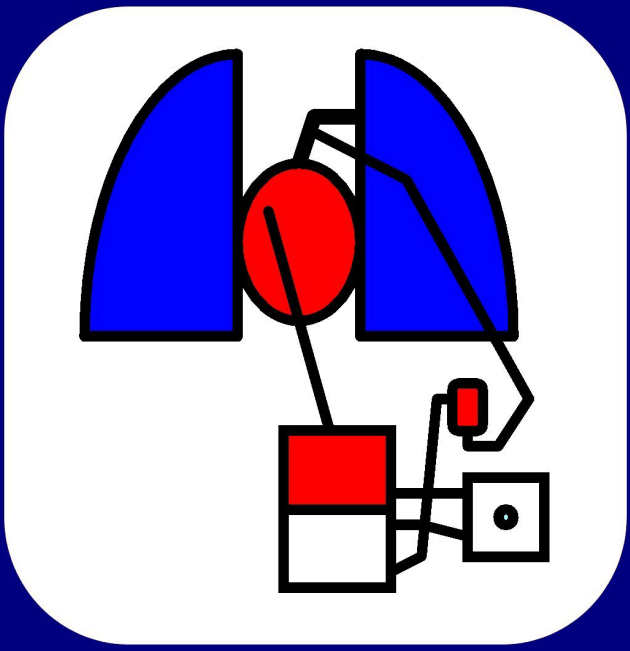
THE MANUAL OF CLINICAL PERFUSION

2nd Edition Updated



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SECOND EDITION UPDATED

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Foreword

This book release, *The Manual of Clinical Perfusion Second Edition Updated*, is an update to the 1997 edition by John Brodie and Ronald Johnson. John and Ronald began this book project in 1994 and quickly developed what has become one of the most valuable publications for perfusionists, perfusion students and other perfusion-related medical professionals. As the new owners of this publication, we will do our best to preserve their concise and straight-forward format that has made this book so popular.

As one might imagine, there have been quite a few advances in perfusion technology in the last seven years. Vacuum assisted venous drainage, modified ultrafiltration and platelet-rich plasma sequestration are just a few that come to mind. There has also been an agglomeration of new products released into the market including; oxygenators, cannulae, filters, medications and hardware devices.

We have spent considerable time and effort revising and updating the contents to bring this manual up-to-date. However, many of the planned updates will not be included until the third edition is released sometime in the fall of 2004. While this is unfortunate, we felt it was prudent to satisfy the immediate customer demand by releasing this interim edition in lieu of the third edition.

With that said, we hope that you will find our new content and revisions practical and useful. We welcome your input and constructive criticism while the third edition of this book is under development.

Bryan V. Lich

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Anatomy

The cells of the body must receive a continuous supply of oxygen and nutrients. Waste products must also be removed. It is the circulatory system that transports the blood from the lungs to all parts of the body with its life giving oxygen and transports the cellular waste products. The average adult human contains over 60,000 miles of blood vessels. The pump that propels the blood through this long network of vessels is the heart.

Heart

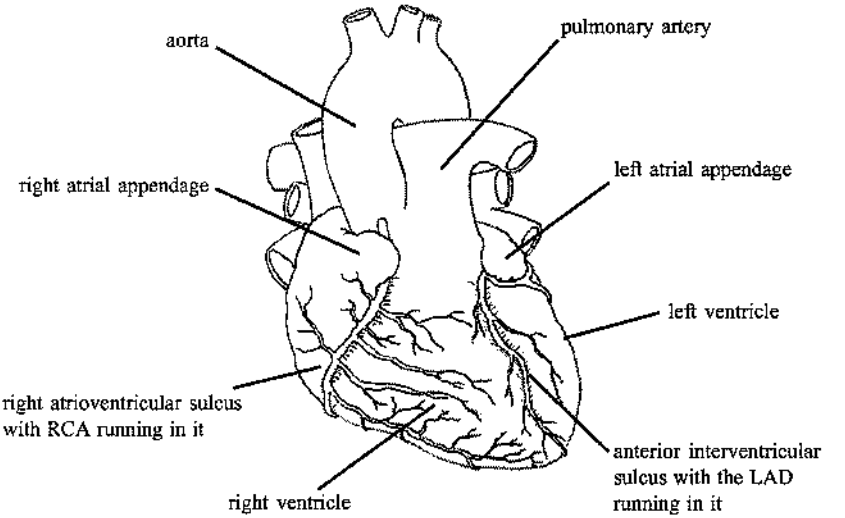
The heart is cone shaped and found between the lungs in the middle of the chest, behind the sternum, in the **middle mediastinum**. The heart lies with the blunt point (the apex) pointing downward and to the left. Approximately two thirds of the heart's volume is in the left thoracic cavity. The heart of the average male weighs about 280-340 grams. The heart of the average female weighs about 230-280 grams. The dimensions of the average heart are a length of 12 cm, a width of 8 cm and a thickness of 6 cm. There are four chambers in the heart with four one-way valves to direct blood flow through the chambers. The upper chambers are the right and left atria. They are divided by a thin muscular wall, the **atrial septum**. The septum contains a central depression of thin fibrinous material known as the **fossa ovalis**.

The lower chambers are the right and left ventricles. They are divided by the **ventricular septum**. This is a thick muscular wall with a small, thin membranous portion at the top.

Frontal View

Most heart surgery techniques require opening the **sternum** to expose the heart. This surgical opening, the **median sternotomy**, displays the right atrium and right ventricle. Seen in the drawing below are the right atrium and right ventricle separated by the right atrioventricular sulcus. This sulcus is a groove in which the right coronary artery runs. Further left is the anterior interventricular sulcus separating the right and left ventricles. The anterior descending branch of the left coronary artery runs through this sulcus. The left atrium and ventricle have only small portions visible in this view because they lie posterior to the right atrium and ventricle.

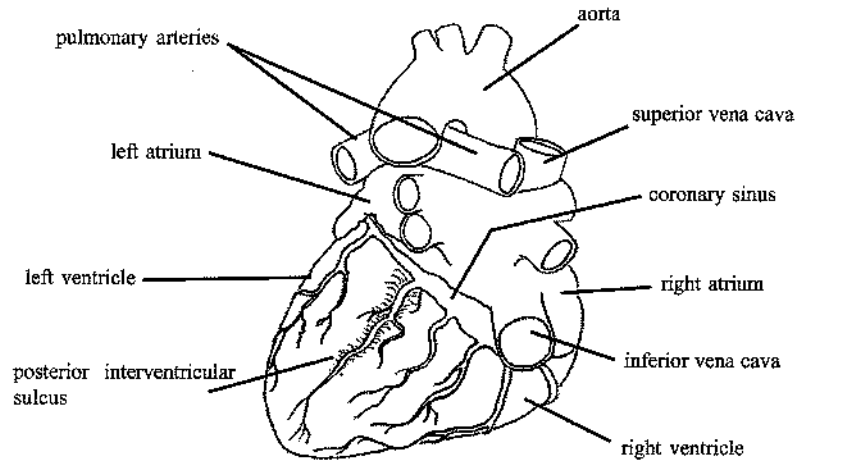
Frontal View



Posterior View

In the posterior view below, large portions of the left atrium and ventricle are visible. Also seen are the pulmonary veins, the **coronary sinus** lying in the posterior atrioventricular sulcus, and the posterior interventricular sulcus containing the descending branch of the right coronary artery.

Posterior View

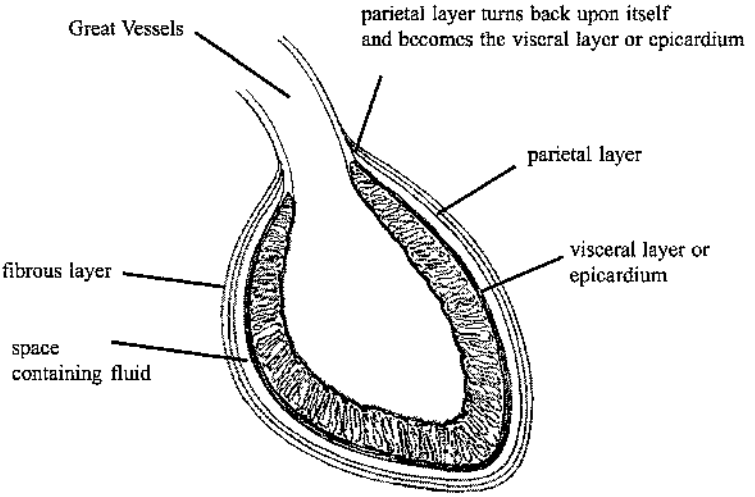


Heart Walls

The heart has a protective covering called the **pericardium**. This two layered covering allows the heart to move with a minimum of friction. The loose outer layer, the **fibrous layer**, is a tough white connective tissue. This layer becomes continuous with the outer layers of the great vessels.

The inner layer is the **serous pericardium**. This layer is further divided into the parietal and visceral layers. The **parietal layer** covers the fibrous layer. It runs superiorly under the fibrous pericardium for approximately 2 cm up the great vessels, then turns back upon itself. At this point, it now becomes the **visceral layer** or epicardium, and continues back to cover the heart. Both layers of the serous pericardium are moist, smooth and mesothelial covered. Between the parietal layer and the epicardium is a space containing approximately 50 ml of pericardial fluid.

There are three layers of the heart wall. The outer most layer is the **epicardium** (the visceral pericardium). This is a single layer of cells of mesothelial cells over a fibroelastic membrane. The cardiac muscle wall is the **myocardium**. This muscle is similar to the voluntary muscles. The myocardium muscle cells are elongated and interconnected to allow coordinated contractions of the heart. There are two bands of muscles that form a latticework arrangement to accomplish these contractions. The inner most layer is the **endocardium**. This is a single layer of endothelial cells that creates a very smooth lining of the interior chambers of the heart. This smooth lining allows blood to move through the heart with a minimum of friction. Lesions of the endocardium caused by inflammation can cause local thrombosis and thromboembolism.



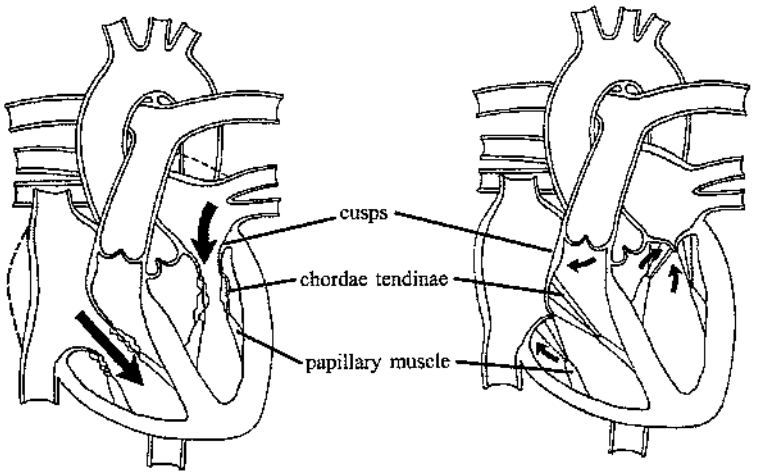
The Heart

Right Heart

The **superior vena cava** returns blood from the upper body to the right atrium. Blood from the head, arms and upper body is drained into the superior vena cava. The **inferior vena cava** drains blood from the lower body into the right atrium. The **right atrium** is the small chamber that is the site of venous cannulation for CPB. The walls of the right atrium are thin, about 2-3 millimeters.

The blood moves from the right atrium through an **atrioventricular (A-V) valve.** A-V valves have leaflets which tethered by fibrous chords. The **chordae tendineae** that are attached to the **papillary muscles** in the walls of the ventricle. These chordae tendineae and papillary muscles prevent the leaflets from going back into the atrium when the heart contracts. Thus, a complete closure occurs. This first A-V valve is the **tricuspid valve** which has 3 leaflets as its name implies.

Atrioventricular Valve



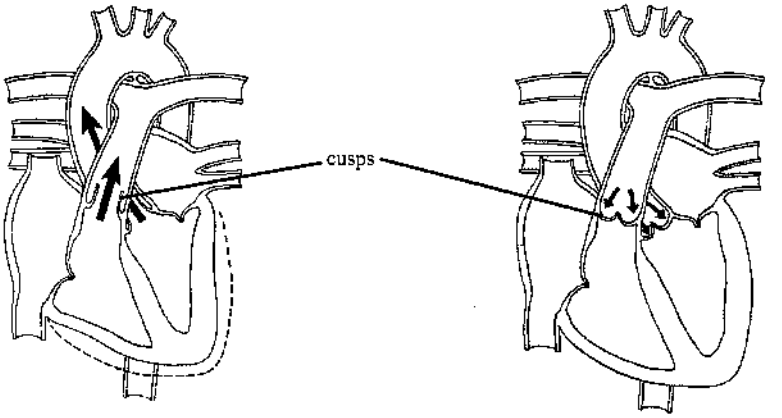
blood flow during diastole blood flow during systole

The **right ventricle** is the lower chamber of the right heart. The top part of the ventricle surface is smooth, but in the lower portion bundles of muscles arise from the walls to form interlocking webs. Contraction of the right ventricle forces blood through the pulmonary valve.

A semilunar valve, the **pulmonary valve**, allows blood to enter the pulmonary artery. This valve has three cusps that form pockets. These cusps balloon out as they are filled and come together to prevent backflow into the ventricle at the end of systole. These half-moon shaped cusps give this type of valve its name, **semilunar valve**. The **pulmonary artery** transports blood from the right ventricle to the lungs. This is the only artery that transports

venous blood. This is a short vessel, only about 2 inches long. It branches into the right and left pulmonary arteries. These enter their respective lung near the base.

Semilunar Valve



blood flow during systole

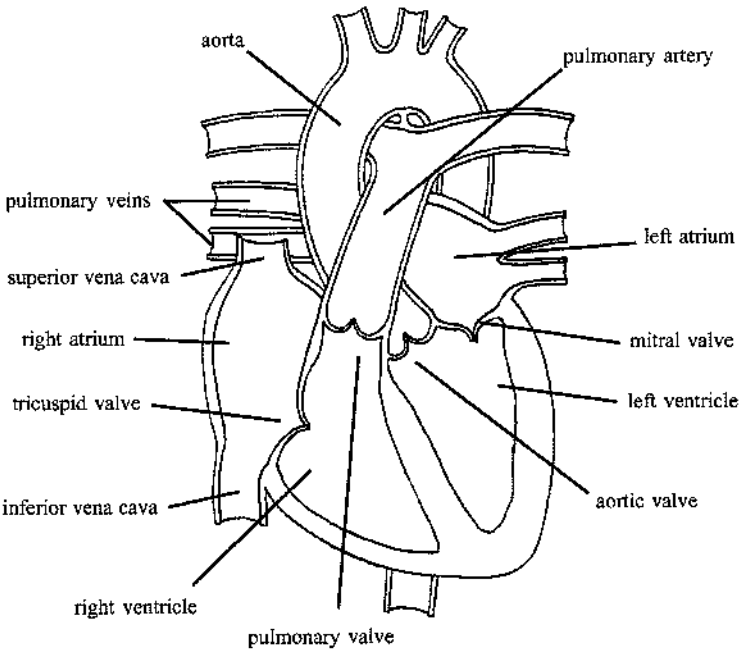
blood flow during diastole

Left Heart

Blood from the lungs that has been oxygenated enters the left atrium through four **pulmonary veins**. The **left atrium** is slightly larger than the right atrium During ventricular diastole, when the heart relaxes, blood flows through the **mitral valve,** an atrioventricular valve, into the left ventricle. This valve has two leaflets held by fibrous chordae tendineae. It is sometimes referred to as the bicuspid valve. The chordae tendineae are attached to the anterior and posterior papillary muscles. The papillary muscles and chordae tendinae assist in ventricular contraction by allowing longitudinal shortening of the left ventricle during systole. Because of this, when a patient has a mitral valve replaced, the posterior leaflet is often spared. This chordal sparing procedure has been shown to help maintain ventricular function. The **left ventricle** is the most powerful chamber with a wall much thicker than the right ventricle. This ventricle has an extra layer of muscle, the deep bulbo layer, that goes from the aortic and mitral valve rings around the left ventricle. The left ventricle contracts, and blood is forced through the aortic semilunar valve into the aorta.

The **aortic valve**, a semilunar valve, has three cusps that balloon and seal to prevent backward flow. This prevents blood in the aorta from returning to the left ventricle during diastole. The aortic and the pulmonic valves are located in an enlarged portion of their great vessels known as the aortic and pulmonary **sinus of Valsalva**.

The Heart



Coronary Arteries

The heart, itself, also needs oxygenated blood supplied to its cells. The blood that passes through the chambers of the heart cannot supply the myocardium. The coronary arteries must supply these needs. The coronary arteries come from the aorta. The openings to the coronary arteries are located in the aortic sinus of Valsalva. These openings are called **ostia** and are found behind the aortic valve cusps. When the valve is closed during diastole, blood flows into the openings to perfuse the coronary arteries. The left and right coronary arteries branch into other arteries and send branches into the myocardium to supply this need.

The **left main coronary artery** supplies the myocardium of the left ventricle. This artery divides into the anterior descending branch and the circumflex branch. The left anterior descending branch runs on the front of the heart, down to the apex. This supplies the apex, anterior surface and anterior septum. The circumflex branch lies in the groove between the left atrium and left ventricle. This branch furnishes a portion of the left ventricle away from the septum with oxygenated blood.

The **right coronary artery** is located in the groove between the right atrium and right ventricle. It runs down and around to the back of the heart. The acute marginal arteries come off the right coronary artery and branch to supply the right ventricle. In most cases the right coronary artery terminates in the posterior interventricular groove as the posterior descending artery which supplies blood to the posterior septum. It also may continue and provide branches to the posterior left ventricular wall. These are known as posterior lateral muscular branches.

Coronary Veins

The venous drainage of the heart consists of the coronary sinus system and the anterior cardiac system. The coronary sinus is a large vessel that allows blood from the superficial veins to enter the right atrium. The coronary veins mirror the coronary arteries to some extent. The great cardiac vein runs beside the LAD and drains into the coronary sinus. Also draining into the coronary sinus are the left marginal, left posterior and middle veins. Draining into the anterior cardiac system are the right marginal, small cardiac and sinus node veins. The small cardiac vein runs along the RCA. The cardiac veins do not have valves as many of the other veins of the body have. This makes it possible to give retrograde cardioplegia.

Arteries

Arteries start this highway that delivers oxygenated blood. They continue to branch into smaller branches. The **arterioles** are the smallest branches of the arteries. **Capillaries** link arterioles and venules, the smallest veins. These microscopic vessels are the smallest blood vessels.

Arteries contain 3 layers of tissue. The inside layer, the intima, is a smooth endothelial lining, connective tissue and some muscle. The middle layer, the media, is made of muscle and elastic fibers. This allows expansion of the vessel. The outside layer, the adventitia, is fibrous connective tissue that prevents the vessel from expanding too much. This ability to expand and contract helps regulate blood flow through the body.

Veins

The blood, after giving up its oxygen and nutrients, picks up carbon dioxide and waste products before returning to the heart. This return trip is by way of the **venules** that become the larger veins. The vein is thin walled, but it does not contain the high pressures that the arteries must accommodate. Most veins contain valves that prevent backflow of the blood.

Aortic Arch Vessels

The blood leaves the left ventricle and enters the aorta, the largest artery of the body. This large vessel has a diameter of about 2.5 cm. The

portions of the aorta are referred to as the **ascending aorta, aortic arch** and **descending aorta**. The descending aorta is further divided into the **thoracic aorta** and **abdominal aorta**. The aorta leaves the heart and turns downward. This turn of the aorta is known as the aortic arch. Three arteries branch from the aortic arch. The **innominate artery** is the first vessel to branch off. It divides into the right carotid and the right subclavian arteries. The next artery to branch off the aorta is the **left common carotid artery**. The third artery is the **left subclavian artery**. The **left internal mammary artery** branches from the left subclavian artery.

The **carotid arteries** supply much of the blood for the brain. The left and right common carotid arteries branch into external and internal carotid arteries. It is the internal carotid artery that supplies the brain and eyes. The middle cerebral artery branches from the internal carotid. This artery branches to supply the brain. Embolisms causing strokes often occur in these arteries. Branching from the internal carotid artery is the posterior communicating artery. Branches of this artery supply the optic cortex. The external carotid artery supplies the neck, face and exterior of the head. This artery branches into the temporal and internal maxillary arteries.

Arteries of the Upper Extremities

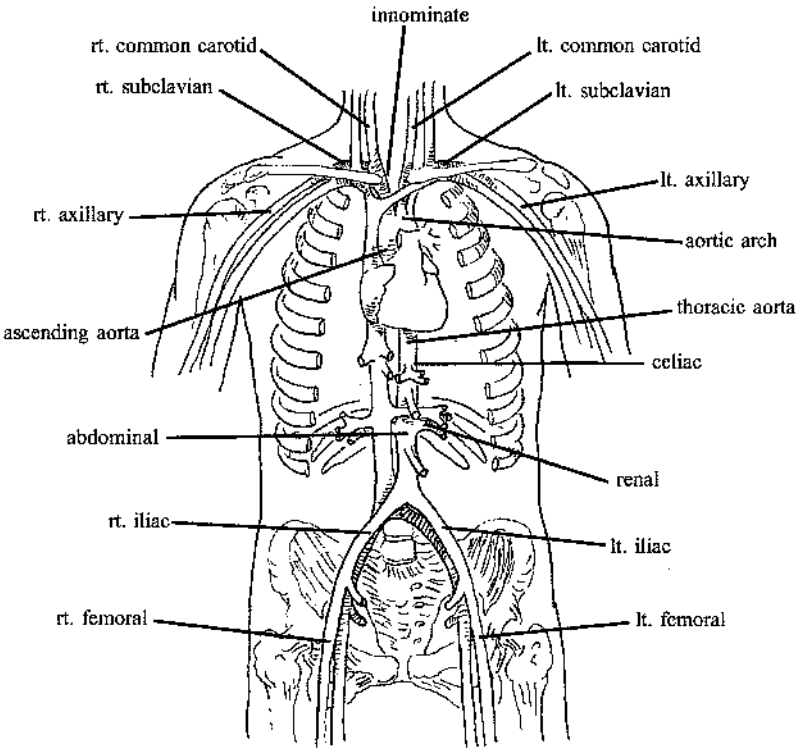
The subclavian artery supplies the upper extremity. The subclavian artery becomes the **axillary artery** at the lateral edge of the first rib. The axillary artery becomes the **brachial artery** in the upper arm at the tendon of the teres major muscle. Also branching from the axillary artery is the thoracic artery which supplies the chest wall. The radial and ulnar arteries come off the brachial artery just below the elbow. The **radial artery** runs down the arm and branches into the palmer arch to supply the thumb. The **ulnar artery** supplies the arm and palm. The palmar arch of the hand branches from the ulnar artery. This arch has two branches. These are the superficial and deep branches. From the palmar arch four digital arteries come off. Each of these arteries extends into a finger.

Branches From the Descending Aorta

The descending aorta is divided into two sections. The first section is the thoracic aorta. The abdominal aorta begins at the diaphragm. The abdominal aorta gives off branches that include the celiac, mesenteric and renal arteries. The **celiac artery** comes off the aorta near the diaphragm and branches into the gastric, hepatic and splenic arteries. The gastric artery supplies the stomach. The hepatic artery is a large artery that branches into left and right branches to supply the liver. The splenic artery supplies the spleen. The superior mesenteric artery supplies the intestines, while the inferior mesenteric artery furnishes blood to the colon and the rectum. The **renal arteries** come off the abdominal aorta to supply the kidneys. These arteries

furnish large amounts of blood due to the filtering mechanisms the kidneys perform. The abdominal aorta bifurcates to form the right and left common iliac arteries. These arteries are only about two inches in length. They bifurcate into the internal and external iliac arteries.

Arteries



Arteries of the Lower Extremities

The **internal iliac artery** has an anterior and posterior branch. It supplies the pelvis, genitals and medial aspects of the thigh. The **external iliac artery** continues along the pelvis to become the femoral artery at the inguinal ligament. The **femoral artery** supplies the leg. At the knee, this artery becomes the popliteal artery. The **popliteal** has branches that furnish blood to the knee, thigh and upper calf. The popliteal artery gives rise to the peroneal artery and the anterior and posterior **tibial artery** branches that supply the lower leg and foot. The posterior tibial artery goes into the foot and branches into the internal and external plantar arteries. The anterior tibial artery branches

from the popliteal at the knee. This artery divides into six other arteries to furnish blood to the leg and foot. The dorsalis pedis artery is a continuation of the anterior tibial artery. It begins at the ankle and divides into five branches to supply the foot and toes.

The **dorsalis pedis artery** can be felt on the dorsal aspect of the foot and is used to check distal pulses. The dorsal metatarsal artery comes off the dorsalis pedis and runs along the outer aspect of the foot to supply the foot and toes. There are two plantar arteries labeled the medial and lateral arteries. The medial artery travels along the inside of the plantar aspect of the foot to supply muscles of the foot and big toe. The lateral plantar artery travels along the base of the metatarsals and supplies the foot and tendons of the toes.

Venous System

The vessels of the venous system parallel the arterial vessels. The blood returns to the heart by veins that lead to the superior and inferior vena cavae. These lead to the right atrium where the blood is moved through the ventricle and pulmonary artery to the lungs. It then returns to the left heart and enters the arterial system.

Veins of the Head

The veins of the brain drain into the venous sinuses of the brain. The **saggital and transverse sinus veins** drain the veins of the brain. The veins outside the skull and the sinuses inside the skull are connected by diploic veins. The venous blood from the head enters the external and internal **jugular veins**. These are bilateral veins. The external jugular vein is the largest vein in the neck. This vein drains the face and scalp, then enters into the subclavian vein. The internal jugular vein is deeper and runs alongside the carotid artery on both sides of the neck. It combines with the subclavian vein to become the innominate vein. As with the other veins, the innominate veins are bilateral. The innominate veins come together to form the superior vena cava.

Vena Cava

The vena cava consists of the superior and inferior vena cava. The vena cavae return venous blood to the right atrium. The **superior vena cava** drains the head, arms and upper body. The **inferior vena cava** drains the lower body and legs.

Veins of the Upper Extremities

The upper extremities have groups of deep and superficial veins. The **subclavian vein** drains the axillary vein from the arm. The brachial and basilic veins of the arms combine and drain into the axillary vein. The **basilic vein** is formed from the median basilic and ulnar veins. The axillary vein also combines

with the cephalic vein before it becomes the subclavian. The **cephalic vein** runs in the arm up to the shoulder. The fingers are drained by digital veins that converge into the palmar arch. The metacarpal vein drains the palm of the hand. It also drains into the palmar arch. The arch joins with the **radial vein** that runs alongside the radial artery. Deep and superficial palmar veins connect the ulnar and radial veins.

Large Veins

There are two veins that receive blood from the stomach. These are the two **gastroepiploic veins**. The smaller of the two corresponds with the hepatic artery. The other corresponds with the gastric artery. They both drain into the inferior vena cava. Veins from the stomach, pancreas, intestines and the spleen drain blood rich in digested nutrients into the large portal vein. The **portal vein** branches to venules that empty into the liver. The capillaries of the liver then combine to form the **hepatic vein** that empties into the inferior vena cava. The **splenic vein** is formed from five or six branches that return blood from the spleen. The splenic vein and **mesenteric veins** combine to form the portal vein. The superior mesenteric vein drains blood from the small intestine and colon. The inferior mesenteric vein drains blood from the rectum and colon. These veins are parallel to their corresponding arteries. There are two renal veins, one from each kidney. The renal veins empty into the inferior vena cava. Due to the filtering process of the kidneys, high volumes of blood are moved through these veins.

