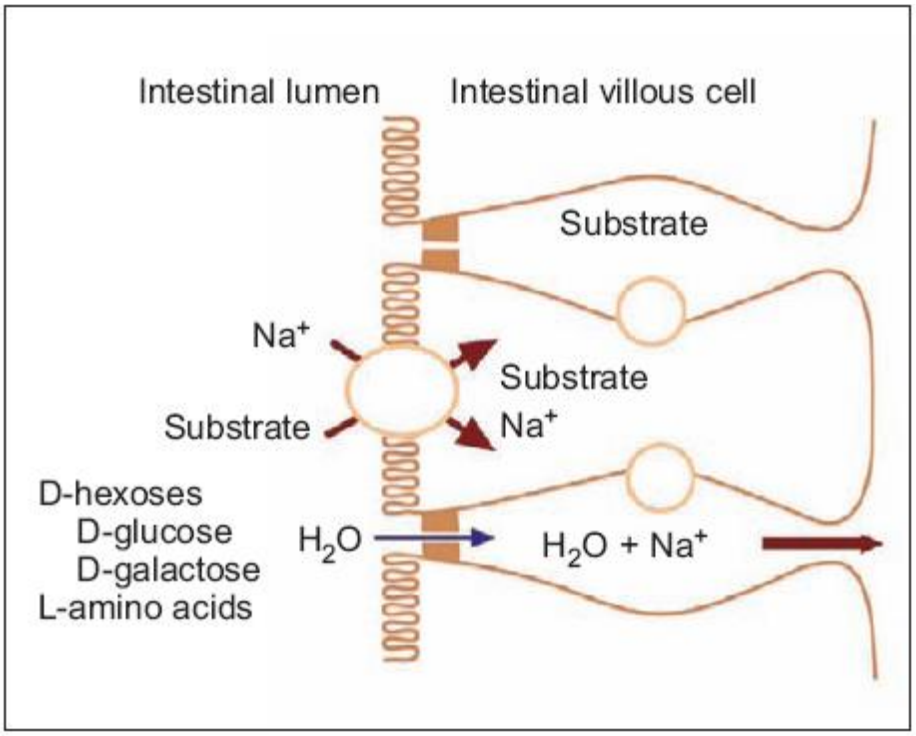
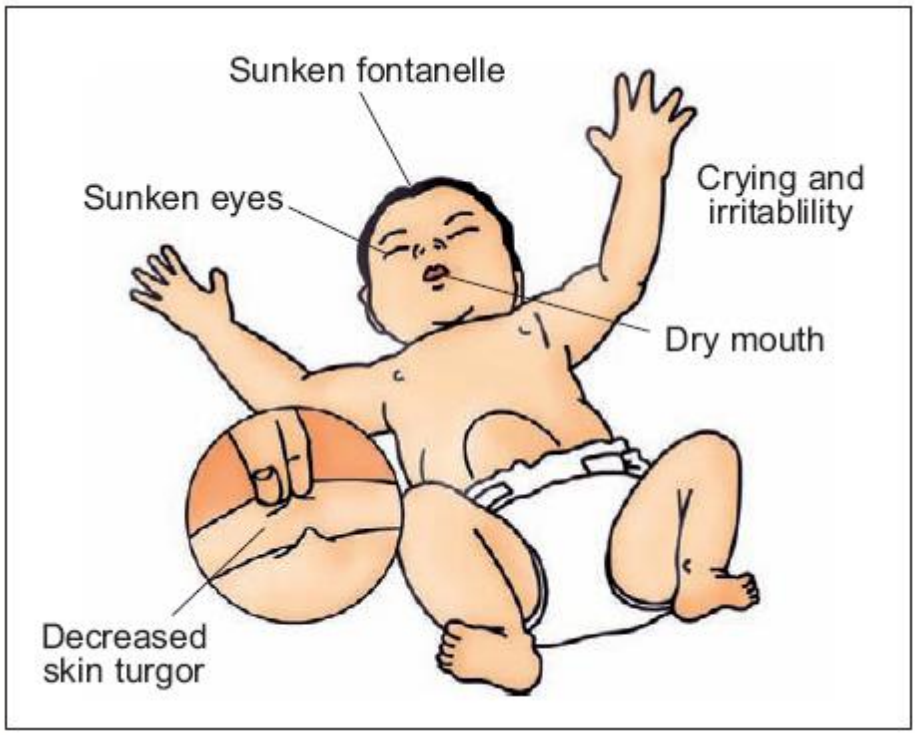
DRAW AND LABEL

