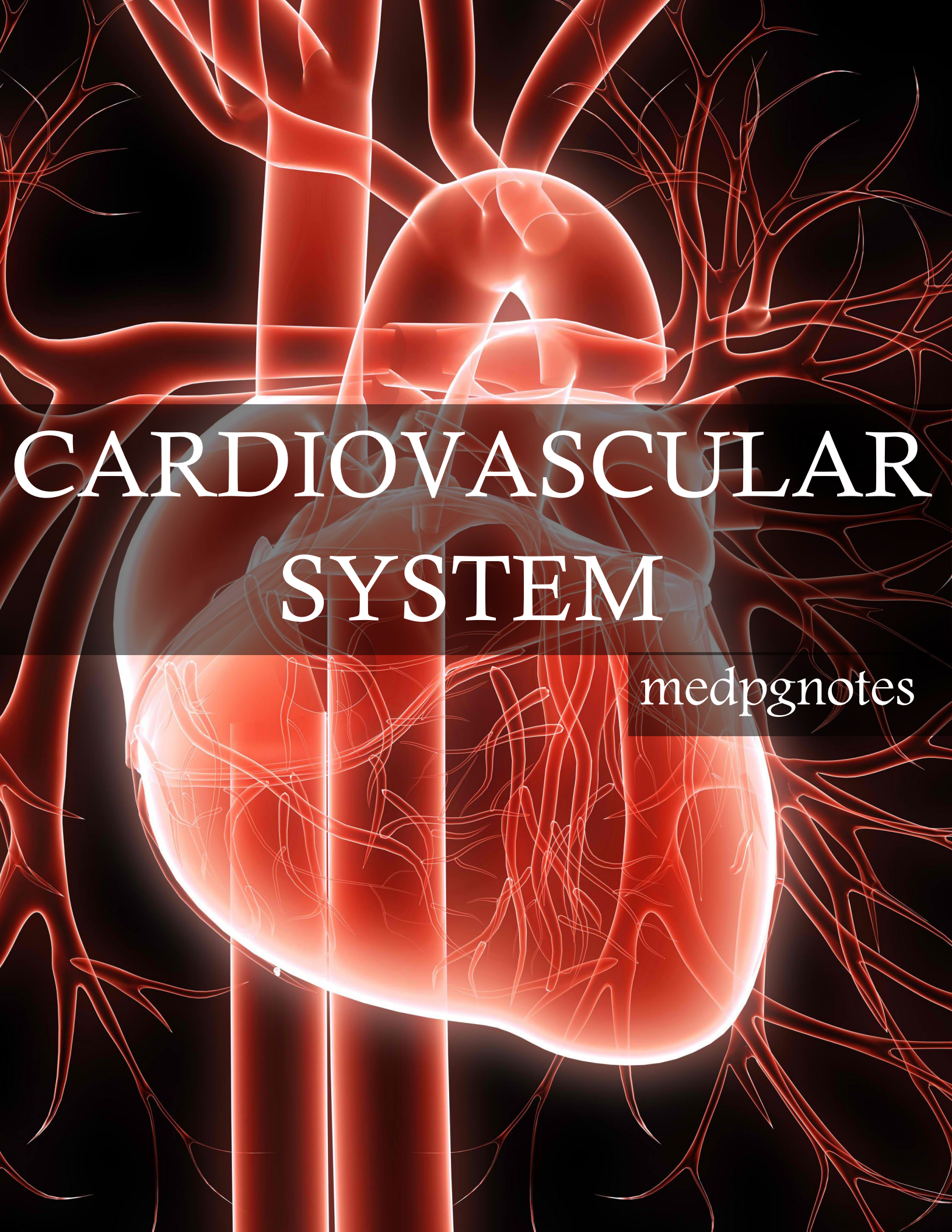

CARDIOVASCULAR SYSTEM

medpgnotes

CONTENTS

CARDIOVASCULAR SYSTEM

CARDIOVASCULAR SYSTEM

KEY TO THIS DOCUMENT

Text in normal font – Must read point. Asked in any previous medical entrance examinations

Text in bold font – Point from Harrison's text book of internal medicine 18th edition

Text in italic font – Can be read if you are thorough with above two.

DEVELOPMENT OF CARDIOVASCULAR SYSTEM

First organ formed during embryogenesis	**Heart**
Development of heart	2nd to 8th week
Development of septal defects in fetal heart	5 – 8 weeks
Ectopia cordis associated with	Heart
Cardiac jelly is secreted by	Myocardium
Cardiac jelly contributes to the formation of	Endocardium
Development of heart	Dorsal mesocardium forms transverse pericardial sinus, Myosites arise from splanchnopleuric mesoderm, Purkinje fibres develop from splanchnopleuric mesoderm, Neural crest cells have role in development of muscular and subpulmonary infunbibulum, Endocaridal cushion develops from cardiac jelly
Development of interatrial septum	*Perforation in septum primum forms foramen secundum, foramen secundum maintains right to left shunt, septum secundum is situated to the right of septum primum, septum primum closes at day 42 of development*
Left horn of sinus venosus forms	*Coronary sinus*
Interventricular septum	*Muscular interventricular septum, conus septum, AV endocardial cushion*
Membranous part of interventricular septum	*Anterior part between RV and LV, posterior part between RA and RV*
Membranous part of atrioventricular part of interventricular septum is between	RA and LV
Premature closure of foramen ovale results in	Right ventricular hypertrophy
Patent foramen ovale is due to failure of fusion of	Septum primum and septum secondum
Aneurysm of sinus of valsalva usually arise from	Right aortic sinus
Muscular component of dorsal aorta develops from	Paraxial mesoderm
Axial artery of Upper Limb is derived from	Seventh Intersegmental artery
Most important structure involved in development of inferior vena cava	Supracardinal vein and subcardinal vein
Vitelline vein forms	Hepatic vein, Inferior mesenteric vein, inferior vena cava
Does NOT derive from vitelline vein	Superior vena cava
Left sided superior vena cava drains into	Coronary sinus
Kommerell's diverticulum	**Anatomical remnant of right aortic arch**
Smooth portion of right atrium is derived from	*Sinus venosus*

ANATOMY OF CARDIOVASCULAR SYSTEM

Thinnest portion of Myocardial wall	Right atrium and Left atrium
Sinoatrial node is situated at	Junction of SVC and Right atrium
AV node lies at	Interatrial septum

Fibres of AV junction	Modified Nerve fibres
Ventricular muscle receives direct innervation from	Purkinje fibres
Triangle of Koch	Coronary sinus opening, Tendon of todaro, Septal leaflet of tricuspid valve
Boundary of Koch triangle NOT formed by	Limbus fossa ovalis
NOT a boundary of Koch's triangle	Origin of Left Coronary artery
Feature of right atrium	Coronary sinus lies between fossa ovalis and IVC
Structures associated with internal surface of right atrium	*Sinus of keith, triangle of Koch, tendon of todaro, Waterson groove*
NOT a part of interior of Right atrium	*Trabeculae carnea*
Coronary sulcus	*Between right atrium and aorta. Right coronary artery passes through it.*
Anatomy of right ventricle	Most prominent trabeculation, Crista supraventricularis separate tricuspid valve and pulmonary valve, Apex trabeculated, TV and PV share fibrous continuity
Right ventricle	*Conus, outflow tract*
Annulus of Vieussen around	*Right ventricular outflow tract*
Base of heart is related to	Descending aorta
Area of mitral orifice in adults	4-6 cm^2
Central fibrous body of heart formed by	*Right fibrous trigone with membranous part of ventricular septum*
Commonest variation in arteries arising from arch of aorta	Left common carotid artery arising from brachiocephalic trunk
Sympathetic supply to heart	T1 to T5
In angina pectoris, pain radiating down the left arm is mediated by increased activity in afferent (sensory) fibres contained in	Thoracic splanchnic nerve
Buffer nerve	*Carotid sinus nerve and vagal fibres from aortic arch*
Superficial cardiac plexus	*Below aortic arch*
Left coronary plexus	*Deep cardiac plexus*
Right coronary plexus	*Both superficial and deep cardiac plexus*

PHYSIOLOGY OF CARDIOVASCULAR SYSTEM

GENERAL FEATURES OF PHYSIOLOGY OF CARDIOVASCULAR SYSTEM

Duration of Cardiac cycle in man	0.8 seconds
During cardiac cycle	*Mitral and aortic valve never open at same time*
Left ventricular systole corresponds to	Auricular diastole
Minimum motion of heart during cardiac imaging	Mid diastole
LEAST correct statement	During exercise systole is shortened more than diastole
Heart stops in Diastole in	Hyperkalemia
AV valves open at the beginning of	Diastole
Isometric relaxation of cardiac cycle ends with	Opening of AV valve
At the end of isometric relaxation phase	AV valves open

CARDIOVASCULAR SYSTEM

Closure of aortic valve corresponds to beginning of	Isovolumetric relaxation
Isovolumetric dilatation of ventricles is at	Closing of Semilunar valves
Aortic valve closes at the end of	*Protodiastole*
During cardiac cycle, opening of aortic valve takes place at	End of isovolumetric contraction
Maximum pressure rise in ventricles during	*Isovolumetric contraction*
Phase of cardiac cycle follows immediately after beginning of QRS wave	*Isovolumetric contraction*
Isovolumetric relaxation precedes	Ventricular ejection
Initiation of opening of aortic valve occurs when	Ventricular pressure is more than aortic pressure
Correlate with isovolumetric contraction	Both valves are open
Preload is associated with	*Isotonic contraction with shortening of muscle fiber*
Volume determining preload	End diastolic volume of ventricles
End diastolic ventricular volume	*130 ml*
End diastolic volume increases in	Decreasing venous compliance
Intrinsic heart rate is determined by	*IV administration of atropine, atenolol*
Cardiac output in L/min divided by heart rate equals	Mean stroke volume
Cardiac output in an adult is nearly	5 litres
Percentage of Cardiac output concentrated by Renal blood flow	25%
Maximum cardiac output during pregnancy	32 weeks
Cardiac output is decreased in	Rapid arrhythmia
Low cardiac output is associated with	Arrhythmia
Cardiac output decreases during	Standing from lying position
Cardiac output can be determined by	Fick's principle, ECHO, Thermodilution
Direct fick method for estimation of cardiac output requires	O2 content of arterial blood, O2 consumption per unit time, O2 content of blood from right ventricle
Fick's law	Passive osmosis along concentration gradient only
Scientific principle for the basis for thermodilution method used in measurement of cardiac output by pulmonary catheter	Stewart Hamilton priniciple
Most recent advance in non invasive cardiac output monitoring	Electrical impedance cardiography technology
Cardiac index	cardiac output/body surface area
Cardiac index in normal person	3.2 (2.6 – 4.2)
Cardiac index of normal person	3.2 l/min/m^2
Cardiac output in L/min divided by heart rate equals	Mean stroke volume
Preload to heart depends upon	Stroke Volume
Stroke volume is decreased by	*Increasing heart rate*
Stroke volume is decreased in	Arrhythmia
Gorlin formula	**Area of flow across valve**
Hakki formula	**Aortic valve area**
Severity of shunt	**Pulmonary blood flow/systemic blood flow (Qp/Qs)**
Venous return	(mean systemic filling pressure – right atrial pressure)/resistance to venous return
Venous return to heart from lower limb is NOT affected by	Arterial pressure
Venous return to heart during quiet standing facilitated	Calf muscle contraction during standing, valves in

by	perforators, sleeves of deep fascia
When a person changes from standing to lying down position	Venous return to heart rises immediately
Supine to upright position	Decrease in central venous pressure, Rise in heart rate, Decrease in cardiac output, Decrease in stroke volume
Shift from supine to upright NOT occur	Rise in central venous pressure
Coronary blood flow	Directly related to perfusion pressure and inversely related to resistance
Amount of coronary blood flow	250 ml/min
Coronary blood flow in left coronary artery is maximum during	Ventricular diastole
Coronary blood flow stops during	Isometric contraction
Blood pressure during exercise is increased in	Coronary circulation
Reflex noradrenergic discharge during fall in blood pressure causes vasodilatation in	Coronary circulation
Most important metabolic factor affecting coronary blood flow	Hypoxia
Cardiac oxygen demand	Has a constant relation to external work done by heart
Myocardial oxygen demand	Correlates with heart rate, Constant relation to external cardiac work, Depends of preload, afterload, intramyocardial tension, myocardial muscle mass
Myocardial oxygen consumption	Directly proportional to mean arterial pressure
Myocardial oxygen demand correlate with	Heart rate
Oxygen utilization by ventricular muscles at rest	10 ml/100 gm/ min
Negative G	Cardiac output increases, Cerebral artery pressure increases, Blood centrifuged towards head, Red out
Aviator subjected to negative G	Cerebral arterial pressure rises
Baroreceptor	Nucleus tractus solitarius
Baroreceptors are mostly sensitive to	Systolic blood pressure
Discharge from baroreceptors causes inhibition of	Rostral ventrolateral medulla
Baroreceptor stimulation produce	Decreased heart rate and BP, Decreased cardiac contractility
Ligature tied proximal to baroreceptors (below carotid sinus)	Hypertension and tachycardia
Means of studying acute physiological response to arterial baroreceptor unloading	*Sinoaortic denervation (lowers body negative pressure)*
Vasomotor centre of medulla	Acts along with cardiovagal centre to maintain blood pressure
Inhibition of Vasomotor centre causes	Decrease in BP
Pressure on carotid sinus cause	Reflex bradycardia
On cutting or severing sinus nerve in dog, cause	Increase in mean blood pressure
Clamping of carotid arteries below (proximal) carotid sinus likely to produce	Increase in blood pressure and increase in heart rate (stimulates vasomotor center – sympathetic response)
Effect of bilateral Carotid compression above Carotid Sinus	Stimulates vagal parasympathetic center
Single most important factor in control of autonomic contractility of heart	Sympathetic stimulation
Features of sympathetic stimulation of heart	Increased contractility, increased heart rate, increased conduction velocity
Sympathetic stimulation	Increased HR, BP, total peripheral resistance, Decreased

	venous capacitance
Sympathetic stimulation does NOT cause	Increase in venous capacitance
cAMP specific phosphodiesterase inhibitor on contractility of isolated strips of rabbit heart	*Increases contractility*
Human heart	Heart rate increases with parasympathetic denervation
Features suggesting denervation	Unregulated firing of individual muscle fibres, presence of positive sharp waves, spontaneous firing of motor units
Denervated heart has	More heart rate
In a patient with transplanted heart, reason for increased cardiac output during exercise	Epinephrine from medulla
Plateau phase of myocardial action potential is due to	Influx of Ca++
Factors increasing length of ventricular cardiac muscle fibres	Increased venous tone, Increased total blood volume, Increased negative intrathoracic pressure
Left ventricle performs more than right due to difference in	Arterial pressure
Ejection fraction (left ventricle)	**Stroke volume/end diastolic volume**
Normal left ventricular ejection fraction	65%
Pressure in right atrium	**Less than 6 mm Hg**
Right Ventricular Systolic Pressure	25 mm Hg
Right ventricular diastolic pressure	**0 – 12 mm Hg**
A 0.5 litre blood loss in 30 minutes will lead to	Slight increase in heart rate and normal BP
A patient with increased BP and decreased heart rate is likely to have	Increased ICT, Brain tumor, Head trauma
Bradycardia can occur in	Myxoedema, during convalescence, complete heart block
Drug linked with increased cardiac mortality	Rofecoxib
When blood flow stops the pressure is given by	Mean Circulatory filling pressure
Main site of Peripheral vascular resistance	Precapillary arterioles
Splanchnic circulation	25-30% of total circulation
Shape of arterial pulse is influenced by	Arterial wall expansion

OXYGEN CONSUMPTION

Whole body	*250 ml/min*
Liver	*51 ml/min*
Skeletal muscle	*50 ml/min*
Brain	*49 ml/min*
Rest of muscle	*44 ml/min*
Heart muscle	*29 ml/min*
Kidney	*18 ml/min*
Skin	*12 ml/min*

MEAN ARTERIAL PRESSURE

Mean arterial pressure	Diastolic + one third of pulse pressure

Peripheral resistance	Mean arterial pressure as it remains constant
Mean arterial pressure	(SBP + 2DBP)/3

PULSE PRESSURE

Pulse pressure lowest in arterial system in	Capillaries
Pulse pressure	Systolic pressure - diastolic pressure
Pulse pressure is increased by	*Decrease in arterial compliance*
Varying pulse pressure with Normal rhythm	Left ventricular failure
Wide pulse pressure NOT seen in	Congestive heart failure

VASCULAR SYSTEM

Circulation	Pulsatile flow affects gene transcription, Increased viscosity increases mean blood pressure, Hematocrit does NOT markedly change peripheral resistance, Pulsatile flow is recommended
Marey's law is	Relationship with heart rate and blood pressure
Law relating distending pressure and tension in a vessel wall	Law of Laplace
Law related to wall tension	*Laplace law*
Laplace law for a cylinder	$P = T/r$
Laplace law has NO role in	Liver
50% reduction in arterial diameter causes	*Reduction in flow 16 times*
Velocity of blood in aorta	22 cm/sec
Velocity of blood is inversely proportional to	Cross sectional area
Reservoirs of blood in cardiovascular system	*Venules*
Velocity of blood is maximum in	Large veins
Flow is laminar in small vessels because	Effective velocity in small vessels is less
Which increase turbulence in blood flow	Increase diameter of blood vessel
Blood flow	Liver > kidney > brain > heart
Local control of blood flow NOT seen in	Skin
Which is NOT increased during exercise	Peripheral Vascular resistance
Storage pool of blood	Vein
Blood supply in splanchnic vessels decrease due to	Venoconstriction with decreased blood flow
In a younger subject whose aorta has high elastin content	Diastolic pressure is maintained, pulse pressure is narrow
Maximum difference of BP occurs between	Femoral artery and femoral vein
Classical finding in AV fistula	Sinus tachycardia
Capillaries	Greatest cross sectional area, Contains 5% blood, Contains less blood than veins, Have single layer of cells bounding the lumen, Site of gaseous exchange, Lined by endothelium
Capillaries	Larger quantity of blood than veins, site of gaseous exchange, lined by endothelium
Capillaries	Greatest cross sectional area, less blood than veins,

	single layer of cells
Changes in blood passing through systemic capillaries	Hematocrit increase, pH decrease, increase in protein content, shift of O2 dissociation curve to right
Pre capillary sphincter relaxation mediated by	Local hormones
Common structural feature seen in all capillaries is	Continuous basement membrane
Pericytes	**Wrap around capillaries**
At a constant blood flow, an increase in number of perfused capillaries improves the exchange between blood and tissue because of	*Greater surface for diffusion of molecules*
Distribution of blood flow is mainly regulated by	Arterioles
Maximum peripheral vascular resistance	Arterioles
Regulation of blood flow is maintained by	Arterioles
Cutaneous shunt vessels	Role in thermoregulation
Quantitatively most important means of increasing flow to an actively metabolizing tissue	*Decreasing local vascular resistance*

