Part I: Fundamentals

Ain Shams Lecture Notes in Cardiovascular & Thoracic Surgery

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To My Students

You will remember

some of what you hear,

much of what you read,

more of what you see,

and

almost all of what you experience and understand fully.

‘Happiness comes when you
believe in what you are doing,
know what you are doing,
and
love what you are doing’

Ezzeldin A. Mostafa, MD, PhD, MBA

Tell me.....and I will forget
Show me.....and I may remember
Involve me.....and I will understand

Confucius, 450 BC
# 1 Cardiovascular Anatomy

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Cardiovascular Surgical Anatomy

Unit Objectives

- To review the surgical anatomy of the cardiovascular system and surgical significance of each part.
- Structural functional relationships and review anatomy and histology and overall anatomy
- To review the anatomical correlation with the investigations like the EKG, ECHO (TTE & TEE), ANGIO, and different imaging studies to orient the surgeon’s perioperative management.
- To know my way to understand the embryology of the CVS and its clinic-physiologic classification of the congenital heart disease (CHD)

Learning Objectives:

By the end of this chapter the student should Know:

- Surface anatomy
- Cardiac chambers
- Right atrium
- Right ventricle
- Left ventricle
- Conduction system
- Cardiac valves
- Descriptive variables

Surface Anatomy

- Right atrium anterior and to the right of left atrium
- Left atrium a midline structure
- Right ventricle anterior and to the right of left ventricle
- Pulmonary artery anterior and to the left of aorta
- Coronary arteries on surface follow A-V groove and IVS

Cardiac Chambers

- **Right atrium**
  1) Wide based blunt appendage, crista terminalis separates trabeculated from non-trabeculated portion

- **Left atrium**
  1) Long, narrow appendage, smooth walls

- Right ventricle
  1) Coarsely trabeculated inlet/sinus, outlet portion

- **Left ventricle**
  1) Fine trabeculations inlet/sinus and outlet portions

What is a ventricle? Anderson RH, Mohun TJ, Moorman AF. Cardiol Young. 2011 Dec;21 Suppl 2:14-22. Review. PMID:22152524

On the basis of both developmental and morphological evidence, we would suggest that a ventricle is best defined as any chamber within the ventricular mass possessing an apical trabecular component. Such ventricles can be of R. or L. morphology, and always coexist. The ventricles are normally formed when possessing all three of the inlet, apical trabecular, and outlet components, but incomplete when lacking one or both of the inlet and outlet components. Ventricles that are incomplete because of lack of the inlet component are always hypoplastic, with incomplete RV being positioned antero-superiorly within the ventricular mass, and incomplete left ventricles located posterio-inferiorly. Patients having such incomplete ventricles because of the lack of the inlet component have FSV, although the functionally univentricular arrangement can also be produced in the setting of normally constituted but hypertrophied ventricles. Full analysis of ventricular morphology, therefore, requires attention not only to component make-up, but also size.
Right Atrium

- SVC - IVC
- Crista terminalis
- Coronary sinus
- Tricuspid valve
- Fossa ovalis
- Triangle of Koch
- Tendon of Todaro
- Inferior isthmus

Triangle of Koch FIGURE 1–6.

A. Location: defined by the following structures:
   1. The ostium of the CS, posteriorly;
   2. The anterior portion of the TV annulus; and
   3. The tendon of Todaro (a tendinous structure connecting the valve of the IVC ostium to the central fibrous body), posteriorly.

Importance in cardiovascular diseases: used as an anatomical landmark for location of the AVN during EPS procedures such as pacing or ablation. VSD closure.

Right Ventricle

- Inlet portion supports tricuspid valve
- Trabecular sinus portion (main body of the RV)
  1) Moderator band
  2) Medial papillary muscle (of conus)
- Outlet portion
  1) Infundibular (Conal) septum (separates semilunar valves)
  2) Crista supraventricularis - separates sinus (chamber) from outlet portion of the ventricle
  3) Septal band (trabecula septomarginalis)
  4) Parietal band (ventriculo-infundibular fold)
  5) Pulmonary valve

Left Ventricle

- Thick wall
- Inlet portion supports mitral valve
- Anterior and posterior papillary muscles
- Outlet portion beneath aortic valve

Conduction System

- Sinoatrial node - anterolateral RA
- Interatrial conduction pathways - not well defined and somewhat controversial
- Atrioventricular node - triangle of Koch
- Bundle of His - AV node to membranous septum, usually located on the inferior/posterior wall of the membranous septum
- Left bundle branch - left ventricular septal surface into multiple branches
- Right bundle branch - below medial papillary muscle via septal and moderator bands to anterior papillary muscle
- Inferior isthmus (right atrium)
- Bachman’s bundle (left atrium)

Ventricular Band (Torrent-Guasp)

- Biventricular myocardial band extending from pulmonary artery to aorta
- Two loops: basal and apical
- Double helix derived from spiral fold
- Apex has figure-8 configuration

See Arrhythmia - Tachycardia
See Arrhythmia - Bradycardia
Cardiac Valves

- **Aortic valve** wedged between mitral and tricuspid, pulmonary valve separated
- **Mitral valve**
  1) Anterior leaflet wide, short, 1/3 of annular circumference
  2) Posterior leaflet narrow, long, 2/3 of annular circumference
  3) Papillary muscles and chordae tendineae
- **Tricuspid valve**
  1) Anterior, posterior, septal leaflets
- **Aortic and pulmonary valves**
  1) 3 cusp, semilunar
  2) Sinuses of Valsalva
  3) Nodulus Aranti and lunulae

Left Ventricular Outflow Tract

- Semilunar aortic valve
- Fibrous annulus is not a ring
- Interleaflet triangles
- Aortoventricular junction
- Sinuses of Valsalva
- Sinotubular junction (sinus rim) = junction of sinus of Valsalva and ascending aorta
- Posterior commissure relates to mid point of anterior leaflet of mitral valve

Coronary Arteries

- **Right (RCA) and Left coronary (LCA) arteries** originate from proximal aorta via respective ostia.
- **Dominant pattern** determined by origin of posterior descending
- **Balanced pattern** occurs when there is no particular dominance
- **Septal blood supply** 2/3 left anterior descending, 1/3 posterior descending
- **Sinus node artery** from RCA - 55%
- **AV node artery** from U bend at crux, just beyond the takeoff of the PDA if circulation is right dominant

**The 4 optimal views for LCA FIGURE 1–19.**

- **R.A.O. 30°**
  - It permits the entire circumflex system to be studied as well as the first part of the LAD.
- **L.A.O. 55/60°**
  - Mainly studies the diagonal arteries and the mid and distal parts of the LAD.
  - On the other hand the circumflex is not well defined.

**L.A.O. 55/60° + 20° cranial or caudal projection.**

The coronary circulation consists of coronary arteries and veins

**Optimal View(s) FIGURE 1–20.**

**The 2 optimal views for RCA**

- **L.A.O. 45° + 15° caudal angulation**
  - This projection allow the whole study of the R.C.A. and clearly defines the region of the crux of the heart.
- **R.A.O. at 45°**
  - It permits the survey of the 2nd (vertical) segment of the RCA, the PDA and the collateral branches (right ventricular and right marginal arteries).
**Surgical Anatomy of The Mitral Valve**

The mitral apparatus includes the leaflets, annulus, chordae tendineae, papillary muscles, and left ventricle.

### A. Leaflets
- The mitral valve has two leaflets, the anterior (aortic) and posterior (mural) leaflets.
- The leaflets are attached directly to the mitral annulus and to the papillary muscles by primary and secondary chordae.

#### 1. Anterior mitral leaflet:
- Is in direct continuity with the fibrous skeleton of the heart.
- This leaflet is contiguous with the left and noncoronary cusps of the aortic valve and the area beneath the intervening aortic commissure, termed the fibrous subaortic curtain.
- Although the anterior leaflet occupies only 35% to 45% of the annular circumference, its leaflet area is almost identical to that of the posterior leaflet.

#### 2. Posterior Leaflet:
- Has two variable indentations or clefts that divide the posterior leaflet into three scallops: the largest or middle scallop, the posteromedial scallop, and the anterolateral scallop.
- Fan-shaped chordae insert into and define the clefts between the individual posterior scallops.
- Motion of the posterior leaflet is more restricted than that of the anterior leaflet; however, both mitral leaflets contribute importantly to effective valve closure.

The surface of the mitral leaflet is divided into three zones corresponding to areas of chordal insertion and leaflet coaptation.

1. **The rough zone**: is the leading edge of the anterior and posterior mitral leaflets. This zone is the contact surface of the mitral leaflets during systole.
2. **The clear zone**: is peripheral to the rough zone and represents most of the body of the leaflet; this portion of the mitral valve billows into the atrium during ventricular contraction.
3. **The basal zone**: between the clear zone and the annulus, receives the insertion of the basal chordae tendineae (tertiary chordae), which originate directly from the trabeculae of the left ventricle. The basal zone is found only on the posterior leaflet.

### B. Annulus
- The mitral annulus is the site of leaflet attachment to muscular fibers of the atrium and ventricle.
- The annulus is flexible and decreases in diameter during each systolic contraction by approximately 26%.
- The orifice of the mitral valve also changes shape, from elliptical during ventricular systole to circular during late diastole. This flexibility increases leaflet coaptation during systole and maximizes orifice area during diastole.
- Anteriorly, the annulus is attached to the fibrous skeleton of the heart. This limits its flexibility and its capacity to dilate with mitral regurgitation (MR). The posterior annulus is more flexible and is not attached to rigid surrounding structures. This accounts for the clinical observation that dilation of the annulus occurs posteriorly with MR.

**Important Anatomic Landmarks:**
- The LCX courses laterally around the mitral annulus in the posterior AV groove.
- The coronary sinus runs more medially in the same groove.
- The artery to the AVN, usually a branch of the right coronary artery, runs a course parallel and close to the annulus of the AML near the posteromedial commissure.
- The aortic valve is situated between the anterior and posterior fibrous trigones. The bundle of His is located near the posterior trigone.

The Mitral Apparatus (Complex)

**Figure 1-11.**

- B. Posterior LA wall.
- C. Annulus.
- D. Leaflets.
- E. Chordae tendineae.
- F. Papillary muscles.
- G. LV wall.
C. Chordae Tendineae

- The chordae tendineae are chords of fibrous connective tissue that attach the mitral leaflets to either the papillary muscles or the left ventricular free wall.
- They often subdivide and interconnect before they attach to the leaflets. The chordae are divided into:
  - **Primary chordae**: attach directly to the fibrous band running along the free edge of the leaflets. These chordae ensure that the contact surfaces (rough zone) of the leaflets coapt without leaflet prolapse or flail.
  - **Secondary chordae**: attach to the ventricular surface of the leaflets at the junction between the rough and clear zones. These chordae contribute to ventricular function. Secondary chordae enable the ventricle to contract in an efficient cone-shaped fashion; when secondary chordae are excised, the left ventricle assumes a globular shape.
  - **Tertiary chordae**: are unique to the posterior leaflet. They arise as strands directly from the left ventricular wall or from small trabeculae to insert into the ventricular surface of the leaflet near the annulus.

D. Papillary Muscles

- The anterolateral and posteromedial papillary muscles each supply chordae tendineae to both leaflets.
- The two groups of papillary muscles subtend the anterolateral and posteromedial commissures and arise from the junction of the apical and middle thirds of the ventricular wall.
- The anterolateral papillary muscle receives a dual blood supply from the anterior descending coronary artery and either a diagonal branch or a marginal branch of the left circumflex artery.
- The posteromedial papillary muscle receives its blood supply from either the left circumflex artery or a distal branch of the right coronary artery.
- Because of the single blood supply to the posteromedial papillary muscle, infarction of the posteromedial papillary muscle is much more common.

E. Left Ventricle

- The posterior left ventricular wall and papillary muscles play an important role in leaflet coaptation and valve competence.
- Papillary muscles are aligned parallel to the ventricular wall and attach via chordae to the free edges of the valve leaflets. These muscles project from the trabeculae and may be single, bifid, or a row of muscles arising from the ventricular wall.
- During isovolumetric contraction the mitral leaflets are pulled downward and together by this interaction. Ventricular dilatation may affect the alignment and tension on the papillary muscles and valve competence.
FIGURE 1−14. The anatomy of the aortoventricular continuity (aortic annulus) and different surgical techniques of aortic annular enlargement (Nicks, Manuagian and Konno techniques).
The Surgical Anatomy of the Aortic Valve

The aortic valve is a structure composed of leaflets supported in a connective tissue tube. It has been defined as a thin mobile layer of tissue located in a canal allowing free flow through the passage way and preventing reflux. Nonetheless, the structures connected by and supporting the leaflets affects both its structure and function. This is significance, because studies on the dimensional changes of the aortic root during each cardiac cycle reveal that the sinuses of valsalva account for significant variations during opening and closing of the valve. **FIGURE 1−12.**

Accordingly, the sinuses have been suggested to play a more active part in the function of the valve than has been previously thought. Thus, the aortic valve sinuses along with the triangles of fibrosis tissue interposed between the sinuses in the subvalvular ventricular out flow tract must be considered as constituent parts of the aortic valve. The normal aortic valve (or root) can be considered as a complex structure made up of several components, namely the leaflets, the aortic sinuses and the fibrous interleaflet triangles of the subaortic outflow tract, “fibrosis skeleton”.

*The aortic sinuses or sinuses of the Valsalva:*

The sinus of valsalva is the space which exists between aortic surface of an aortic cusp and the wall of aorta behind that cusp. It is bounded caudally by basal attachment of the aortic cusp and above by the sinotubular junction, this space serves as a reservoir of the blood during ventricular diastole. The origin of the coronary arteries is the base of the nomenclature for the sinuses and cusps. The ostia of the right and left coronary artekies identify the right and left sinuses and cusps, the third sinus and cusp without an associated coronary artery are named non-coronary. **FIGURE 1−13.**

Some investigators used the term right, left, and posterior for the cusps and the sinuses were correspondingly named. This is because the term non coronary cusp or sinus is best avoided as a coronary artery may originate from the wall of the aorta behind the posterior cusp or a coronary ostium may be absent from one or both anteriorly located cusps. **FIGURE 1−13.**

*Relations of the sinuses:*

---

*Image 1−12:

**Anatomic ventriculo-aortic junction**

- Sinotubular junction
- Crown-like semilunar attachments
- Virtual basal ring
The R. coronary sinus: It is related to the RV infundibulum and its lowermost attachment is to the muscular part of the ventricular septum at the union of its infundibular and muscular parts. Above this level, the infundibular septum, with the parietal limb of the crista supraventricularis, lies against the outer wall of the R. aortic sinus with a wedge of epicardium separating the two. The R. coronary arises from this sinus at a point level with the free edge of the leaflet. At times, the orifice is close to one of the commissures which constitute a point of surgical interest. Also, of interest is the fact that the ostium of the RCA may be located above the sinus in the tubular portion of aorta in some cases. Another important point, is that, the circumflex, the conus or left coronary artery may originate from R. coronary sinus.

The posterior aortic sinus: The aortic origin at the posterior sinus has two main attachment, the R. \( \frac{1}{3} \) and L. \( \frac{2}{3} \).

The R. \( \frac{1}{2} \) of the aortic origin at the posterior sinus is attached to the membranous portion of the ventricular septum. In this way, the sinus lies above the septal leaflet of the tricuspid valve and against the atrial septum. The left two thirds of the posterior aortic sinus in common, with the posterior one third of the left sinus, exhibits attachments which vary greatly from the remainder of the aortic root, it is related and connected to the left ventricle indirectly via anterior mitral leaflet. This part of the aorta is connected with the anterior mitral leaflets via an intervening thin layer of connective tissue, so called “the mitral-aortic intervalvular fibrosa”.

The L. aortic sinus: The most posterior \( \frac{1}{3} \) of the left aortic sinus has the same relationship as does the adjacent part of the posterior sinus. The central part of the L. aortic sinus lies against the epicardium and below the origin of the LCA. The R. most \( \frac{1}{2} \) of the L. sinus is related to L. pulmonary sinus. The LCA arises from the centre of the aortic sinus and in some cases may arise from above the level of the sinus in the tubular portion of the aortic root which can be taken to represent the “ring” or “annulus”, there is a series of three fibrous triangles separating the sinuses on their ventricular aspect. These areas of fibrous tissue have their apices at the attachment of the commissures to the aortic wall and their

The Annulus or the fibrous “skeleton” interleaflets triangles: FIGURE 1–14.

For the echocardiographist, the aortic annulus corresponds to the plane passing through the nadir of the leaflet hinge-lines.

For the surgeon, it generally corresponds to the leaflet hinge-lines, also called the surgical annulus, onto which the prosthetic valve is sewn.

For the anatomist, the VAJ is where the ventricular myocardium terminates and gives way to the wall of aortic sleeve.

The aortic cusps (leaflets) FIGURE 1–14.

The aortic valve is composed of 3 semilunar cusps, the superior edge of each cusp is unattached, the remaining edges of the cusps are attached to the aortic wall. The 3 cusps are oriented in R. anterior, L. anterior and posterior position and are usually named the R., L. and posterior cusps.

The hinge: The part of the valve below this line separates aortic cavity from LV cavity, while on closure, the part of the valve above this line is called lunula and overlies the neighbouring cusp and serve as supporting strut. The lunula are sometimes fenestrated near the commissure. The leaflets overlap in their closed position, as the cross-sectional area of the aortic root is less than the sum of areas of the leaflets. The closure line produced by opposition of the adjacent leaflets is visible on their ventricular surface 2 to 3 mm from free edge.
bases toward the proximal border of the leaflets. The interleaflet triangles complement the semilunar shape of the sinuses, these triangles together with leaflets were considered to compose the arterial root. From the surgical view, the apices of the triangles separate the LVOT from outside of the heart, since tips of the commissures are attached to the aortic wall. The triangle between the R. and L. coronary sinuses faces pulmonary valve and has its base on the septal component of the RVOT, its apex points to the tissue space between the arterial trunk.

The triangle between the RCS and NCS faces the RA and being in direct continuity with the membranous septum, and is closely related to the septal leaflets of the TV. Also, apex communicates with the transverse sinus above the supraventricular crest of the RV.

The triangle between the LCS and NCS is in direct continuity inferiorly with the aortic leaflets of mitral valve, its apex points directly into the transverse sinus in front of the LA. C- it is these triangle which separate and mark the presence of three sinuses in the normal valve.

The coating surfaces: **FIGURE 1–14.**

The commissures: The 3 cusps are oriented in R. anterior, L. anterior and posterior position and are usually named the R., L. and posterior cusps, the junctional zone between two adjacent cusps is known as commissure. At the commissure there is some thickening of the aorta called **commissural mound**, into which each cusp inserts. The commissures are three in number, each named for two cusps which form it, thus, the commissure of the aortic valve are R. posterior, L. posterior and R. left.
### Chest X-Ray (CXR)

#### Cardiovascular Silhouette

- **Mediastinal Border**
  1) Right atrium
  2) Superior vena cava
- **Left Border**
  1) Aortic arch
  2) Pulmonary trunk
  3) Left atrial appendage
  4) Left ventricle

**Mnemonic:** How to read the plain CXR

“Some Bastard Pinched My Toy Dinosaur”

“Some Body Pinched My Toy Dinosaur”

- **S**—Soft tissue
- **B**—Bones—clavicles, ribs
- **P**—Pleura, Pulmonary Parenchyma
- **M**—Mediastinum
- **T**—Thorax, Tubes, Lines
- **D**—Diaphragm

---

### Basic Echocardiography (ECHO)

The cardiac segments (atria, ventricles & great arteries) are analogous to a train. One can readily recognize 3 components (engine or great arteries, cabin or ventricles, and caboose or atria). It is essential to know how the various segments are aligned and connected.

#### Descriptive Variables

**Situs of thoracic viscera and atria**

1) This is best identified from the bronchial anatomy (3 bronchi on the right, 2 on the left)
2) Solitus, inversus, ambiguous

**Situs of ventricles**

1) Usual, concordant, D-loop, right-handedness
2) Inverted, discordant, L-loop, left-handedness

**Dominance of ventricles**

1) Balanced (usual), right (left small), left (right small)

**Cardiac connections**

1) Atroventricular and ventriculoarterial
2) Concordant or discordant (transposed)

**Cardiac and arterial position**

1) Cardiac apex; levo-, dextro-, mesocardia
2) Great arteries; transposition, malposition
3) The patient can have completely normal cardiac structures and still have dextrocardia - this only refers to the position of the cardiac apex

Conventional diagnosis; e.g., TOF
FIGURE 1–49. TEE. Summary of 20 Basic Views.
Developmental Anatomy of the CV System

Basic Principles (Remember # 3)

- Blood circulation by 3 weeks (21 days)
- Heart develops 3-8 weeks
- Critical period for anomalies 3-6 weeks

A. Cardiovascular system is first functional system in embryo
B. Blood circulation by 3 weeks (21 days)
C. Heart develops 3-8 weeks
D. Critical period for anomalies 3-6 weeks

Heart Development

A. Endocardial tubes fuse to form heart tube (21 days)
B. Heart begins to beat (22 days)
C. Heart folding - 21-22 days, folding - 23-28 days
   1) D loop, L loop
   2) Bulboventricular loop --- future ventricles
   3) Cellular differentiation
   4) Bulbus cordis - conus cordis --- RVOT
   5) Truncus arteriosus --- great vessels

Atrial Septation

A. Septum primum forms from roof of atrium
   1) Ostium primum - closed by fusion of septum to endocardial cushion
   2) Ostium secundum - coalescence of fenestrations
B. A-V canal divided by endocardial cushions
C. Septum secundum grows down from roof of atrium
   1) Fuses with endocardial cushions
   2) Overlaps ostium secundum
   3) Foramen ovale remains open until after birth

Ventricular Septation and A-V Valves

A. Muscular interventricular septum forms
B. Fusion of ventricular septum with endocardial cushion must await partition of truncus arteriosus
C. Undermining of myocardium forms valve leaflets
D. Papillary muscles and chordae tendinea derived from ventricular myocardium

Mnemonic: Weeks 2, 3, 4 of development: an event for each
Week Two: Bilaminar germ disc.
Week Three: Trilaminar germ disc.
Heart develops
Week Four: Four limbs appear.
Mnemonic: Teratogenesis: when it occurs
Teratogenesis is most likely during organogenesis—between the: Third and Eighth weeks of gestation.

Clinical Correlates - Septal Defects
A. Atrial septal defect
   1) Ostium secundum = excess resorption of septum primum or inadequate development of septum secundum (foramen ovale defect)
   2) Ostium primum = septum primum fails to fuse with endocardial cushion (low defect with semilunar shape, right above the AV valves)

Clinical Correlates - Septal Defects
B. Ventricular septal defect
   1) Failure of membranous portion to develop from extension of endocardial cushion to fuse with truncobronchial septum
   2) Malalignment
   3) Muscular defect = resorption of septum
Truncoconal Septation

A. Bulbar-truncal ridges form truncoconal or aorticopulmonary septum
B. Streaming of blood flow may account for spiral configuration of truncoconal septum
C. Bulbar-truncal ridges fuse to divide truncus arteriosus (aorta and pulmonary artery)
D. Fused bulbar-truncal ridges extend to fuse with endocardial cushion and muscular septum to partition ventricular septum - Semilunar valves derived from truncoconal swellings

Aortic Arch Derivatives

A. Truncus arteriosus
   1) Proximal ascending aorta
   2) Main pulmonary artery
B. Aortic sac
   1) Ascending aorta, 1/2 arch
   2) Brachiocephalic artery

Aortic Arch Derivatives Part II

A. Aortic arches
   1) 1, 2, 5, R6 disappear
   2) 3 => carotid arteries
   3) 4 => mid arch, R proximal subclavian artery
   4) 6 => RPA and ductus arteriosus
B. Dorsal aorta
   1) Left => descending aorta
   2) Right => R distal subclavian, distal disappears
   3) Internal carotid arteries

Fetal Circulation

A. Three shunts permit most of the blood to bypass liver and lungs
   1) Ductus venosus --- Ligamentum teres, venosum
   2) Foramen ovale -- Fossa ovalis
   3) Ductus arteriosus -- Ligamentum arteriosus
B. Shunts close after birth and become ligamentous

Clinical Correlates - Truncoconal Septation

A. Truncus arteriosus = defective fusion of bulbotruncal ridges
B. Transposition of Great Arteries = failure of truncoconal spiral
C. Tetralogy of Fallot = unequal division of conus cordis
D. Semilunar valve stenosis = failure of development of truncoconal swellings or unequal partition

Clinical Correlates - Aortic Arch Derivatives

A. Coarctation of the Aorta = probably related to ductus incorporation into the aortic wall
B. Fetal blood flow and resorption of the dorsal aorta = may also play a role
C. Double aortic arch = failure of right dorsal arch to disappear
D. Abnormal origin R subclavian artery = R4 arch and R dorsal aorta disappear, leaving 7 intersegmental artery originating as fourth branch of aorta behind esophagus

Mnemonic: Branchial arch giving rise to aorta
"Aor- from Four": Aorta is from fourth arch.

Mnemonic: Vitelline duct: closure time
Vitelline duct normally closes around week VI of intrauterine life.
FIGURE 1–45. Classification of congenital heart diseases.
Developmental Anatomy of The Lungs

### Periods of development

#### A. Intrauterine stage
- Four periods of development: embryonic, pseudoglandular, canalicular, and terminal sac

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<thead>
<tr>
<th>Phases</th>
<th>Time</th>
<th>Comments</th>
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<tbody>
<tr>
<td>Embryonic period</td>
<td>Begins at 26 days with ventral protrusion of the foregut</td>
<td>Ends at 32 days with appearance of 5 lobar bronchi</td>
</tr>
<tr>
<td>Pseudoglandular period</td>
<td>Lasts from 5th to 16th weeks</td>
<td>It is characterized by rapid branching and formation of all conducting airways</td>
</tr>
<tr>
<td>Canalicular period</td>
<td>Lasts from 16th to 25th weeks</td>
<td>It is characterized by capillary ingrowth and appearance of saccules</td>
</tr>
<tr>
<td>Terminal sac or alveolar period</td>
<td>Begins at 25 weeks</td>
<td>Alveolar development begins between the 30th and 36th weeks. Type I and II epithelial differentiation typically occurs at 28 weeks</td>
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#### B. Neonatal stage
- Precursors of typical acinar unit are present at birth: bronchioles, transitional ducts, and terminal saccule
- Alveolar development continues after birth with remodeling and multiplication
- The total adult number of alveoli are not reached until at least age 8
- Alveolar enlargement continues until adulthood, although no new alveoli are added

#### C. Histology
- The mature lung is characterized by closely packed alveoli divided by thin septa, occupied by capillaries
- Capillary endothelium is typically a single cell layer with few organelles and thin cytoplasmic matrix
- Over 95% of alveolar epithelium is type I cells, which are also very thin with few organelles
- Type II alveolar cells are cuboidal and secrete surfactant
- Surfactant synthesis peaks at term and decreases to adult levels shortly thereafter
- An increase in the lecithin-sphingomyelin ratio to more than 2:1 occurs just before birth
- Type II cells can be stimulated to produce this pattern of phospholipid by steroids, thyroxine, estrogens, beta-agonists, and increases in ventilation or tidal volume
- Surfactant stabilizes the alveoli, lowers surface tension to keep alveoli open at low volumes, prevents alveolar wall adhesion, and helps maintain pulmonary compliance

**Mnemonic: Lung development phases “Every Premature Child Takes Air”:**
- E–Embryonic period
- P–Pseudoglandular period
- C–Canalicular period
- T–Terminal sac period
- A–Alveolar period
Anatomic Variants with Normal Parenchyma

T. Superior segment of lower lobe delineated by separate fissure
U. Medial accessory left lower lobe
V. Azygous lobe - mesentery of azygous vein forms double fold of visceral pleura that isolates part of right upper lobe
W. Bilateral bilobation or trilobation
X. Situs inversus thoracis or totalis - totalis form associated with Kartagener's syndrome (situs inversus, bronchiectasis, pansinusitis - immotile cilia syndrome)

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<td>B. Congenital lobar emphysema (CLE)</td>
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<td>C. Congenital cystic adenomatoid malformations (CPAM)</td>
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<td>D. Pulmonary bronchogenic cysts (PBFM)</td>
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<td>E. Tracheal bronchus</td>
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<tr>
<td>F. Accessory cardiac bronchus</td>
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<td>G. Tracheomalacia</td>
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<td>H. Tracheal stenosis</td>
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<td>I. Pulmonary hypoplasia</td>
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Unit Objectives

• To review the basic concepts of functional physiology of the cardiovascular system (heart, blood vessels and blood) and the structural functional relationships and significance of each part in relation to the monitoring, anesthesia, and postoperative care of the surgical cardiac patient. To review the physiological correlation with the investigations like the EKG, ECHO (TTE & TEE), ANGIO, and different imaging studies to orient the surgeon’s perioperative management.

• Relationship of respiratory function and physiology and hemodynamics to previous sections on cardiovascular function and body fluid regulation

• Relationship of renal function and physiology and hemodynamics to previous sections on cardiovascular function and body fluid regulation.

Learning Objectives

By the end of this chapter the student should know:
Cardiovascular Function & Physiology
10 topics related to CVS

HEART
Cardiovascular System Function

Functional components of the CVS:
• Heart
• Blood Vessels
• Blood

General functions these provide
• Transportation: Everything transported by the blood.
• Regulation of the cardiovascular system: Intrinsic vs extrinsic
• Protection: Against blood loss
• Production/Synthesis

Functional Anatomy of the Heart

To create the “pump” we have to examine the Functional Anatomy
• Cardiac muscle
• Chambers
• Valves
• Intrinsic Conduction System
Membrane Biochemistry

- A cell’s phospholipid bilayer limits the passage of charged molecules (especially ions) across the cell membrane (i.e., the lipid part of the cell membrane has high electrical resistance). *Gap junctions, membrane transporters* and *ion channels* provide routes for charged molecules to cross the cell membrane. *Transporters and channels have fundamentally different properties.*

- Fundamental ion channel characteristics include *selectivity* and *gating*. Channels differ in the number and types of ions they will pass (selectivity). Channels can be *mechanically, ligand* and/or *voltage gated*. In addition to gating, some channels have additional *inactivation* mechanisms that can prevent the flow of ions through the channel.

- All cells have a specific complement of channels, transporters and receptors that, in part, defines their unique physiology. By definition, all cells have a membrane potential. *Ohm’s Law* defines the relationship among membrane potential (voltage), current and conductance (the inverse of resistance): \[ I = CV \]. Live cells have a resting membrane potential (V_m or RMP) that is negative with respect to the extracellular fluid. Electrically excitable cells (neurons and myocytes) have a much larger RMP (-30 to -70 mV) because they have a larger number of K⁺ channels open at rest.

- The primary function of neuronal Na⁺/K⁺ ATPase is the establishment of *the concentration gradients for Na⁺ and K⁺* that are needed to generate resting, graded and action potentials. *The actions of Na⁺/K⁺ ATPase are only mildly electrogenic*: the net result of the actions of Na⁺/K⁺ ATPase is a V_m of ~-5 to -12 mV.

- For each ion, the equilibrium (or reversal) potential is the membrane potential where the net flow through any open channels is 0. In other words, at \( E_{rev} \), the chemical and electrical forces are in balance. \( E_{rev} \) can be calculated using the *Nernst equation*. In mammalian neurons, the equilibrium potential for Na⁺ is \( \sim +60 \text{ mV} \) and for K⁺ is \( \sim -88 \text{ mV} \).

- In order to calculate the RMP, we must account for the *RELATIVE* contribution of each channel type, which is expressed in terms of *permeability* (P). The resting membrane potential will be close in value to the reversal potential for the ion that carries the majority of the resting current.

Biologic Circuits

Just like an electrical circuit (think about a flashlight), a biological circuit has 4 fundamental components. Ion channels are responsible for: *carrying the current in the biological circuit*: movement of either cations or anions into or out of the cell (more complicated than an electrical circuit where only electrons are moving); *providing resistance*: a cell that expresses few ion channels on its surface will have a higher resistance than a cell with many ion channels; and determining whether or not flow occurs (acting as an on/off switch).
Heart Muscle Mechanics - 3 concepts

**TENSION (force)** - Elements contributing:
- Contractile element ➔ Active tension
- Elastic element (functional, not anatomic) ➔ Resting tension

**LENGTH** of muscle fibers influences Tension
- Starling’s relationship (Tension (active & resting) vs Length)
- Performance-wise this is PRELOAD effect

**VELOCITY** is influenced by Length and Tension
- Calcium activation
- Total calcium released
- **Sarcomere** length alters calcium sensitivity

---

Cardiac Cycle

- Isovolumetric ventricular contraction
- Rapid ejection phase
  - Reduced ejection phase
- Isovolumetric relaxation
- Rapid filling phase
  - Slow filling period
  - (Atrial contribution (20-30% in failing heart))

Cardiac Cycle Mnemonics: ISRR
In this mind map is represented the VENTRICULAR cardiac cycle. Focusing in the “ISRR” word that represents the 5 phases of the Ventricular cardiac cycle, the Atrial phase is not included (see Dr. Najeeb video).

- **Isometric contraction** (max. O2 consumption)
- **Systolic ejection** (rapid and slow phases)
- **Isometric relaxation**
- **Rapid filling or rapid passive ventricular filling**
- **Reduced filling or slow passive ventricular filling**

See the Aorta & Mitral valves icons and remember Aorta up - Mitral down in the house scheme.
Only the healthy valve sound are in closing, the opening of valves does not produce sounds.
Cardiodynamics: Cardiac Output Controls & Blood Pressure

Cardiac Performance

- Stroke Volume affected by
  - Preload
  - After load
  - Contractility
- Law of La Place relates ventricular pressure and wall tension
  \[ T = Pr \]
  \[ 2h \]

Preload
- Normal heart - increased venous return results in increased cardiac output
- Failing heart - sarcomere length is already maximal; cardiac output increase requires increased contractility or heart rate

Afterload
- Definition
- Increased after load - increase in LVEDV and radius (=preload)
- Anrep effect or homeometric autoregulation
- Contractility - inotropic state of muscle

Indicators of Cardiac performance

- Cardiac index = cardiac output/body surface area
- LVEDP (or approximation)
  - Mean left atrial pressure (Δ LAP)
  - Mean pulmonary wedge pressure (Δ PCWP)
  - Pulmonary artery diastolic pressure (P ADP)
- CI and LVEDP together are better indicators of contractility than either alone
- Ejection fraction (EF) = stroke volume/end-diastolic volume
- Fractional shortening (FS) - calculated from the diameter perpendicular to the midpoint of LV long axis

Law of The Heart (Frank-Starling ‘s Law)

The Cardiac Function Curve (Length-tension Curve)
Coronary flow & myocardial O₂ consumption

- Very efficient oxygen extraction (70% oxygen utilization coefficient)
- Coronary hemodynamics - Q = P/R
- Viscous resistance
- Autoregulatory resistance
- Compressive resistance
- Transmural gradient in myocardium - DPTI x HR = driving pressure
- Myocardial oxygen consumption
- Pressure work, contractility, heart rate, basal cell metabolism, electrical activation

Hemodynamics: Blood Flow & Blood Pressure Controls

BLOOD VESSELS
- Cardiovascular regulation: ensures adequate circulation to body tissues
- Cardiovascular centers: control heart and peripheral blood vessels
- Cardiovascular system responds to: changing activity patterns and circulatory emergencies
  CO tells us how much blood is ejected per minute and is influence by both intrinsic & extrinsic factors
Extrinsic factors (besides ANS) include
- Blood vessels & blood pressure
- Blood volume & viscosity
- Capillary exchange & the lymphatic return
- Cardiovascular disease

Ways To Alter The Vascular Function Curve
- Change The Mean Circulatory Pressure
  Change Blood Volume
  Change Venous Capacity
- Change Total Peripheral Resistance

Medullary Center for Cardiovascular Control & the Baroreceptor Reflex

Sympathetic Activity Summary:
- ↑ chronotropic effects ➔ ↑ heart rate
- ↑ dromotropic effects ➔ ↑ conduction of APs
- ↑ inotropic effects ➔ ↑ contractility

Parasympathetic Activity Summary:
- ↓ chronotropic effects ➔ ↓ heart rate
- ↓ dromotropic effects ➔ ↓ conduction of APs
- ↓ inotropic effects ➔ ↓ contractility
Capillary Dynamics: Capillary & Cellular Exchange

Cardiovascular process involving all three functional systems: heart, blood & blood vessels and physics:
- Velocity of blood flow
- Cross-sectional area of capillaries
- Exchange processes
- Diffusion & transcytosis
- Pressures
- Filtration
- Influenced by capillary hydrostatic pressure
- Colloid osmotic pressures (oncotic pressure)
- Influence bulk flow

The Lymphatic System

Q. What does the lymphatic System do for us?
- Returns the excess fluid
- In doing so prevents edema
- Absorbs and transports fats from the GI tract
- Through specialized lymphatic capillaries called lacteals
- Filters the returning fluid for purposes protection
- Occurs at the lymph nodes
- More on the lymphatic system and its functions later as it relates to digestive system and immunity.

Coagulation Basics

BLOOD
Primary hemostasis
Platelet (a) adhesion, (b) activation, (c) aggregation

Secondary hemostasis
Activation of plasma coagulation (form fibrin)
Extrinsic pathway (via tissue factor)
Intrinsic pathway (subendothelium or foreign contact)

Common pathway
- Inhibition of systemic clotting
- Natural anticoagulants (AT III, Protein C & S)
- Fibrinolytic system, i.e. Plasmin (degrades fibrinogen))

Other reactions
• Complement activation (increased permeability, cell lysis)
  Kinin generation (vascular dilation, increased permeability)

Platelet Function

Contact
• With subendothelium after endothelial injury
• With proteins adsorbed onto synthetic surfaces

Adhesion
• Via attachment mechanisms i.e. Glycoprotein Ib/IX (GP Ib/IX) receptor

Activation
• Begins as platelets spread with a conformational change
• Release TxA2, ADP, serotonin, (PF4, BTG)

Aggregation
• ADP induced change in GpIIb/IIIa receptor permits binding of adhesive proteins, like fibrinogen, between platelets.

Fluid, Electrolyte, and Acid-base Balance

Body Fluid, Fluid Compartments

• Body Water
  Regulation of Gain
  Regulation of Loss

• Movement of body fluids
  between plasma and interstitial fluid between interstitial and intracellular

The Electrolytes
Acid Base Balance

- The pH of arterial blood is maintained at 7.35–7.45 (Better normal range of pH 7.38 – 7.42).
- Normal functioning of the body’s complex enzyme systems depends on this stability.
- Controlled by systems which maintain H+ levels:
  - Buffering Systems,
  - Ventilation Rates, &
  - Renal Function
- Derangements are due to respiratory and/or metabolic dysfunction.
- Compensatory mechanisms are also divided into metabolic and respiratory. The true clinical picture is usually mixed.

