CARDIAC SURGERY

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Heart

The heart is bounded by the 2nd left costal cartilage, the 3rd right costal cartilage, the 6th right costal cartilage, and the 5th left costal cartilage.

Vessels

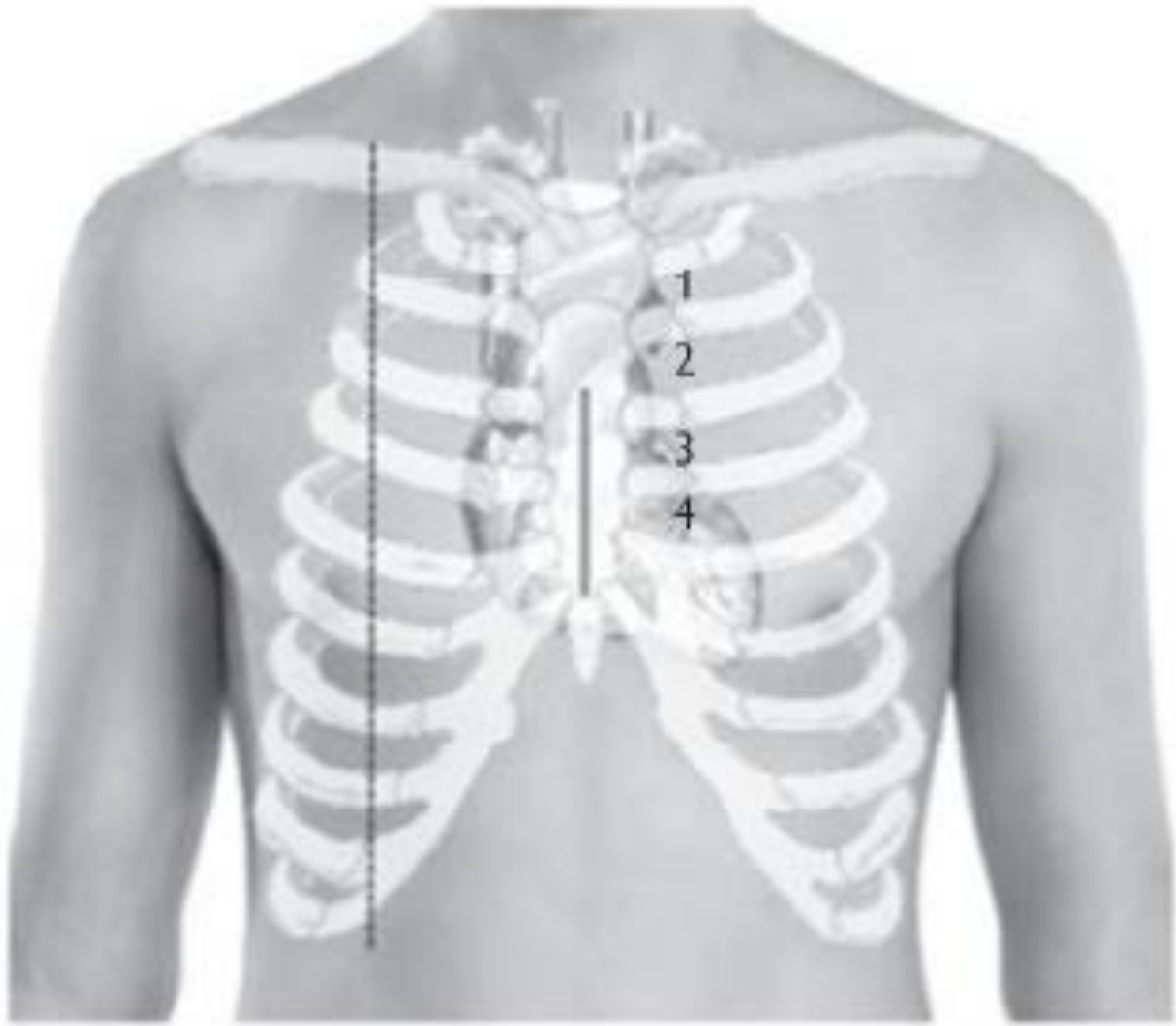
The internal thoracic arteries descend behind the costal cartilages, 1cmlateral to the sternal edge.

The aortic arch arches anteroposteriorly behind the manubrium, the innominate, and left common carotid ascend posterior to the manubrium.

The innominate veins are formed by the confluence of the internal jugular and subclavian veins posterior to the sternoclavicular joints.

The SVC arises from the left and right innominate veins behind the 2nd and 3rd right costal cartilages.

(a)



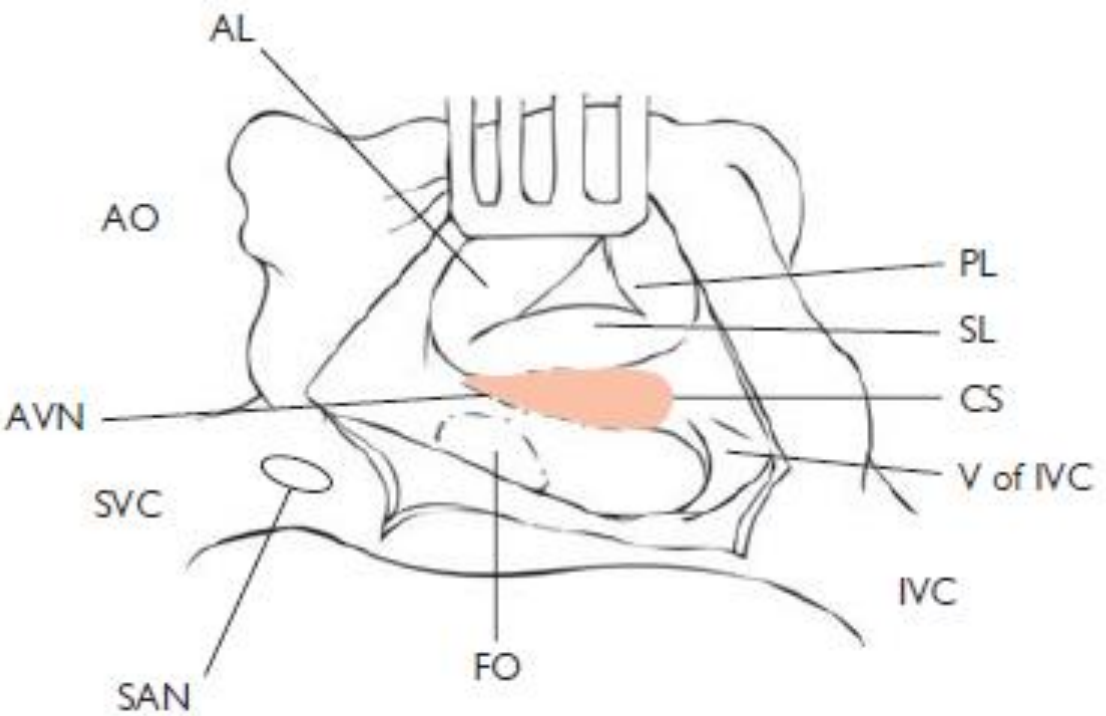


Fig. 2.4 Interior of the right atrium: AO aorta, AL anterior leaflet, PL posterior leaflet and SL septal leaflet of tricuspid valve, AVN atrioventricular node, SVC superior vena cava, FO foramen ovale, IVC inferior vena cava, V of IVC Esutacian valve, CS coronary sinus, T tendon of Todaro, TK triangle of Koch.

