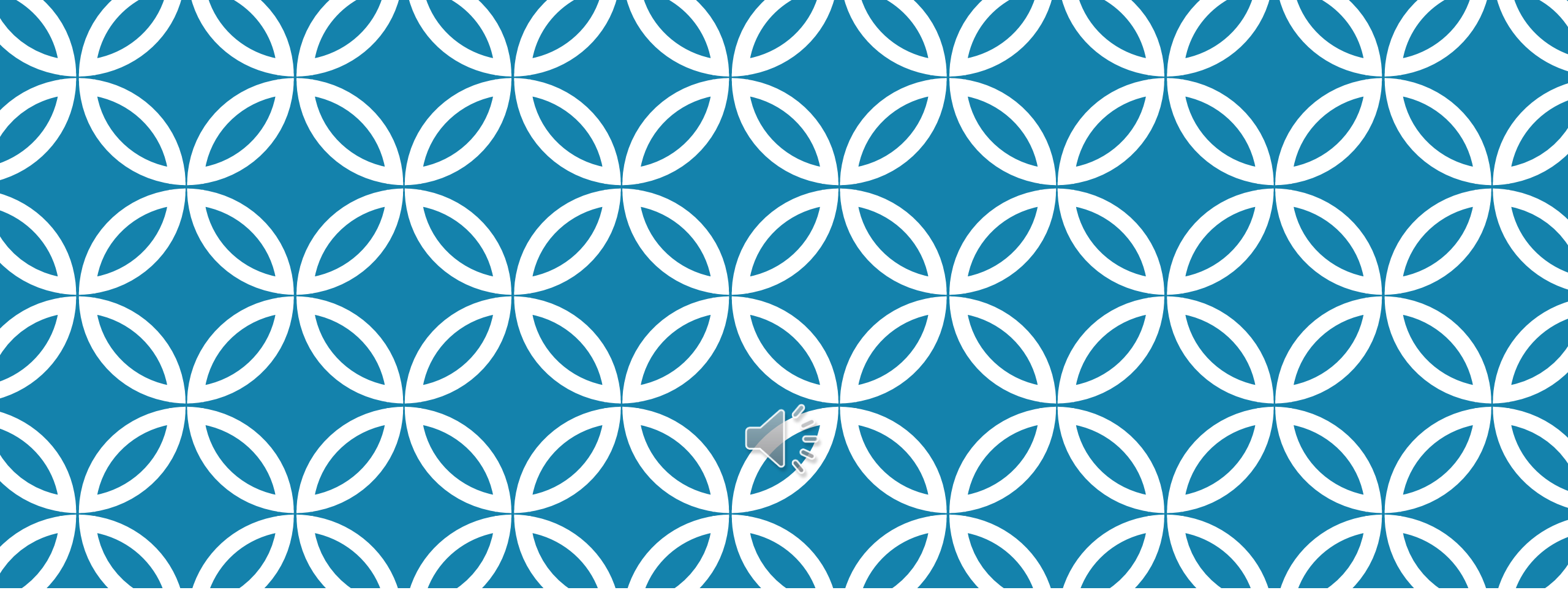


CARDIAC SURGERY

Dr. Bassam M. Hassan

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

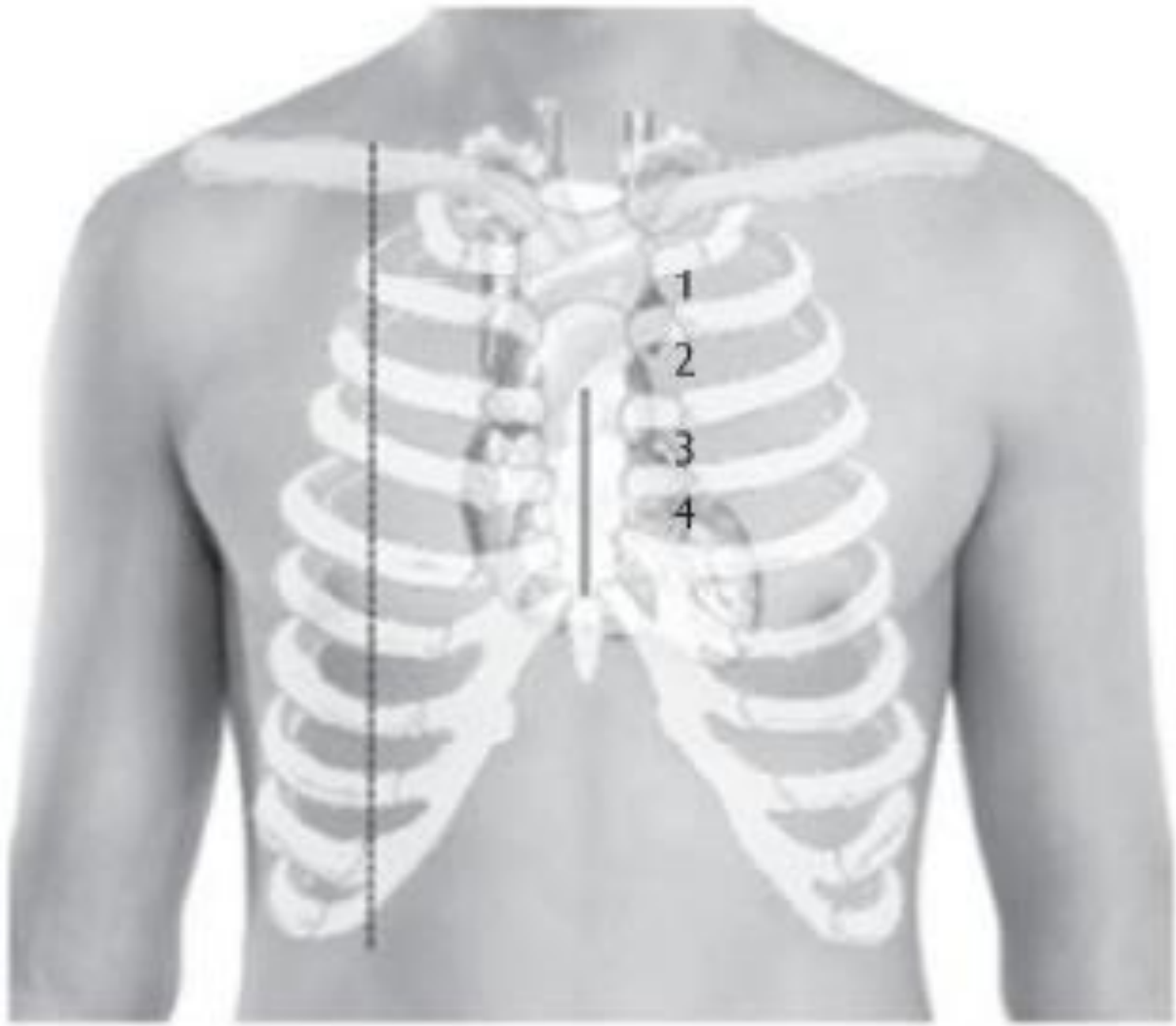
The internal thoracic arteries descend behind the costal cartilages, 1cmlateral to the sternal edge.

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)