Buffering System
1. Buffering system #1 PROTEINS (Hemoglobins)
2. Buffering System #2 Bicarbonate buffering system
3. Buffering System #3 Phosphate buffering system
   - Buffer systems
   - Exhalation of Carbon Dioxide
   - Kidney Excretion

- Acid Base Imbalances
  - Acidosis vs Alkalosis

Respiratory Physiology

Oxygenation

- The amount of oxygen in arterial blood, is described in terms of the partial pressure of oxygen in arterial blood (PaO2), and the percentage saturation of arterial hemoglobin with oxygen (SaO2).

Ventilation,

- The movement of air in and out of the lungs, is described in terms of minute volume, and assessed by measuring the partial pressure of carbon dioxide in arterial blood (PaCO2).
- Oxygenation is independent of minute volumes until they are very low.

In postoperative patients the primary cause of hypoxia is atelectasis and this must be reversed before the patient can benefit from increasing the fraction of oxygen in inspired air (FiO2).
- Oxygen consumption is about 250mL/min O2 in the adult at rest, rising to over 4L/min during exercise.
- Oxygen diffuses from alveolus to pulmonary capillary until arterial PO2 (PaO2) = alveolar PO2 (PAO2).
- The solubility of oxygen in blood is low (0.000225mL O2 per mL of blood per kPa): at a normal PaO2 of 13kPa, there is only 0.3mL O2 dissolved in 100mL of blood.
- Each gram of hemoglobin combines with 1.34mL O2: at a normal Hb 13g/dL there is 20mL O2 bound to hemoglobin in 100mL of blood.
- Hence the percentage of arterial hemoglobin binding sites bound to oxygen
(SaO2) or ‘oxygen sats’ is the main determinant of the O2 content of arterial blood.

Positive end expiratory pressure (PEEP) and continuous positive airway pressure (CPAP) (treat and prevent atelectasis. The balance between oxygen consumption and delivery is assessed by measuring the percentage saturation of mixed venous hemoglobin with oxygen (SvO2) or ‘mixed venous’, which is dependent on cardiac output, hematocrit, oxygenation, and oxygen consumption. Loss of functional residual capacity through atelectasis, supine position, lobar consolidation and collapse, effusions and obesity, results in hypoxia: CPAP, PEEP, and physiotherapy are aimed at limiting this loss.

Renal Function & Physiology

- Relationship of renal physiology and hemodynamics to previous sections on cardiovascular function and body fluid regulation
- Review renal acidification from the perspective of what should be taught to medical students
  - Overview – role of the kidneys
  - HCO3⁻ reabsorption along the nephron
  - Generation of “New HCO3⁻”
  - Regulation of renal acid excretion
- Pressure profiles along the nephrovascular unit and glomerular and peritubular capillary dynamics
- Restriction of macromolecular permeability and role of charge and size selectivity
  - Intrinsic vs extrinsic mechanisms
- Effects of sympathetic stimulation
- Renal autoregulation
  - Myogenic and tubuloglomerular
  - Feedback mechanisms
- Other intrinsic regulations
  - Endothelial factors
  - Renin-angiotensin system
  - Prostaglandins
3 Cardiovascular Pharmacology

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Unit Objectives

1. To understand the inotropic agent receptor basic physiology and to understand the appropriate clinical application of inotropic agents and the new developments.
2. To understand the vasopressor agent receptor basic physiology and to understand the appropriate clinical application of vasopressors and the new developments.
3. To understand the appropriate clinical application of vasodilators and the new developments.
4. To understand the appropriate clinical application of antiarrhythmic agents and the new developments.
5. To understand chronotropic agent receptor basic physiology and to understand the appropriate clinical application of chronotropics.
6. To understand the basics of cardioversion.
7. To understand the basics of defibrillation.
8. To understand the basics of cardiac pacing.
9. To understand the indications, contraindications, and types of component hemotherapy & the principles of transfusion medicine and their relation to cardiovascular diseases and pre-, intra- and postoperative surgical cardiac patients.
10. To understand the appropriate clinical application of fibrinolytic agents and the new developments.
11. To understand the appropriate clinical application of antithrombetics (antithrombolytics, anticoagulants, and antiplatelets) and the new developments.
12. To understand the appropriate clinical application of diuretics and the new developments.
13. To understand the appropriate clinical application of fluid management (Crystalloids & Colloids).
14. To understand the appropriate clinical application of antihyperlipidemic agents and the new developments.

Learning Objectives

By the end of this chapter the student should:

- know the differences between vasopressor and inotropic agents and to understand the appropriate clinical application of vasopressors and inotropic agents and the new developments.
- know the appropriate clinical application of vasodilators (arterial, venous, or mixed balanced) and the new developments of inodilators.
- know the appropriate clinical application of the main 5 groups of antiarrhythmic agents and the new developments.
- know the basics of cardioversion & defibrillation.
- know the basics of cardiac pacing.
- know the indications, contraindications, and types of component hemotherapy & the principles of transfusion medicine.
- know the appropriate clinical application of fibrinolytic agents and the new developments.
- know antithrombetics (antithrombolytics, anticoagulants, and antiplatelets) and the new developments.
- know the appropriate clinical application of diuretics.
- know the appropriate clinical application of fluid management (Crystalloids & Colloids).
- know the appropriate clinical application of antihyperlipidemic agents.
**Inotropes**

- Increase contractility via beta receptors
- Increase CO, but with
- NO guarantee to increase blood pressure

**Vasopressors**

- Vasoconstrict via alpha receptors
- Increase SVR, therefore
- Raise mean arterial pressure (MAP)

**Inotropes, Vasoconstricting**

**Dopamine**
- 1) Low doses - D1 receptors in renal vasculature
- 2) Increasing doses - b-1 receptors activated
- 3) High doses - a-adrenergic receptor activation

**Epinephrine**
- 1) Potent b and a effects

**Norepinephrine**
- 1) Potent alpha effects

**Inotropes, Vasodilating**

**Dobutamine**
- 1) Beta > alpha effect
- 2) Reduces LV filling pressures
- 3) Decreases afterload

**Milrinone, Amrinone**
- 1) Phosphodiesterase inhibitors

**Isoproterenol**
- 1) Inotropic (beta), chronotropic effects

**Recap of some important equations**

\[ CO = SV \times HR \]
\[ SV = EDV - ESV \]
\[ EF = \frac{(EDV - ESV)}{EDV} \]

**Some new equations to remember**

Flow = Difference in pressure / resistance

\[ CO = \frac{MAP}{SVR} \]

Resistance \( \propto \frac{1}{radius^4} \)

**Mnemonic: Clinical use: ABC HIL**

A - Anaphylaxis
B - Bronchospasm
C - Cardiac arrest
H - Haemostatic agent
I - Insulin inhibition
L - Local anaesthetic

**Sara Hates Men On Drugs**

S - Serotonin, H - Histamine, M - Muscarinic agents, O - Opioids, D - Dopamine
Vasodilators & Vasoconstrictors

Vasoconstrictors

Neosynephrine (pure alpha)

Vasodilators

Nitroprusside
- Generalized vasodilatation
- "Steal phenomena"
- Indications - hypertension, acute heart failure
- (thiocyanate toxicity - rare; with renal failure)

Nitroglycerine
- General vasodilatation
- Low doses - venous; high doses - arterial
- Preload reduction and coronary vasodilation
- Useful in management of ischemia
- Decrease LVEDP and pulmonary vascular congestion

NO and Isoproterenol - pulmonary effects

ACE Inhibitors
A. Mechanisms
1) Prevent conversion of Angiotensin I to Angiotensin II - vasodilation
2) Decreased Aldosterone secretion
3) Indication - hypertension, heart failure, prophylactically after MI

B. Agents
1) Captopril
2) Low cardiac output states - improvement in renal blood flow
3) Angioedema/cough/neutropenia/nephrotic syndrome
4) Increase in creatinine - RAS

C. Enalapril
1) Enalaprilat (liver) - delay - long duration
2) Less side-effects

Antiarrhythmics
### Beta Blockers (BBs)

**A. Mechanisms**
1) b-1 and b-2; cardioselectivity

**B. Indications**
1) Hypertension, angina pectoris, arrhythmias, prophylactically after MI

**C. Adverse effects**
1) Bronchospasm, Inhibition of myocardial contractility

**D. Drug interactions**
1) Propranolol, metoprolol, atenolol
2) Esmolol - very short half-life

**E. Agents**
1) Propranolol, metoprolol, atenolol
2) Esmolol - very short half-life

### Calcium Antagonists (CCBs)

**A. Mechanisms**
B. Interference of Ca2+ - mediated smooth muscle contraction - coronary and peripheral smooth muscle relaxation

**C. Selective Ca2+ channel inhibition**
1) Treatment of angina pectoris / supraventricular tachycardia / hypertension

**D. Agents**
1) Verapamil
2) Nifedipine
3) Diltiazem

### Digitalis

- Inhibit Na-K-ATPase - positive inotropic effect
- Parasympathomimetic and anti-adrenergic mechanisms
- Drug interaction - Quinidine, Verapamil, Amiodarone
- Conditions that increase sensitivity

**Mnemonic: Amiodarone - important side effects**

- **PCT** - Proximal Convoluting Tubule
- **P** - Pulmonary fibrosis
- **C** - Corneal microdeposits
- **T** - Thyroid disorders (hypo/hyperthyroidism)

**Mnemonic: Amiodarone: action, side effects 6 P's:**
- **P** - Prolongs action potential duration
- **P** - Photosensitivity
- **P** - Pigmentation of skin
- **P** - Peripheral neuropathy
- **P** - Pulmonary alveolitis and fibrosis
- **P** - Peripheral conversion of T4 to T3 is inhibited -> hypothyroidism

---

### Antiarrhythmic Drug Classification

**Some Block Potassium Channels**

- I - Sodium channel blocker
- II - β-Blocker
- III - Potassium channel blocker
- IV - CCB

**Mnemonic: I to IV MBA College**

**Mnemonic: Beta blockers:**

- B1 selective vs. B1-B2 non-selective
- **A** through **N**: B1 selective: Acebutalol, Atenolol, Esmolol, Metoprolol.
- **O** through **Z**: B1, B2 non-selective: Pindolol, Propranolol, Timolol.

**Mnemonic: Beta blockers with ISA**

- Picture diabetic and asthmatic kids riding away on a cart that rolls on pinwheels.
- Pindolol and Carteolol have high and moderate ISA respectively, making them acceptable for use in some diabetics or asthmatics despite the fact that they are non-selective beta blockers.

**Mnemonic: Beta blockers with CYP2D6 polymorphic metabolism**

- "I Met Tim Carver, the metabolic polymorph."
- The following beta blockers require dose adjustment due to CYP2D6 polymorphic metabolism: Metoprolol Timolol Carvedilol (in patients with lower or higher than normal CYP2D6 activity)
Chronotropics

Mnemonic: Propranolol and related ‘-olol’ drugs: usage “olol” is just two backwards lower case b’s. Backward b’s stand for “betablocker”. - Beta blockers include acebutolol, betaxolol, bisoprolol, oxprenolol, propranolol.

Mnemonic: Beta-blockers: nonselective beta-blockers “Tim Pinches His Nasal Problem” (because he has a runny nose...):
T - Timolol
P - Pindolol
H - Hismolol
N - Naldolol
P - Propranolol

Antifibrinolytic

There are two types of antifibrinolytic:
- Synthetic lysine analogues, e.g., tranexamic acid and aminocaproic acid (Amicar®).
- The non-specific serine protease inhibitor aprotinin (Trasylol®) which was withdrawn from world markets in May 2008 after several non-randomized studies raised safety concerns.

Antithrombotics (Thrombolitics, Anticoagulants & Antiplatelets)
Therapeutic Strategies

- **Degrade fibrinogen/fibrin (fibrinolytic agents)**
  - **GOAL:** eliminate formed clots
- **Inhibit clotting mechanism (anticoagulants)**
  - **GOAL:** prevent progression of thrombosis
- **Interfere either with platelet adhesion and/or aggregation (antiplatelet drugs)**
  - **GOAL:** prevent initial clot formation

---

**Thrombolytic Agents**

- These drugs are used to treat strokes, AMIs, DVT/PE, DIC---all potentially life-threatening conditions.
- The effectiveness of thrombolytics ("clot busters") is inversely related to the time elapsed since the thrombic crisis began; these drugs are most effective within 6 hours of onset of symptoms.
- **Streptokinase** - Indirectly activate plasminogen to plasmin => fibrin into FDP's (non-specific)
- **Urokinase** - Indirectly - thrombolysis (non-specific)
- **tPA (Alteplase)** - Clot specific thrombolytic - binds directly to clot via fibrin
- **APSAC** - Like Streptokinase

---

**DRUGS YOU NEED TO KNOW:**

<table>
<thead>
<tr>
<th><strong>ANTICOAGULANTS</strong></th>
<th><strong>AMINOCAPROIC ACID (EACA)</strong> (generic, Anciclar) (in bleeding disorders handout!)</th>
</tr>
</thead>
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<tr>
<td>ARGATROBAN</td>
<td>THROMBOLYTICS</td>
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<td>BIVALIRUDIN (Angiomax)</td>
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<td>DALTEPARIN (Fragmin)</td>
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<td>DROTRECOGIN ALFA (ACTIVATED PROTEIN C) (Xigris)</td>
<td>TISSUE PLASMINOGEN ACTIVATORS (tPAs): ALTEPLASE (Activase, RETEPLASE (Rellevase))</td>
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<tr>
<td>ENOXAPARIN (Enoxa)</td>
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<td>FONDAPARINUX</td>
<td>ANTIPLATELET DRUGS</td>
</tr>
<tr>
<td>HEPARIN (Calciparin, Hepathrom, Lipid-Heparin, Lipo-Heparin, Panheparin)</td>
<td>ABCIXIMAB (Centorcor)</td>
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<td>HIRUDIN (Desirudin)</td>
<td>ACETYLSALICYLIC ACID (Aspirin)</td>
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<td>4-HYDROXYCOUMARIN (Coumadin)</td>
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<td>LEPIRUDIN (Refludan)</td>
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<td>WARFARIN (Lambeth K, Panwarfin)</td>
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<td>XIMELAGATRAN (Exeral)</td>
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<td>ANTIDOTES</td>
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<td>PHYTONADIONE (Vitamin K)</td>
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<tr>
<td>PROTAMINE SULFATE</td>
<td></td>
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</tbody>
</table>
Anticoagulant Drugs

- The major classes of anticoagulant drugs have distinctly different mechanisms of action, routes of administration and adverse effects. The mechanisms of action include: activation of anticlotting factors (especially antithrombin III), direct inhibition of thrombin, inhibition of synthesis of blood coagulation factor precursors (zymogens), and activation of protein C.
- A unique side effect to the use of HEPARIN is a transient thrombocytopenia (HIT) that occurs in 25% of patients.
- The approved use of direct thrombin inhibitors (DTIs) is for the treatment of HIT.
- WARFARIN:
  - has a narrow therapeutic index
  - is nearly completely (99%) bound to plasma albumin
  - is eliminated by hepatic metabolism (cytp450)
  - ❯ WARFARIN is the prototype for drug-drug interactions!

Drotrecogin Alfa is approved for use in DIC (fatal complication of septic shock). The use of activated protein C as a drug occurred as a result of a change in our understanding of the pathophysiology of sepsis, particularly the intricate interplay between activation of coagulation and inflammation. The major classes of anticoagulant drugs have distinctly different mechanisms of action, routes of administration and adverse effects.

Mechanisms of Action:
1. Activation of anticlotting factors (especially antithrombin III) e.g. HEPARIN
2. Direct inhibition of thrombin e.g. HIRUDIN
3. Inhibition of synthesis of blood coagulation factor precursors (zymogens) e.g. WARFARIN
4. Activated Protein C i.e., DROTRECOGIN ALFA

Drugs That Activate Anticlotting Factors: Heparin, Dalteparin, Enoxaparin, Fondaparinux

HEPARIN
- most commonly given anticoagulant for short term use (>12 million patients/year)
- is a complex mixture of mucopolysaccharides
- isolated from bovine lung or porcine intestine
- normally found in mast cells - unknown physiologic role • strongly acidic

Heparin
- Standard initial dose = 300 U/kg
- Maintain ACT > 300-350 (>300?)
- Monitor with ACT (or direct Heparin concentrations)
- Redose to maintain therapeutic level
- 100 U/kg every 60 - 90 minutes (approx.)
  - Use dose-response curve
- Protamine for heparin reversal
- Estimate heparin present (dose response curve)

Adverse Effects
- HEMORRHAGE (esp. hemorrhagic stroke)
- hypersensitivity
- transient HIT occurs in about 25% of the patients during first 5 days of treatment – severe HIT occurs in 5% of patients o small % of patients develop antibody-mediated thrombocytopenia that is associated with thrombosis (paradoxical) o in those patients, HEPARIN should be discontinued, and treatment initiated with HIRUDIN or LEPIRUDIN, but not WARFARIN (may exacerbate the prothrombotic state)
- prolonged administration of high doses may cause osteoporosis

Anticoagulants Heparin
- Glycosaminoglycan, MW 3K - 100K
- Acts by binding enzyme AT III (AT III inhib's IIa,Xa,IXa,XIa,XIIa)
- Half life is 60-90 minutes
- Monitored with aPTT or ACT
- Complications: bleeding; HIT => thrombosis, "white clot" (Ab versus Hep-PF4 complex); osteoporosis

Alternatives to heparin (future)
- Hirudin (Hirulog, synthetic analog)
  - From leeches, direct inhibitor of thrombin
  - Does not require ATIII
  - Prolongs TT, aPTT, PT, and ACT
- Ancrod
- From venom of Malayan pit viper
- Others

- Anticoagulants: Heparin
Dalteparin, Enoxaparin

- Low MW fragments of HEPARIN Fondaparinux • synthetic formulation of the key pentasaccharide that appears to be the active component in all heparins

Mechanisms of Action
- Bind to antithrombin III, a protease inhibitor that complexes with activated clotting factors II, IX, X and XI (i.e., heparins are INDIRECT thrombin inhibitors)
- Conformational change in ATIII • exposure of ATIII active site • more rapid formation of ATIII-protease complexes
- Release of HEPARIN; activated clotting factors remain bound to ATIII

Pharmacokinetics
- Activity is standardized by bioassay • must be given IV (not active orally; IM administration causes hematomas)
- Rapid effect (within minutes); instantaneously in vitro • metabolized in liver; excreted in urine
- HEPARIN dose is adjusted to double partial thromboplastin time (monitored by aPTT)

### Greater than heparin | Less than heparin
---|---
- Anti-factor Xa activity | Inactivation of thrombin (IIa)
- Bioavailability (SC administration) | Platelet inhibition
- Half-life (decreased dosing) | Vascular permeabilization
- More predictable pharmacology (less need for monitoring) | Plasma protein binding

Protamine Sulfate

- Highly basic peptide that combines with HEPARIN as an ion pair
- Lasts about 2 hours
- Routinely used following cardiac surgery and other vascular procedures
- Excess protamine also has an anticoagulant effect, since it can interact with platelets, fibrinogen and other plasma proteins
- Anaphylactic reactions can occur - approximately 1% of patients with diabetes mellitus who have received protamine-containing insulin experience anaphylaxis • cannot reverse many LMWH! A unique side effect to the use of heparin is a transient thrombocytopenia (HIT).

2. Direct Thrombin Inhibitors (DTIs): ARGATROBAN, Bivalirudin, HIRUDIN, Lepirudin, Ximelagatran

Mechanism of Action
- HIRUDIN was originally isolated from leech; LEPIRUDIN is the recombinant form; BIVALIRUDIN is a synthetic analogue
- Work by 1) inhibiting fibrin binding to thrombin and 2) interacting with the thrombin active site inhibiting thrombin activity even in the presence of bound fibrin

- ARGATROBAN is a synthetic derivative of L-arginine - ARGATROBAN and XIMELAGATRAN bind reversibly to the thrombin active site
- Advantages over HEPARIN:
  1. Inhibition of coagulation via a single mechanism of action
2. Actions are independent of antithrombin iii, which means they can reach and inactivate fibrin-bound thrombin inside clots
3. Little effect on platelets or bleeding time
4. Elimination of hit as a side effect of treatment
5. Not inactivated by platelet or plasma proteins 6. More uniform potency and increased safety
6. Rebound coagulation after discontinuation of the drug is less likely with direct thrombin inhibitors

Pharmacokinetics
- Given IV (note: XIMELAGATRAN is first oral agent in this class Æ promoted as a replacement for both WARFARIN and HEPARIN because of its immediate anticoagulant action
- Metabolized by hydrolysis in liver, excreted in urine The approved use of dtis is for the treatment of heparin-induced thrombocytopenia (HIT).
- Monitored by aptt – ARGATROBAN causes elevated inrs because of test interference, making the transition to warfarin difficult

3. Drugs That Inhibit Synthesis Of Coagulation Factor Precursors: 4-Hydroxycoumarin, WARFARIN

Warfarin
- Acts as Vitamin K antagonist  (Vitamin K required for Fx II, VI, IX, X; Prot C,S)
- Half-life is 36 to 42 hours
- Monitored w/ INR = Pt. PT / Control PT
- Reversed w/FFP (immediate); Vit K (8-24 hrs)
- Complications: Bleeding, skin necrosis (Protein C & S deficiency), fetal abnormalities

Mechanism of Action
- Inhibit epoxide hydrase
- Interfere with the synthesis of vitamin k and thus inhibits activation of vitamin k-dependent clotting factors (ii, vii, ix, x)
- ↓ the activity of protein c (activated by thrombin) – responsible for some side effects

Pharmacokinetics
- Rapid, complete absorption from GI; peak plasma drug concentration in < 1 hr
- WARFARIN is a racemic mixture of R- and S-forms – S is the active enantiomer
- S- and R- WARFARIN are metabolized differently; S is metabolized primarily by CYP2C9, and R by CYP1A2, CYP2C19 and CYP3A4 • several genes play a role in WARFARIN metabolism:

Adverse Effects
- Hemorrhage
- Flatulence and diarrhea are common
- Decreased protein c causes cutaneous necrosis caused during first weeks of treatment; rarely can progress to infarction (venous thrombosis) in fatty tissues, intestine and extremities
- Bone defects (chondrodysplasia punctata) and hemorrhagic disorders in infants born to mothers taking the drug during first trimester of pregnancy IS NOT CONTRAINDICATED IN PREGNANCY

Phytonadione (Vitamin K1)
- Pharmacodynamic antagonist (not due to disappearance of warfarin, but rather the reestablishment of normal clotting factor activity) – takes 24 hours
4. Drotrecogin Alfa (Activated Protein C)
Mechanism of Action
- Recombinant version of protein C; activated by thrombin; requires Ca2+, phospholipid and protein S as cofactors
- Anti-coagulant actions:
  1. Destroys activated factors Va and VIIIa, resulting in ↓ thrombin formation
  2. Inhibits platelet activation
  3. Suppresses tissue factor expression
- Fibrinolytic actions
  1. ↑ thrombin-catalyzed activation of tPA inhibitors
  2. ↓ PAI-1 concentrations
- Anti-inflammatory actions:
  1. Synthesis of tumor necrosis factor
  2. Neutrophil activation
  3. The release of cytokines from macrophages

Therapeutic Indications
- ↓ relative risk of death by 20% (up to 50% of patients die of sepsis within 6 months)

| Antiplatelet Drugs
| ASA, Abciximab, Clopidogrel, Dipyridamole, Eptifibatide, Ticlopidine, Tirofiban
| ASA
Mechanisms of Action
Inhibit platelet adhesion and aggregation by:
1. Inhibiting cyclooxygenase: e.g. ASA
2. Blocking glycoprotein IIb/IIIa receptor: e.g. Abciximab, Eptifibatide
3. Inhibiting the binding of fibrinogen to activated platelets: e.g. Clopidogrel, Ticlopidine
4. Inhibiting cyclic nucleotide phosphodiesterase: e.g. Dipyridamole
5. IV Dextran (40 - MW 40,000 daltons)
   - ↓ Plt-vascular endothelial interaction
   - ↓ von Willebrand factor

| Diuretics
| Diuretics cause net loss of Na+ and water from the body by action on the kidney: their primary effect is to decrease Na+ and Cl− reabsorption from the filtrate: increased water loss is secondary.
| Diuretics act by: (1) direct action on the cells of the nephron (loop, thiazide and K+-sparing diuretics) or (2) modifying the content of the filtrate (osmotic diuretics).
| Although loop diuretics are used to convert oliguric to polyuric renal failure the temporary increase in urine output has no prognostic effect on the progression of acute tubular necrosis. In hypovolemic states loop diuretics may exacerbate acute tubular necrosis by provoking a diuresis, worsening hypovolemia, CO, and renal ischemia.
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A temporary increase in urine output has no prognostic effect on the progression of acute tubular necrosis. In hypovolemic states, loop diuretics may exacerbate acute tubular necrosis by provoking a diuresis, worsening hypovolemia, CO, and renal ischemia.

Antihyperlipidimic Agents

The major classes of drugs for consideration are:

Established
- HMG CoA reductase inhibitors (statins)—lovastatin, pravastatin, simvastatin, fluvastatin, atorvastatin (statin)
- Bile acid sequestrants—cholestyramine, colestipol, colesvelam
- Nicotinic acid—crystalline, timed-release preparations, Niaspan®
- Fibric acid derivatives (fibrates)—gemfibrozil, fenofibrate, clofibrate (fibr)
- Estrogen replacement
- Selective estrogen receptor modulators

Alpha Blockers
Beta Blockers
Ca Channel Blockers
ACE Inhibitors
Anti-Lipemic
Nitrates

atenolol (Tenormin®)
carvedilol (Coreg®)
metoprolol (Toprol XL®, LoPressor®)
propranolol HCl (Inderal®)
benazepril HCl (Lotensin®)
captopril (Capoten®)
enalapril maleate (Vasotec®)
ilisopropil (Prinivil®, Zestil®)
amlopidine besylate (Norvasc®)
diltiazem HCl (Cardizem®, Dilacor®)
nifedipine (Adalat®, Procardia XL®)
verapamil HCl (Calan®, Isoptin®, Covera®)

- atorvastatin calcium (Lipitor®)
- lovastatin (Mevacor®)
- pravastatin (Pravachol®)
- rosuvastatin calcium (Crestor®)
- simvastatin (Zocor®)

- isosorbide dinitrate (Isordil®)
- isosorbide mononitrate (Ismo®)
- Nitroglycerin

doxazosin mesylate (Cardura®)
prazosin HCl (Minipress®)
4 Cardiac Dysrhythmias

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Unit Objectives

- To review the basic concepts of functional physiology of the conduction system and the structural functional relationships and significance of each part in relation to the monitoring, anesthesia, and postoperative care of the surgical cardiac patient.
- Classification of bradydysrhythmias
- Classification of supraventricular tachydysrhythmias
- Classification of ventricular tachydysrhythmias

Learning Objectives

By the end of this chapter the student should know:

- Overview: EKG Monitoring, Basic EKG Interpretation
- Bradydysrhythmia: Lethal Dysrhythmias , Dangerous Dysrhythmias, Benign Dysrhythmias
- Cardiac Pacing: Cardiac Pacing Overview
- Internal Cardioverter Defibrillators (ICD)
- Tachydysrhythmias: Lethal Dysrhythmias, Dangerous Atrial Dysrhythmias, Other Dangerous Dysrhythmias
- Supraventricular Tachycardias (SVT)
- AFB/Flutter, Surgery for SVT
- Cardioversion
- Defibrillation: External Defibrillation, Internal Defibrillation, Internal cardiac massage and defibrillation
- Surgery for AFB, Wolf-Parkinson-White Syndrome (WPW)

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Cardiac dysrhythmias are caused by disorders of impulse formation or disorders of impulse conduction, or both. Cardiac dysrhythmia may be life-threatening because of ↓cardiac output, ↓myocardial blood flow, or precipitation of a more serious arrhythmia.

RF ablation is the therapy of choice for many types of cardiac dysrhythmias.

EPS are used to map out normal and abnormal intracardiac structures. In this process, the mechanism of arrhythmia is delineated, and ablation can be performed at the same time.

Pacing technologies have been developed to treat heart failure →↑pulse pressure, ↑left ventricular stroke volume, ↑cardiac index, and ↑wedge pressure.

Implantable pacemakers are placed for treatment of symptomatic bradycardia with the ability to respond to changing hemodynamic demands.

The development of implantable cardioverter-defibrillators (ICDs) to terminate ventricular tachydysrhythmias by delivering high-voltage shocks to the ventricle has revolutionized therapy for cardiac dysrhythmias.

The main purpose of ICD placement is to prevent sudden cardiac death resulting from hemodynamically unstable ventricular dysrhythmias.

An ICD also can be placed for cardiac resynchronization. Cardiac resynchronization therapy has been shown to improve heart failure symptoms, quality of life, exercise capacity, and electrocardiographic variables.

Anesthetic management of patients for correction of cardiac dysrhythmias depends on associated comorbid illness and the procedure that is planned.
Overview

EKG Monitoring

Indications
All cardiac surgical patients should have a 12-lead EKG preoperatively, and daily 12-lead EKGs postoperatively, as well as EKG monitoring intraoperatively and for 48h postoperatively. This facilitates:

• Diagnosis of ischemia.
• Diagnosis of conduction defects.
• Diagnosis of electrolyte disturbances.

Contraindications
There are no contraindications to EKG.

Basic EKG Interpretation

Normal sinus rhythm (NSR)
Note the P wave, followed in 0.12–0.2s (3–5 small squares) by a QRS complex that lasts <0.12s (<3 squares), and which is followed by a T wave. The ST segment is flat and is at the isoelectric point (the same level as the PQ segment).

| TABLE 4−1. Waves, Intervals, and Common Pathologies |
| Etiology of dysrhythmias |

N.B.: Arrhythmias is a misnomer, the correct is DYSRHYTHMIAS

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Bradydysrhythmia

Bradycardia
Heart Rate < 60 beats/min:
A. Asymptomatic
1) Athletic conditioning
2) Sinus bradycardia
B. Symptomatic
1) Inadequate cardiac blood flow
2) Loss of atrial augmentation
3) Dizziness, syncope (inadequate cerebral blood flow)

Heart Block Definition
A. First Degree - PR interval > 0.2 seconds
B. Second Degree - Intermittent AV conduction
C. Third Degree - Atrial impulses do not conduct

Heart Block - Complete
A. Causes
1) Congenital- often asymptomatic
2) Valvular calcification- Aortic, Mitral
3) Myocardial infarction- inferior, anterior septal
4) Surgical- VSD repair, subvalvular stenosis resection, valve replacement (IHSS)

Sinus Node Dysfunction
Amyloid deposition
A. Surgical damage
1) Fontan repair, atrial switch, Maze II, superior approach for Mitral valve repair  
2) Sick sinus syndrome  
1) Tachycardia, bradycardia  
2) Diagnosis by prolonged ambulatory ECG

**Carotid Sinus Syndrome**  
A. Cardio-inhibitory- Carotid sinus stimulation  
1) 3 second or greater pauses  
2) pacemaker appropriate  
B. Vasodepressor- Carotid sinus depression with Atropine  
1) BP drops without slowing  
2) pacemaker may not help symptoms

### Lethal Dysrhythmias

Complete heart block  
**Features:** wide complex < 35bpm (Fig. 5.14d) with no or poor cardiac output.  
**Therapy:** pace if possible. Commence CPR if no cardiac output. Usually the patient will maintain **Therapy:** this is benign. In a patient that has undergone aortic valve surgery it suggests damage to the conduction pathways passing close to the aortic annulus. This often resolves over the weeks postoperatively as edema and hematoma settle. The patient warrants follow-up by a cardiologist as occasionally insertion of a permanent pacemaker is indicated, but unless first-degree heart block is accompanied by a new left bundle branch block or some cardiac output and chest compressions are not necessary.  
If epicardial wires are present pace immediately.  
If there is an adequate cardiac output do not commence chest compressions. Treat pharmacologically.  
If no cardiac output give atropine 1mg iv once, and epiinephrine 1mg iv (10mL of 1:10,000) at 3min intervals until cardiac output restored. Transvenous pacing wires should be placed immediately if epicardial wires are not functional/present.

### Dangerous Dysrhythmias

**Second-degree heart block**  
**Trifascicular block**  
Left bundle branch block

### Benign Dysrhythmias

**1st-degree heart block**  
**Sinus or junctional bradycardia**

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Cardiac Pacing Overview

- For maximum CO the optimum HR in the immediate PO period is 90bpm, and the optimum rhythm is SR.
- But dysrhythmias and block are common and pacing is often required to optimize HR and rhythm.

Pacing modes

**TABLE 4–4. Temporary pacing modes**

<p>| | |</p>
<table>
<thead>
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<tbody>
<tr>
<td>I</td>
<td>Chamber paced.</td>
</tr>
<tr>
<td>II</td>
<td>Chamber sensed.</td>
</tr>
<tr>
<td>III</td>
<td>Response to sensing.</td>
</tr>
<tr>
<td>A</td>
<td>Atrium.</td>
</tr>
<tr>
<td>V</td>
<td>Ventricle.</td>
</tr>
<tr>
<td>D</td>
<td>Dual chamber (both atrium and ventricle).</td>
</tr>
<tr>
<td>D</td>
<td>Dual effect (inhibits and triggers pacing).</td>
</tr>
<tr>
<td>I</td>
<td>Inhibits pacing.</td>
</tr>
<tr>
<td>O</td>
<td>No effect on pacing.</td>
</tr>
</tbody>
</table>

Atrial pacing

- AOO: asynchronous atrial pacing—useful if using electrocautery.
- AAI: Demand atrial pacing. For effective atrial pacing normal AV conduction is required. It is ineffective during AF and atrial flutter. Pacing amplitude is 3–20mA. Indications include:
  - Sinus bradycardia.
  - Junctional bradycardia.
  - Suppression of PACs and PVCs, if pacing is set at a slightly faster rate. SVT and JT, and AFI can be treated by overdrive pacing.

Ventricular pacing

- VOO: asynchronous ventricular pacing. Not often used because of the risk of VF if pacing spikes coincide with the T wave of intrinsic rhythm. Useful during surgery if patient has no underlying rhythm when electrocautery interferes with pacing.
- VVI: demand ventricular pacing. For effective ventricular pacing set the pacing amplitude to 3–20mA. Ventricular pacing is much less effective than atrial and dual chamber pacing because of loss of synchronized AV contraction. Indications:
  - Slow ventricular response rate to AF and atrial flutter.
  - Any bradycardia with no atrial wires or failure of atrial pacing.
- Overdrive pacing of supra-ventricular tachycardia below.

Dual chamber pacing

- DOO: asynchronous dual chamber pacing. Caution as for VOO.
- DVI: dual chamber pacing, atrial asynchronous and ventricular demand.
- DDD: dual chamber pacing, dual chamber demand. Effective dual chamber pacing requires atrial and ventricular pacing wires, but does not require normal AV conduction. Dual chamber pacing is more effective than ventricular pacing as synchronized AV contraction is maintained.

Overdrive pacing

This is occasionally used to treat supraventricular tachycardias. Select the maximum pacing output (20mA) and atrially pace at a rate 20bpm faster than the atrial rate (up to 600bpm!). When capture is achieved abruptly turn off the pacemaker: sinus rhythm may result.

Pacing System Malfunctions

Problems with pacing

- Failure to pace—no pacing spikes:

Anatomic Principles

Mature conduction system

- Sinoatrial (SA) node: spindle-shaped bundle with hear extending toward intra-atrial groove, tail toward IVC.
- 2) Preferential conduction but not anatomical pathway from SA to atrioventricular (AV) node.
- Atrial muscle tract in ant limbus of fossa ovalis, crista terminalis and continuation to atrial septum.
- No gross anatomical landmarks for AV node and bundle of His- located within Triangle of Koch.
- Tendon of Todaro superiorly.
- Tricuspid annulus inferiorly.
- Coronary sinus posteriorly.
- Apex in central fibrous body and atrial portion of membranous septum.
- AV node is usually far removed (anteriorly) from coronary sinus.
- AV node becomes a penetrating bundle at apex of triangle, passes into ventricular septum.
- Branching and non-branching bundles are sandwiched between muscular ventricular and membranous septum.
- Compact node, penetrating bundle and branching bundle form a continuous axis of cells running the length of the ventricular septum.

Endocardial anatomy

Atrial leads are designed for placement in (pectinate muscle) atrial appendage Ventricular leads– apex (trabeculae).

Physics/Engineering

- \( V=IR \); where \( V \) = voltage, \( I \) = current (mAmPS) and \( R \) = resistance (Kohms).
- \( E=VI; \) where \( E \) = energy (Joules), \( t \) = time (pulselwidth PW in msec).
- \( E=(V^2) \times PW/R \) (??is that V squared or x2?)

The size of the distal tip is inversely proportional to the concentration of the electrical charge, and therefore the amount of energy required to capture myocardium. Sensing intra-myocardial electrical activity: Circuitry includes: sensing amplifier, bandpass filter, threshold comparator.

Electrophysiology of cardiac pacing

- Myocardial cells can be depolarized by artificial electrical stimulation. Impulse initiation and propagation.
- Resting potential (~90 mV in ventricular tissue, ~50 mV in SA and AV nodal tissue.
- Function of intracellular and extracellular K+.
Failure to pace—pacing spikes present:
• Increasing threshold:
• Oversensing:
• Pacing diaphragm

Indications
A. Symptomatic bradycardia
1) Syncope, dizziness, exercise intolerance, CHF
B. Asymptomatic bradycardia
1) profound bradycardia, usually brief
2) congenital third degree heart block with wide QRS complex
3) BBB?? with intermittent second degree heart block post-MI
4) bifascicular block with intermittent second degree heart block
5) IHSS

Lead placing

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<th>B. Ventricular</th>
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<td>3) Sensing &gt;/= 2 mVolt</td>
<td>3) Sensing &gt;/= 5 mVolts</td>
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<td>Sensing &gt;/= 5 mVolts</td>
<td>Slow rate &gt;/= 0.8 mVolts/sec.</td>
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<tr>
<td>Slow rate &gt;/= 0.8 mVolts/sec.</td>
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Factors affecting sensing
• Electrode size,
• Unipolar/ bipolar configuration
• Lead position

Complications
A. Implantation
• Venous access
• Wire trauma
• Generator
B. Post-surgical
• Late perforation
• Venous thrombosis
• Loss of capture in a lead
  • a) Lead fracture- high impedance
  • b) Insulation break- low impedance
  • c) Exit block- fibrosis around electrode inhibits conduction
• Pacemaker mediated tachycardia- macoreentrant circuit
• Infection- treat with generator/ lead removal.

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Internal Cardioverter Defibrillators (ICD)

"ICDs are programmed to detect tachydysrhythmias, deliver the therapy, and detect if the therapy was successful or not. Multiple different zones of detection can be set (e.g., VT, fast VT, and VF), and the therapies can be individualized for each particular zone. Delivered therapy can be in the form of high-energy defibrillation shocks, low-energy synchronized cardioversions, or antitachycardia pacing.

According to ACC/AHA Guidelines, ICDs are indicated (Class I recommendations) for the following patients:

- Survivors of VF or hemodynamically unstable sustained VT in the absence of identified and treated causes that resolved the dysrhythmia (e.g., coronary artery disease, structural cardiac disease)
- Structural heart disease with sustained VT (stable or unstable)
- Sustained or inducible (during study) VT with associated syncope of otherwise indeterminate origin
- EF < 35% (nonischemic or at least 40 days post-MI), with NYHA Class II or III CHF; EF < 30% if Class I CHF
- Non-sustained VT after an MI if EF < 40%
- Cardiac resynchronization therapy (biventricular pacing), with or without an ICD, is indicated for patients with NYHA Class III or IV CHF with EF < 35%, QRS duration ≥ 0.12 sec, and sinus rhythm.