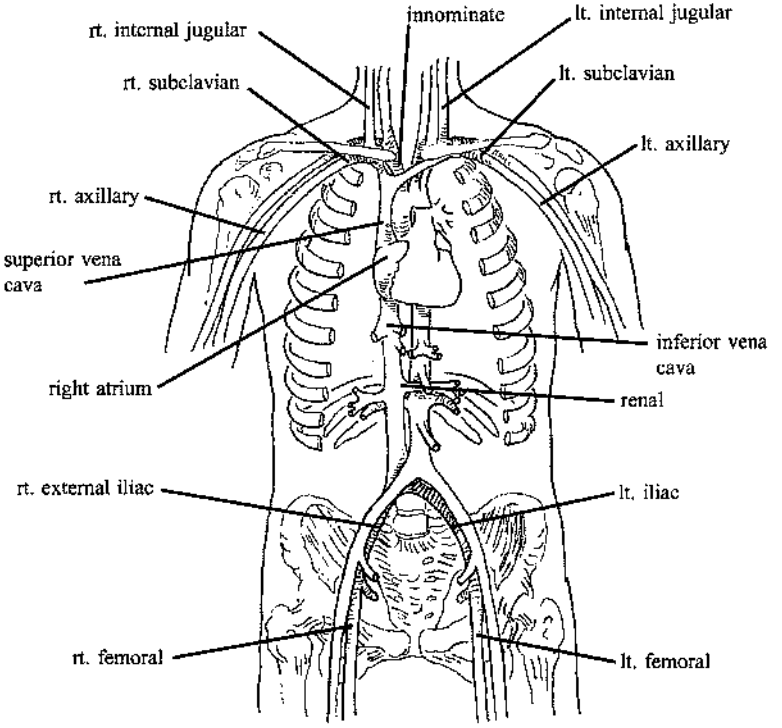
Veins of the Lower Extremities

The lower extremities are drained by a set of superficial veins and deep veins. The superficial veins, the greater and lesser saphenous veins drain the superficial lower leg, then join the deep veins. The deep veins are named after their matching arteries. The left common iliac and right common iliac veins combine to form the inferior vena cava. The **iliac veins** drain the lower extremities and pelvis. The external iliac is an extension of the femoral vein. It joins with the internal iliac vein to form the common iliac vein. The femoral vein runs up the inner aspect of the thigh with the femoral artery to join with the greater saphenous vein to become the external iliac vein. The femoral vein drains most of the blood from the legs.

The **lesser saphenous vein** begins at the outer arch on the top of the foot. It runs along the Achilles tendon to the popliteal vein. There are many branches of this vein receiving blood of the leg and foot. The **greater saphenous vein** starts at the inner arch on the top of the foot. It moves up the inner thigh to join the femoral vein. These veins have many valves that help move the blood and prevent pooling. The greater saphenous vein is harvested for use as conduit during coronary bypass artery grafting.

The **popliteal vein** receives blood from the anterior and posterior tibial veins and runs into the **femoral vein**. The **anterior tibial vein** runs between the tibia and fibula. This vein drains the knee, thigh and upper calf. It combines with the posterior tibial and popliteal veins. The **posterior tibial vein** is the vein before the lateral and medial plantar veins bifurcate. It runs alongside the tibial artery through the leg.

Veins



Diagnostics
Electrocardiograms and Cardiac Angiography

A basic understanding of the physiology of cardiac muscle contraction is needed to interpret cardiac arrhythmias. Discussion of electrocardiography should begin with a review of electrophysiology.

The **cardiac myofibril** is the basic contractile unit of the heart. Through the synchronized contractions of these myofibrils, the atrial and ventricular contractions occur that pump the blood through the heart and out to the lungs and body.

The resting cell sits ready to respond to stimulation. This is termed **excitability**. In this resting state, the cell membrane has a potential energy known as a **resting membrane potential or transmembrane potential**. This is an electrical gradient. Stimulation activates a rapid sequence of events known as the **action potential**. There is a sudden loss in the transmembrane potential called **depolarization**. It is caused by a change in the cell membrane permeability to sodium (Na^+). Upon stimulation, sodium flows into the cell in an effort to reach neutrality. Potassium (K^+) flows out of the cell, to an area of lesser concentration, and it replaces the sodium.

The cell's intracellular fluid becomes positively charged. The "sodium pump" of the cell membrane begins to transport Na^+ out of the cell and K^+ into the cell to return it to the original concentrations. This returns the cell to its resting state. This is the process of **repolarization.** In summary, depolarization is when contraction occurs and the heart is in systole, and repolarization is when the cell returns to its resting state and the heart is in diastole.

The impulse that stimulates the cardiac myofibrils comes from cells that specialize in impulse production. These cells have an unstable membrane potential, while those that contract have a stable membrane potential. These automatic cells have spontaneous depolarization. **Automaticity** is spontaneous depolarization without external stimulus. The impulse these cells produce travels from one myofibril to the next, each stimulating the next, or through tracks of cells that specialize in impulse conduction. The latter is a much faster means of impulse conduction.

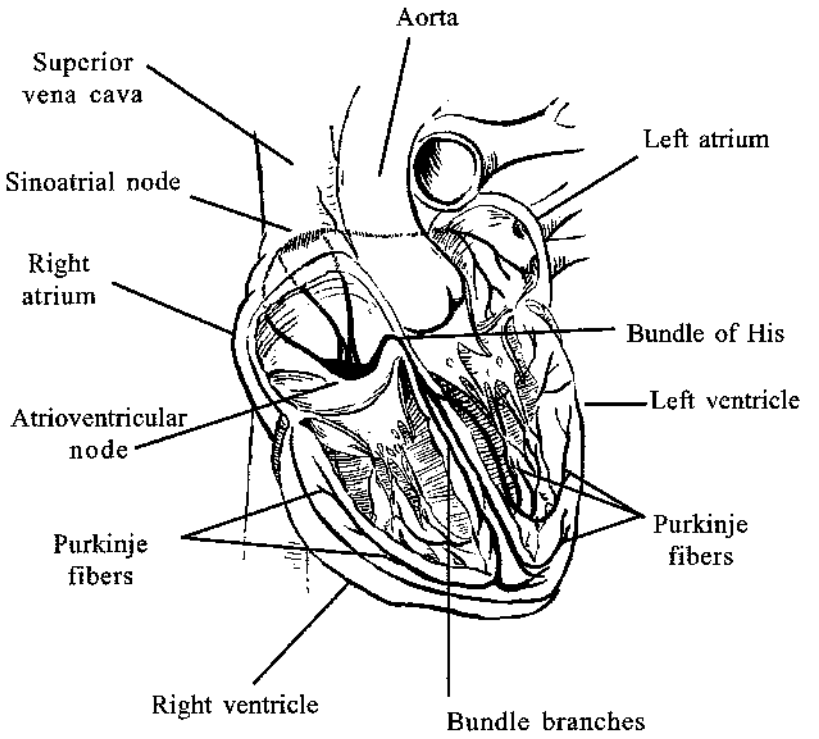
The impulse that causes the heart to contract normally originates from the **sinoatrial (S-A) node**. The S-A node is located in the right atrium at the junction with the superior vena cava. The normal adult heart rate is 60-100 beats per minute. The following sections will examine the heart as the impulse travels through the heart and the corresponding electrocardiograph (ECG) tracing.

Electrocardiograms

The release of the impulse from the S-A node causes the surrounding myofibrils to depolarize and contract. As the impulse travels across the atrium from cell to cell, the ECG has an upward deflection known as the **P wave**.

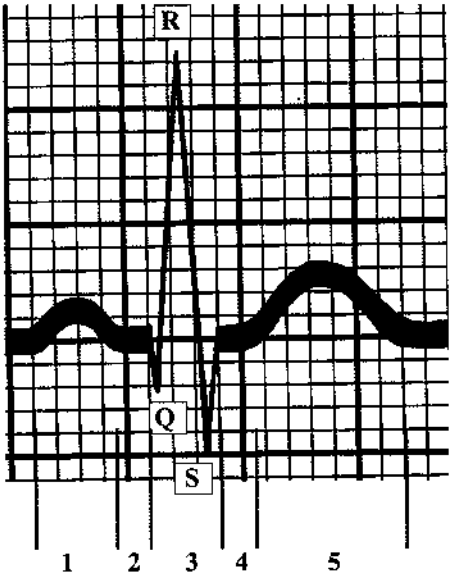
The impulse travels through the cells, reaches the **atrioventricular (A-V) node** and causes it to depolarize. The A-V node is located in the inferior intra-atrial septum. Its function is to relay the signal from the atria to the ventricles. The small electrical activity as the impulse travels through the A-V node is not sensed on the ECG, so it is represented graphically by a straight line or an **isoelectric line** after the P wave.

Electrical Conduction Paths



The impulse next travels through a track known as the **Bundle of His**. This divides in the ventricular septum into the right and left bundle branches. These terminate in the smallest branches known as **Purkinje fibers**, which go throughout the heart muscle walls. This impulse causes ventricular depolarization and contraction. This depolarization travels from the inside to the outside of the ventricles and is displayed by the **QRS complex**.

After the QRS complex, there is a brief period of inactivity called the **S-T segment**, and then repolarization of the ventricles occurs. This is seen on the ECG as the **T wave**. There is occasionally another wave after the T known as the **U wave**. It is very small and often associated with cardiomyopathy, decreased serum potassium or decreased serum calcium.

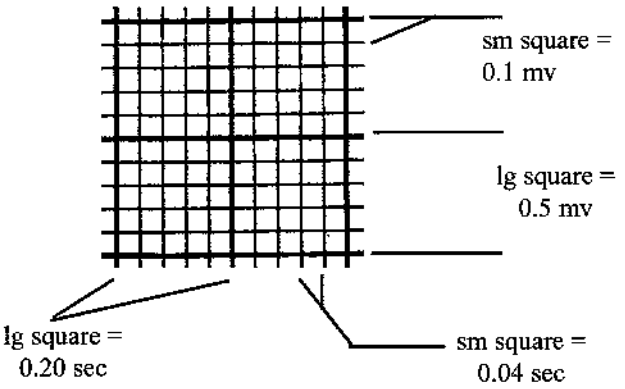


1 - P wave
2 - P-R segment (Isoelectric line)
3 - QRS complex
4 - S-T segment
5 - T wave
1 & 2 - P-R interal

The ECG is recorded on ruled paper. This allows measurements of duration and strength of particular wave forms. Standard paper has **dark thick lines** which form squares. These are subdivided into **smaller blocks (1mm),** five between each set of dark lines.

The **horizontal axis** is used when measuring **time.** Each of the small squares = 0.04 seconds. Therefore, the time between dark lines = 0.20 seconds (5 x 0.04 = 0.20).

The **strength** of a signal is measured on the **vertical axis.** Here each small square = 0.1 mv, and between dark lines = 0.5 mv.



The measurements of both time and strength can have significance on certain portions of the ECG tracing. The normal values for duration are listed below.

P wave	0.08 - 0.12 second
QRS complex	0.08 - 0.10 second
T wave	0.20 second
P - R interval	0.12 - 0.20 second

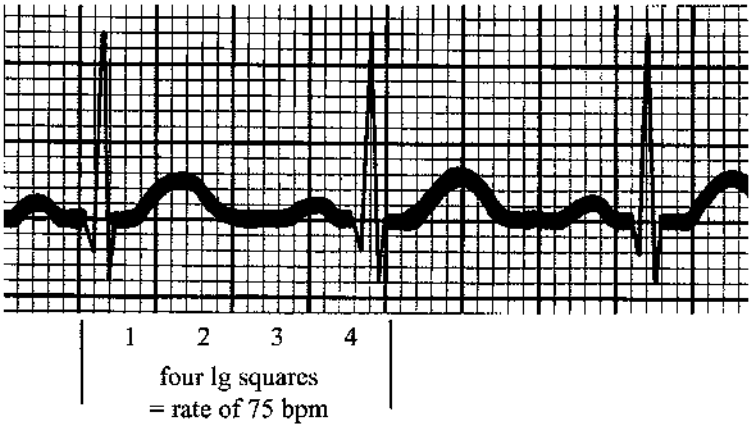
(from the beginning of the P wave to the beginning of the QRS complex)

S - T segment	0.12 - 0.15 second

(from the end of the QRS to the beginning of the T wave, ventricle contraction)

There are several methods of determining heart rates. There are rulers that can be used to measure between beats, but these rulers are easily misplaced. The two easiest methods are the grid method and the scan method.

The **grid method** determines heart rate by looking at two consecutive beats and determining the number of dark grid lines between them. If the QRS does not fall on a dark line, calipers may be useful to measure the distance. An example and the table of values are listed below.



* THIS PARTICULAR EKG HAS A RATE OF ABOUT 85

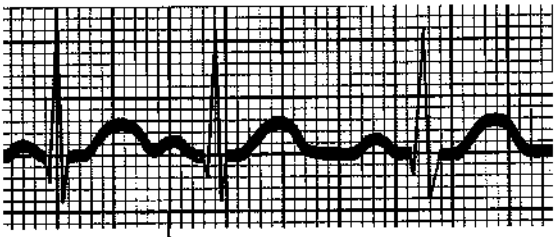
# lg squares	seconds	rate per minute
1	0.2	300
2	0.4	150
3	0.6	100
4	0.8	75
5	1.0	60
6	1.2	50
7	1.4	43
8	1.6	37
9	1.8	33
10	2.0	30

NOTE: Count off the rate as you count off the large squares. As you count the squares from one QRS complex to the next, the rate is 300, 150, 100, 75, 60 and 50.

The other quick method is the **scan method**. Most manufacturers place markers along the bottom edge of the ECG paper at three second intervals. There are 20 of these **three second marker** intervals in a minute. Therefore, simply multiply the number of QRS complexes in one three second interval by 20 to obtain the beats per minute. This method is fairly accurate, but it may be improved by multiplying the beats in two 3-second intervals by 10.

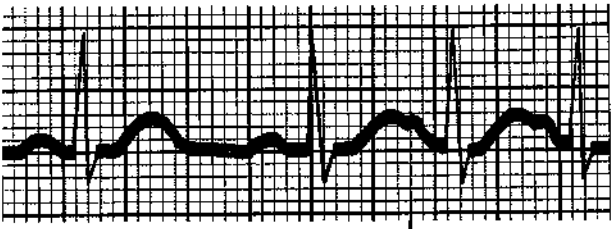
For quick reference, the following are examples of common arrhythmias. Certain stimuli may cause the atrial pacemaker to depolarize at an accelerated rate. When this rate exceeds the normal sinus rate, it preempts the normal sinus beat.

PAC - Premature Atrial Contraction - A single beat that is usually asymptomatic and benign. PACs occur early in the cardiac cycle and may originate from either atrium. The P-wave appears different than the sinus P-wave. The QRS complex and the T wave are usually normal, but if the P-wave appears very early in the cardiac cycle, there may be a prolonged PR interval or even a blocked beat.



PAC- note the P-wave falls early
and the QRS and T wave are normal

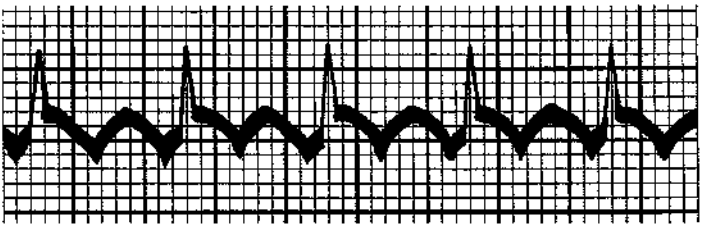
Atrial Tachycardia - May be preceded by frequent PACs, this is a state of continuous discharge from an excited region of the atrium. It usually appears suddenly and subsides just as suddenly. Known as **paroxysmal atrial tachycardia (PAT)** in the past, it is now known as **paroxysmal supraventricular tachycardia (PSVT).** The P waves are abnormal and may be superimposed on the T waves. The rate may range from 120-250 beats per minute. The rhythm is regular with a narrow QRS complex.



normal sinus rhythm

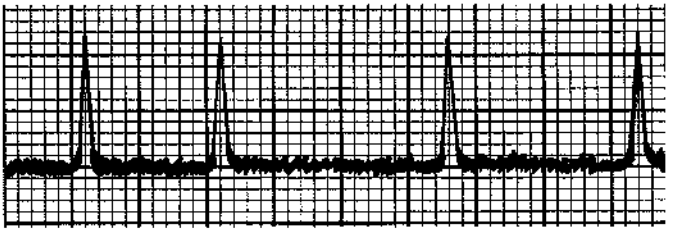
atrial tachycardia begins
note - P on the T wave

Atrial Flutter - An atrial rate between 250 and 350 beats per minute is characteristic of atrial flutter. When the rate becomes this high the P wave becomes bi-directional and takes on its characteristic **saw-toothed** appearance. The A-V conduction may remain constant or variable, but the ventricular rate is typically half of the atrial rate or less.



Atrial Flutter - A-V ratio 2:1
atrial rate - 300 ventricular rate - 150

Atrial Fibrillation - The atria can respond to very high rates of impulses. The rate may be so fast with atrial ectopic focus that the atria have not repolarized when the impulse to depolarize arrives. This leads to total disorder of the atria and the loss of their pumping action. The atria quiver and do not contract normally. Ventricular response to these impulses upon the A-V node is random. **The ventricular response is irregularly irregular. This is the hallmark of atrial fibrillation.** Atrial fibrillation has three types of **ventricular responses. Rapid** ventricular response is a ventricular rate over 100. **Moderate** ventricular response is a ventricular rate of 60 to 100. **Slow** ventricular response is a ventricular rate of less than 60. The scan method, using the three second markers, is the best way to estimate the ventricular rate due to the irregularity of the rate.



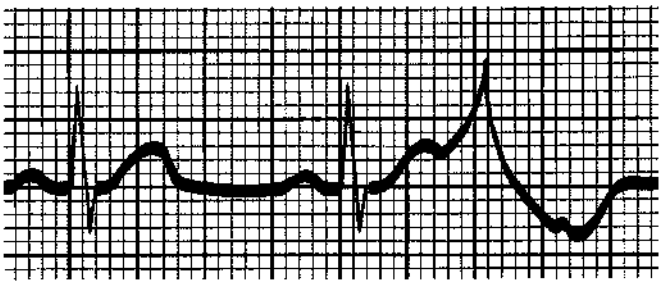
Atrial Fibrillation - moderate ventricular response
rate of approximately 90 bpm

There are various rhythm disturbances that may result from conduction of impulses through the A-V conduction system. **First degree A-V block** is the prolongation of the PR interval greater than 0.20 second (one large block on the ECG paper). This represents a delayed conduction through the atria, A-V node or His-Purkinje fibers.

The conduction velocity may become so depressed that some, but not all, impulses fail to penetrate the A-V node. This results in the absence of ventricular depolarization. This is termed **second degree A-V block** or **intermittent A-V block.** The relationship of beats that results from atrial stimulus is often referred to as a ratio. For example, if one ventricular contraction occurs for two atrial contractions, second degree A-V block with a 2:1 A-V ratio exists.

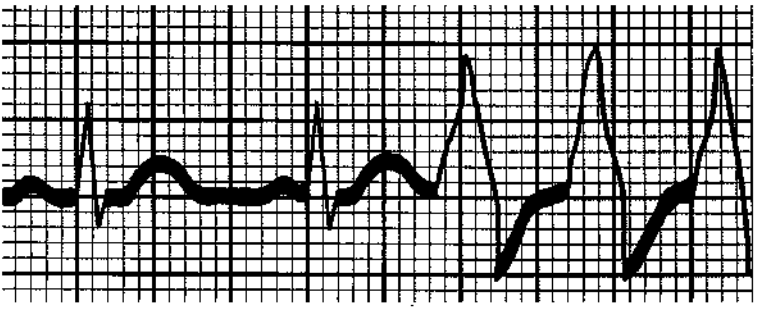
If no impulses are retransmitted through the A-V node, it is termed **third degree** or **complete A-V block.** In this case there may be an impulse from automatic cells farther along the system that stimulates the ventricles. Since the sinus impulse does not reach the ventricles, and the ventricles are receiving impulses from farther along the conduction track, the atria and ventricles may beat at different rates. With complete block and when no impulse is initiated from another source to stimulate the ventricles, cardiac arrest exists.

Premature Ventricular Contractions (PVCs) - Premature firing of a ventricular automatic site causes an early beat known as a PVC. This beat is defined as having no P wave, a wide QRS complex (>0.12sec), and it must be followed by a compensatory pause. **Bigeminy** is where every sinus beat is followed by a PVC. Two sinus beats followed by a PVC are refered to as **trigeminy.** Two consecutive PVCs are **couplets.** Three or more consecutive PVCs are considered **ventricular tachycardia.**

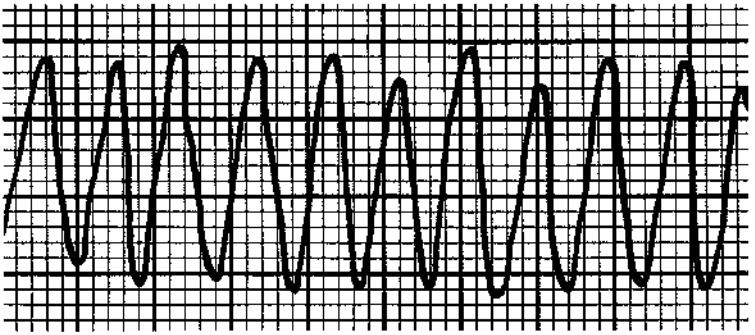


Premature ventricular contraction - PVC

Ventricular Tachycardia (VT) - A series of impulses from automatic cells within the ventricles, at a rate that is more rapid than the normal ventricular rate (20 - 50 BPM). VT may be sustained (persists more than 30 seconds) or nonsustained. Ventricular tachycardia consists of A-V dissociation, QRS complexes that are abnormally wide and of different form, and a rate exceeding 100 beats per minute. Extremely high ventricular rates cause the merging of the QRS and T waves producing a sine wave. The ventricular contraction time is short, and there is inadequate time for the chambers to fill. Cardiac output becomes inadequate and circulatory failure ensues. Without immediate intervention, this usually leads to ventricular fibrillation and cardiac death.

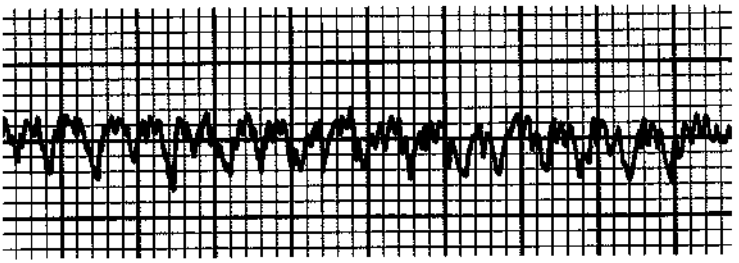


Ventricular tachycardia



Ventricular tachycardia

Ventricular Fibrillation (VF) - When the ventricular impulses become so rapid they cannot be processed, the condition is known as ventricular fibrillation. The onset usually begins with a short run of VT. Like atrial fibrillation, the muscles of the ventricles are depolarizing at different times throughout. The ventricles quiver throughout instead of contracting. Without immediate treatment thiscondition is fatal.



Ventricular fibrillation

Nervous System Control

The **autonomic nervous system** is the major factor outside of the heart itself that influences the actions of the heart. The SA node discharge is under the influence of the **sympathetic and parasympathetic (vagal) divisions** of the nervous system. Although the node will discharge spontaneously if no nervous system influence is present, it is affected by these systems. The main regulation of the heart rate is done by the balancing of the sympathetic discharges, which increase the rate, and the parasympathetic discharges, which slow the rate.

The catecholamine level will increase as the sympathetic system is activated. The catecholamine that influences this system is **norepinephrine**. This increase of the circulating catecholamines will increase both the heart rate and force of contractions. An inotropic response to sympathetic stimulation will be exhibited by the left ventricle.

Contractility of the myocardium is controlled by reflex responses from chemoreceptors and mechanoreceptors. Sympathetic and parasympathetic nerves transmit information to cause changes in the heart rate and contractility. Chemoreceptors are found in the carotid and aortic bodies. These receptors are stimulated by decreasing pO_2, increasing pCO_2 or decreasing pH. The heart

rate, arterial pressure and left ventricular inotropy are all influenced by arterial baroreceptors. Left ventricular inotropy, however, is much less influenced than the heart rate and arterial pressure.

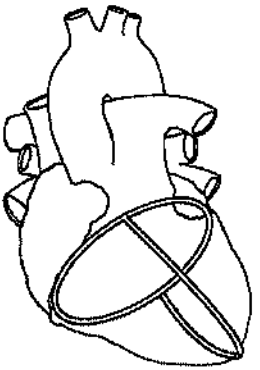
Exercise causes sympathetic stimulation and decreased parasympathetic activity. Hemorrhage causes sympathetic activity to increase the arterial pressure and heart rate. Left ventricular inotropy is, again, much less influenced.

Parasympathetic stimulation decreases heart rate, cardiac output and peripheral resistance. This inhibitory part of the nervous system works to decrease the effects of the sympathetic system. **Acetylcholine** is the chemical that is involved in stimulation of the parasympathetic system. The interaction of the sympathetic and parasympathetic systems is a complex one. Parasympathetic influence on the sympathetic effects can occur at different levels. This influence can work between nerve endings at the prejunctional level to decrease the amount of norepinephrine released from sympathetic nerve terminals. The parasympathetic influence can also work at the postjunctional level. Muscarinic receptors are activated to decrease the effect of cardiac myocytes to beta-adrenergic stimulation. The parasympathetic effect is magnified in the presence of sympathetic activity.

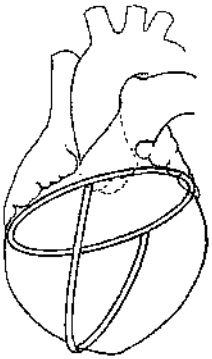
NOTES

Cardiac Angiography

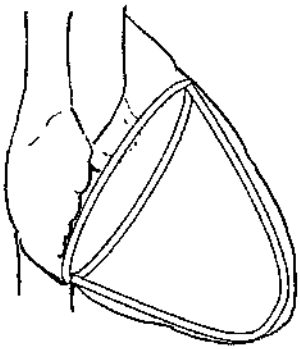
Perfusionists often find themselves with a surgeon and cardiologist looking at the coronary arteriogram of a patient being considered for surgery. As a member of the surgical team the perfusionist should be able to identify the major vessels of the heart and their branches. One method of learning and identifying the vessels uses a circle and loop to form the internal skeleton of the heart. The circle is formed by the margin of the atrioventricular groove. The loop is the margin of the ventricular septum. By understanding the relationship between the coronary arteries, the circle and the loop, and the heart, it becomes much simpler to identify the vessels. The drawings below show the circle and loop in frontal, left anterior oblique (LAO), and right anterior oblique (RAO) views.



Frontal view

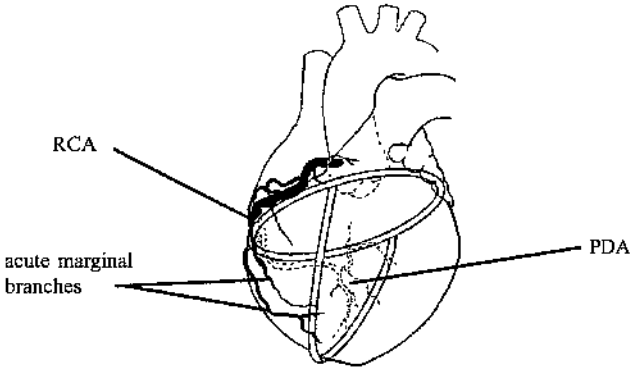


Left anterior oblique (LAO)

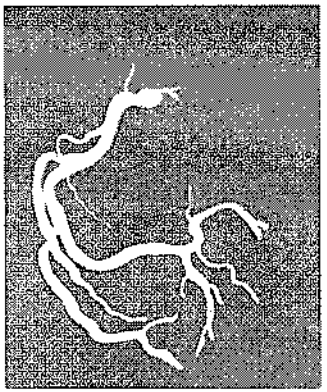


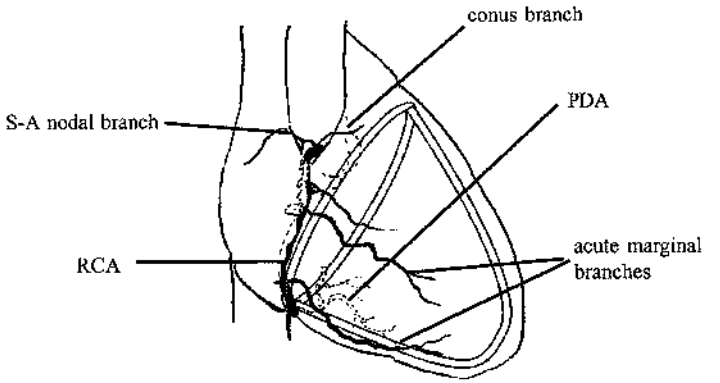
Right anterior oblique (RAO)

The **right coronary artery (RCA)** begins from the right sinus of Valsalva, travels out to the atrioventricular groove (the circle), then right, inferiorly and posteriorly to the crux. The **crux** is a point on the back of the heart where the atrioventricular groove and the atrial and ventricular septums form a cross. The RCA follows the right side of the circle and is best seen in the LAO projection. The first major branch of the RCA is the **conus artery.** It travels anteriorly and to the left. In 50% of patients the conus artery has a separate origin. The next artery to arise in 50% of the patients is the **sinoatrial nodal branch.** It travels posteriorly and medially. Next along the middle of the RCA are several branches that supply the free wall of the right ventricle. These are the **acute marginal arteries.** The RCA divides at the crux in 70% of individuals and branches into the **posterior descending artery (PDA).** The PDA travels along the inferior interventricular septum toward the apex (the bottom edge of the loop). The PDA and acute marginal branches are best seen in the RAO projection. The drawings below show the course of the RCA and its branches, while the simulated radiographs show how the arteries would appear on the catherization film.