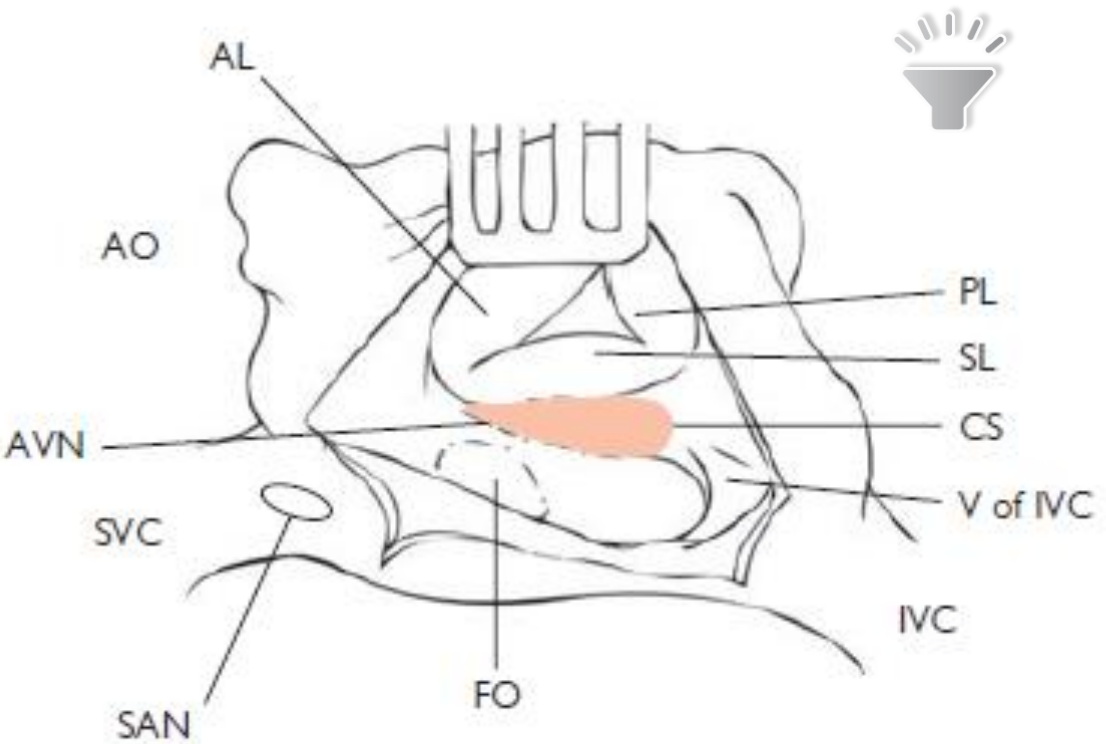


Fig. 2.4 Interior of the right atrium: AO aorta, AL anterior leaflet, PL posterior leaflet and SL septal leaflet of tricuspid valve, AVN atrioventricular node, SVC superior vena cava, FO foramen ovale, IVC inferior vena cava, V of IVC Esutacian valve, CS coronary sinus, T tendon of Todaro, TK triangle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