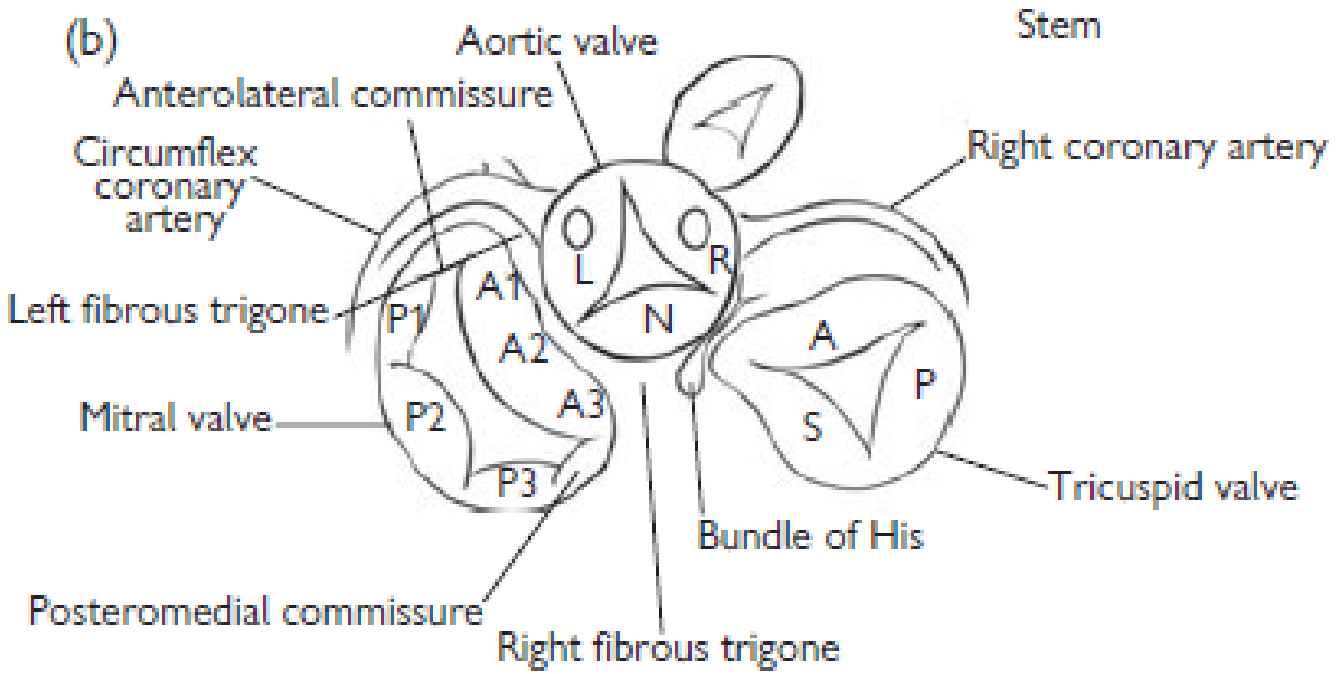


Fig. 2.5 (a) Atrioventricular junction: note the septal atttachment of the tricuspid valve is lower than the septal attachment of the mitral valve. (b) Cardiac valves and their relationships viewed from above, with the atria removed. Note how the commissure of the left (L) and the noncoronary (N) sinuses of the aortic valve points at the midpoint of the anterior leaflet of the mitral valve (A2). The circumflex coronary artery is particularly close to the posterior mitral annulus in the P3, P2 segment. The right coronary artery is close to anterior tricuspid annulus. The bundle of His lies in the membranous septum near the right fibrous trigone located at the junction of the right (R) and the non-coronary cusp (N), extending to the antero-septal commissure of the tricuspid valve.

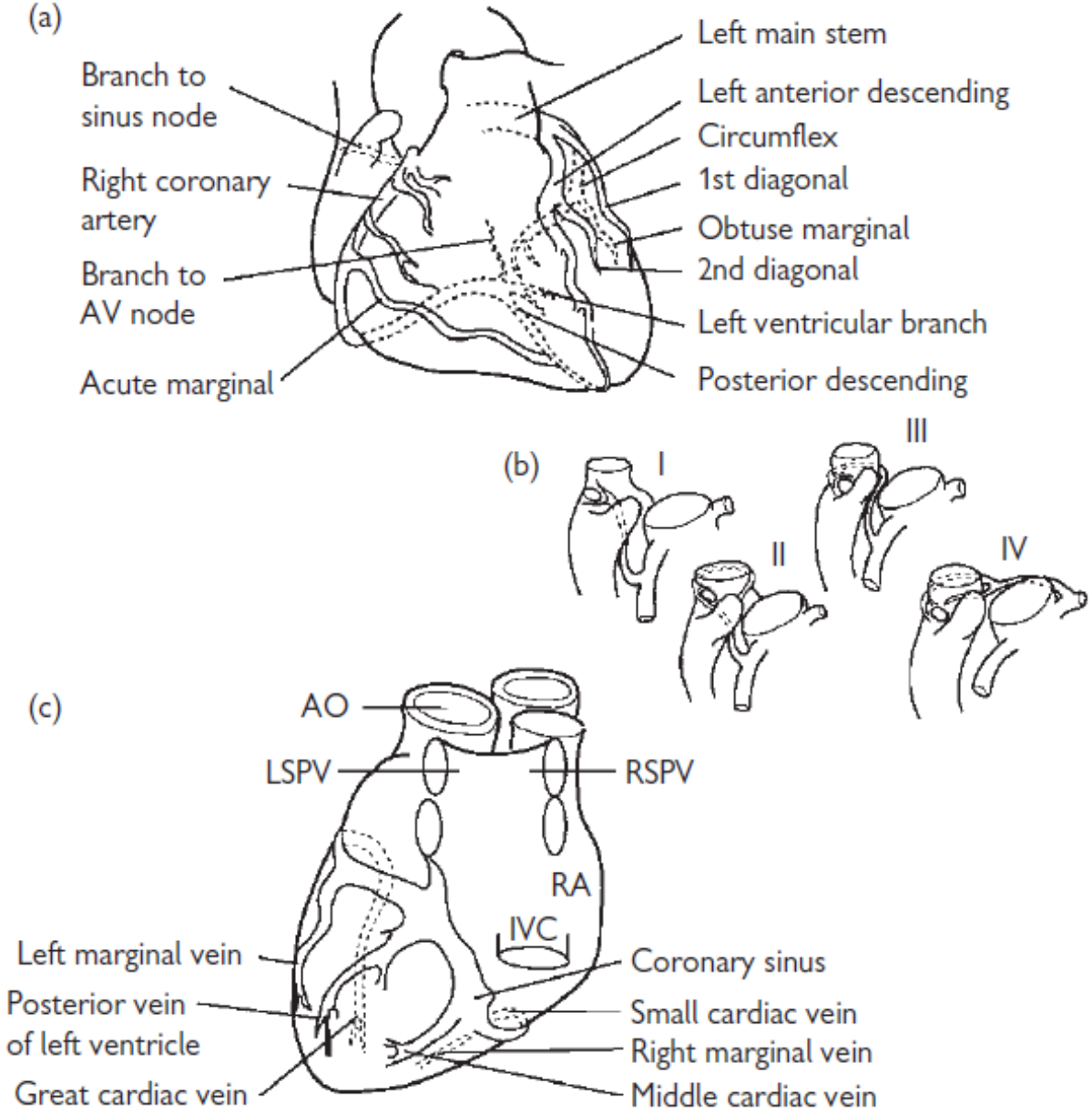


Fig. 2.6 (a) Coronary arteries. (b) Variations in the sinus node artery, which may arise from the right coronary artery (60%) and encircle the base of the superior

CARDIOPULMONARY BYPASS CIRCUIT

Cardiopulmonary bypass (CPB) provides a still, bloodless heart while circulation to the rest of the body is maintained.

Alternatives to standard cardiopulmonary bypass, including circulatory arrest and off-pump surgery,

There are three essential functions of CPB:

- Oxygenation.
- Ventilation.
- Circulation.

The other important function of CPB is temperature control.

THE CPB CIRCUIT

Desaturated blood drains from the RA or vena cava via venous cannulas and the venous line to a reservoir . A pump propels blood from the venous reservoir through a membrane oxygenator , followed by an arterial filter, into the patient's aorta via the arterial line and the aortic cannula .

ADJUNCTS TO THE BASIC CIRCUIT INCLUDE:

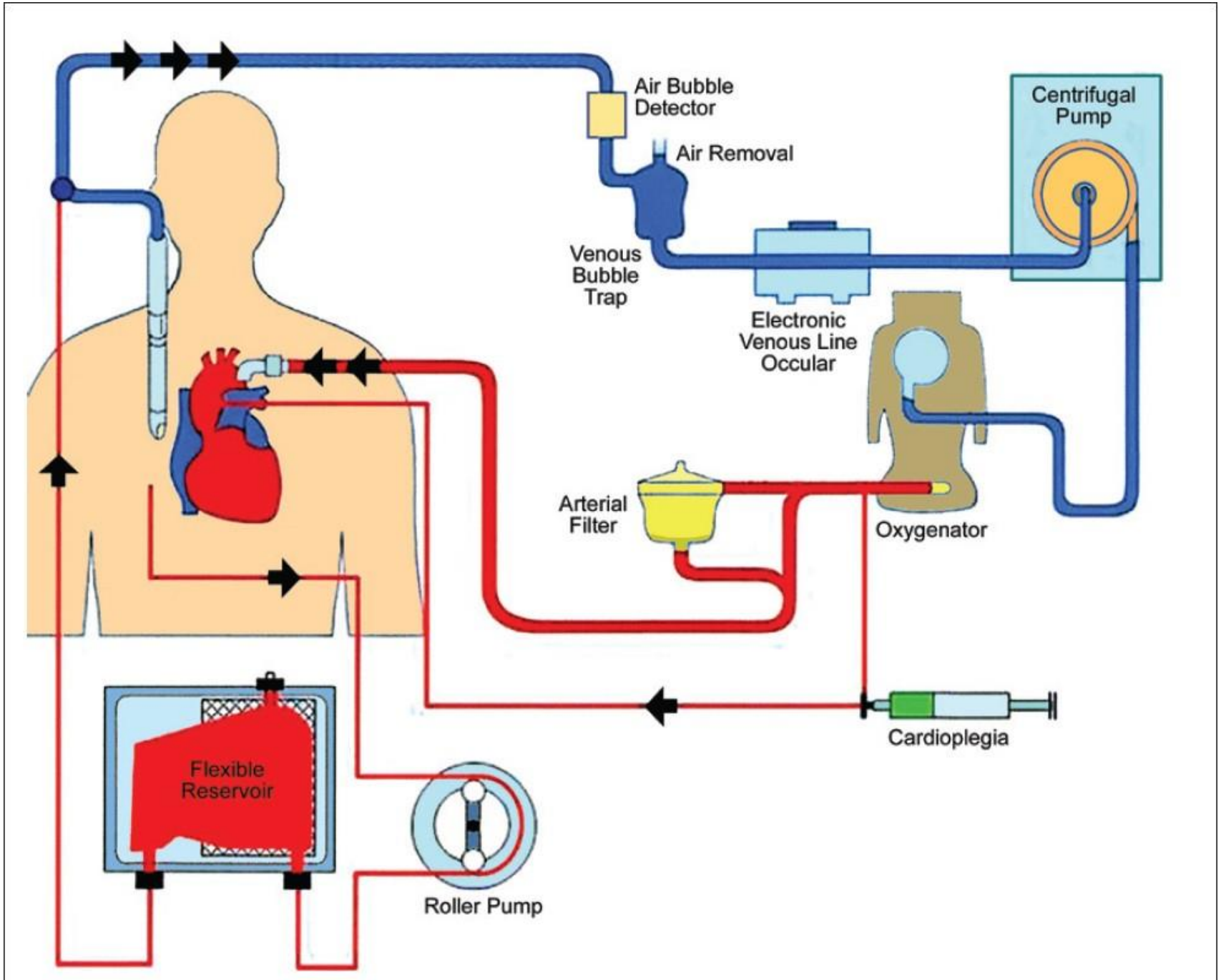
- Venous occlusion (an adjustable clamp on the venous line).
- Ports for drug and fluid infusions to the venous reservoir.
- Cardioplegia delivery system using a separate roller pump.
- Source of oxygen, air, CO2 and anesthetic gases.
- Sampling ports, and in-line blood gas and temperature monitors.
- Bypass line around the arterial filter in case the filter obstructs.
- Low-level reservoir alarm.
- Ultrafiltration.
- Suction tubing leading to cardiotomy reservoir via a pump for removing blood from heart (vents) or the surgical field (pump suckers).

