

Anesthesia Review for DNB Students



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for DNB Students

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Anesthesia Review for DNB Students

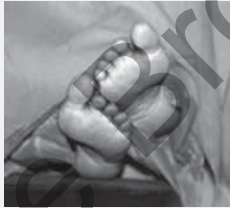
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Dedicated to

Appa, Amma, Sowmya, Jay and Sai



AT THY LOTUS FEET

Preface

It was during my postgraduate years that I realized that the subject of anesthesiology does not have a comprehensive and examination-oriented book. The pursuit of DNB as a postgraduation degree itself is an arduous task, owing to the difficult work schedule, and high expectations from the students at the time of examinations. This problem is compounded manifold for the subject of anesthesiology, as it is a discipline in which the students have to have a wide base of knowledge.

Most of the textbooks available today are not student-friendly as there is a lot of information in them, from which the examinee has to pick and retain in memory only those details which are necessary. As a result, there is a whole lot of unwanted information to which he is subjected, which may be confusing at the time of examinations. Secondly, there is no single textbook which gives all the details in an examination-oriented format. As a result, the student is forced to study from several different textbooks spanning multiple subjects. Also, owing to the importance of knowledge of various clinical guidelines, it becomes mandatory for the anesthesiology examinee to be familiar with the latest guidelines across a wide variety of disciplines. I have attempted to address these problems by compiling a book, which is student-friendly, well researched and is based on the most recent clinical practice guidelines.

This book has been written to cater to the DNB anesthesiology board examinations in a comprehensive and point-based system. Also, most of the references are from standard textbooks in order to prevent confusion arising due to numerous research papers published in the recent past.

Therefore, the information has been provided in a highly concise, crisp and readable manner to help you crack the anesthesiology boards.

Hope you enjoy reading it and wishing you all the best to crack your boards!!

Kaushik Jothinath

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Cardiac Anesthesia

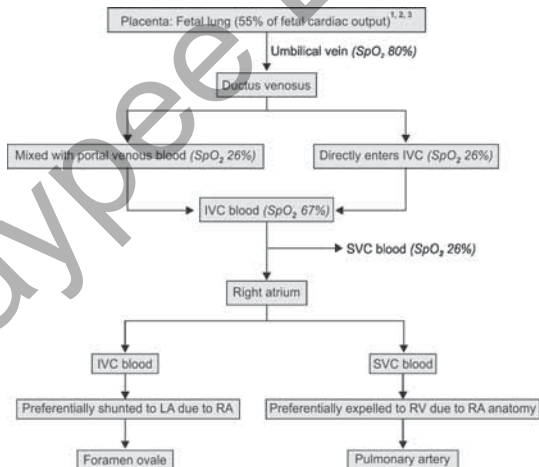
PHYSIOLOGY OF FETAL CIRCULATION^{1,2,3,7}

Introduction

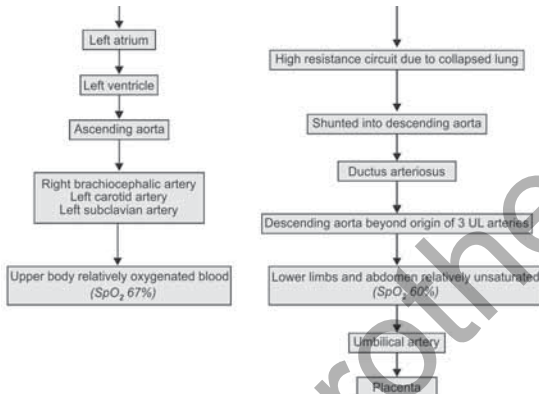
Fetal pulmonary and systemic circulation are essentially in parallel instead of in series as in adults due to two anatomical shunts, the ductus arteriosus and foramen ovale.

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.



Contd...



Changes Occurring at Birth

- The major cardiopulmonary adaptations at birth are:
 - Recovery from birth 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 ultrafiltrate is present in fetal lungs
 - At birth, chest wall is squeezed as it passes through vaginal vault
 - Thus, ultrafiltrate 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}

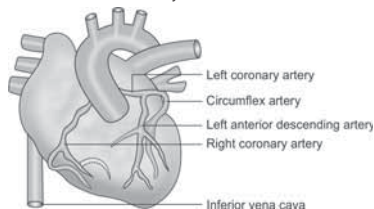
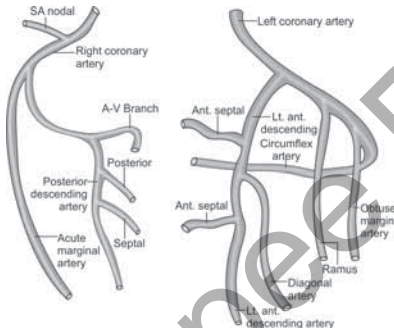
Introduction

- 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 circulation²Fig. 7.2: Coronary circulation schematic²

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%)

Contd...

Contd...