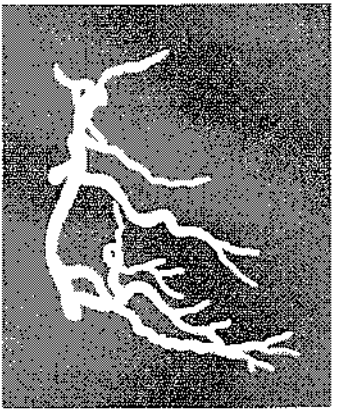


RCA in the LAO view

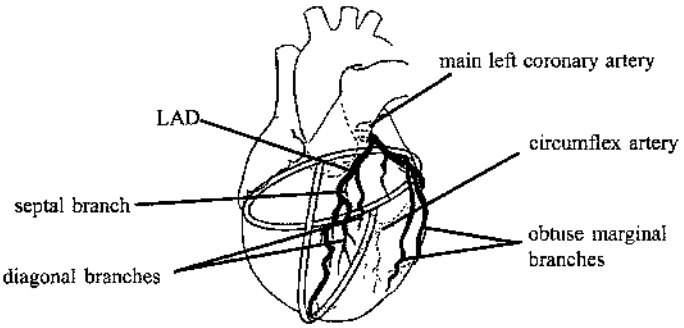




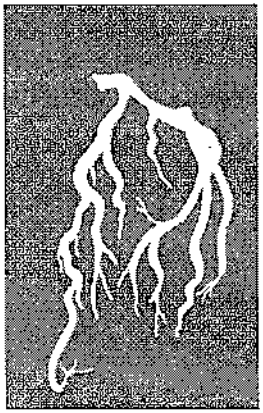
RCA in the RAO view

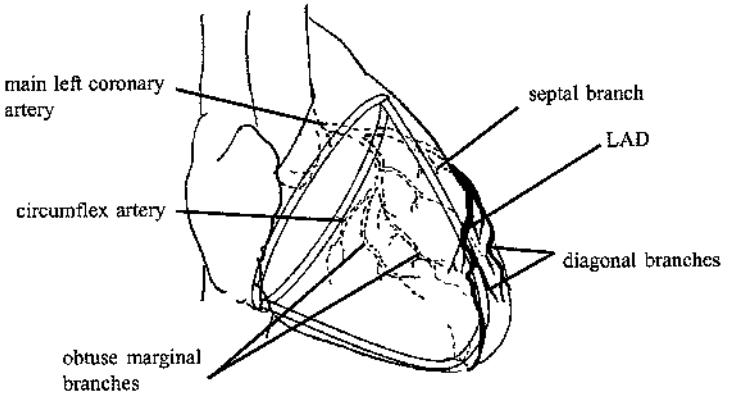


The **left coronary artery** begins from the left sinus of Valsalva and runs behind the pulmonary artery. This portion is the **left main coronary artery.** The left main then bifurcates into the **left anterior descending (LAD)** and the **circumflex (LCX)** arteries. The LAD runs anterior and along the top edge of the loop (which is the interventricular septum). The LAD gives off branches into the septum known as **septal arteries.** It also has branches to the free wall of the left ventricle which are known as the **diagonal arteries.** The circumflex artery travels inferiorly around the left side of the circle. It gives off branches to the free wall of the left ventricle known as the **obtuse marginal (OM) branches.** The LAD and the obtuse marginals are best seen from the RAO projection, and the LCX and the diagonal arteries are best seen from the LAO projection. The LAO and RAO views of the left coronary artery and the simulated radiographs are illustrated below.

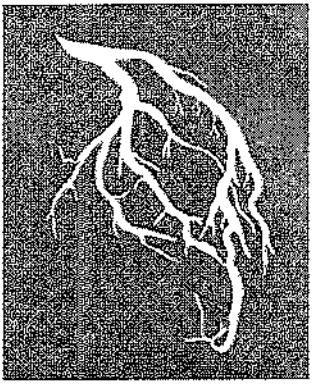


Left coronary in the LAO view

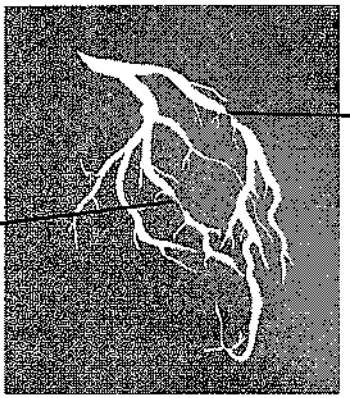




Left coronary in the RAO view



Most of the cath films belonging to open heart patients show lesions of some type. This sample radiograph shows lesions in the LAD and an obtuse marginal branch.



Blood

The perfusionist is necessarily distanced from the operative field and the patient, but still intimately involved with the patient's vital functions. The important link between the patient and the heart lung machine is the blood. Every action that the perfusionist performs to maintain the well-being of the patient is related to the blood. The science of perfusion is, in its simplest description, one of manipulation of the blood. Blood is the essential fluid that delivers oxygen, hormonal messengers and nutrients to the cells while removing carbon dioxide and other waste from the cells.

Blood is the liquid which the heart ordinarily propels through the arteries, veins and capillaries. It moves at a speed of about 1 foot per second. It completes a circulation in about 20 seconds. On bypass, the heart lung machine provides the energy and moves the blood throughout the circulatory system and the pump circuit. The blood is composed of a clear yellow fluid called the plasma, and other elements. These other elements include the erythrocytes (red blood cells), leukocytes (white blood cells), thrombocytes (platelets), proteins, electrolytes, hormones and glucose. The erythrocytes, leukocytes and platelets are described as the formed elements of the blood. Together they comprise about 50% of the total volume of the blood. There are close to 30 trillion blood cells in an adult human. Two and a half million red blood cells die every minute and are replaced. The normal adult has a blood mass of 7% to 8% of the total body weight.

Erythrocytes

The **erythrocytes** or red blood cells (RBCs) are the components responsible for the movement of oxygen and nutrients to the cells and for the removal of carbon dioxide and other wastes from the cells. RBCs are produced in the marrow of long bones. The proerythroblast, with a relatively large nucleus, is the precursor of the erythrocyte. If the bone marrow does not produce enough RBCs, **aplastic anemia** results. **Erythropoietin** is a hormone, formed mostly in the kidneys, but also in the liver, that stimulates RBC production in the bone marrow. Vitamin B12 and folic acid are also important in the production of mature RBCs. Lack of these vitamins in the diet or poor absorption can cause **pernicious anemia.** The RBC is a biconcave disk, about 7.1 microns in diameter containing hemoglobin. The size of the erythrocyte makes it too large to pass through most pre-bypass filters. This requires using a method of going around the pre-bypass filter if adding blood to the prime before the filter is removed. The **hemoglobin's** function is to transport oxygen. It is a complex protein-iron compound that carries oxygen to the cells and carbon dioxide away from the cells. Each molecule of hemoglobin contains several molecules of heme, each of which can carry one molecule of oxygen.

The number of RBCs found in adults is usually 4.5 million to 5 million per cubic millimeter. Sex, health, activity and environmental altitude all affect the number of RBCs present. At higher altitudes, the RBC count will increase dramatically. In patients with pulmonary disease, the count will also increase as the body attempts to compensate. This abnormally elevated hematocrit is termed **polycythemia**. Anemia is a low RBC count. It is caused by various factors including; blood loss, failure to produce adequate RBCs and excessive RBC destruction. The RBCs have a short life span of about 120 days and are then removed from the bloodstream and broken down. Types of RBCs include burr cells, discocytes, macrocytes, meniscocytes and spherocytes.

Patients on CPB are diluted and perfused with lower than normal hematocrits. Hematocrits of 22-30% are considered normal. This lower than normal hematocrit allows better perfusion of the capillaries. Lowering of the hematocrit occurs as a result of hemodilution from the pump prime. It is very rare that a patient's hematocrit would be considered too high while on CPB.

Hemolysis, destruction of the RBC membrane with the breakdown and release of hemoglobin, occurs normally at the end of the life span of a RBC. However, it may occur during bypass at a greatly increased rate and is a major concern of the perfusionist. Hemolysis creates **plasma free hemoglobin** and releases intracellular potassium. Aspiration of blood from around the pericardium containing plasminogen, a cleaving enzyme, is a major cause of hemolysis. The surgeon should attempt to practice sucker discipline. The force of pumping the blood through the tubing of the bypass circuit causes a certain degree of hemolysis. This makes it important to have the proper occlusion set on all roller heads. Occlusion that is too tight can crush cells while occlusion that is not tight enough may produce turbulence (**Reynolds Number** exceeds 2,000) and cause hemolysis. On roller pumps the arterial pump head occlusion should be set by holding the arterial line 30 inch above the pump head and tightening until the level is falling at the rate of about 1 cm per minute. The goal is to produce **laminar flow** in which the middle portion of the blood in the tubing moves rapidly over a slower moving boundary layer of blood.

Leukocytes

Leukocytes or white blood cells (WBCs) are the elements of the blood that protect against pathogens and foreign bodies. WBCs are able to move to an area that is invaded by microorganisms and enter the affected tissue. This movement of the WBCs is known as chemotaxis. The passage of WBCs through the walls of blood vessels without damaging the vessels is known as diapedesis. In a process called **phagocytosis** the WBCs engulf and digest bacteria, viruses, fungi and foreign bodies.

Leukocytes are larger than red blood cells and platelets. There are 5,000 to 10,000 leukocytes per cubic millimeter of blood. WBCs are classified by the presence (or lack) of small particles called granules in the cytoplasm of

the cell. The granulocytes are WBCs that contain granules. These are the neutrophils, basophils and eosinophils. The agranulocytes, or those without granules, are the lymphocytes and monocytes. The presence of infection causes the WBC count to increase. This increase is called **leukocytosis.** Increases of specific types of WBCs may indicate the presence of certain types of diseases. CBC differential counts are valuable for this reason. **Leukopenia** is an abnormal decrease in the number of WBCs to fewer than 5,000 cells per cubic millimeter. Leukopenia may affect all the WBCs or only certain types. Drug reactions, radiation poisoning or other conditions may cause this. A distribution abnormality called margination exists when WBCs move to the walls of the blood vessels. The structure of the cell itself is normal, but the count obtained on a sample from a venipuncture is decreased. In contrast, in conditions with cell survival defects, there would be an increase in immature cells.

White Blood Cells

WBC count	5,000-10,000/cubic mm
Neutrophils	60% of WBC count
Eosinophils	3% of WBC count
Basophils	1% of WBC count
Lymphocytes	30% of WBC count
Monocytes	6% of WBC count

Neutrophils are granulocytes that are produced in the bone marrow. Neutrophils comprise 60% of the WBC count. These white blood cells are able to move from the capillaries to the tissue. These are the white blood cells necessary for removing bacteria, cell debris and solid particles. The phagocytic process involves these white blood cells as they move to the site of an invasion and engulf offending organisms or foreign bodies. An increase in neutrophils above 7500 per cubic millimeter is defined as **neutrophilia.** This may be the result of infection and patients with abnormally high counts should be worked up to determine the cause of the increase.

Neutropenia is a neutrophil count below 1500 per cubic millimeter. Neutropenia is linked to leukemia, infections, rheumatoid arthritis, vitamin B12 deficiency, chemotherapy and an enlarged spleen. Patients with this condition are likely to contract infections due to the decreased ability to attack invading organisms.

Eosinophils are two-lobed white blood cells. They comprise 1 to 3% of the WBCs of the body. They increase in number with allergies and parasitic

infections. Eosinophilia exists when there are greater than 500 per cubic millimeter. In eosinophilic pneumonia, the eosinophils increase and fluid accumulates in the lungs. This condition may be caused by an allergy to fungus, plant fibers, wood dust, bird droppings or other things. Hypereosinophilia is a syndrome with increased eosinophils without an apparent cause.

Basophils are white blood cells that contain a nucleus and granules. Basophils are scant when compared to the other granulocytes and total 1% or less of the white blood cell count. Basophils are phagocytic and release heparin and histamines to keep vessels open at the site of an invading pathogen. Basophilia exists if there are more than 100 per cubic millimeter. The number of basophils increase in bone marrow diseases and decrease in severe allergic reactions. Basophilic leukemia is a cancer of the blood-forming tissues. It manifests as large numbers of immature basophils found in the blood.

Monocytes are the largest of the white blood cells. Their diameter is two to four times the diameter of a red blood cell. Monocytes become macrophages after leaving the blood and entering the tissues. As macrophages they can digest pathogens or foreign bodies due to an increase in intracellular lysosomes. Monocytosis exists when there are greater than 1000 per cubic millimeter. Elevated monocyte counts are found in conditions such as tuberculosis and subacute bacterial endocarditis. In monocytic leukemia the major cells are monocytes. This disease has signs and symptoms of weakness, fever, weight loss, a large spleen, bleeding gums, skin disorders and anemia.

Lymphocytes normally comprise 25% of the total white blood cell count. **Lymphocytosis** exists when their number is greater than 4500 per cubic millimeter. Viral illnesses such as mononucleosis cause lymphocytosis. In mononucleosis many of the lymphocytes may, also become atypical. **Lymphocytopenia** is a lymphocyte count of less than 1500 per cubic millimeter in the blood circulation. This may occur as a result of malnutrition or cancer. Lymphocytes occur in two forms and are involved in the immune system in the production of antibodies. These two forms are B cells and T cells. These two forms are developed differently, and each performs a different function.

The **B cell** searches out, identifies and binds with specific antigens or allergens. The cell membranes of B cells are self injected with antibodies that the B cells have created. B cells reproduce by simple division. All of the cells created have the same identical antibodies on their membranes. The B cell is made active and forms antibodies when it is exposed to a specific antigen or allergen. After moving to the spleen or lymph nodes it produces plasma cells and memory cells. The plasma cells produce large amounts of an antibody. Although memory cells do not secrete antibodies, they develop into antibody secreting plasma cells if they are re-exposed to a specific antigen or allergen.

T cells or killer cells secrete chemicals and assist B cells. T cells have an important role in limiting the spread of cancer. They are lymphocytes that

have gone through the thymus and become thymocytes. These cells multiply rapidly and produce new T cells specific to a particular antigen when exposed.

Platelets

Platelets or thrombocytes are disk-shaped, relatively small particles of cytoplasm. About 35 times more plentiful than the white blood cells, they do not have a nucleus or contain hemoglobin. They are biconcave disk shaped bodies with a diameter of 2-4 microns and 7-8 microns to the third power in volume. The surfaces of the platelets are negatively charged. They are produced in the bone marrow as broken fragments of the cytoplasm of the giant cells of the bone marrow called the megakaryocytes. Normally only two thirds of the platelets are in circulation. Platelets are necessary for clotting and have three properties for this work: adhesiveness, aggregation and agglutination. Plugs are formed at the site of a vascular injury in an antigen-antibody reaction. In addition the platelet membrane contains a phospholipid that aids the clotting factors. This phospholipid is know as **platelet factor 3**. In large vessels the platelets make a clump and the blood clot begins to form around this. A vasoconstrictor, thromboxane A2, is also released by platelets at the site of a vascular injury. Prostaglandins, hormones that precipitate vascular reactions, are produced in the cytoplasm of platelets. **Platelets are not given while on bypass.** It makes no sense to administer them while maintaining the anticoagulation status necessary for bypass. Platelets adhere to the bypass circuit and are sequestered in the liver, spleen and lungs during CPB. In addition, the platelets could clump and clog the oxygenator and filters in the bypass circuit.

A normal platelet count is 150,000-350,000 per cubic millimeter. Decrease platelet counts are described as **thrombocytopenia**. The term critical thrombocytopenia is used if the count is less than 50,000. Post-operatively counts less than 100,000 are usually treated with a platelet infusion. Platelet dysfunction is the most common post-op bleeding cause. Abnormally high platelet counts are termed thrombocytosis. Normothermic bypass preserves platelet function compared to hypothermic bypass. Platelet counts are decreased by 20 to 65% immediately after CPB, but usually return to normal within 24 hours.

Plasma

The liquid part of the blood is the plasma. Also found in the lymph fluid, plasma is a straw colored solution that is 91.5% water and comprises 55% of the blood. Albumin and other plasma proteins are the chief solutes of the plasma. In addition to transporting the blood cells and platelets, the plasma contains electrolytes, proteins, glucose, fats, bilirubin and gases. Plasma and the fluid of the interstitial areas are similar in their protein content. If the protein content of the plasma falls too low the patient becomes edematous as

intravascular fluid is drawn to the interstitial area by the higher **colloid osmotic pressure**. This is why albumin is added on pump if large amounts of crystalloid solutions are used. An abnormal colloid osmotic pressure prevents the proper exchanges between the intracellular, interstitial and intravascular regions. Globulins, also found in the plasma, are proteins that are larger than albumin. The globulins are of different structures and have many functions. Transferrin is a plasma globulin that binds free iron so that it can be used in the production of hemoglobin eventually. Lymphocytes produce antibodies that are globulins found in the plasma.

The plasma normally transports nutrients to the tissues and removes waste products. This is important for maintaining the proper pH and numerous other functions. On the other hand, if the colloid osmotic pressure is allowed to increase greatly by priming with plasma volume expanders such as dextran (Macrodex), intravascular overload may result.

Plasmapheresis and **plateletpheresis** are terms used to describe the removal of those portions from the blood while separating the blood cells and giving them back to the patient. The products may then be given to the patient after bypass. The purpose of this procedure is to conserve the platelets and other clotting factors while preserving the hematocrit of the patient during bypass. The cell saver machine is used for this procedure. Uncontaminated blood is drawn into the centrifuge where it is spun to separate the products. The products are collected in transfer bags where ordinarily the waste liquid is contained and the red blood cells are collected as usual. The collected RBCs can be given back immediately or held until later.

Electrolytes

The electrolytes are ions of elements and are able to carry electric currents. Various electrolyte concentrations are found in the blood plasma. These concentrations are also found in tissues and intracellular spaces. Electrolytes, in the proper concentrations, are necessary for the body to perform various functions and to utilize energy. Maintaining normal electrolyte levels is a major part of the perfusionist's job.

Potassium (K^+), the major intracellular ion, is necessary for the cardiac muscle to perform normal contractions. The intracellular space accounts for 98% of the potassium of the body. Hyperkalemia will cause irregular cardiac contractions and complete cessation if great enough. In the past, hypokalemia was more of a problem. However, with the advent of cardioplegia containing potassium, **hyperkalemia** is now more often encountered. Blood potassium would decrease during CPB without the addition of potassium in the cardioplegia. The insulin production and levels are abnormally low during hypothermic bypass, and the blood glucose rises in response. Insulin enhances the intracellular potassium and glucose uptake. After the crossclamp has been removed and very high levels of potassium occur, it may be necessary to

give insulin in order to maintain a normal sinus rhythm. Levels above 7 mEq/L are high enough that administration of 10 units of insulin is indicated, especially if the glucose is also increased. (Normal K^+ levels are 3.5-5.0 mEq/L.) After the response is determined, more insulin can be given if necessary. Most of the insulin-stimulated potassium uptake enters the liver. A word of caution: In the post-op phase, the blood glucose may drop precipitously if a great deal of insulin is given. Those managing the patient in recovery should be aware of this potential when insulin is administered. If the urine output is scant, furosemide (Lasix) 10 mg is indicated to promote urine production and excretion of the potassium before the insulin is given.

Sodium (Na^+) is the major extracellular ion and has many roles in the distribution of body fluids. The sodium pump keeps sodium out of the cells in order for potassium to stay intracellular. Sodium and potassium are linked in this constant intracellular-extracellular exchange.

Calcium (Ca^{++}) is involved with myocardial contractility, blood clotting, neurotransmission and muscle contraction. There are about 2.5 lb. of calcium in an adult. Blood calcium is regulated by the parathyroid hormone and vitamin D. Levels of **ionized calcium** may drop during CPB and because of the positive inotropic effects of calcium it may be necessary to administer calcium chloride before coming off CPB.

Magnesium (Mg^{++}) is an intracellular ion required for many chemical activities. The average adult contains about 25g of magnesium. Magnesium controls transmembrane electrolytes and energy metabolism. It also activates many enzyme systems and acts as a depressant of the central nervous system if given IV. Cardiac arrhythmias may occur if the magnesium level is decreased (hypomagnesemia). The magnesium level also affects calcium utilization due to the synergistic effect the two elements possess. The kidneys control the amount of magnesium in the body. During CPB, hypomagnesemia often occurs due to poor pre-op health or administration of albumin and citrated blood products.

Chloride (Cl^-) is the major extracellular anion and necessary for maintaining electrical balance with the cations.

Electrolytes

(Normal serum concentrations)

Sodium	136-145 mEq/L
Potassium	3.5-5.0 mEq/L
Chloride	100-106 mEq/L
Calcium	8.5-10.5 mg/dl
Phosphorus	3.0-4.5 mg/dll
Magnesium	1.5-2.5 mEq/L

Other

Glucose	70-130 mg/dl
Serum Osmolality	285-295 mOsm/L

Blood Types

The blood is classified into 4 types determined by antigens on the surfaces of the red blood cells. The types of blood using the **ABO blood** grouping system are A, B, AB and O. The cell wall of the RBC contains a protein known as the antigenic protein. The A and B antigen proteins are among these. In Type A blood, RBCs have an A antigen attached while in Type B blood, the RBCs have a B antigen attached. Type AB blood has A and B antigens attached to its RBCs. Type O blood has no antigens attached to its RBCs. The Type A blood has an anti-B antibody in its plasma, while the Type B blood has an Anti-A antibody in its plasma. Type AB blood has neither in its plasma. Type O blood has both anti-A and anti-B in its plasma. To determine the blood type, the antigens in the red blood cells are found by mixing them with serums that are known. The antibodies in the plasma are found by mixing them with cells of known A or B Type. Keep in mind that the antigens are attached to the RBC wall while the antibodies are found in the plasma. There are other antigens in the blood. Among these are the Rh and Hr types, M and N, Kell and Duffy antigens. The RBC combinations of the various antigens make each person's blood unique.

Percentage of Blood Types

Type O	47%
Type A	41%
Type B	9%
Type AB	3%

ABO Types and Reactions

		Red Cells			Serum		
		Reactions with reagents				Reactions with reagents	
TYPE	Antigens present	anti-A	anti-B	anti-A,B	Antibody present	A cells	B cells
0	Neither	—	—	—	anti-A&B	+	+
A	A	+	—	+	anti-B	—	+
B	B	—	+	+	anti-A	+	—
AB	A&B	+	+	+	Neither	—	—

The **Rh factor** is an antigen of the RBC found in 85% of the population. This factor was so named because it was found in the blood of the rhesus monkey. An individual with the Rh factor is Rh positive. An individual without the factor is Rh negative. An individual that is Rh negative and given Rh positive blood will develop antibodies. If the patient is later given Rh positive blood, RBCs are destroyed and anemia may result. Rh incompatibility may develop if an Rh negative woman becomes pregnant by an Rh positive man and an Rh positive fetus is conceived. During pregnancy, RBCs from the fetus cross the placenta and enter the mother. Most of the RBCs enter the mother at delivery, and the mother produces antibodies against the Rh factor. These antibodies are then transported to the fetus and RBC destruction occurs. The first pregnancy is usually not affected unless the mother has previously been exposed to Rh positive blood by transfusion and has developed antibodies. To prevent this occurrence, anti-Rh gamma globulin (Rhogam) should be given to an Rh negative mother within 72 hours of delivery after every pregnancy. Rhogam prevents the mother from developing permanent antibodies to the Rh factor.

Blood Products Information

COMPONENT	HCT	VOLUME	STORAGE TIME
RBCs	70	300 ml	35 days
WB	40	513 ml	35 days
WB, heparinized	40	477 ml	48 hours
PLTs	n.a.	30 to 50 ml	3 days
FFP	n.a.	220 ml	1 year

Uses of Blood Products

Packed red blood cells	Reduced hematocrit requiring transfusion
Whole blood	Massive blood loss
Platelets	Thrombocytopenia, platelet function abnormality
Fresh frozen plasma	Clotting deficiencies
Cryoprecipitate	Hemophilia, von Willebrand's disease, hypofibrinogenemia, factor XIII deficiency
Albumin	Hypoproteinemia, decreased colloid osmotic pressure, volume expander
Leukocyte-poor red blood cells	To prevent reactions from leukocyte antibodies and patients who are candidates for organ transplants

Reactions to Administration of Blood

Type	Cause	Time of Reaction
Acute hemolytic	Incompatibility	Immediate
Allergic	Antigen transfer	Less than 30 min
Delayed hemolytic	Immune response	Days or weeks later
Febrile	Reaction of antigen	30 to 90 min
Graft versus host disease	WBCs given to individual immunodeficient	Delayed
Noncardiac pulmonary edema	Donor antibodies react with recipient HLA antigen	Immediate to a few minutes

Blood Clotting

The end result of clotting is to change blood from a moving liquid to a semisolid gelatin mass. This process usually begins with injury to the tissues or exposure of the blood to a foreign surface. Within seconds, platelets start moving to the site and forming clumps. Clotting or hemostasis requires certain factors to be present in the blood and performing in their usual manner. These factors are present in the blood at all times but are not activated unless stimulated. The goal of the clotting process is to stop the loss of blood by producing fibrin which forms a mesh over the injury site. This fibrin mesh causes the cells and platelets to become emeshed, stopping the loss of blood. The following represents the fibrin forming process:

Injury or Foreign Surface Contact

Platelets aggregate and break down ——— Prothrombin, Thromboplastin, Calcium

:

:

Thrombin, Fibrinogen

:

:

Fibrin - RBCs become emeshed

There are three phases to the formation of a clot. The first phase, the **vascular phase**, is the constriction response of the vessels at the site of the injury. The second phase is the formation of plugs at the site by the platelets. In a few seconds the platelets adhere, aggregate and release factors that aid in the vasoconstriction. The third phase involves the clotting factors and their cascades of activity.

Clotting occurs through two different pathways, the intrinsic and the extrinsic pathway. The **intrinsic pathway** is initiated by contact with foreign surfaces, while the **extrinsic pathway** is initiated by tissue injury. These two pathways combine into the **common pathway** with Factor X and complete the clotting process. Calcium is involved in many steps of the clotting cascade. The cascades follow the order represented in the table below. The end result is the production of fibrin that enmeshes platelets at the site. The fibrin is reinforced by factor XIII, the fibrin stabilizing factor.

Clots are constantly being produced in response to minor bleeding. Mechanisms must be present to remove the clots after their purpose has been served. The blood would become stagnant if all clots remained after damaged tissue has healed. Plasma proteins, fibrinolysins and plasmins, are constantly dissolving old clots. Lysis occurs in both the intrinsic and extrinsic pathways. In the intrinsic path, plasminogen is split to form plasmin by factor XIIa. In the extrinsic path, plasmin is produced from the plasminogen by tissue plasminogen activators. The fibrin clots are dissolved into small pieces known as **fibrin degradation products**. The final breakdown of these results in products labeled D and E products. These products are removed to prevent inhibition of future clotting. The D and E products are removed from the blood by cells of the reticulo-endothelial system. A large number of these cells are found in the liver and are known as Kupffer's Cells. Disease of the liver will cause a reduction in all clotting factors.

