 mm, 60–80 msec after J point in at least 3 consecutive heart beats with stable baseline
    - ST elevation:
      - In men > 40 yrs old, more than 2 mm in V2–V3 or 1 mm in all other leads
      - In men < 40 yrs old, more than 2.5 mm in V2–V3 or 1 mm in other leads
      - In women, more than 1.5 mm in V2–V3 or 1 mm in other leads.
    - T wave inversion with significant ST changes
  - Imaging findings.

Causes

- Tachycardia:
  - More than 110 bpm associated with high risk
  - Most important determinant of myocardial oxygen consumption
  - Heart rate > 80–90 bpm in patients with preoperative resting heart rate 50–60 bpm increases risk.
- Hypotension
- Hypertension
- Anemia with hematocrit < 28%
- Hypoxemia, hypercarbia
- Hypothermia
- Systolic and diastolic myocardial dysfunction.

Differential Diagnosis

- Anxiety
- Esophageal reflux
- Hyperventilation
- Hypo/hyperkalemia.

Types of Perioperative MI

Two types of PMI:

- Type I: Acute coronary syndrome
- Type II: Myocardial oxygen demand-supply imbalance.

Pathophysiology

- Type I Perioperative MI:
  - Occurs in the presence of an unstable coronary plaque
  - Plaque erosion or rupture occurs due to:
    - Sympathetic overactivity
    - Hemodynamic instability with hypotension and tachycardia
    - Coronary vasoconstriction.
  - This leads to acute coronary thrombosis in the setting of:
    - Increased coagulability with decreased fibrinolysis
    - Recent PCI with stent
    - Premature cessation of Dual Antiplatelet Therapy (DAP)
  - This leads to acute coronary syndrome type of perioperative MI.
- Type II Perioperative MI:
  - Occurs in the presence of severe stable coronary artery disease
  - Occurs due to imbalance between myocardial oxygen demand and supply:
    - Increased myocardial oxygen demand may occur due to:
      - Increased heart rate due to:
        - Increased sympathetic activity
        - Withdrawal of β-blocker therapy
        - Arrhythmias.
      - Increased myocardial wall stress due to:
        - Hypertension
        - Increased LVEDP
        - Pulmonary congestion
        - Atelectasis.
    - Reduced subendocardial oxygen supply may occur due to:
      - Hypovolemia with hypotension
      - Systemic vasodilatation
      - Cardiac decompensation
      - Perioperative anemia
      - Hypoxemia.
  - This leads to prolonged ST depression (more than 30 minutes), causing type II perioperative MI.

Diagnosis[3,5]

Method	Information obtained	Problems	Sensitivity
TEE	• New RWMA • Severe hypokinesia • Akinesia/ dyskinesia • Transmural MI	• Only one view at a time	• Highest
Coronary sinus lactate level	• Myocardial lactate production		
PCWP	• Raised PCWP • *a and c* waves	• Poor • sensitivity and specificity	
ST segment monitoring	• ST depression • ST elevation, T wave changes	• Requires V_4R for RCA • V_7,V_9 leads for post. wall ischemia	
Systemic pressures	• Fall in arterial pressure • Increase in CVP	• Low sensitivity and specificity	• Lowest

Cardiac Biomarkers

Biomarkers used:

- Troponin I
- CK-MB
- Lactate dehydrogenase
- Myoglobin
- Glycoprotein-BB
- AST, C-reactive protein, brain natriuretic peptide (BNP), myocardial lactate.

Biomarker[8]	Onset	Peak	Duration
myoglobin	Immediate	2 hrs	24 hrs
GP - BB	1–3 hrs	7 hrs	36 hrs
Troponin	2–4 hrs	12 hrs	7–10 days
CK-MB	4–8 hrs	10–24 hrs	48–72 hrs
LDH	24–48 hrs	72 hrs	10–14 days

Perioperative MI Predictors

- None are reliable predictors
- Rate pressure product (RPP)and triple index (TI):
  - Rate pressure product = heart rate × Systolic blood pressure
  - Triple index = Heart rate × Systolic BP x PCWP.
  - If RPP ≥ 20,000, patient is at increased risk of PMI
  - Both predictor are not reliable
- *Myocardial oxygen supply*: Demand ratio (DPTI: SPTI)
  - DPTI = Diastolic pressure time index = (MDP – LVEDP) × (duration of diastole)
  - SPTI = Systolic pressure time index = MAP × duration of systole
  - MDP = Mean diastolic pressure
  - MAP = Mean arterial pressure
  - If ratio less than 0.5, patient is at increased risk for subendocardial ischemia
  - Index is unreliable as increase in MvO_2 due to contraclility is not reflected.
- Mean arterial BP-heart rate quotient (pressure-rate quotient):
  - Pressure rate quotient = (Mean arterial pressure)/(Heart rate)
  - PRQ less than 1 predicts increased risk of ischemia
  - Heart rate ≥ 110 bpm is most important determinant of PMI.

Prophylaxis

Intervention[1,3,5,8]	Regimen	Recommendation
Perioperative β blockade	• Atenolol 50-100 mg started 30 days before surgery • Therapy continued IV during surgery • Long term β blockers not discontinued	Class I
α_2 agonists	• 300 µg clonidine (2-5 µg/kg) PO 90 min before surgery • Clonidine continued for 72 hrs postop at 200 µg/kg	Class IIa
Statin therapy	• Atorvastatin 20 mg OD started 45 days before surgery • Continued till 2 weeks postoperatively	Class IIa
ACE inhibitors	• Continue preoperative ACE inhibitor therapy • Watch for intraoperative hypotension	Class IIb
Calcium channel blockers	• Not used as individual agents preoperatively • Continue preoperative CCB therapy	Class IIb
Aspirin	• To be stopped 5–7 days before surgery to reduce bleeding • Only exception is CABG where it is continued	
Dual anti-platelet therapy	• Elective surgery discouraged for 4 weeks after BMS • Elective surgery discouraged for 1 year after DES • DAP therapy has to be continued uninterrupted in this time • Restart thienopyridine as soon as possible after surgery • Aspirin to be continued preoperatively	
Other measures	• Use of regional anesthesia where possible • Thoracic epidural reduces incidence of PMI • Use of volatile anesthetics for preconditioning • Maintain hematocrit ≥ 28 % • Maintain normothermia • Use of postoperative sufentanyl 1 µg/kg/hr	
Coronary revascularization	• PCI provides less complete revascularization • PCI associated with more perioperative complications • CABG provides complete revascularization • CABG is associated with less incidence of PMI	DECREASE trial CARP trial Class IIb
NTG	0.5–3 µg/kg/min	Class III

Goals of Perioperative Management

- Careful perioperative monitoring
- Low threshold for treating and preventing tachycardia
- Avoid hypotension, low cardiac output and cardiac decompensation.

Perioperative Management

- Tight perioperative monitoring:
  - ECG
  - Echocardiography
  - Transesophageal echocardiography
  - Invasive blood pressure
  - PA pressure monitoring.
- Additional tests:
  - Arterial blood gas, troponin I levels
  - Treat any acid base disturbances
  - Correct electrolyte imbalance.
- Tachycardia treated aggressively:
  - Tachycardia with hypotension:
    - *Hypovolemia*: IV fluid challenge
    - *Anemia*: Transfuse blood if:
      - Hematocrit less than 25% normally
      - Hematocrit less than 30% in patients with coronary artery disease.
    - Low systemic vascular resistance:
      - Vasopressors added
      - Cut off volatile anesthetics.
    - *Cardiac failure*: Inotropes, IABP
  - Tachycardia with hypertension or normotensive:
    - Beta blocker therapy:
      - Propranolol 0.5 mg boluses up to maximum of 0.1 mg/kg
      - Metaprolol 2.5 mg/kg increments up to maximum of 0.5 mg/kg
      - Esmolol 0.5 mg/kg bolus followed by 50 µg/kg/min.
    - Calcium channel blockers or nitroglycerine
    - Check appropriate pain control, supplement opioids
    - If tachyarrhythmias, treat rate and rhythm.
- Emergency coronary intervention/anticoagulation with GP-IIbIIIa antagonists only if:
  - Persistent ST depression/elevation (lasting more than 30 minutes)
  - Intractable cardiogenic shock.

ISCHEMIC PRECONDITIONING[1,2,3,5,17]

Introduction

- Myocardial ischemic preconditioning refers to the adaptive mechanism by which a brief period of reversible ischemia increases the heart's tolerance to subsequent longer periods of ischemia
- Anesthetic preconditioning refers to administration of volatile anesthetics before prolonged coronary artery occlusion in order to increase the *critical ischemia time* (CIT_{50})
- CIT_{50} is the duration of circulatory disruption, compatible with 50% tissue survival.

Conditions Which Benefit from Preconditioning

- Extreme CAD with poor collaterals
- Severe LVH with reduced subendocardial perfusion
- Anticipated prolonged ischemic time
- Senescent myocardium prone to tissue damage from calcium overload.