Contraindications

- The presence of active infection is a contraindication for placement of a permanent pacemaker or ICD. If a pacemaker is needed in the setting of infection, a temporary transvenous pacemaker or an externalized pulse generator is recommended.
- Placement of cardiac devices is also contraindicated with inadequately treated coagulopathy.
- ICDs are not recommended for patients with reversible causes of arrhythmia or a life expectancy < 12 months, or for psychiatric patients that may be adversely affected by ICD shocks.

Complications

- Significant complications occur in < 2% of transvenous devices.
- Early
  - Injury to systemic veins and right-sided cardiac structures (including RA perforation with tamponade or
  - Traumatic tricuspid regurgitation),
  - Pneumothorax,
  - Hemothorax,
  - Air embolus,
  - Venous thrombosis,
  - Dysrhythmias.
- Late
  - Infection of the pocket or leads, which occurs in 3% of patients and pocket erosion.
  - Lead fracture, lead malfunction (usually detected by pacing thresholds).
  - Venous thrombosis.

Key Points

Preoperatively

- Identify the generator manufacturer and whether the generator is a pacemaker or defibrillator.
- Have the pacemaker or defibrillator interrogated by a competent authority shortly before administration of the anesthetic.
• Obtain a copy of this interrogation. Ensure that the device will pace the heart.
• Consider replacing any device near its elective replacement period in a patient scheduled to undergo either major surgery or surgery within 25 cm of the generator.
• Determine the patient’s underlying rate and rhythm, which then determines the need for backup (external) pacing support.
• Identify the magnet rate and rhythm, if any.
• Program minute ventilation rate responsiveness off, if present.
• Program all rate enhancements off.
• Consider increasing the lower rate limit to optimize oxygen delivery to tissues for major cases.
• Disable antitachycardia therapy if a defibrillator is present.

**Intraoperatively**
• Monitor cardiac rhythm with pulse oximetry (plethysmography) or arterial waveform analysis.
• Ask the surgeon to operate without the monopolar electrosurgical unit (ESU).
• Use a bipolar ESU if possible; if not possible, a pure cut is better than “blend” or “coag.”
• Place the ESU return pad so that electricity is prevented from crossing the generator-heart circuit, even if the pad must be placed on the distal part of the forearm and the wire covered with sterile drape.
• If the ESU causes ventricular oversensing and pacer quiescence, limit the period or periods of asystole.

**Postoperatively**
• Have the device interrogated by a competent authority immediately postoperatively. Some rate enhancements can be reinitiated, and the optimum heart rate and pacing parameters should be determined. Note that any patient with disabled antitachycardia therapy must be monitored until the antitachycardia therapy is restored.

*Adapted from the ASA Practice Advisory for Perioperative Management of Patients with a Pacemaker or Defibrillator.*

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General Pathologic Considerations

Patient population at risk

- Usually related to some form of cardiac disease
- Patients most at risk for life-threatening are:
  1. Those sustaining sudden cardiac death without identified cardiac disease
  2. Those with known cardiac disease (including post-MI)
  3. Those with structural heart defects with known arrhythmogenic potential
     a) Long QT, arrhythmogenic dysplasia
- **SCD** (350,000-400,000 per year)
  1. Often during mild-to-moderate exercise
  2. More than 30% of survivors are discharged without neurologic deficit
  3. 60% chance of recurrence within 2 years
  4. Increased risk of: proximal LAD stenosis, regional LV dysfunction, LV failure (CHF)
  5. Overall 50% 5 year survival vs. 80% for age matched cohorts
  6. ~70% are not MI related
     a) Greater risk of recurrence
     b) 2/3 will have significant CAD
- VT following MI results in 40-80% mortality
  1. Prior infarct with aneurysm yields an even greater risk
If CAD present, CABG better than anti-anginal treatment

Pathophysiology

- **Wide QRS >14 msec** is diagnostic of Ventricular- tachycardia (nl=<80 msec)
- Polymorphic (vs monomorphic)
  - No constant morphology for > 5 complexes, or
  - No clear isoelectric baseline, or
  - QRS complexes asynchronous in multiple leads
- Frequently degenerates into VF
- **Sustained tachycardia** – at least 15 seconds
- **Possible mechanisms:**
  - Reentry
  - Normal/abnormal automaticity
  - Triggered activity due to after-depolarization
- Action potential
  - Resting membrane potential (electrical diastole = -85 mV)
  - Electrical, mechanical or chemical signal reduces membrane potential
  - At threshold, cell will depolarize (phase 0), reversing polarity to +30 mV
- **Automaticity**
  - RMP (phase 4) is characteristically constant
  - Some specialized cells will automatically depolarize until they reach threshold, depolarize and initiate beat
  - Depolarization is usually most rapid in SA node, which dominates rhythm
- **Peri-MI tachycardias.** Associated with: hypoxemia, hypocalcemia, catecholamines, drugs (digitalis)
  - Usually responsive to: lidocaine, procainamide, Beta- blockade, discontinuing sympathomimetics
  - Phase 4 dependent are not induced or terminated by EP testing and are rarely approached surgically
  - Triggered automaticity. Arises during repolarization when late after-depolarizations reach threshold
- **Re-entrant**
  - Two or more electrically heterogenous pathways of varying conduction or refractoriness
• Unilateral block
• Slow conduction over alternative route
• Delayed excitation just distal to blocked tissue
• Re-excitation of proximal tissue upon return of the impulse
• Pathophysiologic substrate for ventricular
Chronic CAD (ischemia). Usually gives rise to reentrant arrhythmia. Alters conduction and refractoriness
Acute infarction
• Peri-ischemic areas of abnormal, viable tissue
• Alters conduction and refractoriness
• Heterogeneous infarcts are more arrhythmogenic than homogenous infarcts
• Effect of thrombolytic therapy on then arrhythmogenic substrate. Clinically, no significant increase in life-threatening

Treatment
Programmed Electrical Stimulation (EPS)
1) Rapid ventricular pacing. Burst pacing at 250 bpm. Single, double or triple stimulus to terminate arrhythmia. Hemodynamic instability may require instability
2) Premature ventricular stimuli
Depolarization introduced in late stimuli, then earlier, until no ventricular response is elicited
In no VT, double stimuli introduced 50-100ms after refractory period
S1 (fixed), S2 (premature stimulus), S1-S2 interval reduced by 10ms
When S2 no longer initiates ventricular response, S3 is initiated, S2-S3 interval then decreased
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Lethal Dysrhythmias

Ventricular fibrillation
Pulseless ventricular tachycardia
Asystole
Pulseless electrical activity (PEA)
Complete heart block

Dangerous Atrial Dysrhythmias

These are potentially dangerous in patients with: (1) borderline CO, (2) stiff, non-compliant hypertrophic ventricles where ↓ filling times → ↓ SV, and (3) SAM. Loss of synchronized atrial contraction → ↓ SV by up to 30%.

AFib and atrial flutter
Supraventricular tachycardia
Other Dangerous Dysrhythmias

This section describes the management of VT in *hemodynamically stable* patients. If the patient has lost CO get help and follow the ALS algorithm.

**Ventricular tachycardia (pulse present)**

**Features:** wide complex regular tachycardia, impaired CO.

**Therapy:**
- If at any stage the patient loses cardiac output start the ALS algorithm for VF/pulseless VT.
- Correct hypokalemia: give 20mmol KCl in 50mL 5% dextrose via the central line over 10min, and repeat as necessary to obtain serum K⁺ of 4.5–5.0mmol/L.
- Correct hypomagnesemia: give 1–2g MgSO₄ in 50mL 5% dextrose via the central line empirically if it has not already been given, as up to 60% of patients are hypomagnesemic postoperatively, and serum magnesium represents only 1% of total body stores.
- Correct hypoxia (b p160 and acidosis.
- Start weaning potentially arrhythmogenic infusions, e.g., isoprenaline.
- If the patient is hemodynamically compromised sedate with the help of an anesthesiologist and give 1 x 200J synchronized biphasic shock, and repeat if needed.
- Amiodarone 300mg in 50mL 5% dextrose over 1h via central line, followed by amiodarone 900mg in 250mL 5% dextrose over 23h via central line may achieve rate control and is first choice agent in most units.
- Alternatively lidocaine 1mg/kg iv bolus, followed by an iv infusion of 4mg/min for 30min, 2mg/min for 2h, then 1 mg/min may achieve cardioversion.
- Look for and treat any evidence of myocardial ischemia.

**Ventricular ectopy**

**Features:** wide complexes that may occur occasionally or in couplets (ventricular bigeminy), may be unifocal or multifocal, and are usually followed by a compensatory pause.

**Therapy:** although ventricular ectopics occurring less than one per screen are usually benign, particularly if present preoperatively, in a small number of patients they reflect myocardial ischemia and may herald lethal arrhythmia.
- Look for signs of myocardial ischemia and treat accordingly (b p185).
- Correct hypokalemia: give 20mmol KCl in 50mL 5% dextrose via the central line over 10min, and repeat as necessary to obtain serum K⁺ of 4.5–5.0mmol/L.
- Correct hypomagnesemia: give 1–2g MGSO₄ in 50mL 5% dextrose via the central line empirically if it has not already been given, as up to 60% of patients are hypomagnesemic postoperatively, and serum magnesium represents only 1% of total body stores.

**Other Dangerous**

Correct hypoxia and acidosis.
Atrial pacing at a rate greater than the ventricular rate may abolish ectopics and improve cardiac output, but does not address any underlying pathology.

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Junctional tachycardia

Atrial ectopies

Supraventricular Tachycardias (SVT)

AFib/Flutter

General Pathologic Considerations

A. Mechanism
• Multiple macro re-entrant circuits
• Atrial refractory period
• Structural, fibrosis, SVC, IVC, ASD

B. Effects
• Irregular heart beats
• Loss of AV synchrony
• Risk of thromboembolism
• Normal Atrial Activation
• Atrial Flutter
• LA Isolation Procedure

Pharmacological cardioversion: This is indicated in hemodynamically stable postoperative patients. A. Rate Control
1) Digitalis, Beta-blockers, calcium channel blockers
2) Prevention
3) IA- Quinidine, procainamide, disoprymadine,
4) IC- Flecanide, propafenone
5) III- Solatol, amiodarone
Surgery for SVT

Introduction
Radiofrequency (RF) ablation has replaced surgery as treatment of choice for certain re-entrant
• Accessory pathways-- Wolff-Parkinson-White Syndrome
• AV nodal pathways from perinodal pathways
• Ectopic atrial tachycardias
• Certain Type I atrial flutter

Surgery for RF ablation failures
Maze procedure for chronic paroxysmal or sustained AFib/flutter

Accessory atrioventricular connections
Complications of RF catheter ablation (RFCA) (may impact surgical technique)
• Scarring and fibrosing relate directly to the amount of energy delivered
• Direct RF ablation to LA side of A-V groove has resulted in the destruction of planes, injury to Cx, CA and CS
• Excessive energy to RA planes has resulted in destruction of planes

Surgical Treatment
Indications
• Recurrent reciprocating tachycardia
• Poorly controlled (?refractory?) or toxic on medical treatment
• failed RF ablation or require surgery for concomitant disease
• Symptomatic, atrio-His, nodoventricular, fasciculoventricular fibers

Locating pathways
• Locate preoperatively with EP studies and epicardial mapping
• Activation sequence mapping on atrial and ventricular sides of A-V groove
• Accessory pathways are located in: left free wall> posterior septal> right free wall> anterior septal

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Surgical approach

AV Nodal reentrant tachycardia
Dual A-V conduction pathways, one fast, one slow through AV node or perinodal tissues

Surgical treatment
• Surgery for failure of RFCA or concomitant disease
• Monitor A-V conduction time during application of cryolesions
• Apply lesions to boundaries of Triangle of Koch
• Impending heart block signaled by an A-V interval of 200-300ms- "reversible knife"
• A-V nodal reentrant tachycardia and accessory A-V connections should undergo treatment together
• Nodoventricular and nodo-His corrections can be concurrently performed

Surgical Results
• Only a single pathway remains
• No early or late recurrence
• Dissection of tissue anterior or posterior to A-V node has resulted in A-V block plus late recurrence

Ectopic or automatic atrial tachycardias
More common on right than left, may be multifocal

Surgical treatment
Preoperative localization necessary
• GA may suppress ectopic focus
• Intraoperative mapping without sophisticated computerized system is
difficult
• Ectopic tachy dysrhythmias not inducible by standard programmed stimulation techniques

Options
• Cryoablation
• Wide excision with pericardial patch
• Ablation and pericardial patch
• Isolation

**Left atrial focus isolation** (left atrium remains tachycardic)
a) Option is to ablate His bundle, place pacemaker

**Right atrial foci** if circuit or focus cannot be localized
a) Option is to isolate

**Surgical Results**
• No recurrence
• No sequelae

Atrial flutter and fibrillation (§ p)

**It is AFib better than AF (NOT to be confused with VF)**

"Surgical Cure" or AFib
• Elimination of clinical arrhythmia
• Maintenance of SA nodal tissue as driving impulse
• Maintenance of A-V conduction
• Restoration of atrial transport function
• Items 1-4 reduce the risk of thromboembolism

**Idiopathic AFib (Maze procedure)**
• Drug resistant, medically refractory, symptomatic idiopathic (non- rheumatic) fibrillation
• Mean age = 56 y.o., concomitant procedure in 28% or patients
• 40% require postoperative pacemakers- atrial chronotropic incompetence
• Atrial transport- 100% RA, 81% LA
• Local effective refractory period (ERP)- shorter in LA than RA
• RA more susceptible to reentrant atrial flutter
• LA more susceptible to reentrant AFib

**AFib associated with degenerative mitral valve disease- indications for Maze procedure**
• <70 years old
• Normal ventricular function
• History of H/O embolic events secondary to more than one year of AFib
• Medically refractory and severely symptomatic
• LA dimension >60 mm
• Easily repairable valve

**Maze procedure**- indications for rheumatic mitral valve disease. Modifications usually involve LA (forms of LA isolation)

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Cardioversion

Synchronized DC cardioversion is the treatment of choice for tachyarrhythmias compromising cardiac output, such as AF and SVT, and for AFIB refractory to chemical cardioversion.

DC cardioversion for AF and SVT

Complications of DC cardioversion
- Complications of general anesthesia.
- Systemic embolization.
- Failure to cardiovert.
- Burns from incorrect application of gel pads.
- Muscle pain from involuntary contraction.
- Dysrhythmias including asystole and VF. Hemodynamic improvement after cardioversion is more likely when the initial HR is very fast. Patients with a HR <150 b/m are less likely to improve immediately following cardioversion.
- Patients treated with negative chronotrophic agents prior to cardioversion may be pushed in a slow sinus rhythm without the ability to mount a compensatory sinus tachycardia.
- For patients with severe systolic dysfunction, this can precipitate cardiogenic shock.
- For patients with chronic AF in septic shock, attempting to “normalize” the heart rate will reduce the cardiac output. Such patients may benefit from permissive tachycardia.

Common pitfalls
- Failure to deliver a shock:
- Failure to cardiovert:

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Defibrillation

External Defibrillation
- Do not delay defibrillation for maneuvers such as intubation, massage, or administration of drugs.
- Expose chest. Place gel pads on chest the aim is to direct as much of the current as possible through the heart.
- Switch defibrillator on and turn dial on to appropriate power setting (200J for external defibrillation). Press charge button.
- If you are using hand-held paddles instead of adhesive external defibrillator pads, place them firmly on gel electrodes and hold.
- Perform a visual sweep to check that no one is in contact with the patient at the same time as saying clearly, ‘Charging. Stand clear.’ Press the red/orange shock button on the paddles.
- If the shock has been delivered successfully the patient’s muscles will contract violently: personnel in contact with the patient may experience an electric shock.
- Check the rhythm: if VF charge again and repeat the sequence. If the rhythm changes to one compatible with an output check the pulse before proceeding further.
Internal Defibrillation

Familiarize yourself early on with how to connect the internal paddles to the defibrillator console: it’s different in most models, ICU nurses may not be familiar with how to do this, and an emergency resternotomy is not the time to be trying to figure it out.

In some models one of the external paddles disconnects at the handle, and this is the part that the internal paddle lead plugs into, in other models the internal paddles plug directly into the console.

Switch defibrillator on and turn dial on to appropriate power setting (30–50J for internal defibrillation) before you scrub.

Hand out lead for internal paddles and connect them to defibrillator. Nurse will need to press charge button. Place concave surface of paddles squarely and firmly over LV and RV taking real care not to dislodge coronary anastomoses (placing the anterior paddle into the left pleura reduces the risk of damaging the LIMA to LAD) or pacing wires, or lacerate the myocardium (the paddles are relatively sharp edged) and press button on paddle handles to deliver shock.

Common pitfalls

Failure to deliver a shock:
Failure to defibrillate:

Internal cardiac massage and defibrillation

If the chest is open internal defibrillator paddles can be used. Shocks are given starting at 30J to a maximum of 50J. Remove the paddles with caution as rough contact with the myocardium may be enough to cause the heart to revert into VF. Internal cardiac massage should not be performed by inexperienced hands because of the risk of AV dehiscence, RV or LV rupture, or dislodging bypass grafts. The safest method is, standing on the patient’s right, to carefully place your right hand under the ventricles, your left hand over the ventricles and compress carefully with the flat of your hands, not fingertips (Fig. 5.19c). One-handed massage is less effective and more likely to rupture the RV. If the patient is in asystole or complete heart block epicardial pacing wires can easily be placed, so that pacing can commence.

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Surgery for AFib

It is AFib better than AF (NOT to be confused with VF)

Indications:
Patients with chronic AFib (greater than six months) and left atrial size greater than 5.5 cms with associated mitral valve disease. Left atrial size is measured on the transthoracic echocardiogram at the time of aortic valve closure in the M-mode echocardiogram in the parasternal long axis view.

Patients receiving mitral valve surgery (repair/commisurotomy/replacement), with concomitant tricuspid or aortic surgery being performed.

Contraindications

Operative Procedure

The Cox maze III procedure The cut and suture technique:
Radio frequency (RF) maze:
The electrocautery (EC) maze:
The output waveforms are as follows: Valleylab Force 40 S™ – 500 kHz

Multimodal attack of AF (maze + amiodarone + optional atrial pacing) ensures a controlled sinus rhythm throughout the immediate postoperative period and makes it smoother.
damped sinusoidal bursts with a repetition frequency of 31.25 kHz, rated load of 300 ohms and power output being 40 watts. Conmed Excalibur Plus PC™ – 540 kHz damped sinusoidal bursts with a repetition frequency of 20 kHz, a rated load of 500 ohms and power output being 40 watts. The theoretical energy delivery is approximately 40 Joules for every second of cauterization. The right atrial lesions are as follows:

• From the posterior wall of the SVC – RA Junction (just caudal to the level of the SA node), down across the fossa ovalis to the IVC cannula veering towards the mouth of the coronary sinus and burning the inferior mouth of the coronary sinus including as much of the ostium as possible (Figure 1, ).

• From the IVC burning the atrial isthmus and proceeding to the tricuspid valve orifice at 5 o’clock (Figure 1, ).

• From the middle of lesion 1 laterally towards the atrial wall, burning the atrial “Crista” and proceeding further laterally to meet the atriotomy (Figure 1, ). The right sided lesions are performed on the beating perfused heart primarily to see the effect of ablation on the right side. Nearly 60% of cases revert to sinus rhythm or develop a slowing of the heart rate and intermittent P wave formation. The lesion at the coronary ostium can be precisely placed and thus heart blocks can be avoided.

• From the superior end of the atriotomy to the right atrial appendage stopping at the cannulation orifice and then

• Restarting at the diametrically opposite point and continuing the lesion towards the dome of the left atrium.

The retrograde cannula is placed back into the coronary sinus. Any tricuspid procedure that has been planned is performed. The atriotomy is closed and the heart is arrested with antegrade cold blood cardioplegia. During this period, if there is no left atrial thrombus, the left atrium is ligated externally with a silk/linen ligature. The left atriotomy is done after dissecting the interatrial (Sondergaard’s) groove extensively (vertical left atriotomy).

• Any left atrial thrombus is evacuated and all lamellar thrombus in the atrial body is assiduously evacuated. The mitral valve procedure is performed. If the left atrial appendage has not been ligated previously, it is now ligated externally.

• The cautery lesions are placed while controlled warm retrograde normokalemic reperfusion is being done. The lesions are placed circumferentially at one centimeter from the pulmonary vein orifices.

• A lesion touching each of the previous lesions and the mitral annulus at 5 o’clock connects all four lesions. (On a practical basis, if the gap between the left superior and inferior pulmonary vein is small, a common lesion can encircle both pulmonary veins).

• A lesion is placed from this outer lesion to the ligated left atrial appendage.

• In giant left atria with atrial diameter more than 7 cms, an optional cruciate lesion is placed within the circumpulmonary vein lesion.

The left atriotomy is then closed. The heart is de-aired and the patient is weaned off CPB in the routine manner. TEE is done in all the patients. Atrial and ventricular wires were placed in all patients. Care is taken to place the atrial wires as high as possible to enable sinus node recovery time studies.

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**Postoperative Care:** Patients who are in sinus rhythm were put on an infusion of amiodarone (10 mg/Kg/24 hours) and then an oral amiodarone 200 mg/day for three months, and then stopped. Patients who had nodal rhythm were given intravenous aminophylline (18 mg/Kg/24 hours) with an initial 200-mg bolus on CPB, if there was nodal rhythm, and temporary AV pacing was instituted as and when required. All patients who were not on coumadin for valve replacement (i.e., repairs) were placed on 150-mg enteric-coated aspirin life long.
Wolf-Parkinson-White Syndrome (WPW)

General Pathologic Considerations
- Short PR interval wide QRS- delta wave accessory pathway
- Free wall Left Atrium- Type A
- Right Atrium: anterior superior- Type B
- Tricuspid annulus: anterior ventricular septum
- Tricuspid or Mitral posterior ventricular septum
- Reciprocating tachycardia
- AV conduction- anterograde
- Accessory bypass- retrograde
- AFib
- Life-threatening
- Rapid conduction via accessory pathway

Diagnosis: ECHO, Stress testing, and EPS:
- The mechanism of the clinical tachycardia
- The electrophysiologic properties (eg, conduction capability, refractory periods) of the accessory pathway and the normal conduction system
- The number and locations of accessory pathways (necessary for catheter ablation)
- The response to pharmacologic or ablation therapy

Management
- RF ablation of the accessory pathway.
- Antiarrhythmic drugs to slow accessory pathway conduction: termination of acute episodes (± cardioversion) and long-term antiarrhythmics.
- AV nodal blocking medications in adult patients to slow AV nodal conduction in certain situations (ie, Mahaim or atriofascicular pathway-mediated SVT; typically, AV node-conduction blocking medications are avoided in the acute setting)
- For adult WPW patients, address the triggers that perpetuate the dysrhythmia (CAD, ischemia, cardiomyopathy, pericarditis, electrolyte disturbances, thyroid disease, and anemia)/

Radiofrequency ablation
- Patients with symptomatic AVRT
- Patients with AF or other atrial tachydysrhythmias that have rapid ventricular response via an accessory pathway (preexcited AF)
- Patients with AVRT or AF with rapid ventricular rates found incidentally during EPS for unrelated dysrhythmia, if the shortest preexcited RR interval during AF is less than 250 ms
- Asymptomatic patients with ventricular preexcitation whose livelihood, profession, insurability, or mental well-being may be influenced by unpredictable tachydysrhythmias or in whom such tachydysrhythmias would endanger the public safety.
- Patients with WPW and a family history of SCD

Surgical treatment
- RF catheter ablation has virtually eliminated surgical open heart treatments in the vast majority of WPW patients, with the following exceptions:
- Patients in whom RF catheter ablation (with repeated attempts) fails
- Patients undergoing concomitant cardiac surgery (possible exception)
- Patients with other tachycardias with multiple foci who require surgical intervention.

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5 Cardiac Arrest & CPCR

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Unit Objectives

• To review the basic concepts of CARDIAC ARREST AND ITS TYPES.
• The steps & sequence if CPR
• CPR in adults
• CPR in children
• CPR in neonates

Learning Objectives

By the end of this chapter the student should know:

• Cardiac Arrest in Adults: Definitions; Cardiopulmonary Cerebral Resuscitation (CPCR); American Heart Association CPR guidelines
  • Closed Chest Massage (CCM)
  • Airway
  • Breathing
• Ventricular Tachycardia/Fibrillation (VT/VF)
• Asystole
• Pulseless Electrical Activity /Electromechanical dissociation
  • Open Cardiac Massage (OCM)
• Cerebral Protection/Resuscitation
• Cardiac Arrest in Children
• Cardiac Arrest in Neonates

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• Chest compressions performed with minimal interruption are critically important in improving the chance of survival from sudden cardiac arrest.
• Thirty chest compressions followed by two rescue breaths is the recommended ratio for single rescuers resuscitating children and adults.
• Chest compressions should be performed at a rate of 100/min for children and adults.
• The American Heart Association 2005 guidelines recommend that a single shock be delivered when a shockable dysrhythmia exists, followed by resumption of chest compressions as soon as the shock is delivered. Two minutes of chest compressions and ventilation should be performed before reassessing the underlying cardiac rhythm.
• Automated external defibrillators (AEDs) may follow an outdated defibrillation protocol (e.g., three defibrillation shocks before resumption of cardiopulmonary resuscitation [CPR]). In this circumstance, the rescuer should allow the AED to function as programmed until a manual defibrillator becomes available.
• When a rescuer is unfamiliar with the type of manual defibrillator used during resuscitation, a default energy of 200 J is a reasonable energy level for defibrillation.
• In an unwitnessed cardiac arrest or in situations in which initiation of CPR has been delayed, 2 minutes of CPR before the first defibrillation has been shown to have survival benefit.
• Hyperventilation during resuscitation ↑↑ intrathoracic pressure, ↓ venous return to the heart, and has a negative impact on survival from cardiac arrest. The resuscitation team leader must ensure that only 8 to 10 breaths per minute are being delivered during resuscitation attempts.
• Therapeutic hypothermia has demonstrated benefit in improving the neurologic outcomes of victims resuscitated from out-of-hospital VF who remain comatose on hospital admission. This therapeutic intervention
should be considered for victims of in-hospital VF cardiac arrest with decreased neurologic function after resuscitation.

- Resuscitation knowledge declines rapidly, even in anesthesia providers. Review of advanced cardiac life support (ACLS) resuscitation protocols may be necessary in the interim between ACLS certification. ACLS protocols should be made available on resuscitation carts to ensure that more uniform and accepted resuscitation interventions are followed during resuscitation attempts.

**Pediatric and Neonatal Intensive Care**

- Critically ill children cared for in PICU have improved outcomes when compared with children treated in ICUs that do not specialize in the treatment of children.
- The autonomic nervous system has predominantly a parasympathetic vagal tone at birth and gradually shifts to a sympathetic tone in older children.
- Because the Frank-Starling mechanism is less effective in the newborn, a greater but not exclusive dependence on HR is necessary for maintenance of CO.
- Microprocessor technology in ventilators has made volume-preset modes of ventilation possible in babies and small children.
- A multidisciplinary task force has developed a set of guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents.
- The leading causes of death in children 1 to 14 years of age are accidents and trauma.
- Pediatric critical care transport systems not only provide a means of transferring a sick patient to a more appropriate facility but also initiate appropriate intensive care monitoring and treatment of the patient before leaving the referring hospital.
- Mortality from sudden infant death syndrome dropped threefold with institution of the “Back to Sleep” campaign.
- Pediatric cardiopulmonary arrest is usually manifested initially as respiratory compromise or arrest, followed by secondary cardiac arrest.

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Cardiac Arrest in Adults

Definitions

Sudden cardiac death (SCD) is the unexpected, nontraumatic abrupt cessation of effective cardiac function in a patient with either no symptoms or acute symptoms for less than 1 hour. Prodromal symptoms such as chest pain, palpitations, and fatigue may be present within the preceding 24 hours.

Cardiac arrest is the final manifestation for 5 different pathophysiologic conditions:
- Ventricular fibrillation (VF),
- Pulseless VT,
- Asystole,
- Electromechanical dissociation (EMD), also called pulseless electrical activity.
- Pulsless bradycardia

These 5 malignant nonperfusing lethal dysrhythmias have different etiologies, priorities of treatment, and prognoses. Early identification of the underlying dysrhythmia is vital for successful resuscitation.

Post-cardiac Arrest Syndrome ("Post-Resuscitation Syndrome"). This is the abnormal physiological state which occurs when whole-body ischaemia is followed by whole-body reperfusion. In summary, it is a systemic inflammatory state which resembles every other form of vasodilatory shock; the degree of organ dysfunction depends on the sensitivity of those organs to ischaemia, and the duration of ischaemic time.

"Reanimatology" is a term coined by Negovsky of Moscow, meaning “the science of resuscitation medicine.” With “anima” meaning the mind, reanimation means brain orientation. Resuscitation medicine is much more than "tilting, blowing, pumping, and zapping." It encompasses the pathophysiology of acute terminal states and clinical death; their reversibility with basic, advanced, and prolonged life support of the cardiopulmonary cerebral resuscitation (CPCR) system; and the emergency medical services (EMS) delivery system. "Reanimatology" of Europe I consider synonymous with "emergency and critical (intensive) care medicine (ECCM)” in the United States.

TABLE 5-2. Contributing causes of cardiac arrest

<table>
<thead>
<tr>
<th>The 6 Hs</th>
<th>The 5 Ts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemia</td>
<td>Toxins</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>Tamponade, cardiac</td>
</tr>
<tr>
<td>Hydrogen ion (acidosis)</td>
<td>Tension, pneumothorax</td>
</tr>
<tr>
<td>Hypokalemia/Hyperkalemia</td>
<td>Thrombosis (coronary or pulmonary)</td>
</tr>
<tr>
<td>Hypothermia</td>
<td></td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>Trauma</td>
</tr>
</tbody>
</table>

Cardiopulmonary Cerebral Resuscitation (CPCR)

In 2005, the American Heart Association (AHA) released updated guidelines based on the International Consensus Conference on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment recommendations. These recommendations are based on both experimental data and expert consensus. The new guidelines incorporated significant changes in the algorithms in the treatment of cardiac arrest (TABLE 5-3). The AHA also identified future areas of research that may impact outcomes in cases of cardiac arrest. These changes include the manner in which CPR is to be carried out with increased emphasis on the continuity of chest compressions with minimal interruptions. This issue of Emergency Medicine
Practice highlights significant changes in the 2005 AHA guidelines, examines the evidence that prompted the changes, and explores future therapies that may impact outcomes from SCD.

Important Changes In The 2005 AHA Guidelines For CPR And Emergency Cardiovascular Care

### TABLE 5-3. Differences between the recommendations of 2000 & 2005

<table>
<thead>
<tr>
<th>Measure</th>
<th>2000 Recommendation</th>
<th>2005 Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immediate defibrillation for unwitnessed</td>
<td>Recommended</td>
<td>5 cycles of CPR prior to shock is recommended</td>
</tr>
<tr>
<td>Compression: ventilation ratio</td>
<td>15:2</td>
<td>30:2</td>
</tr>
<tr>
<td>Sequence of defibrillation</td>
<td>3 stacked shocks</td>
<td>1 shock only followed by immediate CPR</td>
</tr>
<tr>
<td>Rhythm/pulse check</td>
<td>After each shock</td>
<td>After 5 cycles of CPR following each shock</td>
</tr>
</tbody>
</table>

American Heart Association CPR guidelines

### TABLE 5-4. In 2010, the Emergency Cardiovascular Care Committee (ECC) of the AHA released the Association’s newest set of guidelines for CPR. Changes for 2010 include the following:

- The initial sequence of steps is changed from ABC (airway, breathing, chest compressions) to CAB (chest compressions, airway, breathing), except for newborns
- “Look, listen, and feel” is no longer recommended
- The compression depth for adults should be at least 2 inches (instead of up to 2 inches)
- The compression rate should be at least 100/min
- Emergency cardiac treatments no longer recommended include routine atropine for pulseless electrical activity (PEA)/asystole, cricoid pressure (with CPR), and airway suctioning for all newborns (except those with obvious obstruction).
- Post–cardiac arrest care is covered in a new section.

In its full, standard form, cardiopulmonary resuscitation (CPR) comprises 3 steps: chest compressions, airway, and breathing (CAB), to be performed in that order in accordance with the 2010 American Heart Association (AHA) guidelines.

Note that artificial respirations are no longer recommended for bystander rescuers; thus, lay rescuers should perform compression-only CPR (COCPR). Healthcare providers, however, should perform all 3 components of CPR (chest compressions, airway, and breathing). For an unconscious adult, CPR is initiated using 30 chest compressions. Perform the head-tilt chin-lift maneuver to open the airway and determine if the patient is breathing. Before beginning ventilations, rule out airway obstruction by looking in the patient’s mouth for a foreign body blocking the patient’s airway. CPR in the presence of an airway obstruction results in ineffective ventilation/oxygenation and may lead to worsening hypoxemia.

The techniques described here refer specifically to CPR as prescribed by the Basic Cardiac Life Support (BCLS) guidelines. In the in-hospital setting, or when a paramedic or other advanced provider is present in the out-of-hospital setting, Advanced Cardiac Life Support (ACLS) guidelines call for a more robust approach to treatment of cardiac arrest, including drug interventions, electrocardiographic (ECG) monitoring, defibrillation, and invasive airway procedures.
Closed Chest Massage (CCM)

It is indicated when the unresponsive patient is pulseless. Absence of a pulse is confirmed by palpation of the carotid or femoral artery. Peripheral pulses, i.e., in the radial artery, are unreliable. CCM is performed with the victim supine on a flat, firm surface. If the patient is in bed, a backboard is placed beneath him or her. The heel of one hand is placed on the lower half of the sternum, and the other hand is placed on top of it.

The thrust of compression is delivered directly downward. The recommended rate is 80 to 100 compressions per minute, and the recommended depth in an adult is 4 to 6 cm (1.5 to 2 in). The compression phase should occupy 50% of the cycle. Pressure is released completely after each downstroke to allow ventricular filling and coronary blood flow. The hands should maintain contact with the chest wall during the release phase to avoid having to reposition them between compressions.

Complications Of Closed Chest Massage

- The most common injuries from chest compressions are rib fracture (30%) and sternal fracture (20%).
- Other common complications include aspiration, gastric dilatation, anterior mediastinal hemorrhage, epicardial hematoma, hemopericardium, myocardial contusion, pneumothorax, coronary air embolus, hemotherax, lung contusion, and oral and dental injuries. The intraabdominal organ injuries are liver (2%) of cases and the spleen (<1%).

Rare injuries (incidence < 1%) include tracheal injuries, esophageal rupture, gastric rupture, cervical spine fracture, vena caval injury, retroperitoneal hemorrhage, and myocardial laceration.

Airway

BLS Airway Management

Management of the unconscious patient: After unresponsiveness is verified and the emergency medical system (EMS) system is activated, the initial step is to ensure a patent airway. The patient is positioned supine on a firm surface. The preferred method of opening the airway is the head-tilt-chin-lift maneuver. An alternative technique is the jaw-thrust technique, which is recommended for patients with suspected head and neck trauma. However, the jaw-thrust is technically difficult and more fatiguing than the head-tilt-chin-lift. Posterior displacement of the tongue is the most common cause of airway obstruction in an unconscious patient. Either of these techniques relieves obstruction due to the tongue.

Once an open airway is established, the rescuer looks for signs of breathing, using the "look, listen, feel" method: Look for chest movement, listen for sounds of air flow, and feel for air movement. If no signs of respiration are present after 3 to 5 seconds, rescue breathing is initiated. If unable to ventilate the patient, the rescuer should reposition and attempt to ventilate again. If still unable to ventilate, airway obstruction from a foreign body should be suspected.
FIGURE 5–9. Algorithm for management adult cardiac arrest (adult bradycardia with pulse).

**Personnel and Protocols**

- Within individual institutions, a protocol should be established to determine which personnel attend cardiac arrests in various parts of the hospital. In some ICUs cardiac arrests are managed in-house; in others, the hospital “crash” team attends. For all cardiac arrests, a structure should be established to determine who is responsible for running the arrest. In the ICU, it is commonly the on-call intensive care physician. This person should be in overall command and should be responsible for determining all treatments. If possible, the person running the arrest should not be involved in administering drugs or performing interventions.
- All medical and nursing staff working within the ICU should be familiar with basic and advanced life support and be regularly recertified in these procedures. Staff should also be familiar with the emergency chest reopening protocol for their units.
- During each arrest, one person should be responsible for documenting the time of the arrest, the dosages of all drugs that are administered, and the times of all interventions. In particular, the time to return of spontaneous circulation should be noted. Each cardiac arrest should be subject to postevent review as part of continuing quality assurance.

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Foreign-body airway obstruction: The Heimlich maneuver, or subdiaphragmatic abdominal thrust, is the recommended method for relief of airway obstruction secondary to foreign bodies.

Advanced Airway Management

- Masks and oral and nasal airways: Oropharyngeal airways are not used in conscious patients because of the risk of laryngospasm and regurgitation.
- Endotracheal intubation (ETT): Esophageal airways.
- Transtracheal catheter ventilation (TTC)
- Cricothyroidotomy:

Breathing

For basic lifesaving with no airway device in place, mouth-to-mouth breathing is recommended. It is easily learned, rapidly applied, and effectively provides oxygen.

Exhaled air has an FIO₂ of 0.16 to 0.17, resulting in an alveolar Po₂ of approximately 80 mmHg. Two slow breaths (2 seconds each) are given initially. A tight seal between rescuer and patient is essential. Chest movement during inspiration and air movement during passive exhalation are confirmed. In adults, 800 to 1200 ml of air are needed to move the chest wall. The recommended rate of rescue breathing is 10 to 12 breaths per minute. Maintaining an open airway during both phases of respiration and giving breaths slowly minimize the risk of gastric distension and aspiration. Mouth-to-nose breathing is recommended when the mouth cannot be opened or a tight mouth-to-mouth seal is not achieved. Cricoid pressure, the Sellick maneuver, may be used to decrease the risk of gastric distension and regurgitation when two rescuers are involved. Barrier devices, such as face shields and mask devices, may be used when available to protect medical personnel from exposure. Supplemental oxygen is administered in the highest concentration feasible when it is available.

<table>
<thead>
<tr>
<th>Ventricular fibrillation</th>
<th>Tachycardia/Fibrillation (VT/VF)</th>
</tr>
</thead>
</table>

**Ventricular fibrillation**

Features: irregular, fine, sinusoidal trace (Fig. 5.14a). No cardiac output.

Therapy: immediate defibrillation. Do not delay defibrillation for any other procedures, e.g., intubation, cardiac massage, starting iv therapy. • 3x shock 2001 (biphasic, if monophasic defibrillator give up to 360J).