Some of the clotting factors are dependent on **Vitamin K** to perform their work. Vitamin K is responsible for the insertion of negative charges on Factors II, VII, IX and X. This negative charge allows the factors to link with the positively charge calcium ion. Deficiency of this vitamin results in blood clotting disorders. The vitamin is also given to newborns to protect against hypoprothrombinemia, a bleeding disorder.

Clotting Factors

Normal Lab Values

Factor I	Fibrinogen	0.15-0.35 gm/100 ml
Factor II	Prothrombin	60-140% of control
Factor III	Thromboplastin	
Factor IV	Calcium	
Factor V	Proaccelerin	60-140% of control
Factor VI	None	
Factor VII	Proconvertin	70-130% of control
Factor VIII	Antihemophilic	50-200% of control
Factor IX	Plasma Thromboplastin	60-140% of control
Factor X	Stuart Factor	70-130% of control
Factor XI	Plasma Thromboplastin Anteced	60-140% of control
Factor XII	Hageman Factor	60-140% of control
Factor XIII	Fibrin Stabilizing Factor	

Clotting Pathways

INTRINSIC

Factor XII
Factor XI
Factor IX
Factor VIII

EXTRINSIC

Factor III
Factor VII

Factor IV-Calcium
Found in all pathways at many stages.

COMMON
Factor X
Factor V
Factor II
Factor I
Factor XIII

Heparin

Clotting during CPB is obviously undesirable. The patient is kept in an anticougulated state to prevent clotting. Heparin is the anticoagulant most commonly used during CPB. The primary **anticoagulant** effects are on plasma coagulation. Specifically, heparin works by potentiating **antithrombin III**, which stops the fibrinogen from being changed to fibrin and prothrombin from being changed to thrombin. Without heparin, anticoagulation thrombin is a catalyst to change fibrinogen into insoluble fibrin.

Heparin also works in another way to inactivate thrombin by binding to cofactor II a glycoprotein that inactivates thrombin. In addition, heparin affects platelets in various ways. The half life of heparin is one to two hours. During hypothermia, the half life is increased, while during the rewarming phase, it is decreased. Care should be taken during the rewarming phase to check the ACT often. Elimination is by metabolism via the kidneys and the reticuloendothelial system. Thus, impaired renal function will also prolong the activity of heparin. Heparin is obtained from beef lung, beef liver, beef intestinal mucosa and pork intestinal mucosa. The heparin used in medicine is combined with sodium because of the acidic nature of heparin.

The patient is usually heparinized before cannulation by either the surgeon or the anesthesiologist. The surgeon gives the heparin in the right atrium with a syringe and needle, while the anesthesiologist usually gives the heparin via a central venous line. Giving the heparin by these methods ensures that the heparin reaches the circulating blood. The dose necessary to achieve an activating clotting time of 500 seconds is 300 to 400 units per kg. This heparin bolusing has a side effect of decreasing arterial blood pressure and systemic vascular resistance by 10 to 20%. Serious reactions are rare after this bolus, but anaphylaxis, pulmonary edema and disseminated intravascular coagulation have been reported. In rare cases, a patient may have an **antithrombin III deficiency** and not respond with the necessary increased ACT for bypass. Repeat dosing of 400 units per kg may be necessary. If this does not provide the necessary ACT, fresh frozen plasma (2 units) or the specific component should be given. The fresh frozen plasma contains antithrombin III in amounts that should allow the heparin to work its anticoagulant effect. The ACT is increased, reflecting the IV administration of heparin after about 1 minute. On bypass, during long cases, repeat dosing is sometimes necessary to maintain adequate ACTs. Heparin analyzers calculate these repeat dosage requirements. Many perfusionists simply give 5,000 units and check another ACT for results.

Protamine

Protamine functions as a heparin antagonist by ionically binding with it, which makes it ineffective. It has a weak anticoagulant effect when given by itself, but in the presence of heparin, it forms an inert substance. Protamine is

a polycationic protein obtained from the sperm of salmon. Protamine is usually given IV just before the arterial cannula is removed to reverse the anticoagulant effect of heparin.

There are various methods for determining the amount of protamine given to reverse the heparin. These are heparin assays, heparin response curves to determine active heparin or simply giving a fixed amount for each unit of heparin administered. This fixed amount is from 1.0 to 1.3 mg per 100 units of heparin.

Protamine must be given slowly to minimize reactions. Some authorities recommend not infusing faster than 5 mg/min. The incidence of **reactions** is decreased by slow infusion. Possible reactions are manifested by hypotension, bradycardia and eventual circulatory collapse. The probability of a reaction is increased if the patient has had protamine in the past (redo), is an insulin dependent diabetic or has seafood allergies. Some researchers suggest that men with vasectomies may have an increased incidence of reactions due to the protamine being obtained from salmon sperm. A patient with increased risk factors is often given benadryl and steroids on bypass as a prophylactic measure. **Signs of reactions** are dramatically increased pulmonary arterial and CVP pressure with decreased systemic arterial pressure. Serious reactions require supportive measures including vasoactive drugs, inotropic drugs and pulmonary vasodilators such as isoproterenol and nitroglycerin. Complete collapse may make it necessary to reinstitute CPB. **Full heparinization is required if going back on bypass because of the protamine administration.** The half-life of protamine is less than that of heparin. This may necessitate another small dose (50 mg) after the initial dose. ACTs done after the initial protamine dose are useful for determining whether more protamine is required.

Normal Values

Red Blood Cells

	Males	Females
Red Cell Count	4.5-6.1	4.3-5.3 million/cu/mm
Hemoglobin	14-18	14-18 gm/dl
MCV	82-93	82-93 cu microns
MCH	27-31	27-31 pg
MCHC	32-36	32-36 %
Sed. rate	0-13/hr	0-20/hr

White Blood Cells

WBC count	5,000-10,000/cubic mm
Neutrophils	60% of WBC count
Eosinophils	3% of WBC count
Basophils	1% of WBC count
Lymphocytes	30% of WBC count
Monocytes	6% of WBC count

Platelet Count

150,000-350,000/cubic mm

Coagulation Tests

ACT	110-120 sec
PT	10-12 sec
PTT	24-37 sec
TT	Within 5 sec of control
Bleeding Time	3-9 min
Fibrinogen Level	200-400 mg%

Blood Gases

The interpretation of the information contained in blood gas results is a major responsibility of the perfusionist. Maintenance of homeostasis, the body's ability to maintain its normal physiology, is greatly influenced by the gas exchange that occurs at the cellular level. The first organ affected by inadequate oxygenation is the brain. Other organs are affected soon thereafter. The body's quest for homeostasis is reflected in blood gas chemistry. The exchange of gas between the blood and cells of the tissues is known as internal respiration. External respiration is the exchange of carbon dioxide and oxygen that takes place in the lungs or in the heart lung machine.

Blood gases are routinely performed on CPB to ensure proper oxygenation, carbon dioxide level and pH status. In-line monitoring devices are used by many perfusionists. These devices identify almost instant changes in the patient's status. Some perfusionists use in-line oxygen saturation indicators. These indicators are often placed in the venous side to determine the adequacy of perfusion. (Normal venous O_2 saturations are about 75% with a venous pO_2 of about 40 mmHg.) These indicators are simple and inexpensive, but they do not furnish the information of other more sophisticated devices. Some type of in-line monitoring is now the standard of care. A monitoring device should be used even if blood gases are routinely sent to the lab.

Hemoglobin-Oxygen Effect

Hemoglobin is the major portion of the red blood cell. It is the hemoglobin that is responsible for transporting oxygen to the organs and tissues of the body. About 97% of the blood's oxygen is transported by the hemoglobin. Hemoglobin is protein complex that is formed from amino acids. Hemoglobin has a large transportation task to carry out. The average adult has a **basilar oxygen consumption** rate of about 250 ml/min. The heart has a basilar oxygen consumption rate of 1.3 ml/100 gm of tissue per minute. The brain has a basilar oxygen consumption rate of 3.5 ml/100 gm of tissue per minute. Lack of blood flow to the brain will cause loss of consciousness in about 5-10 seconds. Temperature affects the oxygen consumption. For every 7^0 C that the temperature is lowered, the metabolic rate is decreased by 50%, thus lowering the oxygen consumption. Temperature also affects the solubility of both oxygen and carbon dioxide. Colder temperatures cause increased solubility.

Hemoglobin Oxygen Dissociation Curve

The hemoglobin oxygen dissociation curve represents the relationship of the hemoglobin and the oxygen in the blood. The bell shaped curve displays the effects of increased oxygen on various percentages of saturated hemoglobin.

Oxygen content, capacity and consumption are dependent to a large measure on the hemoglobin. When the hemoglobin is fully saturated, increases in pO_2 result in only small increases in oxygen content. The oxygen will only be transported in the dissolved state. In other words, if a patient has a low hemoglobin content, then administering large amounts of oxygen will have little effect if the oxygen cannot be transported. Shifts of the dissociation curve reflect the affinity of hemoglobin for oxygen. A **shift to the right** is a decreased oxygen affinity. The hemoglobin will then release more oxygen to the tissues. This is found in cases of hyperthermia, decreased hemoglobin, increased 2,3 DPG and conditions where the pCO_2 is increased and the pH is decreased. A **shift to the left** reflects an increased oxygen affinity in which the hemoglobin holds the oxygen and releases less to the tissues. The oxyhemoglobin saturation is, therefore, increased. The average adult has a hemoglobin value of 12-16 gm/100ml of blood. On CPB, the hemoglobin is much lower due to hemodilution.

P_{50}

P_{50} is a reflection of a certain enzyme effect on the hemoglobin affinity for oxygen. The enzyme measured is **2, 3 DPG**. The term can be described as the oxygen tension when 50% of the hemoglobin is saturated at 37°C, pCO_2 40 mmHg and pH of 7.40. Normal adult P_{50} (hemoglobin saturated at 50%) is 27 mmHg under these conditions. Decreased P_{50} indicates increased affinity of hemoglobin for oxygen. Increased P_{50} indicates decreased affinity of hemoglobin for oxygen.

Oxygen Calculations

A fully saturated gram of hemoglobin can carry 1.34 ml of oxygen. Formulas that determine the oxygen status of the blood must consider the hemoglobin concentration. Formulas are based on volume per 100 ml of blood.

The formula for **oxygen carrying capacity**:

$$O_2 \text{ Capacity} = 1.34 \times \text{Hgb} + .003 \times pO_2$$

This formula assumes 100% saturation. **Dissolved oxygen** in plasma is found by $pO_2 \times .003$. The formula for oxygen content represents oxygen combined with the hemoglobin plus dissolved oxygen. It differs from oxygen capacity in that it uses the actual O_2 saturation.

The formula for **oxygen content**:

$$\text{content} = 1.34 \times \text{Hgb} \times \%\text{ saturation (in decimal)} + .003 \times pO_2$$

The formula for **oxygen saturation**:

O_2 saturation = O_2 content / O_2 capacity

On bypass, the formula for **oxygen consumption**:

O_2 consumption = aO_2 content - vO_2 content x flow (L/min) x 10

On bypass, the formula for **oxygen transfer**:

O_2 transfer = [Art - Ven sat in decimal form x 1.34 x Hgb x flow(ml/min)] / 100

Carbon Dioxide

The arterial pCO_2 affects the pH of the blood. Control of the pCO_2 is necessary for proper blood gas management on CPB. Clinically, if the starting point is a pCO_2 of 40 mmHg a mathematical relationship can be shown. If the arterial pCO_2 increases by 20 mmHg, the arterial pH will decrease by 0.10. If the arterial pCO_2 decreases by 10 mmHg, the pH will increase by 0.10. Carbon dioxide comprises .03% of the atmosphere. The metabolic processes of the body produce CO_2 when O_2 is utilized. The **respiratory quotient** is the ratio of CO_2 production to O_2 consumption. The average ratio is 4:5. The cerebral blood flow increases in response to increases in CO_2 or decreased O_2

Carbon dioxide is also transported to a degree by hemoglobin. It combines with the hemoglobin, while some combines with water to form carbonic acid. The carbonic acid breaks down to form hydrogen ions and bicarbonate ions. Some carbon dioxide is also transported dissolved in the plasma. Carbon dioxide has other consequences on the oxygen in the blood. There is an interaction between the two gases. The **Bohr effect** is that the addition of carbon dioxide to the blood causes oxygen to be released from hemoglobin faster. The **Haldane effect** is that oxygen added to the blood causes carbon dioxide to be released from the hemoglobin faster.

pH

The pH indicates the blood's acidity or basicity. Although 7 represents neutrality, normal pH for human blood is 7.35 to 7.45. Thus, the body is maintained in a slightly alkaline state. In medicine, however, values below 7.35 are considered acidotic and values above 7.45 are considered alkalotic (base). Technically, the pH is an expression of a solution's relative amounts of hydrogen and hydroxide ions present. The goal of the perfusionist is to keep the patient in this normal pH range. The cells work best and maintain their normal metabolic functions in the proper environment. Proper pH is among the most critical aspects of this environment. The plasma **bicarbonate-carbonic acid relationship** is actually reflected in the pH. The amount of carbon dioxide present is the

chief facet of this relationship. Thus, the amount of CO_2 blown off by the oxygenator will have great effects on the pH. Increases in the H^+ concentration and decreased pH will also cause increased cerebral blood flow.

Buffer Systems

A buffer system resists changes toward acidity or alkalinity. A buffer system works to keep the pH in the normal physiologic range when conditions are causing the production of an acidotic or alkalotic state. This is done through manipulation of added hydrogen or hydroxyl ions. A buffer is usually a weak acid and its related alkaline component. The buffering occurs when the hydrogen ion is brought into a reaction to form water. Thus, the pH is increased. The reaction can be rapidly reversed to produce a lower pH as well. The four main buffers of the body are the bicarbonate, hemoglobin, phosphate and serum protein.

The **bicarbonate system** is of major concern to the perfusionist. This major buffer system works through reactions to minimize the effects of excessive or insufficient CO_2 that would cause the pH to become acidotic or alkalotic. Bicarbonate ions leave the RBCs and enter the plasma in response to increased CO_2 in the blood. The **chloride ions** enter the RBCs as the bicarbonate ions leave to maintain an electrical balance. This is known as the chloride shift. The kidneys also have a role in the amount of bicarbonate ions in the blood. Bicarbonate can be excreted by the kidneys or reabsorbed and returned to the blood. Ammonia combines with hydrogen, and ammonia ions are released into the urine. This decreases the amount of hydrogen ions and raises the pH. A bicarbonate ion is added for every hydrogen ion that is excreted from the kidneys.

RBCs contain **carbonic anhydrase** which is an enzyme that increases the speed that CO_2 and H_2O are changed to carbonic acid. **Carbonic acid** is the part of the system that lowers the pH, while the bicarbonate ion (HCO_3-) is the component that increases the pH, causing the blood to become more alkalotic. Hyperventilation or blowing off too much CO_2 on CPB causes excessive loss of CO_2 and a drop in the hydrogen ion and bicarbonate levels in the body. This results in an increased pH.

Acidosis

Acidosis is present when the body has a decreased pH. Base deficit is a measurement used to describe the degree of acidosis. Acidosis is reported as a negative number while alkalosis, base excess, is reported as a positive number. Acidosis is either caused by a respiratory or metabolic factor. **Respiratory acidosis** is caused by too little carbon dioxide being removed by the lungs or oxygenator on CPB. On CPB, using a membrane oxygenator, this is caused by insufficient sweep gas to remove the CO_2. Other terms used to describe this increased CO_2 condition are hypercarbia and hypercapnia.

Metabolic acidosis is a condition in which the lungs are blowing off sufficient CO_2, but the kidneys, liver and other organs are allowing the patient's pH to decrease. On CPB, this is usually do to inadequate oxygenation of the tissues. The addition of large amounts of IV solution that has a low pH may also cause a lower pH. Lactated Ringer's Solution, for instance, has a pH of 6.2 and should be buffered with the addition of sodium bicarbonate. For correction of a metabolic acidosis on CPB adequate tissue perfusion should be present. The mean arterial pressure should be above 50 mmHg. The maintenance of an adequate pressure can be accomplished by increased pump flows and the use of vasopressors. Urine output should be adequate. It may be necessary to give diuretics to maintain adequate urine production. The kidneys correction of acidosis is very slow, however. Hydrogen ions must be excreted to effect a lower pH. If all factors are normal and the pH is still decreased, it may be necessary to administer sodium bicarbonate. This has an immediate effect on the pH. The most widely used formula for determining sodium bicarbonate dosage is (base deficit x weight in kg) / 4. Half of the dose is given and the response determined. The remainder of the dose can then be given if necessary.

Alkalosis

Alkalosis is a condition where the blood pH increases out of the normal range. This condition is also classified as either respiratory or metabolic. **Respiratory alkalosis** is caused by excessive removal of CO_2. On CPB sweep gas that is too high causes a drop in the carbonic acid content of the body and an increased pH.

Metabolic alkalosis exists when the pCO_2 is normal, but the pH is increased. If alkaline solutions are administered in such an amount that the kidneys are overwhelmed this condition may occur. Excessive urine output could also cause alkalosis. The addition of solutions with a decreased pH, such as Lactated Ringer's, may correct this condition.

Shunting

Arterial-venous shunting in the capillaries will cause certain unusual blood gas values. The oxygenated blood is not distributed to the tissues, but is shunted back to the venous side. The blood moves across a bridge that bypasses the capillaries. This shunting is normal in certain conditions when it is necessary to use the oxygenated blood in other areas. Shunting may occur inappropriately during CPB, however, and tissue hypoxia may result. The stress of surgery, cooling or certain medications may precipitate this inappropriate condition. There is not a blood gas value that directly indicates this condition. However, the hallmarks of shunting are a high venous pO_2, low systemic arterial blood pressure and decreased A-V pO_2 difference.

Signs of Shunting

- High venous pO_2
- Low systemic arterial blood pressure
- Decreased A-V pO_2 difference

Atmospheric Gases At Sea Level

Gas	% of total	partial pressure mmHg
Oxygen	20.84	159
Nitrogen	78.62	597
Carbon dioxide	.04	0.15
Water	.5	3.85

Alveolar Air

Gas	% of total	partial pressure mmHg
Oxygen	13.6	104
Nitrogen	74.9	569
Carbon dioxide	5.3	40
Water	6.2	47

Dalton's Law states that in a mixture of gases, the total pressure is equal to the combined partial pressures of various gases. Thus, both of the above equal 760 mmHg, the pressure of atmospheric air at sea level.

Corrective Actions

Abnormal blood gas results indicate that correction of one or more factors is required.

Normal Arterial Blood Gas

pH	7.35-7.45
pO_2	75 - 100 mmHg at room air (on bypass this is higher) (see O_2 tensions below)
O_2 sat.	96 - 100%
pCO_2	35-45 mmHg
BE	0
Bicarb	22-28 mEq/L

The following represent blood gases on bypass:

Respiratory Alkalosis
Example: pH 7.63, pO_2 95, pCO_2 24, Bicarb 26, BE +5
Condition: Respiratory alkalosis with moderate hypoxemia.
Corrective action: Decrease sweep gas to blow off less CO_2. Raise pO_2 by increasing FIO_2 percentage on oxygen-air blender. Increasing pump flow to expose more blood to the oxygen or cooling slightly would also increase the PO_2.

Respiratory Acidosis
Example: pH 7.24, pO_2 70, pCO_2 59, Bicarb 25, BE -3
Condition: Respiratory acidosis and hypoxemia.
Corrective action: Increase sweep gas to blow off more CO_2. Raise pO_2 by increasing FIO_2 or increasing blood flow.

Example: pH 7.21, pO_2 72, pCO_2 75, Bicarb 30, BE 1
Condition: Respiratory acidosis and hypoxemia.
Corrective action: Increase sweep gas to blow off more CO_2. Raise pO_2 by increasing FIO_2 or increasing blood flow.

Metabolic Acidosis
Example: 7.25, pO_2 210, pCO_2 29, Bicarb 13, BE -14
Condition: Metabolic acidosis.
Corrective action: Ensure adequate tissue perfusion, (adequate flow, arterial blood pressure and urine output) give sodium bicarbonate.

Alpha-Stat and pH-Stat

Alpha-stat and pH-stat are methods of calculating blood gas values that give somewhat different results. Perfusionists should be aware of the method their institution uses and understand the difference in results between the two methods. The important difference between the two calculating methods is the measurement of the pCO_2. The respiratory center of the brain responds to increased pCO_2 and dilates cerebral vessels. In normal individuals the respiratory rate will increase in response to higher pCO_2. On CPB, this is controlled by the perfusionist. The bicarbonate and base excess calculations are not affected by temperature if the pCO_2 and pH are obtained at the same temperature. The method that was first used in calculating values was the pH-stat technique. This technique requires that the values be corrected for the blood temperature. The idea of this is to keep the pCO_2 and pH at normal values of blood at 37 degrees. This usually makes it necessary to decrease the sweep gas when the patient is at lower temperatures, thus allowing the pCO_2 to rise. This is done because the pCO_2 decreases by 4.4% for every degree that the temperature is reduced. Therefore, the gas values display decreased pCO_2 and are more alkalotic at low temperatures. If the idea is to maintain normal blood gas values, then increased CO_2 would be necessary.

On the other hand, if the values are not temperature corrected as in the alpha-stat method, the pCO_2 would be interpreted as normal and the increased CO_2 level would not be necessary. In addition, the pH would not be increased. If the pCO_2 at 28 degrees is measured at 35 and not temperature corrected (alpha stat), it is reported as 35. If it is temperature corrected (pH-stat), it would be around 20 and it would be necessary to blow off less CO_2 to have a normal pCO_2 and pH. The net result is that those using the pH-stat method tend to keep their patients more acidotic with the idea that cerebral vasodilation and blood flow are improved by the higher pCO_2. Those using the alpha-stat method feel that cerebral blood flow may be independent of pCO_2 during CPB as cerebral autoregulation is disrupted and is more dependent on cerebral perfusion pressure and the mean arterial pressure. They feel that patients managed using alpha-stat results and are more alkolotic do better than those managed using pH-stat results who would be more acidotic. Technically, the advantage with the use of alpha stat management, is that with the constant degree of buffer dissociation, many aspects of metabolic activity, including the removal of metabolic by-products are improved.

Examples of Temperature Correction

Temperature	pH	pCO_2	pO_2
20	7.65	19	27
25	7.58	24	37
30	7.50	30	51
37	7.40	40	80

Oxygen Tensions at Various FIO_2 Concentrations

FIO_2	mmHg
30%	150
40%	200
50%	250
80%	400
100%	500

Normal Arterial Blood Gas

pH	7.35-7.45
pO_2	75 - 100 mmHg at room air (on bypass this is higher) (see O_2 tensions below)
O_2 sat.	96 - 100%
pCO_2	35-45 mmHg
BE	0
Bicarb	22-28 mEq/L

Normal Mixed Venous Blood Gas

pH	7.35-7.39
pCO_2	44-48 mmHg
pO_2	38-42 mmHg
O_2 sat	73-77%
BE	-2.5 to + 2.5
P_{50}	27 mmHg

NOTES

Blood Disorders

Cold Agglutinins

Cold agglutinins or cryoproteins are serum antibodies that work on the antigens found on the surface of red blood cells. This process may cause complement activation and red blood cells to clump at low temperatures and break down. The process does not occur if the patient is kept warm. This condition is classified as an **autoimmune reaction**. This disorder occurs primarily in patients over 50 years of age, but is also found in others. These antibodies are usually the IgM class in cases that the **agglutination** is significant. IgG and IgA antibodies may be involved in cases that are clinically insignificant. Tests can be performed to find the presence of cold agglutinins and at what temperature, if any, agglutination will be precipitated. Cold agglutinins can be caused by an infection such as a virus. These incidences of positive cold agglutinins may resolve with time and any elective cardiac surgery should be delayed if cooling of the patient or cold cardioplegia will be necessary. The level of the cold agglutinins at certain temperatures can also be determined with titers.

The techniques used to perfuse these patients involve keeping the blood at temperatures warm enough that agglutination is not likely to occur. Do not cool these patients below the identified temperature that may cause agglutination. If it is known that the patient must be cooled to temperatures that may cause a reaction, **plasmapheresis** is necessary. Many of the serum antibodies may be removed in this manner. Patients with severe cold agglutinins may have severe sequelae if the usual hypothermic bypass techniques are used. Perioperative myocardial infarctions, renal failure, hemolytic anemia and thrombosis may be the unfortunate results. The team members should all be aware of the potential problem and devise an appropriate strategy to keep the patient warm. There is some evidence that the dilutional effect of the pump prime may provide a measure of safety in preventing the process, but this is not predictable enough to be of practical value. The patient should be kept warm before, during and after bypass. A heating blanket on the OR table is beneficial. Normothermic bypass with a warm prime should be used. Packed RBCs should be warmed if added to the pump during the case. If cold cardioplegia is desired, crystalloid, non-blood, is used. The cardioplegia solution should be started warm to flush the coronary arteries of blood and then switched over to cold after 200-300 ml. The very cold temperatures that cardioplegia is ordinarily brought to will cause almost certain agglutination if blood is introduced to this solution. Blood cardioplegia must be given warm. A warm infusion of cardioplegia just before the cross clamp is removed will provide additional safety for the blood that will be entering the coronary arteries.

Sickle Cell Disease

The sickle cell is an abnormal red blood cell that is crescent shaped. The sickled cells are caused by **hemoglobin S (Hgb-S)**, an abnormal type of hemoglobin. The cells move slowly, clump together and eventually break down. In environments of low oxygen the sickling of the cells may be precipitated. **Hemoglobin A (Hgb-A)** is the major form of hemoglobin in the normal adult while hemoglobin S is the major form in individuals with sickle cell disease. Individuals with a majority of hemoglobin S are in a homozygous state and are described as having sickle cell disease. Those with less than 45% of hemoglobin S are in a heterozygous state and are described as having sickle cell trait as opposed to sickle cell disease. Patients with sickle cell anemia have blood clots and anemia with all of its symptoms. In sickle cell crisis, the blood vessels become occluded and pain, shortness of breath and convulsions may occur.

The strategy for the perfusionist should be aimed at preventing the sickling crisis and its damage. The plan should be to keep the oxygen saturations high and not cool the patient. Patients with this disease will experience sickling at less than 85% hemoglobin oxygen saturations, while those patients with only partial hemoglobin S will have sickling at less than 40% hemoglobin oxygen saturations. Patients with sickle cell trait may be placed on CPB safely if the oxygen saturations are kept at an increased level and acidosis is prevented. Acidosis increases the tendency toward sickling and is particularly injurious to these patients. Hypothermia also causes increased sickling. Thus it is best not to cool these patients and to use warm cardioplegia or crystalloid cardioplegia that is first infused warm to flush the coronary arteries and then changed to cold. This is the same technique described under the cold agglutinins section. Dilution, as with cold agglutinins, is beneficial to lessen the effects of this disorder. The use of vasodilators to keep vessels open is beneficial to prevent blockages.

Since patients with sickle cell disease experience sickling at saturation drops below **85%**, it is necessary to remove some of the blood before bypass and replace it with either whole blood or adequate combinations of components (packed RBCs, FFP, colloids and crystalloids). The blood can be removed with a Y connector in the venous pump line that is connected to a cell salvaging device. The platelets and the plasma can then be separated from the faulty red blood cells for reinfusion. The patient must be infused with volume via the arterial cannula as the blood is removed. On bypass, the oxygen saturations are kept high, and normothermia is maintained.

Other Red Blood Cell Defects

There are other types of red cell defects that are sometime encountered. In most cases, the red cells are fragile and care should be directed at preserving

their function. These patients can be successfully perfused using techniques of more than adequate oxygen saturation, good acid base management, normothermia and special cardioplegia methods. The cardioplegia is given warm, if blood is used. If cold cardioplegia is necessary, it must be crystalloid using the warm flushing method explained earlier in this chapter.

Polycythemia is an abnormal increase in the number of red blood cells. Individuals with pulmonary and heart disease may exhibit this abnormality. Those who live at high altitudes may also have this increased RBC count.