Agents used for Preconditioning

- *Volatile anesthetics*: Isoflurane, halothane, sevoflurane, desflurane
- *Adenosine*: Acts through A1, A2 and A3 receptors
- Acadesine
- Acetylcholine
- Carbechol
- *Opioids:* Acts through δ_1 and K receptors.

Beneficial Effects of Volatile Anesthetics

- Altered neutrophil function
- Improves coronary perfusion
- Reduces myocardial oxygen demand
- Preserves energy dependent vital cellular processes
- Negative inotropic effects
- Negative chronotropic effects
- Negative lusitropic effects
- Reduction in LV afterload.

Beneficial Effects of Individual Agents

- Halothane:
  - Attenuates ST changes caused by brief coronary artery occlusion
  - Preserves contractile function and ultrastructural integrity at reperfusion
  - Inhibits platelet thrombus formation by increasing platelet cAMP.

- Isoflurane:
  - Beneficial to LV diastolic function during ischemia
  - Reduces impact of reperfusion injury
  - Increases ratio of myocardial oxygen supply: Demand ratio
  - Reduces troponin I and CK-MB levels after cardiac surgery.
- Sevoflurane:
  - Reduces impact of reperfusion injury
  - Increases blood flow to collateral dependent myocardium via action on calcium activated K^+ channels (BK_{ca})
  - Improves recovery of coronary vascular reactivity
  - Enhances endometrial nitric oxide release.
- Desflurane:
  - Beneficial to LV diastolic function during ischemia
  - Attenuates effects of free radicals on LV function
  - Reduces adhesion of neutrophils and platelets after myocardial ischemia and reperfusion.

Beneficial Effects of Preconditioning

- *CABG:* Better ejection fraction and cardiac index after CABG
- *OPCAB:* Preconditioning less used now due to use of coronary shunts intraoperatively
- *Heart transplant:* Improves contractile function after global myocardial hypothermia, which occurs when donor heart is stored
- *Cardiomyoplasty:* Skeletal muscle preconditioning prior to cardiomyoplasty augments systolic function and limits diastolic dysfunction.

Types of Preconditioning

- *Early preconditioning*: Limited to 1–3 hours after brief ischemic stimulus
- Delayed preconditioning:
  - Occurs after 24 hours of volatile anesthetic exposure
  - Lasts as long as 72 hours
  - Maybe species specific (occurs with sevoflurane but not isoflurane)
  - Late or second window phase.
- Post conditioning:
  - Myocardial protection provided by volatile anesthetics at the time of reperfusion
  - Administered immediately before or during reperfusion
  - 4% sevoflurane during first 10 minutes of CPB reduces postoperative release of BNP (marker of LV dyfunction).

Biochemical Mechanisms of Preconditioning

- Upregulation and activation of mitochondrial and sarcolemmal KATP channel: Acts by reducing coronary vascular tone
- G proteins and coupled receptor ligands:
  - Due to activation of δ_1 opioid receptors
  - These in turn activate G_i and G_s protein.
- *Protein kinases*: PKC, PKG and $P_{13}K$ involved in cardioprotective signal transduction
- Reactive oxygen species:
  - Volatile anesthetics stimulate small bursts of reactive oxygen species (superoxide, H_2O_2 and peroxynitrite)
  - These paradoxically initiate downstream signaling and protect from subsequent ischemic injury.
- Adenosine receptor (especially α_1 and α_2 subtypes)
- Tyrosine kinase
- Na^+: H^+ exchanger
- COX_2, NFKB and NO systems are implicated for delayed preconditioning.

Inhibitors of Preconditioning

- Adenosine antagonists
- δ_1 opioids
- Glibenclamide:
  - As they close K_{ATP} channels
  - Discontinue glibenclamide 24–48 hours preoperatively
- Pertussis toxin
- Hyperglycemia.

ANESTHESIA FOR PATIENTS WITH INTRACORONARY STENTS[1,2,3,5,11,17]

Introduction

Success of stents requires long term antiplatelet therapy and thus, perioperative management of these patients poses special issues.

Types of Stents

- Bare metal stents (BMS):
  - Eliminated acute vessel closure (occurs within 24 hours) which was seen with PTCA

  - However, acute (< 24 hrs) and subacute (24 hrs-30 days) stent thrombosis can still occur
  - Incidence of stent rethrombosis is reduced with dual antiplatelet therapy
  - Restenosis rate is however high
  - This is due to neointimal hyperplasia (medial hyperproliferation) in coronary artery
  - This occurs due to endothelial damage from free ends of the stent.
- Drug eluting stent (DES):
  - Bare metal stent is coated with polymer containing sirolimus/paclitaxel
  - Sirolimus and paclitaxel inhibit neointimal hyperplasia
  - This lowered restenosis rate seen with bare metal stents
  - However, higher incidence of:
    - Late stent thrombosis (> 30 days)
    - Very late stent thrombosis (> 1 year).
  - Risk factors for thrombosis after drug eluting stent:
    - Acute coronary syndrome
    - Diabetes mellitus, old age
    - Renal impairment
    - LVEF ≤ 30%
    - Premature discontinuation of dual antiplatelet therapy (DAP).

Dual Antiplatelet Therapy

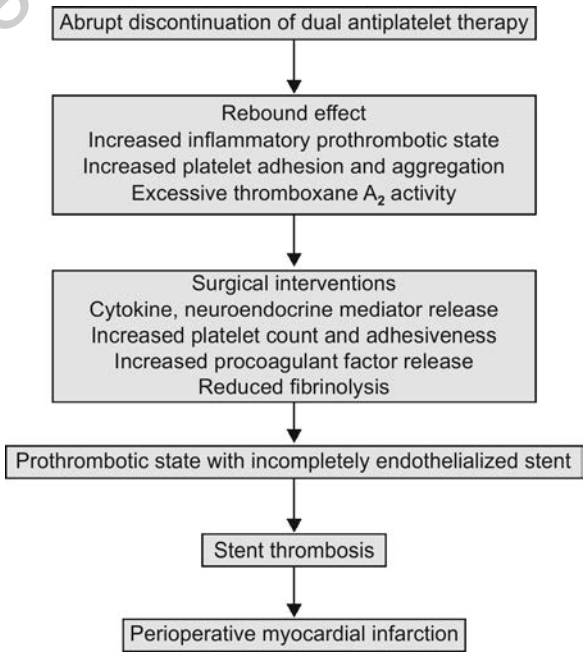
ACC-AHA 2007 recommendations:

- Bare metal stents:
  - Loading dose of 300–600 mg clopidogrel before implantation
  - Aspirin 75–100 mg with clopidogrel 75 mg given for 4–6 weeks postprocedure to allow stent endothelialization
  - Low dose aspirin continued thereafter, for life, as secondary prophylaxis
  - Elective surgery postponed beyond 6 weeks of stent placement (but before 12 weeks when restenosis can occur).
- Drug eluting stent:
  - Loading dose of 300–600 mg clopidogrel given before implantation
  - Aspirin 75–100 mg with clopidogrel 75 mg given for 12 months poststent placement
  - Thereafter, continue low dose aspirin life long, for secondary prophylaxis
  - Postpone elective surgery for more than 12 months after stent placement.
- PTCA alone:
  - Dual antiplatelet therapy given for 2–4 weeks after stent placement
  - Elective surgery postponed for more than 2–4 weeks after stent placement.
- Consider using BMS/PTCA rather than DES in patients due for noncardiac surgery within 12 months
- Discontinue antiplatelet therapy only after discussion with cardiologist.

Perioperative Anesthetic Problems

- *Increased risk of bleeding*: Dangerous in closed compartment surgeries (neurosurgery)
- Acute perioperative stent thrombosis
- Complications arising due to simultaneous neuraxial blockade.