Mechanism of action of ORS



3.4 Diagram to show the processes involved in cotransport of organic solutes and sodium and secondary water absorption.

SIGNS OF DEHYDRATION



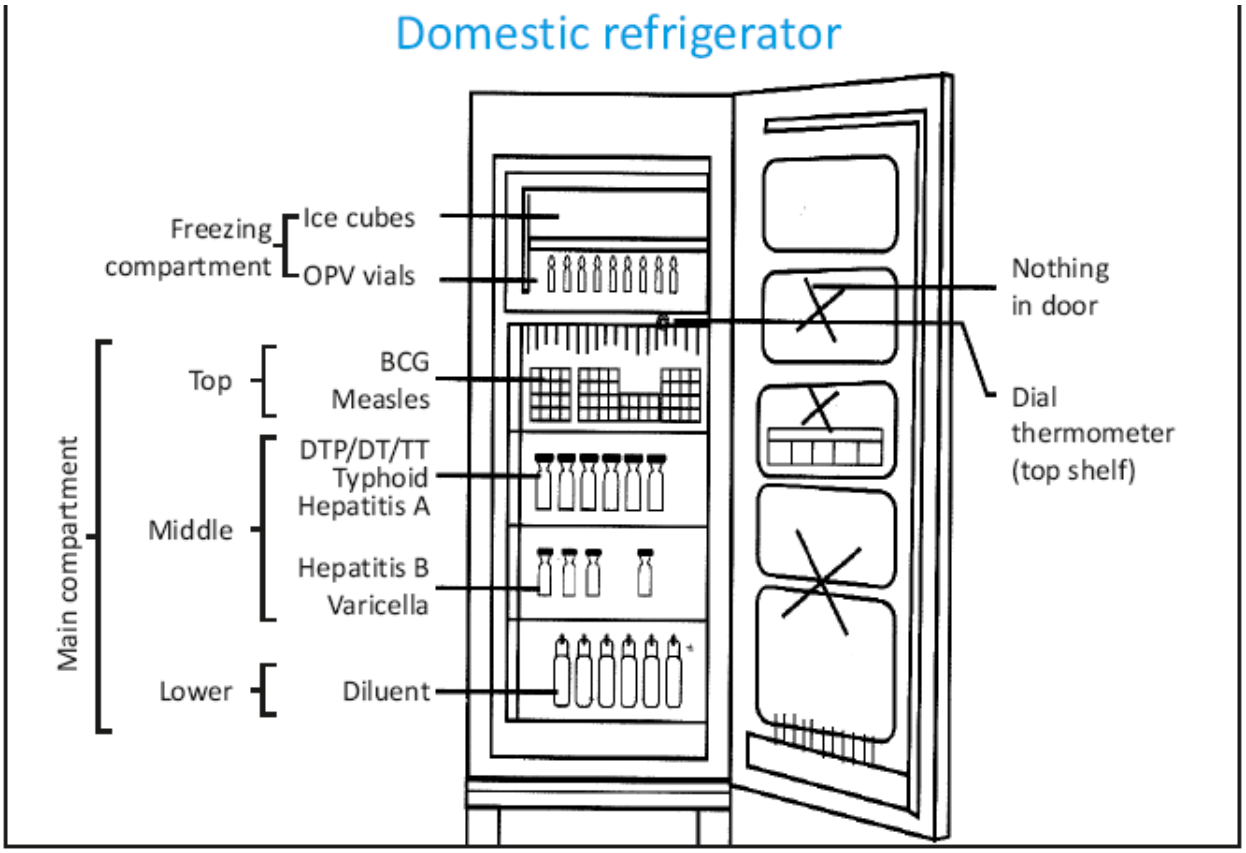
FREEZE WATCH

Box 2: Freeze watch indicators

A freeze watch indicator consists of a small vial of red liquid attached to a white card and covered in plastic. The vial breaks if the temperature where the indicator is located drops below 0° C for more than one hour.

These are very useful for cold sensitive vaccines like DTwP, DTaP, TT, DT, Td, TT, hepatitis B.

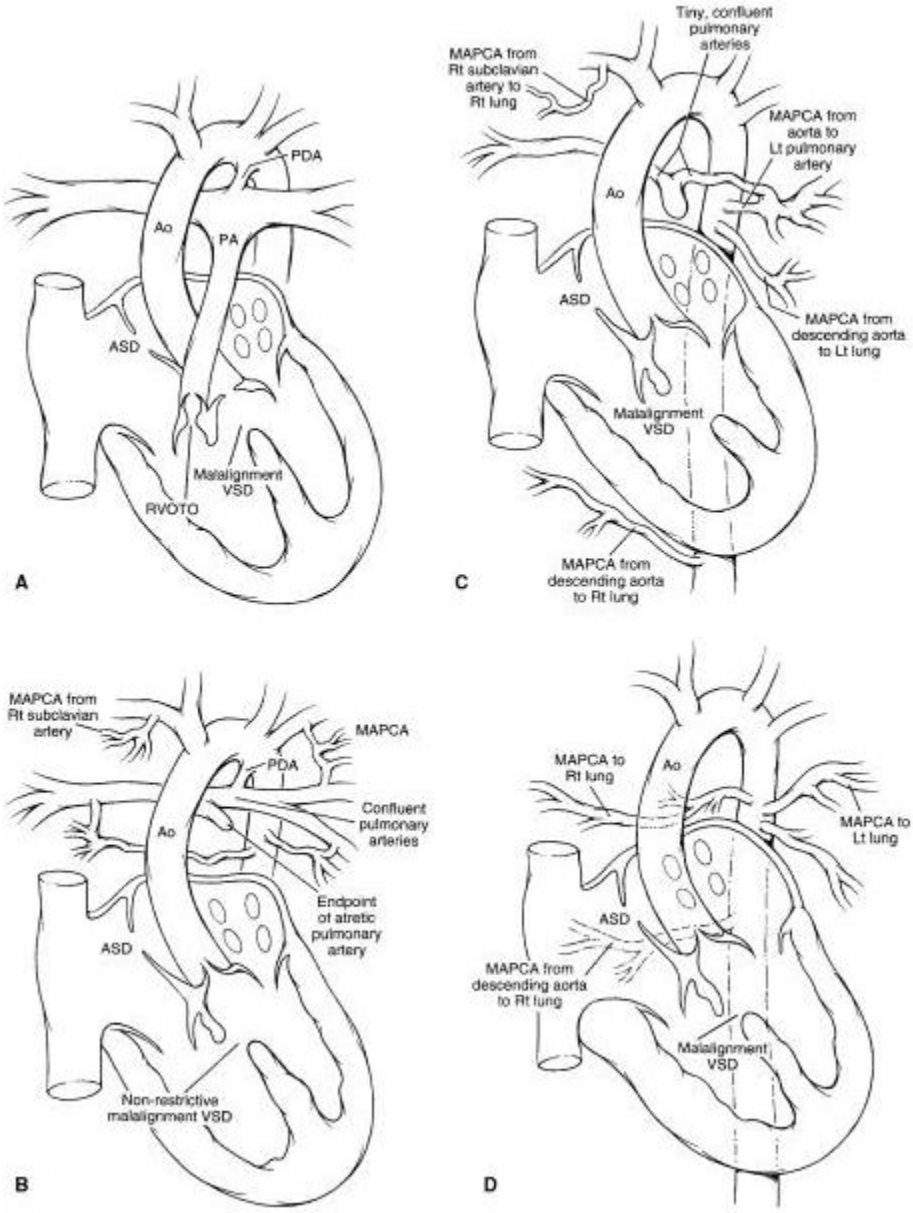
LABEL VACCINES IN ADOMESTIC REFRIGERATOR