THE STANDARD COMPONENTS OF THE CPB CIRCUIT

- Atrial cannula (right atrial, caval, femoral and/or axillary).
- Venous line (PVC . inch diameter 12mm).
- Venous reservoir (integrated with oxygenator).
- Venous outlet (PVC 3⁄8 inch 8mm).
- Pump (peristaltic roller pump or centrifugal).
- Oxygenator (membrane oxygenator, defoamer and heat exchanger).
- Arterial fi lters macro and micro (300 micrometer) and bubble detector.
- Arterial line (PVC 3⁄8 inch).
- Arterial cannula (aortic, femoral, or axillary).

PATHOPHYSIOLOGY OF BYPASS

The pathophysiological changes associated with bypass are due to more than activation of the whole-body inflammatory response as a result of the passage of blood through the non-endothelial circuitry. Changes in temperature, acid–base balance, hemodilution, non-pulsatile flow, drugs, circulating volume, and the mechanics of bypass all contribute to dysfunction of blood constituent cascades and whole organ systems.


Air Bubble Detector
Air Removal
Centrifugal Pump
Venous Bubble Trap
Electronic Venous Line Occular
Arterial Filter
Oxygenator
Cardioplegia
Flexible Reservoir
Roller Pump

CONGENITAL HEART DEFECT TYPES

There are many types of congenital heart defects. If the defect lowers the amount of oxygen in the body, it is called cyanotic. If the defect doesn't affect oxygen in the body, it is called acyanotic.

CYANOTIC HEART DEFECTS

are defects that allow oxygen-rich blood and oxygen-poor blood to mix.

In cyanotic heart defects, less oxygen-rich blood reaches the tissues of the body. This results in the development of a bluish tint—cyanosis—to the skin, lips, and nail beds.

Cyanotic heart defects include:

Tetralogy of Fallot .

Transposition of the great vessels .

Pulmonary atresia .

Total anomalous pulmonary venous return .

Truncus arteriosus .

Hypoplastic left heart syndrome .

Tricuspid valve abnormalities .

ACYANOTIC HEART DEFECTS?

Congenital heart defects that don't normally interfere with the amount of oxygen or blood that reaches the tissues of the body are called acyanotic heart defects. A bluish tint of the skin **isn't common** in babies with acyanotic heart defects, although it may occur. If a bluish tint occurs, it often is during activities when the baby needs more oxygen, such as when crying and feeding.

Acyanotic congenital heart defects include:

Ventricular septal defect (VSD) .

Atrial septal defect (ASD) .

Atrioventricular septal defect .

Patent ductus arteriosus (PDA) .

Pulmonary valve stenosis .

Aortic valve stenosis .

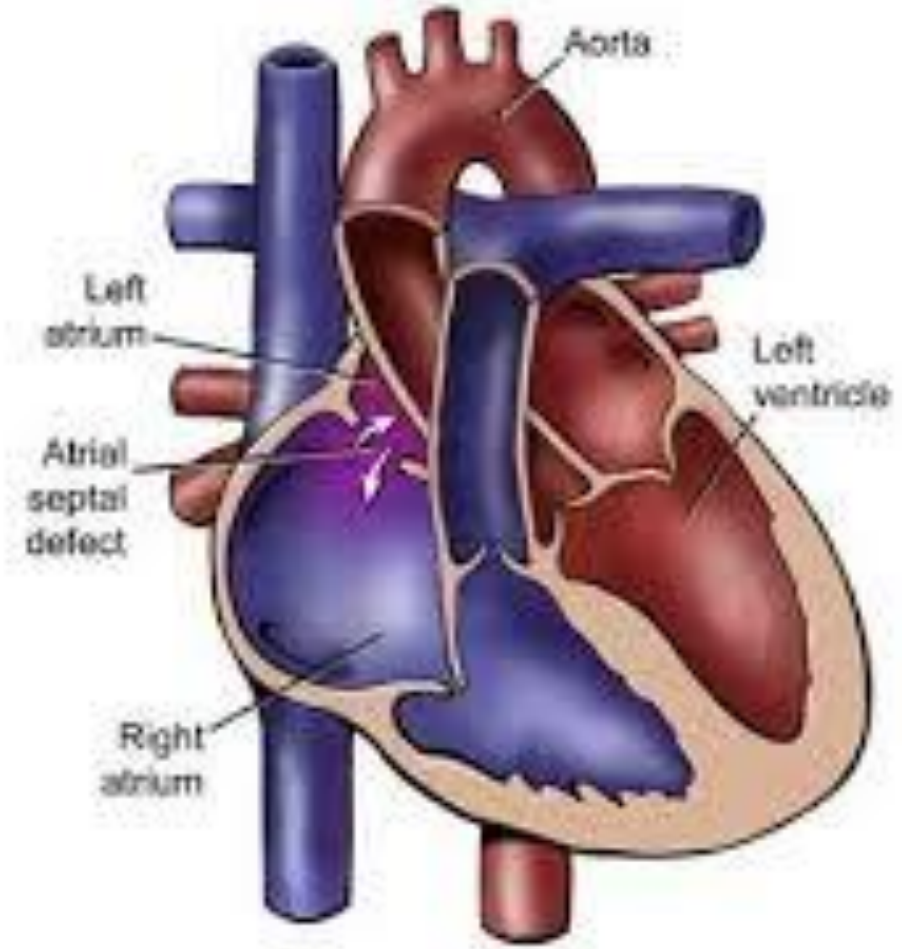
Coarctation of the aorta .

A small hole in the heart, called a patent foramen ovale, is not considered a heart defect. It happens in many healthy people. But typically it doesn't need treatment.

ATRIAL SEPTAL DEFECTS

• ASDs are defects at atrial level leading to shunting of blood between left and right atria. They constitute 10–15% of congenital cardiac defects, and up to 40% of congenital defects presenting in adulthood.

• The commonest defects are of the septum *secundum*, the 'true' septum, these constitute >80% of ASDs. Female:male=2:1.



TYPES

- ***Ostium secundum* defects (70–80%):** commonest type of ASD. They involve the fossa ovalis. Should not be confused with a patent foramen ovale which occurs in 25% of adults, where septum primum lying to the left of the foramen ovale fails to fuse with its sides postnatally, leading to 'probe patency'. Secundum ASDs may extend to the IVC.

- ***Ostium primum* defects (20%):** form of AV defect and as such are by defi nition associated with cleft mitral valve.

- ***Sinus venosus defects* (10%):** majority are superior sinus venosus ASDs, at the mouth of the SVC, associated in 90% of cases with *partial anomalous pulmonary venous drainage (PAPVD)* of anomalous right upper pulmonary vein from the upper ± middle lobe draining into this junction.

- **Coronary sinus defects (<1%):** varying communications between the coronary sinus and the LA, also known as unroofed coronary sinus, almost always associated with persistent left SVC.

PATHOPHYSIOLOGY

ASDs permit left-to-right shunting, causing volume overload of the right heart and increased pulmonary blood flow

NATURAL HISTORY/LONG-TERM SEQUELAE

In patients operated on before the age of 25, long-term survival is normal, but after this age, mortality is higher than in healthy controls. When ASDs are closed in childhood, the long-term risk of arrhythmias is not much higher than the general population, but after the age of 11 this risk increases, so that about 50% of 60-year-olds with ASD have flutter or fibrillation or sinus node dysfunction.

PRESENTATION AND INITIAL MANAGEMENT

ASDs rarely cause symptoms in children, and are usually found incidentally. presenting in their third/fourth decade with fatiguability and reduced exercise tolerance. Rarely, if the defect is very big, there may be heart failure and failure to thrive (1% of patients). In older children and adolescents, there may be reduced exercise tolerance in comparison to peers. Patients are pink, with normal respiratory rate. (Infants may be tachypneic.) If desaturated, indicates right-to-left shunt, and should be further investigated before surgery.