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
Left circumflex artery	Most of interventricular septum	Part of posterior fascicle of LBB
	Left atrium	Sinoatrial node (40%)
Left anterior descending artery	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 *Thebesian 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 Thebesian 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.

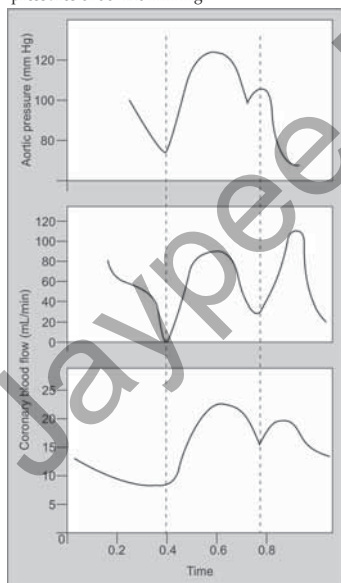


Fig. 7.3: Normal perfusion curves²

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 μ m
 - Critical mediator for autoregulation is oxygen, acting via adenosine.

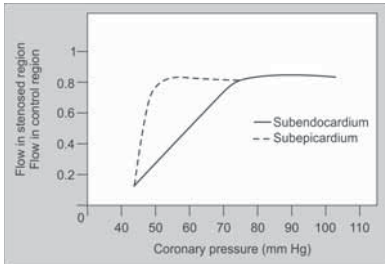


Fig. 7.4: Coronary autoregulation curve²

- 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

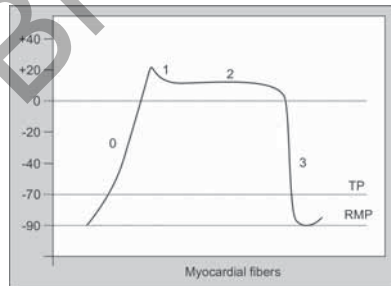
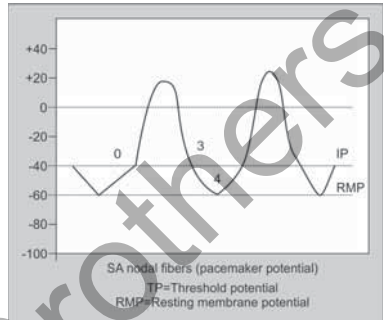
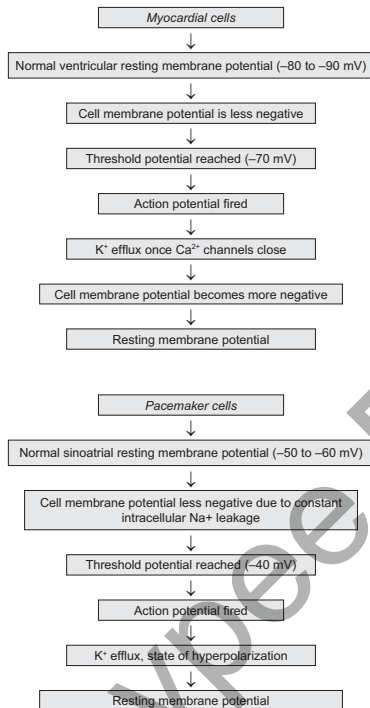


Fig. 7.5: Cardiac action potential²

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



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.

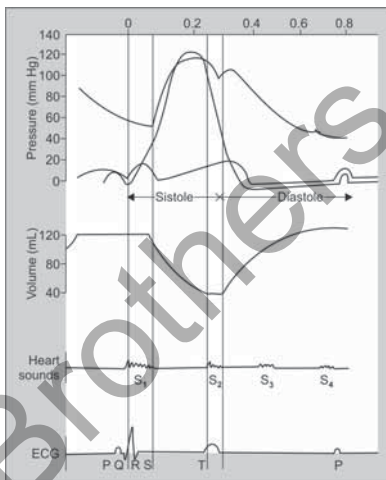


Fig. 7.6: Cardiac cycle²

- 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: S₁ 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: S₁ 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:
 - S₂ produced when semilunar valves close
 - Usually split S₂ 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: S₂ occurs when semilunar valves close.

Rapid Filling Phase

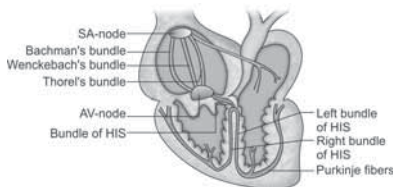
- Electrical event: No events
- Mechanical event: Atrioventricular valves open
- Pressure volume event:
 - Blood from atria enters ventricles
 - Ventricular volume increases rapidly
- Heart sound event:
 - S₃ produced due to rapid passive filling of blood
 - Causes of S₃ gallop:
 - Myocardial infarction
 - 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.