SYNCOPE

NOT a situational syncope	Deglutition syncope, Cough syncope, Micturition syncope
Mess trick and fainting lark is associated with	**Syncope**
Head up tilting for diagnosing	**Syncope**
Least useful investigation in neurocardiogenic syncope	Carotid duplex scan
Drug of choice in carotid sinus syncope	Ephedrine
Biofeedback therapy is not applicable for	*Vasovagal syncope*

HEART SOUNDS

FIRST HEART SOUND

First heart sound occur during the period of	Isovolumetric contraction
First heart sound	Lower frequency than S2, Caused by closure of mitral valve, Better heard with diaphragm of stethoscope
Soft S1	MR, VSD, calcified valve, long standing severe MS, pleural effusion, obesity
Loud S1	Short PR interval, Tachycardia
Loud S1 in mitral stenosis is caused by	Prolonged flow through mitral valve
Reversed splitting of S1	LBBB, atrial myxoma

SECOND HEART SOUND

Incisura of arterial pulse corresponds to	Second heart sound
Aortic component of Second heart sound is best heard	Ludwig's angle to right

at	
Heart sounds occurring shortly after S2	Opening snap, pericardial knock, tumour plop
Heart sound NOT occurring shortly after S2	Ejection click
Tumor plop	Low pitched
Single S2	TOF, pulmonary atresia, severe pulmonary stenosis
Loud pulmonary component of S2	Pulmonary hypertension, Eisenmenger syndrome
Pulmonary component of second heart sound is soft and inaudible on auscultation	Fallot's tetrology
Hang out time is related to	*Splitting of second heart sound*
Wide split S2	ASD, MR, pulmonary stenosis
Fixed(wide) splitting of S2	ASD, Pulmonary stenosis, pulmonary embolism
Wide fixed split S2 with ejection systolic murmur in left second intercostals space, left axis deviation	Ostium primum atrial septal defect
Reversed splitting of S2	Aortic stenosis, Left bundle branch block, systemic hypertension, PDA, post stenotic dilatation in AS
Severe Paradoxical S2 split	AS

THIRD HEART SOUND

Loud S3	Severe MR
Third heart sound	Constrictive pericarditis, ASD, VSD, athletes, LVF
Third heart sound is due to	Ventricular filling NOT at the time of atrial systole
S3 heard over	**Left ventricle**
S3 not heard in	Severe MS

FOURTH HEART SOUND

Fourth heart sound is due to	Ventricular filling
S4	IHD, long standing hypertension, hypertrophic cardiomyopathy, abnormal forceful left ventricular dilatation, Aortic stenosis, Hypertension
S4	Aortic stenosis, Hypertension, HOCM
S4	Thyrotoxicosis, acute MI
Fourth heart sound	Heard during ventricular filling phase
S4 is NOT heard in	Ventricular aneurysm

SNAP, CLICK AND THRILL

Opening Snap	High pitched, Early diastolic
Opening snap in mitral area corresponds to	Dicrotic notch of carotid pulse
Dicrotic notch in arterial pulse is due to	Closure of aortic valve
NOT a diastolic sound	Ejection click
Systolic thrill in second and third intercostal space	Subpulmonic VSD, pulmonic stenosis, Ebstein anomaly
Double apical impulse	Aortic stenosis, HOCM

MURMUR

Still murmur	**In normal children and adolescents**
Right sided murmur	**Usually increase with inspiration except pulmonary ejection sound**
Valsalva maneuver phase I	**Forced expiration against closed glottis, increased BP and decreased heart rate**
Phase of valsalva maneuver in which heart rate decrease	*Phase I*
Valsalva maneuver	**Decreases length and intensity except HOCM, MVP**
Standing	**Diminishes murmur except HOCM, MVP**
Hand grip increases	**MS, PS, MR, AR, VSD**
Continuous murmur	AV communication, Aortic sinus of Valsalva rupture, Coarctation of aorta
Continuous murmur	PDA, shunt between pulmonary and subclavian artery
Continuous murmur NOT seen in	VSD with aortic regurgitation
Continuous murmur NOT seen in	Mitral stenosis with mitral regurgitation, peripheral pulmonary stenosis, VSD with AR
An early systolic murmur may be caused by	Small ventricular septal defect, papillary muscle dysfunction, Tricuspid regurgitation
MC cause of midsystolic murmur in adult	**Aortic stenosis**
Pansystolic murmur	MR, VSD, TR
To and fro murmur	*VSD with AR, AR with MR, Repaired TOF*
Murmur heard in utero	*Gerbode effect (LV to RA shunt)*
New systolic murmur after MI	Rupture of interventricular septum, papillary muscle dysfunction, ischemic cardiomyopathy
Intensity of systolic murmur increased in	Severe AS
Carey comb murmur	Delayed diastolic murmur, rheumatic fever, low pitched murmur
Cole cecil murmur	*AR murmur in mid axillary area*
Gibson murmur	*Machinery murmur in PDA*
Graham Steel murmur	**Pulmonary regurgitation, pulmonary arterial hypertension**
Seagull murmur	**Ruptured chorda tendinae**
Means Lerman scratch	**Uncommon cardiac murmur in hyperthyroidism, may mimic pericardial rub**
Best position for examining cardiac murmurs in child	Recumbent
Abnormal change in pregnancy	Diastolic murmur
Investigation for diastolic murmur	**Echocardiography**

ARTERIAL PULSE AND JUGULAR VENOUS PULSE

GENERAL FEATURES OF PULSE

Asymmetric pulse	Dissection of aorta, Aortoaortitis, Aneurysm of descending aorta
Pulsus parvus et tardus	**Weak and delayed pulse, seen in aortic stenosis**

PULSUS BISFERIENS

Pulsus bisferiens	Two systolic peaks
Dicrotic pulse	*Two peaks one in systole and other in diastole*
Pulsus bisferiens	AR, AR+AS, Hypertrophic cardiomyopathy
Pericardial tamponade is NOT associated with	Pulsus bisferiens
Pulsus bisferiens best felt in	Radial artery

PULSUS PARADOXUS

Pulsus paradoxus is defined as	*Marked and exaggerated inspiratory fall in systolic BP in which the Korotkoff's sound disappears during inspiration*
Pulsus paradoxus	Cardiac tamponade
Pulsus paradoxus is seen in	Constrictive pericarditis, Cardiac tamponade, Massive pulmonary embolism, COPD, severe asthma, emphysema, hypovolemic shock
Pulsus paradoxus is NOT seen in	Hypertension, MI
Pulsus paradoxus is NOT seen in	Aortic regurgitation

PULSUS ALTERANS

Pulsus alterans	Left ventricular failure
Pulsus alterans	Ischemic heart disease

JUGULAR VENOUS PULSE

JVP	a-x descent atrial relaxation, v-y emptying of blood from right atrium into right ventricle, y-a ascent filling of right atrium from vena cava
JVP finding in cardiac tamponade	Prominent x descent, Absent y descent
Typical JVP finding in cardiac tamponade	Absent y descent
Paradoxical Inspiratory rise in JVP	Constrictive Pericarditis, Kussmaul sign
Square root sign in JVP	Constrictive pericarditis
JVP is NOT raised in	Hypovolemic shock
Should NOT rise in pregnancy	JVP
Cannon wave	Complete heart block
Giant 'a' wave is seen in	Tricuspid stenosis, right heart failure, pulmonary hypertension
A wave is exaggerated in	Tricuspid stenosis, Complete heart block, junctional rhythm, pulmonary hypertension
A waves in JVP are absent in	Atrial fibrillation
C wave in JVP	Ventricular contraction
C wave in JVP is due to	Bulging of tricuspid valve into right atrium

c-wave in JVP due to	Isometric Contraction (Bulging of tricuspid)
C wave in JVP	Bulging of tricuspid into right atrium
v-wave in JVP	Filling of Right Atrium due to venous return
V wave in JVP is due to	Atrial filling while TV closed

ELECTROCARDIOGRAM

GENERAL FEATURES OF ECG

Sum of voltage of 3 leads in ECG 5 millivolts	Increased cardiac muscle mass
Depolarization of atria in ECG is seen in	P wave
Depolarization in ECG by	P and QRS complex
P wave size in right atrial enlargement	2.5 mm
Ventricular Depolarization starts from	Left part of Interventricular septum
Time required for ventricular depolarization	0.08 to 0.1 sec
QRS complex indicates	Ventricular depolarization
QRS duration 100 - 120 ms	Normal, Left anterior fascicular block, left posterior fascicular block, Incomplete RBB
Wide QRS complex > 120 ms	Hyperkalemia, WPW syndrome, Ventricular tachycardia
Wide QRS complex typically seen in	Bundle branch block
Massive QRS is associated with	*Pompe's disease*
Athletic syndrome characterized by	Increased amplitude of QRS complex
Low QRS voltage	Pericardial effusion
Low QRS voltage with left ventricular hypertrophy	Cardiac amyloidosis
Component of ECG varying with heart rate	*QT interval*
Ventricular contraction	Beginning of Q wave to end of T wave, Beginning of R wave to end of T wave, if Q wave is absent
SNHL, Syncope, Prolonged QT interval	Jervell Lange Neilson Syndrome
Vagal stimulation of heart causes	Increased RR interval in ECG
Repolarization of ventricles is indicated by	*T wave*
QRS and ST junction	**J point**
Plateau phase corresponds with	ST segment
Brugada syndrome	**Normal structure of heart with ST elevation in V1, V2 and V3**
Brugada syndrome is due to mutation of	**Cardiac sodium channel SCN5A**
Treatment of Brugada syndrome	**Quinidine, Isoprotenol**
J wave (Osborn wave)	**Hypothermia**
Normal axis of ECG in adult male	-30 to +110 *
Left axis deviation is seen as	Positive in lead I and negative in lead II
Instantaneous mean vector	Equal and same as mean QRS vector, It is drawn through centre of vector in a direction from base towards apex, Summated vector of generated potential at particular instant cause by inflowing septal depolarization, When a vector is exactly horizontal and directed toward the person's left side, vector is said to extend in direction of 0*

Electromechanical systole	Between Q and S2
If carotid transducer is NOT functioning we can NOT obtain	LVET and PEP
PEP/LVET ratio is increased in	*Left ventricular systolic failure, aortic stenosis, left bundle branch block*
NOT measured without carotid transducer	LVET, PEP

ECG FEATURES OF HYPERKALEMIA

Hyperkalemia	Peaked T waves, loss of P waves, sine waves Prolonged PR interval, prolonged QRS interval, ventricular asystole
ECG changes in hyperkalemia	Wide QRS, Tall T waves, Prolonged PR interval, Sine wave pattern, disappearance of P waves
Tall T waves	Hyperkalemia
NOT an ECG feature of hyperkalemia	Prolonged QT interval
NOT true about hyperkalemia	U wave
Calcium is indicated in	Hyperkalemia
NOT a treatment of hyperkalemia without ECG changes	Calcium gluconate

ECG FEATURES OF HYPOKALEMIA

Hypokalemia ECG	U wave, ST depression, flattened or inverted T wave, prolonged PR interval
ECG with ST segment of prolongation and late T wave	Hypokalemia
ECG finding in hypokalemia	Increased PR interval with ST depression

ECG FEATURES OF HYPERCALCEMIA

Narrowed QT interval, polyuria, polydipia, nausea, altered sensorium for last 2 days. squamous cell carcinoma	Hypercalcemia
QT interval shortened in	Hypercalcemia

ECG FEATURES OF HYPOCALCEMIA

ECG feature of Hypocalcemia	Prolonged QT interval

ANGINA

FEATURES OF ANGINA

Critical narrowing of coronary vessel to cause angina	*More than 70%*
Most important factor in causation of cardiac arrest	Anoxia
Tietze syndrome usually	Second costal cartilage
Angina pectoris carried by	Middle and inferior cervical cardiac nerve
Potassium channel opener with anti anginal activity	Nicorandil
Potassium channel opener	*Penicidin*
Hypertensive developed angina	Propanolol
Ranolazine	Piperazine derived antianginal agent, may be used as first line agent in chronic angina, may improve glycemic control
Drugs that can worsen angina	Dipyridamole, Thyroxine, Sumatriptan
Does NOT worsen angina	Oxephedrine
Stable angina is associated with	**Physical exertion**
Cardiac markers in stable angina	Unchanged
Unstable angina are true	Recent angina, ST elevation or depression, rest pain
Drug used in Unstable Angina	Eptifibatide
Drug for Variant angina/Unstable angina/Prinzmetal angina	Diltiazem
Drug of choice for variant angina	Nitrates
Propanolol is NOT indicated in	Variant angina
Prinzmetal angina	Transient ST elevation
Prinzmetal angina	Pain at rest, transmural ischemia, ST elevation during attack, ST depression with pain
MC site of focal spasm in Prinzmetal angina	**Right coronary artery**
Drug increasing severity of prinzmetal angina	**Aspirin**

MANAGEMENT OF ANGINA

Ranolazine	*pFOX inhibitor*
Ranolazine is a selective inhibitor of	*iNa current*
Ranolazine is metabolized primarily by	*CYP3A4*
Unique feature of ranolazine	*Anti ischemic effects are achieved without clinically meaningful change in heart rate and blood pressure*
Ranolazine	NO effect of BLOOD PRESSURE, Not indicated in acute angina, Improves glycemic control
Dipyridamole	Adenosine uptake inhibition
Beneficial effect of metoprolol in management of secondary angina	*Increase in diastolic filling time*

NITRATES

Drugs decreasing preload	Glyceryl trinitrate, ACE inhibitors, Sodium nitroprusside
Nitrates	Release NO, cause vasodilatation, high first pass metabolism
Nitrates	Decreases left ventricular end diastolic pressure, Direct reduction of oxygen consumption of myocardial cell, Dilatation of capacitance vessels, Decreasing size of heart
Anti anginal action of nitrates	Decrease myocardial O2 consumption, decrease both pre and after load, cause favourable redistribution of coronary blood flow
GTN given by sublingual route because of	Hepatic first pass metabolism
Nitrates metabolized by	Guanathione reductase
Nitrates does NOT cause	Increase in cardiac work
Nitrates NOT used in	Renal colic
Nitrate does NOT undergo first pass metabolism	Isosorbide mononitrate
NOT given by sublingual route	Isosorbide 5 mononitrate
Longest acting nitroglycerine preparation	Pentaerythritol tetranitrate
Nitrate associated with allergic reaction	*Pentaerythritol tetranitrate*
Nitrate bypassing cysteine dependent pathway	*Molsidomine*
Route of amyl nitrite	Inhalation
Nitroglycerin cause	Hypotension and TACHYCARDIA
Side effects of nitroglycerine	Hypotension, Tachycardia, Methemoglobinemia, Vasodilatation
Treatment of idiosyncratic effects of nitrates	*Atropine*

MYOCARDITIS

Heart muscle	Act as syncitium, Single nuclei, Gap junctions, Has branching
Cardiac muscle is able to function as syncitium because of structural presence of	Gap junction
Intercalated discs present in	Cardiac muscle
Myocarditis may be associated with	Trichinosis, Corynebacterium diphtheria, SLE
Myocarditis can be associated with	*SLE, radiation*
Infantile myocarditis and pericarditis is due to	Coxsackie B
MC non infective myocarditis	**Granulomatous myocarditis**
Giant cell myocarditis	*Rapidly progressing heart failure, may be associated with thyroiditis, tachyarrhythmia are common, steroids are used in treatment*

SUDDEN CARDIAC DEATH

Cut off for sudden cardiac death	1 hour or less
System most commonly involved in sudden death	CVS
Sudden cardiac death may occur in	Dilated cardiomyopathy, Hypertrophic cardiomyopathy, Eisenmenger syndrome
MC ECG abnormality in sudden death	*Prolonged QT interval*

VENTRICULAR INFARCT AND HYPERTROPHY

Factors increasing length of ventricular cardiac muscle	Increased venous tone, increased total blood volume, increased negative intrathoracic pressure
Right ventricular infarct	Nocturia, hepatomegaly, ascites
NOT true about right ventricular infarct	Normal JVP
Left ventricular hypertrophy is caused by	MR,AS,MR
Left ventricular hypertrophy NOT seen in	MS
NOT a sign of RVH	Lower sternal dullness
NOT true about subendocardial hemorrhage	Involves RV wall
Left ventricular function in ventriculography is evaluated by	Technetium
Most accurate investigation for assessing ventricular function	Echocardiography
Cardiotoxicity caused by radiotherapy and chemotherapy is best detected by	Endomyocardial biopsy
Drug of choice in asymptomatic left ventricular dysfunction	Enalapril