- Commence CPR.
- Give 1mg epinephrine iv (10mL of 1:10,000), repeat at 3min intervals.
- Repeat cycle.
- Give 1mg/kg lidocaine, correct acidosis and electrolytes.
- Emergency re-exploration is sometimes indicated for refractory VF in early postoperative period if surgical cause suspected (kinked conduit) or for institution CPB with a view to RVAD or LVAD support.

**Pulseless ventricular tachycardia**

Features: wide complex tachycardia (Fig. 5.14b). No cardiac output.

Therapy: treat as VF.

- Sometimes runs of VT are short and self-limiting. If recurrent these are best treated with lidocaine or amiodarone infusions, magnesium, and correction of acid–base and electrolyte abnormalities.
- Sustained and non-sustained VT may degenerate into VF.

Peter Safar (the father of modern CPR) mnemonics:

A B C D E F G H

The guideline change in 2011: From ABC to CAB
C “hands-only” or “compression-only” CPR (COCPR) or standard CPR
A B
Prognosis
VF/VT has a better prognosis than asystole or EMD, ≥30 % of patients resuscitated successfully. Early defibrillation has been shown conclusively to improve survival in out-of-hospital arrest. It has been reported that an increase in survival to discharge from 7 % with standard CPR to 26 % with defibrillation in the field. Failure to respond to the initial series of shocks is a poor prognostic sign, but efforts should continue. At this point attention should be directed to correcting metabolic problems and providing effective ventilation and circulation, with attempts to defibrillate at appropriate times during the resuscitation.

Notes on ABC:

Asystole

Features: flat EKG trace or low amplitude fibrillation. No cardiac output. Therapy: pace if possible. If not commence CPR immediately.
- If epicardial pacing wires are present pace immediately.
- Give atropine 1mg iv once, and epinephrine 1mg iv at 3min intervals
- Fine, low amplitude VF resembles asystole and may respond to electrical defibrillation: if in doubt defibrillate.
- An absolutely straight line suggests telemetry lead disconnection.
- Treat hyperkalemia aggressively with 50mL 50% dextrose and 15 units insulin iv push.

Prognosis
Asystole has a grim prognosis, with < 2 % of patients surviving to discharge. If asystole occurs after countershock of VF early during a resuscitation, the patient may survive; however, asystole occurs after prolonged CPR, it is typically fatal.

Cardiac arrest in the monitored patient
- The ALS algorithms (see
- Defibrillate VF or pulseless VT.
- Secure an airway.
- Hand ventilate with 100% oxygen at a rate of 10 breaths/min.
- Start external cardiac compressions at a rate of 100/min if defibrillation not possible or not indicated (EMD, asystole).
- Listen for bilateral breath sounds. Look for symmetrical expansion.
- Get iv access, check electrolytes, blood sugars, blood gases.
- Look for reversible causes: mechanical—cardiac tamponade, tension pneumothorax, massive PE, and metabolic—hypokalemia, hyperkalemia, acidosis, hypoxia, and hypovolemia and hypothermia.
- Give 10mL 1:10 000 epinephrine if no response.

Pulseless Electrical Activity /Electromechanical dissociation

Pulseless Electrical Activity (PEA) previously called electromechanical dissociation (EMD). is characterized by organized electrical activity without effective cardiac contractions. The AHA now uses the term pulseless electrical activity (PEA) to encompass EMD, as well as pulseless idioventricular, bradycardic, and ventricular escape rhythms. Pulseless idioventricular, bradycardic, and escape rhythms have an extremely poor prognosis. PEA is the most common proximate cause of death in delayed or difficult resuscitations.

Features: no cardiac output despite EKG trace compatible with output. Therapy: commence CPR immediately and try to pace if possible. Attempt pacing if epicardial wires are present. The priority of treatment of EMD is
identification and treatment of reversible causes. A rapid, narrow-complex rhythm is often an indicator of a treatable etiology of EMD. Patients are rarely resuscitated from EMD if a reversible cause is not found. After reversible causes have been ruled out, the focus is directed toward providing adequate ventilation and effective chest compressions in an attempt to provide effective coronary perfusion. In addition to BLS maneuvers, epinephrine is given to improve cerebral and coronary perfusion pressures. Atropine is indicated if bradycardia is present. Patients with EMD following penetrating chest trauma should undergo emergency left anterolateral thoracotomy. Thoracotomy allows identification and management of reversible causes of EMD, such as tension pneumothorax, cardiac tamponade, and hypovolemia, as well as open cardiac massage and occlusion of the descending thoracic aorta.

TABLE 5–7. Dosage of sympathomimetics.

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dilution</th>
<th>Dose in mcg/kg/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dobutamine (mostly Beta)</td>
<td>500mg in 100ml</td>
<td>2-25</td>
</tr>
<tr>
<td>Dopamine (DA &gt;Beta &gt;Alpha)</td>
<td>400mg in 100ml</td>
<td>5-20</td>
</tr>
<tr>
<td>Norepinephrine (Mostly Alpha)</td>
<td>4mg in 50ml</td>
<td>0.02-0.1</td>
</tr>
<tr>
<td>Epinephrine (Alpha, Beta)</td>
<td>5ml in 50ml</td>
<td>0.02-0.1</td>
</tr>
<tr>
<td>Isoproterenol (Beta)</td>
<td>4mg in 50ml</td>
<td>0.05-0.2</td>
</tr>
<tr>
<td>Milrinone (PDE inhibitor)</td>
<td>100mg in 50ml</td>
<td>0.375-0.75</td>
</tr>
<tr>
<td>Dobexamine (DA1 and Beta2)</td>
<td>50mg in 50ml</td>
<td>0.5-5</td>
</tr>
<tr>
<td>Angiotensin (AI1)</td>
<td>2.5mg in 50ml</td>
<td>0.03-0.3</td>
</tr>
<tr>
<td>Vasopressin (V1)</td>
<td>100 units in 1000ml</td>
<td>0.01-0.08</td>
</tr>
</tbody>
</table>

Only a few drugs are indicated for managing cardiac arrest, and even these have limited evidence supporting their use.

- **Epinephrine** 1 mg every 3 to 5 minutes is recommended for all forms of cardiac arrest without a palpable pulse.
- **Vasopressin** 40 IU may be used, but only as a one-time dose.
- **Atropine** 1 to 3 mg is indicated for asystole and PEA.
- **Amiodarone** 300 mg is recommended for VF/VT that is unresponsive to initial defibrillation and epinephrine and is not thought to be due to torsades de pointes. Additional doses may be required.
- **Magnesium sulfate** 1 to 2 g should be used instead of amiodarone when the rhythm is thought to be torsades de pointes.
- **Sodium bicarbonate** and **calcium chloride** (with nebulized albuterol and glucose/insulin) are indicated specifically for cardiac arrest due to hyperkalemia, but they no longer form part of standard cardiac arrest protocols.

**Open Cardiac Massage (OCM)**

OCM was the preferred technique prior to the introduction of modern CPR in 1960. Presently, open chest massage is indicated for cardiac arrest associated with penetrating thoracic trauma and arrest during a thoracic surgical procedure. OCM also is indicated when chest wall deformity or recent sternotomy precludes effective CCM. Other situations in which to consider OCM include cardiac arrest due to hypothermia, massive pulmonary embolism, pericardial tamponade, or intraabdominal hemorrhage. The 1992 AHA guidelines also recommend consideration of OCM when CCM fails to provide adequate support, but OCM is seldom performed for this reason. It is important to note that OCM is not indicated as a last effort after prolonged closed chest CPR.

**Technique**

OCM is performed via a left lateral thoracotomy unless the patient has a fresh sternotomy. The chest is entered through the fifth intercostal space. The pericardium is opened longitudinally, anterior to the phrenic nerve, if pericardial tamponade and/or a penetrating cardiac injury is present. Otherwise, the pericardium may be left intact. The heart is compressed with
two hands or, alternately, with one hand while the other is used to occlude the thoracic aorta.

**Results of OCM**

Early reports of OCM had success rate (28 - 55 %). However, when events under anesthesia are excluded the survival rate was only 17 %, which is comparable with that found in many recent series of CCM. Complications

Complications include RV perforation, hemorrhage, lung laceration, phrenic nerve injury, esophageal and aortic injury, cardiac lacerations, and empyema. However, the rate of infection is relatively low, approximately 5 %, given the emergent nature of the procedure on an unprepped chest.

**Chest Reopening.**

Early chest reopening, ideally within 10 minutes of the arrest, enhances the survival rates of patients who suffer cardiac arrest after cardiac surgery. Chest reopening affords several benefits. Once the chest is open, cardiac compressions and defibrillation can be performed internally, which is more efficient. Tamponade (and tension pneumothorax) may be identified and relieved. A bleeding site may be recognized and controlled. Inspection of the heart may reveal dehiscence or occlusion of a coronary graft. Pacing wires may be reattached. An operating team is assembled because it may be required for subsequent surgery. If a decision is made to return to the operating room, internal cardiac compressions can continue while the patient is transferred from the ICU. As far as possible, a sterile technique should be maintained during chest reopening in the ICU. Prophylactic antibiotics should be administered.

**What is the role of CPB and ECMO support?**

How to deal with traumatic cardiac arrest? **FIGURE 5−18**

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**Cerebral Protection/Resuscitation**

The objective of CPR is to restore circulation in a neurologically intact individual. The main priority during CPR is to provide sufficient myocardial and cerebral blood flow to prevent irreversible damage prior to definitive intervention. Unfortunately, this goal often is not achieved. **Less than 10 % of CPR attempts result in survival without neurologic damage, whether in or out of a hospital.**

The **postresuscitation syndrome (PRS)** has been defined as a condition of an organism resuscitated following prolonged cardiac arrest, caused by a combination of whole body ischemia and reperfusion, and characterized by multiple organ dysfunction, including neurologic impairment.

During global ischemia, consciousness is lost within 10 seconds. Brain-stem function ceases within 1 minute of cardiac arrest, producing agonal respirations and fixed and dilated pupils. Anaerobic glycolysis, which is the only means of energy production, is incapable of meeting the brain's basal metabolic demands. As a result, high-energy phosphate and glycogen stores are depleted within 5 minutes. Lactic acid accumulates in neurons and has a direct cytotoxic effect.

**TABLE 5−8. The postresuscitation syndrome (PRS)**

<table>
<thead>
<tr>
<th>Phase Interventions</th>
<th>Phase Interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pre-arrest (protect) phase</strong></td>
<td>• Optimize community education regarding child safety</td>
</tr>
<tr>
<td></td>
<td>• Optimize patient monitoring and rapid emergency response</td>
</tr>
<tr>
<td></td>
<td>• Recognize and treat respiratory failure and/or shock to prevent cardiac arrest</td>
</tr>
<tr>
<td><strong>Arrest (no-flow) phase</strong></td>
<td>• Minimize interval to BLS and ACLS (organized response)</td>
</tr>
<tr>
<td></td>
<td>• Minimize interval to defibrillation, when indicated</td>
</tr>
</tbody>
</table>
### Low-flow (CPR) phase

- Push Hard, Push Fast
- Allow full chest recoil
- Minimize interruptions in compressions
- Avoid overventilation
- Titrate CPR to optimize myocardial blood flow (coronary perfusion pressures and exhaled CO2)
- Consider adjuncts to improve vital organ perfusion during CPR
- Consider ECMO if standard CPR/ALS not promptly successful

### Postresuscitation phase: short-term

- Optimize cardiac output and cerebral perfusion
- Treat arrhythmias, if indicated
- Avoid hyperglycemia, hyperthermia, hyperventilation
- Consider mild postresuscitation systemic hypothermia
- Debrief to improve future responses to emergencies

### Postresuscitation phase: longer-term rehabilitation (regenerate)

- Early intervention with occupational and physical therapy rehabilitation
- Bioengineering and technology interface
- Possible future role for stem cell transplantation

### Postresuscitation Cerebral Care: Techniques for induction of hypothermia after cardiac arrest

- Surface cooling
- Large volume ice cold intravenous fluid
- Intravascular catheter cooling
- Extracorporeal cooling
- Partial liquid ventilation with cold fluorocarbons
- Pharmacological approaches
- Isolated brain cooling
- Body cavity lavage

Autoregulation of cerebral blood flow is ineffective following ischemic injury; therefore, blood flow is completely dependent on arterial pressure.

N.B.: Currently, there is no definitive evidence that any therapy improves neurologic outcome in humans. Hypoxia, hypotension, and hypercapnia aggravate cerebral injury and must be corrected.

**FIGURE 5−21. Algorithm for the management for out-of-hospital cardiac arrest victims.**
Outcome

In-Hospital Cardiac Arrest (IHCA)

24hr survival rates ranged from 13 to 59 % (µ 38.5 %).
µ survival to hospital discharge was 14.6 % (range 3 to 27 %).
Of note, there was no substantial difference between survival rates for studies published in the 1960s and those from the 1980s. Another review of in-hospital arrest reports published between 1987 and 1991 found a combined long-term survival rate of 11 % (range 5 to 29 %).
Several theories exist to explain this failure to improve survival. One is that no dramatic advances have been made since the introduction of CCM combined with ventilation and defibrillation in the 1960s. A second theory is that although CPR techniques and training have improved, the inpatient population is older and sicker than in years past. Despite the trend toward older patients with more comorbidities in the inpatient population as a whole, there is no definitive proof that the subset of patients undergoing CPR is different from those in years past.

Factors Associated With Improved Outcomes in Cardiac Arrest

• Presenting rhythm of VT/VF
• Early/bystander CPR
• Early defibrillation
• CPR prior to defibrillation in the circulatory phase of cardiac arrest
• Minimal interruptions to chest compressions
• In-hospital and out-of-hospital use of AEDs
• Amiodarone use in shock-resistant VT/VF
• VSE (Vasopressin + Steroid + Epenephrine)

Out-of-Hospital Cardiac Arrest (OPCA)

In contrast to inpatient arrest, where comorbid diseases play a major role in outcome, time outweighs all other factors for out-of-hospital arrest. Key determinants of survival include initial rhythm, whether the arrest is witnessed, the interval before CPR starts, and the period before definitive treatment. Survival rates are highly variable among reported studies.

Predictors For Out-Of-Hospital Cardiac Resuscitation

It has been reported that the importance of early intervention for survival. Survival for witnessed arrest was 22 % versus 4 % for unwitnessed arrests. If the time to CPR was less than 4 minutes, survival improved from 12 to 28 %. And if definitive care, i.e., defibrillation, was provided within 8 minutes, survival was 40 % compared with 13 % if the first shock was given more than 8 minutes from the time of cardiac arrest. As with in-hospital arrest, presenting rhythm was a major determinant for survival; VF/VT had the best prognosis. Bystander CPR improves survival to discharge rates for victims of cardiac arrest. In addition, several studies found less neurologic morbidity in cardiac arrest victims who received bystander CPR. The decrease in hospital mortality from bystander CPR is primarily due to fewer deaths from anoxic encephalopathy during the postresuscitation hospitalization. Return of consciousness within 24 to 48 hours of arrest is a positive neurologic prognostic sign.

Long-Term Outcome (Survival): Of the few patients who survive to discharge after out-of-hospital arrest, long-term survival is relatively good. The reported 1-year survival rate ranges from 75 to 85 %, and approximately 50 % are still alive 4 years postarrest. The majority of these patients ultimately die of cardiac causes. Positive predictors for long-term survival are cardiac arrest associated with acute MI, no prior history of MI, and short time intervals to CPR and definitive care. Patients with primary arrhythmic events, congestive heart failure, impaired LV function, extensive CAD, and complex premature ventricular depolarizations are less likely to survive long term after discharge.

Morbidity: ≈ 90 % of survivors of CPR return to home, while 5 to 10 % are institutionalized. Between 20 and 30 % of those who return home are home-bound. Only 60 % return to their prearrest level of function, and less than 30 % of those employed before arrest return to work. The rate of significant mental impairment is variable.
Cardiac Arrest in Children

The pathophysiology of cardiac arrest in children is markedly different from that of adults, and to undertake a successful resuscitation these differences must be understood. Most children have a healthy myocardium and healthy, patent coronary arteries. We do not see myocardial infarction, nor arrhythmias as causes of cardiac arrest in children; and antiarrhythmic agents or manoeuvres play virtually no part in their resuscitation. The major causes of cardiac arrest in children are hypoxia and hypovolemia. Therefore the majority of resuscitations will be in successful in restarting the heart if the myocardium is well oxygenated and presented with a pre-load to pump. To understand why children are at a greater risk from hypoxia and hypovolemia than adults, this presentation will highlight some of the physiological differences between them.

**Differences in physiology**

1. Children are smaller than Adults! Weight (in Kg.) = 8 + (Age x 2)
2. Children are a different shape from Adults! Large head, small chest, large belly, small limbs.

The caloric expenditure of a child is greater than an adult for two major reasons. Firstly, calories are required for growth in addition to the normal turnover, and secondly, in order to maintain a normal body temperature, a child has to burn more calories per kilogram of body weight than an adult.

**Temperature Regulation**

Children, as all other mammals, are ‘homeothermic’; that is they attempt to maintain a constant body temperature in the vital central organs of their body, their core temperature. This core temperature is the balance of heat gains against heat losses.

Heat gains can be from the environment, if the environment is at a higher temperature than the body (>370°C), or from metabolism. Metabolic heat generation is from shivering& or thermogenesis. Young children are unable to shiver; but the infant has other sources of heat generation. Both mechanisms of heat generation require the consumption of oxygen and calories.

Heat losses may be from radiation, conduction, convection or evaporation. Children are more susceptible than adults to heat loss because of a number of factors: (a) Large surface area in relation to their mass., (b) No subcutaneous tissue prevent heat loss, (c) Large scalp which has a high blood flow, and (d) An inability to alter their surroundings to prevent heat loss. Therefore in any sick child, all 4 routes of heat loss need to be considered:

1. Radiation. A warm body will radiate heat to their surroundings. Eg. Cold windows, etc. Children need to be screened from large cold surfaces.
2. Conduction. Avoid contact with cold surfaces, particularly those that will conduct a large amount of heat, eg cold mattresses.
3. Convection. Prevent cross currents of cold air around the children.
4. Evaporation. Dry any exposed surfaces when possible.

A child will generate heat by increasing oxygen consumption in response to a cool environment. The environment that is warm enough to produce the lowest amount of heat generation and therefore the least amount of work, is called the ‘Neutral Thermal Environment’. This environment may be as warm as 34°C in an infant.

<table>
<thead>
<tr>
<th>TABLE 5-9. Approximate Caloric Expenditure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (kg)</td>
</tr>
<tr>
<td>-------------</td>
</tr>
<tr>
<td>3 kg</td>
</tr>
<tr>
<td>20 kg</td>
</tr>
<tr>
<td>70 kg</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TABLE 5-10. Mls/Kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Body Water</td>
</tr>
<tr>
<td>Newborn</td>
</tr>
<tr>
<td>Child</td>
</tr>
<tr>
<td>Adult</td>
</tr>
<tr>
<td>Blood Vol.</td>
</tr>
<tr>
<td>Newborn</td>
</tr>
<tr>
<td>Child</td>
</tr>
<tr>
<td>Adult</td>
</tr>
</tbody>
</table>
significant in the child. For example, a four year old child weighing 15 kg may bleed 120 Hs from a scalp laceration. This loss represents more than 10% of his circulating blood volume and can easily produce signs of shock. Eq. Typical I.V. rates: For a baby weighing 5 kg = 20 ml/hr

---

**I. V. Solutions**

Although the water requirements of children are high, the sodium requirements are the same as an adult per kilogram of body weight, and therefore the concentration of solutions required for children contain less sodium than adult solutions. Commonly used solutions are 5% Dextrose + 0.2% Saline or 3.3% Dextrose + 0.3% Saline. The large liver and potentially large stomach, necessary for this caloric intake, can compromise the ventilation of a child; particularly if, during periods of increasing distress, the child swallows large quantities of air.

---

**TABLE 5-11. Water Requirements per Hour**

<table>
<thead>
<tr>
<th>Weight</th>
<th>Water Requirements per Hour</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 - 10 Kg</td>
<td>4 ml/ kg</td>
</tr>
<tr>
<td>10 - 20 Kg</td>
<td>40 ml + 2 ml for each kg over 10 kg</td>
</tr>
<tr>
<td>&gt; 20 Kg</td>
<td>60 ml + 1 ml for each kg over 20 kg</td>
</tr>
</tbody>
</table>

---

**TABLE 5-12. Signs of dehydration**

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimal</td>
<td>&lt; 5%</td>
</tr>
<tr>
<td>Mild</td>
<td>5%</td>
</tr>
<tr>
<td>Moderate</td>
<td>10%</td>
</tr>
<tr>
<td>Severe</td>
<td>15%</td>
</tr>
</tbody>
</table>

---

**Respiratory System**

The high caloric expenditure of the infant and child require a high oxygen consumption. Oxygen uptake is more than twice that of the adult per kilogram of body weight. Alveolar ventilation must be high to eliminate the carbon dioxide produced from this metabolism; but, paradoxically, the respiratory system is poorly equipped to cope with these high demands. The respiratory system in this age group is efficient for growth, but not efficient for gas exchange. To accommodate the rapid swallowing movements necessary to ingest feeds quickly, and yet maintain a rapid respiratory rate, the child has a different anatomical configuration of his upper airway.

- This configuration makes it easier for the child to protect his airway during swallowing but it does increase the chance of aspiration in cases where the conscious level is decreased, and makes the child more susceptible to disease states producing edema of these structures. The differences in the upper airway also make the maintenance of a patent airway in the unconscious child more difficult.
- Development of lung tissue may not be complete until the age of 7 to 8 years, and this immaturity of alveolae leads to high lung closing volumes. The child will not maintain open small airways during normal breathing until the age of 5 years.
- When considering oxygenation, the neonate is at the same risk as the 70 yr. old man; both have a deficiency in elastic recoil, and both will have relative arterial hypoxemia under normal conditions.
- In addition to airway difficulties and pulmonary inefficiency, the child has a highly compliant chest wall.
• Under condition of respiratory stress, the diaphragm can generate considerable power; in the adult this power is transmitted to the lung tissue and the rib cage is firm enough to not only remain stable, but also to contribute to the work required.

• However, the child has a rib cage that is not only inefficient in producing an increase in lung volume, but also will distort under conditions of stress. This distortion of the chest wall is clinically detected as sternal recession, indrawing, rib flaring and a ‘Harrison's Sulcus’.

Treatment

A. Guideline changes in PBLS

Compression:ventilation ratios

• Lay rescuers should use a ratio of 30 compressions to 2 ventilations.

• Two or more rescuers with a duty to respond should use a ratio of 15 compressions to 2 ventilations.

Age definitions

• An infant is a child under 1 year.

• A child is between 1 year and puberty.

Automated external defibrillators

• A standard AED can be used in children > 8yrs. Purpose-made pediatric pads, or programs which attenuate the energy output of an AED, are recommended for children 1-8yr.

• If no such system or manually adjustable machine is available, an unmodified adult AED may be used for children older than 1 year.

• There is insufficient evidence to support a recommendation for or against the use of AEDs in children less than 1 year.

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Prognosis

The outcome of children requiring resuscitation can be reasonably good if they have only suffered a respiratory arrest (mortality 25%), however if the myocardium is subjected to a great enough stress to produce cardiac arrest then the mortality is similar to adult resuscitations. (88% mortality.

Foreign body airway obstruction sequence

A simplified sequence of actions should be used for the management of foreign body airway obstruction (FBAO) in infants and children.

Guideline Changes in ALS

• Where possible, give drugs intravascularly (IV or intraosseous), rather than by ETT.

• Either uncuffed or cuffed tracheal tubes may be used in infants and children in the hospital setting.

• One defibrillating shock, rather than three ‘stacked’ shocks, is recommended for (VF/VT).

• When using a manual defibrillator, the shock energy for children is 4 J kg⁻¹ for all shocks.

• A standard AED can be used in children > 8yrs.

• Purpose-made pediatric pads, or programs which attenuate the energy output of an AED, are recommended for children 1-8yr.

• If no such system or manually adjustable machine is available, an unmodified adult AED may be used for children >1 year.

• There is insufficient evidence to support a recommendation for or against the use of AEDs in children <1 year.

• The dose of adrenaline during cardiac arrest is 10 microgram kg⁻¹ on each occasion.
FIGURE 5–25. Algorithm for management of pediatric cardiac arrest.
Algorithm for management of pediatric cardiac arrest by pediatric BLS healthcare provider.

Cardiac Arrest in Neonates

Passage through the birth canal is a hypoxic experience for the fetus, since significant respiratory exchange at the placenta is prevented for the 50-75 sec duration of the average contraction. Though most babies tolerate this well, the few that do not may require help to establish normal breathing at delivery. Newborn life support (NLS) is intended to provide this help and comprises the following elements:

• Drying and covering the newborn baby to conserve heat;
• Assessing the need for any intervention;
• Opening the airway;
• Lung aeration;
• Rescue breathing;
• Chest compression;
• Administration of drugs (rarely).

Guideline changes in NLS

• The use of food-grade plastic wrapping is recommended to maintain body temperature in significantly preterm babies.
• Attempts to aspirate meconium from the nose and mouth of the unborn baby, while the head is still on the perineum, is no longer recommended.
• Ventilatory resuscitation may be started with air. However, where possible, additional oxygen should be available if there is not a rapid improvement in the infant’s condition.
• Adrenaline should be given by the intravenous or intraosseous route, as standard doses are likely to be ineffective if given via a ETT.
• If there are no signs of life after ten minutes of continuous and adequate resuscitation efforts, then discontinuation of resuscitation may be justified.

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5 Anesthesia for Cardiac Operations

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One Lung Ventilation 21
Unit Objectives

- To review the basic concepts of anesthesia for cardiac operation.
- To review the preoperative assessment
- The different steps & sequence of conduct of anesthesia (premed, induction, prebypass, on bypass, postbypass)
- Anesthesia for special situations.

Learning Objectives

By the end of this chapter the student should know:

- Basics of General Anesthesia: general anesthesia.
- Preoperative Assessment: preoperative assessment; risk stratification & scoring preoperative condition; infective endocarditis prophylaxis; machine checkout; anesthesia setup
- Conduct Of Anesthesia: premedication; transfer; prior to induction; induction
- Pre-Bypass Anesthesia
- Anesthesia on Bypass: maintenance of bypass; common abnormalities on CPB
- Anesthesia Post-Bypass
- Anesthesia For Special Situations: anesthesia For OPCAB; anesthesia for vascular surgery; epidural anesthesia in cardiac surgery; anesthesia for ischemic heart disease; anesthesia for valvular heart disease; anesthesia for HCM; anesthesia for patients recieving anticoagulation & antiplatelet medication; anesthesia for pericardial tamponade; anesthesa for chronic renal failure & cardiac lesions; anesthesia for wwp patients; one lung ventilation

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Basics of General Anesthesia

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1. General Anesthesia

A general anesthetic has 3 components: hypnosis (reversible state of unconsciousness), analgesia, and muscle relaxation.

A. Hypnosis: Hypnosis has 3 phases: induction, maintenance, and emergence.

Induction and maintenance can be by iv or inhaled drugs. Intravenous induction is faster than inhalational induction. Some of the IV drugs used for hypnosis are the same as those used for analgesia—just in higher doses. As the hypnotic effect of IV anesthetic drugs is short, iv maintenance is by continuous infusion. Most induction agents are associated with a degree of cardiorespiratory depression, and usually cause vasodilation: particular care must be taken when inducing patients with acute tamponade, or critical AS to avoid acute decompensation requiring emergency intervention.

B. Analgesia:
- Analgesia ⇧ somatic and ↪ autonomic response to pain.
- Analgesics used in general anesthesia are usually opiates.
- Opiate analgesics ⇪ respiratory depression.

C. Muscle relaxation: Muscle relaxants are classified as depolarizing or non-depolarizing.
- Non-depolarizing muscle relaxants competitively bind with acetylcholine receptors at the neuromuscular junction and last up to 60 min.
- They are reversed by neostigmine, an anticholinesterase.

Depolarizing muscle relaxants non-competitively depolarize the muscle end plate causing fasciculation and rapid onset paralysis. They are effective for 5–15 min. Choice is dictated by duration and cardiovascular effects (vecuronium and rocuronium have no cardiovascular side effects).

TABLE 6–1. Examples of anesthesia for cardiac surgery

Key Points for Anesthesia for ADULT Cardiac Surgery

- The most common causes of CNS injury or dysfunction in cardiac surgical patients are thought to be microemboli and macroemboli.
- After coronary revascularization, mortality was far lower in patients with normal renal function (0.9%) than in patients with acute renal failure (63%).
- In addition to activating the extrinsic and intrinsic hemostatic pathways, CPB directly affects platelet function through the effects of hemodilution, hypothermia, and contact activation by bypass circuit materials.
- When choosing anesthetic agents and doses during induction and maintenance, one should consider any pharmacodynamic properties that might affect BP, HR, or CO, as well as the desirability of “early” extubation (i.e., within a few hours after the operation is completed).
- RV dysfunction or failure may also occur after CPB because of inadequate myocardial protection, inadequate revascularization with resultant right ventricular ischemia or infarction, preexisting pulmonary hypertension, intracoronary or pulmonary air embolism, chronic MVD, or TR.
- Numerous clinical approaches have been shown to measurably ⇪ the inflammatory response in cardiac surgical patients: modification of surgical and perfusion techniques, modification of circuit components, and pharmacologic strategies.

Regime A
- Premed: Temazepam 10–20 mg po 1h preoperatively.
- Induction: Fentanyl 5–10 micrograms/kg and Etomidate 10–20 mg.
- Maintenance: Isoflurane 1–2%, or further boluses of Fentanyl.
- Muscle relaxant: Pancuronium 0.1 mg/kg.

Regime B Total intravenous anesthesia (TIVA)
- Premed: Hyoscine 0.3 mg im and 5 mg Morphine im 1 h preoperatively.
- Induction: Propofol 4–10 micrograms/kg/h.
- Maintenance: Propofol 4–10 micrograms/kg/h + Remifentanil 0.3 micrograms/kg/min.
- Muscle relaxant: Rocuronium 0.6–1.0 mg/kg.
Types of procedures that are performed in the cardiac catheterization laboratory include (1) electrophysiologic assessment of rhythm disorders and management by ablation or by implantation of devices, (2) biventricular pacing for heart failure, (3) stenting of abdominal or thoracic aortic aneurysms, (4) balloon dilation and stenting for valvular and subvalvular lesions, and (5) the use of occlusion or umbrella devices to close an atrial or ventricular septal defect or a patent ductus arteriosus. Sedation or anesthesia improves the efficacy and safety of many procedures.

- Even after uncomplicated cardiac surgery, a midline sternotomy (or thoracotomy) \( \Downarrow \) total lung capacity, \( \Downarrow \) vital capacity, \( \Downarrow \) forced expiratory volume in 1 second, and \( \Downarrow \) functional residual capacity.

Key Points for Anesthesia for PEDIATRIC Cardiac Surgery

- Organ system maturation, from birth through adolescence (e.g., CVS, CNS, pulmonary, renal, hematologic), affects physiologic function and therefore anesthetic and surgical management and outcome.
- Physiologic understanding of CHD and consequent anesthetic management are based on the pathophysiologic determinants of FIVE categories of defects: see my classification.
- Preoperative assessment of cardiac status (e.g., review of history and physical examination, echocardiography, and catheterization data and consulting with the patient's cardiologist) and planning are the keys to a successful anesthetic outcome.
- Selecting an induction technique is dependent on the degree of cardiac dysfunction, the cardiac defect, the degree of sedation provided by the premedication, and the presence or absence of an indwelling venous catheter. The maintenance of anesthesia depends on the age and condition of the patient, the nature of the surgical procedure, the duration of CPB, and the need for postoperative ventilation.
- The physiologic effects of CPB on neonates, infants, and children are significantly different from the effects on adults. During CPB, pediatric patients are exposed to biologic extremes not seen in adults, including deep hypothermia (18°C), hemodilution (threeto fivefold greater dilution of circulating blood volume), low perfusion pressures (20 to 30 mm Hg), and wide variation in pump flow rates (ranging from 200 mL/kg/min to total circulatory arrest).
- After the repair of complex CHD, the anesthesiologist and surgeon may have difficulty separating patients from CPB. Under these circumstances, the underlying cause must be determined, which may be (a) an inadequate surgical result with a residual defect requiring repair, (b) PAH, or (c) RV or LV dysfunction.
- Neonates, infants, and children undergoing cardiac surgery with CPB have a higher rate of postoperative bleeding than that seen in older patients. This is due to several factors: (a) There is disproportionate exposure to the nonendothelialized extracorporeal circuit, which produces an inflammatory-like response. This inflammatory response to CPB is inversely related to patient age; the younger the patient, the more pronounced the response. (b) The type of surgery performed in neonates and infants usually involves more extensive reconstruction and suture lines, creating more opportunities for surgical bleeding than in adult cardiac patients. (c) Operations are frequently performed using DHCA, which may further impair hemostasis. (d) The immature coagulation system in neonates may also contribute to impaired hemostasis. (e) Patients with CHD demonstrate an increased bleeding tendency before and after CPB.

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Preoperative Assessment

Predicting evaluation of difficult mask ventilation and intubation
- Overall incidence of difficult mask ventilation (DMV) is 1.4% with impossible mask ventilation (IMV) 0.15%
- Obesity, snoring, and lack of teeth can probably be overcome with simple airway maneuvers, which is why they are not risk factors for IMV:
- Lack of teeth actually makes DL and intubation easier
- Neck circumference (> 50 or 60 cm at the level of the cricoid cartilage) is the most predictive factor of IMV
- Patients with three or more risk factors have a much higher likelihood of being IMV (odds ratio 8.9)
- Most patients (75%) who are IMV will be easy to intubate.
- Risk Factors Associated with Difficult versus Impossible Mask Ventilation

Risk Stratification & Scoring Preoperative Condition
- Preoperative Risk Assessment
- Algorithms
- Intermediate-Risk Patients

Understanding Cardiac Complications: Treatment to Periop Risk
- β-Blockers
- Statins
- Clonidine
- Calcium Channel Blockers
- Aspirin
- Deep Venous Thrombosis Prophylaxis
- Endocarditis Prophylaxis
- Perioperative Medication Management
- Prophylactic Coronary Revascularization

Infective Endocarditis Prophylaxis
The antibiotic prophylactic regimens below are recommended by the American Heart Association (AHA) only for patients with underlying cardiac conditions associated with the highest risk of adverse outcome from infection
Antibiotic prophylaxis is indicated for the following high-risk cardiac conditions:
- Prosthetic cardiac valve
- History of infective endocarditis
- CHD (except for the conditions listed, antibiotic prophylaxis is no longer recommended for any other form of CHD): (1) unrepaired cyanotic CHD, including palliative shunts and conduits; (2) completely repaired congenital heart defect with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first 6 months after the procedure; and (3) repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibits endothelialization)
Cardiac transplantation recipients with cardiac valvular disease
Preoperative Anesthesia Checklist

Although risk scoring for cardiac surgery has its own dedicated systems (Euroscore and STS Score, all surgical patients are also allocated an ASA grade TABLE 6-2 preoperatively.

Machine Checkout

The following monitors should be available for every anesthetic procedure:
- EKG leads (three- or five-lead)
- Pulse oximeter
- Noninvasive blood pressure monitor
- Temperature monitoring

Additional monitors depending on surgery type and patient history may include:
- Invasive arterial blood pressure monitoring
- Central venous pressure monitoring
- Pulmonary artery catheter monitoring
- Precordial Doppler
- EEG/BIS monitoring

The anesthesia machine should be checked every day and rechecked in between cases if different anesthesia equipment is used or if the anesthesia operator changes.

The following monitors should always be available:
- Oxygen analyzer
- Capnography
- Low- and high-pressure alarms
- Spirometer

Checkup procedure:
- Backup gas cylinders and Ambu-type bag valve mask device should be available in the room
- Turn on the machine master switch and all monitors
- A high-pressure system check should be performed with the oxygen cylinder supply and the central supply
  - A value of 1,000 psi in an O₂ E cylinder indicates 340 L of O₂ (half-full) at atmospheric pressure, that is, it would last for 34 minutes at 10 L/min
- A low-pressure system leak test should be performed (tests for leaks from flow control valves to common gas outlet)
- Check flow meter function
- Calibrate the O₂ sensor
- Check leak test of breathing system
- Check manual ventilation and ventilator bellows
- Check integrity of unidirectional valves
- Check capnography function
- Check scavenging system and CO₂ absorber; change absorbent if necessary
- Ensure APL valve is in the open position and ventilator is set to “manual” prior to patient’s arrival in OR

Additionally:
- Ensure that the MH box or cart is available and stocked
- Ensure defibrillator is in functioning condition and readily available
- If using local anesthetics, ensure lipid rescue is available and ready for use
- Ensure rapid transfuser is available and stocked for use if needed

Anesthetic Setup
Can be remembered with the mnemonic MSMAID.
M—“Machine”
Perform machine check as indicated above
S—“Suction”
Ensure suction is available and attached to appropriate length of tubing to reach patient’s head
Attach Yankauer tip to end of tubing
M—“Monitors”
Ensure that monitors as indicated above are available, calibrated, and ready to be used
All monitors should be of the appropriate size for the patient including blood pressure cuff size, arterial and central catheter sizes
Ensure that disposable monitors are attached and ready to be used for the next patient such as EKG leads, pulse oximeter, and, in some cases, BP cuff
A nerve stimulator should be readily available and functioning
Fluid warmers and patient warming blankets and devices should also be readily available and functioning
Have eye protection and lubricant ready
A—“Airway”
Check handle with ...

Anesthesia Setup
Conduct Of Anesthesia

Premedication

There is wide variation in choice of premedication, which is normally given an hour before the patient is transferred to the surgery area. Options include:
- A benzodiazepine, e.g. temazepam 10mg po, or lorazepam 2–4mg po: lorazepam has a half-life of 10–20h, temazepam is shorter acting.
- Intramuscular morphine 0.1mg/kg and scopolamine 4–8 micrograms/kg.
- Intramuscular morphine 0.1mg/kg and hyoscine 0.3 mg.

Transfer

- Prior to transfer to the surgical area the following are verified:
  - The patient’s identification in the form of a wristband with name, date of birth, and a hospital number.
  - The patient’s name, date of birth, hospital number, operation details, signature, and date of signing, on the consent form.
  - Availability of and patient details on cross-matched blood.
  - Presence of responsible surgeon.