Pathophysiology of Acute Perioperative Stent Thrombosis



Risk Factors for Perioperative Thrombosis with DES

- Factors related to the stent:
  - Stent in LMCA
  - Stent in bifurcation/crossing arterial branch points

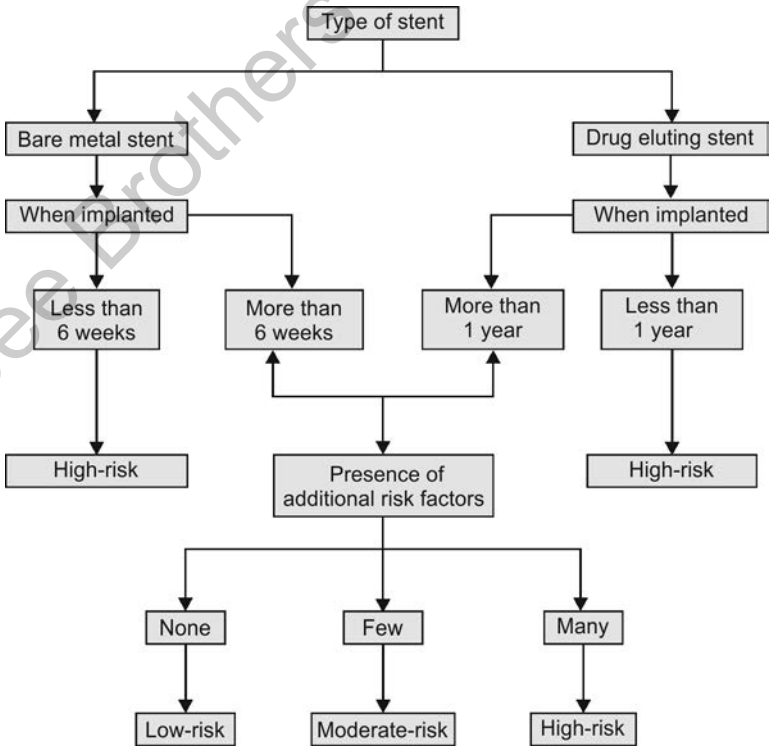
  - In-stent restenosis
  - Greater total stent length, multiple stents
  - Small stent diameter (less than 3 mm).
- Factors relating to patient:
  - Heightened platelet activity (surgery, cancer, diabetes mellitus)
  - Localized hypersensivity vasculitis (due to anti-proliferation drugs)
  - LV dysfunction
  - Diabetes mellitus
  - Plaque disruption into necrotic core
  - Advanced age, prior brachytherapy.
- Factors relating to dual antiplatelet therapy:
  - Resistance to antiplatelet therapy
  - Interruption of DAP therapy within 14 days of stent insertion is the most important factor
  - Inappropriate discontinuation of antiplatelet therapy.

Preanesthetic Evaluation

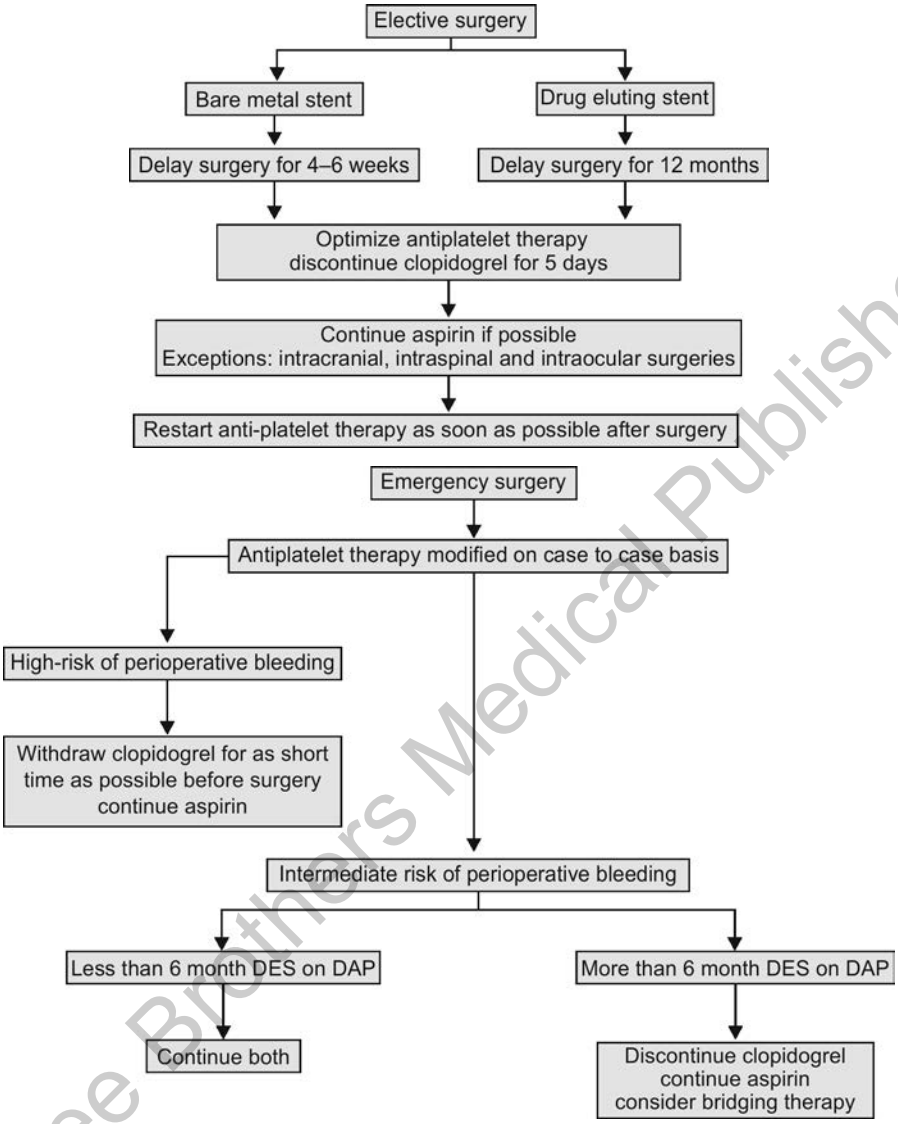
- History:
  - Duration since PCI.
  - Type of stent: BMS/DES
  - Number of stents
  - Location of stents
  - History of adverse cardiac events
  - Previous history of stent thrombosis
  - Drug regimen and any irregularities in the regimen
  - Urgency of surgery
  - *Comorbidities*: DM, CRF, low LVEF
  - History of conditions prone for thrombosis
  - Cardiology consultation.
- Investigations:
  - Routine blood investigations
  - Platelet count and function
  - Bleeding and clotting time
  - Renal function tests
  - Arrange whole blood and platelet concentrates.

Determining Risk of Stent Thrombosis

Circulation 2007



Perioperative Management



Rationale of Continuing Aspirin

- No difference in blood loss between aspirin users and non-users in OPCAB patients
- Protective effects of aspirin provided in vascular surgeries:
  - Improved long term peripheral bypass graft potency
  - Reduced incidence of MI, TIA, stroke, death.
- Increases incidence of bleeding by a factor of 1.5
- Aspirin did not increase severity or perioperative morbidity except in:
  - Intracranial surgeries
  - Spine surgeries
  - Intraocular surgeries
  - TURP (possibly).

Bridging Therapy

- Short acting GP-II_b/III_a antagonists (tirofiban/eptifibatide) is substituted for clopidogrel during perioperative period, if there is high risk of bleeding
- Heparin has no antiplatelet action and is not protective against stent thrombosis
- Use GP-II_b/III_a antagonists in:
  - Patients who have not completed dual anti-platelet therapy
  - Patients whose stent complexities increases risk of developing stent thrombosis.

Dual Antiplatelet Therapy and Neuraxial Blockade

- Neuraxial block not recommended until:
  - Platelet function is within normal limits, or
  - Platelet transfusion is given before surgery.
- If neuraxial blockade is planned:
  - Clopidogrel stopped 7 days before surgery
  - Ticlopidine stopped 14 days before surgery
  - Aspirin alone is not a contraindication for neuraxial blockade
  - Those receiving bridging therapy:
    - Discontinue abxicimab for 48 hrs before NAB
    - Discontinue eptifibatide and tirofiban for 48 hrs before NAB.

PREOPERATIVE ASSESSMENT OF CARDIAC PATIENT FOR NONCARDIAC SURGERY[1,2,3,5,17]

Goals

- To identify risk for heart disease based on risk factors
- To evaluate presence and severity of cardiac disease through symptoms, physical findings and diagnostics tests
- To determine the need for preoperative interventions
- To modify risk factors for perioperative adverse events.

Risk Indices

- Before 1990:
  - NYHA, ASA indices
  - Goldman, Cooperman
  - Detsky, Larsen
  - Penderson, Vanzetto.
- After 1990:
  - ACC/AHA, ACP
  - Lee, ACC updated.

Goldmans Cardiac Risk Index[3]

Characteristic	Score
third heart sound, jugular vein distension	11
Recent MI	10
Nonsinus rhythm, paroxysmal atrial complexes	7
More than 5 ventricular ectopic beats	7
Age more than 70 years	5
Emergency surgery	4
Poor general condition	3
Intrathoracic/intraperitoneal/aortic surgery	3
Aortic stenosis	3

Goldmans score	Cardiac complication rate
0–5 points	1%
6–12 points	7%
13–25 points	14%
More than 26 points	78%

Lee's Revised Cardiac Risk Index (RCRI)

Introduction

It has been validated in several studies as the best scoring system to predict perioperative cardiac risk in patients undergoing cardiac surgery.

Importance

- Traditional risk factors for CAD are:
  - Smoking
  - Male gender
  - Hypercholesterolemia
  - Family history of CAD.
- These are not the same factors which are associated with an increased incidence of perioperative cardiac events
- These are, however, important in assessing significance of chest pain, dyspnea or abnormal ECG.

Components

- High risk surgery:
  - Intraperitoneal surgeries
  - Intrathoracic surgeries
  - Suprainguinal surgeries
  - Vascular surgery.
- Ischemic heart disease by any diagnostic criteria:
  - History of angina
  - Use of sublingual nitroglycerine
  - Positive exercise test
  - Significant Q waves on ECG
  - Patient who has undergone PTCA/CABG.
- Heart failure
- Cerebrovascular disease
- Insulin dependent diabetes mellitus
- Creatinine 2 mg/dl.