MYOCARDIAL INFARCTION

BLOOD SUPPLY TO HEART

Coronary artery	Right coronary artery lies in right anterior coronary sulcus, Left anterior descending artery is a branch of left coronary artery, SINGLE obtuse marginal artery arise from left coronary artery, In 85% cases posterior descending interventricular artery arise from right coronary artery
Vasodilatation associated with hypotension in	Coronary circulation
Right coronary artery lies in	Right anterior coronary sulcus
Right coronary artery	Diameter less than LCA, RCA arises from anterior aortic sinus, RCA gives rise to circumflex coronary branch
Branch of right coronary artery	Acute marginal, Posterior interventricular
SA node is predominantly supplied by	Right Coronary artery
Right coronary artery supplies	SA node, AV node, AV bundle
Branches of right coronary artery	Acute marginal artery, Posterior interventricular artery, Posterior ventricular

In 85% of patients posterior descending interventricular artery arise from	Right coronary artery
Right coronary artery does NOT supply	Right bundle branch
Occlusion of anterior descending branch of LAD will lead to infection of	Anterior wall of left ventricle
Greater volume of myocardial tissue supplied by	Left Coronary artery
If circumflex artery gives of the posterior interventricular artery, then arterial supply is called	Left dominance
Left coronary artery	Anterior Descending, Circumflex
Widow's artery	Left anterior Descending
Involvement of anterior descending branch of left coronary artery	Anterolateral wall
Anterior wall of left ventricle is supplied by	*Proximal part of left anterior descending*
Right bundle branch and left bundle branch are supplied by	*Left coronary artery*
Third coronary artery	*Conus artery (arteria coni arteriosi from anterior aortic sinus)*
Kugel artery	*Arteria anastomotica auricularia magna*
Common site of occlusion of thrombus	Anterior interventricular, Posterior interventricular, Circumflex
NOT a common site of occlusion of thrombus	Marginal artery
Attachment of Thrombus	Firm
Left Common cardinal vein forms	Oblique vein of Left Atrium
Valveless tributary of coronary sinus	*Oblique vein of left atrium*
Vein in cardiac anterior Interventricular groove	Great cardiac vein
Anterior cardiac vein drains into	Right atrium
Middle cardiac vein is located in	Posterior interventricular sulcus
Coronary sinus	Remnant of left horn of sinus venosus, Great middle and small cardiac vein drain into it, Thebesian valve guard its opening
Thebesian veins	Venae cordi minimi (smallest cardiac veins), open directly into all four chambers
Coronary sinus ends in	Right atrium
Vein NOT draining into Coronary sinus	Anterior Cardiac vein
Anterior cardiac vein drains in to	*Right atrium*
Thebesian valve is located in	*Coronary sinus*

FEATURES OF MI

Strongly associated with coronary heart disease	Apolipoproteins
Killik classification	*Myocardial infarction*
Shoulder hand syndrome	*MI*
Levine sign	**Substernal discomfort**
Most susceptible to ischemia	Myocytes
MC cause of acute epigastric pain	Myocardial infarction
Pain is not uniformly present in	**ST elevation MI**
MC artery involved in Myocardial infarction	Left anterior descending artery

Involvement of anterior descending branch of left coronary artery	Anterolateral wall infarct
Occlusion of anterior descending branch of LAD will lead to infarction of	Anterior wall of left ventricle
MC site of MI	Anterior wall of left ventricle
Universal definition of myocardial infarction	Sudden unexpected cardiac death with symptoms of ischemia, Elevation of cardiac biomarkers with new regional wall motion abnormality, Three times increase in troponin levels after PCI
Right ventricular infarction	Associated with inferior wall MI, JVP is raised, diagnosis is confirmed by right side chest leads on ECG, arrhythmia, cardiomegaly, hypotension
Subendocardial infarction	Multifocal in nature, often result from hypotension or shock, epicarditis is NOT seen , does NOT result in aneurysm
Neonate has recurrent attacks of abdominal pain, restless irritability and diaphoresis on feeding. Cardiac auscultation reveals a non specific murmur. believed to be at risk of MI	Anomalous coronary artery
Acute MI associated with	Chest pain, Gallop, Systolic murmur in mitral area
Acute coronary syndrome does NOT include	Prinzmetal angina
Pain of Myocardial infarction does NOT radiate to	Left iliac fossa
Rapid x descent rare in	RVMI
MC cause of death immediately after MI	*Arrhythmia*
NOT true about coronary heart disease in india	CHD presents a decade later than in western countries
NOT true about coronary heart disease	Influence of smoking is only additive to other risk factors for CHD
Best predictor for coronary heart disease	LDL
Used to perform stress echo	Dobutamine
Somatotrophin is contraindicated in	Ischemic heart disease

MORPHOLOGY OF MI

Major histological feature of MI	Coagulative necrosis
Autopsy finding after 12 hours in case of death due to MI is	Coagulative necrosis
Pathology of myocardial infarction	Neutrophilic infiltration around coagulative necrosis
Earliest light microscopic change in myocardial infarction (1 – 3 hours)	Waviness of fibres
2 - 3 hours	*Staining defect*
4 - 12 hours	*Coagulation necrosis*
12 - 24 hours	*Dark mottling*
18 - 24 hours	*Pyknosis*
60 year male, acute chest pain, new Q wave, ST segment depression, succumbed to his illness within 24 hours of admission. Heart revealed presence of transmural hemorrhagic area over septum and anterior wall of left ventricle. light microscopy finding	Necrotic myofibres with presence of neutrophils
Coagulative necrosis with neutrophilic infiltration in	1-3 days

Myocardial infarction seen after	
24 – 72 hours	*Neutrophils and loss of nuclei*
Cells seen after 72 hours in infracted area in MI	Macrophages
In myocardial infarction, infarct acquires hyperaemic rim with a yellow centre	3 – 7 days
Myocardial infarct showing granulation tissue has most likely occurred	Within one week
3 – 7 days	*Macrophages*
Granulation tissue following MI	7 – 10 days
A myocardial infarction showing early granulation tissue has most likely occurred	Within 1 month
10 – 12 days	*Fibrovascular response*
7 weeks	*Fibrosis*
Infracted myocardium completely replaced by scar tissue by	8 weeks
Myocardial scarring completes by	3 months

COMPLICATIONS OF MI

Patient with acute anterior wall MI and hypotension, immediate treatment	Angiography and pulmonary angioplasty
Best modality of treatment in acute inferior wall MI	IV fluids
Most deaths in MI occur during	First 24 hours
Most of deaths in MI	1st day
Reason for shock in inferior wall MI	Right ventricular infarction
Parasympathetic activity is associated with	*Inferior wall MI*
Pansystolic murmur after MI is due to	*Elevated LA pressure which in turn leads to rupture of papillary muscle*
70 year old, hypertensive male, transmural anterolateral MI, stable till 5th day. Painful friction rub and pleuritic chest pain, persisted despite narcotic and steroid therapy. On seventh day morning, marked hypotension. marked distension of jugular veins, electromechanical dissociation	External cardiac rupture
Post MI Rupture, Leakage at	Site of Vascular Anastomosis
Complication of MI occurring between 3 – 7 days	Rupture of left venticular free wall
Dressler syndrome	Occurs 1 to 4 weeks after myocardial injury, Chest pain is common, Recurrence may be seen, Responds well to salicylates
Dressler syndrome is associated with	Pleural effusion
Dressler syndrome is	Autoimmune
NOT true about Dressler syndrome	Myocarditis

ENZYMES IN MI

Correct sequence of increase in enzymes in myocardial infarction	CPK, AST, LDH

Both CKD and LDH are raised in	Myocardial infarction
Increased CK and SGOT levels are seen in	Myocardial infarction
NOT a marker of MI	Calmodulin
Flipped pattern of LDH isoenzymes	Myocardial Infarction
Isoenzyme specific for MI	LDH 1
Serum total LDH raised in	Muscle crush injury, myocardial infarction, hemolysis
First marker in MI	Myoglobin
Earliest enzyme to elevate after MI	Myoglobin
CPK is increased in	Alcoholic myopathy, Clofibrate therapy, After electrocardioversion
Enzyme raised in 4 to 6 hours and decreases in 3 to 4 days	CPK
Heart muscles contain isoenzyme	MM and MB
Investigation of choice for second MI after 1 week of previous MI	CPK-MB
Enzyme elevated in first 2 hours of MI	CPK MB
Enzyme of choice during reperfusion	CK- MB
Best enzyme assay within 3 hours following acute MI	CK -MB
Most Sensitive enzyme for Myocardial Infarction	Troponin
Biomarker of recurrent MI	*CKMB*
Test of choice in patient coming 12 hours following MI	Cardiac troponin
Marker of choice in Myocardial infarction with hypothyroidism	Troponin I
Normal value of troponin T	**0 – 0.01 microgram/L (ng/ml)**
Troponin T preferable to CPK MB in diagnosis of acute MI in	Bedside diagnosis of MI, post operatively after CABG, small infarcts
Best indicator of MI after 72 hours	Cardiac specific troponin T
Troponin T is a marker of	Myocardial infarction
Preferred marker of acute STEMI in athletes	Troponin T
Troponin T is NOT preferable in	Reinfarction after 4 days

DIAGNOSIS IN ISCHEMIC HEART DISEASE

Pain epigastrium, difficulty in breathing, initial investigation	ECG
Feature of acute coronary syndrome	*ST depression and T wave inversion*
ECG is poor in detecting ischemia in areas supplied by	Left circumflex artery
ECG finding associated with acute MI	Tall T wave with increased amplitude
ST segment elevation	Early repolarisation variant, ventricular aneurysm, prinzmetal angina
ST elevation in II, III, AVF indicates	Inferior wall MI
Fresh myocardial infarction in ECG	ST segment elevation
ST elevation and hyperacute T waves in precordal leads V1 to V6 and in lead aVL	Anterolateral wall MI
Characteristic ECG finding of transmural myocardial infarction	Pathological Q waves
Most sensitive lead in detecting intraoperative ischemia	V5
NOT seen in ECG tracing of MI	Biphasic P wave
Coronary angiography can visualize vessels with lumen	0.5 mm

up to	
Drug used to perform stress echo	Dobutamine
Phase of minimum motion during cardiac imaging	Mid diastole
Following an attack of MI, mortality and morbidity of a patient is indicated by	Left ventricular ejection fraction
Ischemic cardiac tissue shows	Anaerobic Glycolysis
Myocardium	Rest injection thallium scan is used in hibernating myocardium, Late Gd MRI enhancement is suggestive of scar but Gd scan is not used for hibernating myocardium detection
Hot spot in acute myocardial infarction	Tc99 strontium pyrophosphate
Infarct Avid imaging/Hotspot imaging	Myocardial Infarction
Myocardial viability is detected by	Thallium scan
Test of choice for reversible Myocardial ischemia	Thallium scan
Nuclear cardiac imaging of heart utilizes	Thallium
Commonly used thallium	**Thallium 201**
Best time to perform myocardial infarct imaging with thallium 201	*12 – 24 hours*
SPECT scan is used for	**Heart**
Bruce protocol for	**Ischemic heart disease**
Modified exercise test	**6 days after MI**

MANAGEMENT OF MI

Best possible intervention of acute myocardial infarction	Early primary coronary intervention
Drug used for Pain relief in MI	Morphine
Low dose aspirin used in myocardial infarction act by	Inhibit cycloxygenase
Immediate intervention in ST segment elevation in inferior leads	Aspirin
Reperfusion is believed to restore contractile function of	Hibernating myocardium
Accelerated idioventricular rhythm is most common arrhythmia associated with	Myocardial reperfusion
Chest pain, ST segment depression, NOT given	Thrombolytic
Antineoplastic drug best avoided in myocardial infarction and congestive cardiac failure	Anthracycline
Drug contraindicated in acute MI	Pentazocine
NOT used in myocardial infarction	Inhibitors of plasminogen activator
Drug NOT given in ischemic heart disease	Isoproternol
NOT used for intraoperative management of myocardial ischemia	Heparin
NOT used in acute myocardial infarction	Calcium channel blocker
NOT a management of Unstable angina/ STEMI	Lignocaine bolus
NOT used in management of acute MI	Warfarin
Anterior wall MI, RBBB left atrial hypertrophy	Temporary pacing
Best treatment for STEMI	PTCA
Immediate modality in acute anterior wall MI and hypotension	Primary angioplasty
Percutaneous coronary intervention through	**Femoral artery**

Fractional flow reserve is used in	*Coronary catheterization*
Most effective management in triple vessel heart disease	CABG
CABG is best indicated in	Double vessel disease with CCF
NOT an indication of CABG	To prevent progress of native blood vessel disease
Tolazoline	Vasodilator in treating coronary artery stenosis during angio procedure
Tolazoline	As vasodilator in treating coronary artery stenosis during angioprocedures
CABG in NOT done to	Prevent progress of native blood vessel disease
Coronary care unit and cancer care facility to be established at the	District level hospital

THROMBOLYSIS

TIMI 0 means	**Complete occlusion**
Thrombolytics can be given in treatment of AMI, if patient comes within	12 hours
Thrombolysis should be started in Ischemic stroke within for Maximum benefit	3 hours
Thrombolysis is CONTRAINDICATED IN	Non STEMI
NOT a contraindication for thrombolytic therapy	Supraventricular tachycardia
Recurrent ischemic events following thrombolysis has been physiologically linked to	Lipoprotein A
Most common cause of death in a patient with thrombolytic therapy?	Intracranial hemorrhage
Widely used thrombolytic agent	Streptokinase
Commercial source of streptokinase	*S. dysgalactiae (subspecies equisimilus)*
Streptokinase and urokinase are contraindicated in	Intracranial malignancy
Complications of streptokinase	Intracranial bleed, fever, anaphylaxis, hypotension
Bolus fibrinolytics	*Tenecteplase, Reteplase*
Plasminogen activator produced by recombinant DNA technology	Alteplase

WARFARIN

Anticoagulant action of warfarin is monitored by	PT
Treatment of warfarin toxicity	Phytonadione

AORTIC DISSECTION

Cardiovascular causes of clubbing	Infective endocarditis, AV fistula, tricuspid atresia
Digital clubbing NOT seen in	Aortic dissection
Factors predisposing to aortic dissection	Systemic hypertension, Coarctation of aorta, Takayasu arteritis, Marfan syndrome

MC cause of dissecting aneurysm of thoracic aorta	Medial degeneration
MC cause of abdominal aortic aneurysm	Atherosclerosis
Dissection of which artery is common in pregnancy	Aorta
MC site of aortic dissection	Ascending aorta
MC site of aortic dissection	*Near aortic valve*
Sudden onset of excruciating pain, radiating to back, shock, distension of abdomen, mild rigidity, precipitated by exertion	Dissecting aneurysm of aorta
Manifestation of acute aortic dissection	Pericardial effusion, AR, AMI, limb ischemia
Apical cap on chest X ray	*Dissecting aneurysm*
Abdominal aortic aneurysm may be complicated by	**Occlusion of artery of adamkiewicz**
Severe back pain in abdominal aortic aneurysm	Enlargement of sac
Most common complication of aortic aneurysm of size 8 cm	Rupture
Most common site of rupture of abdominal artery aneurysm	Laterally into left retroperitoneum (infrarenal)
Abdominal aortic rupture usually ruptures	*Posterolaterally*
Stanford classification for	**Aortic dissection**
Investigation of choice for Aortic Dissection(Stable)	MRI
Investigation of choice for Aortic Dissection(Unstable)	Transesophageal ECHO followed by CT
Diameter of aortic aneurysm is best described by	MRI
Procedure of choice for evaluating aneurysm	Arteriography
Fibroptic endoscopy contraindicated in	Aneurysm of arch of aorta
Treatment of aortic dissection	Propanolol, sodium nitroprusside, labetalol
Bentall's procedure is for	*Aortic root aneurysm repair*
Criteria for endovascular repair of aneurysm	*Asymptomatic infrarenal or common iliac aneurysm, anatomy suitable for repair, aneurysm neck length > 10 mm, external and common iliac arteries must accommodate the device*

ARRHYTHMIAS

CARDIAC CONDUCTION

Resting membrane potential of sino nodal fibres	*-55 mV*
SA node	Situated at junction of SVC and right atrium, Contains specializes nodal cardiac muscle, Initiates cardiac conduction
SA node	**No t tubule**
Sinus arrhythmia is produced by	SA node
Slow depolarizing pre potentials are characteristic of	SA node
SA node is pacemaker because its excitability is	Highest of all
SA node acts as pacemaker because	It generates impulses at highest rate
Blood supply to SA node	*Right posterior interventricular artery*

Right posterior interventricular artery is accompanied by	*Middle cardiac vein*
Resting membrane potential of myocardial fibers	*-90mV*
Initial depolarization is due to	*Rapid Na+ influx*
Plateau phase is due to	*Slow Na+ influx*
Repolarization is due to	*K+ efflux*
Action potential in SA node and AV node owing to	*Ca++ influx*
Initiation of prepotential in cardiac pacemaker is due to	*Closure of K+ channel*
Vagal stimulation on membrane potential of SA node	*Activates hyperpolarizing potassium current*
Stronger than normal stimulus cause excitation during	*Relative refractory period*
Refractory period is NOT affected by	*Sympathetic stimulation*
Pacemaker potential or prepotential	*Decrease in K+ permeability*
Prepotentials are normally absent in	*Purkinje fibres, working myocardial cells*
Predepolarisation phase of SA node action potential is due to	Ca++ entry
Slowest conduction velocity	Atrial myocardial fibres
Least conduction velocity seen in	AV node
Decremental conduction is associated with	**AV node**
AV bundle is related to	*Membranous part of ventricular septum*
Right coronary artery supplies AV node in	*60% of individuals*
Atrioventricular Nodal delay is due to	Resistance to Ion flow
AV nodal delay is increased by	*Stimulation of left vagus*
Ability of AV node to generate its own impulse when sinus node is sick	*Spontaneous diastolic depolarization*
Fibres from AV node to RV	*Moderator band*
Fibres from AV node to fascicles	**Mahaim fibres**
AH interval (conduction time from atria to His bundle)	*60 – 125 ms*
Maximum velocity of Transmission in heart in	Bundle of His
Conduction rate is fastest in	Purkinje fibres
Order of activation after stimulation of Purkinje system	Septum > Endocardium > epicardium
Repolarisation in isolated muscle pierce fibre from	Endocardium to epicardium
Plateau phase of ventricular muscle is due to opening of	Ca – Na channel
Principal determinant of inotrophic state of heart	**Intracytoplasmic Ca++**
Extrasystole in ventricle	Falls to produce radial pulse, Associated with abnormal QRS complex, Tendency to be followed by a compensatory pause
NOT true about extrasystole	Hints at serious heart problem
Patient develops sudden palpitation HR 150/min	Sinus tachycardia
Bradycardia is caused by	Propanolol, clonidine, reserpine
Management of Severe bradycardia	Atropine, pacing, isoproterenol
Should be given safely in patients with sinus	Esmolol

bradycardia	
Asystole	*Absence of cardiac conduction > 2000 ms*
Fibrocalcareous encroachment into the conducting system	Lev's disease
Ashman phenomenon is seen with	Atrial fibrillation (right bundle branch block)
Left bundle branch block	**Coronary heart disease, hypertensive heart disease, aortic valvular disease, cardiomyopathy**