ANATOMY OF TOF

, TOF. Note right ventricular outflow tract obstruction (RVOTO) at the infundibular level. The main pulmonary artery (PA) is small in comparison to the size of the aorta (Ao). RVOTO and the anterior malalignment ventricular septal defect allow desaturated blood to enter the systemic circulation. **B**, TOF with long-segment pulmonary atresia and confluent pulmonary arteries. The right and left PAs are confluent, but the main PA is atretic and discontinuous from the RVOT. Pulmonary blood flow is supplied in part via a patent ductus arteriosus (PDA). Major aortopulmonary collateral arteries (MAPCA) arising from the descending AO as well as right and left subclavian arteries also supply pulmonary blood flow. **C**, TOF with long-segment pulmonary atresia and tiny central pulmonary arteries with MAPCAs showing extensive arborization abnormalities. Note the absence of the PDA. **D**, TOF with pulmonary atresia, absent central PA

and extensive MAPCAs as the sole source of pulmonary blood flow. ASD, atrial septal defect; VSD, ventricular septal defect



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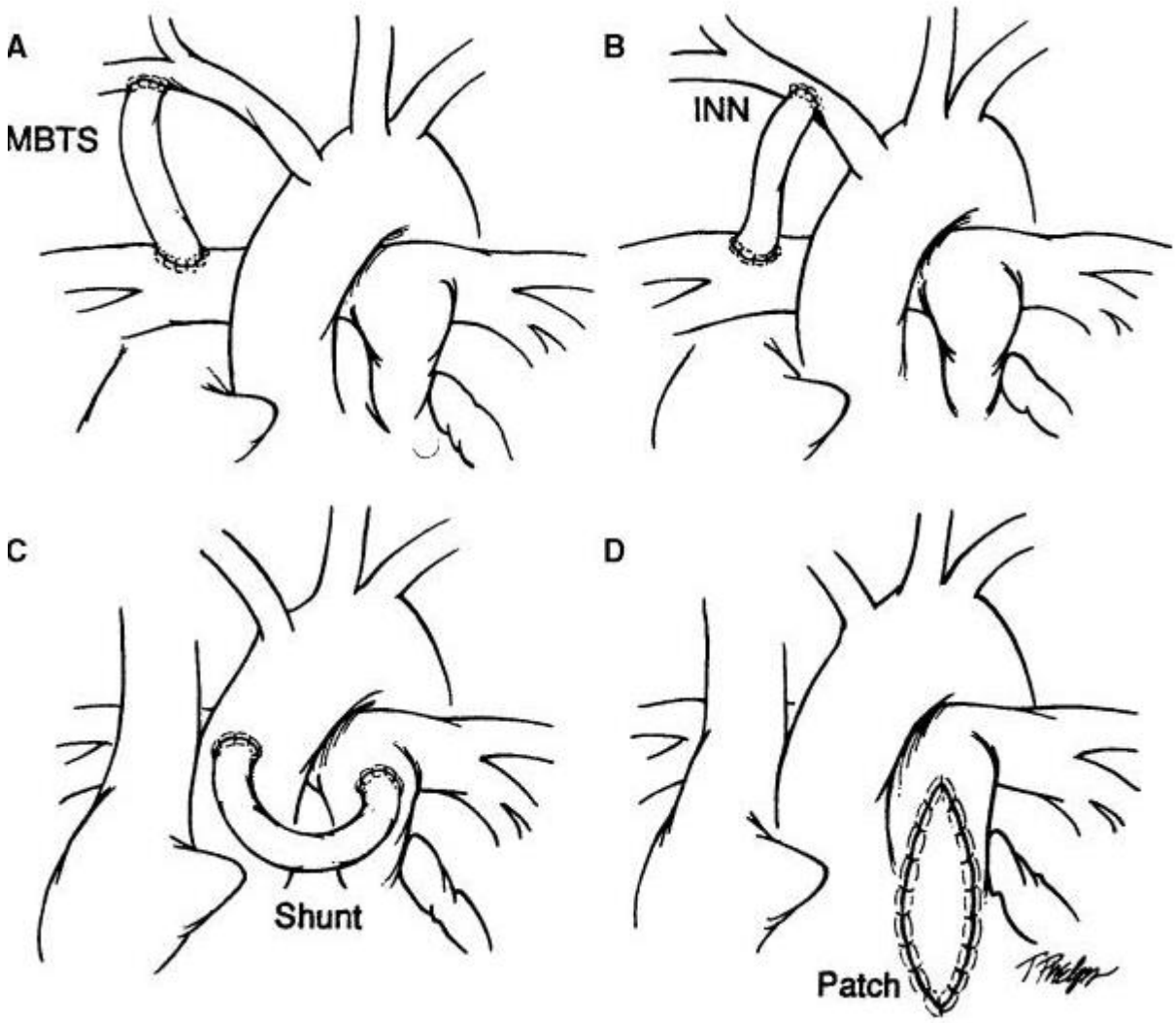
SHUNTS IN TOF