Risk of Major Cardiac Events

- 0 risk factors: 0.4 % risk
- 1 risk factor: 0.9 % risk
- 2 risk factors: 7 % risk
- 3 risk factors: 11 % risk

Detsky Modified Multifactorial Index

No.	Characteristic	Score
1	Class IV angina	20
2	Suspected critical aortic stenosis	20
3	History of MI within the last 6 months	10
4	History of pulmonary edema within last 1 week	10
5	Class III angina	10
6	History of unstable angina within the last 3 months	10
7	Emergency surgery	10
8	History of MI more than 6 months back	5
9	History of pulmonary edema more than 1 week back	5
10	Nonsinus rhythm/PAC	5
11	More than 5 ventricular ectopic beats	5
12	Poor medical status	5
13	Age more than 70 years	5
Score more than 15 suggests high risk		

Eagles Criteria for Risk Assessment

Characteristic	Score
age more than 70 years	1
Diabetes mellitus	1
Angina	1
Pathological Q waves	1
Ventricular arrhythmias	1

Score	Significance
Less than 1	No tests indicated
1–2	Noninvasive testing warranted
More than 3	Angiography warranted

Preoperative Evaluation

- History:
  - Presence, severity and reversibility of CAD:
    - Risk factors:
      - Age
      - HTN, DM
      - Cholesterol
      - Smoking
    - Angina pattern:
      - Stable/unstable
      - Medications
      - Aggravating/relieving factors
    - Previous history of MI
  - Myocardial function:
    - Exercise capacity
    - Pulmonary edema
    - Orthopnea, PND, edema
    - NYHA classification:
      - Class 1:
        - No limitations of physical activity
        - On ordinary activity, no fatigue/syncope/palpitations
      - Class II:
        - Slight limitation of physical activity
        - Fatigue/palpitations/syncope on ordinary activity
      - Class III:
        - Marked limitation of activity
        - Fatigue on less than ordinary activity
      - Class IV: Symptoms occurring at rest.
  - Valvular heart disease:
    - Dyspnea, orthopnea, PND
    - History of medications
    - Hemoptysis
    - Embolic events.
  - Associated cerebral, cardiovascular, carotid, aortic vascular disease
  - Prior cardiac evaluation: Noninvasive testing, angiography.
- Physical examination:
  - Vital signs: Pulse, blood pressure, respiratory rate
  - Cardiac examination:
    - JVP, peripheral edema
    - Displaced apical impulse: Cardiomegaly
    - S_3 gallop/S_4 gallop
    - Apical systolic murmur
    - Pulmonary edema.
- Diagnostic tests:
  - Routine blood tests, blood urea, serum creatinine
  - *Serum electrolytes*: Especially in patients on diuretics
  - If Hb 10 g%, transfuse blood in those with low cardiovascular reserve
  - Brain natriuretic peptide ≥ 100 pg/ml indicates high risk of CCF
  - Chest X-ray:
    - Cardiomegaly
    - *Ventricular dysfunction*: Increased vascular markings edema
    - Pleural/pericardial effusion.
  - ECG:
    - Should not be ordered simply because of advanced age
    - Only certain abnormalities are significant in preoperative assessment:

    - ° Q waves, especially if recent
    - ° Conduction abnormalities and arrhythmias
  - Establishing baseline for comparison is most important reason to obtain ECG preoperatively
  - If previous ECG available and no change in risk factors/no new physical finding, it is unlikely that repeat ECG will be helpful
  - *ACC-AHA 2007 recommendations*:
    - ° Class I recommendations:
      - * Should be performed
      - * For patients with atleast 1 clinical risk factor undergoing vascular surgery
      - * Those undergoing intermediate risk surgery with known case of:
        - → Congestive cardiac failure
        - → Peripheral arterial disease
        - → Cerebrovascular accident.
    - ° Class IIa recommendations:
      - * Reasonable to perform
      - * For patients with no clinical risk factors undergoing vascular surgery.
    - ° Class IIb recommendations:
      - * Maybe considered
      - * For patients with atleast 1 clinical risk factors undergoing vascular surgery
    - ° Class III recommendations:
      - * Should not be performed and not useful
      - * Asymptomatic patients undergoing low risk surgeries.
- Stress testing:
  - For patients with normal ECG who can exercise
  - Patients should be likely to achieve adequate heart rate response
  - Test is adequate when patient can achieve 85% of target heart rate
  - Target heart rate = 220 – age in years
  - Positive predictive value of 5–25%
  - Negative predictive value of 100%
  - Thus, it gives more information about patient who will not have an event rather than one who will.
- Dobutamine stress test:
  - Useful for patients with normal ECG but unable to exercise
  - Avoided in:
    - ° Poorly controlled hypertension
    - ° Bradycardia.
    - ° Aortic/cerebral aneurysm
    - ° Patients with pacemaker.
- Nuclear perfusion imaging:
  - For patients unable to exercise and in whom dobutamine test is contraindicated
  - Myocardium said to have limited blood flow if it is normal at rest but shows reduced isotope uptake with exercise.
- Echocardiography:
  - For evaluating RWMAs and LV ejection fraction
  - Abnormal movement at rest indicates scar tissue
  - Areas which are normal at rest but with abnormality which increases with increasing inotropy and chronotropy indicates stenotic lesions and limited blood flow
  - Classification of LV dysfunction:
    - ° EF ≥ 50%: Normal
    - ° EF 41–49%: Mildly LV dysfunction
    - ° EF 26–40%: Moderate LV dysfunction
    - ° EF ≤ 25%: Severe LV dysfunction.
- Cardiac catheterization: Does not absolutely risk stratify patients.

Dukes Activity Status Index

1 MET is the oxygen consumption by a resting adult (3.5 ml/kg/min or 250 ml/min).

- Dukes status 1 MET:
  - Able to take care of self
  - Eat, dress, use toilet
  - Walk indoors or on level ground at 3.2–4.8 km/hr
- Dukes status 4 METS:
  - Light housework: Dusting, washing dishes
  - Climbs flight of stairs
  - Walks on level ground at 6.4 km/hr or 4 mph
  - Walks 1–2 blocks
- Dukes status 7 METS: Playing singles tennis, dancing
- Dukes status 10 METS:
  - *Strenous sports:* Swimming, singles tennis, basket ball, football
  - Running rapidly for moderate to long distances
- Significance:
  - *Poor functional capacity:* Less than 4 METS
  - *Moderate functional capacity:* 4 to 7 METS
  - *Good functional capacity:* 7 to 10 METS.