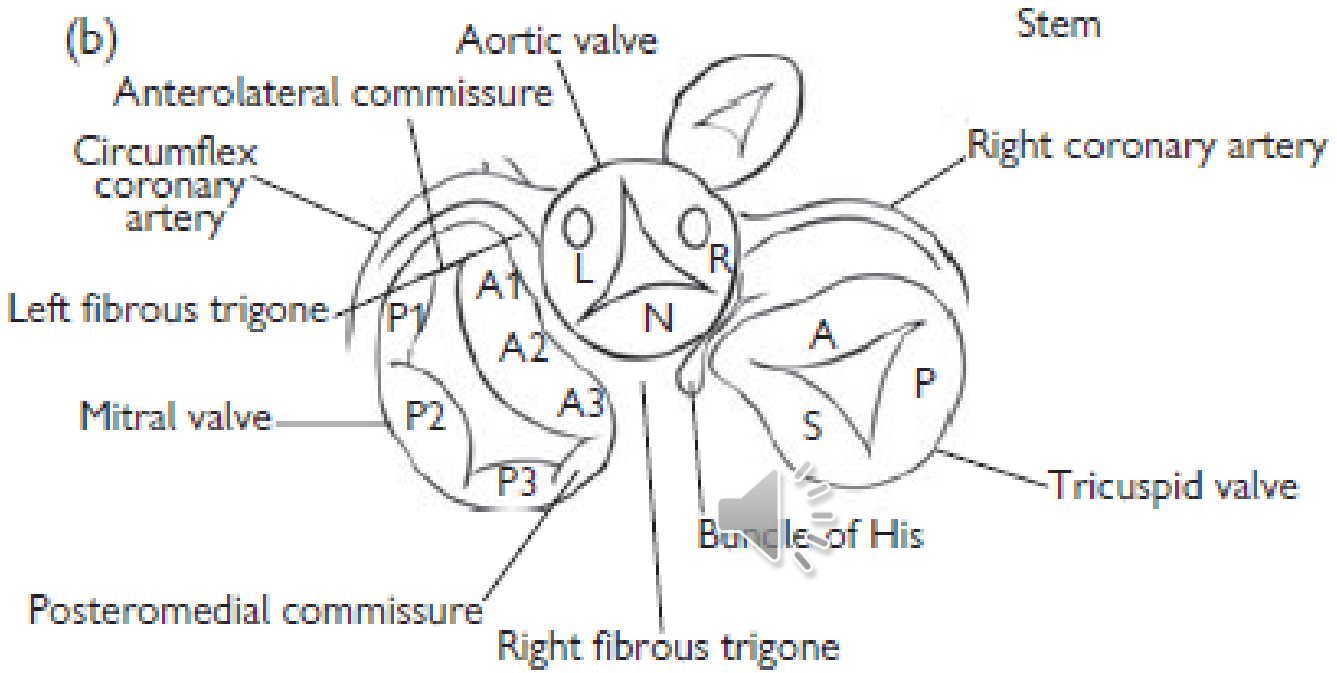


Fig. 2.5 (a) Atrioventricular junction: note the septal atttachment of the tricuspid valve is lower than the septal attachment of the mitral valve. (b) Cardiac valves and their relationships viewed from above, with the atria removed. Note how the commissure of the left (L) and the noncoronary (N) sinuses 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.

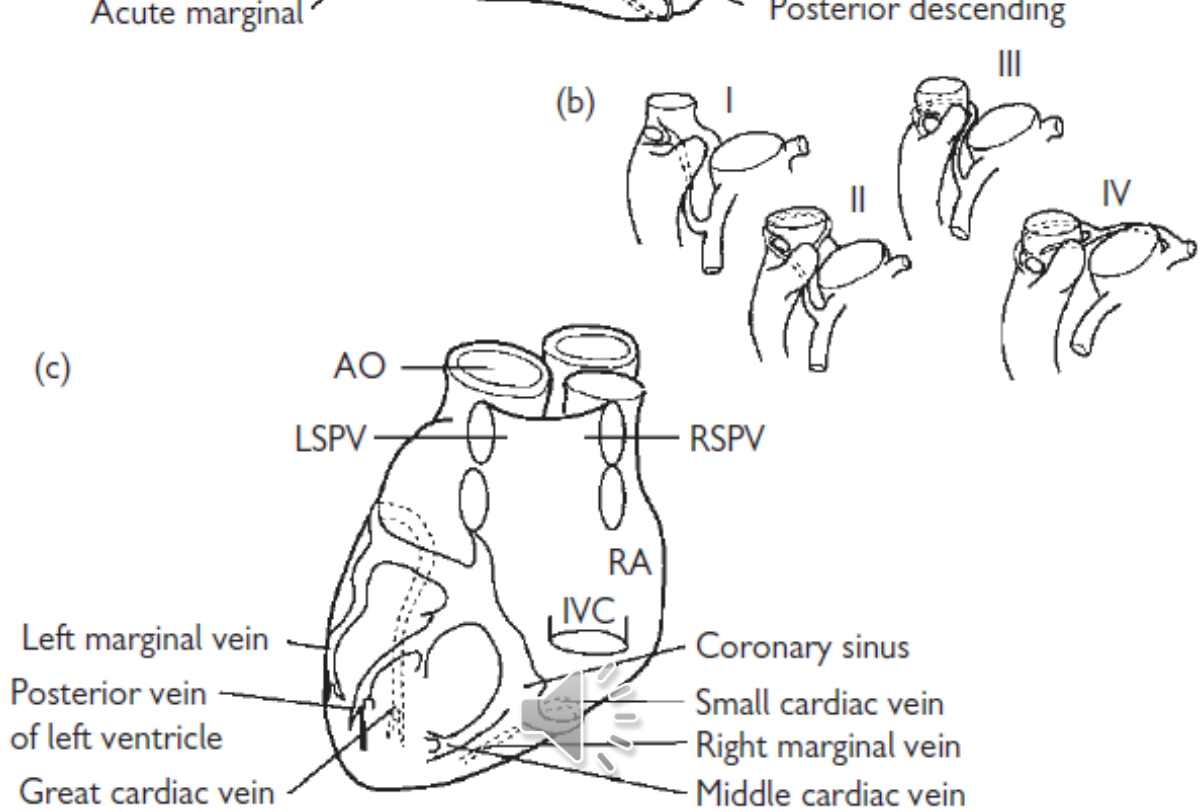


Fig. 2.6 (a) Coronary arteries. (b) Variations in the sinus node artery, which may arise from the right coronary artery (60%) and encircle the base of the superior vena cava in a clockwise (I), anticlockwise (II) or both (III) directions, or arise from the left coronary artery (40% of cases) (IV). (c) Coronary veins.