MURMUR IN TOF

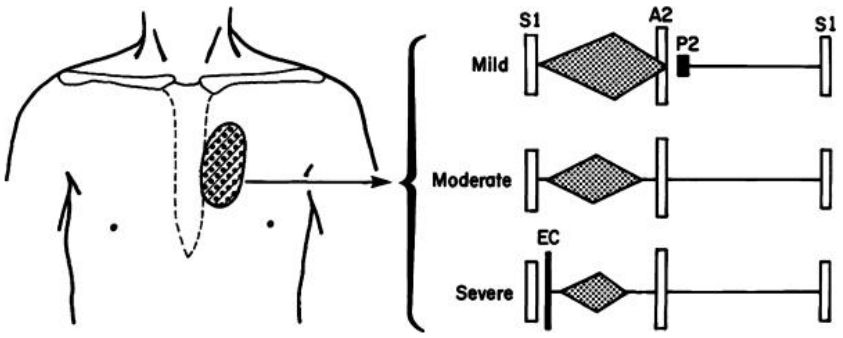
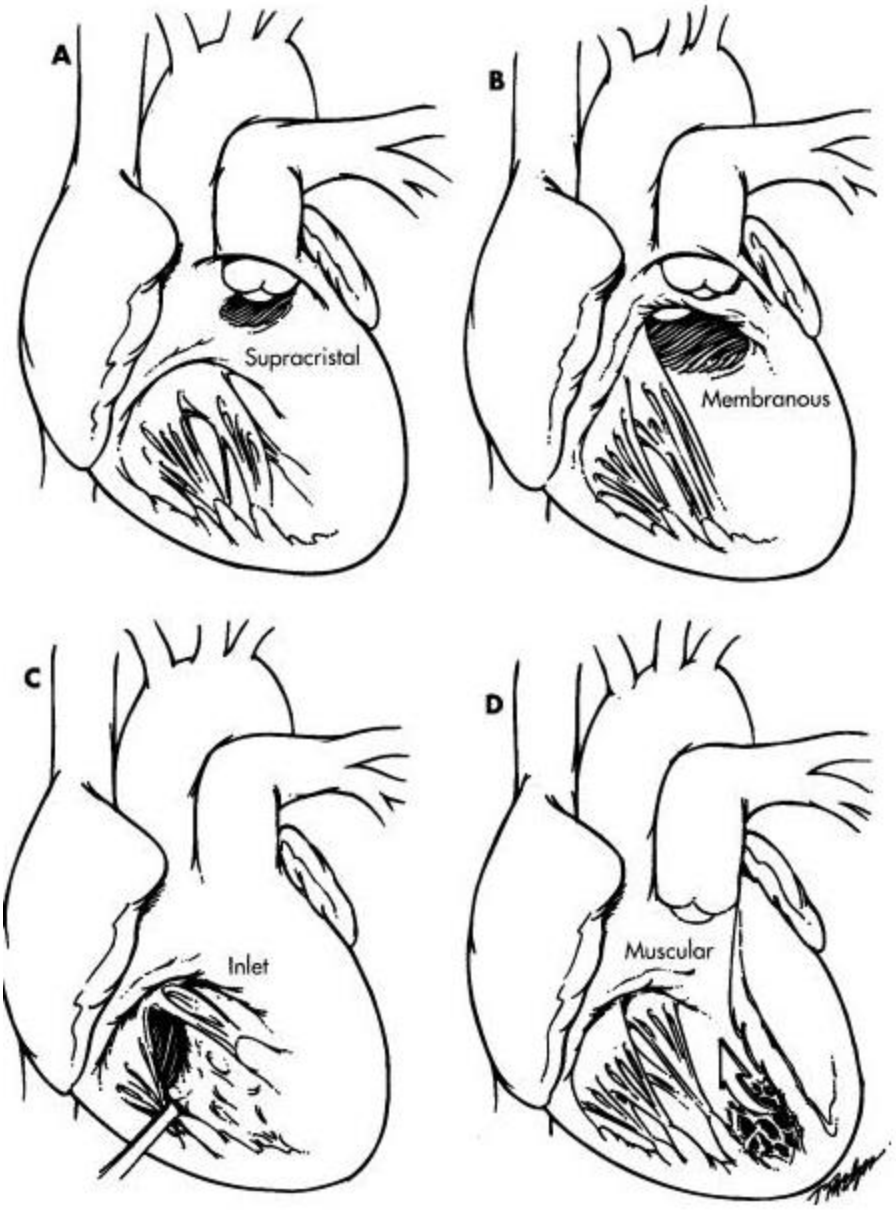