GENERAL FEATURES OF ARRHYTHMIA

Patient taking ketaconazole and terfenadine prone for	Cardiac arrhythmia
MC mechanism of arrhythmia	**Re entry**
MC cause for extrinsic SA node dysfunction	**Drugs**
SA node dysfunction is associated with	**Kearns Sayre syndrome**
Tachycardia bradycardia syndrome	**Autosomal dominant SA nodal dysfunction syndrome**
Transient SA node dysfunction is associated with	**Inferior wall MI**
Frog sign	**AV nodal reentrant tachycardia**
Pseudo R waves are associated with	**AV nodal reentrant tachycardia**
Epsilon wave is associated with	**Arrhythmogenic right ventricular dysplasia**
Naxos disease	**Arrhythmogenic right ventricular dysplasia/cardiomyopathy, woolly hair, palmoplantar keratosis**
Cyclic variation of heart rate	*Sinus arrhythmia*
Respiratory sinus arrhythmia	*Decrease in heart rate during expiration and increase in heart rate during inspiration, abolished by atropine, reduced in elderly*

ATRIAL FLUTTER

Arrhythmia commonly associated with alcohol binge in alcoholics	Atrial flutter
Drug most useful in atrial flutter	Amiodarone
Most effective treatment of Atrial flutter	DC shock

ATRIAL FIBRILLATION

MC arrhythmia associated with alcohol binge in alcoholics	Atrial fibrillation
Atrial fibrillation is due to	Thyrotoxicosis
Atrial fibrillation	Mitral stenosis, constrictive pericarditis, diphtheritic myocarditis
Atrial fibrillation does NOT occur in	Hypothyroidism
Irregularly irregular pulse	Atrial fibrillation
Known mitral stenosis, atrial fibrillation, acute onset of weakness rcovered completely in 2 weeks	Ischemic stroke
NOT true about atrial fibrillation	Anticoagulant NOT required
P waves are absent in	Atrial fibrillation

Treatment of resistant cases of Atrial fibrillation	Amiodarone
Drug of choice in wolf parkinson's syndrome with atrial fibrillation	Procainamide
Digitalis has most profound effect in	Atrial fibrillation
Purpose of digitalis in atrial fibrillation	Slow ventricular rate
Vernalakant is used in treatment of	*Atrial fibrillation*
Drug of choice for ectopic atrial tachycardia	*Amiodarone*
Most effective for conversion of atrial fibrillation to sinus rhythm	*Amiodarone*
Most effective treatment of atrial fibrillation	DC shock
NOT used in Atrial arrhythmia	Lignocaine
Maze operation is done for	*Atrial fibrillation*

VENTRICULAR TACHYCARDIA

Most common type of surpaventricular tachycardia in infants	*AVRT*
Type of SVT	Wolff Parkinson White Syndrome
Features of ventricular tachycardia	Variable first heart sound, Can NOT be relieved by carotid sinus massage, QRS duration >0.14 sec
Patient with wide complex tachycardia, which indicates ventricular tachycardia	AV dissociation, fusion beats, capture beats
Ventricular tachycardia	Fusion beat, Capture beat, AV dissociation, Bizzare QRS complexes
Repolarization alterans is seen in	*Ventricular tachyarrhythmia*
NOT true about ventricular tachycardia	Similar QRS in all leads
Treatment of Ventricular Tachycardia	Lignocaine
Tocainide	Used in ventricular tachycardia, used as lidocaine analogue, orally
Heart rate is slowed by	*Carotid massage*
Used for treatment of supraventricular tachycardia with hypotension under general anesthesia	Carotid sinus massage, adenosine, direct current cardioversion
Mechanism of abruption of SVT by cardiac massage	*Increase parasympathetic discharge to SA node*
Drug of choice for PSVT	Adenosine,Verapamil
Drug of choice in most cases of acute AV nodal tachycardia	Adenosine
Adenosine	Used in PSVT, administered as rapid iv infusion, short lived side effects
Adenosine	Dipyridamole potentiates action, Used to produce controlled hypotension
Arrhythmias that can be treated with Adenosine	Atrial flutter, Paroxysmal atrial tachycardia, supraventricular tachycardia
PSVT Rapid Control in Known Asthmatic	Verapamil
Treatment of antipsychotic induced ventricular tachydysrhthmia	**Sodium bicarbonate**
Drug of choice for ventricular	*Amiodarone*

tachycardia during cardiac arrest	
Digitalis is contraindicated in	Ventricular tachycardia

VENTRICULAR FIBRILLATION

MC electrical mechanism for cardiac arrest	**Ventricular fibrillation**
MC cause of Sudden Cardiac Death	Ventricular Fibrillation
MC cause of death from Aortic stenosis in children	Ischemic heart disease with Ventricular fibrillation
MC cause of death from chloroform anaesthesia	Ventricular fibrillation
Ventricular fibrillation is associated with	Follows ventricular tachycardia, quick fall in cardiac output
VF is treated by	Lignocaine
Drug of choice for ventricular arrhythmia due to MI	Xylocaine
First line of treatment if a patient develops ventricular fibrillation after intravenous infusion of potassium chloride	Defibrillation
Treatment of Ventricular fibrillation	Immediate electrical cardiac version

MANAGEMENT OF ARRHYTHMIA

Class IA antiarrhythmic (Na+ K+ blocker)	*Quinidine, procainamide, disopyramide*
Quinidine	Class IA antiarrhythmic
Quinidine is a	Na+ blocker
Quinidine exerts its action on heart by inhibiting	Na+ channel
Quinidine	Decreases automaticity in heart
Quinidine	Increases effective refractory period, Paradoxical tachycardia, Cinchonism is seen
Quinidine is contraindicated in	Bifascicular block, acute carditis, thyrotoxicosis
Effective refractory period is prolonged by	Propanolol, Verapamil, Lignocaine
Procainamide	Class I antiarrhythmic
Class IB antiarrhythmic (Na+ blocker K+ opener)	*Lignocaine, phenytoin, mexilentene, tocainamide*
Mexiletine	Class IB antiarrhythmic
Lignocaine	Class IB antiarrhythmic
Anti arrhythmic drug decreases action potential duration is purkinje fibres	Lignocaine
Antiarrhythmic NOT proarrhythmic	Lignocaine
Antiarrhythmic drugs which are NOT proarrhythmic	**Verapamil, diltiazem, moricizine**
Class IC antiarrhythmic (Na+ blocker)	*Flecainadine, encainidine, propafenone, moricizine*
Does NOT belong class IC antiarrhythmic	Tocainide
Feature of class IC anti arrhythmic agents	*Pro arrhythmic*
Esmolol	Class II antiarrhythmic
Beta blockers are antiarrhythmogenic agents of type	II
Sotalol	Non selective beta blocker, prolongs action potential duration throughout heart, polymorphic ventricular tachycardia is a common side effect, excreted unchanged in urine

Sotalol excreted mainly by	Kidney
Class III (K+ blocker)	*Amiodarone, bretylium, ibutilide*
Anti arrhythmic least likely to cause torsades de pointes	*Amiodarone*
Amiodarane	Class III antiarrhythmic
Action of amiodarone	Action potential duration is prolonged, effective refractory period is prolonged, conduction is slowed
Antiarrhythmic drug cause prolonged repolarisation of ventricles and ERP	Amiodarone
Arrhythmias refractory to treatment of lignocaine can be treated by	Amiodarone
Dronedarone	*Shown to reduce hospitalization in patients with AF*
Side effects of amiodarone	Pulmonary fibrosis, Hypothyroidism, Hyperthyroidism, Corneal microdeposits, Cirrhosis of liver
NOT an adverse effect of chronic amiodarone therapy	SLE
NOT seen with amiodarone therapy	Productive cough
Dilitazem	Class IV antiarrhythmic
Verapamil	Class IV antiarrhythmic

HEART BLOCK

Bifascicular block	*LBBB and RBBB*
Trifascicular block	*Alternating RBBB with LBBB*
Constant PR interval	First degree block, Mobitz type II second degree block
Earliest sign of diphtheric myocarditis	First degree AV block
Treatment of 1st Degree AV block	Atropine
Digoxin classically causes	Mobitz type 1 block
Mobitz type I block	Varying PR interval, Normal QRS morphology, Regular Atrial rhythm, Atrial rate > Ventricular rate
Type I second degree block	**Progressive prolonging PR interval**
Wenkebach phenomenon is seen in	II degree AV block Mobitz type I
2nd Degree AV block Type I	Atropine
Type II block	**Intermittent failure of conduction, associated with paroxysmal AV block**
Stroke Adams attack occurs	II degree AV block Mobitz type II
2nd Degree AV block Type II	Temporary Pacemaker
Atenolol is NOT indicated in	Partial heart block
Child born to SLE mother	Heart block
Anti SSA(Rho) & Anti SSB(La)	Congenital Heart Block
History of Stroke Adam's attacks, giddiness, Collapse	Unstable block
AV nodal block	Clinical evidence of inferior MI, wenkebach periodicity of conduction, escape focus rate faster than 50 beats per minute
Symptoms of cerebral ischemia is associated with	*Infranodal block*
Treatment of Complete Heart Block	Permanent Pacemaker
Treatment of choice in symptomatic sinus node dysfunction	Permanent pacemaker

Pacemaker therapy is indicated in	Congenital AV block, trifascicular block, sick sinus syndrome
Twiddler syndrome	**Rotation of pacemaker**
Acute symptomatic sinus bradycardia usually responds to	Atropine
NOT a modality of treatment in severe bradycardia	Diltiazem
ICD means	**Implantable cardiac defibrillator**
Implantable cardiac defibrillator	**Primary prevention**

VENTRICULAR PREMATURE CONTRACTION

Premature ventricular beat	Sequential depolarisation of ventricles, Wide bizzare notched QRS complex, Palpitation is a common presenting feature, Wide QRS, Absent P wave, Complete compensatory pause, Fusion beat, Capture beat, AV dissociation
Most characteristic finding of ventricular premature complex	Wide QRS complex
Ventricular premature complexes are associated with	**Full compensatory pause**
Interpolated VPC	**VPC without compensatory pause**
NOT a feature of ventricular premature complex	Narrow QRS complex
Drug of choice for ventricular premature beats due to digitalis toxicity	Diphenylhydantoin
MC arrhythmia encountered in digitalis toxicity	Bigeminous rhythm
Proper treatment of ventricular bigeminy	Cessation of digitalis and administration of potassium

TORSADES DE POINTES

QT prolongation seen in	Hypocalcemia, hypothermia, Romanowand syndrome
Torsades des pointes caused by	Quinidine
Torsades de pointes caused by	Quinidine, Disopyramide, Procainamide
Drug implicated for prolonging QT interval in premature baby	Cisapride
Co administration of ketoconazole and cisapride cause	Torsades de pointes, ventricular fibrillation and ventricular tachycardia
Cisapride for Barrett's ulcer. He develops pneumonia. Physician prescribes erythromycin	Increased risk of ventricular arrhythmia
MC type of long QT syndrome	**LQT1**
LQTS1	*Stress induced*
LQTS3	*Sudden death during sleep*
Congenital long QT syndrome can lead to	Polymorphic ventricular tachycardia
Congenital long QT syndrome is associated with	Neonatal sinus bradycardia
Treatment of long QT syndrome	*Beta blocker*
Best treatment for congenital long QT syndrome	*Implantable cardiac defibrillator*
Feature of Torsades des pointes	Prolonged QTc interval
Torsade de pointes	Hypomagnesemia
Known prolonged congenital QT syndrome and	Metoprolol

intermittent torsade de pointes	
Treatment of Choice for Torsades de pointes	Magnesium Sulphate
Tachycardia NOT amenable radiofrequency catheter ablation	*Torsades de pointes*

WPW SYNDROME

MC accessory pathway	Left free wall
WPW syndrome caused by	Right sided accessory pathway
WPW syndrome	Common in males
WPW syndrome	Right ventricular aberrant is commonly seen, incidence decreases with age, heart is structurally normal
MC reentrant tachycardia associated with WPW syndrome	Orthodromic AV reentry
WPW syndrome is associated with	**Echo beat (echo wave)**
In Wolff Parkinson syndrome connection between atrium and	Ventricles
ECG findings in WPW syndrome	Normal QT interval, slurred tall QRS, Short PR interval
Short PR interval with delta waves	WPW syndrome
NOT a cardiac contraindication to pregnancy	WPW syndrome
Treatment of choice for Wolff Parkinson White Syndrome	Radiofrequency ablation
Asymptomatic child with delta wave short PR interval which drug not to be given	Beta blocker

ATHEROSCLEROSIS

CAUSES OF ATHEROSCLEROSIS

Causative organism for coronary atherosclerosis	Chlamydia pneumonia, CMV, Herpes Simplex
Organism implicated in coronary artery disease	Chlamydia
Aminoacid associated with atherosclerosis	Homocysteine
Increased level of lipoprotein predispose to	Atherosclerosis
Risk factors for atherosclerosis	Increased homocysteine, increased lipoproteins, increased fibrinogen, increased plasminogen activator inhibitors
Atherosclerotic plaque formation is due to	*Persistent endothelial injury*
Increased risk of atherosclerotic plaque formation associated with	Apo E mutation, oxidized LDL, increased homocystine
Susceptibility to coronary artery disease	Nephrotic syndrome
Predisposing factors for CAD	Homocystinemia, Increased fibrinogen, Increased plasminogen activator inhibitor
Predispose to atherosclerosis	Homocystinemia, Fibrinogen, Lipoprotein A
Drug causing Hyperlipidemia	Prednisolone
Highest Risk of CHD is seen in	Familial Hypercholesterolemia

Agatston score is used for	*Quantification of calcified plaque*
NOT a predisposing factor for atherosclerotic plaque	Alpha 2 macroglobulin
Metabolic syndrome is associated with	**Decreased adiponectin**
NOT a criteria for metabolic syndrome	LDL
NOT a risk factor in IHD	Diabetes insipidus
NOT a risk factor for atherosclerosis	Decreased fibrinogen levels
Dietary relation not established in CAD	K+
Atherosclerosis risk decreased with	PUFA intake

FEATURES OF ATHEROSCLEROSIS

MC cause of Renovascular Hypertension in Elderly	Atherosclerosis
MC cause of True Aneurysm in India	Atherosclerosis
MC cause of Peripheral Limb Ischemia in India	Atherosclerosis
MC cause of Abdominal Aortic Aneurysm	Atherosclerosis
MC cause of thoracic aortic aneurysm	Atherosclerosis
MC cause of Aortic aneurysm	Atherosclerosis
MC cause of peripheral limb ischemia in India	Atherosclerosis
Chronic smoking pain in lower limb due to obstruction of femoral artery	Atherosclerosis
Atherosclerosis affects	Medium and Large sized vessels
Coronary artery most commonly involved in atherosclerosis	Left anterior descending artery
MC site of coronary atherosclerosis	**Epicardial coronary artery**
LEAST common site of atherosclerotic lesions	Pulmonary artery trunk
Arteries spared in atherosclerosis	*Arteries of upper extremity, mesenteric arteries, renal arteries*
Elderly man with fusiform dilatation of descending aorta because of	Atherosclerosis
Mortality in emergency abdominal aneurysm repair	>50%

MORPHOLOGY IN ATHEROSCLEROSIS

Thush breast / tigered effect	*Fatty change in heart*
Characteristic lesion of atherosclerosis	Fibrofatty lesion in intima of blood vessel
In atherosclerosis, increased LDL in monocyte macrophage is due to	Lipids in LDL gets auto oxidized
Pathophysiological phenomenon that occurs during atheromatous plaque formation and is used for screening of asymptomatic coronary plaque	Calcium deposition in atheromatous plaque
Atheromatous Plaques do NOT contain	Neutrophils

DIAGNOSIS OF ATHEROSCLEROSIS

Marker to predict serum atherosclerosis in early age	Apoprotein B100
Raised serum level of lipoprotein A is a predictor of	Atherosclerosis

Best predictor for future risk of cardiovascular events	Hs CRP
Triglyceride level risk for IHD	>150
Best marker for dyslipidemia	LDL/HDL ratio
Best predictor of risk of developing cardiovascular disease	LDL/HDL ratio
Most important in causing coronary artery disease	LDL
LDL is checked by	*Skin fibroblast culture*
Most important predictor of coronary artery disease	HDL
Lipoprotein acting as a scavenger and preventing atherosclerosis	HDL
Protective against atherosclerosis	HDL
Inverse relation for increasing risk of Atherosclerosis	HDL
Coronary calcium is quantified by	**Agatston scoring**