Systolic ejection murmur over left second intercostal space—pulmonary flow murmur.

INVESTIGATIONS

- EKG normal in 20–40% of children. May show right axis deviation
- CXR shows iRA, RV, PA, and pulmonary vascular markings.
- Echo is diagnostic and identifies RA, RV, and PA enlargement, septal defect and associated anomalies.
- Catheterization is not indicated in the routine management of secundum ASD. It is necessary if there are concerns about pulmonary hypertension, to measure pulmonary vascular resistance and its reversibility with vasodilators.

TREATMENT

Percutaneous options

Defects up to 40mm within the fossa ovalis, with a 5mm rim between the defect and AV valves/major veins, can be closed percutaneously in the cath lab. The device is placed across the atrial septum via a catheter in the femoral vein, under TEE guidance. Success depends on the anatomy of the defect, but this is becoming the standard treatment for suitable defects.

Complications: **incomplete defect closure, device migration or erosion, local vascular complications.**

Surgical management

Up to 40% of small and moderate sized ASDs close spontaneously by age 4, although this becomes rare after age 2. Secundum ASDs still present after this time should be closed.

Indications

• In adults ASDs should be closed to prevent pulmonary hypertension.

• In older patients with pulmonary hypertension, if PVR <10U/m2, the outcomes are good; but with 15U/m2 or higher, the mortality is high.

• In infants, a large shunt with failure to thrive, recurrent respiratory tract infections or signs of heart failure. Evidence of RV or LV volume

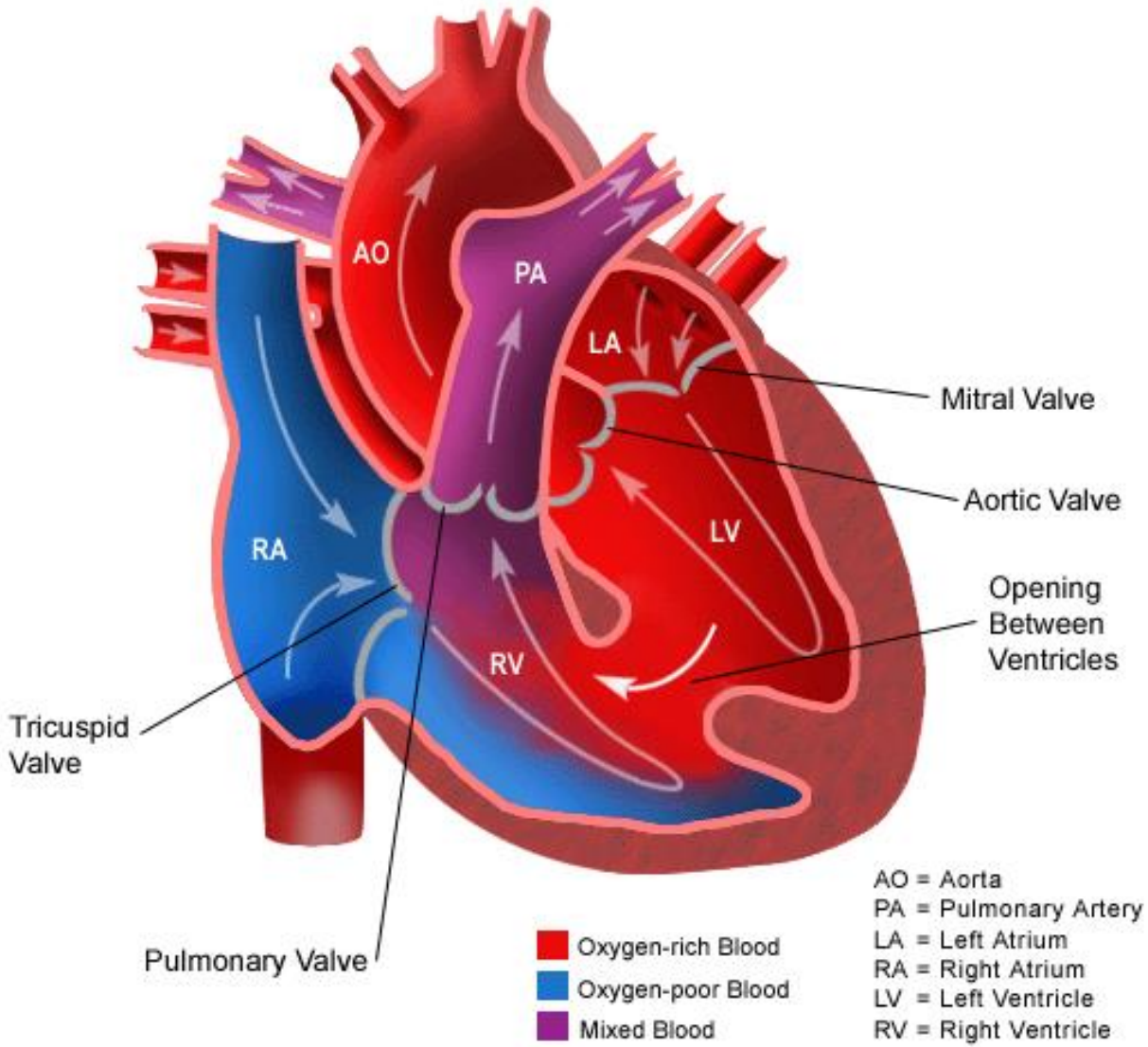
overload. Secundum ASDs should be closed electively in 3–5-year-olds.

• Paradoxical embolus causing TIA/stroke.

VENTRICULAR SEPTAL DEFECTS

- A VSD is a congenital, abnormal defect in the ventricular septum allowing communication of blood between the ventricles.
- They occur as isolated lesions, or in combination with other anomalies constituting 25% of all defects: the commonest congenital heart defect.
- Prevalence increasing over the last three decades with the improvement of imaging technology, and associated with maternal drug and alcohol abuse.

Ventricular Septal Defect (VSD)



VSDs CAN MOST SIMPLY BE CLASSIFIED AS FOLLOWS:

Perimembranous (70–80%)

Perimembranous VSDs: where a margin of the VSD consists of fi brous continuity between the tricuspid and aortic valves. The conduction bundles

run along the inferior rim of the defect. They may be *inlet, outlet, or inlet–outlet VSDs*.

Muscular (5–10%)

• Also called *trabecular* and including central, mid-muscular, apical, marginal, and multiple or 'Swiss cheese' VSDs. • They may be single or multiple, associated with other types of VSD and may occur anywhere within the muscular septum.

• The rim of the VSD is entirely muscular. The conduction bundles are remote from the defect, and in the case of the inlet VSD, they run near the superior margin of the defect. They may be located in the

inlet, apicotrabecular, or outlet portions of the RV.

Juxta-arterial (5–10%)

• Also called *conal septal, supracristal, infundibular, subpulmonary, doubly committed sub-arterial (DCSA) VSDs*.

• The conjoined leaflets of aortic and pulmonary valves form rim of VSD.

• The conduction bundles are remote from the defect.

• They extend up to the aortic and pulmonary annuli, and result in aorticvalve prolapse in up to 50% of cases.

PATHOPHYSIOLOGY

May be *restrictive defect*: small defects, presenting resistance to flow

across the defect, or *non-restrictive defect*: where the cross-sectional area of the defect is equivalent or larger than that of the aortic annulus

Three hemodynamic consequences:

(1) LV volume overload,

(2) increased pulmonary blood flow,

(3) reduced systemic cardiac output.

Presentation

Larger defects present in infancy with CHF, recurrent chest infections, failure to thrive; Qp;Qs >2 is poorly tolerated. Smaller defects may be entirely asymptomatic. Bacterial endocarditis is a risk.

Examination shows failure to thrive (FTT), tachypnea, and recession.

Investigations

- EKG shows LV and RV hypertrophy.
- CXR may show increased vascular markings.
- Echo is diagnostic, and defines the location and size of the defect, the extent of the hemodynamic consequences, and associated anomalies.
- In cases where pulmonary vascular disease is suspected, or there are multiple VSDs, cardiac catheterization may be required.

Natural history

- 30–40% of all VSDs close spontaneously. Up to 70% of small VSDs close spontaneously (50% of small PM VSDs close spontaneously in the first 2 years). Inlet perimembranous VSDs and DCSA VSDs do not close spontaneously, and are candidates for early closure.
- 10% of infants with large untreated VSDs die within fi rst year of life.
- Eisenmenger syndrome may be complicated by fatal hemoptysis, polycythemia, cerebral abscess or infarction, and RV failure.

TREATMENT:

Percutaneous closure

VSDs can be closed by percutaneously placed devices—these are mainly used to close muscular defects, and smaller perimembranous defects. In the latter there is a high incidence of AV node block, up to 10%.

Surgical closure

Operative mortality for VSD closure in pediatric setting is <1%.