Figure 14-18 Cardiac findings in cyanotic tetralogy of Fallot (TOF). A long ejection systolic murmur at the upper and mid-left sternal border and a loud, single S2 are characteristic auscultatory findings of TOF. EC, ejection click.

TYPES OF VSD



Perimembranous (paramembranous) defects comprise up to 80% of all primary VSDs. They are also known as membranous or infracristal defects. They are located between the anterior and posterior divisions of the septal band and between the conal and trabecular interventricular septum. The lateral border is formed by the tricuspid annulus; the superior border is usually the aortic annulus. There may be a variable amount of muscular rim at the superior and lateral borders. The defect can extend into the inlet, trabecular, or outlet portions of the interventricular septum. Extension of the defect to the base of the noncoronary leaflet of the aortic valve may cause aortic regurgitation (AR). The conal septum may be anteriorly malaligned as in tetralogy of Fallot, causing right ventricular outflow tract obstruction, or, less commonly, it may be malaligned posteriorly, causing left ventricular outflow tract obstruction.

Subarterial defects, comprise 5% to 10% of VSDs, are also known as outlet, conal septal, supracristal, or subpulmonary VSDs. They are located beneath the pulmonary valve and their superior edge is a fibrous ridge between the two semilunar valves. They can be associated with prolapse of the right coronary leaflet of the aortic valve with associated regurgitation. This type of defect is more common in the Asian population.

Inlet defects account for 5% to 10% of VSDs and are also called atrioventricular canal-type defects. The posterior margin of the defect runs along the septal leaflet of the tricuspid valve to the anterior leaflet of the mitral valve, which often has a cleft. The defect extends superiorly to the membranous septum.

Muscular defects represent 5% to 10% of VSDs and are located anywhere in the muscular septum. The margins are characteristically muscular. They are frequently multiple. They may be anterior, mid-muscular, apical, or in the inlet septum. The latter differs from the inlet or atrioventricular canal-type VSD in that it is separated from the tricuspid valve and membranous septum by muscle tissue. Infundibular or outlet muscular VSDs differ from subarterial VSDs because of the presence of a rim of muscle separating the defect from the annuli of the aortic and pulmonary valves

VSD MURMUR

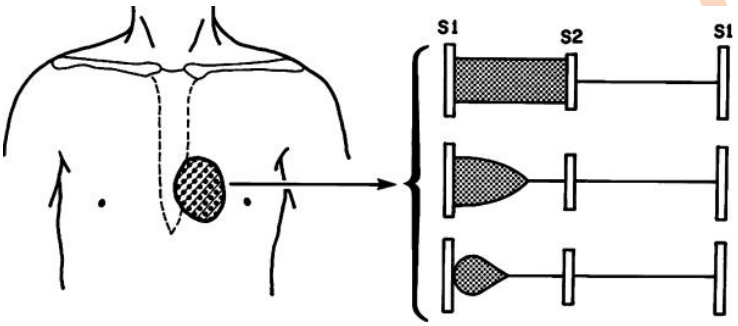
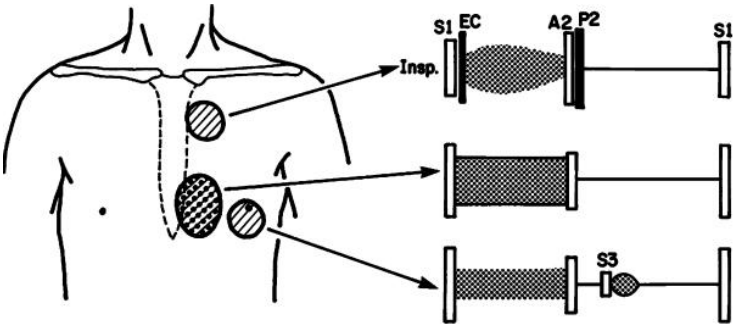


Figure 12-7 *Cardiac findings of a small ventricular septal defect. A regurgitant systolic murmur is best audible at the lower left sternal border; it may be holosystolic or less than holosystolic. Occasionally, the heart murmur is in early systole. A systolic thrill (dots) may be palpable at the lower left sternal border. The S2 splits normally, and the P2 is of normal intensity.*