Prior To Induction

The anesthesia area is prepared well in advance of the patient’s arrival to ensure that all necessary equipment is present and working, and that the specific drugs required for induction are drawn up and labelled. Practice varies, but most anesthesiologists establish all monitoring, with the exception of the central venous catheter, prior to induction, as this ensures that any hemodynamic or respiratory compromise occurring as a result of induction will be rapidly detected and treated.
- The patient is sat at 45°.
- Oxygen is given by face mask.
- A pulse oximeter is attached and oxygen adjusted appropriately.
- EKG leads attached, checked and II and V5 used for display.
- A 14-gauge venous cannula is placed at the left wrist under local anesthesia, unless the left radial artery is to be harvested.
- A radial arterial catheter is placed under local anesthesia, transduced and a baseline blood gas is measured.
Induction

• There is a wide choice of induction agents, and techniques. Use of fentanyl is described here: fentanyl has all the advantages of morphine (overdose is rare, it is not a myocardial depressant, it is non-explosive compared to inhalational agents, it facilitates postoperative management) as well as being a short-acting agent that is safe in the management of critically ill patients. The disadvantage is a risk of awareness.
• The patient is placed flat on a single pillow.
• Preoxygenation with 100% O₂ by face mask helps to avoid pulmonary hypertension in at-risk patients, as well as giving a safety margin by filling the functional residual capacity with O₂ if there are any problems establishing a secure airway later.
• 1mg of pancuronium may help avoid narcotic-induced rigidity.
• Induction: fentanyl 5–10 micrograms/kg and etomidate 10–20mg IV.
• As soon as the patient is unresponsive an appropriate dose of muscle relaxant up to a maximum of 0.1mg/kg of pancuronium is given.
• It may be necessary to give volume or a vasopressor such as metaraminol to treat hypotension at this stage.
• The trachea is intubated, and the ET tube secured.
• The table is placed in Trendelenburg position so that the central venous catheter, and pulmonary artery catheter if indicated, can be inserted.
• The urinary catheter is inserted, and the patient shaved.
• The table is levelled, and the pressure transducers adjusted.
• Prophylactic antibiotics are given: vancomycin is run an hour before incision to maximise serum concentrations.

TABLE 6–9. Problems during induction

Respiratory
• Failure to secure an airway.
• Difficult intubation.
• Aspiration.
• Bronchospasm.
• Pulmonary hypertensive crisis.

Cardiovascular
• Cardiac arrest (critical AS and/or LMS, acute tamponade).
• Hypotension or hypertension.
• Ischemia.
• Arrhythmias.

Other
• Anaphylaxis
• Complications of with invasive monitoring.
• Dental and oropharyngeal trauma.
• Brachial plexus, ulnar and radial nerve injury, occipital alopecia, heel and sacral tissue necrosis may result from improper positioning.
• The EKG leads for the defibrillator and any TEE probe are placed, and an electrocautery pad is placed.
• The patient’s arms are carefully secured to avoid compression injury.

Anesthetic Agents for Adult Cardiac Surgery
• Narcotic Based: Fentanyl and Sufentanil (8 x more potent)
• Hemodynamic stability without myocardial depression
• Typical total dose during cardiac procedure
  • Fentanyl: 10-100 mcg/kd
  • Sufentanil: 8-15 mcg/kg
  • Remifentanil: 1-3 mcg/kg/min
• Muscle Relaxation: Choice depends on desired heart rate response
  • Pavulon: Increases heart rate– may be useful in balancing the vagolytic effect of narcotics
  • Vercurium: No effect on heart rate or blood pressure
• Other new long-acting relaxants are similar to Vercurium

TABLE 6–8. Patients at high risk of hemodynamic instability
Patients with the following lesions are at high risk of hemodynamic decompensation during the induction sequence, and a surgeon should be immediately available, and in the case of acute tamponade the patient prepped and draped ready for incision prior to induction:
• Tight LMS stenosis.
• Severe aortic and mitral stenosis.
• Acute cardiac tamponade or mediastinal bleeding.
• Severe pulmonary hypertension.
• Very poor LV function or cardiogenic shock.
• Pregnancy.
Induction and Maintenance of Anesthesia (Adult)

Induction
- Narcotic
  - Remifentanil 1-2 mcg/kg
  - Fentanyl 5-35 mcg/kg or Sufentanil 1-8 mcg/kg plus
- Sedative-Hypnotic
  - Pentothal
  - Etomidate
  - Valium or Versed
  - Propofol

Maintenance
- Narcotic PLUS
- Propofol, Benzodiazepine, or low dose inhalation drug (Ethrane, Forane, Desflurane, Halothane, Sevoflurane)
- "Fast Track" Anesthesia

Pediatric Anesthetic Agents
- Induction is usually not intravenous
- Ketamine 5 mg/kg intramuscular
- Inhalation using Halothane or Sevoflurane
  - "Halothane sensitizes myocardium to arrhythmogenic influence of catecholamines"
- IV established after induction
- Maintenance
  - Narcotic PLUS
  - Hypnotic or Inhalation

Pre-Bypass Anesthesia

Anesthesia management is aimed at optimizing several parameters over a period of time when surgical activity results in variable levels of sympathetic stimulation, as well as manipulation that impact cardiac function, namely:
- The ratio of myocardial oxygen supply: demand to prevent ischemia.
- Preload and afterload, particularly in patients with valvular defects.
- Heart rate, rhythm, and ventricular contractility
- Systemic and pulmonary vascular resistance, particularly in patients with pulmonary hypertension or congenital heart disease.

Median sternotomy

Skin incision, sternal division, sternal retraction, and dissection of the adventitia over the aorta result in high levels of sympathetic stimulation. An adequate depth of anesthesia is necessary to avoid tachyarrhythmias and hypertension. The surgeon should check that the anesthesiologist is happy with the level of anesthesia before starting. Sternotomy occasionally results in significant blood loss. Some surgeons ask for the lungs to be deflated prior to sternal division to reduce the minimal risk of damage to the lung parenchyma. A baseline ACT may be measured at this point. Redo-sternotomy. The main differences in redos are:
- The patient must have external defibrillator pads on.
- Check cannulation and conduit plan with the surgeon as this dictates possible sites for siting arterial lines (e.g., patient will need left radial line if right axillary artery cannulation or radial harvest planned).
- There is a risk of injuring grafts, ventricle, or great vessels on sternal division: in high-risk cases the patient will be cannulated and even placed on bypass prior to resternotomy to reduce the risk.
- The dissection that must take place before the heart can be cannulated is longer, and involves more mechanical disturbance and bleeding.
Conduit harvest

- This is a period where sympathetic stimulation is minimal, and the anesthesiologist may need to treat bradycardia and hypotension.
- Compression of the left subclavian artery by sternal retractors may mean left radial lines do not accurately reflect systemic pressures.
- Major occult blood loss may occur into the pleurae or the leg wound.
- Brachial plexus injury can result from overabduction of the arm when positioning it for radial artery harvest.
- For harvesting the IMA the table needs to be raised, sometimes tilted away from the surgeon, and sometimes the lungs deflated temporarily.
- Heparin (300 units/kg iv) is given before the IMA is divided otherwise it could thrombose: the surgeon will ask for heparin to be given and the anesthesiologist should acknowledge the request.
- Check the ACT 3min after heparin is given.

Preparing to cannulate

- Hitching the pericardium to the sternal edges frequently leads to transient hypotension as a result of vagal stimulation, and mechanical constriction of the SVC and IVC: if this does not respond to adequate volume resuscitation the surgeon should ‘drop’ the pericardium. TEE is helpful to reassess valve and ventricular function, guide coronary sinus catheter and assess aorta.
- Handling the RA while placing purse-strings may result in AF: synchronized DC cardioversion 10–50J with internal paddles is safest when patient is heparinized and cannulated as VF is occasionally induced.
- Blood pressure should ideally be <100mmHg systolic at aortic cannulation to reduce the risk of aortic dissection and bleeding.
- Once the aorta is cannulated and pipe connected, volume can be given rapidly by the perfusionist if necessary. Blood loss can be drained by pump suckers directly back to the pump if the ACT is >400.
- Once the venous cannula is in and connected the patient can be put on bypass if needed, but in order to minimize bypass time the surgeon will usually check conduit and site additional vents first.

Management Of Conditions Which Affect Bypass

- **Antithrombin III deficiency:** If ACT remains <400 despite large heparin dose, give FFP to treat antithrombin III deficiency and recheck ACT.
- **Pregnancy:** Although maternal mortality is the same as for a non-pregnant patient, fetal mortality approaches 50%. Placental ischemia occurs as a result of microemboli, elevated IVC pressure which reduces venous drainage, and pump flows inadequate for the hyperdynamic circulation associated with pregnancy. Uterine blood flow is not autoregulated. Dilution of progester- one may result in induction of labour. Management is targeted at establish- ing adequate pump flows, and avoiding hypoxia and hypothermia.
- **Heparin-induced thrombocytopenia (HIT):** Heparin substitutes such as danaparoid may be used.
- **Sickle cell:** Cardiopulmonary bypass exacerbates the tendency of erythrocytes to sickle because of vasoconstriction due to hypothermia, stasis, hypoxia, and acidosis. Even patients with sickle cell trait are at risk of a bypass- induced sickle cell crisis. This condition is managed with preoperative partial exchange transfusion to reduce Hb-S from 100% to less than 33%. Hypoxemia, acidosis and dehydration must be avoided preoperatively.
Anesthesia on Bypass

Maintenance Of Bypass

Blood concentrations of drugs are diluted by the prime in the bypass circuit, and additional muscle relaxant and anesthetic agent may be required on bypass. Once full flow is achieved the patient is disconnected from the ventilator (just switching off the ventilator can result in air trapping, leaving the lungs inflated, interfering with the operative field).

Routine checks during bypass

• **Anticoagulation**: the ACT is checked after initiating bypass and every 30min thereafter, with additional heparin given in 5000-unit increments if necessary to maintain the ACT >500.

• **Blood gas and acid base status**: this is checked after initiating bypass and every 30min thereafter, with the PaO₂ maintained at >14kPa (105mmHg) by adjusting FiO₂ to the oxygenator, and pH adjusted according to whether management is pH stat or alpha stat.

• **EKG**: checked for inappropriate electrical activity during arrest and ischemic changes and arrhythmias pre-and post-arrest.

• **CVP and MAP**: these are recorded at 5–10min intervals, and should be 1–10mmHg and 50–70mmHg respectively (arterial pressures are maintained 70–80mmHg in elderly/arteriopathic patients).

• **Urine output**: the bag is usually emptied before bypass so that total urine output during bypass can be measured.

• **Face**: periodically check that there is no facial edema, or asymmetry in colour or temperature, and no pupil asymmetry, due to misplacement of the venous or aortic cannulas. Sweating, lacrimation, mydriasis may indicate inadequate depth of anesthesia.

• **Core and peripheral temperature**: nasopharyngeal or tympanic temperature probes measure core temperature which the perfusionist maintains at the temperature requested by the surgeon.
Common Abnormalities On CPB

Problems of high line pressures, poor venous drainage and poor electromechanical arrest.

**Hypotension**
- Flow rate deliberately decreased by perfusionist, e.g., when surgeon clamping aorta or dealing with bleeding.
- Inadequate flow as a result of inadequate arterial cannula size for patient’s BSA, kinked or clamped arterial line, poor roller head occlusion.
- Low SVR due to vasodilation (give phenylephrine) or hemodilution.
- Cytokines in sudden return of pooled blood via pump suckers.
- Transducer error, calibration error, tubing disconnection, kinking or compression of subclavian, brachial or radial artery.
- Arterial cannulation problems including selective cannulation of carotid arteries, aortic dissection, or rarely reversed cannulation.

**Hypertension**
- High SVR due to vasoconstriction mediated by endogenous catecholamines, hypothermia, awareness, and hyperoxia: treat the likely cause.
- Pump flows deliberately increased by perfusionist, e.g., to speed rewarming.
- Selective cannulation of subclavian proximal to radial artery catheter.
- Transducer too low.

**High CVP**
- Poor venous drainage, inadvertently snaring SVC proximal to cannula, obstructing venous return.
- Catheter abutting SVC wall, venous pipe or snared by surgeon.

**Slow nasopharyngeal cooling or rewarming**
- Excessive vasoconstriction.
- Low pump flows.
- Temperature probe misplaced or malfunctioning.
- High CVP impeding cerebral perfusion pressure gradient.
- Carotid artery malperfusion (e.g., inadvertent selective cannulation).
- Rarely increased intracranial pressure.

**Hypoxemia**
- Inadequate FiO₂ to the pump oxygenator.
- Low pump flows.
- Pump oxygenator malfunction.
- Causes of low SvO₂ are as given earlier, and O₂ consumption secondary to hyperthermia, shivering, and rarely malignant hyperpyrexia.

**Oliguria**
- Post-renal problems such as kinked catheter tubing, misplacement, clamping, and catheter occlusion by gel or clot.
- ↓ glomerular filtration rate as a result of ↓ renal perfusion due to inadequate pump flows, hypotension, renal vasoconstriction by drugs and hypothermia, ↑ IVC pressures, non-pulsatile perfusion, clamping of descending aorta and circulatory arrest.
- Pre-existing renal impairment.

**Hemoglobinuria**
- Hemolysis due to pump and pump sucker trauma.
- Trauma to UT in setting of heparinization.
- Blood transfusion reaction.
- Rarely contamination of pump circuit with water from heat exchanger.

**Electrolyte imbalance**

**Hyperkalemia**
- Improper electrolyte mixture in the prime solution.
• Frequently administered or improperly mixed cardioplegia
• Inappropriate exogenous administration to correct hypokalemic state
• Hemolysis
  • Immune: transfusion reaction, improper type and cross-match. Inadequate patient–unit identification cross-check
  • Mechanical: long bypass time, excessive suction, excessive pump head occlusion pressure
  • Inadequately occlusive aortic clamp with consequent leakage of cardioplegia solution into the circulation
  • Pretransfusion warming of blood at excessive temperature (>42°C)
  • Transfusion of blood that is G6PD deficient or hemoglobinopathic
  • Improperly mixed with a hypo- or hyperosmotic solution
  • Stored or transported at improper temperature
• Hypercarbia (inadequate gas sweep, hypermetabolic state)
• Temperature-related potassium shifts
• Metabolic acidosis
• Renal insufficiency/failure

Hypokalemia
• $\downarrow K^+$ due to dextrose and insulin administration, polyuria.

Proposed practical approach for RV failure

Diuretics
• Slowly increased
• Oral: bumetamide up to 10 mg (b.i.d. or t.i.d.)
• i.v. drip if necessary (1 mg per h)
• Association with spironolactone 50–100 mg and chlorthalidone 50 mg
• Switch from bumetamide to furosemide up to 600 mg
• Sodium and fluid restriction
• Sometimes haemofiltration
• Intermittent paracentesis for therapy-resistant ascites and pleural effusion

Inotropes
• 7-day course
• Dobutamine 4–6 µg·kg$^{-1}$·min$^{-1}$ (peripheral administration is acceptable)
• Low blood pressure: dopamine 4–6 µg·kg$^{-1}$·min$^{-1}$
• Heart rate >110 beats per min + digoxine 0.5 mg i.v.

Vasodilators
• Seldom needed
• Inhaled NO 10–20 ppm
• Inhaled iloprost 150 µg per day (every 2–3 h or even continuously)

Awareness
About 1% of patients have recall of intraoperative events. This is due to a combination of dilution of anesthetic agents by the pump prime and absorption of fentanyl into the prime circuit: some patients are too unwell to tolerate higher doses of anesthetic agent. It is commonest when the patient is warm, as hypothermia induces unconsciousness. Variation between individuals means there is no dose of drug that guarantees unconsciousness. Careful titration of anesthetic agents is critical.
Anesthesia Post-Bypass

• The perfusionist gradually allows the heart to fill and eject by progressively occluding the venous line at the same time as reducing pump flows, until pump flow ceases and the patient is ‘off bypass’.
• Bypass can be terminated immediately at any point during this procedure, and it is usually better to come off relatively underfilled as excessive preload and afterload increase myocardial oxygen demand at the time when the heart is least able to deal with this.
• Rapid, controlled transfusion is given by perfusionist in aliquots of 50–100mL from the pump via the arterial line on request, and pump suckers can still be used to return blood to the pump, so there is no need to give volume intravenously.
• Inotropes, chronotropes, vasodilators, or vasoconstrictors are titrated.
• If hemodynamic instability or evidence of ischemia it may be necessary to return go back on bypass for a period to reduce the demand on the heart, and allow it to recover from stunning.
• Failure to wean from bypass is discussed.
• Protamine slow push (3mg/kg/IV or dose calculated by Hepcon®) is given to reverse heparin after bypass is finally terminated.
• The surgeon will spend a period of time checking hemostasis while protamine is given, allowing time for full reversal of heparin.
• The surgeon decannulates the aorta once the pump is empty or the heart is full: systemic pressure should be <110mmHg systolic to reduce the risk of hemorrhage or dissection at this point.

Giving protamine

Once the surgeon is sure that bypass will not be required again they will ask the anesthesiologist to give the protamine. It is good practice not to draw up protamine until this request is made as bypass has been inadvertently and catastrophically terminated by giving protamine by mistake instead of a drug in a nearby syringe. Although bypass is terminated blood can still be transfused to the patient from the pump via the arterial line if required, but use of pump suckers should be discontinued once protamine starts.

Surgical maneuvers that cause hypotension

• Always look at the operative field before treating hypotension: the cause is usually surgical and temporary.
• Aortic decannulation: (1) surgeon may compress heart or occlude venous inflow to deliberately lower arterial pressure in order to decannulate safely, (2) if significant bleeding occurs.
• Oversewing the atrial purse-string if arrhythmias or bleeding result.
• Lifting the heart to inspect anastomoses.
• Pacing the pericardium.
• Hitching the pericardium: most surgeons unhitch at this stage.
• Approximating the sternal edges if there are packs around the heart.
• Deliberate occlusion of the IVC by the surgeon to reduce BP rapidly (see aortic decannulation).
• Pacing problems: use of electrocautery while on demand pacing if slow (or no) underlying rhythm, deliberate or inadvertent disconnection.
• Use of electrocautery interfering with IABP in EKG mode.

Variations in sequence

Decannulating before protamine is given

If the surgeon expects that attempting to decannulate the aorta may cause sufficient hemodynamic compromise that bypass must be re-instituted, they may elect to decannulate before protamine is given. Some surgeons routinely decannulate the aorta before protamine is given to eliminate any risk of thrombus formation at the tip of the cannula.

Placing the aortic cannula into the right atrium after decannulation

If the surgeon anticipates substantial bleeding from the aortic cannulation site, they may elect to place the arterial cannula into the RA via the purse-string previously used for the venous cannula, so that blood transfusion from the pump may be continued if required.
Anesthesia For OPCAB

While off-pump surgery confers some benefits it presents surgeon and anesthesiologist with two major challenges, which anesthetic and surgical technique must overcome:

- Reduction in CO when positioning the heart.
- Interruption to coronary blood flow during each distal anastomosis.

Anesthetic technique

- One important reason for choosing off-pump surgery is that patients can sometimes be extubated earlier, but this can only be accomplished with appropriate choice of anesthetic agents and analgesia.
- Smaller doses of short-acting benzodiazepines are given as premedication, which may be supplemented by midazolam if necessary.
- Fentanyl and remifentanil are shorter-acting anesthetic agents that may be used in preference to propofol and opioids.
- Lines inserted by the anesthesiologist are the only means of giving rapid transfusion in the absence of an aortic cannula: there must be adequate wide-bore venous access.
- To help maintain CO when the heart is positioned, patients are actively hydrated.
- Heparin is frequently given at a lower dose but check with the surgeon: doses vary between 100–300 units/kg IV.
- Most units routinely use TEE to assess LV function intraoperatively, and routinely place PA catheters to help detect ischemic changes (e.g., ischemic MR, elevated PA pressures).
- Continual measurement of ST segment changes is a very useful adjunct.
- Make sure that a sterile warming blanket is placed on the patient, as there will be no other way of securing normothermia intraoperatively.

Hemodynamic management during anastomoses

Hemodynamic changes occur rapidly, but can be anticipated by knowing the sequence of surgery, as well as by adjuncts to standard monitoring: ST segment analysis, TEE, and CO measurements. Pressor support may be required during the distal anastomosis, during which preload must be optimized. The systemic pressure needs to be relatively high prior to the distal anastomoses, as positioning the heart inevitably results in a decrease in CO. For the proximal anastomoses the systemic pressures need to be lower, to minimize the risk of aortic tears and dissection. It is possible to perform the proximal anastomoses first: this may be indicated to minimize the period of relative ischemia, and exploit low systemic pressures. The use of intracoronary shunts helps to reduce myocardial ischemia while performing distal anastomoses. The use of TEE may allow the anesthesiologist to assess whether the heart is likely to tolerate particular positions, by identifying valvular regurgitation, and ventricular impairment before the heart decompensates. Tachycardia makes distal anastomoses more difficult. It is easier to maintain hemodynamic stability when the pH is physiological. BP and CO should return to baseline between each anastomosis.

Respiratory management

Sometimes it is necessary to stop ventilating for short periods to optimize surgical exposure. This is usually during anastomoses to the obtuse marginal vessels. The table will also be placed in steep Trendelenburg position for the right-sided anastomoses, which results in a reduction in pulmonary compliance and functional residual capacity. This may result in hypoxia, hypercarbia and atelectasis, which should be addressed by careful hyperventilation.

Postoperative management and extubation
Not all surgeons choose to reverse heparinization with protamine. If the patient is normothermic, not acidotic, and adequately ventilating, hemodynamically stable and not bleeding once the sternum is closed, they are candidates for waking and extubation in theatre, or shortly within intensive care. Adequate analgesia is imperative. Although intrathecal morphine has been used in some centres, most units use patient- and nurse-controlled analgesic IV morphine infusions. High $O_2$ flows, bronchodilator therapy, and chest physiotherapy are important adjuncts.

Patients with good ventricular function and a full revascularization are usually hypertensive and tachycardic. Intraoperative low CO state. As bypass cannot be used to warm these patients they may be hypothermic.

They do not produce a diuresis as they are often relatively underfilled. A significant metabolic acidosis frequently develops during the first 6h postoperatively, resolving within 12h. The cause of the acidosis is unclear but may reflect an

Ensure these patients are adequately filled.

Use warming blankets and infusion warmers to achieve normothermia.

These patients sometimes receive a reduced dose of intraoperative heparin depending on surgical preference, and should therefore receive early aspirin (75mg po) and LMWH (5000 units sc) to ensure graft patency and avoid TE complications.

What is ???
The role of anesthetist in OPCAB.
The role of anesthetist in awake cardiac surgery.

**TABLE 6−13. Endovascular Procedures**
Anesthesia for Vascular Surgery

- Recent advances in our understanding of the biology of atherogenesis suggest that endothelial dysfunction is a critical element in the pathogenesis of atherosclerotic cardiovascular disease and its complications. Inflammation in the artery wall probably plays a fundamental role as well.

- Major vascular surgery is particularly challenging to the anesthesiologist because these are high-risk operations in a patient population with a high prevalence of either overt or occult CAD, which is the leading cause of perioperative and long-term mortality after vascular surgery.

- Accurate clinical assessment of the pretest probability of significant CAD is necessary for prudent use and rational interpretation of preoperative cardiac testing.

- Guidelines on perioperative cardiovascular evaluation and care suggest that coronary intervention is rarely necessary to simply lower the risk of surgery unless such intervention is indicated irrespective of the preoperative context. Current evidence does not support the role of prophylactic coronary revascularization as a means to reduce perioperative or long-term morbidity after major vascular surgery.

- Perioperative management of patients undergoing vascular surgery requires an understanding of the underlying pathophysiology of the specific vascular lesion.

- Patients should be maintained on their usual cardiovascular medications throughout the perioperative period. Antiplatelet therapy requires special consideration and must be individualized to each patient.

- Prevention and treatment of perioperative myocardial ischemia require careful control of the determinants of myocardial oxygen supply and demand. ST-segment monitoring, particularly with computerized ST-segment analysis, should be used to detect myocardial ischemia during the perioperative period.

- Clinical trial data suggest that initiation of perioperative β-blocker therapy has potential benefits and risks.

- The clinical usefulness of any intraoperative monitoring technique ultimately depends on patient selection, accurate interpretation of data, and appropriate therapeutic intervention.

- Evidence suggests that maintenance of vital organ perfusion and function by the provision of stable perioperative hemodynamics is more important to overall outcome after aortic surgery than is the choice of anesthetic agent or technique.

- The pathophysiology of aortic cross-clamping and unclamping is complex and depends on many factors, including the level of the cross-clamp, the extent of coronary artery disease and myocardial dysfunction, blood volume and distribution, activation of the sympathetic nervous system, and the anesthetic agents and techniques.

- The degree of preoperative renal insufficiency is the strongest predictor of postoperative renal dysfunction.

- Endovascular aortic surgery has recently emerged as a less invasive alternative to conventional open aortic repair. Endoleak, or the inability to obtain or maintain complete exclusion of the aneurysm sac from arterial blood flow, is a complication specific to endovascular aortic repair.

- The primary clinical utility of cerebral monitoring during carotid endarterectomy is to identify patients in need of carotid artery shunting; secondarily, such monitoring is used to identify patients who may benefit from blood pressure augmentation or change in surgical technique.

- Postoperative hypothermia is associated with many undesirable physiologic effects and may contribute to adverse cardiac outcome.
TABLE 6–14. Pros and Cons of Minimally Invasive Technique

### Epidural Anesthesia In Cardiac Surgery

This technique is also used in cardiac surgery requiring CPB. An epidural is sited at either T1/2, T2/3, or T3/4. This may be done as early as the night before surgery. 8mL 0.5% ropivacaine with 20 micrograms of fentanyl is infused before induction of anesthesia so that sensory spread can be assessed. After induction the infusion is continues at 5–15mL/h. A sensory blockade of T1–T10 can be achieved. The epidural is discontinued on postoperative day 3. The most important complication is paraplegia from a spinal cord haematoma (risk approximately 1:3000). The benefits of this technique include:

- Improvement in pain relief.
- ↓ opioid and NSAID requirement.
- ↑ physiotherapy compliance.
- ↑ respiratory function.
- ↓ length of ICU and hospital stay
- ↓ incidence of depression.

### Anesthesia For Ischemic Heart Disease

- Goal: prevent myocardial damage
- Optimum myocardial oxygen demand : supply ration
- MVO2 is directly related to
  - Heart rate
  - Contractility
- Oxygen supply is directly related to
  - Coronary blood flow
  - LV wall tension
- Minimize Ischemia
- Avoid tachycardia
- Increased heart rates are correlated with post-operative MI
- Maintain resting (ischemia-free) hemodynamics
- Slow: maximize diastolic time
- Small: minimize wall tension
- Well Perfused: Adequate coronary pressure

### Anesthesia For Valvular Heart Disease

**Aortic Stenosis**

- LVH
- May have coronary artery disease
- Requires high filling pressures
- Anesthetic goals
  - Normal heart rate (avoid tachycardia)
  - Atrial "kick" crucial for adequate preloading of LV.
Anesthesia for HCM

Hemodynamic Principles
Avoid factors that worsen obstruction:
Tachycardia (sympathetic stimulation, vagolysis)
Positive inotropes
Peripheral vasodilators
Hypovolemia
Have immediately available:
Esmolol, diltiazem
Phenylephrine
Induction
Balanced technique:
Avoid induction agents that decrease afterload (e.g., propofol) or increase HR (e.g., ketamine):
Etomidate and/or midazolam ...

Anesthesia for Patients Recieving Anticoagulation & Antiplatelet Medication

For AF, assess thromboembolic risk based on the CHADS2 score (0–6 points).
0 points: no indication for chronic anticoagulation; discontinue warfarin 5 days before surgery; do not resume unless other indication
• 1–2 points: discontinue warfarin 5 days before surgery; resume 5 days after surgery
• 3 points or more: discontinue warfarin 5 days before surgery; LMWH, or IV UFH relay
• For valve replacements:
• Discontinue warfarin at least 5 days before elective procedure, or longer if INR >3.0
• Assess INR 1–2 days before surgery; if INR >1.5, consider 1–2 mg of oral vitamin K
• Reversal for urgent surgery: consider 2.5–5 mg of oral or intravenous vitamin K
• Immediate reversal for emergent surgery: consider fresh frozen plasma, prothrombin complex concentrate, or recombinant factor VIIa

Patients at high risk for thromboembolism:
• For patients who have a mechanical valve, high risk includes those who have mitral valve prostheses, older aortic valve prostheses, or had a CVA or TIA in the past 6 months
• For patients who have atrial fibrillation, high risk includes a CHADS2 score of 5–6, a CVA or TIA within the past 3 months, or rheumatic valvular heart disease
• For patients who had a venous thromboembolism (VTE), high risk includes a VTE within the past 3 months, severe thrombophilia
• Bridge with...
Anesthesia for Pericardial Tamponade

- Acute drop in BP on induction
- \( \downarrow \) venous return with controlled ventilation
- \( \downarrow \) sympathetic tone due to anesthetic state
- Management
  - Prep awake with spontaneous ventilation
  - Ketamine induction
  - Sympathomimetic
  - Spontaneous ventilation possible

Anesthesia for Chronic Renal Failure & Cardiac Lesions

- Remember CRF a multiorgan affection: renal; neurologic; cardiovascular; respiratory, hematologic; immunodeficiency; others
- Positioning: Careful positioning of arms with attention to fistula
- Induction:
  - Minimize sedative agents
  - Rapid sequence induction if delayed gastric emptying is suspected
  - Succinylcholine may be used if preoperative \( K^+ < 5 \) mEq/L
  - Avoid rocuronium or vecuronium; preferred NMB is cisatracurium
- Fluids:
  - Minimize fluids for minor surgery
  - For major and intermediate surgery:
    - Replace fluid loss (blood loss and insensitive losses) with lactated Ringer (LR) or other balanced salt solutions, not normal saline (NS)
    - NS causes hyperchloremic acidosis that worsens hyperkalemia and may preclude extubation
- Drugs:
  - Normal metabolism (independent from renal function):
    - (Cis-) atracurium, succinylcholine, esmolol, remifentanil
  - Titrate all other drugs to effect:
    - Vecuronium, rocuronium, fentanyl, midazolam, hydromorphone
  - Avoid (or titrate carefully) drugs with renally eliminated metabolites:
    - Morphine, vecuronium, meperidine, midazolam
  - Sevoflurane is probably safe but avoid low fresh gas flow

Extubation:

- Check arterial blood gas prior to extubation for any longer cases
- If there is significant metabolic (anion gap) acidosis: ...
Anesthesia for WPW Patients

- Management of paroxysmal tachycardia associated with WPW is similar to that of other SVTs. Treatment is required when there is clinical poor tolerance.
- Be cautious when administering anesthetics that cause an increase in sympathetic tone or the production of extrasystoles that may precipitate tachycardia:
  - Desflurane is sympathomimetic and can increase AV nodal conduction time, which may result in greater conduction via the AP and tachycardias.
  - Atropine, glycopyrrolate, ketamine can resulting in PSVT or AF and should be avoided.
  - Neostigmine slows AV nodal conduction and facilitates AP conduction. Therefore, it should be avoided.
- Patients who develop an atrial arrhythmia intraoperatively (AF or atrial flutter) and have underlying WPW should not be given nodal blockers (including adenosine, calcium channel blockers, beta-blockers, or digoxin) or carotid sinus massage. Nodal blockers slow AV nodal conduction and allow for greater conduction through the faster conducting AP. This promotes degeneration of AF to VF:
  - Cardioversion and/or sodium channel blockers (i.e., procainamide) are first-line therapy for patients with AF and WPW. Sodium channel blockers block the AP.
  - However, patients who develop a narrow complex arrhythmia such as ORT can be given nodal blockers to slow the rate.

One Lung Ventilation

- To facilitate surgery on the lung or thoracic aorta.
- Absolute indications:
  - Lung abcess
  - HeartPort
- Useful in:
  - Pulmonary hemorrhage
  - V.A.T.
- Hypoxemia- commonly due to increased shunt.
- Minimize hypoxemia by:
  - 100% FiO2
  - Decrease volatile agents to < 1%
  - Increase ventilation to the dependent lung (non-operative)
  - PEEP the dependent lung
  - CPAP on the operative lung
  - Occlude the pulmonary artery of the operative lung.

TABLE 6–16. Indications for One-Lung Ventilation (OLV)
7 Cardiopulmonary Bypass

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Unit Objectives

- To understand the **history and goals of CPB**
- To understand **equipments & mechanics of CPB**
- To understand **physiologic parameters of CPB** (perfusion pressure during CPB; nonpulsatile versus pulsatile perfusion; pump flow during bypass; bypass temperature management strategy; acid-base strategy; and fluid management).
- To understand **end-organ effects of CPB**: myocardial injury; brain injury; renal dysfunction; gastrointestinal effects; endocrine and inflammatory responses.
- To understand **management before CPB**: vascular cannulation and other preparations.
- To understand **initiation and discontinuation of CPB Support**: initiation of CPB; preparation for separation and separation from bypass.
- To understand **perfusion emergencies and how to prevent and manage**: arterial cannula malposition; aortic or arterial dissection; massive arterial gas embolus; venous air lock and reversed cannulation.
- To understand **special patient populations**
- To understand **care of the gravid patient during CPB**

Learning Objectives

By the end of this chapter the student should KNOW:

- History & Goals.
- Equipments & Mechanics: pumps; oxygenator; circuitry & cannulation techniques; cardiotomy suction & venting; circulatory assist devices: applications for ventricular recovery or bridge to transplant.
- Hematology: coagulation basics; anticoagulation for CPB; heparin neutralization; hematologic effects of CPB; management of coagulopathy associated with CPB; blood transfusion & blood conservation.
- Physiology & Pathology: hemodilution & priming solutions; hypothermia; surgical myocardial protection; changes in the pharmacokinetics & pharmacodynamics of drugs administered during CPB; systemic inflammatory response after bypass (SIRAB); embolic events; endocrine, metabolic, & electrolyte responses; CPB & the lung; CPB & the kidney; splanchnic, hepatic, & visceral effects; neurologic effects.
- Clinical Applications: institution of CPB; conduct of CPB; management of unusual problems encountered in initiating & maintaining CPB; perfusion emergencies; termination of CPB; CPB in infants & children; ECMO for respiratory or cardiac support; noncardiovascular applications of CPB; perfusion for thoracic aortic surgery; CPB for port-access cardiac surgery; special patient populations; care of the gravid patient during bypass.

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Key Notes

- CPB provides **4 functions** extracorporeal maintenance of respiration and circulation at hypothermic and normothermic temperatures. CPB permits the surgeon to operate on a quiet, or nonbeating, heart at hypothermic temperatures, thus facilitating surgery in an ischemic environment.
- CPB is associated with a number of **profound physiologic perturbations**. The central nervous system, kidneys, gut, and heart are especially vulnerable to ischemic events associated with extracorporeal circulation.
- Controversy regarding the optimal management of **blood flow, pressure**, and **temperature** during CPB.
and temperature during CPB remains. Perfusion should be adequate to support ongoing oxygen requirements; mean arterial pressures of more than 70 mmHg may benefit patients with cerebral and/or diffuse artherosclerosis. Arterial blood temperatures should never exceed 37.5°C.

- **The initiation and termination** of CPB are key phases of a cardiac surgery procedure, but the anesthesiologist must remain vigilant throughout the entire bypass.
- **CPB is a complex system that is vulnerable to accidents.** Careful and constant communication among the anesthesiologist, surgeon, perfusionist, and nurse is critical for patient safety.
- **Minimally invasive cardiac surgery** can be performed with port-access bypass circuits. This approach adds considerable complexity to the procedure and is not as popular as off-pump techniques for coronary artery bypass grafting.
- **Bloodless cardiac surgery** is possible, and numerous approaches including autotransfusion and hemodilution facilitate this goal.
- **Total CPB** can be tailored to produce deep hypothermic, circulatory arrest, or partial bypass. The special techniques require sophisticated monitoring and care.
- **The physiology of CPB and hypothermia has been gradually elucidated,** pathophysiologic effects determined, and therapies addressed. Our technical ability to address the most complex of problems surgically has improved during the last decade, with a reduction in mortality rates to less than 5% for most complex lesions in neonates and children. However, CPB is still associated with significant morbidity. Miniaturization of bypass circuitry, modulation of the inflammatory response, advancement of technology, and improvement in techniques of perfusion will maximize the safety of extracorporeal circulation and provide the next major step in producing better outcomes.

### History & Goals

**History**

A. 1953 - Gibbon - first use of CPB for open heart surgery in a human - screen oxygenator

B. Early screen, bubble, and disc oxygenators were traumatic to blood à frequent bleeding diatheses

The CPB circuit is designed to perform **four major functions:**

1. **Oxygenation and carbon dioxide elimination,**
2. **Circulation of blood,**
3. **Systemic cooling and rewarming,** and
4. **Diversion of blood from the heart to provide a bloodless surgical field.**
FIGURE 7-2. Cardiopulmonary bypass circuits.
### Pumps

- Roller pumps are slightly non-occlusive, resistance-independent, and may cause less blood trauma.
- Centrifugal pumps are dependent on inflow or outflow resistance; will cease flow at very low inflow resistance and very high outflow resistance.
- Venous drainage can be active or siphoned.
- Active drainage requires vacuum through the venous reservoir or negative pressure from the pump.

### Oxygenator

- Largest foreign surface contact area.
- Membrane oxygenators can be microporous, hollow fiber, or silastic (true membrane).
- Gas flow is titrated to maintain PaO2 between 85 and 250mmHg to avoid O2 toxicity.
- PCO2 is regulated by gas and blood flow through the membrane.
- pH is controlled by adjusting the PaCO2.
- pH stat corrects the pH to the temperature of the patient's blood, with the goal of relative hypercarbia to increase cerebral blood flow.

### Heat Exchanger

- The cooling or warming gradient is usually within 10-14 degrees of the patient's temperature.
- This minimizes the tendency for gas to come out of solution and risk of air embolism.
- Mixed blood temperature should be less than or equal to 38.5C.
- The water bath should stay between 15 and 42C to prevent organ damage (too cold) and hemolysis (too warm).

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### Circuitry & Cannulation Techniques

### Cardiomyopathy Suction & Venting

### Circulatory Assist Devices: Applications For Ventricular Recovery Or Bridge To Transplant
Hematology

Coagulation Basics

Overview
Primary hemostasis
- Platelet (a) adhesion, (b) activation, (c) aggregation
Secondary hemostasis
- Activation of plasma coagulation (form fibrin)
- Extrinsic pathway (via tissue factor)
- Intrinsic pathway (subendothelium or foreign contact)

Common pathway
- Inhibition of systemic clotting
- Natural anticoagulants (AT III, Protein C & S)
- Fibrinolytic system, i.e. Plasmin (degrades fibrinogen))
- Other reactions
- Complement activation (↑ permeability, ↑ cell lysis)
- Kinin generation (vascular dilation, ↑ permeability)

Platelet Function
- Contact
  - With subendothelium after endothelial injury
  - With proteins adsorbed onto synthetic surfaces
- Adhesion
  - Via attachment mechanisms i.e. Glycoprotein Ib/IX (GP Ib/IX) receptor
- Activation
  - Begins as platelets spread with a conformational change
  - Release TxA2, ADP, serotonin, (PF4, BTG)
- Aggregation
  - ADP induced change in GpIIb/IIIa receptor permits binding of adhesive proteins, like fibrinogen, between platelets

Anticoagulation for CPB

HEPARIN
- Glycosaminoglycan, MW 3K - 100K
- Acts by binding enzyme AT III (AT III inhib's IIa,Xa,IXa,XIa,XIIa)
- Half life is 60-90 minutes
- Monitored with aPTT or ACT
- Complications: bleeding; HIT => thrombosis, "white clot" (Ab versus Hep-PF4 complex); osteoporosis.