Indications

- Moderate to large VSD after age 1 year.
- Congestive cardiac failure resistant to medical therapy in infants.
- Elevated pulmonary vascular resistance after the age of 6 months.
- Evolving aortic regurgitation in outlet VSDs.
- Multiple muscular VSDs (Swiss cheese defects) with significant shunt

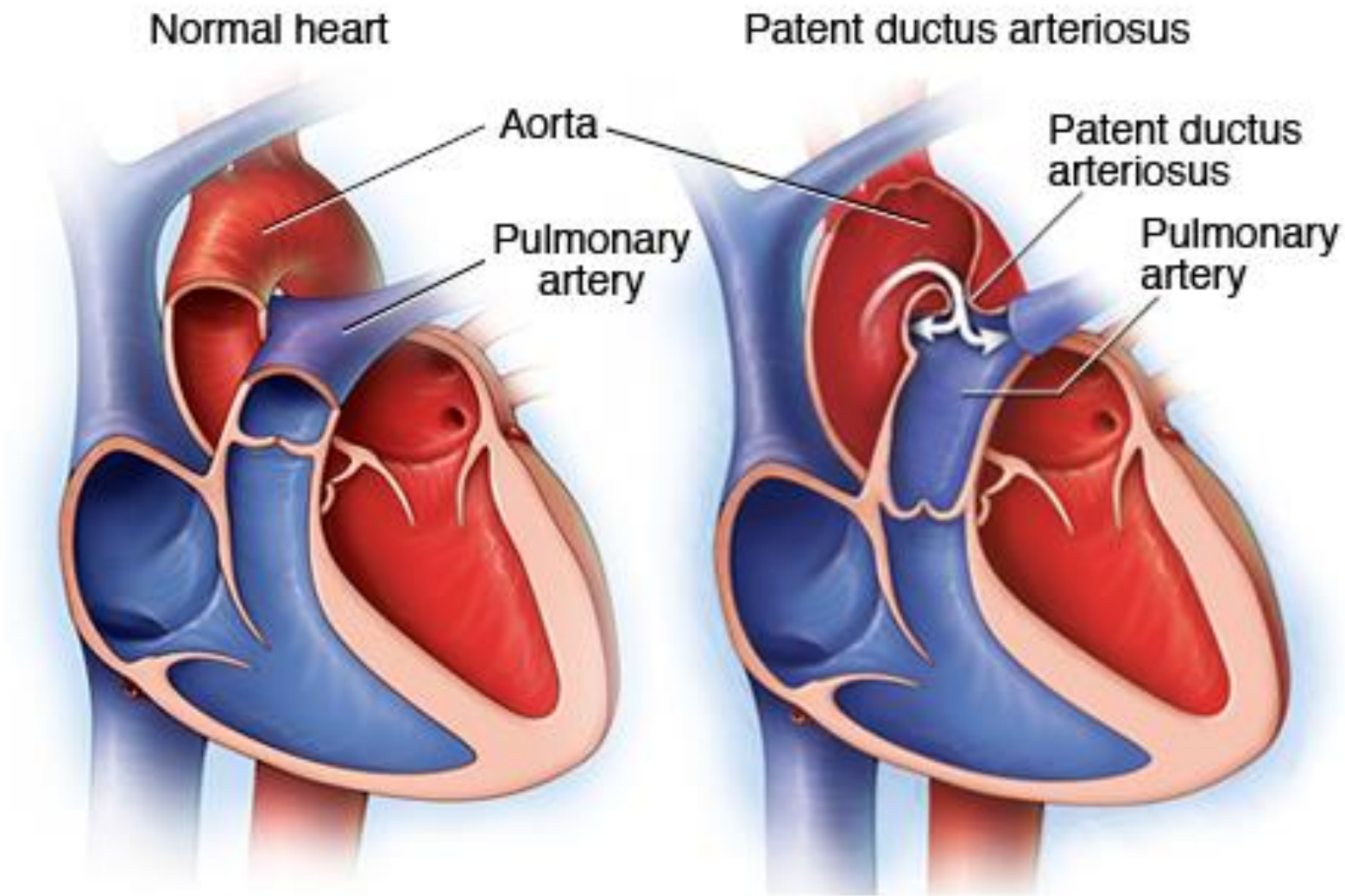
undergo PA band

PATENT DUCTUS ARTERIOSUS

• The arterial duct connects the pulmonary artery with the descending aorta, and in fetal life shunts blood from the pulmonary artery to the aorta, bypassing the immature fetal lungs.

• Most close within 2–3 weeks of birth, initially by contraction of the medial muscle of the duct, then by intimal hyperplasia. Persistence of the lumen after this time, with reversal of the flow, is called *patent ductus arteriosus (PDA)*.

• This anomaly makes up 12–15% of congenital heart defects, and up to 30% of defects in premature infants. 80% of premature infants <1200g present with this. Isolated PDA occurs 1 in 2000 live births.



PATHOPHYSIOLOGY

• When the PDA is large, aortic and PA pressures are equal, and the magnitude and direction of shunting depends on the PVR.

• As neonatal PVR falls the left-to-right shunt increases and congestive

cardiac failure ensues.

• Pulmonary vascular changes may lead to irreversible pulmonary hypertension and reversal of the left-to-right shunt (Eisenmenger syndrome) which has a very poor prognosis, even though the initial reduction in the left-to-right shunt leads to a brief improvement in the clinical picture

Presentation

Presentation depends on the size of the PDA.

- **Large PDAs present with symptoms of severe heart failure in preterm babies, a wide pulse pressure, increase JVP, and a systolic murmur.**
- **The findings are similar in smaller PDAs but develop later; additionally the ductus may be calcified.**
- **Premature infants with this defect may be ventilator-dependent, or require long periods of non-invasive ventilation.**
- **Many children may be asymptomatic. Large shunts may present as congestive heart failure early in life.**

Examination

- **Bounding pulses due to wide pulse pressure.**
- **In premature infants, there is usually a systolic murmur at the second left intercostal space; with increasing size of the shunt, this murmur may extend into diastole and become the classic harsh, continuous 'machinery' murmur.**

Investigations

- The EKG shows left and sometimes right ventricular hypertrophy.
- CXR may be normal, or show cardiomegaly, an enlarged PA, and pulmonary congestion in large shunts.
- Echo is diagnostic, demonstrating the shunt flow from descending aorta to pulmonary artery, and enlarged left-sided chambers. Associated anomalies must be carefully excluded.
- Cardiac catheterization is not usually required, but catheter closure is usually possible in infants >2kg.

MANAGEMENT

Indomethacin and ibuprofen are equally effective in closing the patent duct in pre-term infants, and improve outcomes even in asymptomatic pre-term infants.

If medical therapy fails, surgical ligation is considered for all symptomatic infants, and in all infants in whom the duct has not closed after 3 months.

Indications for closure

- Persistent PDA is an indication for elective closure.
- Urgently if there is evidence of cardiac failure.

Atrioventricular septal defects

• AVSDs, also called *endocardial cushion defects, AV canal defects,* or *atrioventricular communis*, are a spectrum of defects resulting from incomplete development of the atrial septum, the inflow portion of the ventricular septum, and the atrioventricular valves.

• These defects make up about 5% of congenital cardiac anomalies.

• Congenital heart disease is present in 45% of children with Down's syndrome, and 45% of these have an AVSD.

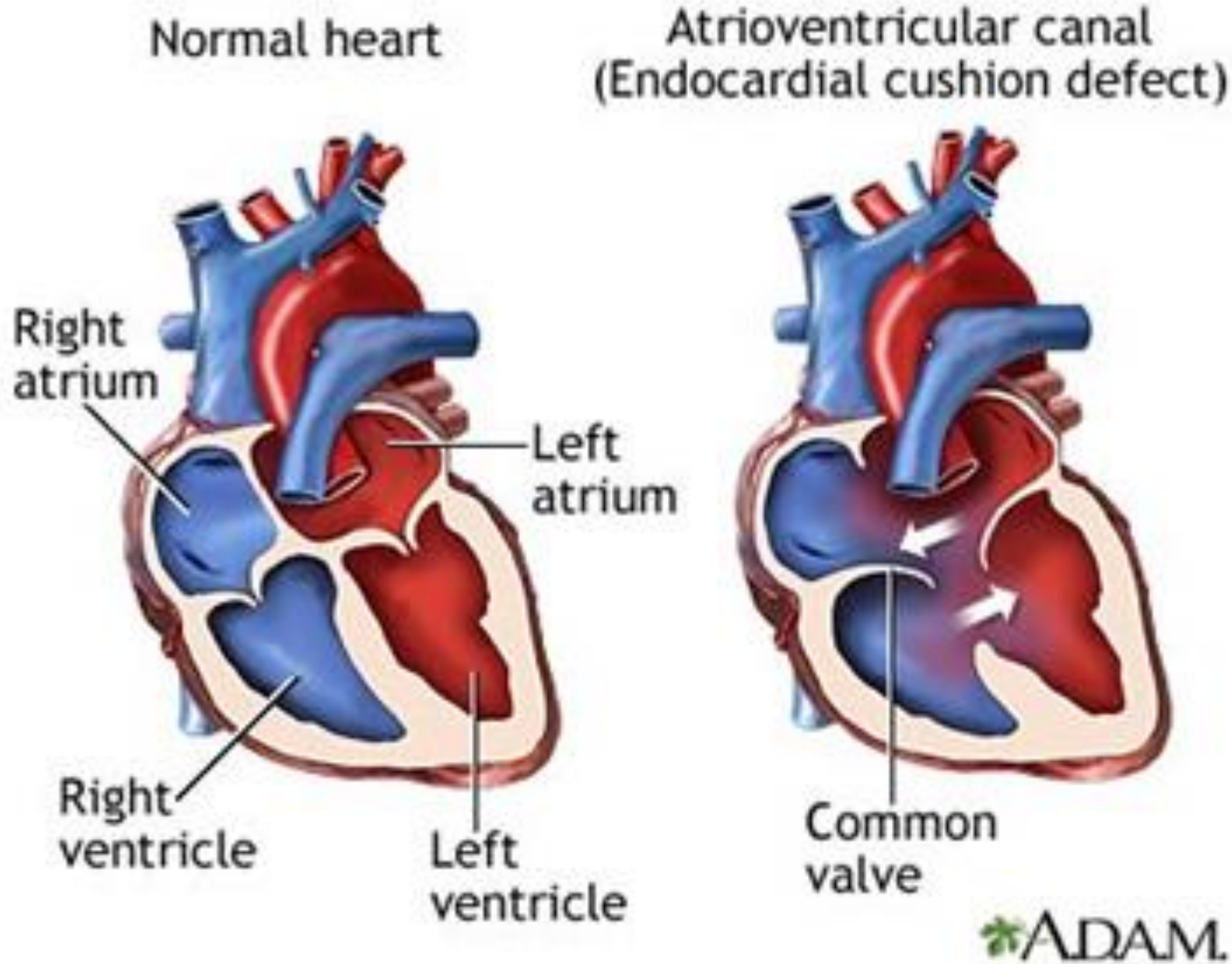
TYPES

Partial AVSD (primum ASD)