Thalassemia is a disease caused by insufficient hemoglobin synthesis that occurs in two forms. The red blood cells are microcytic, hypochromic and short-lived. In **thalassemia major, (Cooley's anemia)** the disorder is recognized early in life. The infant is anemic with splenomegaly and suffers from failure to thrive. The red blood cells are destroyed at too great a rate and transfusions are required. Iron from the breakdown of the cells is deposited in many organs causing damage. **Thalassemia minor** patients display mild anemia and slight red blood cell changes. These patients do not display symptoms and are thought to do well on bypass. Anemia is treated with packed RBCs if necessary. The cell walls are not fragile, however, and increased hemolysis should not occur as a result of bypass.

Elliptocytosis is a defect of the blood in which there are increased numbers of elliptocytes. The elliptocyte is an abnormal oval red cell. The red cells affected usually number less than 15%. In anemia this may be increased.

Spherocytosis, an inherited blood disorder, is the presence of abnormal spherical red blood cells. The cells are fragile and have shortened life spans due to premature removal by the spleen. Patients with this disorder often suffer acute episodes of stomach pain, fever and spleen enlargement. They are treated with transfusions, and splenectomy.

Hemosiderosis is characterized by increased deposits of iron in some tissues. This disorder is often found in patients with various red blood cell destructive disorders such as thalassemia.

Hemochromatosis is a rare condition in which iron deposits are built up in the total body with total body iron greater than 15 gm. Signs of this disease are a palpable liver, bronze discoloration of the skin, diabetes mellitus in over 50% of these individuals and cardiomyopathy. This disease is most often found after middle age.

Erythroblastosis fetalis is a form of anemia that affects newborns. These infants have Rh positive blood, but their mothers have Rh negative blood. The mothers have developed antibodies to the Rh positive blood and these may be passed to the fetus. The destruction of the RBCs in the fetus results in this anemia. The antigen-antibody reaction does not occur in first pregnancies ordinarily. It is in the succeeding pregnancies, after antibodies have developed, where the condition becomes apparent.

Platelet Disorders

There are three main types of platelet disorders. These are: (1) thrombocytopenia in various forms, (2) hereditary disease and (3) acquired disease.

Thrombocytopenia may be the result of decreased bone marrow production, increased sequestration in the spleen or accelerated destruction. Production defects may be he result of preleukemia, aplasia, metastatic cancer or anemia. Increased sequestration is associated with splenomegaly, liver disease, storage diseases or tumors. Excessive destruction may be the result of drugs, immune disorders, sepsis, vasculitis or foreign body contact (CPB, IABP, cardiac valves).

Thrombocytopenic purpura is an immune response that results in decreased platelets. This may be due to infection, drugs or toxic substances producing an autoimmunity. Symptoms include petechiae and bruising easily. The acute form usually occurs in children from 2-6 years of age and is a benign condition. It is self-limiting and usually resolves in 6 weeks. In the chronic form, adults from 20 to 50 years of age are affected. This condition requires treatment with steroids and splenectomy.

Hereditary Coagulation Disorders

von Willebrand's disease (vWD) is the most common inherited bleeding disorder. It is found in 1 in 800 individuals. The von Willebrand's factor (vWF) facilitates platelet adhesion and is the plasma carrier for factor VIII. It is manifested by the usual signs of bleeding disorders: bleeding gums, nose bleeds and slow clotting of lacerations. There are 3 types of this disorder. **Type I** disease patients have a mild decrease in plasma vWF with some impairment of hemostasis. **Type II** individuals have normal levels of vWF, but the factor itself is impaired and this results in mild cyclic thrombocytopenia. **Type III** is a severe form of the disease with very low vWF levels. These patients may experience hemarthroses.

The strategy for patients undergoing cardiac surgery is to infuse 10 bags of cryoprecipitate after coming off bypass. Cryoprecipitate is a plasma fraction enriched in vWF. This cryoprecipitate infusion should be repeated twice a day for 2 to 3 days postoperatively. Desmopressin Acetate (**DDAVP**) is beneficial in some of these patients. DDAVP raises the plasma level of vWF in patients with Type I disease and in normal individuals. DDAVP, however, does not improve hemostasis in Type II disease and should not be given to these patients. Hemostasis could be impaired because of thrombocytopenia due to platelet-vWF aggregates caused by the DDAVP.

The Hemophilias are hereditary bleeding disorders due to a lack of one of the clotting factors. Hemophilia is inherited as a sex-linked recessive trait with clotting problems found only in males. Females carry the abnormal

gene, but do not manifest bleeding disorders. The severity of the disorder depends on the percentage of the factor that is missing. These disorders are usually diagnosed in childhood from unusual bleeding occurrences. Chronic nosebleeds, dental bleeding and bleeding of the joints are all symptoms. On occasion internal bleeding may be a life-threatening circumstance. The forms most often found are hemophilia A and hemophilia B. Hemophilia C is a less severe form of the A classification.

Hemophilia A individuals exhibit a lack of factor VIII. Approximately 80% of hemophiliacs have the A form. The PTT is abnormal in cases that are severe and is a good screening test for hemophilia. Specific factor assays can then be obtained for those with abnormal results. Treatment for uncontrolled bleeding of hemophilia A individuals includes infusion of factor VIII that is commercially available.

Hemophilia B individuals lack factor IX. Factor IX is stable and active in plasma for long periods. This factor is commercially available.

Acquired Coagulation Disorders

Disseminated intravascular coagulation (DIC) is a severe, life threatening, bleeding disorder that is caused by the clotting substances being produced too rapidly in response to various conditions. This condition is sometimes encountered in open heart surgery. Clotting occurs throughout the body and the clotting factors are soon exhausted. This results in widespread, uncontrolled bleeding. Early signs of this condition are purple spots on the torso and then petechial hemorrhage. Treatment is to replace the blood components: fresh frozen plasma, cryoprecipitates, platelets and packed RBCs.

Vitamin K dependent deficiency is a coagulation disorder that displays decreased factors II, VII, IX and X. These factors are dependent on vitamin K for their production in the liver. A lack of this vitamin causes blood disorders. The vitamin is found in many foods such as leafy green vegetables egg yolks and alfalfa. It is also prepared artificially.

Primary fibrinolysis is a condition where the body's continuous process of dissolving fibrin to breakdown small fibrous clots becomes activated in the general circulation. Widespread hemorrhage occurs as the fibrinogen becomes depleted. This may be precipitated by surgery and CPB. This condition is treated with Amicar.

Control of Post-Op Bleeding

Excessive bleeding in the post-op cardiac surgery patient may require re-exploration if the blood loss is:

- Greater than 200 ml/hr for 4 hr
- Greater than 300 ml for 3 hr
- Greater than 400 ml for 2 hr
- Greater than 500 ml for 1 hr
- Bleeding with hemodynamic instability

This assumes that the bleeding is not due to a deficiency of platelets or fibrinogen. Measures to decrease bleeding without taking the patient back to surgery include blood pressure control, additional protamine administration, increased positive end-expiratory pressure (PEEP), DDAVP administration and blood product administration (platelets and FFP). A decreased hematocrit should be treated with reinfusion of the shed blood by autotransfusion or by administration of packed red blood cells. Evaluation of the bleeding by various tests and actions to follow may be summarized by the following:

Abnormal	Action
ACT	Administer 50 mg of protamine
PT, PTT	Administer 2 units of FFP
Platelets <100,000	Administer 6 pack of platelets
Bleeding Time >6 min	Administer DDAVP, 6 pack of platelets
Fibrinogen <100 mg/dl	Administer cryoprecipitate
Fibrin Split Products Increased	Administer Amicar, cryoprecipitate

Blood Conservation Techniques

The necessity of blood conservation and the avoidance of using **homologous blood** (donor blood) has grown in the past few years. The danger of using banked blood has been recognized, and there is a growing move to find new ways to conserve blood. There is risk of transmitting certain diseases. Practitioners are now aware that non-screenable, transmittable diseases are a risk when homologous blood is given. With this new awareness, blood usage has dropped considerably and will continue to fall as new conservation techniques are devised. Much of the decreased blood usage has come from perfusionists developing ways to remove all blood from the perfusion circuit at the end of the case. Manufacturers have also contributed to this reduction in homologous blood use. Lower prime oxygenators, circuits and other blood conservation devices have quickly gained acceptance. The standard of care is now that one should employ all measures to prevent homologous blood use. Blood banks have benefited from decreased usage by having more blood available for emergencies. Practitioners have benefited with reduced exposure to the hazards inherent in banked blood.

Autologous Blood

The patient's own blood, **autologous blood**, eliminates many of the disadvantages of homologous blood. Many patients will accept their own blood, but will not accept transfusions of homologous blood. Some adherents to the Jehovah's Witnesses religion will allow this type of transfusion. As mentioned above, the risk of disease transmission is eliminated. Cross-matching errors are also eliminated. Some patients are difficult to obtain blood for if they have multiple red blood cell antibodies or unusual blood phenotypes. There are several ways that the patient's own blood can be salvaged and returned to the them.

Autologous Blood Removal

The patient can donate his or her blood in preparation of a major surgery. The procedure is excellent for nonemergent cases where there is enough time to employ the technique. The patient's blood is collected gradually in the weeks before surgery. The patient is normally brought in every two weeks, and a small amount of blood is removed and stored. The total amount collected this way is usually about the equivalent of 2 units of packed red blood cells. The major drawbacks of this system are the time and numerous visits required to accumulate a sufficient amount of blood. The cost of drawing and storing the blood can also be considerable.

In heart surgery, another method of obtaining the patient's blood can be adopted. Blood can be removed just prior to starting CPB. After heparinization and cannulation, blood can be removed by way of the venous line and a Y connection to a collection bag. The venous line is unclamped and the priming volume is allowed to enter the venous reservoir. When the patient's blood reaches the Y connection, a clamp is applied and the blood is diverted to the collection bag. The patient can be infused through the arterial cannula to maintain adequate arterial blood pressure. The process is done slowly to prevent drops in blood pressure and arrhythmias. One of the safety features of this technique is that, in most cases, CPB can be initiated immediately if the patient becomes unstable. The patient is heparinized for the bypass procedure before the blood is removed, therefore the blood is anticoagulated when given back to the patient after CPB. Heparin in this blood must be taken into consideration when the protamine reversal dose is given. The blood collected this way retains all clotting factors. This blood contains platelets that will not be exposed to the pump circuit and bypass. Some teams remove blood from the central line or 14 gage IV line. Removing blood in this manner requires more time and eliminates the safety of being able to initiate CPB if done before cannulation. Blood saved before CPB is begun and reinfused after CPB has been shown in some studies to decrease post-op bleeding and coagulation defects. This method is valuable in preventing homologous blood administration. The problem with removal of blood in the OR is that the patient must have a high enough hematocrit to allow removal of the blood. With the dilution of CPB, small patients or those with a low hematocrit are not candidates for this procedure. Some surgeons are reluctant to use this method because of the dangers associated with ischemic heart disease.

Autotransfusion Blood Processors-Washers

Autotransfusion machines or **Cell Savers** have become common place in heart surgery and their employment is likely to increase as new uses are conceived. These machines allow the salvage of blood that is apparently lost. Blood on the surgical field can be aspirated with a special sucker line into a holding cardiotomy. In addition, blood remaining in the CPB oxygenator and circuit, can be salvaged at the end of the operation. Blood can also be aspirated from post-op drainage devices such as chest tube collection containers.

The main concept of the processor is to collect the blood in a heparinized container, to concentrate and wash the RBCs and to return the RBCs to the patient. The aspiration tube is a double line with heparinized saline constantly dripping down the line and back to the collection cardiotomy with the aspirated blood and fluid. The central component of the standard processor is the centrifugal bowl, know as **a Latham bowl**. Fluid from the cardiotomy containing blood enters a tube in the middle of the spinning bowl and travels to the bottom. The fluid moves to the outside where the heavier red blood cells

separate and concentrate. The leukocytes and platelets form a buffy coat layer on top of the red blood cells. The plasma and waste fluid accumulate on top of this layer. An exit tube on top of the bowl allows the plasma and effluent waste to leave the bowl to a waste bag. A sensor recognizes when the red blood cell level has risen to the top of the bowl and a wash with normal saline begins. The **normal saline wash** removes any debris or other harmful elements. It is important not to use sterile water for this wash as hemolysis would occur due to the hypotonicity of the water. Next, the blood is emptied to a holding bag for reinfusion.

The hematocrit of the processed cells suspended in saline is over 50% (possibly as high as 70%). The volume of a processed bowl in the adult set is **225 ml**. The effluent that is removed contains platelets, plasma fractions, leukocytes, free hemoglobin, heparin, saline and debris. The blood should be infused through a filter **within 6 hours** of salvage if kept at room temperature. A danger of the use of the processor is that insufficient washing may leave heparin in the red blood cells to be reinfused and bleeding may occur. Slow washes are better than fast washes for removing contaminants. Often large volumes are processed in cases of massive bleeding and it should be remembered that the product returned is devoid of platelets and clotting factors. It would be necessary, in this case, to also administer platelets and fresh frozen plasma.

Contraindications of Processor Use

- Gross contamination
- Malignancy of the surgical area
- Cesarean Section (Amniotic fluid should not be aspirated.)
- Use of topical hemostatic agents

Hemostatic Agents That Preclude Processor Use

- Antivene
- Gelfoam
- Helistat
- Hemopad
- Instat

Detrimental Factors Affecting Processor Use

- Antibiotics aspirated should be washed slowly and thoroughly.
- Betadine solution should not be aspirated due to hemolysis.
- Hot solutions should not be aspirated due to hemolysis.

Other Uses of Processors

Uses of these machines other than the traditional blood salvaging have increased recently. Blood left in the pump circuit can be salvaged. The blood can be pumped through the arterial cannula after the case into the processor sucker. There the blood enters the cardiotomy and can be processed. It is necessary to flush the circuit with priming solution to remove all the blood. This leaves a pump and circuit that is primed and ready to use in case of the reinstitution of CPB. The blood salvaged from the circuit is excellent for processing due to the lack of contamination and debris.

Use as a **rapid infusion device** is another capability of processors. The processor tubing is adapted by disconnecting the tube before it enters the centrifugal bowl. This tubing can be connected to a 1/4 inch line that leads to various IV sites for infusion. The air detector with automatic stop makes an excellent safety feature during this adaptation. The speed of the pump should be low to avoid overpressuring the IV infusion site.

Plasmapheresis and Plateletpheresis

Plasmapheresis and **plateletpheresis** are terms used to describe the removal of those portions from the blood while separating the blood cells and giving them back to the patient. The products may then be given to the patient after bypass. The purpose of this procedure is to conserve the platelets and other clotting factors, while preserving the hematocrit of the patient during bypass. The blood processor is used for this procedure. Uncontaminated blood is drawn into the centrifuge where it is spun to separate the components. The components (platelets or plasma) are collected in transfer bags where the waste liquid normally exits, and the red blood cells are collected as usual. The collected RBCs can be given back immediately or held until later.

Hemoconcentrators

Excess fluid can be removed from the patient's vascular system with a hemoconcentrator. The hematocrit of the patient will increase as the fluid is removed. During CPB these devices may be connected to the pump circuit and are a great asset for fluid management. There are no absolute contraindications to the use of these devices as there are with the autotransfusion blood processors-washers. Perfusionists do not ordinarily use the dialysate irrigating solution to affect electrolyte control. The fluid removed is plasma water along with solutes. Other solutions can be added to the pump volume to control electrolyte concentrations if necessary. This is often used for control of **potassium**. Plasma water, potassium and other electrolytes are removed and normal saline is added to reduce the amount of circulating potassium. If a normal saline irrigating dialysate was used, the process might be faster because the gradients of solutes in the solution and the blood would be different. However, this is usually not necessary during CPB.

The hemoconcentrator works by forcing fluid and small solutes across a semipermeable membrane. The formed elements of the blood are too large to cross this membrane. Globulins and other large proteins (fibrinogen and albumin) are also not removed. The sizes of the pores range from **15,000-55,000 Daltons** and this allows sodium, potassium, chloride, creatinine, urea and glucose to cross the membrane for removal. Heparin is also removed in small amounts. The size of heparin is 6,000-20,000 daltons, therefore a portion will cross the membrane and activated clotting times should be monitored often. The amount of circulating drugs may be decreased by the removal of the plasma and this should be considered when determining the patient's response to medications.

There are many places to incorporate the device in the pump circuit. Some perfusionists let the pressure of the arterial line provide the pumping force to remove the fluid. This is done by a Y connector and tubing coming off the arterial line. Pump flows should be increased to account for the flow that is diverted to the hemoconcentrator. Care should be taken to clamp this line when the pump flow is decreased due to the backflow that can occur. Other perfusionists desire a dedicated pump head to control the flow through the hemoconcentrator. This allows more control and avoids problems with diminished arterial pump flow. The disadvantage of this technique is that it requires a pump head for use. A method to gain the ability to quickly utilize a hemoconcentrator is to have a Y connector with a short length of tube built into the pump circuit. This tube comes off the arterial line and is kept clamped if use of a hemoconcentrator is not needed. If it becomes necessary to hemoconcentrate, one can be connected quickly to the tube.

The blood remaining in the pump circuit can be concentrated with the hemoconcentrator after CPB is completed. The blood is circulated through joined arterial and venous lines or through an A-V bridge and the hemoconcentrator is allowed to filter. An alternative technique is to concentrate the blood from the pump circuit and infuse it directly into the patient. This practice of **modified ultrafiltration** (MUF) is a method many perfusionists employ to reduce the excess fluid volume in the patient's vascular system after cardiopulmonary bypass.

NOTES

Conduct of Perfusion

Preparation of the case begins with review of the procedure to be performed. The type of operation, such as coronary artery bypass grafting (CABG) or atrial septal defect (ASD) repair, must be known in order to prepare for the operation. The type of case to be done will dictate what circuit and other equipment is to be used. The condition of the patient may also require special preparations. The patient's chart is reviewed before the case for patient size, history of prior surgery, general physical condition, neurological deficits, carotid insufficiency, blood disorders, pulmonary function, allergies and other factors that may influence the conduct of the perfusion. Lab results are reviewed for hematocrit, platelet count, fibrinogen level, serum creatinine, serum albumin, electrolyte levels and the presence of cold agglutinins. Abnormal findings may influence the pump prime or setup. There are many possibilities. Is blood required in the pump prime? Is the serum albumin low requiring extra albumin addition? Is an ultrafiltrator required? Is the patient allergic to drugs usually included in the prime? Are anatomic anomalies present requiring special techniques?

Pre-Bypass

Calculations are performed to determine necessary blood flow and if blood or other products are needed. The pump can then be primed and the sizes of the cannulae determined. Drug doses in the prime can also be determined. The surgeon should be consulted with regard to the size and the selection of the cannulae. In some cases, the surgeon may find an unusual condition requiring different or smaller cannulae. For example, the patient's heart may be smaller than usual, or the aorta may be severely calcified.

Predicted Hematocrits

The hematocrit that will result from the dilution of the volume used in priming the CPB oxygenator and circuit, should be calculated. Charts included in this book give the volume of oxygenators, filters and tubing. The volume of the circuit can be obtained from this information. The blood volume of the patient can be determined using blood volume charts and calculations below. The effects of the dilution can then be determined. The **volume-concentration formula, V1 x C1 = V2 x C2** is used for this calculation. For instance, the predicted hematocrit of a 75 kg patient, with a hematocrit of 30% and a circuit priming volume of 2200 ml would be determined by:

70 ml x 75 = 5250 blood volume of patient, this is V1
5250 + 2200 = 7450 total volume on bypass, this is V2
C1 is .30, the present hematocrit
C2 is unknown
V1 x C1 = V2 x C2
5250 x .30 = 7450 x C2
1575 = 7450 x C2
1575 / 7450 = C2
.211 = C2 the concentration or hematocrit on bypass
or stated differently a hematocrit of 21.1%

If a certain hematocrit is desired the amount of packed RBCs, if any, needed to achieve this hematocrit can be found for the same patient using the following. If our desired hematocrit is 25% the volume-concentration formula, V1 x C1 = V2 x C2, is used again.

V1 x C1	V2 x C2
5250 x .30	7450 x .25
1575 = patients RBC volume	1862.5 = RBC volume for 25 Hct

then
1862.5 - 1575 = 287.5 RBCs needed
The hematocrit of packed RBCs is 70% so,
287.5 / .70 = ?
410.7 ml of packed RBCs needed for hematocrit of .25

If it is necessary to add crystalloid solution, the effects can also be determined. For instance if 500 ml is added to our patient.
In this case
V1 = 7450 volume on bypass
C1 = .25 Hct on bypass
V2 = 7950 volume after adding 500ml
C2 = new hematocrit

V1 x C1 = V2 x C2
7450 x .25 = 7950 x C2
1862.5 = 7950 x C2
.23 = C2, the new hematocrit after adding 500 ml of crystalloid

If a 350 ml unit of packed RBCs is added the effect can also be determined. The RBC volume divided by the total volume equals the hematocrit. The hematocrit of packed RBCs is usually 70%.

.70 x 350 = 245 ml of RBCs, then adding to the RBC volume
(1862.5 + 245) / (7950 + 350)
2107.5 / 8300
.25 = the new hematocrit after adding 350 ml of packed RBCs

Pre-Bypass Checklist

A pre-bypass check should be performed and the results recorded on a permanent form. These checks can become routine and often there is the propensity to perform them quickly and superficially. This is, of course, exactly when an error will occur. The design of the checklist should include many checks common with other checklists, however it should also be tailored to the specific setup being used. Certain aspects of some perfusion circuits may be unusual and these items may not be found on the standard checklists. Could these items be included on the checklist? The checklist may have many checks that are unique to a particular institution.

The pre-bypass list should include the following:

____Chart Revied and Procedure Verified
____Patient data entered into pump computer
____Oxygenator holder in right place and secure
____Pump circuit tubing secure without kinks
____Luer connections tight
____Gas lines connected
____Gas lines not leaking and unobstructed all the way to source
____Gas supply operational, blenders and vaporizers working
____Appropriate purge lines opened
____Gas exhaust cap removed and scavenger line, if any, unobstructed
____Power cords secure on both ends
____Backup power available
____Handcranks available
____Backup light source available
____Waterlines connected
____Water heater-cooler operable and warming
____Oxygenator checked for leaks (before priming)
____Occlusion set on roller pumps
____Arterial filter primed
____Cardioplegia system primed and at proper temperature
____Drugs added to cardioplegia, if necessary
____Suckers and vent in proper direction in pump housing
____Vent valve in proper direction
____Pressure transducers calibrated
____Stopcocks closed properly
____Drugs added to prime as required
____Level detector operable
____Bubble detector operable
____Pressure warning-turn off devices operable
____Temperature probes connected
____Oxygen analyzer calibrated
____In-line sensors calibrated
____Supplies and backup components available

The pump is in the operating room by this time and circulating to ensure proper operation and to remove any air that may be trapped in the oxygenators, filters or circuit. The water heater-cooler is keeping the prime warm. This keeps the blood warm when going on bypass and prevents cardiac fibrillation from a cold priming solution.

Heparin Administration

Heparin is administered by the surgeon directly into the right atrium or by the anesthesiologist through the central line. If the heparin is given through the central line it must be flushed into the patient to ensure rapid heparinization. Three to five minutes after the heparin has been given an activated clotting time (**ACT**) is started to determine adequate anticoagulation. The goal of adequate heparinization is to maintain the patient's ACT at 480 seconds. Patients with an antithrombin III deficiency due to long hospital stays and heparinization or other reasons may require additional heparin doses. It is sometimes necessary to give large doses to reach the proper anticoagulation status. If the extra heparin does not help the anticoagulation status, it may be necessary to give fresh frozen plasma containing **antithrombin III** or the specific component.

Cannulation

The cannulae are now placed by the surgeon with purse string sutures. A test transfusion through the arterial cannula is performed to ensure proper placement. If the pump arterial line pressure rises greatly during this test transfusion, the cannula opening may be occluded by the aortic wall. Even worse, it may be protruding into the media which would cause a dissection. The retrograde cardioplegia cannula is usually placed, also, before bypass is begun. Some teams remove blood from the patient after cannulation via the venous cannula. This temporary removal preserves the platelets and clotting factors that would be lost during bypass for reinfusion at the end of the bypass period. This blood has the advantage of having platelets, clotting factors and red blood cells. The disadvantage is that the hematocrit of the patient drops for the bypass run.

Initiation of Bypass

The initiation of bypass is begun after the surgeon has instructed the perfusionist to go on bypass. The perfusionist should repeat this instruction loudly enough for everyone to hear. This is a good safety measure to ensure that the perfusionist has heard the surgeon correctly and that the surgeon is, indeed, ready for bypass to begin. Accidents have occurred when perfusionists have thought they heard the surgeon tell them to go on bypass when, in fact, they had not. The anesthesiologist stops the ventilator after bypass is initiated. A practiced routine of going on bypass should be used to initiate bypass. This

routine may be unique to the institution or to the perfusionist. The repetition of following this pattern is an obvious safety asset. The oxygen flow is started and the arterial clamp removed. The pump flow is begun slowly, while observing the arterial line pressure to make sure there is no obstruction of the arterial line and that the cannula is functioning properly. If the arterial line pressure suddenly rises there may be several causes. Terminate bypass and sytematically review possible causes.

Causes of Aortic Cannula High Line Pressures

1. Kink in arterial cannula or line.
2. Cannula improperly positioned.
3. Clamp too near cannula. (This occurs when crossclamp is applied.)
4. Cannula too small.
5. Arterial systemic blood pressure very high.
6. Aortic dissection.
7. Blockage in arterial filter.

The venous clamp or occluder is opened and venous return is checked. Flow is slowly increased to a cardiac index of 2.4 L/min/M^2. Poor venous return prohibits the establishment of adequate bypass. It is impossible to maintain adequate flow if the return doses not equal the arterial flow. This problem should be dealt with early in the case. The problem can be assessed systematically and corrected.

Causes of Poor Venous Return

1. Kink in the venous line or cannula.
2. Airlock in the venous line or cannula.
3. Oxygenator or venous reservoir is not positioned low enough.
4. Noncardiac suction being used instead of pump suckers.
5. Fluid rapidly moving to interstitial area, due to decreased intravascular colloid oncotic pressure (COP)
6. Venous cannula placed too far down or up, and vena cava not draining.
7. Vent or cardioplegia line inadvertently open and draining blood on field.
8. Bleeding due to accidental laceration or puncture in back of heart.
9. Bleeding due to other causes such as a bleeding ulcer.

Occasionally, the venous line may "**chatter**" due to excessive negative pressure causing a suction effect. The easy way to correct this is to place a clamp on the venous line that partially occludes the return. If the pump has a built in venous occluder this could, of course, be slightly closed.

Checks After Bypass Is Initiated

Immediately after bypass is established and full flows are obtained, standard operating procedures are reviewed. This review ensures that some aspect of the perfusion is not being overlooked. Some perfusionists use a written checklist for this stage also. These checks should be repeated mentally every few minutes if possible.

On Bypass Safety Checks

1. Blood flow at proper rate.
2. Arterial line pressure is normal.
3. Oxygen started at proper flow and concentration.
4. Oxygen saturations normal.
5. Patient's arterial pressure 50-90 mmHg.
6. Temperature appropriate.
7. Coagulation status acceptable.
8. Vaporizer turned on at appropriate level.

A check of the **safety devices** and other factors as required should be done at least once.

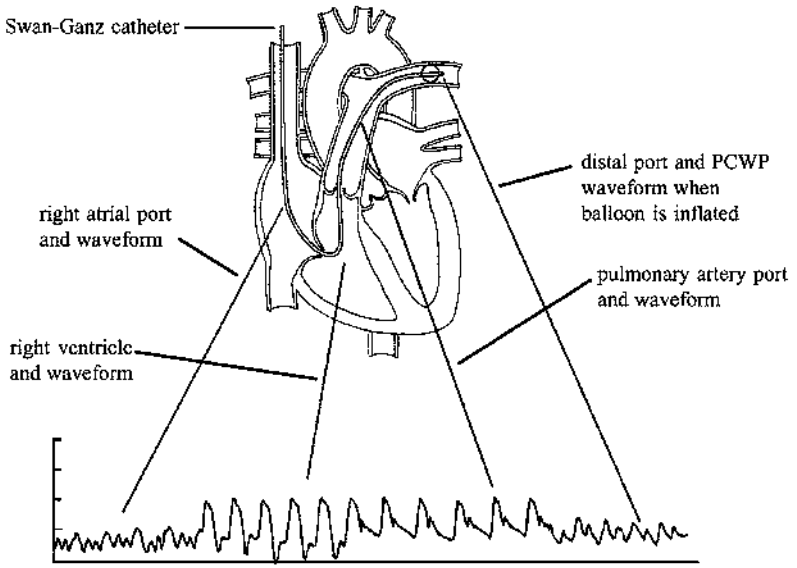
1. Bubble detector on.
2. Level detector on.
3. Manifolds in right position.
4. Drugs given as required.
5. Oxygen analyzer on.