Heparin dosing
- Standard initial dose = 300 U/kg
- Maintain ACT > 300-350 (>300?)
- Monitor with ACT (or direct Heparin concentrations)
- Redose to maintain therapeutic level
- 100 U/kg every 60 - 90 minutes (approx.)
- Use dose-response curve
- Protamine for heparin reversal
- Estimate heparin present (dose response curve)

Alternatives to heparin (future)
Hirudin (Hirulog, synthetic analog)
From leeches, direct inhibitor of thrombin
Does not require ATIII
Prolongs TT, aPTT, PT, and ACT

Ancrod
From venom of Malayan pit viper

Others

**Heparin Neutralization**

**PROTAMINE**

- Basic protein, binds heparin
- 1 mg protamine = 100 U heparin
- Give 1.1 - 1.5 mg protamine : 100 U heparin
- Confirm reversal to baseline
- Adverse reactions
  - Transient systemic hypotension
  - Related to infusion rate, total dose
  - Anaphylaxis - pulmonary hypertension, systemic hypotension, bradycardia
  - (Risk factors - prior exposure, DM's/NPH)

---

**TABLE 23.3. Classification of protamine reactions**

<table>
<thead>
<tr>
<th>Type</th>
<th>Horrow</th>
<th>Moorman, Zapol, Lowenstein</th>
</tr>
</thead>
<tbody>
<tr>
<td>I.</td>
<td>Hypotension resulting from rapid administration</td>
<td>Pharmacologic histamine release</td>
</tr>
<tr>
<td>IIA.</td>
<td>Anaphylactic reactions</td>
<td>True anaphylaxis (IgE-mediated)</td>
</tr>
<tr>
<td>IIB.</td>
<td>Immediate anaphylactoid reactions</td>
<td>Anaphylactoid reactions</td>
</tr>
<tr>
<td>IIC.</td>
<td>Delayed anaphylactoid reactions</td>
<td>Pulmonary vasoconstriction (IgG/complement-mediated)</td>
</tr>
<tr>
<td>III.</td>
<td>Catastrophic pulmonary vasoconstriction</td>
<td>Noncardiogenic pulmonary edema</td>
</tr>
</tbody>
</table>

**Hematologic Effects of CPB**

Hemostasis with CPB
Basic Considerations with CPB (CPB)
CPB leads to:
- Activation of clotting cascades
- Activation of fibrinolytic system
- Platelet activation and removal
- Kinin system activation
- Complement activation
- Results in hemostatic derangement
- Results in systemic inflammatory responses
A. Initial events of blood-surface interactions

- Adsorption of fibrinogen and other plasma proteins to foreign surface is initial event.
- Contact activation of factor XII (intrinsic pathway).
- Platelet adherence, release of cytoplasmic granules, thromboxane A-2.
- Contact activation initiates complement cascade and kallikrein/kinin system.
- Decreased velocity from hemodilution may β damage to formed elements in blood, β net blood loss, improve capillary perfusion.
- Frothing, high shear rates, and turbulence in pump damage formed elements:hemolysis, plt activation.
- Bubble oxygenator (blood-gas) contributes significantly to impaired hemostasis after 2-3 h. total bypass time.
- Intracardiac suction, “pump sucker”

B. Dynamics of plasma coagulation during CPB

- Significant amounts of plasma proteins are not lost in extracorporeal circuit.
- Though diluted (£50%), clotting factor levels remain adequate.
- Prolonged clotting times post-op correlate poorly w/bleeding.
- Fibrinolysis
  - ?? responsible for derangements of clotting tests early post-op.
- Activated plasmin degrades fibrin and fibrinogen.
- FDP’s act as anticoagulants
- Aprotinin (see below)

C. Platelet dynamics during CPB

Number
- To 40-50% baseline in 1st 10-15 min, then stabilizes.
- Reduced plt adhesiveness.
- Rarely < 75,000/mL
- Plt ct returns to normal 3-5d post-op (?sequestration in liver).
- Microembolus formation contributes to platelet consumption.

Function - substantially altered
- Plasma levels of Tx A2, plt-specific .proteins rise at onset of CPB.
- Plt stores of ADP & ATP depleted.
- Clot retraction impaired by heparin:
  - High concentrations of heparin impair vWF-platelet binding.
  - Reduction in clot retraction correlates w/post-op bleeding.
- Hypothermia, plasmin, other proteases.
- Neutrophil activation by surface glycoprotein (GMP-140 or P-selectin)
- Attempts to inhibit plt activation during CPB (ASA, dextran) à excessive hemorrhage.
Management of Coagulopathy Associated with CPB

Hemorrhage Post-CPB
- Consider Surgical bleeding
- Heparin excess
- Incomplete neutralization; reinfusion of anticoagulated blood; heparin rebound
- Clotting cascade procoagulant deficiency
- Platelet dysfunction or thrombocytopenia
  DIC, depleted fibrinogen (preop thrombolytics)
- Exploration (< 3 – 5%)
  - >500/h x 1 hr; >400/hr x 2 hrs; >300/hr x 3 hrs;
  - >1000 total in 4 hrs; >1200 total in 5 hrs

Aprotinin
- Mechanism: proteolytic enzyme inhibitor
- **blood fibrinolysis, kinin activation, platelet activation**
- **Benefits**
  - ↓ blood loss, ↓ systemic response to CPB
- **Risks**
  - Prothrombotic effects, renal failure (?)
  - Anaphylaxis with re-exposure (cutaneous testing, predose)
- Usage guidelines
  - Patient risk should influence use – high risk patients (reoperations, long procedures, coagulopathy, need to avoid transfusions)
  - ACT monitoring

Other agents
- Amicar (Epsilon-amino caproic acid)
- Desmopressin (DDAVP)

Blood Transfusion & Blood Conservation
- Cell saver recycling
- Hemoconcentration of excess CPB blood
- Reinfusion of shed blood from chest tubes
  (Consider time, volume, infection hazard)
- Prevention/reversal of bleeding diathesis
- Optimization of heparin/protamine use
- Autologous plasma, fresh whole blood
- Aprotinin (Trasylol)
- Epsilon-amino caproic acid (Amicar)
- Heparinized CPB circuits
- More pharmacodynam, more thrombo resistant
- Autologous blood donations (with erythropoietin)

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Mechanisms of Injury

Mechanical
• The foreign surfaces of the bypass circuit (boundary layer of oxygenator, heat exchanger, filters, tubing) interact with the blood.
• Shear stresses include the pump, cardiotomy suction, and cannulae.
• Microemboli can form as particles from the oxygenator, platelet aggregate, or fibrin aggregates, and are greatest within the first 15 minutes of bypass.

Humoral
• Factor XII (Hageman factor), the alternative complement cascade (C3a), kallekrein, and plasminogen are activated in various degrees.
• Other factors interrelate and amplify the inflammatory reaction, including the arachidonic acid cascade, interleukins, TNF, and PAF.

Cellular
• Neutrophils play a major role in humoral activation and are sequestered in the lung, releasing cytotoxin and free radicals which increase vasoreactivity and vascular permeability.
• Monocytes and mast cells also participate, although their role is unclear.
• Lymphocytes have a minor role, if any.
• Platelets are activated and elaborate GPIB, IIB, and IIIA.
• Absolute number of platelets is reduced by 40% by the end of bypass, and the number of receptors is also decreased.
• Endothelial cells are affected by abnormal flow, humoral factors, and local ischemia.
• A wide variety of substances are expressed by the endothelium, including prostaglandins, thromboxanes, leukotrienes, and interleukins.

End-Organ Effects Of CPB

End Organs That Can Be Adversely Affected by CPB
• Heart
• Lungs
• Brain
• Kidneys
• Gastrointestinal tract
• Endocrine system & immune system

Factors Associated with Myocardial Injury during CPB
• Abnormal perfusate composition
• Persistent ventricular fibrillation
• Inadequate myocardial perfusion
• Ventricular distention
• Ventricular collapse
• Coronary embolism
• Catecholamines
• Aortic cross-clamping
• Reperfusion

D. J. Wheatley; Protecting The Damaged Heart During Coronary Surgery; Heart; 2003; 89: 367–368.

Physiology & Pathology

Hemodilution & Priming Solutions
• Advantages of hemodilution include the following:
  • ↓ blood viscosity
  • ↑ regional blood flow
  • ↑ oxygen delivery to tissues
  • ↓ exposure to homologous blood products
  • ↑ blood flow at lower perfusion pressure (lower shear stress), especially during hypothermic perfusion

Hypothermia

<table>
<thead>
<tr>
<th>Hypothermia</th>
<th>Hyperthermia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Favorable balance between $O_2$ supply and demand</td>
<td>Imbalance between $O_2$ supply and demand</td>
</tr>
<tr>
<td>↓ Excitotoxic neurotransmitter release</td>
<td>↑ Excitotoxic neurotransmitter release</td>
</tr>
<tr>
<td>↓ Blood-brain barrier permeability</td>
<td>↑ Blood-brain barrier permeability</td>
</tr>
<tr>
<td>↓ Inflammatory response</td>
<td>↑ Inflammatory response</td>
</tr>
<tr>
<td>↑ Free radical production</td>
<td>↑ Intracellular acidosis</td>
</tr>
<tr>
<td></td>
<td>Destabilized cytoskeleton</td>
</tr>
</tbody>
</table>
SURGICAL MYOCARDIAL PROTECTION
See chapter 8.

Changes in the Pharmacokinetics & Pharmacodynamics of Drugs Administered during CPB

Systemic Inflammatory Response After Bypass (SIRAB)

The Inflammatory Response to CPB
• CPB exposes the body to extreme, nonphysiologic conditions that initiates a global inflammatory response.
• Blood–artificial surface interaction results in activation of several amplifying protein cascade systems including those of the coagulation, fibrinolytic, kallikrein, and complement systems.
• Additional ischemia–reperfusion injury occurs as a result of the altered flow states and hypothermia. White blood cells, platelets, and endothelial cells are activated, leading to production of additional inflammatory mediators capable of further activation of humoral and cellular elements resulting in amplifying positive feedback loops. Ultimately capillary integrity is altered and multisystem organ dysfunction occurs.
• Contact of the blood with foreign surfaces results in production of kallikrein. Kallikrein enters into a positive feedback loop with factor XII, activating both the coagulation cascade and the fibrinolytic system. Kallikrein results in production of bradykinin. In addition kallikrein activates the renin–angiotensin system and complement cascades.
• The damaging effects of CPB and the subsequent inflammatory response are the result of the extreme conditions encountered during extracorporeal support, including:
  • Cell activation on contact with the foreign surfaces of the bypass circuit;
  • Mechanical shear stress;
  • Tissue ischemia and reperfusion;
  • Hypotension;
  • Nonpulsatile perfusion;
  • Hemodilution with relative anemia;
  • Blood product administration;
  • Heparin and protamine administration; and
  • Hypothermia.

Embolic Events
• The 3 categories of emboli are biologic, foreign material, and gaseous.
• Bloodborne emboli consist of cellular and noncellular products or aggregates.
• Most fat emboli are derived from cardiotomy-suctioned blood.
Endocrine, Metabolic, & Electrolyte Responses

- A variety of pituitary-related hormonal activities is influenced by CPB: ADH and adrenocorticotropin levels and thyroid-stimulating hormone levels are typically normal, but T3 and T4 responses to TSH “sick euthyroid syndrome.”

- Adrenal responses affected by CPB include catecholamine, aldosterone, and cortisol levels that can be attenuated to varying degrees by deeper anesthesia, thoracic epidural anesthesia, and pulsatile perfusion; and the aldosterone levels is stimulated by activation of the RAS.

- Hypothermic nonpulsatile CPB

- Hypomagnesemia commonly occurs during CPB, and prophylactic treatment of this deficiency with IV magnesium supplementation decreases the incidences of post-CPB atrial and ventricular dysrhythmias and may decrease the incidences of coronary vasospasm and myocardial ischemia.

- Plasma potassium concentrations fluctuate during CPB under the influence of a variety of factors, especially cardioplegia composition and dosing.

CPB & The Lung

- The lung is a target organ for injury induced by CPB.
  - Atelectasis
  - Smoking, chronic bronchitis, chronic obstructive pulmonary disease, and obesity all predispose patients to atelectasis.
  - Atelectasis decreases oxygenation and C0₂.
  - Acute lung injury
  - Lung injury related to CPB and microemboli is decreased by blood filtration.
  - Complement activation correlates with lung dysfunction after CPB.

- Inflammatory response produced by “contact activation” of blood (activation of neutrophils and endothelium, generation of complement and cytokines) contributes to lung injury after CPB.

- Noncardiogenic pulmonary edema is rare but has a high mortality rate, is most commonly associated with complement activation and protamine, and is treated by volume expansion, mechanical ventilation, and cardiac support.
  - Acute bronchospasm
  - Acute bronchospasm is related to complement activation.
  - Cold 8armacody can be treated with H₁ and H₂ blockade.
  - Inaccuracy of EtCO₂
  - Prevention of lung injury is by:
    - Blood filtration
    - Membrane oxygenators
    - Hemodilution
    - Pharmacology, such as steroids (controversial), prostaglandins (i.e., leukocyte and platelet inhibition), and aprotinin (inhibits plasmin, kallikrein, bradykinin).
  - Exclusion of lung circulation during CPB impairs normal metabolic functions, such as clearance of prostaglandins, serotonin,
bradykinin, and norepinephrine and generation of prostaglandins and angiotensin.

### CPB & The Kidney

- The incidence of renal failure requiring dialysis after CABG is approximately 1%, but is 2.7% in congenital heart surgery.
- Mortality rate from postoperative renal failure after CPB is more than 50%.
- Lesser degrees of renal impairment still increase intensive care unit and total hospital length of stay significantly.
- Preoperative renal function impairment and length of CPB correlate with postoperative renal impairment.
- Low cardiac output (and surrogates such as low ejection fraction, NYHA class IV congestive heart failure, use of intra-aortic balloon counter-pulsation, IABP) and advanced age are also predictive of postoperative renal failure.
- Diabetes is not a consistent predictor.
- CPB procedural factors that influence renal function are as follows: hemodilution is protective, pulsatile perfusion is theoretically protective, and membrane oxygenators and arterial line filters may be protective.
- Prophylactic therapies to decrease the risk of renal failure after CPB are as follows:
  - Dopamine stimulates DA-1 receptors to increase renal blood flow and may improve GFR and tubular function.
  - Dopamine benefit is separate from inotropic effect and increased cardiac output is controversial.
  - Clonidine and calcium channel blockers may be protective.
  - ANPs and analogues appear to be protective.

### Splanchnic, Hepatic, & Visceral Effects

### Neurologic Effects

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Preparation for Bypass: Pre-bypass Checklist

1. Anticoagulation
   a. Heparin administered
   b. Desired level of anticoagulation achieved

2. Arterial cannulation
   a. Absence of bubbles in arterial line
   b. Evidence of dissection or malposition?

3. Venous cannulation
   a. Evidence of SVC obstruction?
   b. Evidence of IVC obstruction?

4. Pulmonary artery catheter (if used) pulled back

5. Are all monitoring/access catheters functional?

6. TEE (if used)
   a. In “freeze” mode
   b. Scope in neutral/unlocked position

7. Supplemental medications
   a. Neuromuscular blockers
   b. Anesthetics, analgesics, amnestics

8. Inspection of head and neck
   a. Color
   b. Symmetry
   c. Venous drainage
   d. Pupils

Uncomplicated Initiation

Checklist for Bypass Procedure

1. Assess arterial inflow
   a. Is arterial perfusate oxygenated?
   b. Is direction of arterial inflow appropriate?
   c. Evidence of arterial dissection?
      Patient’s arterial pressure persistently low?
      Inflow line pressure high?
      Pump/oxygenator reservoir level falling?
      Evidence of atrial cannula malposition?
      Patient’s arterial pressure persistently high or low?
      Unilateral facial swelling, discoloration?

2. Assess venous outflow
   a. Is blood draining to the pump/oxygenator’s venous reservoir?
      Evidence of SVC obstruction?
      Facial venous engorgement or congestion, CVP elevated?

3. Is bypass complete?
   a. High CVP/low PA pressure?
   b. Impaired venous drainage?
   c. Low CVP/high PA pressure?
   d. Large bronchial venous blood flow?
   e. Aortic insufficiency?
   f. Arterial and PA pressure nonpulsatile?
   g. Desired pump flow established?

4. Discontinue drug and fluid administration

5. Discontinue ventilation and inhalation drugs to patient’s lungs
Conduct of CPB

Best Practices Are Still Developing in Regard to
- Perfusion pressure
- Pump flow
- Temperature management
- Central nervous system monitoring
- Pulsatility

Management of Unusual Problems Encountered in Initiating & Maintaining CPB

Perfusion Emergencies
- **Acute aortic dissection** should be suspected if the CPB arterial line pressure unexpectedly increases and there is a simultaneous decrease in systemic pressure and/or venous drainage.
- Venous or arterial air embolism may occur from improper operation of CPB, surgical technique, or through intravenous lines.
- **Systemic air embolism** carries a greater risk than venous air embolism because of the potential for cerebral involvement.
- Air embolism originating from CPB may enter the patient’s systemic circulation from the arterial line or by other mechanisms such as improperly operated vents, cardioplegia delivery, or vacuum-assisted venous drainage or from pressurized CPB components.
- Improperly de-aired intravenous lines or inappropriate ventilation of the patient during insertion of cannulas or vents or during surgical de-airing maneuvers can result in air embolism.
- If massive air embolism from the CPB circuit occurs, bypass should be stopped immediately, the source of air determined, and efforts made to remove the air from the circuit and patient’s vasculature.
- Retrograde cerebral perfusion may be an effective treatment if significant air is suspected to have entered the patient’s cerebral circulation.
- The sterility of preassembled CPB circuits must be maintained by ensuring all blood-contacting surfaces and components are kept secure and covered until time of use.
Preparation for Separation-from-Bypass Checklist

1. Air clearance maneuvers completed
2. Rewarming completed
   a. Nasopharyngeal temperature 36-37°C
   b. Rectal/bladder temperature ≥ 35°C, but ≤ 37°C
3. Address issue of adequacy of anesthesia and muscle relaxation
4. Obtain stable cardiac rate and rhythm (use pacing if necessary)
5. Pump flow and systemic arterial pressure
   a. Pump flow to maintain mixed venous saturation ≥ 70%
   b. Systemic pressure restored to normothermic levels
6. Metabolic parameters
   a. Arterial pH, Po₂, Pco₂ within normal limits
   b. Hct: 20%-25%
   c. K⁺: 4.0-5.0 mEq/L
   d. Normal ionized calcium
7. Are all monitoring/access catheters functional?
   a. Transducers re-zeroed
   b. TEE (if used) out of freeze mode
8. Respiratory management
   a. Atelectasis cleared/lungs reexpanded
   b. Evidence of pneumothorax?
   c. Residual fluid in thoracic cavities drained
   d. Ventilation reinstituted
9. Intravenous fluids restarted
10. Inotropes/vasopressors/vasodilators prepared

Termination of CPB

TABLE 29.1. Mnemonic for preparations before termination of CPB

**LAMPS: Laboratory Data, Anesthesia, Monitors/Machine, Patient, Support**

**Laboratory data**
- pH, PCO₂ of arterial blood
- SO₂ of venous blood
- Serum NA⁺, K⁺, Ca²⁺, glucose
- Hematocrit
- Activated clotting time, heparin concentration, thromboelastogram

**Anesthesia/machine**
- Analgesia–supplemental opioid
- Amnesia–benzodiazepine
- Muscle relaxation–if needed
- Airway and functional oxygen delivery system
- Anesthesia machine on
- Adequate oxygen supply
- Breathing circuit intact
- Endotracheal tube connected, unkinked
- Ventilator functional
- Ability to ventilate both lungs confirmed
- Vaporizers off

**Monitors**
- Invasive blood pressure monitors-zeroed and calibrated
- Arterial catheter–radial, femoral or aortic
- Pulmonary artery catheter
- Central venous (right atrial) catheter

<table>
<thead>
<tr>
<th>C</th>
<th>V</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cold</td>
<td>Ventilation</td>
<td>Predictors</td>
</tr>
<tr>
<td>Conduction</td>
<td>Visualization</td>
<td>Pressure</td>
</tr>
<tr>
<td>Cardiac output</td>
<td>Vaporizer</td>
<td>Pressors</td>
</tr>
<tr>
<td>Cells</td>
<td>Volume expanders</td>
<td>Pacer</td>
</tr>
<tr>
<td>Calcium</td>
<td>Potassium</td>
<td>Protamine</td>
</tr>
</tbody>
</table>
• Left atrial catheter
• Electrocardiogram
• Rate—pacing capability
• Rhythm
• Conduction
• Ischemia—review all available leads
• Bladder catheter—urine output
• Pulse oximeter
• Capnometer/mass spectrometer
• Safety monitors—oxygen analyzer, circuit pressure alarm, spirometer
• Transesophageal echocardiogram
• Temperature (37°C nasopharangeal, 35°C rectal or bladder)

**Patient/pump**

• The heart
• Cardiac function—contractility, size
• Rhythm
• Ventricular filling
• Air removed
• Vent removed
• The lungs
• Inflation/deflation
• Compliance
• The field
• Bleeding
• Oxygenation—blood color
• Movement—a sign of inadequate anesthesia

**Support**

• Pharmacologic
• Inotropes
• Vasodilators
• Vasoconstrictors
• Antidysrhythmics
• Electrical
• Atrial and/or ventricular pacing
• Mechanical
• Intraaortic balloon counterpulsation
• Left and/or right ventricular assist device

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CPB in Infants & Children

- Historically, the need to correct serious congenital cardiac abnormalities has been a primary impetus to the advances in the discipline of cardiac surgery.
- The pathophysiology of CPB in neonates and infants generally parallels that in older children and adults but differs in some significant ways.
- During hypothermia, the “Q10” is greater for infants than for adults, so that a greater degree of metabolic suppression occurs for a given degree of hypothermia.
- DHCA may be best conducted with two different pH management strategies:
  - pH stat during cooling to augment brain blood flow and therefore improve cooling.
  - Alpha-stat before and during the circulatory arrest period.
- The significant physiologic and metabolic characteristics of the immature myocardium require protection strategies different from those used in adults.
- The immature lung may respond to CPB differently than does the adult lung, and in some cases it may benefit from liquid ventilation with fluorocarbon compounds.
- Neurologic abnormalities following congenital heart surgery are common; however, the pathophysiology is not uniform and the causes are often unclear.
- Preexisting abnormalities of the CNS are a possibility.
- Hypothermia per se does not appear to be responsible for CNS damage.
- Steroid therapy, to be most effective, should be started several hours before the circulatory arrest period.
- The most severe inflammatory responses to CPB appear to occur in patients at the extremes of age.
- Smaller blood volumes in infants compound the mechanical difficulties of CPB.
- Prime volumes and their content must be carefully controlled.
- Nonporous silicone rubber membrane oxygenators may be beneficial for long-term perfusion.
- The need for miniature tubing and cannulas presents a significant technical challenge in small patients.
- Anticoagulation can be more difficult to manage in neonates and infants.
- The pharmacokinetic and pharmacodynamics properties of heparin and protamine differ between pediatric patients and adults.
- Intrinsic coagulation abnormalities are present, especially in deeply cyanotic patients.
- Modified ultrafiltration (MUF) is a very valuable method for controlling body fluid balance during and after CPB in neonates and infants.

Difference between infants and adults

- Smaller circulating blood volume
- Higher oxygen consumption rate
- Reactive pulmonary vascular bed
- Presence of intracardiac and extra cardiac shunting
- Immature organ systems
- Altered thermoregulation
- Poor tolerance to microemboli

ECMO For Respiratory Or Cardiac Support

- Cardiopulmonary Bypass
Noncardiovascular Applications of CPB

- The noncardiovascular applications of CPB comprise four major categories:
  - DHCA for control of surgical bleeding and for organ (central nervous system) protection.
  - Noncardiac thoracic (pulmonary) applications.
  - Surgical procedures requiring occlusion of the IVC.
  - Miscellaneous unusual procedural applications.

Perfusion For Thoracic Aortic Surgery

CPB For Port-Access Cardiac Surgery

Special Patient Populations

Care of the Gravid Patient during Bypass

Maternal and Fetal Monitor Information
Conducting the Bypass Procedure
Blood Flow
Blood Pressure
Temperature

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8 Myocardial Protection

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- Normal Myocardial Perfusion 2
- Myocardial Ischemic Injury 3
- Strategies for Myocardial Protection 3

Cerebral & Spinal Protection 5
- Hypothermia 5
- Deep hypothermic circulatory arrest 5
- Left heart bypass 6
- Retrograde cerebral perfusion 6
- Antegrade (selective) cerebral perfusion 6
Unit Objectives

- To review the basic concepts of functional pathophysiology of surgical ischemia-perfusion injury.
- To review strategies of myocardial protection: cardioplegic and non-cardioplegic.
- To review cerebral and spinal protection.

Learning Objectives

By the end of this chapter the student should know:

- Pathophysiology of Surgical Ischemia-Perfusion Injury: Normal Myocardial Perfusion; Myocardial Ischemic Injury; Strategies for Myocardial Protection.
- Cerebral & Spinal Protection: Hypothermia; Deep hypothermic circulatory arrest; Left heart bypass; Retrograde cerebral perfusion; Antegrade (selective) cerebral perfusion.


Textbook

Chapters 2 and 3: Hypothermia, Circulatory Arrest, and Cardiopulmonary Bypass; Myocardial Management During Cardiac Surgery with Cardiopulmonary Bypass. Cardiac Surgery (Kirklin and Barratt-Boytes), 2nd ed., 61-165.


Pathophysiology of Surgical Ischemia-Perfusion Injury

Normal Myocardial Perfusion

- Normally, subendocardial flow exceeds subepicardial flow.
- Myocardial perfusion, however, is altered by cardiopulmonary bypass.
- Narrow pulse pressure and variable mean pressure affects coronary perfusion pressure.
- Wall tension is increased in the empty, smaller heart.
- Ventricular fibrillation also increases wall tension.
- Regulatory and inflammatory factors are released which affect coronary resistance.
- Microemboli from the circuit and hemodilution impair oxygen delivery.
- Endothelial and myocardial edema further affect perfusion.
- Subendothelial vulnerability is increased by hypertrophy, coronary disease, fibrillation, cyanosis, shock, and chronic heart failure.
- The acutely ischemic heart may have poor reflow to the injured area.


This article covers the particular challenge of protecting the heart compromised from infarction. The Buckberg and Emory reperfusion protocols are outlined and their merits discussed.
Myocardial Ischemic Injury

- **Intraoperative myocardial injury** can occur before, during, or after cardiopulmonary bypass.
- **Injury before cardiopulmonary bypass** is commonly "unprotected" (i.e., lacking cardiopulmonary bypass or cardioplegia).
- **Injury during cardiopulmonary bypass** can be multifactorial, related to the composition or delivery of cardioplegic protective solutions or to the reperfusion period after cardioplegic arrest.
- **Injury after cardiopulmonary bypass** commonly results from postischemic reperfusion.

The degree of permanent myocardial injury after ischemia is a function of the severity and duration of ischemia, which can be modified by numerous factors. **Reperfusion injury** is defined as additional myocardial injury incurred after restoration of blood flow to ischemic myocardium. Important contributors to this injury include calcium influx into cells, oxygen radicals, neutrophil activation and extravasation, complement, and edema. Impaired microvascular blood flow ("no-reflow" response) can result from these injury mechanisms. Endothelial dysfunction and injury contribute to this phenomenon. Reperfusion injury can cause atrial and ventricular dysrhythmias, reversible systolic and diastolic left ventricular dysfunction (stunning), myocardial necrosis, endothelial dysfunction, and apoptosis.

Strategies for Myocardial Protection

A variety of **strategies for myocardial protection** have been identified, which can be divided into established, emerging, and experimental strategies:

**Established Strategies:**
- **Chemically induced cardiac arrest** in diastole, most often induced by controlled hyperkalemia localized to the heart.
- **Hypothermia** to decrease myocardial oxygen consumption. The benefits of this approach appear to be optimal at myocardial temperatures between 24° and 28°C.
- **Avoidance or reduction of myocardial edema** by limiting the pressure of cardioplegia infusions and by providing moderately hyperosmolar cardioplegia solutions that contain blood.
- **Buffering the acidosis** that results from ischemia by including tromethamine (THAM), histidine-imidazole, or both in the cardioplegia solution.
- **Close management of myocardial calcium balance** to avoid extremes of intracellular hypercalcemia or hypocalcemia, especially during reperfusion.

**Emerging Strategies:**
- Therapies to avoid oxygen radical injury, such as superoxide dismutase and xanthine oxidase inhibitors.
- Amino acid enhancement with glutamate and aspartate to sustain anaerobic glycolysis during ischemia.
- Addition of adenosine to cardioplegia or "reperfusion" solutions.

**Manifestations of reperfusion injury**

The multiple etiologies of reperfusion injury following nonsurgical or surgical reperfusion can also present as numerous abnormalities in electrophysiology, contractile function, biochemistry, and morphology.

**The clinical manifestations of reperfusion injury, separate from those of ischemic injury, can be grouped into the categories listed below.**

**Reperfusion dysrhythmias;**
**Postischemic systolic and diastolic dysfunction;**
**Endothelial dysfunction; and**
**Myocardial necrosis;**
**Myocardial apoptosis**

**What is differences between apoptosis & necrosis?**
Apoptosis, or genetically programmed cell death, as opposed to the explosive and inflammation-initiating process of cellular necrosis.

**Apoptosis vs. Necrosis**
"LIFELESS" (since cells are dead):
- **L** -- Leaky membranes
- **I** -- Inflammatory response
- **F** -- Fate
- **E** -- Extent
- **S** -- Swell or shrink
- **-** -- Energy dependent

**Cordell AR. Milestones in the development of cardioplegia.**

A historical article with highlights major contributions in cardioplegia. A second article in the same issue specifically covers warm cardioplegia.
Experimental Strategies:
These include stimulation of nitric oxide production, antineutrophil therapy, complement pathway inhibition, hyperpolarizing agents, sodium–hydrogen exchange inhibitors, ischemic preconditioning, and strategies aimed at protecting the beating heart during surgical revascularization procedures performed without cardiopulmonary bypass.
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Cardioplegia
- Studies in animals have inconsistent correlation with clinical results due to species differences, extent of disease, and perioperative events that precipitate, extend, or enhance myocardial damage
- The goals of cardioplegia are to protect against ischemic injury, provide a motionless and bloodless field, and allow for effective post-ischemic myocardial resuscitation
- Cardioplegic techniques vary according to perfusate (blood vs. crystalloid), duration (continuous vs. intermittent), route (antegrade vs. retrograde), temperature (warm vs. cold), and additives
- Special consideration is required for the acutely ischemic heart and the neonate

Mechanisms of Cardioplegic Protection
- Mechanical arrest (potassium-induced) will reduce oxygen consumption by 80%
- Hypothermia will reduce consumption by another 10-15%
- Aerobic metabolism can be maintained with oxygenated cardioplegia
- Hypothermic arrest is sustained with readministration every 15-30 minutes
- Retrograde delivery protects the left ventricle more completely than the right ventricle
- Prevent myocardial rewarming with systemic hypothermia, aortic and ventricular vents, and caval occlusion
- In acute ischemia, use warm induction with substrate enhancement (glutamate, aspartate)
- Reperfusion should be controlled, using warm, hypocalcemic alkaline cardioplegia
- This approach combats intracellular acidosis and rapid calcium infusion injury
- Retrograde or low-pressure antegrade perfusion is preferred for reperfusion
- Ensure uniform warming

Cardioplegia Composition
- Blood has the advantage of oxygen carrying capacity, histidine and hemoglobin buffers, free radical scavengers in RBCs, and metabolic substrates
- Blood also has improved rheologic and oncotic properties, which may lessen myocardia edema
- Buffers such as THAM, histidine, and NaHCO3 form a slightly alkaline solution for reperfusion that can counteract intracellular acidosis
- Small amounts of calcium (0.1-0.5 mM/L) restores calcium that has been chelated by citrate
- Potassium concentrations range from 10-25 mM/L, with the first dose being the highest
- Other substrates are being evaluated, including allopurinal, SOD, deferoxamine, adenosine, nucleoside

| TABLE 8–1. Composition of Whole Blood (WB) and del Nido (DN) cardioplegia solutions |
|-------------------------------------------------|-----|----------------|-----|
| Na (mmol/L)                                      | 136-152 | 143-153          |
| K (mmol/L)                                       | 24    | 24              |
| Cl (mmol/L)                                      | 126   | 132             |
| Mg (mmol/L)                                      | 2     | 6.2             |
| Ca (mmol/L)                                      | 0.4   |                 |
| Lidocaine (mg/L)                                 | 200 (before XC release) | 140 |
| Mannitol (g/L)                                   | 12.5  | 2.6             |
| NaHCO3                                          | 50    | 26              |
transport inhibitors, and potassium-channel openers

**Neonates** and **Children**

- Children older than 2 months have similar myocardial physiology to adults
- The neonatal myocardium, however, is different in several ways
  - Hypoxia is more easily tolerated
  - There are greater glycogen stores and more amino acid utilization
  - ATP breakdown is slower due to deficiency in 5’ nucleotidase
  - Multidose cardioplegia is disadvantageous
  - Cyanosis may worsen resistance to ischemia
  - Amino acid substrate enhancement is beneficial

---

**Cerebral & Spinal Protection**

Cerebral protection is necessary when bypass cannot supply the head vessels, e.g., during surgery of the distal ascending aorta and arch. There are two modalities: hypothermia, which is by far the most important, and retrograde or antegrade selective cerebral perfusion.

Spinal cord protection includes hypothermia and CSF drainage aimed at reducing risk of paraplegia in thoracoabdominal surgery.

**Hypothermia**

During coronary and valve surgery most surgeons cool actively or passively (drift) to about 32°C. At 37°C irreversible cerebral damage occurs after 2–3min of ischemia. Cerebral O2 consumption is reduced by approximately 5% per 1°C drop between 37°C and 22°C. At 20°C cerebral O2 consumption is 20% of normothermia. Although this suggests a safe circulatory arrest time of 15–20min, clinical evidence suggests that 30min is tolerated, rising to 45min at 18°C as other factors including pH and suppression of neuronal function may pay a role.

**Deep hypothermic circulatory arrest**

After institution of bypass the patient is cooled to a point where bypass can be discontinued. This technique is used in distal ascending aorta and arch repair, and sometimes during pulmonary embolectomy and stage II elephant trunk. Due to decreased tissue metabolism and O2 demand, even a small degree of systemic hypothermia allows lower pump flows, less blood trauma, better myocardial protection, and better organ protection than normothermic perfusion. Hypothermia decreases blood flow to vascular beds in proportion with reduced metabolic demands, most pronounced in skeletal muscle and the extremities.

**TABLE 8–5. Safe duration of circulatory arrest (in minutes)**

<table>
<thead>
<tr>
<th>Temperature (°C)</th>
<th>Time (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>36</td>
<td>&lt; 2–3min.</td>
</tr>
<tr>
<td>32</td>
<td>3–5min.</td>
</tr>
<tr>
<td>28</td>
<td>10min (moderate hypothermia)</td>
</tr>
<tr>
<td>20</td>
<td>30min (DHCA).</td>
</tr>
<tr>
<td>15</td>
<td>45min (DHCA).</td>
</tr>
</tbody>
</table>

**TABLE 8–6. Alpha- & pH-stat**

- **PH-stat** tries to keep the pH stable by adding CO2 as temperatures fall and removing it as they rise.
- **Alpha-stat** does not add anything. PaCO2 rises and pH falls with falling temperature.
Left heart bypass

This is a useful adjunct in surgery on the descending aorta, where the heart does not need to be arrested, but the distal descending aorta and lower limbs need perfusion during the period of descending aorta cross-clamp. The LA is cannulated and oxygenated blood is siphoned into a reservoir, and pumped into a femoral line. An oxygenator is not required as blood is oxygenated by the lungs. A heat exchanger is optional as the patient is allowed to cool passively.

Without these components there is less need for heparinization: 100U/kg heparin is normally given, and the ACT is maintained at <200s. Flows are maintained at 75% of full flow and MAPs distal to the clamp at 55–70mmHg.

Retrograde cerebral perfusion

Bicaval cannulas and a venous to arterial bridge are required: when retrograde perfusion is required bypass is discontinued, the IVC cannula is clamped, the SVC cannula is snared, and the perfusionist uses the bridge to pump oxygenated blood up the venous line. Deoxygenated blood is drained from the head and neck vessels.

Low, near-continuous flow is used by some surgeons during hypothermic circulatory arrest. The common observation of dark, deoxygenated blood flowing out of the neck vessels suggests that cerebral oxygen utilization occurs.

Antegrade (selective) cerebral perfusion

This is used in arch surgery to reduce DHCA time. The brachiocephalic artery and left common carotid artery can be directly cannulated with a retrograde cardioplegia cannula. Or, if the axillary artery has been cannulated and the head and neck vessels anastomosed to a trifurcation graft the head can be selectively perfused from the axillary artery (b p449). This reduces the risk of embolization from instrumenting the vessels, and keeps the field clearer.

Flows are maintained at 10mL/kg, MAPs 40–50mmHg, and temperature around 20°C.

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Unit Objectives
This chapter will briefly
1. discuss the major pathophysiologic derangements and their management during the first 24 hours after surgery.
2. summarize the postoperative care to more specific procedures.
3. summarize the management of common postoperative complications will be discussed.

Learner Objectives
By the end of this chapter the student should know:
• Clinical Assessment & Monitoring.
• Cardiovascular management: coagulation; bleeding; hemotherapy & transfusion medicine.
• Respiratory management
• Fluid, electrolyte and renal management: fluid resuscitation; the acidic patient; renal management; fluid management
• Management of Sepsis
• Postoperative Nutrition
• Gastrointestinal Management
• Neurological Management
• Pain Management: postoperative pain relief; pain management and techniques; anesthesia & pain control in children
• Postoperative Anticoagulation: thrombosis; prosthetic valves
• Perioperative TEE
• Management of postop complications

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Clinical Assessment & Monitoring

- Although a stethoscope should be present in every anesthetizing location, continuous stethoscopy is an insensitive method for early detection of untoward hemodynamic events.
- Most automated noninvasive blood pressure measuring devices use an oscillometric measurement technique and rarely cause complications. Caution should be exercised in patients who cannot complain of arm pain, those with irregular rhythms that force repeated cuff inflation, and individuals receiving anticoagulant therapy.
- Direct arterial pressure monitoring should be widely used in operative patients with severe cardiovascular diseases or those undergoing major surgical procedures that involve significant blood loss or fluid shifts.
- The Allen test for palmar arch collateral arterial flow is not a reliable method to predict complications from radial artery cannulation. Despite the absence of anatomic collateral flow at the elbow, brachial artery catheterization for perioperative blood pressure monitoring is a safe alternative to radial or femoral arterial catheterization.
- The accuracy of a directly recorded arterial pressure waveform is determined by the natural frequency and damping coefficient of the pressure monitoring system. Optimal dynamic response of the system will be achieved when the natural frequency is high, thereby allowing accurate pressure recording across a wide range of damping coefficients.
- Rather than the common placement at the midaxillary line, the preferred

Vincent Mnemonic: FAST HUG
F—Feeding
A—Analgesia
S—Sedation
T—Thromboprophylaxis
H—Head of bed elevation
U—Ulcus prophylaxis
G—Glycemic control
position for alignment (or “leveling”) of external pressure transducers is ≈ 5 cm posterior to the sternomanubrial junction. When using external transducers and fluid-filled monitoring systems, this transducer location will eliminate confounding hydrostatic pressure measurement artifacts.