PDA MURMUR

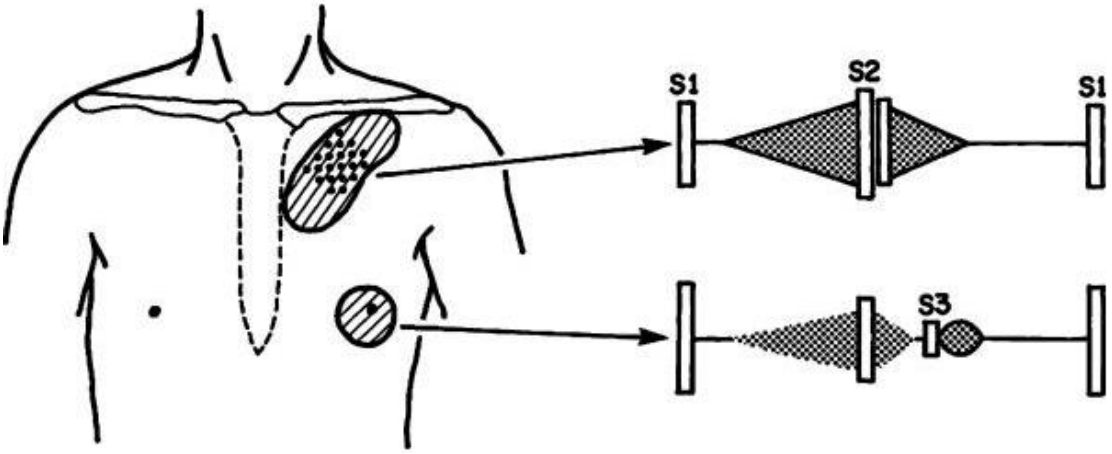


Figure 12-13 *Cardiac findings of patent ductus arteriosus. A systolic thrill may be present in the area shown by dots.*

ENDOCARDIAL CUSHION DEFECT

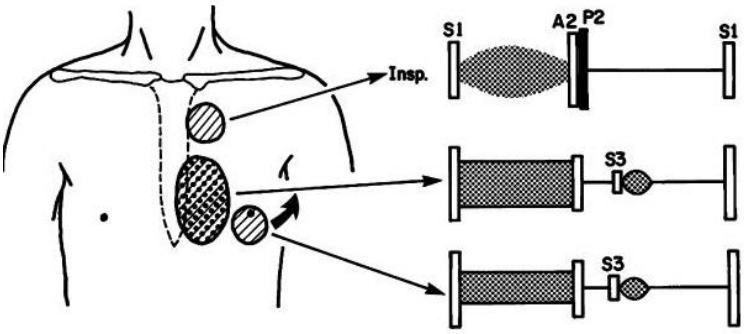


Figure 12-17 *Cardiac findings of complete endocardial cushion defect, which resemble those of a large ventricular septal defect. An apical holosystolic murmur (due to mitral regurgitation) may transmit toward the left axilla. A systolic thrill may be present at the lower left sternal border (dotted area), where the systolic murmur is loudest. Insp., inspiration.*

ASD MURMUR

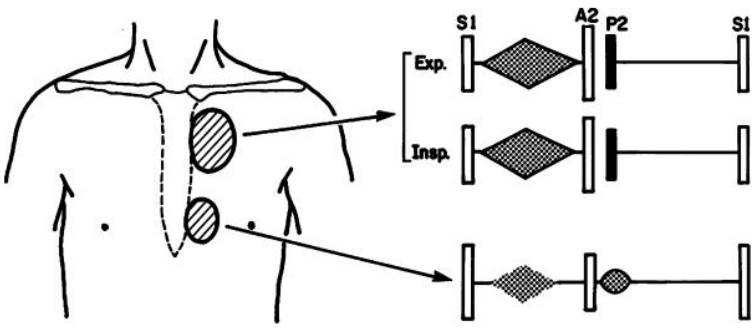


Figure 12-2 *Cardiac findings of atrial septal defect. Throughout this book, heart murmurs with solid borders are the primary murmurs, and those without solid borders are transmitted murmurs or those occurring occasionally. Abnormalities in heart sounds are shown in black. Exp., expiration; Insp., inspiration.*

RHEUMATIC MR

2. The S1 is normal or diminished. The S2 may split widely as a result of shortening of the LV ejection and early closure of the aortic valve. The S3 is commonly present and loud. The hallmark of MR is a regurgitant systolic murmur starting with S1, grade 2 to 4/6, at the apex, with good transmission to the left axilla (best demonstrated in the left decubitus position). A short, low-frequency diastolic rumble may be present at the apex (see Fig. 21-2).