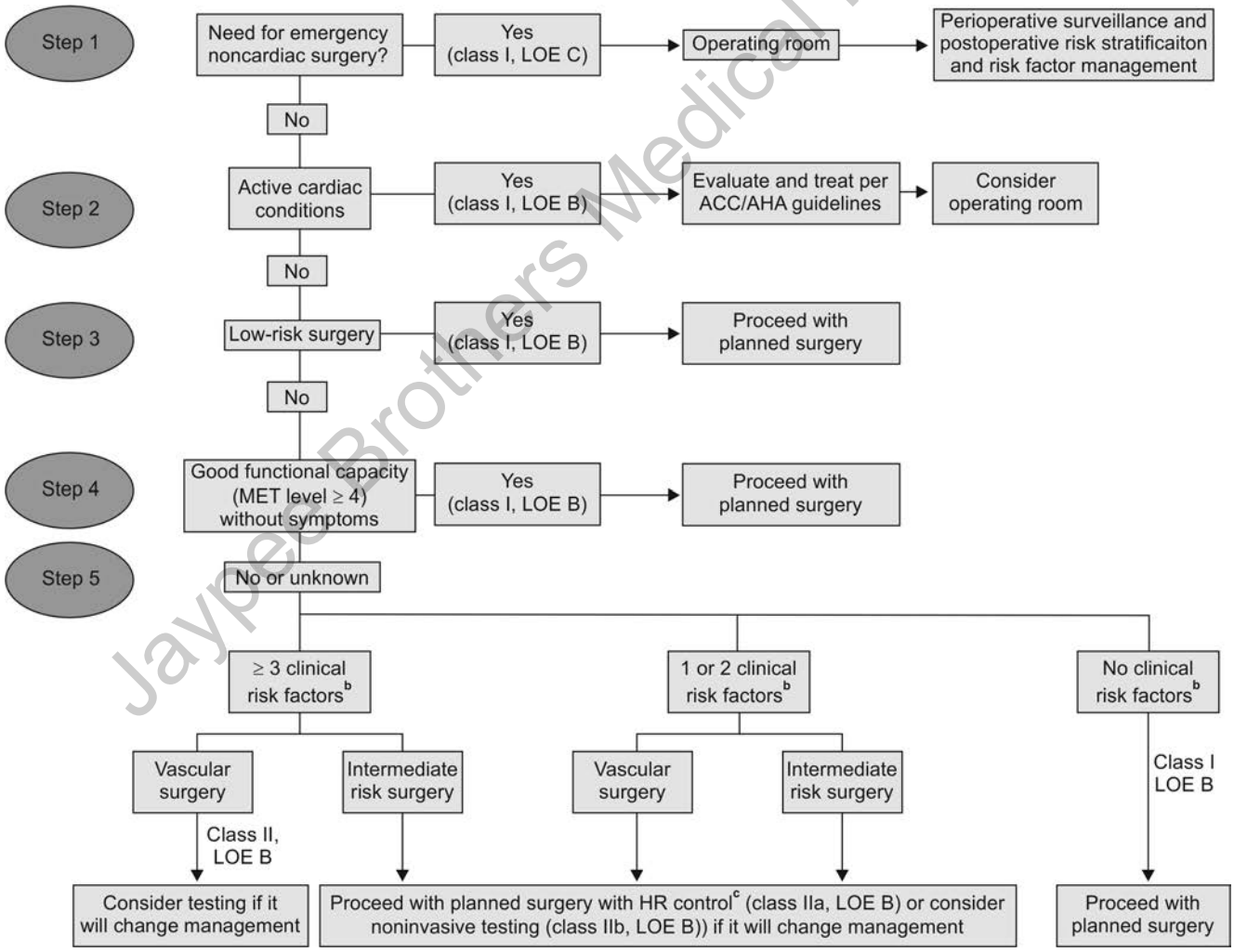
Surgery Specific Risk Factors

- High risk surgery (> 5% risk):
  - Emergency major surgery in elderly
  - Prolonged surgical procedures associated with large fluid shifts/blood loss
  - Aortic/other major vascular surgery
  - Peripheral vascular disease.
- Intermediate risk surgery (< 5% risk)
  - Carotid endarterectomy
  - Head and neck surgery
  - Intrathoracic surgery
  - Intraperitoneal surgery
  - Orthopedic surgery
  - Prostrate surgery.
- Low risk (< 1% risk):
  - Superficial surgeries
  - Endoscopic surgeries
  - Breast, cataract surgery.

ACC-AHA 2007 Perioperative Guidelines *(See Flow Chart)*

Risk factors are:

- Ischemic heart disease
- History of congestive cardiac failure
- History of cerebrovascular accidents
- Diabetes mellitus
- Serum creatinine more than 2 mg/dL.



INTRA-AORTIC BALLOON PUMP[1,2,3,5,17]

Introduction

- Circulatory assist device (CAD) which is designed to augment myocardial perfusion, by increasing coronary blood flow during diastole and unloading left ventricle during systole
- Simplest and most readily available CAD.

Major Types of Circulatory Assist Device

- Counter pulsation devices:
  - IABP
  - Noninvasive counter pulsations.
- Cardiopulmonary assist devices: ECMO
- Ventricular assist devices:
  - LVAD
  - RVAD
  - BiVAD.

Classification of CAD

- By indication:
  - Urgent (unstable angina/uncontrolled CCF):
    - IABP
    - VAD.
  - Emergent (uncontrolled hypotension, inability to wean off CPB):
    - IABP
    - ECMO.
- By duration of therapy:
  - Short term:
    - IABP
    - Abiomed VAD
    - ECMO.
  - Long term:
    - Thoracic VAD
    - Implantable VAD
- By degree of support provided:
  - *Augmentation*: IABP
  - LVAD/RVAD alone
  - Total support (Bivad/ECMO).

Composition of IABP

- 25 mm sausage shaped balloon
- Mode of nonthrombogenic polyurethane
- Mounted on 90 cm long vascular catheter
- Helium/carbon dioxide used as distending gas (helium is preferred)
- Volume of balloon is 30–50 ml.

Insertion of IABP

- Inserted into femoral artery either percutaneously/via surgical exposure
- Length to be inserted calculated by measuring distance between angle of Louis and femoral artery
- Distal tip lies below left subclavian artery (to prevent emboli to brain)
- Proximal tip lies above renal artery
- When peripheral vascular disease present, IABP inserted via ascending aorta after median sternotomy.

Other Approaches

- *Ascending aorta:* If PVD present
- *Iliac artery:* In children where size of femoral artery is small
- Subclavian artery
- Axillary artery.

Indications

- Cardiogenic shock:
  - Postmyocardial infarction
  - Myocarditis
  - Cardiomyopathy
  - Pharmacological.
- Failure to wean from CPB:
  - Right/left ventricular failure
  - Increased inotropic requirements
  - Hemodynamic deterioration.
- Stabilization of cardiac patient for cardiac surgery:
  - MI with VSD
  - MI with mitral regurgitation
  - Unstable, refractory acute coronary syndrome.
- Stabilization of unstable patient for noncardiac surgery:
  - Emergency cholecystectomy
  - Gastrectomy, enterolysis
  - Repair of parastomal hernia
  - Resection of intracranial masses
  - Septic shock.
- Procedure support during coronary angiography
- As a bridge to transplantation
- Trauma/hemorrhagic shock.

Contraindications

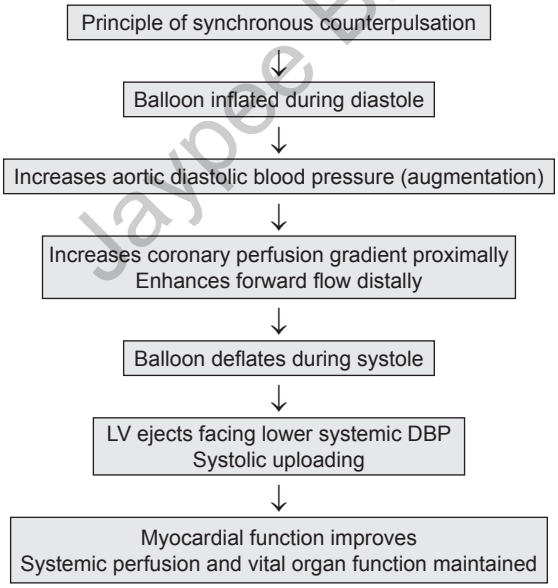
- Absolute contraindications:
  - Abdominal aortic dissection/aneurysm
  - Severe calcific aorta
  - Peripheral vascular disease
- Relative contraindications:
  - Severe AR
  - Irreversible cardiac disease (not a transplant candidate)

- Irregular rhythm
- Irregular pulse pressure
- Irreversible brain damage
- Severe noncardiac systemic disease
- Massive trauma, Do Not Resuscitate (DNR) patients
- Local infection, inability to insert.

Timing and Goal of Inflation and Deflation

- Inflation:
  - Timed to coincide with aortic valve closure
  - Produces rapid rise in diastolic BP
  - Inflated just prior to dicrotic notch
  - Late inflation: Reduces perfusion pressure to coronary artery
  - Early inflation:
    - Called early if inflation occurs more than 40 msec before dicrotic notch
    - Causes aortic regurgitation and LV strain.
- Deflation:
  - Reduces afterload by reducing aortic end-diastolic pressure
  - This reduces myocardial oxygen demand and increases cardiac output
  - *Early deflation*: Causes inappropriate loss of afterload reduction
  - *Late deflation*: Will increase LV work by causing increased afterload
- Factors affecting IABP performance:
  - Position of balloon within the aorta
  - Balloon volume
  - Cardiac rhythm.

Principles[1,5]



IABP Triggers

- *ECG:* More than 120 µV deflation/R wane
- Pacemaker spike
- *Arterial pressure:* More than 15 mm Hg deflation
- Internal trigger.

Associated Therapies

- Heparin to prevent thrombus
- Increased inotropic support while weaning off IABP
- Antibiotic cover
- IV fluids to maintain preload.

Limitations

- Not useful when LV is unable to eject blood into aorta
- Not useful in irregular/fast cardiac rhythms.

Weaning Criteria

- Clinical criteria:
  - Absence of shock syndrome (hypotension, cool extremities)
  - Minimal need for vasopressors (< 5 µg/kg/min dopamine)
  - Cardiac catheterization/major surgery is planned.
- Hemodynamic criteria:
  - Cardiac index more than 2.2 L/min/m^2
  - Wedge pressure < 18 mm Hg
  - Mean BP > 70 mm Hg
  - Less than 10% change in cardiac index, wedge pressure and heart rate during weaning.
- Balloon augmentation is reduced in steps from 1:1 counterpulsation to 1:2, 1:4
- Appropriate intervals required to assess:
  - Hemodynamic and neurological stability
  - Cardiac output and MvO_2
- After appropriate observation at 1:8 counterpulsations, IABP can be removed.

Complications

- Vascular complications:
  - Arterial injury (perforation/dissection)
  - Aortic dissection
  - Femoral artery thrombosis and pseudo-aneurysm
  - Peripheral embolization
  - Lower extremity ischemia (most common)
  - Compartment syndrome
  - Visceral ischemia (mesenteric infarction)
  - Femoral venous catheterization.