HYPOLIPIDEMIC DRUGS

HDL specifically increased by	Niacin
Favourable lipid profile is provided by	Nicotinic acid
Nicotinic acid	**Decreases VLDL product**
HDL levels are increased by	Nicotinic acid
In a patient with poor glycemic control, hypertriglyceridemia, low HDL, which drug will be best without myositis as side effect	Nicotinic acid
Class of hypolipidemic drug used safely in pregnancy	*Niacin*
Laropiprant is used with	*Niacin*
Drug contraindicated in severe hypertriglyceridemia	Niacin
Most potent drug to reduce plasma cholesterol level	Statins
First step in cholesterol synthesis inhibited by	Statins
HMG CoA reductase inhibitors	CNS accumulation of simvastatin and lovastatin is high and less for pravastatin and fluvastatin, Simvastatin is rapidly and pravastatin is least metabolized, Bioavailability is minimally modified when pravastatin is taken with food
HMG coA reductase inhibitors	CNS accumulation of simvastatin and lovastatin is high and less for provastatin and fluvastatin, simvastatin is rapidly metabolized and provastatin is least, bioavailability is minimally modified when provastatin is taken with food, fibrinogen levels are decreased by provastatin
Statins on HDL	Increases
Lovastatin	HMG CoA reductase inhibitor
Chinese yeast rice contains substance having action of	**Lovastatin**
Pravastatin	Decrease fibrinogen levels
Severe myopathy is a common side effect of	Rosuvastatin
Grape juice should not be given to patients on	Atorvastatin
First step in cholesterol synthesis inhibited by	Lovastatin
Teratogenicity of statins	*VACTREL*
Fibrates	Increase lipoprotein lipase activity through PPAR alpha and cause increased lipolysis of triglycerides, cause

	utricaria, rash, alopecia, myopathy, GI distress, first line of drugs in severe dysbetalipoproteinemia and hypertriglyceridemia, absorption increased when taken with food
Fibrates	Absorbed good when taken with food, Drug of choice for type III hyperlipoproteinemia and severe hypertriglyceridemia, Activate PPAR to stimulate LPL Side effects are rash, utricaria, myalgia
Newer fibrates	*Can be given with or without food*
Action of clofibrate	Activating lipoprotein lipase resulting in VLDL degradation
Fibrates are contraindicated in	*Gall stones*
Drug used in treatment of hypertriglyceridemia in primary stage	Gemfibrozil
Ezetimibe acts by	Decreased absorption of cholesterol
Drug reducing cholesterol levels by reducing cholesterol absorption in intestine by acting on NPC1L1 receptor	Ezetimibe
Ezetimibe	Inhibition of Intestinal cholesterol absorption
Probuchol	Inhibition of LDL oxidation
Cholestyramine	Basic ion exchange resin, cause compensatory increase in HMG CoA reductase activity, may cause constipation, steatorrhoea
INH can be used in	CAD
Icosapent is used in treatment of	*Hyperlipidemia*

PREVENTION OF ATHEROSCLEROSIS

HDL in CAD should not be less than	32
Effect of omega 3 fatty acids	Increased LDL, decreased cholesterol
NOT a dietary goal in patient with risk of coronary heart disease	Avoid alcohol

CARDIAC TUMOURS

Cardiac polyp	Fibrinous clot
Cardiac tumor in childhood	Rhabdomyoma, Myxoma, fibroma
MC Cardiac tumor of Children	Rhabdomyoma
MC tumor of infancy	Rhabdomyoma
MC soft tissue tumor in child	Rhabdomyosarcoma
Marker of Rhabdomyosarcoma	Desmin
MC tumor of cardiac valves	**Papillary fibroelastoma**
NAME syndrome	**Nevi, Atrial myxoma, Myxoid neurofibroma, Ephelide**
MC Primary cardiac tumour of Adults	Myxoma
MC primary tumour of heart	Myxoma
MC intracavitary benign cardiac tumour	Myxoma
Cardiac myxoma common in	Left atrium
Favoured site of origin of Myxoma	Fossa ovalis in atrial septum

Features of Myxoma	Fever, clubbing, embolic phenomenon
Blue naevi and Multiple lentigenes associated with	Atrial myxoma
Lipidic cells	Myxoma of heart
Substance accumulated in Cardiac Myxoma	Mucopolysaccharides
Gradient in pulmonary artery wedge pressure and left ventricular end diastolic pressure is seen in	Left atrial myxoma
Atrial myxoma may mimic	*Infective endocarditis*
NOT true about myxoma	Hypertension, familial
Intracardiac mass is detected by	**2D TTE**

RHEUMATIC FEVER

FEATURES OF RHEUMATIC FEVER

Serotype frequently associated with Rheumatic fever in India	*M5*
MC cause of acquired heart disease	Acute rheumatic heart disease
Percentage of coincidence between Sore throat and Acute Rheumatic fever	3%
Rheumatic fever	Caused by beta hemolytic streptococci
Age group for Rheumatic fever	5 – 15 years
Mechanism of acute rheumatic fever	Cross infectivity endogenous antigen
Mechanism of autoimmunity in rheumatic fever	*Molecular mimicry*
MC Site of Rheumatic Fever Vegetations	Mitral > Mitral + Aortic
Valve least affected in rheumatic fever	Pulmonary
Rheumatic fever	Joint pain, ST segment elevation, increased PR interval, cardiomegaly
Rheumatic fever in children	Polyarthritis, mitral valve commonly affected
Characteristic manifestation of cardiac involvement in Rheumatic fever	Pancarditis
Marker for carditis in rheumatic fever	*Subcutaneous nodules*
MC cause of enlarged cardiac shadow in X ray of child	Rheumatic carditis
NOT a definite sign of carditis	Prolonged fever
Carey coomb murmur	Low pitched murmur, Seen in rheumatic fever
Low pitched delayed Diastolic Murmur in Rheumatic Fever	Carey Coomb murmur
Carey Coomb murmur of Rheumatic carditis	Apical Mid diastolic murmur
Rheumatic fever	Chorea aggravated during pregnancy
Syndemham's chorea	*Triad of emotional liability, uncoordinated movements, muscle weakness (hypotonia).*
Tongue in syndenham's chorea	*Bag of worms*
Configuration of hands	*Extended, dish configuration, milkmaid grip*

NOT true about Rheumatic chorea in children	Within 8 -12 weeks of disease
Manifestation of Rheumatic fever disappears completely	Arthritis
Subcutaneous nodule in rheumatic fever	Tender
Rheumatic nodules	Rarely occur unless active carditis is present
Subcutaneous nodule in rheumatic fever	Non tender, usually located on extensor aspect
Erythema marginatum in acute rheumatic fever	Usually associated with carditis
Smoke rings beneath skin	*Erythema marginatum*
PANDAS	*Pediatric autoimmune neuropsychiatric disorder associated with streptococci, tics, OCD, diagnosis made rarely in high incidence RF*
NOT a major manifestation of Acute rheumatic fever	Fever, ESR
NOT included in Jones major criteria	High ESR
NOT true about rheumatic fever	Communicable disease
NOT true about rheumatic fever	Communicable disease
NOT true about epidemiology of RHD	Mitral regurgitation is commonest cardiac lesion

CRITERIA OF RHEUMATIC FEVER

NOT a criteria for acute rheumatic fever	Erythema nodosum
Major criteria	Chorea, arthritis, carditis
NOT a major criteria for Rheumatic fever	Increased CRP
NOT a major criteria for Rheumatic fever	Prolonged PR interval
Minor criteria of rheumatic fever according to modifies Jones criteria	Fever

MORPHOLOGY OF RHEUMATIC FEVER

Rheumatic heart disease	Aschoff nodule(seen in any of the layer), McCallum patch, fibrinous pericarditis
Fine warty vegetations along the line of closure of valves is due to	Rheumatic heart disease
Exudative in Rheumatic fever is	Fibrinous
Vegetations in rheumatic heart disease	Along line of closure
Calcification of heart wall	Endomyocardial fibrosis
Intracardial calcification involves	Rheumatic valves
Fibrinoid necrosis due to Rheumatic fever occurs in	Endocardium
Anitchkow cells are pathogonomic of	Acute rheumatic fever
Most distinctive lesion in rheumatic fever	Aschoff bodies
Aschoff Bodies	Pathogonomic of Rheumatic Fever
Aschoff bodies constitute foci of swollen eosinophilic collagen surrounded by	Lymphocytes and monocytes
Aschoff body in rheumatic heart disease does NOT show	Epitheloid cells
McCallum patch is seen in	Rheumatic fever (Left atrium)
McCallum plaques are seen in	Left atrium

DIAGNOSIS OF RHEUMATIC FEVER

Acute phase reactants in rheumatic fever is elevated for	*12 weeks*
Most common serologic test for RF	*ASO and Anti DNAase B*
Confirmation of Rheumatic fever	ASLO Titre
A child comes with migratory polyarthritis, investigation of choice to confirm diagnosis	ASLO titre
Diagnosis of rheumatic fever is best confirmed by	ASLO
Rheumatic fever is diagnosed by presence of	ECG evidence of prolonged PR interval

MANAGEMENT OF RHEUMATIC FEVER

Drug of choice for syndenham's chorea	*Phenobarbitone*
Drug of choice to suppress lactation in mother with Rheumatic fever	Pyridoxine
Treatment of aspirin resistant rheumatic fever	*Naproxen*
Treatment of penicillin allergy rheumatic fever	*Sulfadiazine*
Drug of choice for rheumatic fever prophylaxis in penicillin allergic patient	Erythromycin
Course of full dose steroids in Rheumatic carditis	3 weeks
Acute rheumatic carditis with fever	Valve replacement will ameliorate CCF
Jai Vigyan mission project for	*RF/RHD in India, involves epidemiological studies, registration and vaccine development*

CARDIOMYOPATHY

FEATURES OF CARDIOMYOPATHY

Keshan Disease	Endemic cardiomyopathy, Deficiency of Selenium
Mineral associated with cardiomyopathy	**Cobalt chloride**
Cardiomyopathy is associated with	Ducchne muscular dystrophy, Friedrich ataxia, Type II glycogen storage disese
Cardiomyopathy is associated with	**Barth syndrome (mutation in tafazzin gene), Glycogen storage disease type II and III, HCV, Chaga's disease**
Trastuzumab	Cardiomyopathy
Doxorubicin Causes Cardiomyopathy	>550 mg/m2
Cardiomyopathy NOT seen in	Alkaptonuria
Cardiomyopathy is NOT a feature of	Lowe syndrome
Tako Tsubo cardiomyopathy	**Global ventricular enlargement, basal constriction, shape of**

	narrow necked jar, also known as apical ballooning syndrome
Tako tsubo cardiomyopathy	*Females are affected, coronary artery is normal, hypokinesia on ECHO, management is with beta blockers*

DILATED CARDIOMYOPATHY

Beta adrenoceptor has been implicated as an autoanigen in pathogenesis of	*Dilated cardiomyopathy*
Mutation in dilated cardiomyopathy	*Tafazzin*
MC cause of cardiac transplantation in pediatric patients	*Dilated cardiomyopathy*
MC type of cardiomyopathy	Dilated cardiomyopathy
Dicrotic pulse	Dilated cardiomyopathy
Contractile dysfunction is a dominant feature of	Dilated cardiomyopathy
NOT true about alcoholic cardiomyopathy	Systemic vasodilatation
Important investigation must be performed before a diagnosis of dilated cardiomyopathy	*Coronary artery angiography*

RESTRICTIVE CARDIOMYOPATHY

Least common cause of cardiomyopathy	**Restrictive cardiomyopathy**
MC cause of restrictive cardiomyopathy	Amyloidosis
Kussmaul sign	Restrictive cardiomyopathy
Restrictive cardiomyopathy differentiated from constrictive pericarditis by	Diastolic pressures are equalized, Thick pericardium

HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY

Mode of inheritance of HOCM	**Autosomal dominant with complete penetrance**
Recurrent chest pain, intensity increased by nitroglycerine	HOCM
Died while playing. autopsy, myocyte hypertrophy	HOCM
Pathology is HOCM	Diastolic dysfunction
Outflow Obstruction, Dilatation of Atria and Asymmetrical septal Hypertrophy	Hypertrophic Obstructive Cardiomyopathy
HOCM	Asymmetrical hypertrophy of septum, dynamic LV outflow obstruction, double apical impulse
Feature of HOCM	*Myocardial hypertrophy without ventricular dilatation*
Disrangement of myofibrils is found in	Hypertrophic cardiomyopathy
HOCM	Double/Triple apical impulse, Diamond shaped murmur
HOCM	Crescendo decrescendo systolic murmur, Brisk carotid upstroke, Increase in murmur during valsalva or

	standing
Harsh systolic murmur of HOCM	Does NOT radiate to neck, DECREASE with hand grip
Valsalva maneuver increases loudness of murmur in	Hypertrophic cardiomyopathy
Systolic ejection murmur in HOCM is disseminated when a patient	Squatting
Murmur of HOCM decreased in	Supine position
Brockenbrough Braunwald sign	**HOCM**
Aggravation of symptoms of angina when nitrates are given	HOCM
Noonan syndrome associated with	Hypertrophic cardiomyopathy
Echocardiography features of HOCM	**Systolic anterior motion of mitral valve (SAM), spade like appearance**
Idiopathic HOCM is associated with	*Ground glass pattern*
HOCM associated with Friedrich's ataxia	*No disarray*
Drug of choice for Hypertrophic Obstructive Cardiomyopathy	β – blocker
NOT true about HOCM	Beta agonists are useful
Digoxin is contraindicated in	Hypertrophic obstructive cardiomyopathy
Contraindication of Digoxin	Hypertrophic cardiomyopathy
Digoxin & Nitrates are CONTRAINDICATED in	HOCM

CONGENITAL HEART DISEASE

FEATURES OF CONGENITAL HEART DISEASE

MC congenital cyanotic heart disease	TOF
Least common cause of heart disease in India	Congenital
MC Mode of Inheritance in Congenital Heart Disease	Multifactorial
MC type of congenital heart disease seen in adults	Bicuspid aortic valve
MC type of bicuspid aortic valve	*Valvular aortic stenosis*
Cause of death in congenital heart disease Birth to 7 hours	Pulmonary, Mitral & Aortic atresia
Cause of death in congenital heart disease	Hypoplastic left and right heart syndrome, Transposition & Malposition of Great arteries
Best to declare the case as interatrial septal defect rather than other cardiac abnormalities	Elevated pressure in right atrium
Congenital heart disease which cause death in first week of life	Hypoplastic left ventricle
7 days child, severe respiratory distress and shock. discharged 2 days back healthy	Hypoplastic left heart syndrome
Surgery for Hypoplastic left heart syndrome	*Nortwood*
Syndrome best associated with congenital heart disease	Holt Oram syndrome
Unapposable fingerized thumb	**Holt Oram syndrome**
Congenital defect existing without any manifestation	Dextrocardia
Absence of conotruncal septum gives rise to	*Patent truncus arteriosus*

Right sided aortic arch is most strongly associated with	Truncus arteriosus
NADA's criteria	Assessment of children for presence of heart disease
Least clinical significance	Incompetent patent foramen ovale
Taussg Bing anomaly	*DORV with TGA*
A taussig bing malformation is best treated by	Diversion of septal defect
Pulmonary plethora is seen in	Truncus arteriosus, TAPVC, VSD
Pulmonary plethora	Diameter of descending pulmonary artery > 16 mm
In a patient with pulmonary plethora due to left to right stunt, large left atrium and normal aorta	VSD
In a patient with pulmonary plethora due to left to right stunt, large left atrium and large aorta	PDA
In a patient with pulmonary plethora due to left to right stunt, small left atrium and normal aorta	ASD
Objective sign of identifying pulmonary plethora in a chest radiograph	Diameter of descending right pulmonary artery > 16 mm
Lung fields are oligemic in	TOF, Tricuspid atresia, Ebstein anomaly, single ventricle with pulmonary stenosis
Ductus arteriosus dependent flow is mandatory for	Hypoplastic left heart, TGA with intact ventricular septum, obliterated aortic arch
Duct dependent systemic circulation	*All left sided obstructive lesions*
Duct dependent pulmonary circulation	*All right sided obstructive lesions*
Duct independent condition	*TAPVC, Truncus arteriosus, ALCAPA*
Congenital heart diseases associated with loud s3	ASD,VSD,PDA
Right axis deviation	ASD, VSD, Pulmonary atresia
Right axis deviation is associated with	*TOF, TGA, TAPVC*
Left axis deviation is associated with	*TA, PA with intact IVS*
Flask shaped heart	Ebstein anomaly, Tetrology of fallot, Transposition of great vessels
Newborn cyanosis, oligemic lung field normal sized heart	Pulmonary atresia
Cyanosis is seen in	Tricuspid atresia, Eisenmenger complex, TOF
Brain abscess in cyanotic heart disease	Parietal lobe
Cyanosis, blood from peripheral vein observed to be chocolate brown colour	Methemoglobinemia
Uhl's anomaly	*Aplasia of right ventricular muscle*
Apert syndrome is associated with	*VSD*
VATER syndrome is associated with	*VSD*
Crouzon syndrome is associated with	*PDA, Coarctation of aorta*
Incontinenta pigmenti is associated with	*PDA*
Cockayne syndrome is associated with	*Accelerated atherosclerosis*
CHARGE syndrome is associated with	*TOF*
CHARGE syndrome	*Choanal atresia, coloboma of eye, ear anomalies*
Pallid spell	*Child becomes pale*