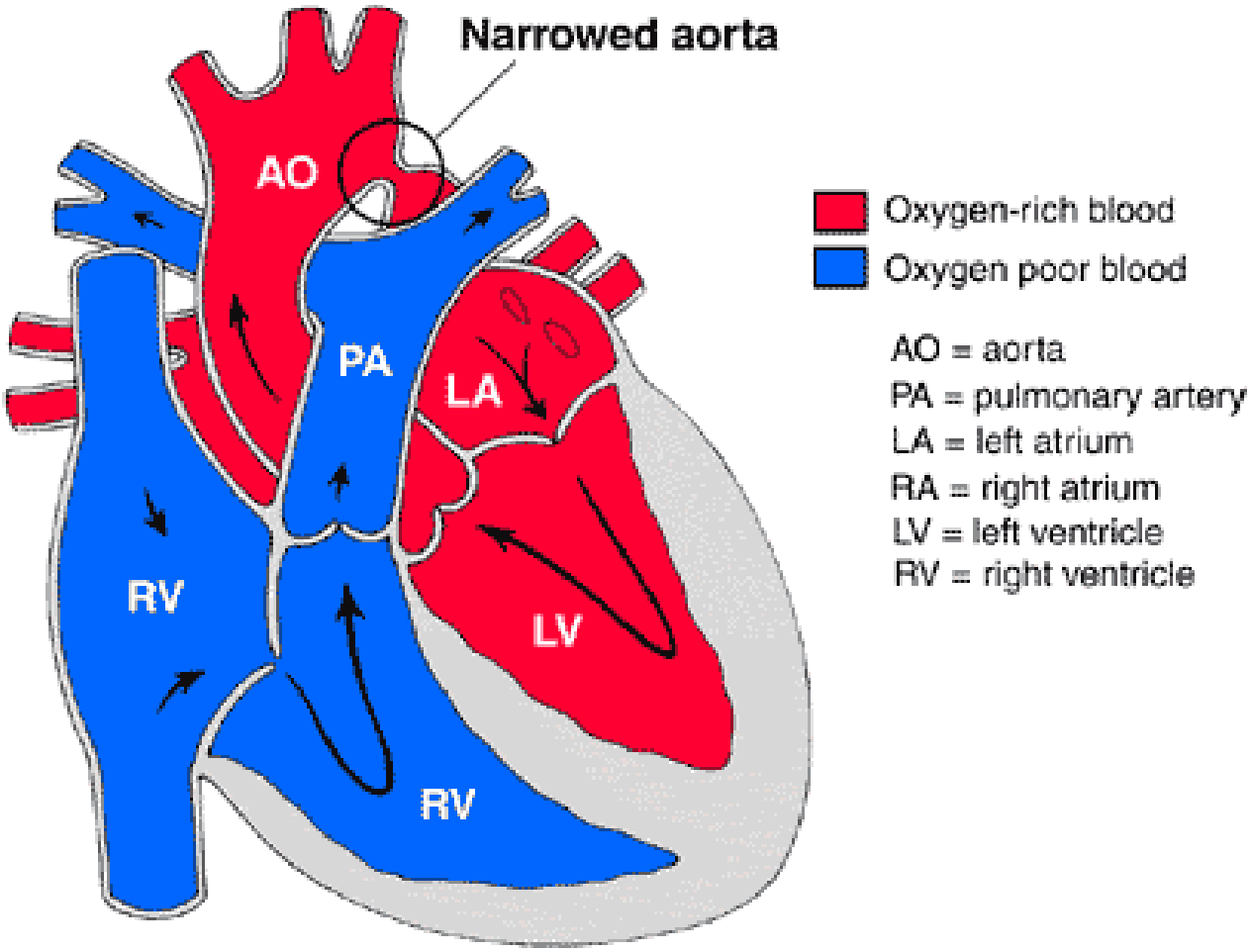
Inferior ASD immediately superior to the separate left and right AV valves; the left AV valve is trileafl et, and in 10% it is incompetent.

Complete AVSD

Single AV valve common to the right and left atrioventricular junction


Normal heart
Atrioventricular canal
(Endocardial cushion defect)
Right
atrium
Left
atrium
Right
ventricle
Left
ventricle
Common
valve
A.D.A.M.

Coarctation of Aorta



COARCTATION OF THE AORTA

• Coarctation of the aorta is defined as a congenital narrowing of the upper descending aorta, opposite the ductus arteriosus.

• This accounts for 5–8% of all congenital heart defects.

• It may be isolated, but is associated with bicuspid aortic valve and VSD.

It is the most common cardiac defect in *Turner syndrome*. (15–20%),

PATHOPHYSIOLOGY

• The hemodynamic consequences are high afterload on the LV,

increasing LV wall stress and causing *LV hypertrophy*.

Hypertension develops due to the mechanical obstruction and

possibly, renin–angiotensin-mediated pathways.Systemic perfusion depends on ductal flow

PRESENTATION AND NATURAL HISTORY

- Depends on the existence of coexisting abnormalities, as well as the location and severity of the location:

- *Neonates*: collapse, acidosis, hypotension, heart failure; absent femoral pulses on routine review.
- *Infancy*: upper extremity hypertension with absent/reduced femoral pulses; congestive heart failure causing dyspnea and failure to thrive.
- *Children/young adults*: headaches, lower extremity weakness, exertional dyspnea, fatigue; hypertension.

- Examination:

findings also depend on the age and presentation:

- In the shocked neonate, all pulses may be weak; however, absent femoral pulses should not be disregarded.
- There may be differential cyanosis, Systolic murmur, discrepancies of >20mmHg between upper and lower

INVESTIGATION

- EKG: LV hypertrophy.
- CXR: in sick children, cardiomegaly, pulmonary congestion.
- Echo: shows the arch,
- MRI/CT useful in older patients or reoperations to plan approach.

MANAGEMENT

Neonates

In the shocked neonate, the initial management is supportive, improving peripheral perfusion by *reopening the duct* if possible

Older children

Balloon angioplasty of the coarctation is now the fi rst-line treatment.

Hypertension should be treated, usually with β-blockers, but intervention

should not be delayed while waiting for normotension.

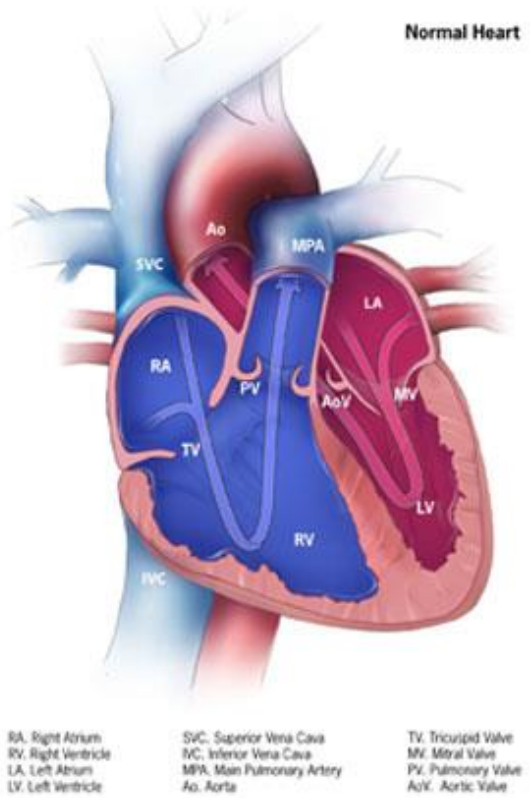
Indications for surgery

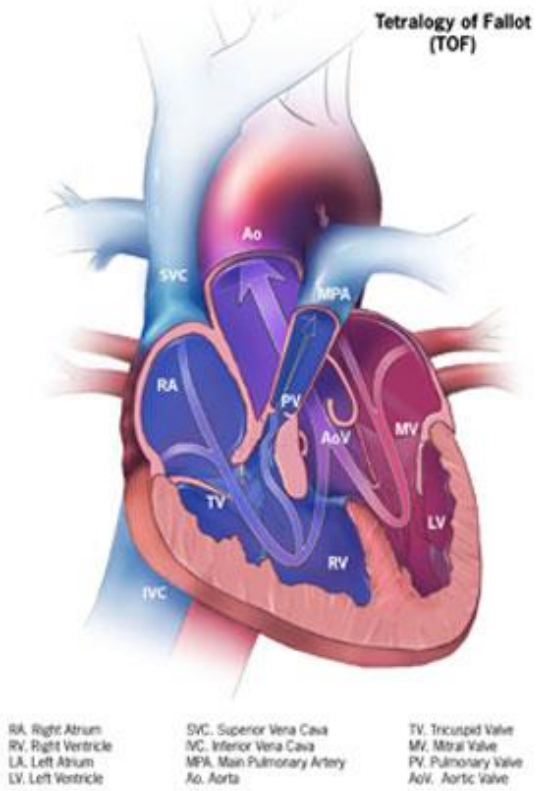
Isolated coarctation is an indication for surgical repair, once critically ill

infants have been stabilized, and within 4–6 weeks of presentation in stable

children.