Monitoring

Monitoring of various parameters is necessary and a vital component of the case. Many of these parameters are not monitored by devices on the heart-lung machine. The **EKG** is monitored throughout the case. During the crossclamped, arrested period electrical activity would be detrimental. In the stage before the crossclamp is applied the EKG is observed for ST segment elevation, fibrillation or other abnormalities. The period after removal of the crossclamp is similarly monitored. Electrical interference is a problem sometimes encountered that makes the EKG difficult to interpret. Power cords or cables placed over other equipment can cause this problem. Another cause is the pump roller heads creating an electrical charge on the plastic tubing and transferring this to the patient. The cell salvaging machine can also create this interference. This problem can be lessened if the electrode pads are not allowed to become dry. Using pads that are only removed from the wrapper just prior to use will help prevent this interference. The heart lung machine and all other electrical equipment should be routinely checked for electrical leakage. Maximum acceptable leakage is **100 microamperes.**

The pulmonary artery diastolic pressure is the indicator of volume

status after the heart is allowed to fill. The monitoring of this pressure is accomplished through a **Swan-Ganz catheter.** This catheter is placed pre-operatively through the internal jugular vein, into the right atrium and out into the pulmonary artery. This is used for the termination of bypass and may indicate distention of the heart if the values increase dramatically in the post crossclamp period. The normal pulmonary artery pressure is 25/8 mmHg. Pulmonary artery pressures are usually increased post-CPB.



Hypotension

Adequate blood pressure is one of the factors necessary for adequate perfusion of vital organs. Although acceptable ranges are sometimes debated, it is generally agreed that a mean systemic arterial pressure of **60-90 mmHg** is proper. Cerebral blood flow is autoregulated when the mean arterial pressure is above 60 mmHg. The circulating catecholamines (epinephrine and norepinephrine) levels are decreased at the onset of bypass due to dilution and the arterial pressure may experience a transient drop. The systemic vascular resistance **(SVR)** is reduced causing hypotension. The catecholamines are secreted by the adrenal medulla. Later in the surgery large amounts of the catecholamines are released by the reaction of the sympathetic nervous system to the stress of surgery. This raises the level to higher than normal. This causes the blood pressure to rise and blood is shunted to the most vital organs and to the skeletal muscles. Therefore, caution should be used when treating this transient hypotension. Ensure that the pump flow rate is correct and that flow

is not being diverted through an unclamped takeoff line of some kind. Before undertaking any aggressive measures verify that vasodilators such as nitroglycerin or nitroprusside are not inadvertently being given. The transducer monitoring the arterial pressure should be flushed and calibrated. The transducer should be at the appropriate level, also.

Vasopressor drugs may be necessary if the pressure does not rise after a brief while. Neosynephrine is usually tried first. Doses of 40-50 micrograms are appropriate in adult patients. The dose can be adjusted according to the patient's response. If the patient does not respond to Neosynephrine it may be necessary to give stronger vasopressors. Epinephrine is usually the next drug given to increase the pressure.

Hypertension

Just as hypotension may be harmful to the patient hypertension may also be harmful. In addition, excessively high pressures may cause leaking around suture and cannulation sites. Operations that require opening the aorta, such as aortic valve replacements, may have severe leaking or tearing after crossclamp removal. Pump line pressures may rise to dangerous levels. Generally, it is desirable to keep the patient's mean arterial pressure below 90 mmHg. Often it may be necessary to maintain the pump flow at a lower rate until the pressure drops. Oxygen saturations are monitored during this period and the FIO_2 is increased as required. Various agents are used to achieve this goal. The patient must be sufficiently anesthetized. Fentanyl is an intravenous drug also often used. This drug inhibits pain in the ascending pathways of the central nervous system. Often times, giving anesthetic agents is all that is needed to lower the blood pressure. Most heart-lung machines have vaporizers to deliver anesthetic inhalation agents such as **Forane. Nitroprusside** is a vasodilator that can be used during bypass. Venous and arteriolar smooth muscles are directly relaxed by this agent's actions. The drug can be given by a constant minidrip infusion into the reservoir. This drug must be kept in a container that is wrapped to prevent light from reaching the solution. Nitroprusside cannot be given faster than 10 mcg/kg/min due to possible cyanide toxicity.

Temperature

The priming solution is warmed before bypass is initiated. This prevents the heart from fibrillating due to the sudden introduction of a cold solution. Traditionally, cooling during bypass was routinely performed. Temperatures of 28^0 to 30^0 were felt to provide a margin of safety if the pump had to be stopped for any reason. The metabolic oxygen consumption is decreased with cooler temperatures. For every 7^0 C that the patient is cooled the oxygen consumption is decreased by 50%. Improvements in equipment and techniques have led to many teams using limited or no cooling.

Normothermic bypass causes less platelet dysfunction. It also requires higher flows that may increase blood trauma, however. Normothermic bypass is possible with today's oxygenators. Even large patients at warm temperatures can be oxygenated well with today's oxygenators. If cooling is used a temperature gradient **less than 10°C** between the arterial and venous blood is maintained. This prevents the formation of microbubbles. Methods of monitoring patient temperatures include placing esophageal, rectal, tympanic and bladder probes. If oxygen saturations are falling it may be due to several causes. The patient may not be adequately paralyzed and may be experiencing subclinical shivering. This can be corrected by giving additional paralyzing agents. The blood flow may not be high enough. It may be necessary to increase the blood flow to a 2.4 L index or higher. As stated above, lower temperatures cause the patient to utilize less oxygen. Therefore, cooling the patient may be obligatory if oxygen saturations cannot be sustained. Adequate flow ensures that organs are receiving sufficient oxygenation. The average adult's blood flow to the brain is about 750 ml/min. Blood flow to the coronary arteries is about 225 ml/min. About 70% of the oxygen is extracted from the coronary blood as it travels through the arteries. The blood flows to various organs while at rest or on bypass are:

Brain	Heart	Kidneys	Liver
15%	4 -5%	27%	29%

The following table illustrates acceptable settings that can be used initially for most membrane oxygenators. Refer to the operator's manual for recommended settings. Of course, blood gas calculation results are used to adjust the settings. Blood flow is calculated by multiplying the cardiac index by the body surface area.

Temperature	Cardiac Index	FIO_2	Gas/Blood Flow Ratio
37° C	2.4 L	.80	1 : 1
34° C	2.2 L	.70	.8 : 1
30° C	2.0 L	.65	.7 : 1
28° C	1.8 L	.60	.6 : 1
22° C	1.6 L	.50	.5 : 1

Renal Function on Bypass

Good renal function is an **indicator** of the adequacy of perfusion. The kidneys receive about 27% of the cardiac output but only require 7% of

the body's oxygen. The function of the kidneys as a filter requires that large amounts of blood pass through to remove waste products. If cardiac output, or blood flow on bypass, is decreased the kidneys are the first organ deprived of blood because of this. Short periods of no urine production are better than reduced blood flow to the brain or other vital organs. Although the production of urine on bypass does not guarantee good renal function post-operatively, it is desirable to have some urine output. Adequate urine is generally considered to be about 0.5 to 1.0 ml/kg/hr or about a ml a minute in the average adult patient. This production is a good indicator that other organs are being perfused. In addition, prevention of hyperkalemia is accomplished with good urine production. Excessive volume can be reduced, also. The urine output should be checked after 15 minutes on bypass to ensure renal function. No urine production after 30 minutes is usually treated. Obstructed Foley catheters are a common cause of this problem. Kinks in the tubing should be ruled out. Ensure that the catheter is connected to the tubing and not leaking on the OR table. At times excessive gel used to introduce the catheter may block the opening at the tip. Sometimes having the personnel scrubbed in at the table push on the bladder will remove this blockage.

Fluid that is moving to the interstitial area decreases the volume available for urine production. Blood pressure must be sufficient for proper perfusion and the filtering mechanisms of the kidneys. Mannitol 25%, 25 gm is often given during bypass and this may be all that is needed to stimulate urine output. If no production occurs the next drug used is Lasix 0.25 mg/kg.

Causes of No Urine Production	Corrective Action
1. Kinked or disconnected Foley catheter or tubing.	1. Straighten or connect tubing.
2. Catheter with tip obstructed by gel.	2. Push on bladder.
3. Decreased blood pressure.	3. Give vasopressors.
4. Low pump flows.	4. Increase flows.
5. Fluid moving to interstitial space.	5. Use mannitol or Lasix.

Crossclamp Period

The surgeon identifies the target sites, the harvest of the saphenous vein is completed and the revascularization of the heart is now ready to begin. The perfusionist is instructed to cool by those teams that practice hypothermic techniques. The crossclamp is placed at this stage. Those practicing normothermic techniques, of course, do not cool. The pump is turned down to decrease flow through the aorta while the crossclamp is placed. The flow is then brought back up slowly while carefully checking the line pressure. If the crossclamp is placed too close to the arterial cannula or the arterial cannula opening is facing the clamp, the pressures will be excessive.

Cardioplegia Administration

Cardioplegia is usually started antegrade for the initial dose. After arrest is accomplished the dose is then completed retrograde by those using the retrograde technique. A method of detecting a nonocclusive crossclamp when giving the cardioplegia antegrade can be used. The arterial pump flow is decreased for a moment while cardioplegia is being given. The cardioplegia line pressure should not drop if the crossclamp is occlusive. There are many variations of the cardioplegia solution and warm and cold methods of delivery. A more detailed discussion is found in the chapter on cardioplegia. All solutions use potassium to accomplish arrest of the heart. Antegrade cardioplegia is given with a line pressure of about 150 mmHg. Few teams monitor the pressure directly in the aorta at this time, but pressures monitored in the aorta would be about 50 mmHg if the line pressure is 150 mmHg using today's cardioplegia cannula. Retrograde administration requires that the pressure be monitored directly in the coronary sinus. Retrograde cardioplegia cannulae have lumens for this purpose that are connected to pressure transducers. The retrograde cardioplegia pressure is not allowed to rise above **50 mmHg** in order to prevent damage to the coronary sinus. The retrograde cardioplegia travels through the coronary veins, capillaries, arteries and then exits into the aorta. Here it must be removed by venting. (Antegrade administration exits into the right atrium where it is removed by the venous cannula.) The EKG should display a flat line if the cardioplegia is reaching its target. At times electrical interference will mimic cardiac activity and confuse the evaluation. A total dose of about 10 ml/kg is commonly given.

Additional doses are given about every 15 minutes to maintain myocardial protection. Most teams are moving toward giving these subsequent doses retrograde. Cardioplegia can also be given down vein grafts as they are attached. This is used to determine flow in the graft and to protect that region of the myocardium. Some teams monitor this temperature with a myocardial temperature probe, while others simply infuse cardioplegia periodically. Myocardial temperatures are kept below 15^0 C. Many teams use a warm dose of cardioplegia (36-38^0 C) just prior to crossclamp removal to uniformly rewarm the heart and to provide arrest during the early reperfusion period. This warm dose flushes the coronary arteries of detrimental metabolic products and air accumulated during the crossclamped period. Replenishment of high energy phosphates is also accomplished by this warm dose before the crossclamp is removed.

Rewarming the patient is done gradually, keeping the water bath temperature below 42^0 C. The warming water and the blood temperature gradient should be kept below **12^0 C in the adult and 8^0 C** in the pediatric patient. Warming the blood too rapidly may cause the red blood cells to absorb fluid causing hemolysis.

Venting

The left ventricle or aorta is vented to remove any air that may accumulate. Venting also removes blood that may distend the ventricle. Blood may return to the left ventricle through the bronchial vessels and through the pulmonary veins even if the venous cannula is removing all blood entering the right atrium. In cases of aortic insufficiency the aortic valve allows cardioplegia to enter the ventricle. Congenital abnormalities and septal defects will also allow the ventricle to become distended. This distention may stretch and injure the heart. One of the problems with femoral bypass is the lack of venting and decompression of the heart. A vent cannula can be placed directly in the left ventricle or the aorta can be vented through a Y line off the antegrade cardioplegia cannula. This venting off the antegrade cardioplegia cannula cannot be used when cardioplegia is being given antegrade. The venting can also be done through the right superior pulmonary vein, across the mitral valve into the ventricle. In some cases it is necessary to place a vent in the pulmonary artery to remove blood going to the lungs and back to the left atrium. One-way suction valves prevent excessive suction and accidental introduction of air into the heart. These safety valves should be used on all vents.

Fluid Management

The introduction of solutions to the circulating blood volume is necessary on bypass. The dilution of adding the priming solution to the patient's blood volume has consequences. The hematocrit is going to decrease. The hematocrit on bypass is kept from **22 to 29 percent**, ordinarily. This reduced hematocrit circulating solution perfuses the capillaries and tissues better that blood with a normal hematocrit. The arterioles have an average critical closing pressure of 20 mmHg and this reduced viscosity helps to maintain flow and pressure. The membrane oxygenator and tubing of the pump circuit also maintain a smooth **laminar flow** with the reduced viscosity. The flow varies inversely with the viscosity of the fluid. Addition of large amounts of crystalloid solution may also decrease the colloid osmotic pressure and cause third spacing of fluid. If large amounts of crystalloid have been added 25% albumin should be added also. Patients with decreased serum albumin and colloid osmotic pressure become edematous, often preventing closure of the sternum.

Termination of Bypass

The first step in terminating bypass is to ensure that all factors are satisfactory. The EKG should be acceptable and the pacemaker capturing as required. The hematocrit should be in an acceptable range. The potassium level should be in a normal range 3.5 - 5.0 mEq/L. Hyperkalemia causes the EKG to display tall, peaked T waves. Progressive hyperkalemia causes widening of

the QRS complexes and disappearance of the P waves. The QRS eventually becomes a sine wave pattern. Hypokalemia causes arrhythmias. The EKG displays ST segments that are decreased with U waves that become greater than the T waves.

The perfusionist should check to see if any vents are still working and inform the surgeon if this has been overlooked. The termination begins with the gradual clamping of the venous line while the arterial flow is decreased. The anesthesiologist turns on the ventilator. The pulmonary artery diastolic pressure is the indicator of the volume level of the patient. The patient is filled until a CVP level in the mid-teens is obtained in the normal patient. The arterial pressure waveform starts to show the effects of the heart ejecting the volume. The arterial systolic pressure should rise to an acceptable level of at least 90-100 mmHg. The pump is then completely stopped as the venous line is totally clamped. The arterial line is clamped after the pump is stopped. Manifolds are turned to the off position. The perfusionist should announce loudly that bypass is terminated. This announcement is a reminder to everyone that bypass is, indeed, terminated and the patient is being supported on the ventilator. Arterial pressures may drop after bypass and it is usually necessary to infuse volume slowly. Cardiac outputs are obtained to determine the pumping status of the left ventricle at this time. The perfusionist should not infuse volume while this output is being obtained. Infusion during this period would provide inaccurate cardiac outputs. Normal cardiac output for an adult is 5-6 LPM at rest. The cardiac index should be greater than 2.2 L/M^2. The beginning of protamine administration starts a different stage for the perfusionist. The pump suckers are turned off to prevent blood in the cardiotomy and oxygenator from clotting. If it is necessary to go back on bypass, re-heparinization is required. The surgeon decannulates at this time to prevent clots from forming on the tips of the cannulae. The venous cannula is usually removed first.

Salvaging Remaining Blood

There are many different methods of salvaging the blood remaining in the pump circuit and oxygenator. Cell savers can be used to remove and concentrate the remaining volume. This removes plasma proteins and formed elements except the red blood cells. Some perfusionists use an ultrafiltrator to concentrate this volume. This has the advantage of saving plasma proteins. There are advantages and disadvantages of both methods. If a cell saver is used for the case routinely, the ultrafiltrator would increase costs. On the other hand, if the ultrafiltrator is routinely used the costs would not increase.

Protamine reactions and other problems sometime occur that require going back on bypass. The perfusionist should always be prepared to go back on bypass and delay breaking down the pump circuit until the sternum is closed. ACTs can be obtained after the protamine is completed to ensure reversal of the heparin.

NOTES

Myocardial Protection

One of the greatest concerns of cardiac surgery is the protection of the heart during the operation. The repair of the heart is of no use if the heart is not adequately protected. The surgeon desires a bloodless, motionless heart on which to operate. However, he also desires a well-protected heart that recovers from the arrest period. The administration of a **cardioplegia solution** is the preferred method to accomplish both of these goals.

The development of myocardial protection strategies and cardioplegia solutions has undergone many improvements since the beginning of heart surgery. The earliest operations used no myocardial protection. It was eventually proven that these patients suffered extensive myocardial damage. One of the first methods of protection was to place ice around the heart, as was already being done with other organs at that time. The next improvement was the idea of injecting chemicals into the aorta between the crossclamp and the aortic valve. This idea was the beginning of the era of cardioplegia. The types of solutions have undergone many alterations, as have the methods of delivery. Numerous studies have shown that administration of cardioplegia during the ischemic time results in improved post-operative ventricular function .

Today, **blood cardioplegia** with high potassium and a relatively high hematocrit, has become the type protection used by most surgical teams. Crystalloid, oxygenated crystalloid, high sodium and low hematocrit cardioplegias have all been widely used at one time. The administration techniques have progressed from unmonitored pressurized delivery in the aorta to carefully controlled retrograde delivery into the coronary sinus. The concepts employed today provide a soft, pliable heart through arrest in diastole, while nourishing the heart muscle with oxygenated blood.

Myocardial Muscle

The myocardium is the thick muscular layer of the heart. The inside of the myocardium is covered by a membrane called the endocardium. The muscle fibers of the heart have many branches and interlace often. The purpose of the these muscle fibers is to contract the heart. The heart muscle uses oxygen rapidly and efficiently. About 70% of the oxygen in the blood that enters the coronary arteries is utilized. The fuel used by the myocardium to produce contractions is free fatty acids. Glucose and amino acids are also used as fuel in lesser amounts.

Cardiac muscle can be distinguished from both skeletal and smooth muscle in that it is striated. The muscle is made of tracts of cells that have a single, centrally placed nucleus. The spaces between the cells are occupied by the endomysium. This is composed of fibroblasts, collagen, reticulum fibers, capillaries and nerves.

The cardiac muscle is made of fibers that are in turn made of fibrils. The fibril contains the sarcomere, which is the main functioning component of striated muscle. The sarcomere is composed of pairs of parallel filaments, the thick myosin and thin actin filaments. These filaments are responsible for contraction. Cross bridges from the myosin filament to the actin filament move causing a sliding action of the parallel filaments on each other. **Calcium** is necessary for contractions, as it binds with the actin. The calcium then dissociates from the actin and is pumped back into the sarcoplasmic reticulum by an ATP powered transfer. This causes muscle relaxation.

Cardiac muscle is formed during embryonic development. The mesenchymal cells of the myoepicardial part of the embryonic heart are the cells that develop into cardiac muscle.

Cardioplegia Components

The search for the optimal cardioplegic solution has resulted in many variations. It seems that every institution has a different prescription that is followed. **Potassium** is the ingredient used to chemically arrest the heart. Infusion of potassium in large doses causes electromechanical arrest. There are varying concentrations used, but 15-30 mmol of potassium per liter of solution as it is delivered into the heart is typical. This requires the concentration of potassium in the cardioplegia solution to be much higher if a mixture of blood and cardioplegia is used. For instance, if blood cardioplegia is used in a 4 to 1 ratio, it is necessary to have 5 times the required delivery strength in the one liter solution. Thus, for a delivery of 20 mmol of potassium chloride per liter, there must be 100 mmol of potassium chloride in the liter of cardioplegia solution. This discounts the potassium already in the patient's blood that would actually increase the delivery strength 3-5 mmol. The pH of the solutions is usually in the 7.6 to 7.8 range, a mildly **alkaline state**. The buffering agents used to create this alkaline state are bicarbonate and tromethamine (THAM). THAM creates this alkaline environment by combining with hydrogen ions to form bicarbonate. Chloride is included in the solution to effect electrical neutrality.

Cellular edema is decreased by having a slightly hyperosmolar solution. The sodium in the solution contributes to this hyperosmolarity and prevents increased intracellular calcium. There is a balancing act that must be performed with the hyperosmolarity. Excessive hyperosmolarity would cause cellular dehydration. The proper level of hyperosmolarity prevents fluid from entering the cells at excessive rates. Glucose is another substrate that increases osmolarity and prevents edema. Mannitol is included in some solutions to increase osmolarity.

Perfusionists should be cautious when adding any drugs to cardioplegic solution. There are many factors affected by the addition of drugs to a cardioplegic solution. The pH, osmotic pressure and intracellular

concentration can all be affected by seemingly unrelated drugs because of the complex relationships of the chemicals present. In addition, the effect of the cold temperature on the cardioplegia is a factor. Some drugs may crystallize at the cold temperatures at which cold cardioplegia is given. The drugs and strengths of the various cardioplegic solutions in this chapter have all been determined with the effects on the above factors taken into consideration.

Actions of Ingredients of Cardioplegia Solutions

Ingredient	Action
Potassium	Electromechanical arrest
Sodium	Prevents edema and intracellular calcium buildup
Calcium	Membrane stabilization and prevents intracellular calcium buildup
Bicarbonate	Increases pH
Tromethamine	Increases pH
Glucose	Increases osmolarity to prevent edema, also a substrate
Mannitol	Increases osmolarity to prevent edema

Cardioplegia Solutions

The composition of the cardioplegia solutions in this chapter describe the crystalloid portion only, **before** they are mixed with blood in a **4:1 blood:crystalloid** ratio.

Composition of Common Cardioplegia Solution

	High Potassium	*Low Potassium*
KCl	100 mmol/l	30 mmol/l
THAM	12 mmol/l	12 mmol/l
$MgSO_4$	9 mmol/l	9 mmol/l
Dextrose	250 mmol/l	250 mmol/l
CPD-Adenine	20 ml	20 ml

4:1 Blood to Solution Mixture as Delivered

Na	105 mmol/l
K	25 mmol/l (High K), 9 mmol/l (Low K)
Cl	100 mmol/l
HCO_3	18 mmol/l
Mg	3.0 mmol/l
Phos	0.5 mmol/l
Ca (ionized)	0.7 mmol/l
Glucose	35 mmol/l
Hct	20%
PO_2	350 mmHg

The following is another common cold blood cardioplegia solution composition as it is **delivered** into the heart.

Potassium	25 mmol/l
Sodium	110 mmol/l
Chloride	114 mmol/l
Bicarbonate	27 mmol/l
Glucose	28 mmol/l
Mannitol	54 mmol/l

Another solution often used for warm induction arrest and before the crossclamp is removed contains **glutamate** and **aspartate.** Listed below are the ingredients before it is mixed with blood. This cardioplegia solution is always given in a 4 : 1 blood to crystalloid ratio. The main advantage of this solution is the addition of glutamate and aspartate (MSA/MSG). The amino acids comprising these substances are intermediaries in the Krebs Cycle and cause the ATP myocardial stores to be replenished.

Tromethamine 0.3M Comp. Sol.	225 ml
CPD	225 ml
Dextrose 50%	40 ml
MSA/MSG 0.46 M	250 ml
Dextrose 5%	200 ml
KCl, 2 mEq/ml	15 ml

The approximate electrolyte totals of the above solution per liter are:

Na^+	186.52 mEq/L
K^+	31.41 mEq/L
Cl^-	31.41 mEq/L

Oxygen Free Radicals

Oxygen derived radicals are altered oxygen molecules that are created in the cross-clamped ischemic period and the early stages of reperfusion. The reperfusion stage is the period immediately after the crossclamp is removed and blood begins flowing through the heart again. The chemical reactions that occur at this time result in the oxygen molecules having unpaired electrons added to their outer orbital shells. These oxygen free radicals react in detrimental ways with many structures in the heart. They have been shown to cause cell membrane damage, arrhythmias, myocardial injury and many other harmful effects.

One of the goals of cardioplegia solution design is to create **scavengers** that render these free radicals harmless. The beneficial effects of current free radical scavengers are often debated However, many researchers feel that inclusion of scavengers such as superoxide dismutase (SOD), N-2-mercaptopropionyl glycine (MPG), catalase, allopurinol or oxypurinol may be advantageous.

Cardioplegia Delivery Systems

Many different cardioplegia delivery systems are used today. Various manufacturers use different designs to build their systems. Almost all the systems allow delivery of warm and cold solutions and allow the mixing with blood. These systems also allow the monitoring of the infusion line pressure. This is accomplished by supplying a luer lock and high pressure monitor line for connection to a transducer.

The system should have the ability to deliver all crystalloid solution or all blood if necessary. This is usually accomplished by an arrangement of dual lines that run through the roller head together. One line moves crystalloid while the other moves blood. The ratios of blood to cardioplegia delivered are determined by the size of the tubing through the roller head. Blood cardioplegia in a 4:1 ratio has a larger size tubing supply the blood, while the smaller tubing supplies the crystalloid portion. If all blood solution is desired, the line delivering the crystalloid solution through the roller head must be clamped and removed from the roller head. Some systems have tubing arrangements that do not require removal of the tubing through the pump head. These systems allow blood to move through both tubes by a clamping arrangement before the pump head. Fluid of some kind must be moving through the tubing if it is left through the roller head raceway. If fluid is not moving through the tubing, air will be sucked out of solution and large bubbles will appear. One of the tubes may become partially pinched in the tubing holder if not seated properly. This pinching can cause decreased amounts of either the crystalloid solution or blood to be administered. The tubing must be **seated perfectly** in the tubing holder when the holder is snapped shut to prevent this problem.

There are newer systems that allow the concentration of the solution and blood to be set with a dial. The concentrations can be changed as required during the operation.

Antegrade Infusion

The most common method of cardioplegia administration involves giving a dose antegrade for the initial infusion after the crossclamp is applied. Antegrade cardioplegia administration is pumped through a special cardioplegia cannula into the aorta between the crossclamp and the aortic valve. A vent can also be used in conjunction with this cannula by means of a stopcock or a Y-connector. This allows blood to be aspirated through the vent when cardioplegia is being given in a retrograde fashion. The Y-connector type arrangement must be clamped and the vent turned off when cardioplegia is given antegrade, or the solution will be aspirated back through the vent and not reach the heart. Infusing between the crossclamp and the aortic valve forces the cardioplegia to enter the coronary arteries. The cardioplegia enters the coronary arteries by way of the right and left **coronary ostia**. The coronary ostia are open completely when the aortic valve is closed in diastole. The heart is arrested in **diastole** and full flow of cardioplegia into the os is possible. The initial dose is usually 10 ml per kg. The most common ratio of blood to crystalloid solution is 4 to 1. This means that if 1000 ml of cardioplegia is to be infused only 200 ml of crystalloid is actually given. The remaining 800 ml is blood.

Subsequent doses, after the arresting dose, are given down the vein grafts and retrograde. Infusion down the vein grafts should be done carefully. In some institutions, the perfusionist gives this dose with the roller pump head. In this situation, the pressure should be maintained at about 50 mmHg. This pressure is usually achieved at a flow rate of about 50-100 ml/min. The size of the graft will influence the amount of flow possible. The surgeon may also give the cardioplegia with a syringe down the distal vein grafts. The distal anastamosis is checked for leaking and adequacy of flow when cardioplegia is directed into the vein graft. Myocardial temperatures are monitored by some teams, while others prefer to follow standard administration schedules. Those monitoring myocardial temperature may alter their administration schedule if temperatures rise. Temperatures in the range of **10-15^0 C** are desirable.