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:

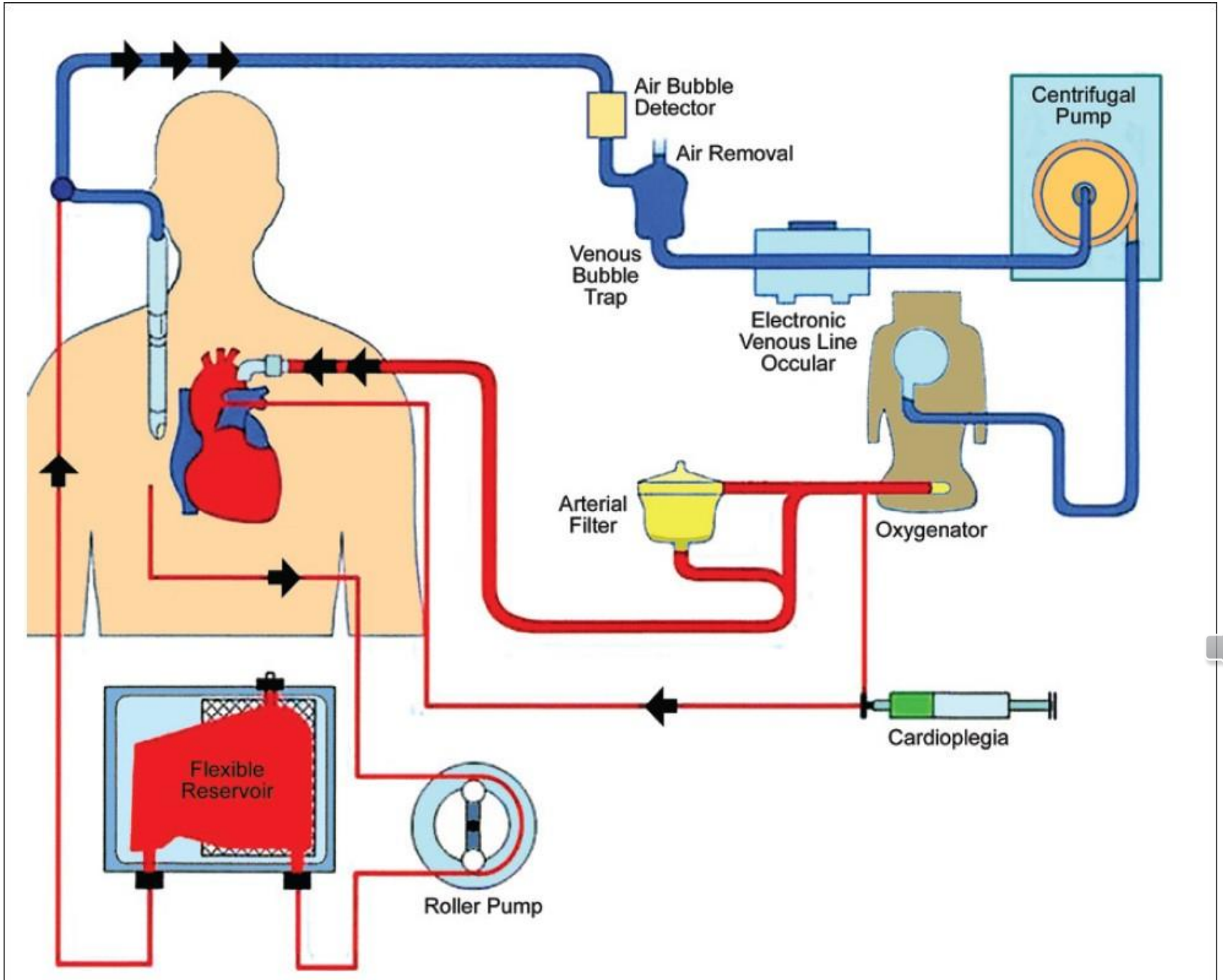
- Venous occlusion (an adjustable clamp on the venous line).
- Ports for drug and fluid infusions to the venous reservoir.
- Cardioplegia delivery system using a separate roller pump.
- Source of oxygen, air, CO2 and anesthetic gases.
- Sampling ports, and in-line blood gas and temperature monitors.
- 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 .