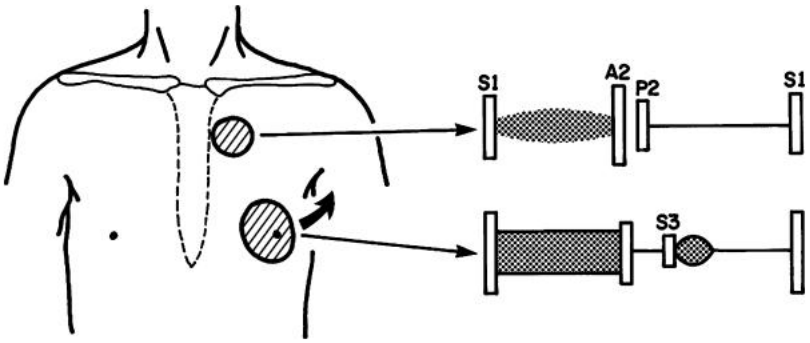
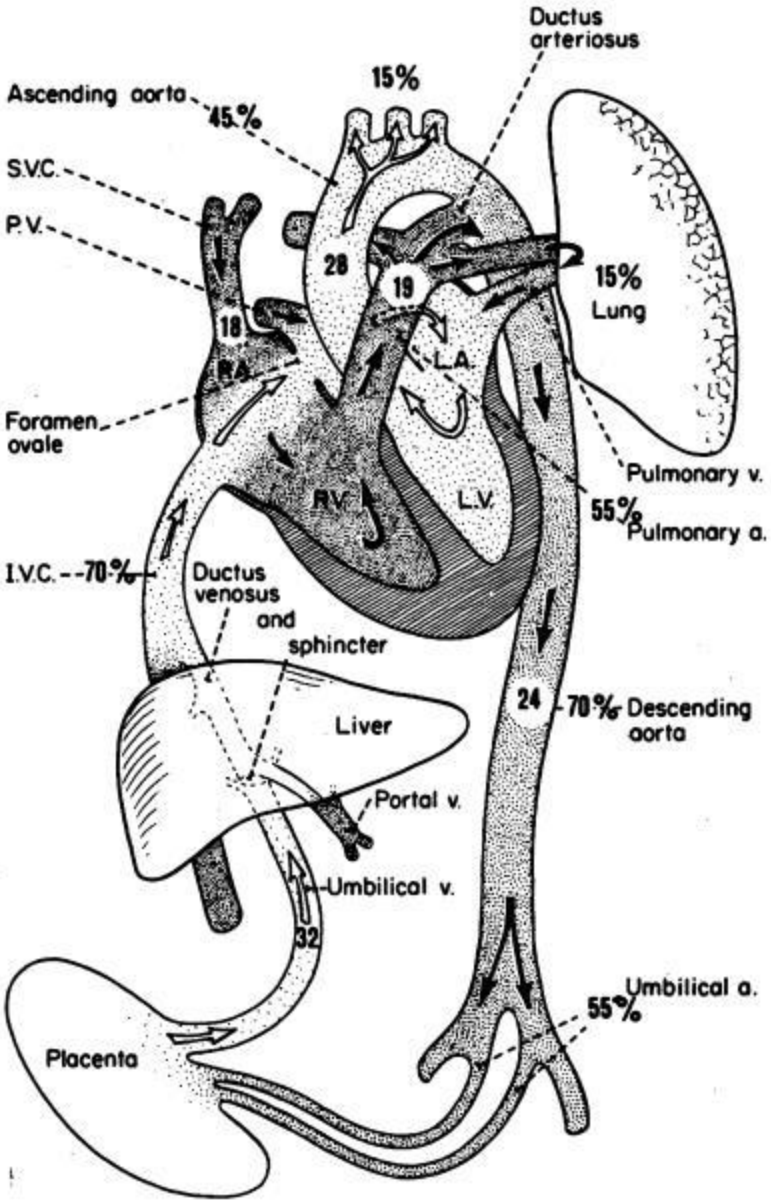


Figure 21-2 Cardiac findings of mitral regurgitation. Arrow near the apex indicates the direction of radiation of the murmur toward the left axilla.

FOETALCIRCULATION


Ductus arteriosus
15%
Ascending aorta
45%
S.V.C.
P.V.
28
19
15%
Lung
18
R.A.
L.A.
Foramen ovale
R.V.
L.V.
Pulmonary v.
55%
Pulmonary a.
I.V.C.
70%
Ductus venosus
and sphincter
24
70%
Descending aorta
Liver
Portal v.
Umbilical v.
32
Umbilical a.
55%
Placenta

STRUCTURES FORMING CARDIAC BORDER

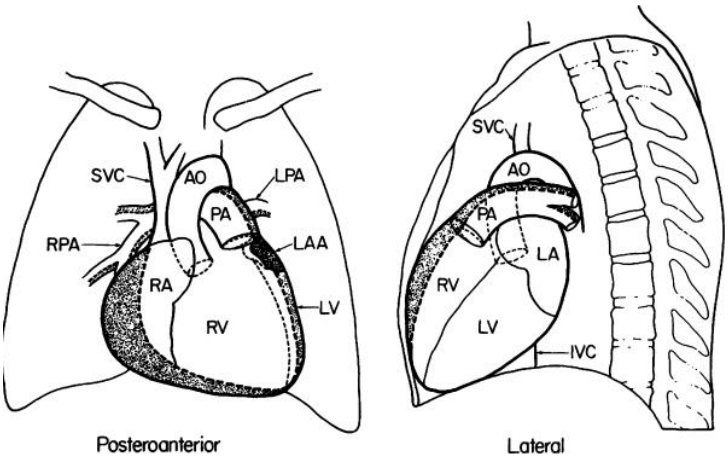


Figure 9-3 Posteroanterior and lateral view diagrams of chest roentgenograms. Enlargement of the right atrium (RA) and pulmonary artery (PA) segment and increased pulmonary vascular markings are present in the posteroanterior view. The right ventricular enlargement is best seen in the lateral view. AO, aorta; IVC, inferior vena cava; LA, left atrium; LAA, left atrial appendage; LPA, left pulmonary artery; LV, left ventricle; RPA, right pulmonary artery; RV, right ventricle; SVC, superior vena cava.