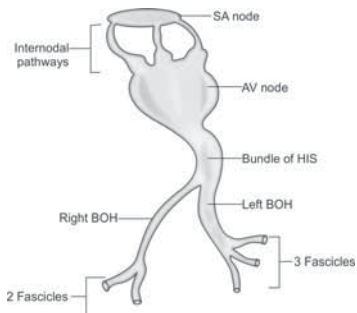
Second Rapid Filling Phase

- 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: S₁ produced.

CONDUCTION SYSTEM OF HEART^{1,2,3,5}

Contd...

Contd...

Fig. 7.7: Conduction system of heart²

Sinoatrial Node

- Location:
 - RA wall at SVC-RA junction
 - Located 1 mm below epicardial surface
 - *Crista terminalis*
 - 50% cells have pacemaker 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:
 - *Bachmann's* anterior bundle
 - *Wenckebach's* middle bundle
 - *Thorel's* 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.

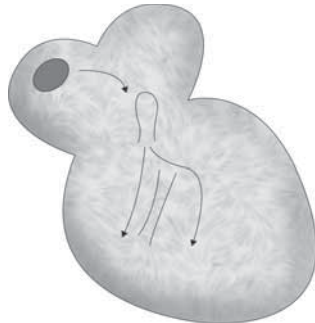


Fig. 7.10: Mahaim fibers

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 dysrhythmias:
 - *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/m² (2.5–4.2 L/min/m²)
- Body surface area calculated from nomogram based on weight and height.

Formula

- Cardiac output = stroke volume \times 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

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

- 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 (arterial 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

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

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

- Normal SVR = 900–1500 dynes seconds/cm⁵
- Normal PVR = 150–250 dynes seconds/cm⁵
- 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

- Myocardium's 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_i)
- 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 = $(220 - \text{age in years})$
 - Normal intrinsic heart rate = $118 \text{ bpm} - (0.57 \times \text{age})$
- *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.

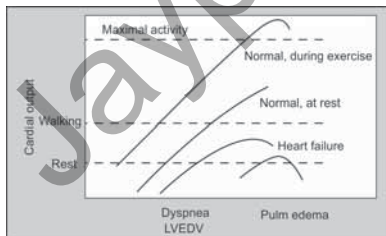


Fig. 7.11: Frank–Starling's law³

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.

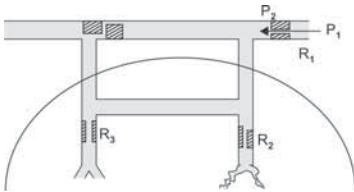


Fig. 7.12: Collateral coronary steal phenomenon

Transmural Coronary Steal

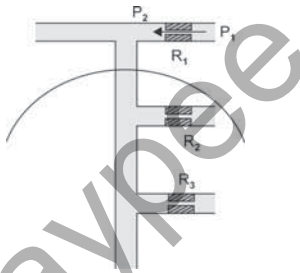


Fig. 7.13: Transmural coronary steal phenomenon

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

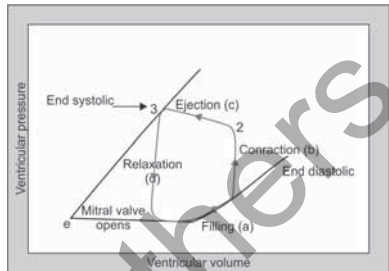


Fig. 7.14: Normal pressure-volume loop^{2,3,5}

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 <i>peak systolic pressure</i>
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.

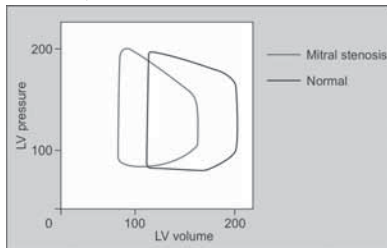


Fig. 7.15: Pressure volume loop in mitral stenosis⁵

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.

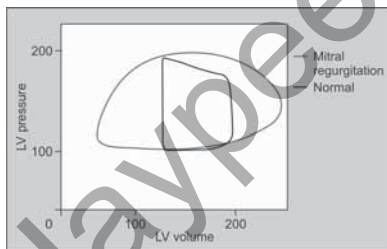


Fig. 7.16: Pressure volume loop in mitral regurgitation⁵

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.

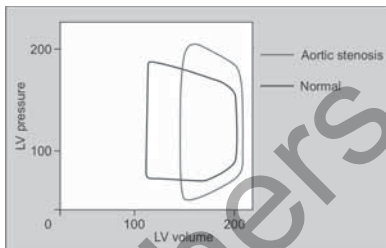


Fig. 7.17: Pressure volume loop in aortic stenosis⁵

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.

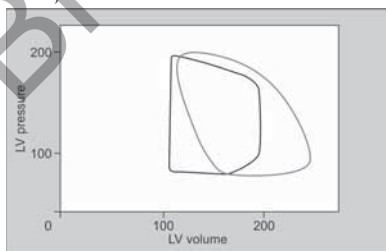


Fig. 7.18: Pressure volume loop in aortic regurgitation⁵

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.