- Miscellaneous:
  - Hemolysis, thrombocytopenia
  - Infection, sepsis
  - Claudication pain (after removal)
  - Hemorrhage
  - Paraplegia, spinal cord ischemia
  - Entrapment of balloon
  - Left internal mammary artery occlusion
  - Coagulopathies.
- Complications related to balloon:
  - Perforation of balloon (preinsertion)
  - Balloon tear during insertion
  - Incorrect positioning
  - Gas embolism, cerebrovascular accidents
  - Inadvertent removal.

ENDOVASCULAR AORTIC REPAIR (EVAAR)[1,5,17]

Indications

- Aortic aneurysms
- Aortic dissection
- Aortic tears and rupture.

Surgical Technique

- Arterial access:
  - Most commonly bilateral femoral artery access
  - Depends on size of blood vessel and degree of obstructive arterial disease
  - Balloon dilation and local endarterectomy done if femoral artery is small
  - Adjunctive retroperitoneal procedure done if:
    - Femoral artery is very small
    - Iliac artery aneurysm is present.
- Delivery system:
  - Under fluoroscopic guidance along with stent grafts
  - Stent graft position confirmed by fluoroscopy/TEE.

Preoperative Preparation

- History and examination of patient
- Chest X-ray, CT scan and MRI to delineate aortic anatomy
- NPO guidelines
- Patient reassurance and consent
- Blood grouping and cross matching
- 10–15 units blood kept ready to cope with any emergencies
- Stabilize coexisting diseases and continue preoperative medications.

OT Preparation

SOAP-ME

- Suction apparatus
- Oxygen delivery systems, oxygen cylinders
- *Airway:* Laryngoscope, ET tube, oral airway, LMA
- *Pharmacy:* Thiopentone, propofol, fentanyl
- Monitors
- *Emergency drugs:* Atropine, adrenaline, dopamine, dobutamine, NTG, SNP
- Availability of cardiac surgeon and resuscitation and transport plan if aortic tear/rupture.

Monitors

- Pulse oximetry, E_TCO_2
- ECG, urine output
- Temperature, NIBP, TEE
- Radial arterial IBP:
  - Useful to assess volume, resuscitation and CPR
  - Useful for ABGs
  - Important as hypotensive anesthesia practiced.
- CVP and PA catheter usually not required
- Somatosensory evoked potentials, motor evoked potentials.

Anesthetic Technique

- Local/general anesthesia can be used
- GA is technique of choice as:
  - Procedure may be long
  - TEE may be required throughout the procedure
  - In case of any emergency, airway is already secured.
- If aneurysm is thoracoabdominal, bronchial blockers can be used
- In case of emergencies, blocker can be inflated and left lung can be isolated
- Systemic anticoagulation with heparin done before device placement
- Hypotensive anesthesia is used during placement of stent graft.

Contraindications

- Very short diseased segment
- Increased diameter of diseased segment
- Aortic wall disease of proximal segment.

Complications

- Migration of stent, kinking/leak around stent
- Paraplegia:
  - Common in thoracoabdominal aneurysms
  - Consider CSF drainage to maintain cerebral perfusion pressure.

- Contrast nephropathy:
  - Risk factors:
    - Diabetes, hypertension, old age
    - Renal insufficiency
    - Repeated exposure at short intervals
    - Use of high osmolality nonionic contrast
    - ACE inhibitor therapy.
  - Treatment:
    - Hydration, N-acetyl cysteine
    - Antioxidants, vasodilators
    - Dopamine and phenoldopam tried
    - Dialysis
    - Diuretics not used during first 24 hours.
- Stroke, abdominal pain, mesenteric ischemia
- Endovascular leaks:
  - Type I: Leak occurs between graft and proximal/distal segment
  - Type II: Leak occurring through collateral
  - Type III: Due to problem with in-stent graft.

Advantages

- Reduced mortality and morbidity
- Reduced blood loss
- Reduced postoperative pain
- Reduced ICU stay
- Reduced postoperative pulmonary complications
- Can be done in very old/very sick patients in whom open surgery is contraindicated.

Disadvantages

- Risk of converting to open surgery exists
- Risk of bleeding, stroke, paraplegia, endovascular leaks and contrast nephropathy.

PHYSIOLOGICAL CHANGES DURING AORTIC CROSS CLAMPING[1,2,3,5,17]

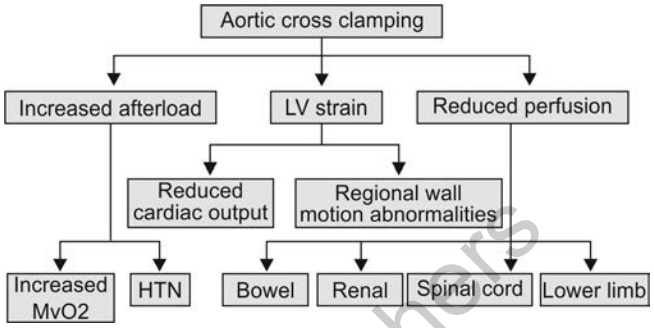
Introduction

Aorta at thoracic/abdominal level, is cross clamped during surgery of abdominal/thoracic/thoracoabdominal aneurysms.

Complications of Cross Clamping

- 30 day mortality: 8–35%
- Complete/partial paraplegia (16–38%)
- Myocardial infarction (11%)
- Respiratory failure (36%)
- Renal failure (18–27%)
- *GI complications*: Mainly hemorrhage: (7%)

Physiological Changes During Clamping



Hemodynamic Changes

- Increased preload
- Increased afterload
- Increased LV wall tension
- Increased regional wall motion abnormalities
- Increased arterial BP, systemic vascular resistance
- Reduced ejection fraction
- Reduced cardiac output
- Reduced renal and splanchnic blood flow
- Increased pulmonary capillary wedge pressure
- Increased central venous pressure
- Increased coronary blood flow.

Metabolic Changes

- Reduced total body oxygen consumption
- Reduced total body oxygen extraction
- Increased mixed venous oxygen saturation
- Reduced total body carbon dioxide production
- Increased epinephrine and norepinephrine levels
- Respiratory alkalosis
- Metabolic acidosis.

Renal Changes

- 80% reduction in renal blood flow with suprarenal clamping
- Redistribution of renal blood flow:
  - Increased blood flow to cortical and juxtamedullary layers
  - Reduced blood flow to ischemia-prone renal medulla.
- Renal hemodynamic changes persists for 30 min after systemic hemodynamics return to baseline
- Increased chances of dialysis dependent ARF:
  - 2–3% incidence regardless of clamp position
  - 13% incidence if post-suprarenal clamping
  - 5% incidence if postrenal clamping.
- Causes of acute renal failure:
  - Reduced glomerular filtration rate

- Reduced renal blood flow
- Ischemia reperfusion injury.

Visceral and Mesenteric Changes

- Reduced blood flow occurs through superior and inferior mesenteric artery
- Ischemia of left colon is more common due to reduced blood supply through inferior mesenteric artery
- Gut ischemia also causes increased gut permeability and bacterial translocation
- High dose methylprednisolone given at induction reduces this response
- Visceral ischemia aggravated by:
  - Preexisting medical conditions
  - Renal dysfunction
  - Stage of aortic disease
  - Level of cross clamping
  - Duration of cross clamping
  - Perioperative hypotension.

CNS and Spinal Cord Changes

- Reduced blood velocity in middle cerebral artery
- Unclamping causes transient dilatation followed by sustained vasoconstriction of pial blood vessels due to:
  - Hemodynamic changes
  - Carbon dioxide accumulation and wash-out
  - Acidosis
  - TXA_2 release
- Supraceliac cross clamping causes:
  - Reduced anterior spinal artery pressure
  - Increased CSF pressure
  - High CVP.

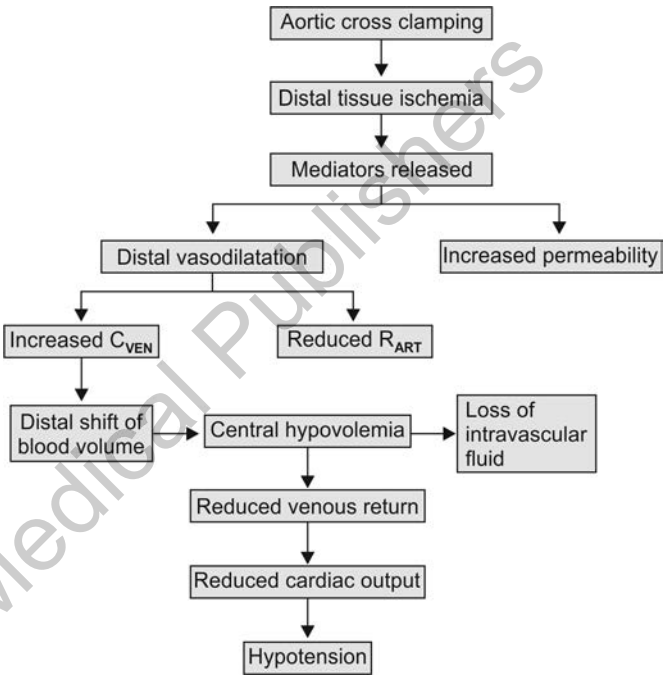
Coagulation Changes

- Increased clotting factor activity occurs during cross clamping
- Reduced speed of clot formation after unclamping.