HYPOKALEMIA

Fig. 2.3 EKG changes of hypokalemia: Sinus rhythm. Normal axis. Narrow QRS complex. ST depression V3–V5. "Apparent" prolonged QT interval. T-waves unseparable from giant U-waves seen V2–6 (Webster A et al. Emerg Med J 2002; 19:74–77)

HYPERKALEMIA

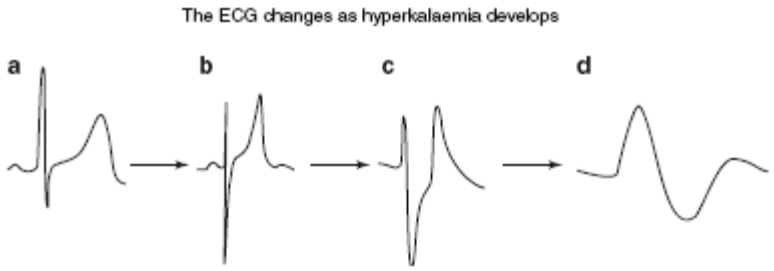
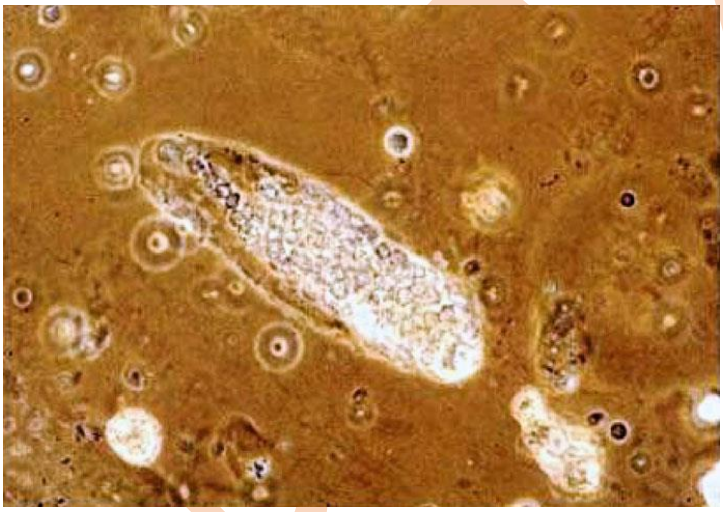
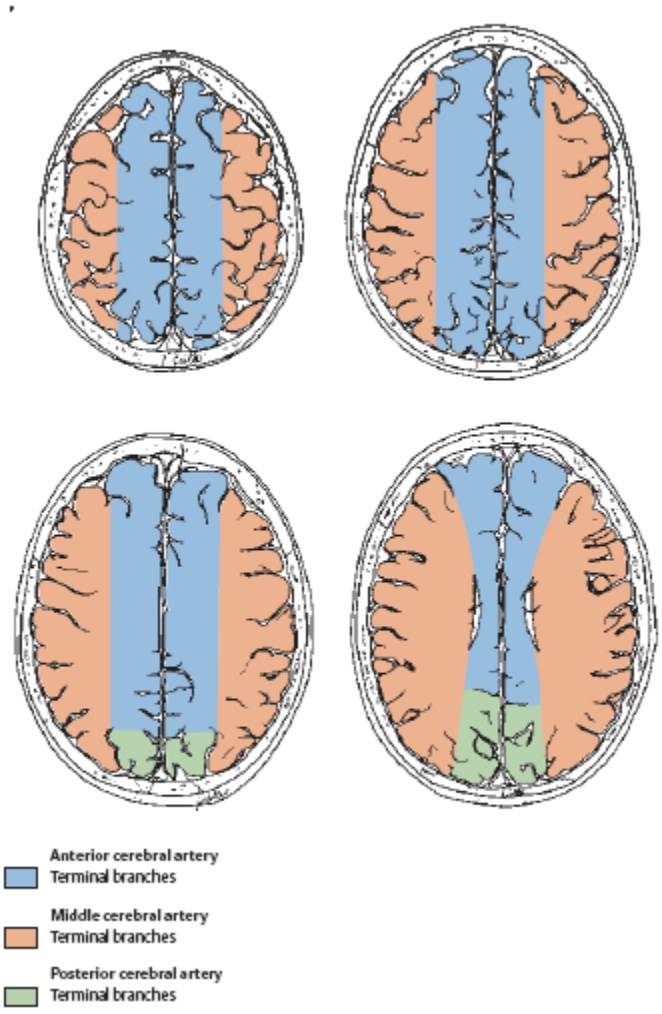


Fig. 2.4 The EKG changes as hyperkalaemia develops. (a) A normal complex. (b) Loss of P-waves, tenting of the T-waves. (c) Broadening of the QRS complex. (d) Sine wave appearance (Webster A et al. Emerg Med J 2002; 19:74–77)

RBC CAST



BLOOD SUPPLY BRAIN



BAG AND MASK PARTS

LIVER BIOPSY NEEDLE

BM NEEDLE

LP NEEDLE STRUCTURES PIERCED

RICKETS CHANGES

PRIMARY COMPLEX

ET TUBE

LARNGEAL MASK AIRWAY

PEFR

NEBULISER

MDI