- Because of wave reflection and other physical phenomena, arterial blood pressure recorded from peripheral sites has a wider pulse pressure than central aortic pressure does.
- Selecting the best site, catheter, and method for safe and effective central venous cannulation requires that the physician consider the purpose of the catheterization, the patient's underlying medical condition, the intended operation, and the skill and experience of the physician performing the procedure. Right internal jugular vein cannulation is favored by most anesthesiologists because of its consistent, predictable anatomic location and its relative ease of access intraoperatively.

Most randomized prospective clinical trials have failed to show that pulmonary artery catheter monitoring results in improved patient outcome. Reasons cited for these results include misinterpretation of catheter-derived data and failure of hemodynamic therapies that are guided by specific hemodynamic indices.

- Methods to reduce mechanical complications from central venous catheters include the use of ultrasound vessel localization, venous pressure measurement before insertion of large catheters, and radiographic confirmation that the catheter tip rests outside the pericardium and parallel to the walls of the superior vena cava.
- PCWP is a delayed and damped reflection of left atrial pressure. The PCWP provides a close estimate of pulmonary capillary pressure in many cases but may underestimate capillary pressure when postcapillary pulmonary vascular resistance is increased, as in patients with sepsis.
- Use of CVP, PADP, or PCWP as an estimate of LV preload is subject to many confounding factors, including changes in diastolic ventricular compliance and juxtacardiac pressure.
- Thermodilution CO monitoring, the most widely used clinical technique, is subject to measurement errors introduced by rapid IV administration, intracardiac shunts, and TR.
- SVO₂ is a measure of the adequacy of cardiac output relative to body oxygen requirements. It is also dependent on arterial hemoglobin oxygen saturation and hemoglobin concentration.
- Newer methods of CO monitoring, including esophageal Doppler and pulse contour analysis, allow beat-to-beat estimation of left ventricular stroke volume and measurement of other cardiovascular variables.

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**Cardiovascular Management**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO = cardiac output</td>
<td>4.5–8L/min</td>
</tr>
<tr>
<td>SV = stroke volume</td>
<td>60–100mL</td>
</tr>
<tr>
<td>BSA = body surface area</td>
<td>2–2.2 m²</td>
</tr>
<tr>
<td>CI = cardiac index</td>
<td>2.0–4.0L/min/m²</td>
</tr>
<tr>
<td>SVI = stroke volume index</td>
<td>33–47 mL/beat/m²</td>
</tr>
<tr>
<td>MAP = mean arterial pressure</td>
<td>70–100mmHg</td>
</tr>
<tr>
<td>DP = diastolic pressure</td>
<td>60–80mmHg</td>
</tr>
<tr>
<td>SP = systolic pressure</td>
<td>110–150mmHg</td>
</tr>
</tbody>
</table>

**DEFINITIONS OF COMMON TERMS**

- **Cardiac output** = SV x HR
- **Cardiac index** = CO/BSA
- **Mean arterial pressure** = (DP – SP)/3
- **Systemic vascular resistance** = ((MAP – CVP)/CO) x 80
- **Systemic vascular resistance index** = SVR/BSA
- **Pulmonary vascular resistance** = ((PAP – PCWP)/CO) x 80
- **Pulmonary vascular resistance index** = PVR/BSA

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Mixed venous = $\text{SaO}_2 - (\text{VO}_2/(\text{Hb} \times 1.39 \times \text{CO})) \times 10$

Mnemonic: Causes of SHOCK

Spinal (neurogenic), Septic

Hemorrhagic

Obstructive (e.g. tension pneumothorax, cardiac tamponade, PE)

Cardiogenic (e.g. arrhythmia, MI)

Anaphylactic

| SVR = systemic vascular resistance | 800–1200 dyne/s/cm$^5$ |
| CVP = central venous pressure | 0–12mmHg |
| SVRI = systemic vascular resistance index | 400–600 dyne/s/cm$^5$/m$^2$ |
| PVR = pulmonary vascular resistance | 50–250 dyne/s/cm$^5$ |
| PAP = pulmonary artery pressure | 20–30mmHg |
| PAWP = pulmonary artery wedge pressure | 8–14mmHg |
| PVRI = pulmonary vascular resistance index | 20–125 dyne/s/cm$^5$/m$^2$ |
| SaO$_2$ = arterial saturation | 95–100% |
| VO$_2$ = oxygen consumption | 200–250mL/min |

### Coagulation

- Under normal physiologic conditions, clot formation requires participation of vascular endothelium, platelets, and plasma-mediated hemostasis.
- Tissue factor (extrinsic pathway) initiates plasma-mediated hemostasis, whereas factor XI (intrinsic pathway) amplifies the response.
- Thrombin generation proves the key regulatory enzymatic step in hemostasis.
- Platelets participate in clot formation as
  - (1) anchoring sites for coagulation factor activation complexes;
  - (2) delivery "vehicles" releasing hemostatically active proteins; and
  - (3) major structural components of the clot.
- A carefully performed history of bleeding remains the most effective means for identifying bleeding and thrombotic tendencies.
- Thrombosis is potentiated by stasis, vascular endothelial injury, and underlying hypercoagulable conditions.
- Heparin-induced thrombocytopenia (HIT) comprises a heparin-mediated autoimmune response potentiating platelet activation as well as venous and arterial thromboses.

Under normal physiologic conditions, clot formation requires participation of vascular endothelium, platelets, and plasma-mediated hemostasis.

Heparin-induced thrombocytopenia (HIT) comprises a heparin-mediated autoimmune response potentiating platelet activation as well as venous and arterial thromboses.
FIGURE 9–22. A stepwise approach to the diagnosis and initial treatment of the hemodynamically unstable postoperative cardiac patient
**Bleeding**

- Blood Products, Antifibrinolytics
- Transfusion Reactions
- Re-Exploration for Bleeding

### TABLE 9–25. Defined Adverse Reactions

<table>
<thead>
<tr>
<th>Presenting With Fever</th>
<th>Presenting Without Fever</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acute</strong></td>
<td><strong>Delayed</strong></td>
</tr>
<tr>
<td>≤ 24 hrs after transfusion</td>
<td>&gt; 24 hrs after transfusion</td>
</tr>
<tr>
<td>- Acute Hemolytic (AHTR)</td>
<td>- Delayed Hemolytic (DHT)</td>
</tr>
<tr>
<td>- Febrile Non-hemolytic (FNHT)</td>
<td>- TA-GVHD</td>
</tr>
<tr>
<td>- Transfusion-related Sepsis</td>
<td>-</td>
</tr>
<tr>
<td>- TRALI</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Acute</strong></th>
<th><strong>Delayed</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 24 hrs after transfusion</td>
<td>&gt; 24 hrs after transfusion</td>
</tr>
<tr>
<td>- Allergic</td>
<td>- Delayed Serologic (DSTR)</td>
</tr>
<tr>
<td>- Hypotensive</td>
<td>- Post-transfusion Purpura (PTP)</td>
</tr>
<tr>
<td>- Tx-associated Dyspnea (TAD)</td>
<td>- Iron Overload</td>
</tr>
<tr>
<td>- TACO</td>
<td></td>
</tr>
</tbody>
</table>

**Hemotherapy & Transfusion Medicine**

- In terms of the transfusion-transmitted infectious diseases, the American blood supply has never been safer than it is today.
- In the setting of massive transfusion, assuming maintenance of isovolemia, critical dilution of clotting factors and platelets will occur after an average replacement of 140% and 230% of blood volume, respectively.
- Coagulation factor and platelet replacement should be determined by laboratory assessment and/or observation of clinical coagulopathy and not EBL-driven formulas.
- Platelet administration thresholds relevant to anesthesiologists will lie usually between 50,000 and 100,000/ul.
- A patient who has received 10 to 12 units of group O RBCs should not be switched back to his or her own ABO group unless testing has been performed to confirm that significant titers of Anti-A or Anti-B antibodies are not present.
- The classical, dual-cascade (intrinsic and extrinsic pathway) model of coagulation is an inadequate representation of coagulation, as it occurs in vivo.
- In vivo, coagulation is initiated principally by contact of factor VII with extravascular tissue factor leading first to platelet activation followed by the generation of large amounts of thrombin by activated clotting factors acting on the phospholipid surface provided by activated platelets.
- Von Willebrand's disease is the most common hereditary bleeding disorder and some form of the disease, which may be subclinical prior to surgery, is present in approximately 1% of the population.
- Factors II, VII, IX, and X and Proteins C and S are dependent on vitamin K for their synthesis. Vitamin K deficiency occurs frequently in hospitalized patients because of dietary insufficiency, gut sterilization, and malabsorption. A high index of suspicion should be maintained.
- PAD became accepted as a standard practice in certain elective surgical settings such as total joint replacement surgery, so that by 1992 more than 6% of the blood transfused in the United States was autologous. Subsequently, substantial improvements in blood safety have been accompanied by a decline in PAD as well as an interest in ANH as an alternative, lower cost strategy.
- The criteria for autologous donors are not as stringent as those for allogeneic donors. Transfusion service policies, implemented under the auspices of hospital transfusion committees, differ regarding collection and

---

**Mnemonics for transfusion reactions:** **AFH**

- **A llergy;** Febrile; and Hemolytic

**The three leading causes of transfusion-related death in the United States are**

1. ABO incompatibility,
2. Transfusion-related acute lung injury (TRALI), and

**The red blood cell (RBC) transfusion “trigger” for most patients will lie between hemoglobin values of 7 and 10 g/dL.**

**Normal coagulation can be achieved with clotting factor levels of 20 to 30% of normal. Those levels can usually be achieved by administration of 10 to 15 mL/kg of fresh frozen plasma (FFP).**

**As many as 5% of patients who receive heparin therapy for 5 days will develop heparin-induced thrombocytopenia/thrombosis. The clinical manifestations are more often the result of thrombosis and thromboembolism than thrombocytopenia.**

**The three types of autologous blood transfusion are**

1. Preoperative autologous donation (PAD),
2. Acute normovolemic hemodilution (ANH), and
3. Intraoperative and postoperative blood recovery (salvage).

**Bloodless medicine and surgery is defined using a team approach that reduces blood loss and employs the best available alternatives to allogeneic transfusion therapy while...**
use of autologous blood with positive viral markers. It is common practice to preclude the blood reactive for hepatitis B surface antigen and human immunodeficiency virus because of concerns for the safety of both patients and personnel. Contraindications include evidence of infection and risk of bacteremia, scheduled surgery for correction of aortic stenosis, and unstable angina.

• Although autologous blood collections have become popular, the costs associated with their collection are higher than those associated with the collection of allogeneic blood.

• ANH is the removal of whole blood from a patient while restoring the circulating blood volume with an acellular fluid shortly before an anticipated significant surgical blood loss. The chief benefit of ANH is the reduction of red blood cell losses when whole blood is shed perioperatively at lower hematocrit levels after ANH is completed.

• Because there is no good evidence that either PAD or ANH is effective at eliminating allogeneic blood transfusions, these autologous blood collection techniques cannot be considered cost-effective alternatives to allogeneic blood.

• Postoperative blood collection denotes the recovery of blood from surgical drains followed by reinfusion, with or without processing. Postoperative autologous blood transfusion is practiced widely but not uniformly.

• Fibrin glue is derived from a source of fibrinogen and factor XIII (fibrin-stabilizing factor), in which a solution of fibrinogen is mixed with a solution of thrombin and applied to a surgical field. These preparations represent additional allogeneic blood donor exposure. Patients should be made aware of the potential complications as well as the potential benefits.

• Recombinant factor VIIa (rFVIIa) has been approved for treatment of bleeding in hemophilia patients with inhibitors. It has also been successful in patients without hemophilia with acquired antibodies against factor VIII (acquired hemophilia). Pharmacologic doses of rFVIIa enhance the thrombin generation on already activated platelets and, therefore, may also be of benefit in providing hemostasis in other situations, such as those characterized by profuse bleeding and impaired thrombin generation.

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Respiratory management

• Removal of $CO_2$ is determined by alveolar ventilation, not by total, minute ventilation.

• Dead space ventilation can be dramatically $\uparrow$ in patients with COPD and PE to more than 80% to 90% of minute ventilation in the extreme case.

• Breathing at low lung volume $\uparrow$ airway resistance and promotes closure of airways.

• Hypoxemia can be caused by alveolar hypoventilation, diffusion impairment, ventilation-perfusion mismatch, and right-to-left shunt.

• Almost all anesthetics $\downarrow$ muscle tone $\downarrow$ FRC to close to awake residual volume.

• $\downarrow$ FRC during anesthesia $\downarrow$ ventilation with a high $O_2$ concentration $\rightarrow$ atelectasis.

• General anesthesia $\rightarrow$ ventilation-perfusion mismatch (airway closure) $\uparrow$ shunt (atelectasis).

• Hypoxic pulmonary vasoconstriction is blunted by most anesthetics, thereby enhancing any ventilation-perfusion mismatch.

• $\uparrow$ Respiratory work during anesthesia, $\leftrightarrow$ respiratory compliance ($\downarrow$ lung volume available for ventilation?) $\uparrow$ $\uparrow$ airway resistance ($\downarrow$ FRC $\rightarrow$ $\downarrow$ airway dimensions?).

• Hypoxemia $\leftrightarrow$ $\downarrow$ $P_{O_2}$, hypoventilation, $\uparrow$ ventilation-perfusion ($V/Q$) heterogeneity, $\uparrow$ shunt, and $\uparrow$ diffusion nonequilibrium. Hypercapnia is focusing on the provision of the best possible medical care to all patients. Some patients, however, object to receiving blood or blood products as part of their medical treatment on religious grounds or because of concern about the safety of blood transfusions, regardless of their religious background.

The term *intraoperative blood collection or recovery* describes the technique of collecting and reinfusing blood lost by a patient during surgery. The oxygen-transport properties of recovered red blood cells are equivalent to stored allogeneic red blood cells. The survival of recovered blood cells appears to be at least comparable to that of transfused allogeneic red blood cells.

Lung Function After Heart Surgery
Ventilation Management
Respiratory Support Modes
Poor Gases In Ventilated Patients
Respiratory Failure On Ventilator Weaning From Ventilation
Minitracheostomy or Tracheostomy

Preoxygenation before and during induction of anesthesia is a major cause of atelectasis.

A clinically useful approximation to the alveolar gas equation for $O_2$ is given by $P_{O_2} = (P_b - 47) \times Fio_2 - 1.2 \times Pco_2$. Exchange of $O_2$ and $CO_2$ takes place independently in the lung.

Pulse oximetry is a rapid, reliable indicator of oxygenation status in surgical and critically ill patients. Newer oximeters feature reduced capability.

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almost always due to hypoventilation.

- During mechanical ventilation in the operative and intensive care settings, hypoxemia is most often due to \( \frac{V}{Q} \) heterogeneity and shunt.

- The alveolar-arterial (a-a) gradient \( \uparrow \) with age and supplemental \( O_2 \). The \( P_{aO_2}/FiO_2 \) and a/a ratios typically do not change with increased age or inspired \( O_2 \).

- When derangements in gas tensions are noted on ABG analysis, it is important to verify that the sample was obtained and analyzed in an appropriate and timely manner.

- Refinements and further studies on continuous intravascular blood gas monitors may one day lead to widespread routine use of these devices.

- Multiwavelength pulse oximeters are commercially available and allow measurement of carboxyhemoglobin and methemoglobin. Pulse oximetry may one day prove to be a reliable noninvasive monitor of volume status and fluid responsiveness.

- A sudden \( \downarrow \) \( Petco_2 \) \( \leq \) a circuit disconnection, airway obstruction, abrupt \( \downarrow \) cardiac output, or pulmonary embolism. \( Petco_2 \) is not always a reliable approximation of \( Paco_2 \), particularly during general anesthesia or in the critically ill.

- Mapping of pressure-volume curves in patients with ARDS and acute lung injury (ALI) can provide valuable information about lung mechanics and help guide PEEP and tidal volume settings. Sustained high airway pressure is needed to open collapsed alveoli, and PEEP stabilizes the recruited lung units.

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Fluid, electrolyte and renal management

Fluid Resuscitation

The Acidotic Patient

- The presence of a significant acid-base abnormality often signals a serious underlying problem.
- **Metabolic acidosis** is caused by decreased SID or increased $A_{TOT}$. Decreased SID results from accumulation of metabolic anions (shock, ketoacidosis, and renal failure), hyperchloremia, and free water excess. Increased $A_{TOT}$ results from hyperphosphatemia.
- **Metabolic alkalosis** is caused by increased SID or decreased $A_{TOT}$. SID increases as a result of sodium gain, chloride loss, or free water deficit. $A_{TOT}$ decreases in hypoalbuminemia and hypophosphatemia. This is particularly common in critical illness.
- Most acid-base disorders are treated by reversal of the cause.

All acid-base abnormalities result from alterations in the dissociation of water.

Only three factors independently affect acid-base balance—the $Paco_2$, the strong ion difference (SID), and the total concentration of weak acids ($A_{TOT}$).

Respiratory acidosis is caused by hypercarbia, and respiratory alkalosis is caused by hypocarbia.

**TABLE 9-52. Major consequences acid-base abnormalities**

<table>
<thead>
<tr>
<th>Organ system</th>
<th>Acidemia</th>
<th>Alkalemia</th>
</tr>
</thead>
</table>
| Cardiovascular | • Impairment of cardiac contractility  
• Arteriolar dilatation  
• Hypotension  
• e cardiovascular responsiveness to catecholamines  
• e sensitivity and e threshold for arrhythmias  
• ePVR | • Arteriolar constriction  
• ↓ coronary blood flow  
• ↑ threshold for arrhythmias |
| Respiratory | • Hyperventilation  
• ↓ strength of respiratory muscles and promotion of muscle fatigue  
• Respiratory failure | • Hypoventilation  
• Hypercapnia and hypoxemia |
| Metabolic | • Inhibition of anaerobic glycolysis  
• Hyperkalemia  
• Insulin resistance  
• ▼ ATP synthesis | • Stimulation of anaerobic glycolysis  
• Hypokalemia  
• ▼ ionized calcium  
• Hypomagnesemia  
• Hypophosphatemia |
| Cerebral | • Inhibition of metabolism and cell volume  
• Regulation, altered mental status | • ▼ cerebral blood flow  
• Seizures  
• Altered mental status  
• Tetany |
Renal Management

- To cross the filtration barrier between plasma and tubular fluid, a molecule must pass in succession through the endothelial fenestrations, the glomerular basement membrane, and the epithelial slit diaphragm. The capillary endothelium restricts the passage of cells, but the basement membrane filters plasma proteins. All three layers contain negatively charged glycoproteins, which retard the passage of other negatively charged proteins. Thus, the filtration barrier is size selective and charge selective.

- A primary determinant of glomerular filtration rate (GFR) is the glomerular filtration pressure, which depends not only on the renal artery perfusion pressure but also on the balance between afferent and efferent arteriolar tone. In the presence of ↓ afferent arteriolar pressure or blood flow, low levels of catecholamines, angiotensin, and arginine vasopressin (AVP) induce preferential efferent arteriolar constriction, which maintains glomerular filtration pressure. This is reflected by an increase in calculated filtration fraction (FF), which is the GFR expressed as a fraction of the renal plasma flow (RPF), that is, FF = GFR/RPF. High levels of catecholamines and angiotensin (but not AVP) ↑ afferent arteriolar tone and ↓ glomerular filtration pressure (and GFR) out of proportion to RPF, and FF decreases.

- Tubuloglomerular feedback may be a primary mechanism in renal autoregulation. When GFR is increased, distal tubular NaCl delivery is enhanced. The increase in chloride is sensed by the macula densa, which triggers the release of renin from the adjacent afferent arteriole. Angiotensin is elaborated and arteriolar constriction ensues, which ↓ GFR. When the thick ascending loop becomes ischemic, reabsorption of NaCl ceases, the ability of the tubule to concentrate urine is lost, and, theoretically, intractable polyuria should result. It has been suggested that the ↑ delivery of NaCl to the macula densa triggers angiotensin-mediated arteriolar constriction, which ↓ GFR, induces oliguria, conserves intravascular volume, and protects the organism from dehydration—so-called acute renal success.

- Autoregulation enables the kidney to maintain solute and water regulation independently of wide fluctuations of arterial blood pressure. It is noteworthy that urinary flow rate is not subject to autoregulation. Tubular water reabsorption determines urinary flow rate and is closely related to the hydrostatic pressure in the peritubular capillaries. Hypotension, whether induced or inadvertent, results in ↓ urinary flow rate that may be correctable only when the arterial blood pressure is restored toward normal.

- The juxtaglomerular apparatus consists of three groups of specialized tissues. In the afferent arteriole, modified fenestrated endothelial cells produce renin; in the juxtaposed distal tubule, cells of the macula densa act as chemoreceptors; and in the glomerulus, mesangial cells have contractile properties. Together these provide an important regulating system for blood pressure, salt, and water homeostasis.

- All anesthetic techniques and drugs ↓ RBF, but filtration fraction is usually ↑ which implies that angiotensin-induced efferent arteriolar constriction limits the ↓ GFR. However, these effects are much less significant than those caused by surgical stress or aortic cross-clamping and after emergence from anesthesia usually resolve promptly. Any anesthetic technique that induces hypotension ↓ urine flow because of altered peritubular capillary hydrostatic gradients, even if renal autoregulation is preserved (as it usually is during anesthesia). Permanent injury seldom results, unless the kidneys are abnormal to begin with or the hypovolemic insult is prolonged and exacerbated by nephotoxic injury.

- Clinically significant renal injury with the use of low-flow sevoflurane...
anesthesia has not been reported in patients, even with moderate preexisting renal dysfunction. The relationship between compound A formation, biochemical injury, and clinically relevant renal dysfunction remains unclear and unproven. Nonetheless it appears prudent to follow current FDA guidelines, which recommend a fresh gas flow of at least 2 L/min to inhibit compound A formation and its rebreathing and to enhance its washout.

- Regardless of the position of the aortic cross-clamp, RBF is $\downarrow$ to 50% of normal during surgical preparation of the aorta, presumably due to direct compression or reflex spasm of the renal arteries. After release of the suprarenal cross-clamp, RBF increases above normal (reflex hyperemia), but GFR remains depressed to one third of control for up to 2 hours. After 24 hours, GFR is still only two thirds of control. Tubular functions (concentrating ability, sodium, and water conservation) are markedly impaired, but urine flow is maintained. It has been observed that these changes resemble an attenuated form of acute tubular necrosis. In the above study all patients received mannitol pretreatment, which probably limited the tubular insult because oliguria was uncommon and recovery was relatively rapid. Cross-clamp times longer than 50 minutes are associated with prolonged depression of GFR and transient azotemia.

- The beneficial effect of AVP on renal function in sepsis may in part be due to its ability to $\uparrow$ low renal perfusion pressure back into the autoregulatory range. Another important factor is that, unlike norepinephrine, even at high local concentrations AVP preferentially constricts the effenter arteriole, thereby improving filtration fraction and GFR.

- The mechanism for perioperative ARF is complex and most commonly involves multiple factors such as ischemia/reperfusion, inflammation, and toxins.

- Repeated direct perioperative assessment of renal hemodynamics, tubular function, or pathogenesis of perioperative renal dysfunction is impractical; therefore, indirect assessments, such as serum creatinine trends, are the best practical currently available perioperative tool to assess renal function.

- Intraoperative urine formation depends on a number of factors and is an insensitive and unreliable method for assessing postoperative risk of renal dysfunction.

- Serum chemistries and urine indices such as blood urea nitrogen, creatinine, fractional excretion of sodium, and free water clearance are generally late indicators of renal function deterioration and do not enable the clinician to clearly delineate the cause of renal failure.

- Creatinine clearance is the most sensitive and specific clinical method for determining renal function, but it is limited by time and measurement restrictions.

- Early biochemical markers for renal function hold promise and are a current focus for research that may soon lead to new tests able to provide prompt clinical information.

- The removal of sodium and water cannot be dissociated when using diuretics or in certain renal replacement techniques. Diuretics $\rightarrow$ natriuresis, whereas dialysis $\rightarrow$ hypotonia or hypertonia, depending on the effect of dialysis on the diffusion and on the removal of molecules, including urea and other electrolytes.

- The ultrafiltrate composition produced from continuous venovenous hemofiltration (CVVH) and from hemofiltration in general is similar to plasma water, but the sodium concentration in the UF can be significantly affected by the sodium concentration in the replacement solution.

- Sodium removal can be dissociated from water removal in CVVH, thus obtaining a real change of the sodium pool in the body. This effect on sodium cannot be achieved with any other technique.

- The UNLOAD trial is the first randomized comparison of intravenous diuretic therapy alone against an alternative therapy, ultrafiltration, in hypervolemic patients. The principal findings of this trial were (a) in hypervolemic patients with congestive heart failure (CHF), ultrafiltration led very steep gain. Even mild dehydration results in a rapid antidiuresis, and urine osmolality can increase from 300 to 1200 mOs/kg as plasma AVP levels rise from 0 to 5 pg/mL. Decreases in intravascular volume also stimulate AVP secretion, mediated by stretch receptors with vagal afferents in the left atrium and pulmonary veins.

Hypovolemia-induced secretion of AVP overrides osmolar responses and contributes to the perioperative syndrome of inappropriate antidiuretic hormone secretion (SIADH): fluid retention, hypo-osmolality, and hyponatremia. The situation is exacerbated by administration of large volumes of hypotonic solutions that decrease serum osmolality. Psychiatric stress, via cortical input, also induces AVP release and can override osmotic and volume sensors.

Perioperative acute renal failure (ARF), although uncommon, is associated with extremely high morbidity and mortality rates.

In contrast to dopamine, there does appear to be increasing evidence to support a renoprotective effect for infusion of low-dose fenoldopam infusion (0.1–0.3 µg/kg/min) during cardiac surgery.

Risk factors included elevated preoperative serum creatinine (>1.5 mg/dL), age older than 70 years, diabetes, and previous cardiac surgery. Patients who received fenoldopam had a decreased incidence of acute kidney injury and requirement for dialysis.

Slow continuous ultrafiltration (SCUF) produces an ultrafiltration (UF) that varies from plasma water minimally due to Donnan effects. The UF from SCUF is iso-osmotic and isonatremic because sodium elimination is linked to the sodium concentration in plasma.

The best evidence to date supports a renal replacement therapy dose of at least 35 mL/kg/hr—spKt/V 1.4—for CVVH, continuous venovenous hemodiafiltration (CVVHDF), or daily intermittent hemodialysis (IHD).

There is evidence that, when the circuit set-up is perfectly optimized, anticoagulants are only a relatively minor component of circuit patency: in fact, when patients have bleeding
to greater weight and fluid loss than intravenous diuretics at the doses used in this trial; (b) volume removal with ultrafiltration at the index hospitalization was associated with significant reductions in the rate and durations of subsequent hospitalizations and fewer unscheduled medical visits for CHF; and (c) the benefits from the short-term use of ultrafiltration over 90 days was achieved without significant adverse effects.

- It is now possible to generate **ultrapure replacement fluid** and administer it in the ICU with a lower cost than **continuous renal replacement therapy (CRRT)**, in greater amounts and for shorter periods of time. The choices are now almost limitless; 3 or 4 hours of IHD with standard settings or CRRT at 35 mL/kg/hr of effluent flow rate can be selected. **Slow low-efficiency extended daily dialysis (SLEDD)** at blood and dialysate flow rates of 150 mL/min for 8 hours during the day or SLEDD for 12 hours overnight can be considered as an alternative.

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### Fluid Management

- **Calcium** is the key component that mediates muscle contraction; exocrine, endocrine, and neuromuscular secretion; cell growth; and the transport and secretion of fluids and electrolytes.
- **Magnesium** is essential for many biochemical reactions; its pharmacologic properties have only more recently been appreciated.
- **Phosphate** stores and releases energy through high-energy phosphate bonds and is integral to the structure of proteins, lipids, and bone.
- **Chloride** is the predominant anion in the ECF.
- **Glucose** is a crucial fuel source, and insulin facilitates glucose movement into cells in a process that also requires potassium and phosphate.
- **Diabetes** affects multiple organ systems, and the perioperative effect of diabetes can be profound.
- The most common causes of **metabolic alkalosis** are antacid therapy, incidental administration of citrate with blood products, sodium bicarbonate administration, gastric drainage, and renal bicarbonate retention.
- **Metabolic acidosis** is commonly caused by low CO and end-stage liver disease.
- **Transfusion of blood products** improves tissue oxygenation and decreases bleeding, but it also increases the risk of transmission of infectious diseases, transfusion reactions, immunosuppression, and alloimmunization.
- **Anesthetics** may blunt the normal physiologic responses to hypovolemia and the stress response.
- **Shock** is dysfunction of intracellular processes caused by the lack of energy.

- **Disorders** (i.e., prolonged clotting times, thrombocytopenia), renal replacement therapy can be safely performed without the utilization of any anticoagulant.

**Mnemonic: Renal dysfunction**

- CBC
  - **Creatinine** >1.2mg/dL (126µmol/L), slightly lower in females.
  - **BUN** >21mg/dL (urea >7.0 mmol/L).
  - **Creatinine clearance**: normal is 90–130mL/min.

**Mnemonic: Indications for Dialysis**

- (refractory to medical therapy)
  - **AEIOU (vowel letters)**
  - **Acidosis**
  - **Electrolyte imbalance** (**K**+)
  - **Intoxication**
  - **Overload** (fluid)
  - **Uremic encephalopathy**, **pericarditis**, **urea** >35-50 mM

**Water** is the major component of all fluid compartments within the body and represents approximately 60% of body weight.

**Sodium** is the most abundant positive ion of the extracellular fluid (ECF) compartment and is crucial in determining the extracellular and intracellular osmolality.

**Potassium** is the most abundant positive ion in the intracellular fluid and plays an important role in the membrane potential of cells.

**Mnemonic: Hypo OR Hyperbatrenia**

- **Hypo**
  - **Onatremia** = **swollen cells**
  - **Hyp**
  - **O**
  - **natrema** = **shrunken cells**

**Mnemonic: Rx of Hyperkalemia:**

- **C**
  - **BIG K DROP**
  - **Cal**
  - **cium gluconate**
  - **B**
  - **I**
  - **G**
  - **agonist**,
  - **Bicarb**,
  - **Insulin**, **Glucose**
  - **K**
  - **ayexalate**, **D**
  - **urotics**, **D**
  - **ialysis**
Management of Sepsis

- Sepsis, SIRS And MOD

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Postoperative Nutrition

Gastrointestinal Management

- Sepsis, trauma, and surgery activate complex metabolic and inflammatory responses that affect all body systems.
- The metabolic response to stress response is characterized by catabolism, hypermetabolism, hyperglycemia (diabetes of injury), and enhanced lipolysis.
- The counterregulatory hormones (cortisol, glucagon, catecholamines) along with the cytokines (e.g., IL-1, TNF) are major mediators of this response.
- Certain intraoperative anesthetic and postoperative analgesic techniques can modulate the stress response.
- During the acute phase of illness, patients unable to eat should receive parenteral or enteral nutritional support.
- Nutritional support during the acute phase of critical illness is a supplementary therapy designed to provide patients suffering from underlying metabolic disarray with sufficient nutrients to aid cellular biochemical functions and attenuate further loss of body mass.
- Only once the ravages of the stress response have abated can lost lean and fat mass be repleted.
- Overfeeding or aggressive refeeding should be avoided.

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Neurological Management

- CBF is tightly coupled to local cerebral metabolism. When cerebral activity in a particular region of the brain increases, a corresponding increase in blood flow to that region takes place. Conversely, suppression of cerebral metabolism leads to a reduction in blood flow.
- Systemic vasodilators (nitroglycerin, nitroprusside, hydralazine, calcium channel blockers) vasodilate the cerebral circulation and can, depending on mean arterial pressure, increase CBF. Vasopressors such as phenylephrine, norepinephrine, ephedrine, and dopamine do not have significant direct effects on the cerebral circulation. Their effect on CBF is dependent on

The brain has a high metabolic rate and receives approximately 15% of cardiac output. Under normal circumstances, cerebral blood flow (CBF) is approximately 50 mL/100 g/min. Gray matter receives 80% and white matter receives 20% of this blood flow.

≈ 60% of the brain's energy consumption is used to support

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Fundamentals
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Postoperative Management
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Pain Management

Postoperative Pain Relief

- The incidence of respiratory depression from opioids does not appear to be significantly different among the various routes of administration (i.e., intravenous versus intramuscular versus subcutaneous versus neuraxial). Appropriate monitoring of patients receiving opioid analgesics is essential to detect those with opioid-related side effects such as respiratory depression. Whether patients receiving neuraxial opioid analgesics require monitoring in an intensive care unit is debatable, although there is literature demonstrating the relatively safe use of single-dose and continuous-infusion neuraxial opioids on routine surgical wards under appropriate monitoring conditions.
- Judicious use of adjuvant agents, such as nonsteroidal anti-inflammatory drugs (NSAIDs), may improve postoperative analgesia and diminish analgesic-related side effects.
- When compared with systemic opioids, perioperative epidural analgesia may confer several advantages, including a facilitated return of gastrointestinal function and decrease in the incidence of pulmonary complications, coagulation-related adverse events, and possibly cardiovascular events, especially in higher-risk patients or procedures. However, the risks and benefits of epidural analgesia should be evaluated for each patient, and appropriate monitoring protocols should be used during postoperative epidural analgesia.
- Epidural analgesia is not a generic entity because different catheter locations (catheter-incision congruent versus catheter-incision

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their effect on systemic blood pressure. When mean arterial pressure is below the lower limit of autoregulation, vasopressors increase systemic pressure and thereby increase CBF. If systemic pressure is within the limits of autoregulation, vasopressor-induced increases in systemic pressure have little effect on CBF.
- All modern volatile anesthetics suppress the cerebral metabolic rate (CMR) and, with the exception of halothane, can produce burst suppression of the electroencephalogram. At that level, CMR is reduced by about 60%. Volatile anesthetics have dose-dependent effects on CBF. In doses lower than the minimal alveolar concentration (MAC), CBF is not significantly altered. Beyond doses of 1 MAC, direct cerebral vasodilation results in an increase in CBF and cerebral blood volume.
- Barbiturates, etomidate, and propofol decrease CMR and can produce burst suppression of the electroencephalogram. At that level, CMR is reduced by about 60%. Flow and metabolism coupling is preserved and therefore CBF is decreased. Opiates and benzodiazepines effect minor decreases in CBF and CMR, whereas ketamine can increase CMR (with a corresponding increase in blood flow) significantly.
- Barbiturates, propofol, ketamine, volatile anesthetics, and xenon have neuroprotective efficacy and can reduce ischemic cerebral injury. Anesthetic neuroprotection is sustained only when the severity of the ischemic insult is mild; with moderate to severe injury, long-term neuroprotection is not achieved. Administration of etomidate is associated with regional reductions in blood flow, and this can exacerbate ischemic brain injury.

CBF is autoregulated and held constant over a MAP range conservatively estimated at 65 to 150 mm Hg, given normal venous pressure. There is probably appreciable intersubject variability. CBF becomes pressure passive when MAP is either below the lower limit or above the upper limit of autoregulation.

Brain stores of oxygen and substrates are limited and the brain is exquisitely sensitive to reductions in CBF. Severe reductions in CBF (less than 10 mL/100 g/min) lead to rapid neuronal death. Ischemic injury is characterized by early excitotoxicity and delayed apoptosis.

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incongruent), durations of postoperative analgesia, and analgesic regimens (local anesthetics versus opioids) may differentially affect perioperative morbidity.

- **Postoperative pain management** should be tailored to the needs of special populations (e.g., ambulatory surgical, elderly, opioid-tolerant, pediatric, and obese patients, as well as those with obstructive sleep apnea) who may have different anatomic, physiologic, pharmacologic, or psychosocial issues.

**Pain Management and Techniques**

**Anesthesia & Pain Control in Children**

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**Postoperative Anticoagulation**

**Thrombosis**

**CABG Graft Patency**

- Vein patency rate = 75-90% at 1 year
- Technique is important
- Avoid endothelial injury
- Antiplatelet therapy
- ASA, before or within POD 1 to > 1 year
- Ticlopidine if allergic to ASA, or with coronary endarterectomy
- Persantine, likely adds nothing

**Prosthetic Valves**

**Thromboembolic complications of prosthetic valves**

**A. INR**

1. DVT - 2.0-3.0
2. Prosthetic valves - 2.5-3.5

**B. Mechanical valves**

1. Thromboembolic rate
   a) 0.5-3%/PT-yr - overall
   b) MVR = 1-3
   c) AVR = 0.5-2
2. Addition of an antiplatelet agent further reduces risk (ASA 160mgQD or dipyridamole 400mgQD)
3. Bleeding complications 0.7-6.3%/pt-yr

**C. Bioprosthetic valves**

1. Thromboembolism - 2%/pt-yr
2. More common in first 6-12 wks after operation
3. Recommendation - INR 2.0-3.0 for 3 months

**Mechanical valves**

T-E rate = 2 - 4% per patient-year

- Coumadin, INR=2.5-3.5, any position
- Bleeding complication rate = 2-3% per patient-year

- Adding anti-platelet drug ➔ T-E, ↑ bleeding
- Reserved for T-E despite therapeutic coumadin

**Bioprosthetic valves**

T-E: greatest 6-12 wks post-op then 2% per patient-year

- Coumadin, INR=2-3 x 3 months (Opt for AVR)
- With large, LA, LA clot, prior CVA - extend x 3-12 mos

**Valve thrombosis**

- Thrombolytics emerging as front-line
4. Benefit from long-term ASA

D. Complicating
1. Child-bearing
   a) Warfarin is teratogenic, crosses placenta - bad for fetus
   b) Self-administration of SC heparin to PTT 1.5-2 x control
   c) Antiplt tx alone?
2. Vascular and prosthetic grafts
   a) SVG - 75-90% 1-yr patency
   b) ASA + dipyridamole helps - ASA early post-op, dipyridamole pre-op
   c) ASA alone may be effective

CAD
   • Acute MI
   • Heparin => decreased LV thrombus/embolism
   • Especially large (anterior) MI’s, LV dysfunction
   • Coumadin - possibly beneficial
   • Unstable angina
   • Heparin + ASA
   • To download 1.9. wikislides: http://www.ezzeldinmostafa.com

Perioperative TEE

• The speed of sound in the heart is assumed to be constant at 1540 m/sec.
• The higher the transducer frequency, the better the image quality, but the more limited the depth of penetration.
• Doppler echocardiography is used to measure the velocity of blood in the cardiac chambers and across the valves.
• The modified Bernoulli equation transforms velocities into pressure gradients. Pressure gradient = 4V^2, where V = velocity in m/sec.
• Continuous wave Doppler measures high velocities but does not precisely identify their location.
• Pulsed wave Doppler permits measurement of velocities at an exact location but is limited in its ability to measure high velocities.
• Color Doppler codes for flow: blue away from the probe, red toward the probe (BART).
• TEE has been shown to be more sensitive than electrocardiography for the intraoperative detection of myocardial ischemia.