Therapeutic Interventions

- Afterload reduction:
  - SNP, milrinone
  - Volatile agents
  - Aortofemoral bypass.
- Preload reduction:
  - NTG
  - Controlled phlebotomy
  - Atriofemoral bypass.
- Other changes:
  - Induced hypothermia
  - Reduced minute ventilation
  - Sodium bicarbonate.

Physiological Changes During Unclamping



Hemodynamic Changes

- Reduced afterload
- Distal pooling of blood
- Reduced venous return
- Reduced cardiac output
- Reduced myocardial contractility
- Reduced arterial blood pressure
- Increased pulmonary artery pressure
- Reduced central venous pressure.

Metabolic Changes

- Increased total body oxygen consumption
- Increased total body oxygen extraction
- Reduced mixed venous oxygen saturation
- Increased serum lactate levels
- Reduced activated complement levels
- Increased prostaglandin levels
- Increased myocardial depressant factor levels
- Metabolic acidosis
- Hypothermia.

Therapeutic Interventions

- Reduce volatile agents
- Increased IV fluid administration
- Increased vasoconstrictors
- Reduced vasodilators
- Reapply cross clamp if severe hypotension
- Consider mannitol
- Consider sodium bicarbonate administration.

Humoral Factors Which Cause Organ Dysfunction

- Acidosis
- Activation of RAS
- Activation of sympathetic nervous system
- Oxygen derived free radicals
- Prostaglandins
- Platelet and neutrophil sequestration
- Complement activation
- Cytokine release
- Myocardial depressant factor.

Pulmonary Complications

- Pulmonary edema
- Pulmonary microembolization
- Complications due to one lung ventilation
- Atelectasis due to lung retraction
- Increased ARDS postoperatively due to remote reperfusion injury and capillary leak.

Spinal Cord Protection

- Increase anterior spinal artery pressure:
  - Aortofemoral shunting
  - Maintain proximal hypertension
  - *Gott shunt*: Between proximal thoracic ascending to descending thoracic aorta.
- Reduce CSF pressure:
  - Avoid cerebral vasodilators
  - *CSF drainage*: Role is controversial
  - Corticosteroids, barbiturates.
- Reduce central venous pressure:
  - Controlled phlebotomy
  - NTG, $MgSO_4$, calcium channel blockers
  - Atriofemoral bypass.
- Esoteric measures:
  - Cooling (temperature drift)
  - Oxygen free radical scavengers:
    - N-acetyl cysteine
    - Mannitol
    - Superoxide dismutase
    - Allopurinol.
  - Limit cross clamp time to less than 30 minutes
  - Application of sequential aortic cross clamp
  - Enhanced spiral cord monitoring
    - Somato Sensory Evoked Potentials
    - Motor Evoked Potentials.
  - Use EVAAR where possible
- Avoid postoperative hypotension.

Renal Protection

- Optimize oxygen demand/supply:
  - Reduce tubular reabsorption (loop diuretics)
  - Cooling (temperature drift)
  - Maintain tissue oxygenation (heart, lung, hemostasis)
  - Limit aortic cross clamp time to less than 30 minutes
  - Sequential cross clamping.
- Increase renal tubular flow:
  - Fluid loading (most effective)
  - Loop diuretics
  - Mannitol 0.5–1 gm/kg before reperfusion
  - Dopamine 1–3 µg/kg/min
  - Fenoldopam 0.1–0.3 µg/kg/min
  - Maintain cardiac output.
- Use endovascular techniques where possible (EVAAR)
- Avoid nephrotoxins:
  NSAIDs
  - ACE inhibitors
  - Angiotensin receptor blockers
  - Aminoglycosides.
- Other techniques:
  - Thoracic epidural for sympatholysis
  - Prostaglandins
  - Isovolemic hemodilution
  - Maintain euglycemia
- Renal vasodilators:
  - Prostaglandins
  - Atrial natriuretic peptide
  - Theophylline.

DEEP VEIN THROMBOSIS[1,2,3,5,10,17]

Introduction

- The presence of a thrombus within a deep vein and accompanying inflammatory response in the vessel wall is called venous thrombosis/thrombophlebitis
- Superficial venous system in lower extremity includes greater and lesser saphenous veins and tributaries

- The deep veins are those which accompany major arteries.

Etiology

Conditions associated with increased risk of DVT:

- Surgery:
  - Orthopedic, thoracic surgeries
  - Abdominal, genitourinary surgeries.
- Neoplasms:
  - Pancreas (very common)
  - *Genitourinary*: Ovary, testes
  - Lung carcinoma
  - Breast cancer
  - Stomach tumors.
- Trauma:
  - Fracture spine, hip
  - Fracture of femur, tibia
  - Pelvic fractures
  - Traumatic brain injury:
    - Upto 25% patients with isolated brain injury develop DVT
    - Prophylaxis started 45 hours after injury to prevent increased risk of bleeding.
- Venulitis:
  - *Burgers disease*
  - *Behcets disease*
  - Homocysteinuria.
- *Immobilization*: MI, CCF, stroke, postop convalescence
- Hypercoagulable states:
  - Pregnancy, OCP use
  - Myeloproliferative disorders, APLA syndrome, SLE
  - Multiple myeloma
  - DIC, factor V Leiden deficiency
  - Protein C and S deficiency, dysfibrinogenemia
  - Previous history of DVT.

Pathogenesis

- *Virchows triad* describes factors which predispose to VTE:
  - Stasis of blood
  - Vascular endothelial damage
  - Hypercoagulability.
- Initially, the thrombus contains principally platelets and fibrin
- RBCs then become interspersed with fibrin and thrombus propagates in the direction of blood flow
- Inflammatory response in blood vessel is due to:
  - Granulocyte infiltration
  - Loss of endothelium and edema

Sites of Occurrence

- *Lower extremity:* Femoral vein, popliteal vein, iliac vein
- Pelvic vein
- Superior/inferior vena cava
- Upper extremities:
  - Due to increased use of subclavian or IJV catheters
  - More common in ICU population
  - Can result in pulmonary embolism in up to 2/3rd of cases
  - Also increases risk of post-thrombotic syndrome.

Clinical Features

- 50 % of cases develop within 1 week (usually within 4 days)
- Calf pain is *most common* complaint
- Unilateral leg swelling, warmth, erythema
- Increased tissue turgor, prominent venous collaterals, distended superficial veins
- Tenderness along course of vein, cord may be palpable
- In markedly edematous limb, interstitial tissue pressure exceeds capillary perfusion pressure and causes pallor: *Phlegmasia alba dolens*
- In some patients deoxygenated Hb in stagnant vein imparts cyanotic hue to the limb: *Phlegmasia cerulea dolens*
- Bedside diagnosis may be difficult as only 1 of multiple veins may be involved allowing adequate venous return through remaining patent vessels
- *Homans sign:* Increased resistance or pain during foot dorsiflexion
- *Moses sign:* Pain on squeezing calf muscles.

Complications

- Short-term complications:
  - Prolonged hospitalization
  - Bleeding due to treatment
  - Pulmonary embolism
  - Local extension of DVT
  - Further embolization
  - Pulmonary embolism.
- Long-term complications:
  - Post thrombotic syndrome
  - Pulmonary hypertension
  - Recurrent DVT
  - Embolic stroke through patent foramen ovale.

Investigations

- Investigations to diagnose DVT:
  - D-dimer assay:
    - Sensitive but not specific
    - Increased D-dimer levels in patients with DVT.
  - Duplex venous ultrasonography:
    - Noninvasive test
    - Most often used
    - Low sensitivity in absence of symptoms
    - Thrombus detected by:
      - Direct visualization
      - By inference when vein does not collapse on compression
      - By flow abnormality.
  - MRI: Used for DVT of SVC/IVC/pelvic veins
  - Venography:
    - Confirmatory, *gold standard test*
    - Diagnosed by presence of filling defect
    - Contrast medium injected into superficial veins of foot
    - This is directed to deep venous system by application of tourniquet
- Investigation to rule out pulmonary embolism:
  - Spiral CT/HRCT
  - Pulmonary angiography: *Gold standard*
  - TEE:
    - Demonstrates RV performance, TR
    - May help to see thrombus in PA/right heart
  - *V-Q scan:* Defect in perfusion with normal ventilation.

Differential Diagnosis

- Muscle rupture
- Ruptured popliteal cyst
- Arterial occlusive disease
- Lymphedema
- Trauma/muscle hemorrhage.