TETRALOGY OF FALLOT

NOT true about trilogy of fallot	*VSD*

Unequal division of conus cordis resulting from anterior displacement of conotruncal septum gives rise to	Tetrology of fallot
Fallot's tetrology present with	Central cyanosis and clubbing
TOF presents with	Central cyanosis with clubbing
Clubbing	TOF
Boot shaped heart	Tetralogy of Fallot
Pulmonary Oligemia	Fallot's tetrology
Couer en sabot	Tetrology of fallot
S1 in TOF	Normal
TOF	Ejection systolic murmur in second intercostal space, single second heart sound, normal JVP
Systolic murmur in TOF is due to	*Pulmonic stenosis*
TOF	Squatting relieves pain, Cyanosis, O2, morphine useful, LVH
Tetrology of Fallot	MC association of Right sided aortic arch
Anoxic spells in TOF is precipitated by	Fever, Exertion, Crying of feeding
Pink fallot	*TOF with mild pulmonary outflow obstruction*
Rare complication of TOF	Congestive cardiac failure
Recurrent respiratory tract infection does NOT occur in	Tetrology of fallot
NOT an essential criteria for TOF	Valvular stenosis
NOT a feature of Tetralogy of fallot	Atrial septal defect
NOT true regarding Tetralogy of fallot	Predominantly left to right shunt
Condition NOT associated with cardiac arrest	Tetrology of Fallot
NOT a potentially treatable cause of cardiac arrest	Tetrology of fallot
Reversal of shunt is NOT associated with	TOF
Radiological features of TOF	Prominent cardiac apex, Prominent Pulmonary bay, Normal right atrial shadow
Treatment of cyanotic spell	*Sudden abdominal aortic compression by applying clenched fist per abdomen*
Drug AVOIDED in Tetrology of Fallot with cyanotic spells	Isoprenaline
Drug NOT used in treatment of cyanotic spell	*Calcium chloride*
Oxygen therapy does NOT correct cyanosis in	Fallot's tetralogy
Blalock Taussing shunt between	Subclavian and pulmonary artery
Potts shunt	Descending aorta to left pulmonary artery
Waterson shunt	*Ascending aorta to pulmonary artery*

ATRIAL SEPTAL DEFECT

MC Heart disease in Pregnancy in Developed countries	ASD
Congenital heart disease asymptomatic till adult life	ASD
Non syndromic ASD is due to mutation in	*NKX 2.5*
ASD is seen with	Ellis van creveld syndrome, Down's syndrome, Holt Oram syndrome
Component of pentology of fallot	Atrial septal defect
MC cause of ASD	Ostium Secondum
MC type of ASD	Septum Secundum

Secundum ASD is associated with	*Holt Oram syndrome*
Primum ASD is associated with	*Goose neck defect of AV valves*
Holt Oram syndrome is associated with	*Thumb hypoplasia*
ASD	Left parasternal heave is due to increased pulmonary artery flow
Presence of pansystolic murmur of mitral regurgitation in a patient with ASD	Ostium primum with floppy mitral valve
ASD with murmur similar to MR and LAD on ECG is having	Ostium primum defect
Aorta in ASD is	Small
Bacterial endocarditis is rarely seen in	Ostium secundum ASD
Infective Endocarditis NOT seen in	ASD
Left atrium NOT enlarged in	ASD
Heart lesion NOT found in Congenital rubella infection	ASD
NOT true about ASD	Left atrial hypertrophy
Does NOT produce cyanosis in first year of life	ASD
Great Hilar Dance (Pulmonary Plethora) On Fluoroscopy	ASD
Characteristic X ray finding in ASD	Pulmonary plethora
Contraindication for ASD operation	Severe pulmonary arterial hypertension

VENTRICULAR SEPTAL DEFECT

MC Type of Congenital Heart Disease	VSD
MC Congenital Lesion complicated by Infective Endocarditis	VSD
Cardiac anomaly most commonly seen in Down's syndrome	VSD
A patient with VSD develops pulmonary hypertension, characteristic feature	Cyanosis
A 29 day old child presents with features of congestive cardiac failure and left ventricular hypertrophy. auscultation shows short systolic murmur	VSD
A child with perimembranous VSD has congestive heart failure. cause of improvement of cardiac failure in this patient	Vascular changes in pulmonary circulation
Type of VSD associated with aortic regurgitation	*Supracristal type*
Management of supracristal type	*Surgery irrespective of size*
Cornelia de lange	*VSD*
Katz Watchal phenomenon	*Equiphasic QRS complex on non restrictive VSD*
Maladie de Roger Defect	Small VSD
CCF in perimembranous VSD is due to	Changes is Pulmonary vasculature
A child with VSD presents with development of cyanosis because of Eisemenger syndrome. correct sequence	Left to right shunt, pulmonary hypertension, right ventricular hypertrophy, right to left shunt
Child with perimembraneous VSD with CCF gets better due to	Spontaneous closure
Natural course of events in untreated ventricular septal defects	Spontaneous closure, Sub acute bacterial endocarditis, A normal life without symptoms

X ray picture of VSD	Dilated left atrium, Dilated pulmonary vein, Dilated pulmonary arteries

PATENT DUCTUS ARTERIOSUS

Ductus arteriosus	May cause machinery murmur by its patency
Ligamentum arteriosus is a remnant of	Ductus arteriosus
Anatomical closure of ductus arteriosus	10^{th} day
Complete Obliteration of Ductus arteriosus	6-12 days
Heart disease commonly associated with rubella infection	PDA
NOT a cyanotic heart disease	PDA
PDA is more common in	Females
Child with PDA	Bounding pulses, pulmonary hemorrhage, necrotizing enterocolitis
Differential Cyanosis	PDA
Ductus arteriosus	May cause machinery murmur by its patency
MC cause of death in adult with PDA	CCF and infective endocarditis
Most important complication of PDA in child	Cardiac failure
Least likely finding in PDA	*CO2 washout*
Features of PDA	Tachycardia
Most appropriate management for maintaining patency of ductus arteriosus in a neonate	Prostaglandin E1
Drug for Maintenance of Patency of PDA	Alprostadil, Misoprostol
Closure of patent ducuts arteriosus stimulated by	Prostaglandin inhibitors
Management of PDA in term child	*Indomethacin is not useful, surgery is indicated*
Gianturo coil	*PDA*
PDA needs surgery to prevent development of	Bacterial endocarditis
Contraindication of surgery in PDA	Reversal of shunt

TRICUSPID ATRESIA

Congenital cyanotic heart disease with pulmonary oligemia	Tricuspid atresia
A patient presents with LVH and pulmonary complications, left axis deviation. most likely diagnosis	Tricuspid atresia
Central Cyanosis, Left Ventricular Hypertrophy, Left axis deviation	Tricuspid atresia
LVH, pulmonary complication.ECG shows left axis deviation	Tricuspid atresia
Central cyanosis with enlarged left ventricle	Tricuspid atresia
Ebstein anomaly associated with	Tricuspid atresia
Ebstein anomaly is associated with	*Pleural effusion, pericardial effusion, ascites*
Himalayan P wave is associated with	*Ebstein anomaly*
Globular cardiomegaly with oligemic lung	*Ebstein anomaly*

fields	
Tricuspid atresia	Left axis deviation, Right ventricular hypoplasia, Diminished pulmonary vascularity
Left ventricular hypertrophy and central cyanosis	Tricuspid atresia
Great Box Shaped Heart	Tricuspid Atresia
Plethoric lung field NOT seen in	Ebstein anomaly
Intracavitatory echocardiography is a diagnostic aid in	Ebstein anomaly of tricuspid valve
Surgeries for tricuspid atresia	*Fontann surgery, Glenn shunt*

COARCTATION OF AORTA

Rib notching is produced by	Coarctation of aorta, Neurofibromatosis, SVC obstruction
Coarctation of aorta	Systolic murmur across anterior chest and back and a high pitched audible diastolic murmur in left sternal border, Persistent hypertension despite complete surgical repair
MC Site of Coarctation of Aorta	Distal to Origin of Left Subclavian artery
MC condition associated with Coarctation of Aorta	Bicuspid Aortic Valve
Coarctation of aorta associated with	Bicuspid aortic valve, Turner syndrome, PDA
Shone Complex	Coarctation of aorta, Left Sided Heart Lesions
Coarctation of aorta common in	Turner's syndrome
15 year old girl Short stature, Webbed neck, Sexual infantilism, Coarctation of Aorta	Turner's Syndrome
Ribnotching of 4-9 ribs with double bulging	Coarctation of aorta
Child presenting with headache, dizziness, intermittent claudication, occasional dyspnea	Coarctation of aorta
1 month old boy, failure to thrive, feature of congestive cardiac failure, femoral pulses are feeble compared to brachial pulses	Coarctation of aorta
10 year boy, seizures, BP in upper extremity 200/140 mm Hg, femoral pulses NOT palpable	Coarctation of aorta
A child with 4 weeks of birth acyanotic, ejection systolic murmur is detected. causes are	Coarctation of aorta
Ejection Systolic murmur, Acyanotic child	Coarctation
Intermittent claudication, dizziness, headache, likely lesion	Coarctation of aorta
Femoral pulse weak compared to radial and carotid pulse	Coarctation of aorta
Tortuous bronchial arteries	Coarctation of Aorta
MC extracardiac abnormality associated with coarctation of aorta	Notching of 1st and 2nd vertebra
Dock's sign (Inferior Rib Notching), '3' Sign, 'E' Sign	Coarctation of Aorta
Reverse 3 sign	Coarctation of aorta
Collateral in post ductal coarctation formed from	Suprascapular artery, Subscapular artery/internal thoracic, Axillary artery
In post ductal coarctation of aorta blood supply to lower limb NOT maintained through	Vertebral artery, superior epigastric artery
Collateral in post ductal coarctation NOT formed from	Vertebral artery
Cause of death in coarctation of aorta	Infective endocarditis, CCF, Intracranial hemorrhage

NOT a cause of death on coarctation	Anterior MI
NOT a characteristic of Infantile coarctation	Diastolic murmur is audible
Coarctation of aorta NOT associated with	Renal artery stenosis, pulmonary stenosis
NOT true about coarctation of aorta	Inability to augment cardiac output with exercise
Coarctation of aorta best diagnosed by	MRI
Valvuloplasty NOT done in	Coarctation of aorta

TOTAL ANOMALOUS PULMONARY VENOUS CONNECTION

Total anomalous pulmonary connection	Total pulmonary venous flow reaches right atrium, oxygen saturation of blood in the pulmonary artery is higher than that in aorta, infracardiac type is always obstructive
TAPVC	All pulmonary veins enter by single trunk, Need not always be associated with septal defects, Cyanotic heart disease
MC Type of TAPVC	Supracardiac
Snowman Sign, Figure of 8 sign, Cottage Leaf Sign, Double Contour	TAPVC (Supracardiac)
Groundglass Appearance, Edematous Septal line A>B	TAPVC (Infradiaphragmatic/Obstructive)

TRANSPOSITION OF GREAT VESSELS

Infant of diabetic mother has	Transposition of great vessels
Transposition of great arteries is common in	**Males**
d-TGA	Aortic valve is to the right of pulmonary artery, Right ventricular pressure is elevated, A balloon arterial septostomy should be performed to improve systemic oxygenation
ECHO of a cyanotic 2 day old infant suggests right ventricular enlargement	Transposition of Great vessels
5 day old, full term male, severely cyanotic at birth, prostaglandin E was initially administered and lateral ballooned atrial septostomy done, improvement in oxidation	Transposition of great vessels
7 day old baby presented in the emergency department with unconsciousness, blue in appearance with 85% in oxygen saturation	TGA
Neonate central cyanosis, short systolic murmur 2nd day of birth	Transposition of great vessels
Long Smooth curve to left border	Physiologically Corrected TGA (L loop Transposition)
Egg on Side/Egg on String appearance	Uncorrected TGA (d- TGA)
Surgery is always indicated in	Transposition of great arteries
Rastelli procedure for	**TGA**
Mustards Procedure	TGA
Definite treatment of TGA	Arterial switch
Surgeries for TGA	*Jatenus, Mustard, Senning, Rashkind*

EISENMENGER SYNDROME

Eisenmenger syndrome	Pulmonary veins NOT distended, Pruning of peripheral pulmonary arteries, Dilatation of central pulmonary arteries
Eisenmenger complex is complex in adult in	VSD, ASD, PDA
Signs of impending Eisenmenger syndrome	Single S2, Increased intensity of P2, Graham Steel murmur
Worst prognosis in Pregnant woman	Eisenmenger syndrome
Heart disease having highest mortality in pregnancy	Eisenmenger syndrome
Maternal mortality is highest in	Eisenmenger syndrome
Tardive cyanosis	*Eisenmenger syndrome*
Heath Edwards classification for	*Severity of Eisenmenger syndrome*
NOT a feature of Eisenmenger syndrome	*LVH*
NOT a feature of Eisenmenger syndrome	Return of left ventricle and right ventricle to normal size
NOT true about eisenmenger syndrome	RV and LV valve come back to normal size
NOT an indication of caesarean section in pregnancy	Eisenmenger syndrome

HEART FAILURE

FEATURES OF HEART FAILURE

Cardiotoxic drugs	Adriamycin, 5-FU, cyclophosphamide
Features of cardiac failure on patient with doxorubicin	Anthracycline induced cardiac risk
Anthracyclines cause	**Vacuolar degeneration, myocyte loss**
MC cause of Heart Failure in Infants	Myocarditis
MC cause of Right Heart Failure	Left Heart Failure
Pure right heart failure is due to	Cor pulmonale
MC cause of heart failure in infancy	Congenital heart disease
MC cause of congestive cardiac failure in infancy	Congenital heart disease
Chief danger in children with paroxysmal atrial tachycardia	CCF
Earliest cause of CHF in 1st week of life	Pulmonary atresia
NOT a precipitating cause of heart failure	*Polycythemia*
Congestive heart failure triad	Tachycardia, tachypnea, tender hepatomegaly
Heart failure cells are seen in	Lung
Heart failure cells are seen in	Chronic venous congestion of lung
Heart failure cells contain	Hemosiderin
Nutmeg pattern of Liver	Right sided heart failure
CCF is associated with increase in	Right atrial mean pressure, serum urea, serum norepinephrine
CCF associated with increase in	Urea, right atrial mean pressure, nor epinephrine
Pure right sided heart failure is seen in	Cor pulmonale
Pure left sided failure	Aortic stenosis, Patent ductus arteriosus
MC cause of LVH	Hypertension
Orthopnea in heart failure due to	Reservoir function of leg veins

Peribronchial edema in the setting of CCF	Cardiac asthma
Congestive cardiac failure in infants is diagnosed by	Liver enlargement
Congestive heart failure in children is best assessed by	Tachycardia, Tender hepatomegaly
Uncommon finding in CCF in newborn	Pedal edema
Grade I edema	Pitting up to ankle
Idiopathic edema of women	NOT related to menstrual cycle, increased water retention in upright position, ACE inhibitors useful in some cases
Uncommon finding in congestive cardiac failure in newborn	Pedal edema
Patients having acute cardiac failure do not show edema because	Fall in systemic capillary hydrostatic pressure
NOT a characteristic feature of Right sided heart failure	Pulmonary edema
NOT a feature of right sided heart failure	Increased PCWP
CCF NOT associated with increase in	Sodium
NOT a characteristic of right sided failure	Pulmonary edema
NOT a Framingham major criteria for diagnosis of heart failure	Hepatomegaly
NADA criteria	Assessment of child for presence of heart disease
Pulmonary edema in CHF is due to	Decreased plasma oncotic pressure
Congestive cardiac failure	Kerley B lines, Pleural effusion, Cardiomegaly
Kerley B lines are seen if pulmonary pressure	25 mm Hg
Diagnosis of CCF in Infants	Hepatomegaly
Paroxysmal atrial tachycardia may be terminated with	Vasopressors, Valsalva manoeuve, Digitalis, Eye ball pressure

DIAGNOSIS OF HEART FAILURE

Chest X ray finding in CCF	Cardiomegaly, Thick interlobar septum
Feature of CCF	*Serum B type BNP is elevated*
NOT a radiological feature of left ventricular heart failure	Oligemic lung field

MANAGEMENT OF HEART FAILURE

Drugs used in CHF	Nesiritide, Digoxin, Spironolactone, Losartan
Digoxin used in CHF due to	Atrial fibrillation and high ventricular rate
Most important pharmacological action of digoxin in congestive cardiac failure	Increase in ventricular contractile force
Drug of choice for congestive heart failure with hypertension	ACE Inhibitors
Inotropic drug	Dopamine, amrinone, isoprenaline
Best inotrope agent to use in right heart failure secondary to pulmonary hypertension	Milrinone
Best ionotrope for use in right heart failure	Milrinone
Drug that can be administered in LVF	Morphine
Calcium channel sensitizer approved for use of CCF	Levosimenden
In heart failure, which of these drugs sensitizes	Levosimendan

tropomyosin towards calcium	
BNP is degraded by	Neutral endopeptidase
Aminopeptidase inhibitors	Bestatin, Apstatin
Niseritide	Brain natriuretic peptide analogue
Niseritide	Used in acutely decompensated heart failure, short half life
Niseritide	BNP analogue, Used in decompensated CHF, IV, Causes loss of Na+ in urine
NOT true about niseritide	*Given orally*
ANP analogue	*Caperitide, uralitide*
In Alternative Medicine, used for Heart Failure	Terminalia Arjuna
Drug NOT prolonging survival in chronic congestive cardiac failure	Digoxin
Drug avoided in elderly	Digoxin in CCF
Drugs NOT used in CHF	Nitroglycerine
NOT used in CHF	Clopidogrel
Beta blockers NOT indicated in	Acute CHF
Beta blocker in heart failure	Absolutely contraindicated in acute decompensated heart failure, Initiated at very low dose, Slow upward titration of dose is required, Carvedilol most widely used in this condition
NOT true about beta blocker therapy in congestive heart failure	It should be started in optimum doses
NOT true about use of beta blockers in heart failure	Most effective in new onset decompensated heart failure
NOT used for treatment of congestive heart failure	Trimetazidine
Calcium channel blocker NOT used in	Congestive heart failure
NOT used in congestive cardiac failure	Trimetazidine
Medication NOT used in management of CCF in congenital heart disease	Soda bicarbonate
Ivabradine is used to	*Reduce heart rate*
Istaroxime	*Na+ K+ ATPase inhibitor*
Management of heart failure	**Biventricular pacing (also known as cardiac resynchronization therapy)**