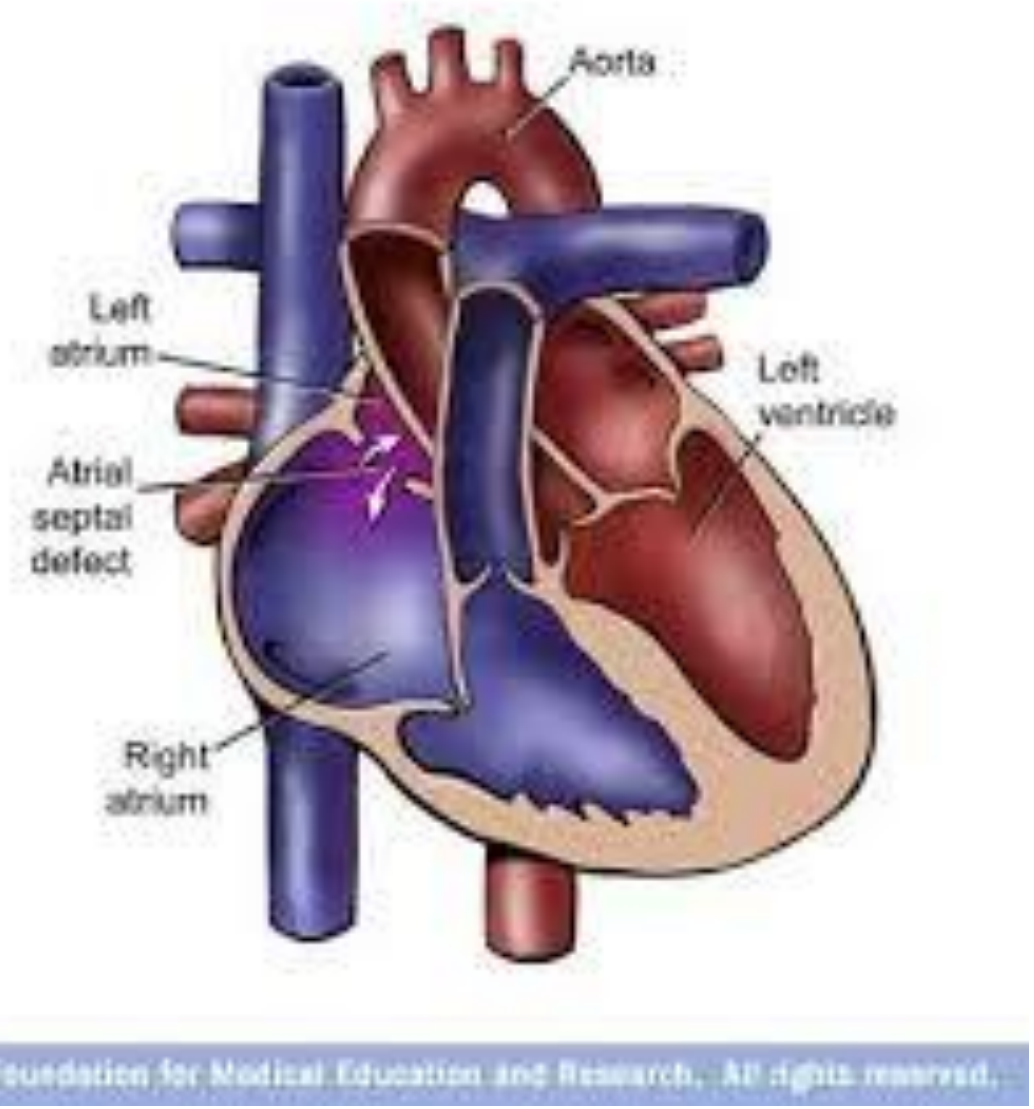
Coarctation of the aorta .

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.