Antegrade Cardioplegia Pressures

The pressure of the cardioplegia pumped into the aorta must be high enough to ensure proper distribution, but not so high as to injure the aorta or the aortic valve. Excessive pressures may cause myocardial edema and injury. Pressures of about **50 mmHg** would be appropriate if a special line were used to measure pressures directly in the aorta. Few teams use this type of monitoring of the aortic pressure through a dedicated pressure needle, line and transducer. Most teams monitor the infusion pressure of the cardioplegia

through the cannula. This is accomplished by a pressure monitor on the heart-lung machine and a line coming from the cardioplegia system. These pressures should range from about **125-150 mmHg.** Pressures should be high enough to force cardioplegia into the distant vascular beds.

Problems Preventing Arrest

The EKG is monitored for electrical activity as the cardioplegia is given. The goal is to produce minimal fibrillation and activity while providing an early arrest. Electrical interference may make it difficult to determine the level cardiac activity. Common problems causing electrical activity to continue, indicating inadequate protection, can be classified as **improper delivery, inappropriate washout or inadequate solution.**

Improper delivery can be caused by lesions in the coronary arteries that may block flow into that region of the heart. Left main coronary artery lesions may prevent protection of a large part of the left ventricle. The solution for this would be to limit the antegrade dose and to initiate the retrograde dose as soon as possible.

Another cause of ineffecive arrest can be aortic insufficiency, which would allow cardioplegia to enter the heart, decreasing flow down the coronary arteries. The heart may become dangerously distended. If the crossclamp is not completely occlusive, delivery is also impaired and arrest may not occur. A technique to detect malocclusion of the aortic crossclamp is to decrease the arterial pump flow while infusing cardioplegia at a constant rate. If the line pressure of the cardioplegia infusion decreases in conjunction with the decrease in arterial pressure, then the clamp is allowing cardioplegia to pass through. This allows the cardioplegia to enter the systemic circulation, while allowing systemic blood to enter the heart.

Inappropriate washout is caused by the venous cannula not allowing venous drainage of the heart. If the heart becomes distended, cardioplegia is not distributed, and the heart does not cool properly. The venous cannula may have to be repositioned.

Defective cardioplegia solution may be another potential problem. It is wise to make certain the potassium has been added to the solution. The cardioplegia solution may not work because it is not being delivered in the proper ratio due to pinching of the tubing. Pinching of the crystalloid line would greatly decrease the amount of potassium infused.

Retrograde Infusion

Retrograde cardioplegia administration is now used by most surgical teams. The retrograde administration technique has increased as surgeons have become more skilled at placing the cannula in the coronary sinus. The cannula is placed through the right atrium into the coronary sinus. This requires the surgeon to lift the heart and palpate the insertion site. The cardioplegia

cannula is best inserted after the venous cannula is placed to prevent dislodgment. Retrograde cannulation can be performed on bypass if the the right heart is kept full.

The cannula used has a balloon tip at the end of the cannula to hold it in the coronary sinus. Inflation of this balloon after insertion keeps it in place. The cannula is a dual lumen with a line coming off for monitoring the pressure in the coronary sinus. This line is connected with high pressure monitoring line to a transducer. The cannula and line should both be flushed of air before insertion. The monitored pressure of the coronary sinus should be maintained at **30-50 mmHg** during infusion. High pressure may indicate that the coronary sinus is becoming distended or that the cannula has entered a coronary vein. Both of these circumstances may cause rupture. Flow of approximately **200 ml/min** is normal in an average sized, adult patient. This is especially important when the myocardium is hypertrophied or if continuous warm cardioplegia is being utilized. Low pressure may indicate that the cannula is not in the coronary sinus or that the balloon is not seated properly. A sudden drop in pressure usually indicates that the cannula has become dislodged, and the surgeon should be informed. Another cause that is rarely encountered is rupture of the coronary sinus. To prevent this, it is important to initiate the retrograde flow slowly. High flow may cause dislodgment. The cardioplegia line pressure should also be observed. Obstruction of the cannula would cause high line pressure while the coronary sinus pressure would fall.

The cardioplegia enters the coronary sinus and flows through the veins, capillaries, arteries and then out of the coronary ostia into the aorta. The aorta must be vented to remove the cardioplegia and prevent overload. The retrograde technique has many advantages and a few disadvantages. The problem of distributing cardioplegia through coronary arteries that are blocked is overcome. Aortic valves that are incompetent are not a problem with this method. Retrograde administration may not protect the right side of the heart because the right coronary vein drains into the right atrium and not the coronary sinus. As a result, other forms of protection are usually given in conjunction with the retrograde method.

Valve surgery is another area where retrograde cardioplegia administration has proven beneficial. The method formerly used for aortic valve surgery required that the cardioplegia be directed into the coronary ostia. This was easy because the aorta was already opened. The cannulae used for this administration into the ostia are awkward to use and sometimes cause damage. Antegrade administration during mitral valve surgery is also difficult. Although the aorta is not generally opened, the intra-atrial retractors cause the aortic valve not to close properly. This allows cardioplegia to enter the heart causing distention.

Warm Cardioplegia

Some institutions practice myocardial protection using warm cardioplegia. The idea of this technique is that the electromechanical arrest causes the oxygen requirements of the heart to be sufficiently reduced. The logic is that the oxygen requirement is already reduced below a level that would make cooling necessary. Cooling would not offer any advantage if the oxygen requirement was already lowered via cardioplegic arrest. In addition, normothermic oxygenated blood cardioplegia furnishes more oxygen to the arrested myocardium than cold cardioplegia.

Retrograde cardioplegia cannulation makes it possible to deliver continuous warm cardioplegia in an unobstructive fashion. During the crossclamped ischemic time, the heart is protected more with the continuous warm cardioplegia administration. The continuous retrograde infusion is delivered at around 100-200 ml/min if the pressure allows it.

A disadvantage of continuous administration is the hemodilution of the patient with the large volume infused. This may require that packed red blood cells be administered. The perfusionist must constantly monitor the pressure and flows. This can be a major distraction. Also, the hypothermic safety afforded with cold cardioplegia is lost. Various techniques to achieve a bloodless anastamosis are necessary. The cardioplegia may have to be interrupted periodically, and the warm heart is more at risk of ischemic injury. There may also be regional disparity of delivery that leaves that portion of the heart at risk if warm cardioplegia is used.

NOTES

The Effects of Cardiopulmonary Bypass

CPB is one of the marvels of modern medicine, but it is a marvel that carries a price. Injury to some degree or another occurs in all patients who undergo CPB. In addition, the longer the bypass runs the more serious the injuries are likely to be. Membrane oxygenators, arterial filters, bubble detectors and other innovative products have all decreased the incidence of serious injury over the years and new inventions are likely to continue to improve the safety of patients. It is important to understand the injuries that do occur in order to decrease factors that may worsen the effects of CPB. The goal of the perfusionist is to return the patient to a normal physiologic state in spite of the recognized insults that occur.

Effects on the Lungs

The lungs receive blood from the pulmonary arteries and from the bronchial arteries that supply blood to nourish the lung tissues. Most of the blood from the lungs returns to the heart through the pulmonary veins. This makes left heart venting necessary during CPB. The lungs cradle the heart. Each lung is cone-shaped and has a peak, a base, three borders and two surfaces. The bases of the lungs are supported by the diaphragm. The peaks of the lungs extend about 4 cm above the first ribs into the root of the neck.

Damage to the lungs is the **most common serious** injury of CPB. The thickness of the respiratory membrane is only about 6 microns. The very act of bypassing the lungs lends itself to the creation of abnormal physiologic changes. The longer the pump run the more likely there will be pulmonary dysfunction. The lungs are, also, particularly subject to injury from the blood mediated inflammatory responses to contact activation caused by the pump circuit. Complement activation of **C3a and C5a** leads to activation of leukocytes. This may cause leukoembolization to occur in the lungs along with the release of oxygen free radicals and proteolytic enzymes by the neutrophils. **Pump lung** (congested lungs with intra-alveolar edema, interstitial edema and atelectasis) is a form of acute respiratory failure. The increased vascular permeability causing capillary leaking in the lungs is a result of complement activation, neutrophil arachidonic acid metabolites and other factors. The reduction in colloid osmotic pressure (COP) due to hemodilution may contribute to interstitial pulmonary edema. Overfilling the pulmonary vasculature may cause increased hydrostatic pressure resulting in fluid accumulation.

Atelectasis, collapsed lung, is a common event after heart surgery. This complication can often be predicted by the condition of the patient pre-operatively. The patient with poor pulmonary function pre-operatively is likely to have pulmonary complications post-operatively. A history of smoking, chronic bronchitis, obesity or pulmonary edema will most likely predispose

the patient to some degree of atelectasis. Most often the left lower lobe is affected, mainly due to the normal right deviation of the tracheal bronchial junction and resulting lack of drainage of the left lung during suctioning. The lungs are maintained in an altered state during bypass (deflation, static inflation or intermittent inflation) and this contributes to atelectasis.

The strategy for the perfusionist is to use an arterial filter, as is done in most institutions anyway. This filter removes leukocyte clumps, platelet aggregates and debris. There is some evidence that leukocyte depleting arterial filters may be beneficial and improve pulmonary function post-operatively. All packed red blood cells added to the pump should be administered through a filter for obvious reasons. In addition, the COP should be prevented from falling too far below normal by the addition of albumin to the pump prime. Priming with the addition of albumin (25 gm in adult patients) also provides a coating of the circuit that may prevent platelets from adhering to the circuit and activating. Lastly, the pulmonary artery pressure should not be allowed to increase to prevent distention of the heart and the pulmonary vasculature. This can be accomplished with left heart venting and temporary reduction in pump flows. The perfusionist should review the patient's chart to determine the pre-op pulmonary functions and optimize measures that may curtail post-op pulmonary complications. The **best indicator** for the potential of post-op pulmonary complications is the pre-op condition of the patient's lungs.

Pulmonary Function Abbreviations

CC	**Closing capacity**
Cdyn	**Dynamic lung compliance**
CSTAT	**Static lung compliance**
CV	**Closing volume**
ERV	**Expiratory reserve volume**
FEV1	**Forced expiratory volume in 1 sec in liters**
FEV3	**Forced expiratory volume in 3 sec in liters**
FVC	**Forced vital capacity**
FRC	**Functional residual capacity**
[H+]	**Concentration of hydrogen ions in nanomoles/liter**
IRV	**Inspiratory reserve volume**
MEF 50% VC	**Mid-expiratory flow at 50% vital capacity in l/sec**
MIF 50% VC	**Mid-inspiratory flow at 50% vital capacity in liters per second**
MMEF	**Mean maximal expiratory flow in l/sec**
MVV	**Maximal voluntary ventilation**
PEF	**Peak expiratory flow in l/sec**
PTP	**Transpulmonary pressure in mmHG**
Q	**Perfusion in l/min**
RAW	**Airway resistance**

RV	**Residual volume**
TLC	**Total lung capacity**
V	**Lung volume in liters**
VC	**Vital capacity**
V*	**Ventilation in l/min**
V*A	**Alveolar ventilation in l/min**
V*CO2	**CO_2 production in l/min**
V*O2	**O_2 consumption in l/min**

Effects on the Kidneys

Renal dysfunction after bypass is a relatively common occurrence. The kidneys filter large volumes of blood ordinarily and adequate perfusion of the kidneys on bypass is subject to many factors. Ordinarily about **25% of the cardiac output** is sent to the kidneys. The regulation of the excretion of water and solutes controls the blood pressure. Blood flow through the kidneys is affected by the composition of the pump prime, sympathetic nervous system and hormones (epinephrine, angiotensin, prostaglandins and bradykinin). The volume of the blood filtered may be decreased as a result of low blood pressure or reduced flows. Endocrine changes from the dilution of bypass may result in decreased renal function. The kidneys may be required to filter damaged red blood cells and other components that have been damaged in the bypass circuit.

The two kidneys are found in the retroperitoneal region. The kidneys consist of an outer cortex and an inner medulla. The cortex contains tubules and the glomeruli while the medulla contains only tubules. The **nephron** is the basic functional unit of the kidney. There are about one million nephrons in each kidney. The nephron is composed of the renal corpuscle, proximal tubule, Henle's Loop, distal tubule and the collecting duct system. The glomerular filtration rate (GFR) is the rate that the glomerulus permits passage of water, electrolytes, and other small molecules but, not blood cells and large proteins. The amount produced by each kidney is about **35ml/min/m^2**. Most of this large amount is reabsorbed by the proximal tubules.

Destruction of red blood cells (hemolysis) results in hemoglobin being released in the plasma. This may result in hemoglobin casts. If the amount of hemoglobin released is great the kidneys are unable to reabsorb filtered hemoglobin and hemoglobinuria results. The degree of renal failure varies but there is failure in about 13% of the patients. As with the lungs, the best predictor of failure post-op is poor renal function pre-op. Extended bypass times, as with pulmonary dysfunction, are also related to renal failure. Other factors besides poor renal function pre-op and extended bypass runs that contribute to renal failure are the copious use of homologous blood, multiple exposures to angiographic dyes and the use of the intraaortic balloon pump.

Serum creatinine level is an excellent indicator of current renal function. Normal serum creatinine is less than 1.0 mg/100 ml. Higher levels indicate renal failure and indicate the possibility of failure post-op. Perfusionists should routinely review patients' charts for serum creatinine levels. Hemodilution from the pump prime reduces the viscosity of the blood and this causes increased renal blood flow and greater urine output with its beneficial effects.

Excessive addition of albumin to the pump prime may have a detrimental effect on the function of the kidneys. This large protein in too great amount can be harmful. However, there is definitely a need for the addition of some albumin to prevent excessive interstitial fluid buildup and grossly edematous patients.

The patient will usually experience a weight gain from the fluid accumulation of CPB and it is usually days before the patient's weight returns to normal. The patient may have to be treated with diuretics such as furosemide and 25% mannitol. Dopamine in renal doses increases creatinine clearance, sodium excretion and urine output. In 1.5% of patients renal failure occurs that does not respond to pharmacological agents and dialysis is required.

Renal failure may be **acute or chronic**. In acute renal failure nitrogen wastes accumulate in the blood rapidly. In chronic renal failure urine decreases in amount and failure signs may occur. Increases in fluid intake do not affect the urine output. Mental status changes, seizures, gastrointestinal bleeding and yellowing of the skin may be present. Uremic frost, a crystal-like substance, may cover the skin.

Neurological Effects

Neurological changes to some degree are prevalent after CPB. These changes are often minimal and cause no lasting problems. However, in a small percentage of the cases serious, permanent damage occurs. This permanent damage is usually the result of an embolic event (cerebrovascular accident, stroke). An embolus in the brain from air, blood clot, fat, atheroma, calcific debris or circuit debris blocks an artery and results in lack of oxygen delivery to the affected region of the brain. An air embolus is primarily nitrogen which does not dissolve rapidly. This is why the pump circuit is flushed with highly dissolvable CO_2. Indicators of air embolisms include seizures, cardiac arrhythmias and ventricular dysfunction. Over two thirds of these events occur intraoperatively with the others in the post-op period. Embolization due to cannulation and clamping of the aorta is thought to cause most neurological damage. The incidence of stroke may be as high as 5% according to some studies. These obvious neurological injuries are not hard to diagnose.

The effects of a stroke differ according to the location and the extent of damage. In some cases death may occur. In other cases paralysis, loss of ability to speak or inability to understand language may be present. After a few days some function may return due to reduction in brain edema. Before

the operation, the patient's chart should be reviewed for any **previous neurological injury** or current deficit. Pre-op neurological assessments will include asking the patient about memory loss, periods of unconsciousness, weakness, numbness, tremors, personality changes and unexplained confusion. The physical examination should note speech pathology, emotional state, appropriate facial expression, attention span, ability to follow instructions, responses to stimulation, coordination, balance and strength of extremities. The reflexes are tested for appropriate response. The eyes should be observed for ocular movements and the presence of nystagmus (jerky eye movements). The pupils are checked for roundness, size and reaction to light. If there is neurological injury this baseline information is valuable in assessing the extent of injury and devising a treatment protocol. The **CT scan** is the best method to determine the extent of brain injury in adults.

Subtle injuries are much more difficult to determine. What some may judge to be emotional change due to possible injury may just be a very normal psychological reaction to having major surgery. In addition, signs and symptoms such as loss of vibratory sense and reduced hand-eye coordination are difficult to ascertain. Neuropsychological studies that test such factors as cognitive functioning are controversial. Whether these tests with abnormal results indicate true neurologic dysfunction is not clear. Neurological exam of the recuperating cardiac patient for subtle injury is difficult and depends on the thoroughness of the pre-op exam. The convalescent period also serves to allow recovery from these lesser injuries. Thus, by the time a thorough exam is performed the injuries have changed or improved considerably.

The task for the perfusionist is to eliminate all possible sources of embolism from the pump circuit. Arterial filters have long been recognized as a main line defense in this quest. Pre-bypass filters that filter the circuit prime pre-op and are then discarded, dramatically reduce pump debris. A gas line filter may help prevent embolism, also. Cooling and warming gradients should be kept within guidelines to prevent air from coming out of solution. In essence, prevention of embolism from the pump circuit is central to the art of the perfusionist. The perfusionist should continuously guard against this devastating injury. The surgeon should be particularly careful removing the crossclamp. The OR table should be placed in the **Trendelenberg position**, causing the patient's head to be lower than the rest of the body during the crossclamp removal. The perfusionist should be sure that the aorta is vented vigorously during this time to remove any air coming from the heart.

Low pump flow rates and hypoperfusion may be related to cerebral injury according to most studies. Other studies show that low arterial pCO_2 may lead to vasoconstriction and injury. The advantage and disadvantages of alpha stat blood gas techniques and pH stat techniques is discussed in other sections of the book. Very high pO_2 may also cause cerebral vasoconstriction and, therefore, should not be maintained at excessive levels.

Hematological Effects

Exposing the patient's blood to the perfusion circuit and the use of systemic heparinization causes changes in the blood and its coagulation ability. The presence of plasma free hemoglobin in the patient post-operatively indicates that the red blood cells are damaged by bypass. Dilution with the priming solution is another major factor affecting the blood. Perfusionists should be aware that patients who are taken back to the operating room and require being placed on CPB again will almost assuredly require the administration of blood products. Post-operative hemorrhage is a common complication of cardiac surgery. Patients returned to the operating room for post-operative bleeding may number as high as 5%. Patients with preexisting diseases that compromise coagulation or those on certain medicines such as aspirin are especially at risk for cardiac surgery. Liver disease, uremia and other illnesses can leave the patient vulnerable to coagulopathy. The presence of heparin in the patient that has not been neutralized is a cause of post-operative bleeding. Accurate reversal of the heparin given for CPB is important to prevent bleeding. Extra protamine is often given if there is any doubt of the adequacy of heparin reversal.

Platelet dysfunction may be the result of the dilutional effect of bypass or the interaction of the blood with air and material of the pump circuit. Copious use of cell saver blood and the use of intraoperative drugs may also be precipitating factors. Critical thrombocytopenia is defined as a platelet count below 50,000 per cu/mm. This is usually treated with the administration of platelets. Platelet counts post CPB greater than 100,000 normally do not require treatment.

Disseminated intravascular coagulation (DIC) occurs rarely post-bypass. In this serious problem the coagulation factors of the patient become inappropriately activated and bleeding occurs systemically. Fibrinolysis does occur as a result of contact with the pump circuit. Heparin inhibits fibrinolysis and maintenance of appropriate activated clotting times is necessary to prevent significant complications.

Serum electrolyte changes may also occur. The serum potassium may be diluted, lowered with copious urine output or lowered due to intracellular shifts. The potassium may rise in some cases due to large doses of cardioplegia, or defective transport of intracellular potassium and glucose. Calcium, magnesium and sodium all usually decrease due to dilutional effects.

Effects on the Complement System

The complement system involves complex proteins in the blood that bind with antibodies against infection and foreign bodies. Severe allergic reactions may also involve the complement system. The activation of the complement system includes over 18 plasma proteins. The complement proteins react sequentially and mediate a number of the immune responses. Inappropriate

activation of the complement system can result in injury to the patient. Anaphylatoxins that increase vascular permeability, edema and smooth muscle contractions may be produced. Chemotactic factors that cause white cells to migrate to an area of inflammation and release lysosomal enzymes and toxic **oxygen free radicals** that destroy tissue are also generated. Red blood cells may also be destroyed by complement activation as in cases of autoimmune hemolytic anemia.

The bypass circuit causes a certain degree of complement activation. The production of **C3a and C5a** anaphylatoxins causes migration of neutrophils to the walls of vessels. **Arachidonic acid** metabolites are also released causing increased vascular permeability. This causes capillary leaking in the lungs.

Effects on the Endocrine System

The endocrine system is defined as the ductless glands that secrete hormones directly into the bloodstream. They cause target organs to react in certain ways affecting many functions of the body. The endocrine glands also affect secretions of each other. The endocrine system includes the adrenal glands, thyroid and the parathyroid, the pituitary, the pancreas, the gonads and the pineal gland. The thymus gland, once thought of as an endocrine gland, is now classed with the lymph system. The supraoptic and paraventricular nuclei of the hypothalamus in conjunction with the pituitary gland secretes **vasopressin**, also known as antidiuretic hormone, that decreases urine output. The adrenal glands are bilateral and each one is found on top of each kidney. The gland consists of the medulla and the cortex. The adrenal medulla secretes epinephrine and norepinephrine **(catecholamines)**. These catecholamines increase blood pressure by vasoconstriction and are increased during bypass after an initial dilutional effect.

The thyroid gland releases the thyroid hormone. This is released as **thyroxine (T_4) and triiodothyronine (T_3)**. These hormones increase the heart rate, contractility and cardiac output. The thyroid hormone also regulates the agonist sensitivity of beta-adrenergic receptors. Other functions such as body temperature and metabolic functions are also affected by these hormones. The thyroid stimulating hormone (TSH) governs the release of T_4 and T_3. T_4's normal half-life is 6 to 7-days. T_3 has a normal half-life of about 2 days or less. T_3 is reduced in response to CPB. T_3 is used by some open heart teams for patients who cannot be weaned from bypass after the usual measures. It is given after going back on bypass and continued in the recovery phase if successful.

Insulin is a hormone released by the pancreas in response to increased blood glucose levels. Insulin regulates the metabolism of glucose and other processes necessary for metabolism of fats, carbohydrates and proteins. The transport of glucose into the cells is promoted by insulin. The insulin response is decreased during hypothermic bypass and glucose levels rise. During the

rewarming phase, the insulin response increases. Hyperglycemia usually continues for an hour or two after bypass is terminated.

Hepatic Effects

The liver is a complex organ and the largest organ of the body. The liver consists of four lobes and is supplied blood by two means. The hepatic artery supplies oxygenated blood from the heart while the hepatic portal vein supplies nutrient filled blood from the stomach and the intestines. The liver contains about 13% of the total blood, and processes glucose, proteins and fats.

Studies at The Milton Hershey Medical Center have shown that during bypass, hepatic oxygen consumption is maintained if pump flows are at least 2.2 liters/m^2/min. Liver enzymes are often elevated post bypass, indicating possible hepatic injury. As with other organs longer bypass times are associated with increased injuries. Some patients become jaundiced after surgery. This may be the result of hepatic injury, excessive bilirubin due to blood transfusions or blood trauma. This jaundice is usually self-limiting and clears in about a week.

Summary of Detrimental CPB Effects

Lungs
Atelectasis
Increased interstitial water
Decreased surfactant production
Pulmonary edema
Leukoembolization

Kidneys
Decrease glomerular filtration rate
Emboli to kidneys
Renal failure

Neurological
Cerebral emboli
Decrease cerebral blood flow
Cerebral hemorrhage
Stroke
Temporary deficits

Hematological
Heparin rebound causing coagulopathy
Reduced platelet counts and impaired function
Decreased hematocrit
Decreased serum electrolytes

Complement System
Production of C3a and C5a anaphylatoxins
Release of arachidonic acid metabolites
Release of oxygen free radicals

Endocrine
Catecholamine increase
ADH (Vasopressin) increase
Increased renin, angiotensin and aldosterone levels
Decreased ACTH
Decreased T_3, thyroid stimulating hormone
Decreased insulin response and elevated blood glucose

Hepatic
Elevated liver enzymes
Jaundice

NOTES

Accidents

Perfusion accidents occur suddenly and require a thoroughly planned response to correct the situation. The unexpectedness of the problem may cause an improper response unless the perfusionist follows a systematic, deliberate procedure. Naturally, the best way to handle accidents is to avoid them. The perfusionist should eliminate or alter any part of the circuit or procedure that might be a potential hazard. Occasionally, something is noticed during the procedure that may be a safety problem. Action should be taken at this point to remedy the potential problem. The perfusionist should always strive to correct problems before they occur.

The accidents and corrective actions presented are those that have been reported over the years. These accidents and responses should be studied. Practice the procedures routinely, and the real emergency will be handled with greater ease. Listed below are emergencies and sequential procedures to follow to correct or alleviate the problem.

Massive Air Embolism

1. TURN OFF PUMP IMMEDIATELY AND CLAMP ARTERIAL AND VENOUS LINES.
2. PLACE PATIENT IN DEEP TRENDELENBERG POSITION (HEAD DOWN).
3. DISCONNECT ARTERIAL CANNULA FROM ARTERIAL TUBING
4. COMPRESS CAROTID ARTERIES.
5. REMOVE AIR FROM AORTA AND CANNULA.
6. REFILL ARTERIAL LINE TUBING WITH FLUID.
7. DETERMINE CAUSE OF ACCIDENT AND CORRECT PROBLEM. ENSURE THAT OXYGENATOR GAS OUTLET VENT IS NOT OBSTRUCTED.
8. PLACE VENOUS CANNULA IN SUPERIOR VENA CAVA AND SLOWLY BEGIN HYPOTHERMIC RETROGRADE PERFUSION ACROSS THE A-V BRIDGE. CONTINUE FOR 1-2 MINUTES WHILE VENTING AORTA.
9. GIVE PHENOBARBITAL.
10. PACK HEAD IN ICE.
11. DISCONTINUE RETROGRADE PERFUSION AND RESTART BYPASS WITH 100% O_2.
12. GIVE VASOPRESSORS TO MAINTAIN HIGH PERFUSION PRESSURES.
13. GIVE STEROIDS.
14. CONSIDER HYPERBARIC CHAMBER.

Water to Blood Leak

SIGNS OF LEAK:

1. UNEXPLAINED RISE IN VOLUME. ENSURE THAT INCREASED VOLUME IS NOT FROM SUCTION OF IRRIGATING SOLUTION OR INADVERTENT ADDITION OF CRYSTALLOID SOLUTIONS.
2. DECREASING pH.
3. DECREASING HEMATOCRIT.
4. HEMOLYSIS WITH HEMOGLOBINURIA.

ACTION: CHANGE OXYGENATOR.

Oxygenator Failure

SIGNS OF FAILURE:

FALLING ARTERIAL PO_2 OR FALLING VENOUS SATURATION IN THE PRESENCE OF 100% FIO_2 AND FLOWS AT A NORMAL TO HIGH CARDIAC INDEX AND ADEQUATE ANESTHESIA.

ACTION:

1. ENSURE THAT OXYGEN LINES ARE CONNECTED THROUGH THE ENTIRE CIRCUIT (FROM WALL, TO BLENDER, TO OXYGENATOR).
2. CHECK THE O_2 ANALYZER TO ENSURE THE BLENDER IS DELIVERING THE PROPER FIO_2. IF ANALYZER IS NOT IN GAS CIRCUIT, CONNECT 100% O_2 FROM A PORTABLE TANK.
3. IN THE PRESENCE OF A SLOW FAILURE AND TIME PERMITTING, EVALUATE THE OXYGENATOR'S O_2 TRANSFER, USING THE TRANSFER FORMULA FOUND IN THIS BOOK.
4. CHANGE OUT OXYGENATOR.

Oxygenator Changeout

REASONS FOR CHANGEOUT:

1. OXYGENATOR FAILURE. UNABLE TO OXYGENATE.
2. OXYGENATOR LEAKING BLOOD.
3. HEAT EXCHANGER LEAK. WATER TO BLOOD LEAK.

STEPS IN CHANGEOUT:

IF TIME PERMITS, AS IN A SLOW OXYGENATOR FAILURE, FOLLOW STEPS 1-4. IF TIME DOES NOT PERMIT BEGIN AT STEP 5.