CARDIAC GLYCOSIDE

DIGOXIN

Digoxin useful in	Complete heart block with CHF
Digoxin NOT indicated in	High output failure
Children does NOT tolerate better	Digoxin
Digoxin is contraindicated in	HOCM
Contraindication for digitalis	Acute rheumatic carditis, Thyrotoxicosis, WPW syndrome
Oubain acts by inhibiting	Na+K+ ATPase
Positive inotropic effect of digitalis is due to inhibition	Increased intracellular Na+ causing increased efflux of

of Na+/K+ ATPase pump in cardiac muscle cell membrane leading to	Na+ and increased influx of Ca++ through Na+/Ca+ exchanger in sarcolemma
Digitalis has positive inotropic effect by virtue of its effect on	Na+ K+ ATP ase pump
Decreases AV conduction	Digoxin
Biochemical mechanism of digitalis is associated with	Decrease in calcium uptake by sarcoplasmic reticulum
Mechanism of action of digoxin is associated with	*Increase in systolic intracellular calcium levels*
Digoxin	Oral dose more than parenteral dose, Onset of action 6 hours, T half 48 hours, Preterm child needs more than term
Digoxin	70-80% protein bound
Digitalis	95% protein bound
Drug deposited in muscle	Digoxin
Digitalis	Excretion is mainly renal, oral absorption is good, lipid soluble
Digoxin is eliminated from body by	Glomerular filtration
Digoxin can accumulate in toxic levels in patients with	Renal failure
Digoxin action NOT affected in	Hepatic disease
Compared to oral digitalizing dose, parenteral digitalizing dose should be	2/3
Dose of digoxin in child as mg/kg	0.04 – 0.06
Time taken for digitalization	5 days
Dose of IV digoxin	0.25 mg
Dose of digoxin orally In adult	0.05 – 0.1 mg/kg
Therapeutic level of digoxin in	0.8 to 1.5 ng/ml
Toxic level of digitalis	>2.4 ng/ml
Significant drug interaction with digoxin	Cholestyramine, Thiazide diuretic, Quinidine
Dose of digoxin reduced when given with	Quinidine, verapamil, calcium

DIGITOXIN

Does NOT contribute to digitoxin toxicity	Hyponatremia

FEATURES OF DIGITALIS TOXICITY

Thiazide induced hypokalemia	Increases digitoxicity
Digoxin toxicity enhanced by	Hypokalemia, Quinidine, Hypomagnesemia, hypothyroidism
Digoxin toxicity precipitated by	Hypokalemia, hypomagnesemia, hypothyroidism
Digoxin toxicity aggravated by	Hypokalemia, Hypercalcemia
Digoxin toxicity NOT enhanced by	Hyperkalemia
ECG findings of digitalis toxicity	Bigeminy, Junctional tachycardia, atrial tachycardia with variable block
QT interval shortened in	Digitalis toxicity
Most characteristic arrhythmia with digitoxicity	PAT with block
Features of digitoxicity	PAT with block, xanthopsia
AV block with atrial tachycardia is seen in	Digitalis toxicity with K+ depletion

Digoxin	Paroxysmal AV tachycardia with AV block
Digoxin induced arrhythmia	Paroxysmal atrial tachycardia with variable AV block, ventricular bigeminy, may be used to treat AF
Rhythm disturbance characteristic of digitalis	Paroxysmal atrial tachycardia
Drug producing emesis by acting centrally and peripherally	Digitalis
Digitalis induced arrhythmia is due to	Increased trigger activity
Digitalis Toxicity causes	Hyperkalemia
Digoxin produces	SA block, AV block, hyperkalemia
Mitral regurgitation and atrial fibrillation presents with syncope. on examination person has a heart rate of 55.most probable cause	Digitalis toxicity
Chronic atrial fibrillation, regular heart rate	Digitalis toxicity
Appearance of VT with the use of quinidine in treatment of atrial fibrillation is usually prevented by prior administration of	Digitalis
60 year old man with rheumatic mitral stenosis with atrial fibrillation is on therapy for fast ventricular rate. on treatment he developed regular pulse	Digoxin
NOT seen in digitalis toxicity	Paroxysmal atrial tachycardia with fast ventricular rate

MANAGEMENT OF DIGITALIS TOXICITY

Digibind	Treat digoxin toxicity
Best treatment of digitalis toxicity	Fab fragments of digitalis antibodies
NOT a treatment of digitalis toxicity	Hemodialysis
NOT indicated in digitalis toxicity	Dialysis
NOT used in treatment of digitalis toxicity	Hemodialysis
NOT a treatment of digitalis induced arrhythmia	Calcium gluconate
NOT given in Digitalis toxicity	Quinidine

VALVULAR HEART DISEASE

GENERAL FEATURES OF VALVULAR DISEASES

Typical movement of mitral valve calcification	*Side to side*
Lambl excrescences are seen in	*Aortic valve*
Fluoroscopy is used in diagnosis	Left ventricular function, Valve calcification, Diaphragmatic palsy
Contraindication for mitral valvuloplasty	*Heavy calcification of mitral valve*

MITRAL STENOSIS

Area of mitral orifice in adults	4-6 cm2

Lutembacher syndrome	Ostium secundum with Mitral stenosis
Parachute mitral valve	*Congenital mitral stenosis*
Mimic physiological sign of Mitral stenosis	Left atrial myxoma
MC heart disease associated with pregnancy	Mitral stenosis
First symptom of mitral stenosis	Dyspnea
Hemoptysis in Mitral stenosis	High pulmonary artery pressure
Mitral stenosis is associated with	Right ventricular hypertrophy
Typical movement of mitral calcification	Upwards and downwards
Mitral stenosis cause Congestive heart failure in pregnancy during	30^{th} week
Mid diastolic murmur with presystolic accentuation	Mitral stenosis
Fishmouth valve is seen in	**Mitral stenosis**
Second stenosis in mitral stenosis	**Pulmonary arterial constriction**
Left ventricular end diastolic is NOT common elevated in	Early mitral stenosis
NOT a feature of Mitral stenosis	Obliteration of retrosternal shadow on lateral X ray
Severity of mitral stenosis is best assessed by	LA enlargement, loudness of S1, Loudness of opening snap
Severity of mitral stenosis assessed by	Length of murmur
Severity of mitral stenosis	S2-OS gap, prolonged diastolic murmur
NOT indicate severity of MS	Opening snap delayed from S2
NOT true about severe MS	Delay of opening snap
Double atrium, Lifting of left bronchus, Straightening of left heart border, Posterior displacement of esophagus	Mitral stenosis
Radiological features of mitral stenosis	Double contour of right heart border, Straightening of left heart border, Splaying of carinal angle, Kerley lines Lifting up of left bronchus, Posterior displacement of esophagus on barium swallow, Pulmonary hemosiderosis, Straight left border of heart
X ray finding of mitral stenosis	Lifting up of left bronchus, double atrial shadow, posterior displacement of oesophagus on barium swallow
Inverted moustache sign	Mitral stenosis
Ideal time for surgery for Mitral stenosis	14 weeks
Balloon valvotomy is NOT successful in	Calcified mitral stenosis
Mitral balloon valvuloplasty is NOT indicated in	Calcified Mitral valve
NOT indicated in mitral stenosis in pregnancy	Methergine at delivery of anterior shoulder

MITRAL REGURGITATION

Earliest valvular lesion in case of acute rheumatic fever	Mitral regurgitation
Commonest rheumatic valvular disease	Mitral regurgitation
MC Rheumatic Valvular Disease	Mitral Regurgitation
Cause of Mitral Regurgitation	Myxomatous degeneration
Myxomatous degeneration is associated with	*Mid systolic click*
Common valvular lesion in myocardial infarction	Mitral regurgitation
Nocturnal angina pain, severe diaphoresis	Chronic severe MR
18 year girl, rheumatic carditis, mitral insufficiency	Decreased functional residual capacity

Murmur in mitral regurgitation	Pan systolic murmur
Valsalva increases pansystolic murmur of	Mitral regurgitation
Severity of mitral regurgitation decided by	Presence of diastolic murmur across mitral valve, wide split second heart sound, presence of left ventricular S3 gallop
Severity of mitral regurgitation judged by	Left ventricular S3
Severity of mitral regurgitation is NOT decided by	Intensity of systolic murmur across mitral valve
59 year old woman, severe myxomatous mitral regurgitation is asymptomatic with left ventricular ejection fraction of 45% and an end systolic diameter index of 2.9 cm/m2. most appropriate treatment is	Mitral valve repair or replacement

MITRAL VALVE PROLAPSE

In mitral valve prolapse syndrome, mitral valve HPE shows	Myxomatous degeneration
Mitral valve prolapse	Autosomal dominant, myxomatous degeneration in valve leaflets, asymptomatic, common cardiovascular manifestation of Marfan's syndrome
Mitral valve prolapse	Common in females, benign clinical course, transient cerebral ischemia is a known complication
Young patient, systolic murmur at apex, murmur increases on both handgrip and valsalva maneuver	MVP
Auscultatory findings of Mitral valve prolapse	Mid systolic click, late systolic murmur, non ejection click
Midsystolic click in asymptomatic young female	Mitral valve prolapse
Complications of mitral valve prolapse	*Mitral regurgitation, arrhythmia, sudden death, transient ischemic attack, infective endocarditis*
Asymptomatic young woman, systolic murmur arrhythmia, mid systolic click	Echocardiography

AORTIC STENOSIS

Complication of Hypervitaminosis D	Supravalvular Aortic stenosis
Supravalvular aortic stenosis	William's syndrome
Heyde syndrome	**Aortic stenosis with GI bleeding**
NOT a common manifestation of congenital rubella	Aortic stenosis
Angina pectoris and syncope most likely to be associated with	Aortic stenosis
Angina, syncope, congestive heart failure	Aortic stenosis
Physical sign in patient with severe aortic stenosis	Delayed peak of systolic murmur
Supravalvular aortic stenosis is associated with	Absence of ejection click, Concentric hypertrophy of left ventricle, Aortic regurgitation murmur
Pressure difference of 5 mm Hg between two upper limbs in	Supravalvular aortic stenosis
Pressure volume curve is shifted to left in	Aortic stenosis
Sustained heaving cardiac impulse	Aortic stenosis

Calcification of aortic valve	Aortic stenosis
Gallavardin effect	**Coarse systolic murmur of AS, sound high pitched and pure at apex**
Gallavardin phenomenon	**Seen in aortic stenosis, murmur is misinterpreted as mitral regurgitation**
Angina pectoris is common with	AS
MC cause of death in aortic stenosis in children	Ischemic heart disease with ventricular fibrillation
Paradoxically split second heart sound signifies	Severe AS
Severe aortic stenosis	ST and T wave changes
Exercise testing is absolutely contraindicated in	Aortic stenosis
Vasopressor of Choice in Aortic Stenosis	Phenylephrine
Ross procedure for	**Aortic stenosis**
Surgeries for congenital aortic stenosis	*Ross, Konno*

AORTIC REGURGITATION

Diastolic mitral regurgitation	*Aortic regurgitation*
Characteristically result in aortic valve insufficiency	*Syphilitic heart disease*
Aortic regurgitation seen in	Marfan syndrome, bacterial endocarditis, ankylosing spondylitis
Acute aortic regurgitation NOT seen in	Acute MI
Young basket ball player, ht 188 cm arm span 197 cm, diastolic murmur best heard in 2nd right intercostals space	Aortic regurgitation
Cardiac lesion having highest risk of occurrence of infective endocarditis	Valvular aortic regurgitation
Aortic regurgitation	Collapsing pulse, Duroziez's murmur, mid diastolic murmur, LVH
LVH commonly seen in	Aortic incompetence
Water hammer pulse	Aortic regurgitation
Low pitched delayed Diastolic Murmur in Severe Chronic AR	Austin Flint Murmur
Hill sign	*More than 20 mm Hg difference in popliteal and brachial systolic cuff pressure*
Blood pressure in severe aortic regurgitation	60-75 mm Hg
NOT a murmur in aortic regurgitation	Pansystolic murmur

TRICUSPID REGURGITATION

MC cause of tricuspid regurgitation	Secondary to dilatation of right ventricle
Commonest cardiac manifestation in carcinoid	Tricuspid regurgitation
Essential for diagnosis of tricuspid regurgitation	Systolic murmur in tricuspid area, pulsatile liver
Hepatomegaly and liver pulsation	Tricuspid regurgitation
Enlarged pulsatile liver	Tricuspid regurgitation
Carvallo sign	**Tricuspid regurgitation murmur increases with inspiration**
Positive hepatojugular reflex	Tricuspid regurgitation, PS, right heart failure, right

	ventricular infarction, pre capillary pulmonary hypertension
Positive hepatojugular reflex NOT found in	Decreased afterload

PULMONARY STENOSIS

MC form of isolated right ventricular obstruction	**Valvular pulmonary stenosis**
MC type of pulmonary stenosis	*Valvular*
Pulmonary stenosis is associated with	*Noonan syndrome, Alagille syndrome*
Peripheral pulmonary stenosis is associated with	William syndrome, Rubella
Rapidly progressing pulmonary stenosis	Amiodarone poisoning
Obstruction in pulmonary stenosis	Supravalvular, valvular, subvalvular
Obstruction in pulmonary stenosis may occur at	Supravalvular, Valvular, Subvalvular
Cardiac abnormality well tolerated during fetal life, serious problem at birth	Pulmonary valve stenosis

ENDOCARDITIS

ETIOLOGY OF ENDOCARDITIS

HACEK	Hemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella
HACEK group does NOT include	Acinetobacter barumannii
MC cause of Acute bacterial Endocarditis	Staphylococcus aureus
MC cause of endocarditis in IV drug users	Staphylococcus aureus
MC cause of infective endocarditis in pediatric age group	*Congenital heart disease*
MC cause of tricuspid valve vegetation	*Staphylococcus aureus*
Anti teichoic acid antibodies in endocarditis	*Staphylococcus*
Cause of infective endocarditis associated with IV drug abusers	*Pseudomonas aureginosa, serratia*
MC cause of infective endocarditis associated with indwelling catheter	*Coagulase negative staphylococci*
MC cause of Prosthetic Valve Endocarditis	Staphylococcus epidermidis (Coagulase negative)
Serious prosthetic valve infection caused by	**Staphylococcus lugdunensis, S. schleiferi**
MC cause of sub acute endocarditis	α Hemolytic Streptococci (Viridans)
MC cause of native valve endocarditis	Streptococcus viridians
MC cause of infective endocarditis associated with dental procedures	*Viridians streptococci*
MC cause of infective endocarditis associated with large bowel and genitourinary manipulation	*Group D enterococci*
Cause of infective endocarditis associated	*Fungal*

with open heart surgery	
Infective endocarditis in elderly with colonic polyp or cancer is associated with	*Streptococcus bovis*
Infective endocarditis due to pseudomonas is most commonly seen in	Intravenous abuse of pentazocin
Most common pathogen in culture negative endocarditis	**Coxiella burnetti, bartonella**
Most common cause of endocarditis	VSD
Least likely to cause infective endocarditis	Salmonella typhi
Least common cause of endocarditis	ASD
Acute infective endocarditis with abscess formation	Staphylococci
A patient of RHD developed infective endocarditis after dental extraction. most likely organism	Streptococcus viridians
A 2 year old known case of RHD presents with 3 weeks history of fever, hematuria and palpitation. diagnosis	Staphylococcal endocarditis

SITE OF ENDOCARDITIS

Valve pocket vegetations are seen in	*Libman Sack endocarditis*
Flat small reddish tan vegetations in the cusps of tricuspid and mitral valve	*Libman sack endocarditis*
MC Site of Vegetations in Libman Sack Endocarditis	Mitral & Tricuspid Valve
MC Site of Non Bacterial Thrombotic Endocarditis (Marantic Endocarditis)	Mitral>Aortic>Tricuspid
MC affected valve in Prosthetic valve Endocarditis	Aortic
Heart valve most likely to be involved in infective endocarditis following septic abortion	Tricuspid valve
MC heart valve involved in IV drug user	Tricuspid

FEATURES OF ENDOCARDITIS

Most friable vegetation	Infective endocarditis
Maximum destruction of valves	*Acute infective endocarditis*
Site of lesion of endocarditis of RHD	Along line of closure of valves
Endocarditis is commonest in	MR
Immune complex lesions in SBE	Osler's nodes, Microscopic hematuria, Roth spot
Flat vegetations in pockets of valves are due to	Libman sacks endocarditis
Vegetations on undersurface of AV valves are found in	Libman sack's endocarditis
Libman sack's endocarditis	*Medium sized vegetations on both side of valve leaflets*
Vegetation in Libman Sach's Endocarditis	Sterile vegetation
Libman sack's endocarditis	SLE
Non bacterial thrombotic endocarditis is commonly associated with	*Terminal neoplastic diseases*
Not firmly fixed to valve	*Non bacterial thrombotic endocarditis*
Woman having septic abortion done, vegetation on tricuspid valve is likely to go to	Septic infarcts to lung
Tricuspid valve endocarditis in septic abortion most	Lungs

likely affect	
Osler's nodes are typically seen in	Acute staphylococcal endocarditis
Osler node seen at	Tips of palms and soles
Roth spots seen in	Infective endocarditis
Roth spots are due to	*Retinal hemorrhage*
Roth spots (White central hemorrhage) may arise in	Anemia
NOT included in Duke's minor criteria	Endocardial involvement
NOT a diagnostic criteria for infective endocarditis	Raised ESR, Rheumatic factor
NOT true about infective endocarditis	May lead to acute regurgitant valvular lesion
NOT a feature of infective endocarditis	Thrombus in left atria
NOT a complication of infective endocarditis	Suppurative pericarditis
NOT a complication of infective endocardits	Myocardial infarction
Mitral valve vegetations do NOT embolise to	Lung
Splenomegaly is more common in	*Sub acute endocarditis*

MANAGEMENT OF ENDOCARDITIS

Antibiotic prophylaxis for infective endocarditis in	Coarctation of aorta
Antibiotic prophylaxis for oral cavity procedures in infective endocarditis	Amoxycillin
Initial therapy for all HACEK except Eikenella	**Ceftriaxone**
Therapy for Eikenella	**Ampicillin**