CYANOTIC HEART DEFECTS
TETRALOGY OF FALLOT





ToF makes up 10% of congenital heart defects and is the commonest cyanotic lesion. 25% of patients have DiGeorge syndrome or deletion/ abnormalities of chromosome 22.

• It is usually an isolated defect, and is characterized by four lesions caused by anterior deviation of the outlet septum:

1- Pulmonary stenosis (at the infundibulum, valve or the PA).

2- RV hypertrophy.

3- Overriding aorta.

4- VSD.

• Children present soon after birth with cyanosis, depending on the severity of the pulmonary stenosis, and are usually managed with surgical correction.

PATHOPHYSIOLOGY

The RVOT obstruction *reduces pulmonary blood flow* and also increases the RV pressure causing *a right-to-left shunt* across the large, unrestricted VSD. Both these effects cause *cyanosis*. Immediately after birth, the ductus arteriosus supplies blood to the pulmonary vasculature, but after it closes there may be a sudden onset of cyanosis, and the RV will start to hypertrophy. In a small subset, the RVOT obstruction is so mild as to avoid cyanosis: '*acyanotic*' *or* '*pink*' *tetralogy*. The pulmonary vasculature is protected from the effects of the VSD by the outflow obstruction, and some patients may be well balanced for years.

PRESENTATION AND NATURAL HISTORY

In children with a very hypoplastic annulus (pulmonary stenosis), the presentation is usually soon after birth, with cyanosis due to the obstruction in pulmonary blood flow and thus increased flow across the VSD from right to left. In children with a larger pulmonary valve annulus, presentation is usually later, and may be with 'tet spells'—cyanotic episodes that occur when there is muscular spasm causing acute obstruction to RV outflow and reversal of the shunt(also due to falling SVR with exercise). 'Squatting' is a characteristic compensatory maneuver, seen in right-to-left shunts developing on exercise.

Hemoptysis may be present late. Examination shows RV heave and a systolic ejection murmur. The degree of RVOT obstruction is the main determinant of outcome in untreated ToF: most children develop signs and symptoms by 6 months of age, and <10% survive to the age of 21 without surgical intervention. 25% of untreated patients die within 1 year of life, 40% of untreated patients die within 4 years, and 95% of untreated patients die within 40 years.

INVESTIGATIONS

- EKG shows right-axis deviation, RVH and RA hypertrophy.
- CXR shows a 'boot-shaped' heart as a result of RVH and the small or absent MPA; and decreased pulmonary vascularity.
- Echo is diagnostic, and defines the severity and extent of the RVOT obstruction, the size of the pulmonary valve annulus, the size and position of the VSD, the coronary artery arrangement, and any associated anomalies.
- Cardiac catheterization is used if there is a need to further image the coronary arteries, or if there are concerns about the size of the pulmonary arteries.

SURGICAL MANAGEMENT

There is an increasing trend towards earlier complete repair, but there is still a place for early palliation with a *systemic–PA shunt* followed by repair at a later stage. A sensible approach is to aim to repair all tetralogies between 3–6 months. If they present with cyanotic spells they can be repaired earlier, or if institutional policy dictates, undergo a *B-T shunt* with elective repair when they are bigger.

TRANSPOSITION OF GREAT ARTERIES

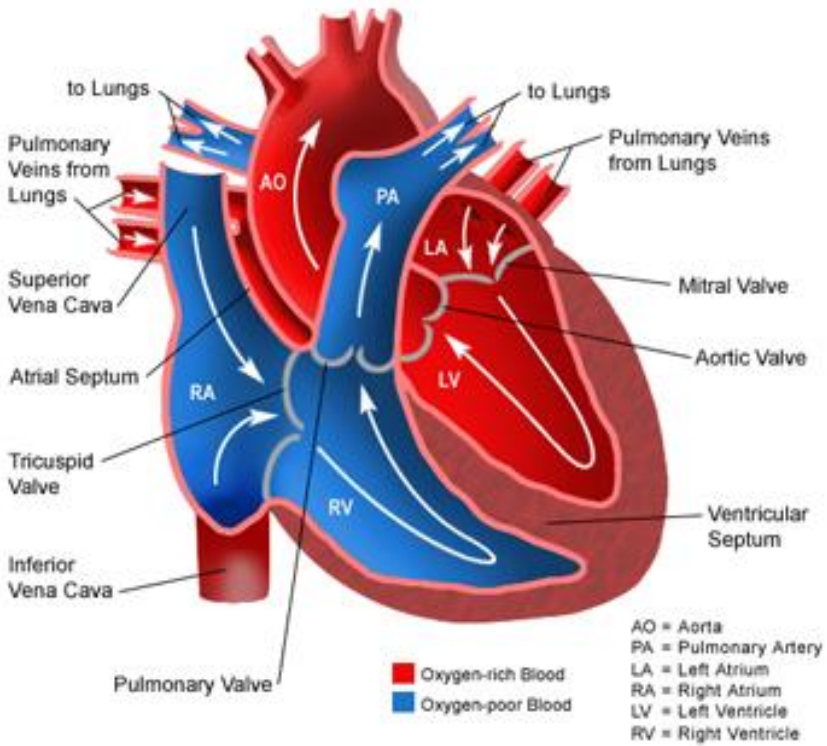
• TGA is a congenital cardiac anomaly in which the aorta arises from the RV and the pulmonary trunk arises from the LV.

• Complete TGA is the most common cyanotic lesion presenting in the neonatal period, and makes up 5% of congenital cardiac lesions. In 90% it is isolated, with no associated extracardiac malformations.

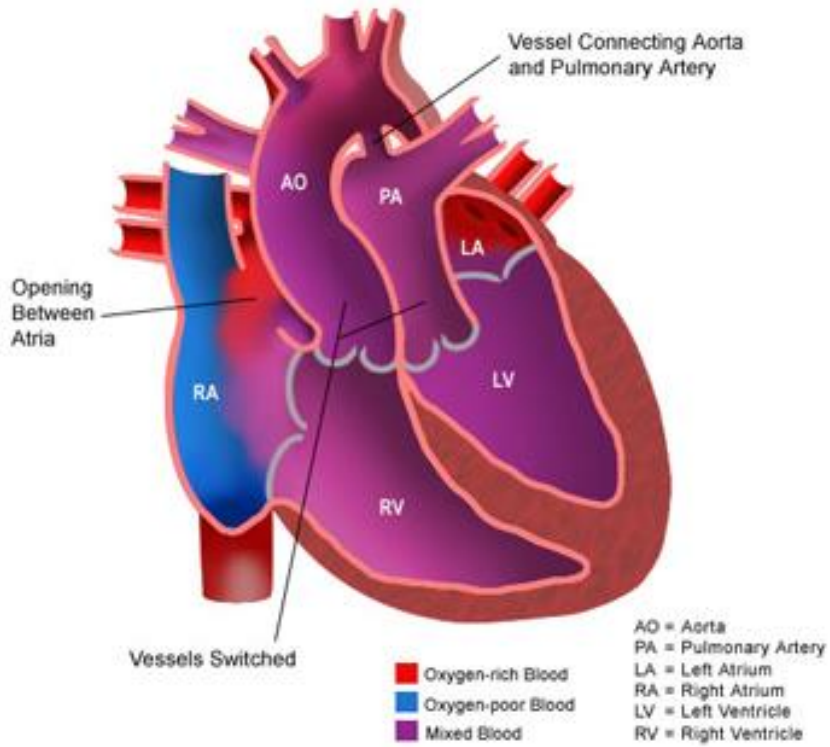
• Infants present with cyanosis and in the absence of an ASD or VSD infants die within a few hours of closure of the ductus arteriosus.

• A corrective arterial switch procedure can be performed in neonates with low mortality.