Treatment

- Anticoagulation:
  - Prevents thrombus propagation and allows lytic system to act
  - Heparin:
    - UFH 7500–10000 IU IV bolus dose followed by infusion of 1000–1500 IU/hr
    - Rate titrated to keep APTT value twice that of control value
    - Subcutaneous enoxaparin 1 mg/kg BD used as alternative
  - Direct thrombin inhibitors:
    - Lepirudin 0.4 mg/kg followed by 0.15 mg/kg/hr
    - Argatroban 2 µg/kg/min
    - Bivalirudin 0.75 mg/kg followed by 1.75 mg/kg/hr
    - Used in patients with Heparin Induced Thrombocytopenia.
  - Warfarin:
    - Administered during first week of therapy with heparin
    - Started as early as 1st day of heparin therapy if APTT is therapeutic
    - Overlap warfarin with heparin therapy for at least 4–5 days as action of warfarin is delayed
    - Dosage of warfarin adjusted to maintain PT at INR of 2–3.
  - Duration of therapy:
    - Continued for 3–6 months in acute DVT to reduce chance of recurrence
    - Duration of therapy is indefinite for:
      - Recurrent DVT
      - Hypercoagulable states
      - Malignancy.
- IVC filter/IVC plication:
  - Transvenous filter placement done through femoral venous access
  - SVC and IVC filters available
  - Used when anticoagulants are contraindicated to protect from pulmonary embolism
  - Does nothing to already formed thrombus
  - Some filters can be left permanently
  - Removable filters are removed once anticoagulants are started.
- Thrombolytics:
  - Used in:
    - Massive pulmonary embolism
    - Cor pulmonale
    - Hemodynamically compromised patients.
  - Streptokinase/urokinase/t-PA used
  - Early administration of thrombolytics:
    - Accelerates clot lysis
    - Preserves venous valves
    - Reduces chances of developing post thrombotic syndrome.
- Management of pulmonary embolism:
  - Anticoagulation
  - Thrombolytic agents in patients with:
    - Severe hemodynamic compromise

- Cardiac arrest unresponsive to resuscitative measures despite increased risk of hemorrhage.

– Symptomatic patients:
  - Intubate and positive pressure ventilation
  - Fluids and inotropes (amrinone)
  - Continuous IBP, CVP monitor
  - Pulmonary embolectomy with partial CPB .

DVT PROPHYLAXIS[1,2,3,5,17]

Introduction

Venous thromboembolism is a major cause of death after surgery or trauma to lower limbs.

Incidence

- Venous thromboembolism develops in 40–80% of orthopedic patients without prophylaxis
- 1–28 % patients develop pulmonary embolism
- Fatal pulmonary embolism occurs in 0.1–8% patients
- VTE develops in 40–80% orthopedic patients
- Late initiation of prophylaxis (≥ 4 days after trauma) triples the risk of VTE.

Anderson and Spencer's Risk Factors for Development of DVT[3]

Strong Risk Factors

- Fracture of hip/leg/spine injury
- Hip/knee replacement
- Surgeries of hip/pelvis
- Major trauma.

Moderate Risk Factors

- CNS disorders, paralytic stroke
- CVP lines, malignancy
- Thrombophilia, polycythemia
- Malignancy
- CCF, nonvalvular atrial fibrillation with previous cerebral embolus
- Patients with prosthetic valves (Mitral > Aortic)
- Respiratory failure
- Irritable Bowel Disease/DM
- Pregnancy/postpartum
- Knee arthroscopy
- Chemotherapy, hormone replacement therapy, OCP, radiotherapy for pelvic neoplasm.

Weak Risk Factors

- Bed rest more than 4 days
- Immobility due to sitting (car/plane travel)
- Increased age, obesity
- Laparoscopic surgery with duration of surgery more than 30 min
- Pregnancy/antepartum
- Varicose veins
- Trauma, severe infections.

Other Risk Factors

Hypercoagulable states:
- Factor V Leiden deficiency (most common disorder associated with VTE)
- Antithrombin III, protein C and S deficiency
- APLA, lupus anticoagulant, malignancy, OCP therapy.

Preoperative Considerations

- Risk stratification for development of DVT done for patients
- Patients with previous history of DVT:
  – Risk of recurrence:
    - It is 50% within 3 months of previous DVT without anticoagulant therapy
    - If 1 month of warfarin therapy is added, risk reduces to 10%
    - If 3 months therapy is added, risk is reduced to 5%
  – Elective surgery:
    - To be postponed in 1st month after episode of venous/arterial thromboembolism
    - Ideally 3 months of anticoagulation is recommended before surgery
    - If elective surgery cannot be postponed preoperative heparin given while INR < 2
    - Heparin not to be restarted for 12 hours after major surgery
    - Check APTT 12 hrs after restarting therapy
- Patients on anticoagulant therapy:
  – Stop warfarin:
    - INR to be checked during preoperative visit
    - Withhold warfarin for 5 days
    - This allows PT/INR to normalize, if INR was chronically maintained between 2–3
    - Withhold more doses if INR is prolonged
  – Bridging therapy:
    - During the time without warfarin, patients are at increased risk for VTE

- Heparin can be used to bridge anticoagulation therapy in perioperative period
- Use of bridging therapy with heparin, however, is controversial
- LMWH can be used as it can be self administered without need for monitoring
- For patients receiving anticoagulation for 1–3 months after an acute episode of VTE, preoperative IV heparin is not justified
- For those receiving anticoagulation for > 3 months after acute episode of VTE, preoperative heparin is not indicated at all
- However postoperative heparin should be administered until warfarin is resumed and INR is > 2.

Intraoperative Thromboprophylaxis

Risk Stratification[3]

Category	Treatment
low Risk No risk factors Uncomplicated surgery Short duration surgery	Comfortable position Knees flexed at 5° Avoid constriction and external pressure Early mobilization Prefer RA Good postoperative analgesia Good physiotherapy
Moderate Risk Age > 40 years with no other risk factors Procedure lasting > 30 minutes OCP use	Proper positioning *Intermittent pneumatic compression devices* of calf and ankle (applied prior to sedation) and continued till patient is awake and moving Compression stockings Frequent alteration of OT table Early mobilization Aspirin therapy
High Risk Age > 40 years with other risk factor Procedure lasting > 30 minutes under GA OCP use	Treated as per patients with moderate risk Preoperative hematology consultation Consider periop antithrombotic therapy

Thromboprophylactic Regimens

ACCP 2004 guidelines:

- Hip and knee arthroplasty, hip surgeries:
  - LMWH:
    - Enoxaparin 30 mg S/C or IV BD or 40 mg OD
    - Started 12 hours before surgery or 12–24 hours after surgery or 4–6 hours after surgery at half usual dose and then increasing to usual dose the next day
  - *Fondaparinux:* 2.5 mg 6–8 hours before surgery (selective Factor X_a inhibitor)
  - Warfarin started preoperatively or the evening after surgery (INR between 2–3)
  - Intermittent pneumatic compression along with aspirin
  - Sole use of aspirin for prophylaxis not recommended
  - Early mobilization in those at high risk of bleeding.
- Spinal cord injury:
  - LMWH once primary hemostasis achieved
  - Intermittent pneumatic compression devices if anticoagulation is contraindicated
  - During rehabilitation phase, conversion to warfarin therapy via 'bridging'
  - INR maintained between 2 and 3
  - Clotting factor activity maintained at 20–40%.
- *Elective spine surgery:* Routine use of thromboprophylaxis apart from early mobilization not recommended in patients without other risk factors
- *Knee arthroscopy*: Routine use of thromboprophyaxis apart from early mobilization not recommended
- Duration of LMWH therapy:
  - At least 10 days for routine procedures
  - 28–35 days extended prophylaxis in high risk patients.
- *Role of neuraxial anesthesia*: Neuraxial anesthesia reduces incidence of VTE through the following mechanisms:
  - Blood flow changes:
    - Causes rheological changes
    - Results in hyperkinetic lower limb blood flow due to vasodilation
    - This reduces venous stasis.
  - Systemic effects of local anesthetics:
    - Anti-inflammatory property of LA
    - Circulatory effects of adrenaline used as adjuvant.
  - Altered coagulation responses:
    - Prevents increases in Factor VIII and vWF levels
    - Attenuated postop reduction in antithrombin III levels
    - Reduced platelet reactivity.
  - Avoids positive pressure ventilation and its effects on circulation
- Although neuraxial anesthesia reduces risk of VTE, risk of VTE remains significant and warrants pharmacological thromboprophylaxis.

- Continuous psoas compartment/femoral nerve catheters may be alternatives to neuraxial block in these in anticoagulants.

Thromboprophylaxis in ICU

- High index of suspicion if:
  - Unexplained tachycardia, tachypnea, fever
  - Asymmetric limb edema
  - Increased dead space ventilation.
- Anticoagulation:
  - LMWH used in high risk patients
  - UFH (low dose) used in moderate-low risk patients
  - Only TID regimens are effective for UFH
  - Fondaparinux use has not been studied in ICU.
- IVC filters:
  - No evidence supporting preventive placement of filters
  - Can be placed in patients for whom LMWH is contraindicated
  - Removable filters available.
- Other measures:
  - Flush catheter tips with heparin:
    - Heparin concentration should be 1 IU/ml
    - 3–4 ml of heparin added to 500 ml saline
  - Serial compression devices:
    - No evidence of benefit
    - Useful in patients in whom LMWH therapy is contraindicated.