PERICARDIAL DISEASES

GENERAL FEATURES OF PERICARDIUM

Most common non penetrating cardiac injury	**Myocardial contusion**
Hemopericardium	Ruptured aortic aneurysm, myocardial infarction, chest injury
Pericardial friction rub heard frequently at	End of Expiration

CARDIAC TAMPONADE

Engorged neck veins, BP 80/50 pulse rate 100 following blunt trauma to chest	Cardiac tamponade
Postoperative cardiac surgical patient developed sudden hypotension, rapid central venous pressue, pulsus paradoxus in 4th postoperative hour	Cardiac tamponade
Carcinoma lung, respiratory distress, electrical alterans	Cardiac tamponade
Diastolic collapse of right ventricle on echocardiogram	Cardiac tamponade
Beck's triad in Cardiac tamponade	Muffled Heart Sounds/Silent Heart, Distended Neck Veins, Hypotension
Beck's triad seen in	Cardiac tamponade

Electric alterans is seen in	Cardiac tamponade
Pulsus paradoxus is characteristic of	Cardiac tamponade
Kussmaul sign is NOT seen in	Cardiac tamponade
NOT a feature of Cardiac tamponade	Warm periphery
Cardiac tamponade	Low voltage ECG
Cardiac tamponade	ABSENT y descent
Feature of JVP in cardiac tamponade	Prominent x descent with absent y descent
Best investigation for Cardiac tamponade	2D echocardiography
In case of tamponade after trauma	Pericardiocentesis at once may be life saving
Treatment of acute cardiac tamponade	Emergency paracentesis

PERICARDITIS

Drugs associated with pericarditis	Hydralasine, Procainamide, Methysergide, Emetine
Drugs causing pericarditis	Methysergide, Hydralasine, Minoxidil
Emetine, Methysergide, Procainamide	Pericarditis
Drug used in HIV causing pericarditis	Didanosine
Cause of pericarditis	Tuberculosis, Uremia, Rheumatic fever
MC cardiac manifestation of SLE	Pericarditis
Pericarditis is always painful	Pyoperitoneum
Typical feature of pericardial involvement on physical examination	Friction rub
In case of chest pain with pericarditis and pericardial effusion, pain is referred by	Phrenic nerve
Pain in pericarditis increases on	Leaning forward
Pain of acute pericarditis is relieved by	Sitting
NOT seen in pericarditis	Pulsus paradoxus
ECG finding in acute pericarditis	Global ST segment elevation in early pericarditis, sinus tachycardia is common finding, PR segment depression is present in majority of patients
Hemorrhagic pericarditis	Transmural myocardial infarction, Dissecting aneurysm of aorta, Metastatic disease of pericardium
Hemorrhagic pancreatitis does NOT occur in	Constrictive pericarditis
Commonest cause of constrictive pericarditis in India	Tuberculosis
Least likely to cause constrictive pericarditis	Acute rheumatic fever
Chronic constrictive pericarditis	Ascites is NOT in proportion to edema, Right ventricular end diastolic pressure is raised
During ventricular pressure pulses, square root wave sign	Constrictive pericarditis
Constrictive pericarditis	Ascites, Retractile apex, Pericardial knock
NOT a cause of ST elevation	Constrictive pericarditis
ST elevation NOT seen in	Constrictive pericarditis
Kussamaul sign	Constrictive Pericarditis
Pericardial Calcification, thickened pericardium, Square root sign, Egg in cup appearance	Constrictive Pericarditis
Friedrich sign (rapid or exaggerated y descent)	*Constrictive pericarditis*
Broadbent sign	**Reduced apical impulse in chronic constrictive pericarditis**
Definitive treatment of constrictive pericarditis	**Pericardial resection**

Water bottle configuration of heart	Pericardial effusion
Water can appearance of chest X ray	Pericardial effusion
Ewart's sign	Pericarditis with effusion
Ewart's sign	*Cardiac tamponade*
Best investigation for pericardial effusion	ECHO
Investigation used to differentiate Pericardial effusion and Heart dilatation	Echocardiogram
Signs of pericardial effusion are difficult to detect	500 ml

HYPERTENSION

BLOOD PRESSURE

Immediate change after lying down	Increased venous return
Mean circulatory filling pressure	Arterial pressure taken at point when heart stops beating
During diastole, arterial pressure is maintained by	Elastic recoil of aorta
Positive pressure head in aorta during diastole is maintained by	*Elastic property of aorta*
Blood pressure	Peripheral resistance * cardiac output
Blood pressure	peripheral resistance * cardiac output
Blood pressure	Cuff width should be 40% of arm circumference, Diastolic blood pressure is indicated by 5th koratkoff sound, Small cuff measures spuriously elevated diastolic BP, Monkenberg sclerosis causes pseudohypertension
Blood pressure measurement	Cuff width should be 40% of arm circumference, small cuff measures spuriously elevated diastolic blood pressure, monkenberg sclerosis causes pseudohypertension
B.P measurement correct is	80% of circumference of a limb
SI unit of blood pressure	KPa
Investigation of flight induced stress on blood pressure. BP twice measured. Once before takeoff and once after space craft entered orbit. For proper comparison preflight BP should be recorded in	Lying down position
Pressure required to occlude blood flow with a tourniquet that exceeds systolic pressure	25 – 50 mm Hg
Blood pressure measured by sphygmomanometer	Higher than intraarterial pressure
Sphygmomanometer blood pressure is	More than interarterial pressure
Experiment for BP on dog. Rakesh uses sphygmomanometer on right femoral artery. Arif pressure transducer on left femoral artery. Mean arterial pressure both 100 mm hg, 5 mins after adrenaline . Rakesh 130.arif 120-	Falsely low values at high pressure in pulse tracing
Spuriously high BP NOT seen in	Ausculatatory gap
A blood pressure of 120/80 mm Hg is elevated for children aged	4 years
Average BP of one year old child	95/50

Systolic BP in stage 2 hypertension	>160
Hypertension in children	*Greater than or equal to 95th percentile for age, sex and height at 3 occasions*
Least likely cause of secondary hypertension in pediatric age group	*Wilm's tumor*
Most accurate non invasive method for measuring BP	*Oscillometric method*
Drug useful for hypotensive anaesthesia	Trimethaphan
NOT used for induced hypotension during surgery	Mephentermine

CAUSES OF HYPERTENSION

MC cause of Systemic Hypertension in Children	Acute Glomerulonephritis
MC cause of Secondary Hypertension in Children	Renal Disease
Acute onset of hypertension in children	Acute Glomerulonephritis
Cause of Persistent hypertension in Children	Renal Parenchymal Lesion
Monogenic autosomal dominant cause of hypertension	Gordon syndrome, Pregnancy Exacerbated hypertension, glucocorticoid remediable aldosteronism
Hypercarbia characterized by	Hypertension
Hypersecretion of aldosterone causes	Hypertension
Hypertension caused by	Erythropoietin, cylclosporine, NSAID
Renovascular hypertension is aggravated by	ACE inhibitors
NOT a cause of hypertension with hypokalemia	End stage renal disease
Hypertension is NOT caused by high intake of	Potassium

FEATURES OF HYPERTENSION

Rapidly evolving end organ damage is a feature of	Hypertensive emergency
Hypertensive urgency	Can be managed without extensive monitoring on an outpatient basis
Accelerated hypertension associated with	Metabolic alkalosis
Features of essential hypertension	Concentric hypertrophy of LV, increased heart size, increased size of heart muscles, myohypertrophy
Multifactorial disorder	Systemic hypertension
J curve phenomenon is associated with	*Hypertension*
Pseudo resistant hypertension occurs in	*Patients going to office*

RENOVASCULAR HYPERTENSION

MC cause of Renovascular Hypertension in Young in India	Takayasu arteritis
MC cause of Renovascular Hypertension in young in western world	Fibromuscular Dysplasia
NOT a feature of renovascular hypertension	Muscle cramps
Most specific and sensitive investigation in case of renovacular hypertension	MRI
Most sensitive and specific investigation for screening	Spiral CT

of renovascular hypertension	
Renovascular hypertension	Captopril

MORPHOLOGY OF HYPERTENSION

Monkenberg sclerosis	Pseudohypertension
Calcification of tunica media is mostly related to	Monckberg sclerosis
Monckeberg sclerosis involves	Media
Monckeberg calcific sclerosis affects medium sized arteries by involving structure of	Media
Changes in heart in essential hypertension	Cardiac cell hypertrophy
Pathological change in kidney in benign hypertension	Hyaline arteriosclerosis
Hyaline arteriosclerosis is seen in	Benign nephrosclerosis, Hypertension, Diabetes
NOT a feature of benign hypertension in kidney	Fibrinoid necrosis
Characteristic pathologic feature of Malignant hypertension	Fibrinoid Necrosis
Characteristic feature of Kidney in Malignant hypertension	Fibrinoid necrosis
Pathological change in malignant hypertension	Hyperplastic arteriosclerosis
Malignant hypertension is associated with	Malignant nephrosclerosis
Hyperplastic arteriosclerosis is associated with	*SLE*
Onion skin thickening of arteriolar wall is seen in	Hyperplastic arteriosclerosis
Hyperplastic arteriosclerosis in malignant hypertension is NOT seen in	Heart
Does NOT occur in malignant hypertension	Hyaline arteriosclerosis

MANAGEMENT OF HYPERTENSION

Primary prevention of hypertension	Weight reduction, exercise promotion, reduction of salt intake
Management of uncomplicated essential hypertension	Diet modification, exercise drugs
First line management of Hypertension in Diabetes	ACE Inhibitors, ARB
Drug of choice In hypertension due to pheochromocytoma	Phentolamine
Antihypertensives having neutral role in lipid metabolism	Losartan, Prazosin
Antihypertensive devoid of central action	Indapamide
Antihypertensive drug causing hypothyroidism	Amiodarone
Antihypertensive NOT contraindicated in pregnancy	Labetalol
Antihypertensive NOT used in type II diabetes	Thiazides
NOT a preferred agent of hypertension in diabetes	Hydrochlorthiazide
NOT used in severe hypertension on elderly on empirical basis	Prazosin
Combination NOT recommended for treatment of hypertension	ACE inhibitors and Beta blockers
NOT true about hypertension	In hypertensive patients with gout, diuretics are preferred

Antihypertensives to stopped before Surgery	ACE Inhibitors, ARB
Diazoxide cause	Hyperglycemia
True about vasomotor centre	Acts along with cardiovagal centre to maintain blood pressure
Methyldopa, Clonidine acts on	Vasomotor centre
Postural hypotension is common with	Prazosin

VASODILATORS

Vasodilators	NO, CO2, N2O, CO, Helium
Drug causing peripheral vasodilatation by direct action which is useful where other therapy is inadequate or where severe hypertension is present	Minoxidil
Minoxidil is a	Antihypertensive
Only vasodilator that can be inactivated by proteolytic enzymes	*Vasoactive intestinal peptide*
Side effects of directly acting vasodilators	Hypertrichosis, Hypotension

ARTERIOLAR DILATORS

Arteriolar dilators	Hydralazine, Nifedipine, Prazosin, Enalapril
Hydralazine	Direct relaxation of arterioles, postural hypotension is NOT common, increases plasma rennin activity
Hydralazine	Predominant arterial dilator
NOT an predominant arteriolar dilator	Sodium nitroprusside

VENOUS DILATORS

Venodilator	Nitroprusside
Mechanism of action of sodium nitroprusside	Stimulation of guanylate cyclase
Side effects of sodium nitroprusside	Lactic acidosis, Psychosis, Headache

ACE INHIBITORS

Action of angiotensin II	Systemic vasoconstriction and retention of water
Angiotensin II does NOT cause	Vasodilatation
ACE inhibitor and ARB	*Decrease total peripheral resistance*
ACE inhibitors	Omission of prior dose decreases risk of postural hypotension
Antihypertensive of choice in diabetes and microalbuminuria	ACE inhibitors
ACE inhibitors NOT contraindicated in	Diabetes
ACE inhibitors NOT useful in	Pheochromocytoma
Most significant adverse effect of ACE inhibitor	Hypotension
Long term use of ACE inhibitors cause	Reduction in filtration fraction

ACE inhibitors cause	Persistent cough, Taste changes, First dose hypotension, Angioedema
Antihypertensive causing renal failure	ACE inhibitors
ACE inhibitors cause	Hyperkalemia
Cough and angioedema in patients taking ACE inhibitor due to	Bradykinin
Maximum bicarbonate excretion is seen with	ACE inhibitors
NOT an adverse effect of ACE inhibitors	Hypokalemia
Captopril exerts antihypertensive effect by	Inhibits conversion of angiotensin I and II
Drugs causing afterload reduction	Captopril
Side effects of captopril	Cough, Hyperkalemia, Renal dysfunction
Dysguesia is a side effect of	Captopril
NOT used in pregnancy associated hypertension	Captopril
Provides hemodynamic stability and prolong survival in congestive heart failure	Lisinopril
Side effects of Lisinopril	Dizziness, Cough, Angioneurotic edema
ACE inhibitors are contraindicated in	Bilateral renal artery stenosis, Renal failure, Elderly
Enalapril	Prodrug, more effective than captopril, less adverse effect, NOT a dipeptide
Enalapril	Useful in heart failure, longer acting than captopril, prodrug
Young patient 190/120 mm Hg, without any clinical symptom and normal fundus examination	Oral enalapril
ACE inhibitor whose bioavailability NOT affected by food	Enalapril
Antihypertensive agent can be used in gout with diabetes mellitus	Enalapril
Antihypertensive NOT used in pregnancy	Enalapril
Antihypertensive Contraindicated in pregnancy	Enalapril
Enalapril is contraindicated in	Single kidney, bilateral renal artery stenosis, hyperkalemia
Best for reducing proteinuria in diabetic patient	Perindopril

ARB

ARB inhibitors	Continued till the day of operation
Losartan	Competitive angiotensin receptor antagonist, long acting metabolite, associated with negligible cough
NOT true about losartan	Cause hyperuricemia

CALCIUM CHANNEL BLOCKER

Calcium channel blocker used in treatment of hypertension	Verapamil, Nifedipine
When nitrates combined with calcium channel blockers	Arterial pressure will decrease
Antihypertensive inhibiting labour	Nifedipine
Antihypertensive causing gingival hyperplasia	Nifedipine
Racemic mixture of two enantiomers with different	Verapamil

pharmacokinetic and pharmacodynamic property is seen in	
Maximum effect of contractility of heart	Verapamil
Calcium channel blocker with maximum effect of conduction of heart is	Verapamil
ECG change caused by verapamil	*Increases PR interval*
NOT a dihydropyridine	Verapamil
Drug causing constipation	Verapamil
Verapamil is carefully used in presence of	Beta blockers
Propanolol should NOT be given to patient on treatment with	Verapamil
Combination of beta blockers and calcium channel blockers cause	Heart block, Bradycardia
Verapamil contraindicated in	Complete heart block
NOT a calcium channel blocker	Pirenzepine
NOT a ca channel blocker	Dantrolene
Ion does NOT causing Vasodilatation	Ca++
Does NOT significant drug interaction with digoxin	Amlodipine
Calcium channel blocker NOT used in	Sick sinus syndrome
Side effect of calcium channel blocker	**Gingival overgrowth**
Antidote for calcium channel blocker overdose	Calcium gluconate

BETA BLOCKERS

First line drug for management of hypertension in patients with angina	Beta blocker
Anti hypertensive agent associated with maximum incidence of impotence	Beta blockers
Drug NOT used in Hypertensive patient with Glaucoma	Beta blockers
Drug contraindicated in hypertensive cardiac failure	Atenolol
Antihypertensive causing decreased libido and impotence	Atenolol
NOT a frontline antihypertensive agent	Atenolol
NOT used to reduce afterload	Propanolol

CLONIDINE

Clonidine	Alpha adrenergic agonist, Dry mouth as adverse effect, Inhibits sympathetic flow, Prazosin does NOT completely antagonize its action
Clonidine	Increases parasympathomimetic outflow, decreases sympathetic outflow by blocking central alpha receptor, used in hypertension
Clonidine	Alpha 2 selective agonist
Clonidine	Reduction in central sympathetic outflow, combined with vasodilators, sedation and xerostomia are common side effects
Antihypertensive causing sedation	Clonidine
Sudden withdrawal of which drug result in serious	Clonidine

adverse cardiovascular changes in patient taking the drug over long time	
NOT true about clonidine	Increase in LDL cholesterol on prolonged usage
Clonidine withdrawal Hypertension	Phentolamine, Tolazoline

METHYLDOPA

Methyldopa acts by	Altering central sympathetic nervous activity
Methyldopa primarily used in	Pregnancy induced hypertension
Antihypertensive agent decreasing libido	Methyldopa
Warm antibody type of hemolytic anemia associated with	Methyldopa

MANAGEMENT OF HYPERTENSIVE EMERGENCY AND URGENCY

Hypertensive crisis is pheochromocytoma caused by	Propanolol, saralasin, captopril
Rapid reduction of blood pressure indicated in	Acute aortic dissection, hypertensive encephalopathy, intracerebral hemorrhage
Drug of choice in hypertensive emergency	Sodium nitroprusside IV
Drug of choice for malignant hypertension in 6 year old child	Sodium nitroprusside
Sodium nitroprusside	Increased guanylate cyclase
Sodium nitroprusside infusion may result in	Cyanide toxicity
To prevent toxic accumulation of cyanide during rapid infusion of sodium nitroprusside ,best drug to be given	IV sodium thiosulphate
Fenoldopam is used in	Hypertensive emergencies
Drug used in hypertensive crisis	Diazoxide
Hypertensive Emergency in pregnancy	Hydralazine
Treatment of hypertensive crisis in patient with autonomic dysreflexia	Nifedipine, clonidine
NOT used for hypertensive crisis	Indapamide, clonidine, phenoxybenzamine, methyldopa
Inappropriate choice of pharmacological management in hypertensive emergency	Nifedipine
Grade II hypertensive retinopathy with BP 230/110 mm Hg. NOT a treatment	Sublingual nifedipine
NOT an IV therapy for hypertensive emergencies	Nifedipine