Management of Postop Complications

Managing the TEN (10) commonest calls (ABCDE2FGHI)

1. Atrial fibrillation
2. Bleeding
3. Cardiac tamponade
4. D
5. Encephalopathy
6. Focal
7. Fighting ventilator
8. Gases (poor)
9. Hypotension (profound)
10. Poor Urine

One of the category I indications for transesophageal echocardiography (TEE) is evaluation of intubated, hemodynamically unstable patients.

Guidelines expected in 2009 will probably recommend the use of TEE during all cardiac and other major surgical procedures in which severe hemodynamic instability is anticipated.

The abbreviated TEE examination will be adequate for the practice of basic TEE as defined by the 1996 SCA/ASA TEE guidelines.
# 10 Venous Thromboembolism (VTE) & Pulmonary Embolism (PE)

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**Unit Objectives**

- To review the definition, pathophysiology, clinical picture, diagnosis and management of venous thromboembolism (VTE).
- To review the definition, pathophysiology, clinical picture, diagnosis and management of the acute pulmonary embolism (PE).
- To review the definition, pathophysiology, clinical picture, diagnosis and management of the chronic thromboembolic pulmonary hypertension (CTEPH).

**Learning Objectives**

By the end of this chapter the student should Know:

- Definition
- Thrombosis - DVT: general pathologic considerations; clinical findings & Diagnosis; Management.
- Pulmonary Embolism: definition; general pathologic considerations.
- Acute Pulmonary Embolism: definition; clinical findings & diagnosis; treatment; prognosis.
- Chronic Thromboembolic Pulm. Hypertension: definition: general pathologic considerations; clinical findings & diagnosis; treatment: medical therapy; prognosis.

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**Definition**

Deep venous thrombosis (DVT) primarily affects the veins of the lower extremity or pelvis and rarely affects the venous system elsewhere. The process may involve superficial as well as deep veins, but superficial venous thrombosis does not generally propagate beyond the saphenous—femoral vein junction and therefore rarely causes PE. Venous thrombosis of the upper extremity almost always is associated with trauma, indwelling catheters, or other pathologic states and is an uncommon cause of PE but can be fatal. Pulmonary emboli that do not originate from the deep venous system of the legs and pelvis are thought to come from a diseased right atrium or ventricle or from retroperitoneal and hepatic systems.

Pathogenesis (THS)

In 1856 Rudolf Virchow made the association (Virchow’s triad) between DVT and PE and suggested that the 3 causes of DVT were related to Local vein wall trauma (injury), hypercoagulability (procoagulants in the blood), and venous stasis. (mnemonic = THS)

- **Mnemonic: Risk Factors (Virchow’s Triad), (THS)**
  - **T**: Local vein wall trauma (injury) Vein injury
  - **H**: Hypercoagulability (procoagulants in the blood) Hypercoagulable states, BCP’s, malignancy
  - **S**: venous stasis Stasis - immobility, surgery, CHF/AF, obesity 48% S/P CABG
General Pathologic Considerations

**Incidence:** General 10%, with 3 risk factors 50% (see below)

**Risk factors for DVT & sources of pulmonary emboli:**

Q. What are the major risk factors for development of DVT or PE? 13
   (=What are the predisposing causes or the stressors that may precipitate PE?
   What are the coexisting conditions? = What are the non-surgical causes of PE? =
   What are the high risk factors in PE?)

**TABLE 10-1.** The triad of VTE and the associated risk factors:

- 96% arise in the lower extremities; 4% arise in the upper extremities.
- Of symptomatic lower-extremity DVTs, 88% involve the proximal veins; the rest
  only involve the calf veins. Almost all lower-extremity DVTs arise from the calf
  veins and extend proximally.
- 90% of PEs arise from DVTs.
- 50% of symptomatic proximal lower-extremity DVTs have asymptomatic PEs.
- 70% of PEs have asymptomatic DVTs.
- 28% of symptomatic DVTs will have post-thrombotic syndrome after 5 years.

**FIGURE 10–10.** Risk stratification in DVT/PE. V/Q-based algorithm for suspected PE.
Clinical Findings & Diagnosis

A. Symptoms & Signs: Of patients with DVT do NOT have clinical symptoms; therefore, the diagnosis depends on a high degree of clinical suspicion and a variety of objective diagnostic tests. The classic presentation of PE is the abrupt onset of pleuritic chest pain, shortness of breath, and hypoxia. However, most patients with PE have no obvious symptoms at presentation. Rather, symptoms may vary from sudden catastrophic hemodynamic collapse to gradually progressive dyspnea. The diagnosis of PE should be suspected in patients with atypical symptoms, such as the following:
- Seizures
- Syncope
- Abdominal pain
- Fever
- Productive cough
- Wheezing
- Decreasing level of consciousness
- New onset of atrial fibrillation
- Hemoptysis
- Flank pain
- Delirium (in elderly patients)

Physical signs of PE include the following:
- Tachypnea (respiratory rate >16/min): 96%
- Rales: 58%
- Accentuated second heart sound: 53%
- Tachycardia (heart rate >100/min): 44%
- Fever (temperature >37.8°C): 43%
- Diaphoresis: 36%
- S₃ or S₄ gallop: 34%
- Clinical signs and symptoms suggesting thrombophlebitis: 32%
- Lower extremity edema: 24%
- Cardiac murmur: 23%
- Cyanosis: 19%

B. Testing:
A hypercoagulation workup should be performed if no obvious cause for embolic disease is apparent, including screening for conditions such as the following:
- Antithrombin III deficiency
- Protein C or protein S deficiency
- Lupus anticoagulant
- Homocystinuria
- Occult neoplasm
- Connective tissue disorders

Potentially useful laboratory tests in patients with suspected PE include the following:
- D-dimer testing
- Ischemia-modified albumin level
- White blood cell count
- Arterial blood gases:
- Serum troponin levels
- Brain natriuretic peptide
- Imaging studies:
- CTA: Multidetector-row CTA (MDCTA) is the criterion standard for diagnosing PE
- Pulmonary angiography: Criterion standard for diagnosing PE when MDCTA is not available
- CXR: Abnormal in most cases of PE, but nonspecific
- V/Q scanning: When CT scanning is not available or is contraindicated
- ECG: Most common abnormalities are tachycardia and nonspecific ST-T wave abnormalities
- MRI: Using standard or gated spin-echo techniques, pulmonary emboli demonstrate increased signal intensity within the PA
- Echocardiography: TEE may identify central PE
- Venography: Criterion standard for diagnosis.
• Duplex ultrasonography: Noninvasive diagnosis of PE by demonstrating the presence of a DVT at any site

Management

Prophylaxis

• General measures such as compression stockings probably should be prescribed more often and be used in most nonambulating patients in the hospital. Intermittent pneumatic compression (IPC) is more expensive and more cumbersome but is also effective. Both methods reduce the incidence of DVT after general surgery to ≈ 40 % of control patients.

• Low-dose SC heparin or LMWH

• Anticoagulation: Six months of warfarin anticoagulation are recommended for patients who have DVT ± PE as prophylaxis against recurrent disease.

Definitive Treatment (Chest 2004;126:401s–428s).
The goals of treatment for VTE are (i) anticoagulation to prevent further clot generation and (ii) thrombolysis if the thrombus is large enough to cause hemodynamic compromise.

Anticoagulation: Reduces further clot formation

• Unfractionated heparin (UFH):
• LMWH:
• Fondaparinux:
• Rivaroxaban:

Chronic anticoagulation: For prophylaxis against future VTE

• Vitamin K antagonists (e.g. warfarin):
• Direct thrombin inhibitors (e.g. dabigatran):
• Direct Xa inhibitors (e.g. rivaroxaban):
• Aspirin: Although this antiplatelet agent is classically used to prevent arterial thrombosis, new evidence suggests that it can also be used for recurrent VTE prevention. Daily aspirin (100mg/day used in trials) can reduce VTE recurrence by approximately 1/3. Aspirin, although not as effective as other anticoagulants, may be used if the patient is intolerant of anticoagulants.

Thrombolysis: Breaks down the thrombus

Tissue plasminogen activator (tPA):

Contraindications to anticoagulation

• Thrombectomy: If a large thrombus creates hemodynamic compromise, and there are contraindications to thrombolysis, the clot can be surgically removed or by interventional radiology.

• Inferior vena cava (IVC) filter:

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Pulmonary Embolism

Definition

- **Anatomical Definition:** Partial or complete occlusion of pulmonary vasculature from detached venous thrombi in the deep veins of the lower extremity (the more proximal femoral and iliac veins) floating (as a single embolus or fragment into smaller clots) in the moving bloodstream through the right heart into the pulmonary circulation.
- **Clinical Definition:** The occlusion of more than 50% of the pulmonary vasculature that causes hemodynamic instability, but may occur with much smaller occlusions, particularly in patients with preexisting cardiac or pulmonary disease.
- **Scintigraphic Definition:** It is the perfusion abnormality that correlates (in size and distribution) with the conventional chest film OR, the abnormal scan that shows the relation between the regional perfusion and ventilation in comparison of perfusion patterns after variable time intervals.

General Pathologic Considerations

**Pathology and Pathogenesis (2 mechanisms)**

A. **Mechanical (blockage) mechanism:**

B. **Reflex (humeral) mechanism:**

Natural History & Complications

- **Migration & Death:** The mortality of untreated PE is 18–33%, but with therapy overall mortality is under 8% (75–90% within the first few hours of the primary event). RV shock, RV standstill and RV stunning is the unequivocal cause for sudden death in PE. It occurs very early. Once the patient survives the initial RV scare (say 24-48 hours) usually do well if prompt treatment is administered.
- **Autolysis:** On average, 20% of the clot disappears by 7 days, and complete resolution may occur by 14 days. For many patients, up to 30 days are needed to dissolve small emboli and up to 60 days for massive clots.
- **Chronic thromboembolic pulmonary hypertension (CTEPH):** Only in 0.1%, PE do not lyse, and CTEPH develops. The mechanism is unknown.
- **Recurrence.**

Acute Pulmonary Embolism

Definition

**Massive PE** is truly life-threatening and is defined as a PE that causes hemodynamic instability. It is sometimes associated with occlusion of more than 50% of the pulmonary vasculature, but may occur with much smaller occlusions, particularly in patients with preexisting cardiac or pulmonary disease. The diagnosis is clinical, not anatomical. To save PE patients, first we must understand how they die. Death is due to hemodynamic collapse rather than hypoxemia. The physiology involves RV dilation with impairment of LV filling and hypoperfusion of the RV myocardium.
Clinical Findings & Diagnosis

A. Symptoms & Signs: The acute disease is conveniently stratified into minor, submassive (major), or massive embolism on the basis of hemodynamic stability, ABGs, and lung scan or angiographic assessment of the% of blocked PAs. Most pulmonary emboli are minor or submassive. These patients present with sudden, unexplained anxiety, tachypnea or dyspnea, pleuritic chest pain, cough, and occasionally streak hemoptysis.

Examination may reveal tachycardia, rales, low grade fever, and sometimes a pleural rub. Heart sounds and systemic BP are often normal; ↑ PaO2; < 30% have clinical DVT. Room air ABG’S indicate a PaO2 between 65 and 80 torr and a normal PaCO2 around 35 torr. Pulmonary angiograms show < 30% occlusion of the PA vasculature or less.

Clinical Syndromes
- Patients with massive PE
- Patients with moderate to large PE
- Patients with small to moderate PE
- Nonthrombotic PE may be easily overlooked

TABLE 10-4. Classification of PE.

B. Blood Tests Th e quantitative plasma D-dimer enzyme-linked immunosorbent assay (ELISA) level is elevated (>500 ng/ML) in more than 90% of patients with PE, reflecting plasmin’s breakdown of fibrin and indicating endogenous (though clinically ineffective) thrombolysis.

Data from the Prospective Investigation of PE Diagnosis (PIOPED) indicate that, contrary to classic teaching, arterial blood gases (ABGs) lack diagnostic utility for PE.

C. EKG Findings: Classic abnormalities include sinus tachycardia; new-onset atrial fibrillation or flutter; and an S wave in lead I, a Q wave in lead III, and an inverted T wave in lead III. Often, the QRS axis is greater than 90'. T-wave inversion in leads V1 to V5, reflects RV strain (25%).

2 Helpful Aphorisms:
- If you find yourself diagnosing inferior infarction from limb leads, and anteroseptal damage or infarction from chest leads, think of PE.
- Unexplained atrial flutter may be due to PE (check PO2 if <50mmHg, it is PE).

D. Radiologic Findings: The chest film may be normal but usually shows some combination of parenchymal infiltrate, atelectasis, and pleural effusion. Radiologic abnormalities in PE:

Manifestations of embolization without infarction:
- Oligemia (10%): Local, or Unilateral (Westermark sign), or Generalized.
- Changes in the vessels (PAs) size (25%): Hilar & Descending [Enlargement and/or distortion], Midzone (diminution, distortion, pruning, amputation, or abrupt tapering (Knukle sign)].
- Alterations in the size and configuration of the heart (10%): Cardiomegaly, dilatation of azygos vein and SVC = CHF, cor-pulmonale or pulmonay edema = bad prognosis.
- Loss of lung volume (40%): Elevation of hemidiaphragm (Stein’ sign).

N.B.: A zone of hypovascularity or a wedge-shaped pleural-based density raise the possibility of PE, but the diagnosis cannot be made with certainty on the basis of a CXR. A normal CXR in the presence of sudden hypoxia and collapse is suggestive of massive PE.

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age older than 65 y</td>
<td>1</td>
</tr>
<tr>
<td>Previous DVT or PE</td>
<td>3</td>
</tr>
<tr>
<td>Surgery (under general anesthesia) or fracture (of the lower limbs) within 1 mo</td>
<td>2</td>
</tr>
<tr>
<td>Active malignant condition (solid or hematologic, currently active or considered cured &lt; 1 y)</td>
<td>2</td>
</tr>
</tbody>
</table>

**Symptoms**
- Unilateral lower limb pain: 3
- Hemoptyisis: 2

**Clinical Signs**
- Heart rate 75–94 beats/min: 3
- Heart rate ≥95 beats/min: 5
- Pain on lower limb deep venous palpation and unilateral edema: 4

**Clinical Probability Score**
- Low: 0–3 total
- Intermediate: 4–10 total
- High: ≥11 total


### Triads in PE

1. Tachycardia, Tachypnea, Fever (Allen sign)
2. Cough, Chest Pain, Syncope
3. Chest Pain, Fever, Hemoptysis
4. Tachycardia, Hemoptysis, Shock
5. NO dyspnea, NO chest pain, NO tachycardia = NO PE
6. Hemoptysis, Cough, Diaphoresis
7. Hemoptysis, Chest Pain, Dyspnea
8. Dyspnea, Chest Pain, Apprehension
9. Increased LDH, Nomal SGOT, Increased Bilirubin, (Walker’s triad)
10. (D.D.) CHF(COPD), Pneumonia, AMI

**DDx: Dyspnea (6 Ps)**
- Pneumothorax
- Pneumonia
- PE
- Possible FB
- Pulm. Bronchial constriction
- Pump failure (CHF)

**DDx: Chest Pain**
- AMI
- DAA
- PE

### TABLE 10–3. Modified Wells Prediction Rule for Diagnosing PE: Clinical Evaluation Table for Predicting Pretest Probability of PE*

<table>
<thead>
<tr>
<th>Clinical Characteristic</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous PE or deep vein thrombosis</td>
<td>+ 1.5</td>
</tr>
<tr>
<td>Heart rate &gt;100 beats per minute</td>
<td>+ 1.5</td>
</tr>
<tr>
<td>Recent surgery or immobilization (within the last 30 d)</td>
<td>+ 1.5</td>
</tr>
<tr>
<td>Clinical signs of deep vein thrombosis</td>
<td>+ 3</td>
</tr>
<tr>
<td>Alternative diagnosis less likely than PE</td>
<td>+ 3</td>
</tr>
<tr>
<td>Hemoptysis</td>
<td>+ 1</td>
</tr>
<tr>
<td>Cancer (treated within the last 6 mo)</td>
<td>+ 1</td>
</tr>
</tbody>
</table>

**Clinical Probability of PE**

- Low: 0-1
- Intermediate: 2-6
- High: ≥6
E. Echocardiographic Findings: It shows right heart dilatation raises the possibility of major or massive PE. TTE or TEE with color flow Doppler mapping can provide reliable information about the presence or absence of major thrombi obstructing the MPA. More than 80% of patients with clinically significant PE have abnormalities of RV volume or contractility or acute TR by TTE. In some patients abnormal flow patterns can be discerned in major pulmonary arteries during TEE. It differentiates among illnesses that have radically different treatment, including AMI, pericardial tamponade, dissection of the aorta, and PE complicated by failure. Detection of RV dysfunction stratify the risk, delineate the prognosis, and treatment. Intravascular ultrasound imaging is a new diagnostic method that requires venous access but that can be done at the bedside and may prove useful.

McConnell’s sign is a distinct ECHO sign that occurs in Acute PE, where RA and RV dilates. RV shows a distinct regional wall motion abnormality in which RV free wall shows akinesia (or severe hypokinesia) with well-preserved RV apical contraction. This is visible in A4C view.

F. V/Q Scanning Findings: It may provide confirmatory evidence, but these studies are less reliable, since pneumonia, atelectasis, previous pulmonary emboli, and other conditions may cause a mismatch in ventilation and perfusion and mimic positive results. Lung scans may be:

G. MRI is a better noninvasive method BUT not suitable for hemodynamically unstable patients.

H. Pulmonary Angiographic Findings: It provides the most definitive diagnosis, but collapse of the circulation may not allow time for this procedure.

**TABLE 10-4. Classification of PE.**

<table>
<thead>
<tr>
<th>Class</th>
<th>Symptoms</th>
<th>(ABGs)</th>
<th>PA Occlusion %</th>
<th>Hemodynamics</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>None</td>
<td>Normal</td>
<td>&lt; 20</td>
<td>Normal</td>
</tr>
<tr>
<td>II</td>
<td>Anxiety, hyperventilation</td>
<td>Po2 &lt; 80, Pc02 &lt; 35</td>
<td>20 - 25</td>
<td>Tachycardia</td>
</tr>
<tr>
<td>III</td>
<td>Dyspnea, collapse</td>
<td>Po2 &lt; 60, Pc02 &lt; 30</td>
<td>30 - 50</td>
<td>CVP elevated</td>
</tr>
<tr>
<td>IV</td>
<td>Shock, dyspnea</td>
<td>Po2 &lt; 50, Pc02 &lt; 30</td>
<td>&gt; 50</td>
<td>CVP elevated, BP &lt; 100 mmHg</td>
</tr>
<tr>
<td>V</td>
<td>Dyspnea, syncope</td>
<td>Po2 &lt; 50, Pc02 &lt; 30</td>
<td>&gt; 50</td>
<td>CVP elevated, CO low</td>
</tr>
</tbody>
</table>

**Normal scan:** virtually excludes PE. **Abnormal scan:** is a sign of any of the numerous causes of decreased regional PBF. Abnormal scans most likely to be due to PE show (=Scintigraphic Definition): 4 items

- Size and distribution of perfusion defects: Large + Lobar (= high probability scan).
- Correlation with the conventional CXR: area free of radiologic abnormality.
- The relation between the regional perfusion and ventilation: without ventilation defect.
- Comparison of perfusion patterns after variable time intervals: decrease in defect on sequential examinations.
Treatment

Integrated Diagnostic Approach: Consensus guidelines from the American College of Chest Physicians are summarized as follows.

Primary versus Secondary Therapy: Primary therapy consists of clot dissolution with thrombolysis or removal of PE by embolectomy. Anticoagulation with heparin and warfarin or placement of an IVC filter constitutes secondary prevention of recurrent PE rather than primary therapy. Primary therapy should be reserved for patients at high risk of an adverse clinical outcome. When RV function remains normal, patients typically have good clinical outcomes with anticoagulation alone.

Adjunctive Therapy: Important adjunctive measures include pain relief (especially with nonsteroidal anti-inflammatory agents), supplemental oxygenation, and psychological support. Dobutamine – a B-adrenergic agonist with positive inotropic and pulmonary vasodilating effects – may successfully treat R. heart failure and cardiogenic shock. Volume loading should be undertaken cautiously because ↑ RV dilatation ⇒ ↓ LV forward output.

Management of major or submassive PE

Major or submassive PE is defined as an acute episode that causes hypoxia and mild hypotension (BP > 90 mmHg), but that does not cause cardiac arrest or sustained low CO and cardiogenic shock. Management of these patients is nearly always done after definitive diagnosis by internists. By definition there is sufficient time in these patients to definitely establish the diagnosis and to attempt pharmacologic therapy and possibly removal of embolic material by catheter suction. However, even though a satisfactory circulation is established, a few patients may become unstable and require management as outlined in the following under the diagnosis of massive PE. This subgroup of patients may be those with ECHO evidence of significant and progressive RV failure.

Monitoring & Resuscitation: The first priority after sudden collapse of any patient is to establish adequate ventilation and a survival circulation. This may require intubation and mechanical ventilation and always requires added oxygen. The initial treatment of low cardiac output includes calcium, epinephrine, and sodium bicarbonate. Later if enough blood reaches the left ventricle to sustain the circulation, dobutamine, dopamine, or norepinephrine may be added or substituted for epinephrine. In some patients phosphodiesterase inhibitors also may be useful to sustain an adequate cardiac output. Once the circulation has been stabilized, both arterial and central venous catheters are placed for access and for continuous pressure monitoring.

The ECG is monitored, a Foley catheter is placed for recording urine output, and ABGs are obtained.

Anticoagulant Therapy: If the patient’s circulation can be stabilized, IV heparin is started with an initial bolus dose of 70 U/kg followed by 18 U/kg/h if there are no contraindications to heparin. Heparin prevents propagation (as opposed to movement) of existing clots but does not dissolve thrombus. In most instances the patient’s own fibrinolytic system lysed fresh thrombi over a period of days or weeks. Without heparin up to 15% of patients with documented DVT developed PE, and 2.3% had fatal PE. When heparin was used in patients with PE, mortality was 4%, and the recurrence rate was 10%. Heparin is monitored by measurement of activated partial thromboplastin times, which are maintained between 51 and 68 seconds (twice control), every 6–8 hours. Platelet counts should be obtained at the beginning of heparin and every 2–3 days to detect the presence or appearance of HIT. Prothrombin times also are obtained at baseline to prepare for long-term anticoagulation with warfarin later.

Contraindications to conventional heparin therapy (= pre-existing hemostatic
defects):
• Recent (8-10 days) operation on C.N.S., eye, major joints, genitourinary tract. Recent CVA or other intracranial disease.
• Extensive denuded or traumatized areas- especially if inaccessible.
• Blood dyscrasias.
• Bleeding from GIT.
• Esophageal varices.
• Bleeding lesion of bronchi or urinary tract. Severe or malignant hypertension.
• Acute bacterial endocarditis.

**TABLE 10-5. Management of PE.**

<table>
<thead>
<tr>
<th>MEDICAL</th>
<th>SURGICAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anticoagulant therapy</td>
<td>Venous thrombectomy</td>
</tr>
<tr>
<td>Unfractionated heparin</td>
<td>Emergency extracorporeal life support (ECLS)</td>
</tr>
<tr>
<td>Low-molecular-weight heparin</td>
<td>Interruption of IVC</td>
</tr>
<tr>
<td>Factor Xa Inhibitors</td>
<td>Early (emergency) pulmonary thromboembolectomy</td>
</tr>
<tr>
<td>Fondaparinux</td>
<td>IVC filter insertion</td>
</tr>
<tr>
<td>Warfarin</td>
<td>Transvenous catheter embolectomy_ ECLS</td>
</tr>
<tr>
<td>Thrombolytic therapy</td>
<td>Delayed pulmonary thromboembolectomy</td>
</tr>
<tr>
<td>Alteplase</td>
<td></td>
</tr>
<tr>
<td>Reteplase</td>
<td></td>
</tr>
<tr>
<td>Urokinase</td>
<td></td>
</tr>
<tr>
<td>Streptokinase</td>
<td></td>
</tr>
<tr>
<td>Anti-platelet agents</td>
<td></td>
</tr>
</tbody>
</table>

**Thrombolytic therapy:** The addition of streptokinase, urokinase, or recombinant tissue plasminogen activator (rt-PA):
• ↑ the rate of lysis of fresh thrombi;
• ↑ the rate of lysis of fresh pulmonary clots over that of heparin alone during treatment;
• CHF → ↓ rate of death and ↓ recurrence.

It usually achieve the following
• Dissolves the anatomically obstructing PA thrombus;
• Prevents the continued release of serotonin and other neurohumeral factors might otherwise exacerbate PH; and
• Dissolves much of the source of the thrombus in the pelvic or deep leg thereby decreasing the likelihood of recurrent PE.

**Indications**
• PE with obstructive shock
• PE with RV dysfunction (?)
• No contraindication for thrombolytic therapy

**Contraindications:**
• Patients with fresh surgical wounds, recent stroke, peptic ulcer, or bleeding disorders.
• Severely anemic patients and
• Patients with potential sources of catastrophic intracranial, retroperitoneal, or GIT bleeding.

**Therapeutic Regimen**
• Continuous infusion heparin is used with lytic therapy. Infusion can be continued during drug delivery.
• Standrad thrombolytic regimen:
  o Alteplase: 100mg infused over 2hrs.
• Regimens aimed at accelerated clot lysis:
  o Alteplase:0.6mg/kg infused in 15 min.
  o Reteplase: 10 U by IV bolus and repeat in 30 min.
• Systemic drug administration is preferred to pulmonary artery instillation.

**Complications:**
• Major hemorrhage: 9-12% (3X of heparin)
• Intracranial hemorrhage: 1-2%
Pervenous Catheter Embolectomy: Mechanical removal of pulmonary thrombi is possible by a catheter device inserted under local anesthesia into the femoral (preferred) or jugular vein. The catheter, which has a small, terminal cup, is steered into the pulmonary artery using fluoroscopy for guidance. Syringe suction is applied to the cup as a thrombus is engaged, and the whole assembly is removed through the venotomy. The procedure may be repeated.

Successful extraction of clot with meaningful reduction in PAP varies between 60 and 85%. 60 to 70% of patients with major, massive, or chronic PE survive.

Management of Massive PE

Time becomes paramount if the circulation cannot be stabilized at survival levels within several minutes or if cardiac arrest occurs after a massive PE. Most deaths from acute PE occur before effective treatment is instituted or are not diagnosed in life (10% of patients with fatal PE die within the first hour, 45–80% within 2 hours, and 85% within 6 hours). To a great extent, circumstances and the timely availability of necessary equipment and personnel determine therapeutic options. Mitigating factors such as advanced age, irreversible underlying health problems, and likelihood of brain damage also enter decisions. A decision to treat medically in an effort to stabilize the circulation at a survival level may preemt life-saving surgery but also may make surgery unnecessary. For many reasons retrospective studies are of limited relevance for this decision. Sometimes surgical treatment is not available immediately; at other times deteriorating patients are referred to surgery too late after failing medical therapy. The relative infrequency of treatment opportunities in massive PE, mitigating factors, and the lack of clear criteria for prescribing medical or surgical therapy leave the management of massive PE unsettled.

Newer understanding and new technology offer a reasonable, if untried, algorithm for dealing with “probable massive PE with life-threatening hemodynamic instability” in hospitalized patients. In otherwise healthy patients in whom a thoracotomy poses little risk or morbidity, emergency thromboembolectomy with preoperative confirmation of the diagnosis in the operating room by TEE offers the surest chance of survival. Although the patient may undergo an “unnecessary operation,” the patient is highly likely to survive. When surgery is not immediately available or in patients who are older, who may have suffered irreversible organ damage, or in whom an alternate diagnosis seems more likely, emergency extracorporeal life support (ECLS) using peripheral cannulation is an attractive alternative. In prepared institutions ECLS can be instituted rapidly outside the operating room, and the morbidity is much less than thoracotomy. ECLS compensates for acute cor pulmonale and hypoxia and sustains the circulation until the clot partially lyases, pulmonary vascular resistance falls, and pulmonary blood flow becomes adequate.

Extracorporeal Life Support (ECLS)

Emergency Pulmonary Thromboembolectomy

Indications: It is indicated for suitable patients with life-threatening circulatory insufficiency (persistent and refractory hypotension despite maximal resuscitation) with massive PE unquestionably documented (by either ECHO, or lung scan or ANGIO).

Contraindications: It should not be done without a definitive diagnosis. A clinical diagnosis of PE is often wrong. Unsuitable patients.

Operative Technique: ECLS is better conducted first. A midline sternotomy incision is used. The ascending aorta and both cavae are cannulated after 3 mgm/kg of heparin. With full CPB the heart may be electrically fibrillated or arrested with cold cardioplegic solution after the aorta is clamped. Hypothermia is not necessary since only a short period of complete bypass is needed. The MPA is opened several centimeters downstream to the valve, and the incision is extended into the proximal LPA and into the proximal RPA behind the ascending aorta.

4 techniques can be used: Clot is removed by (1) Manual technique by forceps (long sponge or gall stone forceps), (2) Suction catheters or Fogarty
catheter technique. (3) Lavage and aspiration technique (3) If a sterile, pediatric bronchoscope is available, the surgeon can use this instrument to locate and remove thrombi in tertiary and quartenary pulmonary vessels. (4) Squeeze technique: The pleural spaces are entered, and each lung is gently compressed or squeezed to dislodge small clots into larger vessels and the suction cannula.

The arteriotomy is closed with a fine running suture (e.g., 5-0 prolene). After restarting the heart, the patient is weaned from bypass, decannulated, and closed.

**Vena caval filter insertion**: Greenfield recommends placement of an IVC filter (= as complementary part of the operation) before closing the chest. A variety of filters are available and are effective in preventing recurrent PE. European surgeons generally clip the intrapericardial IVC (= vena caval interruption) at the end of the operation to prevent migration of large clots into the pulmonary circulation. This clip increases venous pressure and stagnant flow in the lower half of the body and causes considerable morbidity in >60% of patients.

Although recurrent PE is always a threat, the likelihood during the immediate postoperative period is statistically small. Modern diagnosis of proximal DVT, knowledge of risk factors, and efficacy of anticoagulant therapy permit brief deferral of the decision to place a filter. Anticoagulation for 6 months is recommended for most patients with PE, but an IVC filter is recommended for patients with (1) contraindications to anticoagulation or complications of them, requiring its termination, (2) or with recurrent PE, or (3) those who require pulmonary thrombendarterectomy. The cone-shaped Greenfield filter is most widely used and is associated with a lifetime recurrent embolism rate of 5% and has 97% patency rate.

**Complications**: Patients are subject to all complications associated with open heart and major lung surgery (arrhythmias, atelectasis, wound infection, pneumonia, mediastinal bleeding, etc.) but also may develop complications specific to this operation. These include reperfusion pulmonary edema, malignant PAH, hemorrhagic lung, and neurologic disorders related to deep hypothermia and cerebral ischemia.

### Prognosis

Survival mortality rates vary widely between 40 and 90% Results are best if CPB is used to support the circulation during pulmonary arteriotomy.

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FIGURE 10–25. Algorithm for the management of PE.
Chronic Thromboembolic Pulm. Hypertens

Definition

Chronic thromboembolic pulmonary hypertension (CTEPH) is defined as PH associated with an elevated mPAP (>25 mm Hg) caused by thromboemboli in the pulmonary arterial system.

General Pathologic Considerations

- Incidence: 0.1–0.2%
- Pathogenesis and Pathology
Pulmonary microvascular changes manifesting as pulmonary hypertensive arteriopathy likely contribute to disease progression. Mechanisms for significant distal disease may involve:
1. Obstructions of “small” subsegmental elastic pulmonary arteries;
2. Classic pulmonary arteriopathy of small muscular arteries and arterioles distal to nonobstructed vessels; and
3. Pulmonary arteriopathy of small muscular arteries and arterioles distal to totally or partially obstructed vessels.
4. Distal pulmonary vasculopathy of both occluded and nonoccluded pulmonary vasculature is characterized by lesions considered typical for IPAH, including plexiform lesions.

Clinical Findings & Diagnosis

A. Symptoms & Signs: There are no signs or symptoms specific for CTEPH. The most common symptom, as with all other causes of PAH, is exertional dyspnea. Like complaints of easy fatigability, dyspnea that initially occurs only with exertion is often attributed to anxiety or being “out of shape.” Syncope, or presyncope (light-headedness during exertion) is another common symptom in PAH. Generally, it occurs in patients with more advanced disease and higher PAP.

Angina like chest pains occur in 50% of patients with more severe PAH; the pain is similar to that typical of LV ischemia and is generally felt to represent RV ischemia.

Hemoptysis can occur in all forms of PAH and probably results from abnormally dilated vessels distended by increased intravascular pressures. Peripheral edema, early satiety, and epigastric or right upper quadrant fullness or discomfort may develop as the right heart fails (cor pulmonale). Some patients present after a small acute pulmonary embolus that may produce acute symptoms of right CHF. Sometimes hemoptysis occurs. A careful history brings out symptoms of dyspnea on minimal exertion, easy fatigability, diminishing activities, and episodes of angina-like pain or light-headedness. Further examination reveals the signs of PAH.

A. Radiologic Findings: It may show either apparent vessel cutoffs of the lobar or segmental pulmonary arteries or regions of oligemia suggesting vascular occlusion. Central pulmonary arteries are enlarged, and the RV may also be enlarged without enlargement of the LV.

B. ECG Findings: RVH (RAD, dominant R-wave in V1).

C. Pulmonary Function Tests: They are necessary to exclude obstructive or restrictive intrinsic pulmonary parenchymal disease as the cause of the hypertension.

Independent risk factors for CTEPH
Anticardiolipin antibody (= 10% of patients)
Elevated levels of factor viii.
Splenectomy, Ventriculo-atrial shunt, Infected intravenous lines, and Chronic inflammatory states

The physical signs of PAH are the same no matter what the underlying pathophysiology. Initially the JVP is characterized by a large A-wave. As the right heart fails, the V-wave becomes predominant. The RV is usually palpable near the lower left sternal border, and pulmonary valve closure may be palpable in the 2nd LICS. Occasional patients with advanced disease are hypoxic and slightly cyanotic. Clubbing is an uncommon finding.
The S2 is often narrowly split and varies normally with respiration; P2 is accentuated. A sharp systolic ejection click may be heard over the pulmonary artery. As the R. heart fails, a R. atrial gallop usually is present, and TR develops. Because of the large pressure gradient across the tricuspid valve in PAH, the murmur is high pitched and may not exhibit respiratory variation. These findings are quite different from those usually observed in tricuspid valvular disease. A murmur of PR also is frequently detected.

Although some risk remains, the benefit of establishing the presence of a treatable cause of the hypertension far outweighs the small risk; selective and subselective injections with small amounts of contrast material pulmonary angiography should be performed whenever there is a
D. The V/Q Lung Scanning: It is the essential test for establishing the diagnosis of unresolved PE. A normal lung scan excludes the diagnosis of both acute or chronic, unresolved PE.

E. Acute Vasodilator Testing should be performed in all IPAH patients who might be considered potential candidates for long-term therapy with oral CCBs. IPAH patients in whom chronic CCB therapy would not be considered, such as those with overt CHF or hemodynamic instability, need not undergo acute vasodilator testing.

F. CT Findings: includes pouching, webs, or bands ± poststenotic dilatation, intimal irregularities, abrupt narrowing, or total occlusion.

G. Pulmonary ANGIO Findings: Organized thromboembolic lesions do not appear like intravascular filling defects seen with acute pulmonary emboli, and experience is essential for proper interpretation of pulmonary angiograms in patients with unresolved, chronic embolic disease. Organized thrombi appear as unusual filling defects, webs, or bands, or completely thrombosed vessels that may resemble congenital absence of the vessel. Organized material along a vascular wall of a recanalized vessel produces a scalloped or serrated lumenal edge. Because of both vessel-wall thickening and dilatation of proximal vessels, the contrast-filled lumen may appear relatively normal in diameter. Distal vessels demonstrate the rapid tapering and pruning characteristic of PAH.

### Treatment

**Surgical and Invasive Therapy**

Standard anticoagulants and thrombolytic agents have not proven successful in treating these patients; clots are organized and fibrotic and are no longer amenable to pharmacologic therapy. Once the diagnosis is established, evaluation for surgical thromboendarterectomy or PTE (delayed PTE) should proceed.

Placement of an IVC filter is advised to prevent recurrent emboli and is recommended in most patients prior to PTE. Operation relieves the vascular obstructions → ↑ PBF → ↑ the distribution of PBF → ↓ PAP → ↓ R. heart sequelae.

**Indications:** Failure of resolution OR Recurrent embolism (provided the distal PA is patent as seen by collaterals).

**Contraindications:** Serious concomitant cardiopulmonary disease OR Bad collaterals.

**Postoperative care:** Patients are anticoagulated with coumadin and low-dose aspirin (80 mgm every other day) for life. The target prothrombin time is an INR of 2.8–3.5. Patients are discharged when O₂ Sat ≥ 90 % on room air, but some patients need supplemental oxygen for a few weeks for activity. Improvement in RV pressures and volumes occurs early PAPs usually ↓ to 50–60 % of preoperative values to normal or slightly higher than normal. TR improves or disappears. PCWP does not change; however, there is considerable improvement in LV diastolic function with ↑ diastolic and SV.

**Complications:** Patients are subject to all complications associated with open heart and major lung surgery (arrhythmias, atelectasis, wound infection, pneumonia, mediastinal bleeding, etc.) but also may develop complications specific to this operation. These include reperfusion pulmonary edema, malignant PAH, hemorrhagic lung, and neurologic disorders related to deep hypothermia and cerebral ischemia.

### Prognosis

Operative mortality: 5 and 25 %. Risk factors for operative death include preoperative PVR >1100 dyne-sec-cm and postoperative ventilator dependency (> 5 days) that was partially related to prolonged CPB, presence of ascites, and need for ≥ 4 blood transfusions.
Medical Therapy

As in PAH, diuretics and oxygen are used as indicated. Lifelong anticoagulation therapy is prescribed. Pharmacologic therapies approved for use in treating IPAH are being studied in CTEPH in view of the potential for nonocclusive small vessel vasculopathy. It may be beneficial in 4 settings.

- **PTE is not possible due to significant distal disease, medical therapy may be the only possibility, with transplant considered for the most severely ill individuals.**
- **High-risk patients with extremely poor hemodynamics, IV epoprostenol may be considered as a therapeutic bridge to PTE (those with functional class IV symptoms, mPAP >50 mm Hg, CI <2.0 L/min/m², and PVR >1,000 dyn-s-cm⁻⁵).** While this approach may improve surgical success, medical therapy should not significantly delay PTE.
- **Patients with persistent PH after PTE (about 10% to 15%), and who often have significant residual distal pathology.**
- **Surgery is contraindicated due to significant comorbidity.**

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