Normal Heart



Transposition of Great Arteries



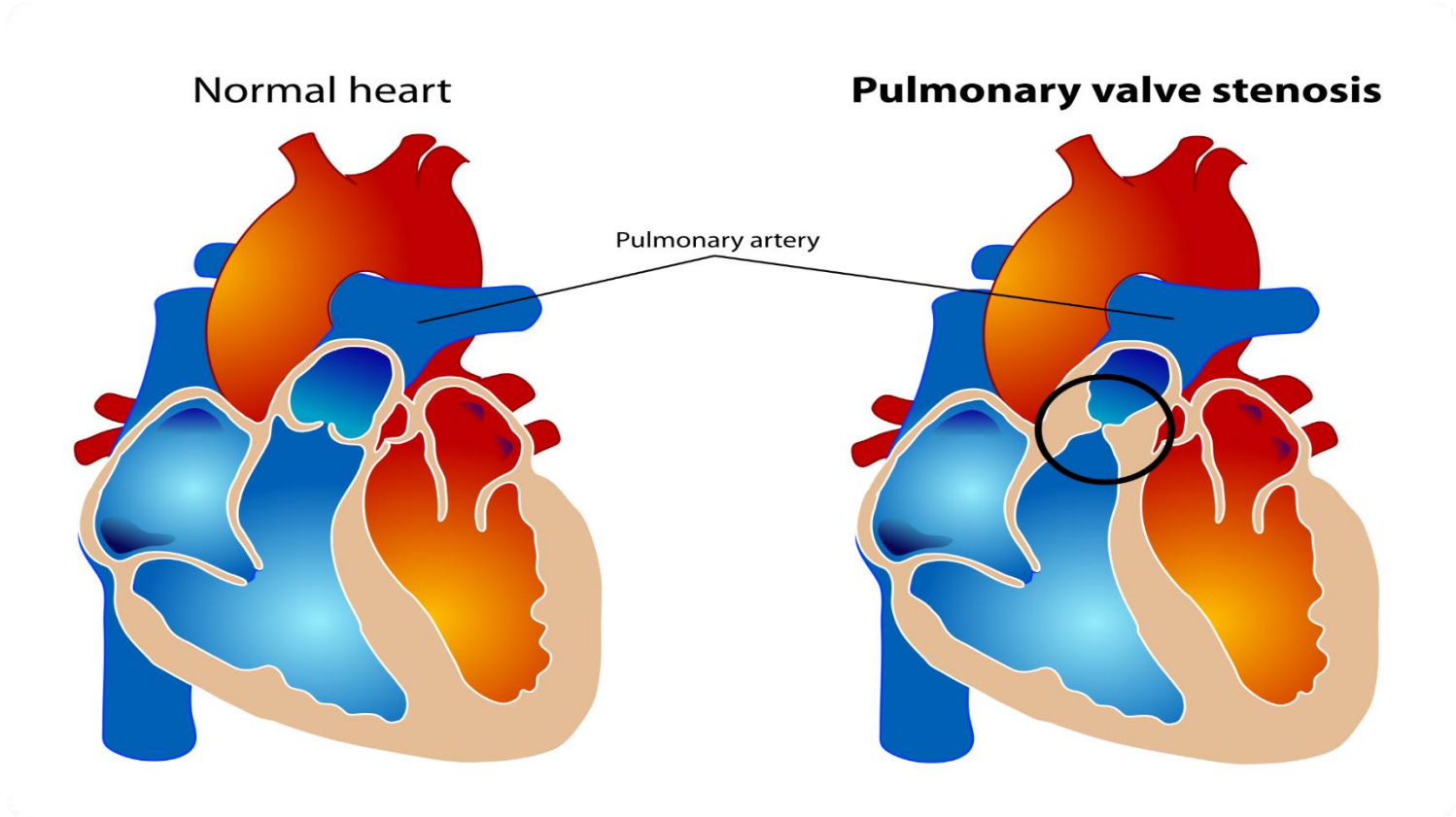
PULMONARY STENOSIS

Key facts

This defect makes up 8–10% of all congenital cardiac defects, and usually involves valvar PS, but includes subvalvar and supravalvar PS.

Morphology

The *pulmonary valve is stenotic*, and usually is tricuspid with fusion of the commissures, thus producing a dome-like structure with a variable central opening. There is often a *PFO or ASD* (40%). There may be *RV hypertrophy*, even at birth, and *subvalvar stenosis* from infundibular muscle hypertrophy. There may be *poststenotic dilatation* of the pulmonary arteries.


Normal heart
Pulmonary valve stenosis
Pulmonary artery

ISCHEMIC HEART DISEASE

Pathophysiology

• Stenotic coronary artery disease (CAD) is narrowing of the coronary arteries caused by thickening and loss of elasticity of the arterial walls (atherosclerosis).

• Stable plaques are mostly responsible for the lesions seen on angiography and stable angina: MI is due to unstable plaque rupture and thrombosis: 50% of MIs occur distal to angiographically ‘normal’ vessels.

Symptoms

Exertional angina: this is common. It results from reduction of coronary flow reserve, and the severity depends on the mismatch between myocardial oxygen supply and demand, i.e., both the severity of the CAD and the amount of work the myocardium is required to do.

Dyspnea: graded in the same way as angina A number of mechanisms contribute. Acutely, transient systolic, and/or diastolic LV dysfunction results from worsening myocardial supply:demand mismatch

caused by increases in preload, afterload, hypotension, or exertion

. Orthopnea results from the sudden increase in preload on lying flat. Paroxysmal nocturnal dyspnea is due to

pulmonary edema, and poorly understood changes in respiratory drive.

Nausea: parasympathetic stimulation results in nausea and vomiting.

Unstable angina: this term, which covers a multitude of syndromes all of which refl ect an adverse prognostic turn, applies to patients with severe and persisting angina. It is most commonly caused by non-occlusive thrombus at the site of an unstable plaque. Unstable angina normally recurs as either another episode of angina or as an acute MI. Patients can be divided into low-, medium-, and high-risk categories depending on the duration of their angina, severity of symptoms, presence of EKG changes, and hemodynamic compromise.

Acute coronary syndrome (ACS)

- This includes unstable angina, NSTEMI, and STEMI.
- Unstable angina is differentiated from NSTEMI by raised cardiac enzymes (CK-MB or troponin) that are present in NSTEMI.
- STEMI is differentiated from NSTEMI by EKG changes.

VALVULAR HEART DISEASE

Aortic stenosis

Etiology

• Congenital (unicuspid rare, *bicuspid valve* more common around 1–2%). Bicuspid valve morphology varies—may be two equal cusps with central opening, or two unequally sized cusps with raphe in larger cusp indicating where two leafl ets have fused. Bicuspid aortic valves usually functionally normal in younger patients, but leaflets may become increasingly sclerotic with age, leading to accelerated stenosis.
Around 50% of bicupisd aortic valve are stenotic by age 60 years.
Bicuspid valves associated with aortic root dilatation .
• *Rheumatic* (b pp372 and 398) due to commissural fusion, leaflet thickening.
• *Calcific degeneration:* commonest cause, occurring in otherwise normal valves. Rheumatic and bicuspid valves eventually calcify.
• Infective endocarditis (rare cause of AS, usually causes AI).
• Hyperlipidaemia (rare).
• Subvalvar (membrane and muscular) and supravalvular.
• Prosthesis failure (pannus, thrombosis, endocarditis, calcification).

Aortic insufficiency

Etiology

• Myxomatous degeneration (common cause, causes leaflet prolapse).
• Rheumatoid (often mixed picture with degree of AS).
• Infective endocarditis (leaflet perforation).
• Root dilatation (quite common—due to rheumatic, atherosclerotic, aneurysmal, Marfan syndrome, syphilis, ankylosing spondylitis).
• Prosthesis failure: paraprosthetic leak, leaflet perforation.

MITRAL STENOSIS

ETIOLOGY

- CHRONIC *RHEUMATIC* HEART DISEASE (COMMONEST CAUSE).
- *CONGENITAL* MITRAL STENOSIS (RARE).
- *MITRAL ANNULAR CALCIFICATION* (MAC) (LESS USUAL CAUSE OF SEVERE MS).

Mitral regurgitation

Aetiology

- Degenerative mitral valve disease. Commonest cause in West.

Represents spectrum from fibroelastic deficiency (small valves, single segment prolapse), to Barlow's disease (large valves, multisegment prolapse). Also called floppy valve, prolapse, myxomatous disease.

- Rheumatic heart disease.
- Infective endocarditis
- Connective tissue disorders, e.g. Marfan and Ehlers–Danlos syndromes.
- Ischemic heart disease.
- Congenital cleft valve leaflet (associated with primum ASD).
- Endomyocardial fibrosis (common in sub-Saharan Africa).
- Iatrogenic (balloon valvuloplasty of stenotic valve).
- Prosthesis failure (paraprosthetic leak, leaflet perforation).

Tricupid valve disease

TS is rare—usually rheumatoid. TR may be caused by:

- *Functional* secondary to mitral valve disease (commonest).
- *Rheumatic* heart disease.
- *Infective endocarditis.*
- Ebstein's anomaly.
- C*arcinoid* syndrome (usually associated with pulmonary regurgitation).
- Endomyocardial fi brosis.
- Prolapsing cusp.

PULMONARY VALVE DISEASE

Acquired pulmonary valve disease is unusual. Severe pulmonary hypertension may cause dilatation of the pulmonary valve ring causing pulmonary regurgitation.

TYPES OF VALVE PROSTHESIS

Key facts

• Valve prostheses are either *mechanical* or *bioprosthetic* ('tissue').

• Tissue valves are mounted on a metal frame (*stented*), or supported by pig aorta and cloth (*stentless)*. Stented most commonly used.

• Stented valves are either *porcine* aortic valves or *bovine* pericardium.

• *Homografts* are human cadaveric aortic roots, complete with aortic valves *in situ* .

• A *pulmonary autograft* is the patient's own excised pulmonary valve used in the Ross procedure