1. NOTIFY SURGEON OF THE NEED TO CHANGE OUT OXYGENATOR.
2. ASSEMBLE THE EQUIPMENT NEEDED.
3. IF POSSIBLE PRIME THE NEW OXYGENATOR.
4. IF COLD, COOL FURTHER. IF WARM, STAY WARM. IF YOU ARE WARM ENOUGH AND THE HEART IS STILL EJECTING, VENTILATE, COME OFF BYPASS TO CHANGE THE OXYGENATOR.
5. COME OFF BYPASS.
6. DISCONNECT WATER LINES.
7. DISCONNECT O_2 LINE.
8. DOUBLE CLAMP THE BLOOD INLET AND OUTLET LINES. ALLOW ENOUGH LINE BETWEEN CLAMPS TO DIVIDE THE LINES AND HAVE ENOUGH LINE TO CONNECT TO NEW OXYGENATOR.
9. DO THE SAME TO ANY OTHER BLOOD LINES COMING OFF OF THE OXYGENATOR, SUCH AS BLOOD CARDIOPLEGIA OR RECIRCULATION LINES.
10. DIVIDE LINES USING STERILE SCISSORS.
11. REMOVE THE OLD OXYGENATOR FROM HOLDER AND SALVAGE BLOOD LATER IF POSSIBLE.
12. PLACE NEW OXYGENATOR IN HOLDER.
13. CONNECT THE BLOOD INLET LINE AND ANY BLOOD CARDIOPLEGIA OR RECIRCULATION LINES.
14. PUMP SLOWLY THROUGH THE OXYGENATOR UNTIL BLOOD REACHES THE OUTLET PORT. IF OXYGENATOR WAS PRIMED, THIS STEP IS NOT NECESSARY.
15. CONNECT THE OUTLET LINE AND CIRCULATE THROUGH A RECIRCULATION LINE OR A-V BRIDGE TO REMOVE AIR.
16. CONNECT O_2 LINE.
17. CONNECT WATER LINES.
18. RESUME BYPASS.

Methemoglobinemia

This is a condition in which hemoglobin has an ion altered from the reduced form to the oxidized ferric molecule which is not capable of binding with or releasing oxygen. This condition is associated with prolonged periods of nitroglycerin administration (greater than 10 mcg/kg/min).

SIGNS: PRESENTS AS DARK BLOOD IN THE PRESENCE OF NORMAL OR HIGH PO_2.

ACTION: ADMINISTER METHYLENE BLUE AT 1 MG/KG. THIS REDUCES THE FERRIC COMPOUND TO ITS FERROUS STATE.

Malignant Hyperthermia

Malignant hyperthermia is a life-threatening syndrome caused by the administration of anesthetics. Inhalation agents such as halothane, enflurane and isoflurane have been identified as causative agents for this syndrome. Succinylcholine has also been identified as a causative agent. The syndrome manifests with metabolic acidosis, hypercarbia, skeletal muscle rigidity, cyanosis and acute hyperthermia. Temperatures may rise above 42^0 C.

SIGNS: ON BYPASS THE TEMPERATURE RISES UNEXPECTEDLY, VENOUS O_2 SATURATIONS DECREASE AND UNEXPLAINED ACIDOSIS OCCURS.

ACTION: ADMINISTER DANTROLENE INTRAVENOUS PUSH AT A MINIMUM DOSE OF 1MG/KG UNTIL SYMPTOMS SUBSIDE OR UNTIL A MAXIMUM CUMULATIVE DOSE OF 10 MG/KG IS GIVEN. IF SYMPTOMS RECUR THE TREATMENT MAY BE REPEATED. THE PATIENT SHOULD BE COOLED ON BYPASS TO PREVENT EXCESSIVE TEMPERATURES UNTIL THE DANTROLENE TAKES EFFECT.

Cardiac Valve Surgery

The valves of the heart are located between the atria and the ventricles, between the left ventricle and the aorta and between the right ventricle pulmonary artery. The valves must work properly to allow blood to flow between these chambers. Charles Hufnagel performed the first artificial valve replacement in 1952. He placed a device consisting of small balls inside a plastic tube into a patient's aorta. The mechanical valve helped ease the work load of the patient's damaged aortic valve. Albert Star, a surgeon, and Lowell Edwards, an engineer, developed the Starr-Edwards ball-and-cage valve and used it successfully in 1960. Today's valves are of two types, mechanical and bioprosthetic. **Mechanical prosthetic valves** are made from metal and other synthetic material. These valves are very durable and often last over 20 years. Mechanical valves last much longer than bioprosthetic valves. Today, these valves are tilting disk valves and double-tilting half-disk (bileaflet) valves. Mechanical valves, although lasting longer than bioprosthetic valves, require that the patient be maintained on anticoagulants permanently. Blood will clot on the valve and cause an embolism if the patient is not kept in an anticoagulated state. The anticoagulation is done with **Coumadin.** Coumadin antagonizes vitamin K. Coagulation factors dependent on vitamin K are **II, VII, IX, and X.**

Bioprosthetic valves are of two types. Animal, usually porcine, or human tissue valves are these types. Valves from animals are termed **heterographs**, while those from human tissue are termed **homografts.** These valves have the disadvantage of not lasting as long as the mechanical valves. About 10-20% will require a new valve within 15 years. The advantage of these valves is that anticoagulation is not required. Anticoagulation carries a risk to the patient of hemorrhage and may limit activities. Homograft valves are used to replace aortic valves. These valves are more durable than heterografts, but are in limited supply.

The cardiac valve is chosen by the surgeon who considers the age, health and life-style of the patient as well as the surgical aspects of the valve. Generally, older patients receive bioprosthetic valves while younger patients receive mechanical valves.

Infection of the valves may occur well after the surgery. This is a common complication of patients receiving cardiac valves.

Evaluating Valvular Function

The most accurate method to evaluate cardiac valvular function is to measure pressure gradients across the valve. This method determines the difference of pressures on both sides of a particular valve. Mitral or tricuspid stenosis cause high atrial pressures with slow fall early in diastole. Mitral or tricuspid insufficiencies cause atrial systolic increases in pressure during

ventricular systole. Aortic stenosis displays a slow-rising aortic pulse pressure while insufficiency causes a collapsing aortic diastolic pressure.

The **echocardiogram** is a noninvasive method of accessing cardiac features. In mitral stenosis, the altered movement of the valve leaflets can be recognized. Mitral valve prolapse can also be seen on the echocardiogram as downward displacement of the leaflet or leaflets. Evaluations of the aortic valve are less helpful. In aortic stenosis the number of echoes is increased and a thickened valve can be detected. Tricuspid stenosis displays an increased diastolic slope. Evaluation of the pulmonary valve is limited to finding pulmonary hypertension and stenosis.

Valve Areas and Flows

Measurement	Value
Aortic Valve Area	2.6 - 3.5 sq cm
Aortic Valve Flow	250 ml/SEP/sec.
Mitral Area	4 - 6 sq cm
Mitral Valve Flow	150 ml/DFP/sec

Mitral Valve Surgery

Conditions that require mitral valve surgery can be classified as those that limit flow through when the valve is open and those that permit backflow when the valve is closed. These conditions are termed mitral stenosis and mitral insufficiency, respectively. The mitral valve consists of only 2 leaflets. The 2 leaflets are attached by fibrous cords called the **chordae tendineae**. These are attached to papillary muscles in the apex of the ventricle. Separate cannulations of the superior vena cava and the inferior vena cava are required to ensure proper drainage. Even if the right atrium is not opened, retractors in the left atrium will distort drainage if a two stage cannula is used.

Mitral stenosis is a narrowing of the mitral valve. This is an acquired condition usually caused by rheumatic fever. The anterior lateral and posterior medial leaflets are fused and do not open properly. The left atrium does not empty and blood backs up in the pulmonary system and right ventricle. The left atrium becomes enlarged. Compliance diminishes and dyspnea and congestive heart failure develop. Right sided heart failure occurs. Surgery is performed to allow the unimpeded flow through the valve. This is a mitral commissurotomy. The surgery leaves the mitral valve in place. The commissures are incised to separate the leaflets. The valve is replaced if the leaflets are calcified and the valve is not pliable. Mitral valve replacement requires removing the diseased valve and suturing the replacement valve to the annulus using the sewing ring that surrounds the replacement valve. Mitral valve repair is always preferred to replacement.

Mitral insufficiency is a condition of the mitral valve that allows blood to regurgitate through to the left atrium even when the valve is closed. This condition may be congenital or acquired. The left ventricle must work harder and dilation and hypertrophy result. This condition can be acquired from myocardial infarctions causing papillary muscle rupture. **Mitral valve prolapse** is when one or both leaflets protrude into the atrium when the ventricle contracts. This causes blood to flow back into the atrium. The surgery for this condition is the annuloplasty. Mitral valve annuloplasty is the placing of a ring around the mitral annulus. The ring is sutured at the base of the leaflets to tighten the annulus.

Aortic Valve Surgery

The aortic valve separates the left ventricle from the aorta. The valve consists of 3 cusps. Aortic insufficiency or regurgitation is the incomplete closure of the valve allowing blood to flow backwards into the left ventricle. Aortic stenosis is a narrowing of the aortic valve. This abnormality may be congenital with a bicuspid valve morphology. It may also be acquired as a result of senile degradation or rheumatic fever. The blood is partially blocked from flowing from the left ventricle into the aorta. This causes an overworked left ventricle, lower output and lung congestion. Pulses are diminished and a murmur is found in these patients. Chest pain and exercise intolerance are symptoms. If patients are symptomatic replacement of the valve is usually indicated. In children, reconstruction of the valve is sometimes attempted.

Venous cannulation is with a two-stage cannula. Cardioplegia must be given retrograde or through coronary perfusion cannulae into the coronary ostia once the aorta is opened. The aorta is opened to gain access to the valve for excision. The replacement valve is either a mechanical or bioprosthetic. The decision of which type valve to use is based on surgical considerations, as well as the age, health and life-style of the patient.

Tricuspid Valve Surgery

The tricuspid valve allows blood to flow from the right atrium into the right ventricle while preventing backflow. The valve consists of 3 leaflets. These 3 leaflets are described as the anterior, posterior and septal leaflets. They are strong fibrous tissue. The anterior leaflet is the largest with the posterior leaflet being the smallest. The three leaflets are attached by fibrous cords called chordae tendineae to papillary muscles in the ventricle. The tricuspid valve may be stenotic or insufficient. Mitral valve disease may cause increased pressures in the right heart causing insufficiency of the tricuspid valve.

Bicaval venous cannulation is used due to the opening of the right atrium. Tricuspid annuloplasty is, as with the mitral valve, suturing of a prosthetic ring around the valve to decrease the size of the opening. The size

of the ring is determined with a ring sizer. Tricuspid commissurotomy or valve replacement is performed in much the same manner as surgery of the mitral valve.

Pulmonary Valve Surgery

The pulmonary valve allows blood to travel from the right ventricle to the pulmonary artery. The valve is made of 3 cusps that grow from the lining of the pulmonary artery. In cases of pulmonary stenosis the procedure usually performed is the balloon valvuloplasty. This is performed in the cardiac cath lab. Surgical commissurotomy can be performed if necessary.

Pulmonic regurgitation is usually associated with other anomalies such as left heart failure, mitral valve obstruction or pulmonary vascular disease. In newborns it may be associated with a VSD. In some cases valve replacement may be indicated if the valve is deficient, however this is rare.

NOTES

Special Cases

This chapter serves as a reference for many of those special cases that the perfusionist must occasionally perform. These are the difficult cases that require extensive knowledge of the patient's disease state and a keen understanding of the perfusion strategies necessary to perform such cases. The perfusion considerations for these special cases are covered by topic in this chapter.

Surgery of the Aorta

Surgery of the aorta requires the perfusionist to have knowledge of the anatomy of the abnormality and the area affected. An **aneurysm** is an expansion of the wall of a blood vessel caused by atherosclerosis, hypertension, injury, infection, Marfan's syndrome or pregnancy. Aneurysms of the aorta may rupture causing immediate loss of systemic blood volume and pressure. The dissection may cause blockage to critical organs. In a dissection, there is a tear in the layers of the aortic wall causing blood to flow between the layers of the aorta. This tear may block or redirect flow to some of the arteries that arise from the aorta.

Ascending Aortic Arch Repair

Surgery for an ascending aortic aneurysm or dissection requires that the affected area of the aorta be clamped out, removed and repaired with a graft. Ideally, arterial blood pressure monitoring is accomplished by a left radial arterial line and a femoral line. The femoral arterial monitoring line offers a means of comparing pressures with the radial. If a dissection occurs that occludes one of the vessels coming off the aortic arch the radial pressure will be affected. The left radial artery is used because the right may be compromised by the clamp on the ascending aorta. The right radial artery arises from vessels off the innominate artery, which is often too close to the affected area. Cannulation for these procedures is accomplished by placing the arterial cannula distal to the area that is clamped out. The aorta or the femoral artery can be cannulated depending on the location of the aneurysm. Flow to the area that is clamped out will be prevented, but flow to the arch vessels is not ordinarily compromised. In cases where it is compromised, complete circulatory arrest may be required. The venous cannulation is done with either a two stage cannula in the right atrium or a single stage cannula in the superior vena cava and a single stage cannula in the inferior vena cava. The heart is protected in the usual way with retrograde cardioplegia. The various perfusion techniques required for these operations are covered later in this chapter.

Aortic Dissection

A **dissecting thoracic aortic aneurysm** is a dire, life threatening condition, that often finds the perfusionist involved in an emergency operation. These operations can be very challenging to even the most experienced perfusionist. Many variations of the perfusion circuit may be required to provide oxygenation to the various regions involved in the dissection. In addition, this problem sometimes occurs during routine cardiac surgery requiring immediate modifications to the bypass circuit.

A dissecting thoracic aortic aneurysm may present with symptoms similar to those of a myocardial infarction and is often misdiagnosed. Aortic dissections rarely occur in young people but most often occur in males between 40 to 60 years of age. The pain of this condition is severe chest pain, possibly radiating to the back. **Cerebral, coronary, renal and peripheral vessels** coming off the aorta may become obstructed. During cardiac surgery aortic cannulation, aortic cross clamping, partial clamping or removal of clamps will occasionally cause a dissection. The arterial perfusion line pressure may rise immediately and then possibly fall as the dissection extends.

Physical findings associated with a thoracic aortic dissection are acute neurological deficit, difference in blood pressure in extremities, limb ischemia, new aortic insufficiency murmur and signs of pericardial tamponade. The chest x-ray displays a widened mediastinum, pleural effusion, and mainstem bronchus or esophagus displacement and an enlarged aortic silhouette beyond intimal calcification. Aortography is the definitive evaluation of this condition. CT scan with IV contrast does not offer great detail. MRI and transesophageal echocardiography are rapidly gaining acceptance as definitive tests.

Debakey Classification of Thoracic Aortic Dissections

Type I: Begins in the ascending aorta just above the aortic valve and extends into the descending aorta.

Type II: Involves the ascending aorta only.

Type III: Originates distal to the left subclavian artery and involves the descending aorta only.

Arterial pressure monitoring is ideally accomplished with right and left radial lines, in addition to a femoral line. Comparison of the radial lines with the femoral is a good indicator of the perfusion of the cerebral region. If the right radial pressure is decreased significantly, then the right common carotid artery may not be receiving required flow. This is because the right radial artery flows from a vessel off the innominate artery. The left radial artery branches off the left subclavian artery, which connects directly to the aorta.

A **cerebral oximeter** can be used to monitor the oxygen saturation in both sides of the brain. This device can provide the perfusionist with an indirect assessment of the cerebral blood flow. This technique is particularly useful during retrograde cerebral perfusion (RCP), as described below.

The **major perfusion question** concerning surgery for dissecting thoracic aortic aneurysms and aortic arch surgery is whether the vessels coming off the aortic arch will be perfused. If the surgery does not require clamping out this area, then the question is whether the dissection or other abnormality will allow blood to flow into these vessels. Surgery that does not allow flow to these vessels can be managed in several ways. Aortic cannulation is accomplished through either femoral artery, while venous cannulation is through either a large two stage cannula or two single stage venous cannulae. Two single stage cannulae permit slow retrograde perfusion through the superior vena cava (SVC) cannula to supply the cerebral region.

Retrograde cerebral perfusion (RCP) of the brain is a technique used by some to provide some oxygenation to the cerebral region. Flow rate is slow, less than 500 ml/min and pressures monitored at the SVC are kept below 25 mmHg. This flow can be continuous or intermittent. There are several circuit designs that allow this type of perfusion. The SVC cannula can be connected to a line that is Y connected from the arterial tubing. The arterial tubing is then clamped distal to the Y connection. Another circuit uses a bridge between the arterial and venous lines with a clamp placed on the arterial line. Oxygenated blood then crosses over the bridge and travels the venous line to the SVC cannula. Another method is to place a retrograde cardioplegia catheter up the SVC, with the balloon inflated and the SVC clamped below the cannula. The SVC pressure is conveniently measured via the retrograde catheter, and the blood is administered in the normal fashion.

Direct cannulation of the innominate, left common carotid and left subclavian arteries is another technique also performed. This method of **antegrade cerebral perfusion** is accomplished by connecting three lengths of quarter inch tubing to Y connectors coming off the arterial tubing. The amount of flow to these vessels is autoregulated by these vessels. A centrifugal pump is excellent for this type of procedure. Most of the flow is directed to the femoral cannula with, ideally, the cerebral vessels acquiring what they need from the flow.

Circulatory arrest is another means of allowing the surgeon to work on this area of the aorta (see section later in this chapter). The bloodless field allows the surgeon to repair or replace the aneurysm. The area is clamped out and the affected area removed. An aortic graft is sutured to the distal aorta. A portion of the superior aspect of the graft is removed to allow attachment of the head vessels. At this site a longitudinal strip of the aorta with the head vessels is removed from the aorta and sutured into the graft. The graft can then be clamped on the proximal end, air removed, distal clamp removed and flow

established to the head vessels. The proximal end of the graft is sutured, completing the procedure.

Descending Thoracic and Thoracoabdominal Aneurysms

These cases are often simpler for the perfusionist due to the location of the aneurysm. These aneurysms are located below the left subclavian artery origin, and flow to the cerebral region is not compromised. Thoracoabdominal aneurysms usually require repair if larger than 6 cm due to an increasing risk of rupture. CT scan and/or aortography confirms the diagnosis. These operations are performed using the clamp and go method and CPB is usually not employed. Some surgeons clamp out the affected aorta and replace it with a graft without perfusing the lower regions distal to the clamp. They quickly perform the surgery and restore flow to the lower region. This surgical technique has been performed for many years with excellent results, however, complications may occur. Using this method the kidneys, liver and gastrointestinal regions are not perfused. In addition the left ventricle must eject against a clamped aorta. This increases afterload of the left ventricle. (Blood left in the ventricle after ejection.) A complication termed **anterior spinal artery syndrome** sometimes results. This syndrome is manifested by paraplegia and sphincter disturbances. The blood flow to the spinal cord is through the anterior spinal artery and two posterior spinal arteries. The anterior spinal artery supplies the lower two thirds of the spinal cord. The anterior radicular artery also contributes to the blood supply of the anterior spinal artery. Neurologic deficits may occur if these vessels are occluded for long periods of time. Safe clamping times are considered by some authorities to be as short as 20 minutes, while others feel that times as long as 60 minutes may be tolerated. As in all cases of absent flow to an area of the body, the shorter the period of no flow, the fewer the complications that occur.

A flexible tube called a Gott shunt may be used to direct blood around a clamped out area of the aorta. The shunt is attached above the superior clamp and below the distal clamp. The blood is propelled by the heart's pumping around the area.

The use of a centrifugal pump and perfusion around the clamped area provides controlled flow. This can be accomplished by cannulating the left atrial appendage to remove some of the blood flowing through this chamber. The blood is then returned below the clamped out area through the femoral artery. Little heparinization is required for this technique and doses of 100 units per kg are adequate. Use of bioactive surfaced circuits and cannulae can reduce this even more. ACTs are kept around 200 seconds. Flow of about 75% of full calculated flow is maintained and the patient is kept warm with a warming blanket or by a heat exchanger incorporated into the perfusion circuit. Arterial pressures monitored below the distal clamp should be kept above 60 mmHg to ensure distal perfusion. Radial pressure monitoring above the proximal clamp

is also used to maintain arterial pressures in the upper region. This type of bypass is referred to as **left heart bypass** and may have better results than the *clamp and go* method.

Circulatory Arrest

Some types of surgeries or complications may require a period of circulatory arrest. The perfusionist should prepare the patient for the circulatory arrest period. In anticipation of the acidosis of the arrest period, as the patient is cooled, the patient is brought into an alkalotic state with a low pCO_2 and the addition of sodium bicarbonate. The patient is cooled to deep hypothermia. Colder patient temperatures allow longer arrest periods. The table below shows temperatures and times generally regarded as tolerable. It is accepted that circulatory arrest causes uncertain sequelae and shorter periods of arrest are desired. The surgeon attempts to limit these periods and should be informed of the arrest time periodically. Cardioplegia is given antegrade and retrograde. Circulatory arrest is then initiated. Temperatures of **15 degrees C** and below are thought to cause cessation of the brain's electrical activity and provide optimal protection. Packing the head in ice is thought to limit cerebral activity (especially in pediatric patients) and is used by some institutions. If the head is packed in ice, care should be taken to protect the ears from frostbite. The head down, Trendelenburg, position is used to prevent air from traveling up the cerebral vessels. After the pump is stopped, the blood is drained from the patient into the venous reservoir by leaving the venous line open. Some research shows that hyperglycemia in circulatory arrest patients may cause damage to the central nervous system. Therefore, insulin should be given if blood glucose levels rise too high prior to circulatory arrest. Sodium Pentothal given before the circulatory arrest causes the brain's electrical activity to decrease. Forane also exhibits a barbiturate effect on the brain. EEG monitoring can be used to indicate cessation of cerebral activity.

The blood in the oxygenator and portions of the circuit can be circulated if an A-V bridge is incorporated into the circuit. The blood can then be warmed slightly and oxygenated before the circulatory arrest ends. A temperature gradient less than 8 degrees should be maintained, when rewarming. More oxygen is held in solution in cold blood, and warming the patient too rapidly may cause microbubbles.

Classification	Temperature
Mild	**32 - 37° C**
Moderate	**28 - 31° C**
Deep	**18 - 28° C**
Profound	**0 - 18° C**

The following circulatory arrest times are considered tolerable by most surgeons. The perfusionist should be aware that all circulatory arrest is detrimental to some degree and should notify the surgeon of the arrest time at regular intervals.

Patient Temperature	Arrest Period
32° C	under 10 min
28° C	10 - 15 min
18° C	16 - 45 min
<18° C	46 - 60 min

Pulmonary Embolism

A **pulmonary embolism (PE)** may result in circulation to a lung being stopped or compromised. Massive pulmonary embolisms cause death in over two thirds of affected individuals. The embolism may be a blood clot, fat, air or debris. Conditions of decreased blood flow such as major surgery, CPB, abnormal or damaged blood vessel walls, childbirth or cardiac dysfunction can all cause embolisms. The patient experiences anxiety, difficulty breathing, chest pain and cyanosis. Distended neck veins, fever, cardiac arrhythmias and hemoptysis may occur. The formation of clot in a lung leads to rapid pulmonary infarction. Diagnosis is accomplished with radioisotope perfusion lung scans and pulmonary arteriography. Thrombolytic therapy is beneficial in these patients. A **pulmonary embolectomy** may be necessary in rapidly deteriorating patients.

There are criteria to determine which patients should have emergency surgery to remove the embolus. Arterial pO_2 less than 60 mm Hg, systolic blood pressure less than 90 mm Hg and urine output less than 20 cc an hour are indicators that surgical removal is necessary. An alternative approach is to use a catheter to remove the clot from the pulmonary artery. Surgery should be performed as early as possible to prevent irreversible damage. This condition has a 100% mortality rate if untreated. The mortality from this surgery is 70%. Once the pathological processes have begun it is difficult to reverse them even though the clot is removed. Femoral-femoral cannulation may be required due to the emergent nature of the surgery. If time permits, two single stage venous cannulae should be used for bicaval cannulation with the usual aortic cannulation. Heparinization is performed although prior use of thrombolytics may complicate hemostasis management postoperatively. The embolus is then extracted from the pulmonary artery on CPB.

Pregnancy

The pregnant patient requires special consideration by the perfusionist. The perfusionist not only manages the mother while on bypass but also manages the unborn fetus. Pregnancy lasts about 38 weeks from conception to birth, although it may be 40 weeks from the first day of the last menstrual period. The placenta is formed from parts of the woman and the embryo. The placenta is responsible for providing nutrients to the fetus and for removing waste products. However, the blood of the mother does not cross the placental barrier to mix with the that of the fetus. The mother experiences numerous physiologic changes during pregnancy. These changes often unmask preexisting cardiac problems such as congenital defects. **Valvular disorders are the most common problems** that occur during pregnancy.

The heart rate increases to a rate of about 80 to 90 BPM. The cardiac output increases 30 to 50 percent. This increased output begins as early as the 6th week and increases until the 16th week. At the end of pregnancy, about 20% of the total cardiac output is sent to the uterus. At around the 30th week, the output begins to decrease. About 6 weeks after birth, the cardiac output is back to normal. The blood pressure falls slightly after the 12th week and returns to normal after the 26th week.

The pulmonary changes reflect a higher respiratory rate with decreased lung reserves. The vital capacity remains the same. Renal blood flow is increased. The blood passing through the kidneys for filtration is increased 30 to 50 percent. Due to pressure from the uterus on major vessels and the ureters, the kidney function is better when the mother is lying on her side.

CPB is statistically safe for the mother, but severe fetal injury and death are as high as 50%. Therefore, surgery is only performed when the condition is life-threatening. The **second trimester** is the safest period for the mother and the fetus for the surgery to be performed. The first trimester is dangerous because the embryo is easily injured by hypoxemia, drugs and other factors. The last trimester is dangerous for the mother and fetus because premature labor may occur. Also during this time, the blood volume of the fetus and the output directed to the uterus is at its greatest.

Heparinization is possible because heparin does not cross the placental barrier. Coumadin does cross the placental barrier and should be stopped when pregnancy is discovered. Patients taking Coumadin before pregnancy must be switched to heparin during pregnancy. If mitral valve replacement is necessary, porcine valves that do not require anticoagulants are used.

The patient is prepared preoperatively by having her turned slightly to the left side on the OR table and elevating the right flank to decompress the vena cava. A fetal heart rate monitor is setup in the room to detect fetal distress. The blood volume of the pregnant patient is 90-100 ml/kg. Adequate venous reservoir capacity should be present for the extra volume. The hematocrit of the patient is kept in a normal CPB range of 22-25 percent. The

patient is not cooled because the fetal heart rate would decrease. In addition, uterine contractions may be precipitated by rewarming. CPB flows are maintained at a high rate. A cardiac index of 3 to 3.2 L/min/M sq may be necessary to ensure the fetal heart beat is kept above 60 beats per minute. A rate **less than 60 beats per minute** places the fetus in danger. The mother's blood pressure is kept above 65 mmHg to provide optimal flow to the uterus. **Epinephrine** can be used to increase the blood pressure during CPB. Epinephrine, at the doses used on bypass, is a beta stimulator and does not affect uterine blood flow. Neosynephrine and levarterenol should not be used. These drugs are alpha agonists and can compromise blood flow to the uterus. Hypertension should be treated with hydralazine, which does not compromise uterine blood flow. Sodium nitroprusside is not used because of the cyanide toxicity possible and its effect of the fetus. The increased oxygen consumption of pregnancy may make increased oxygen concentrations and increased gas flow necessary.

Cardioplegia should not be used excessively due to the possible effects on the heart of the fetus. Retrograde infusion of the cardioplegia is best with venting of the aorta to a separate, clamped out cardiotomy or waste bag. This method of removing the cardioplegia prevents it from being returned to the blood that supplies the uterus.

Cold Agglutinins

(Editor's note: This section is also found in the chapter blood disorders. It is repeated here because the authors feel that this topic is critical and not generally well understood by perfusionists.)

Cold agglutinins or cryoproteins are serum antibodies that work on the antigens found on the surface of red blood cells. They may cause complement activation and red blood cells to clump at low temperatures and break down. This process is classified as an **autoimmune reaction**, and does not occur if the patient is kept warm. This disorder occurs primarily in patients over 50 years of age, but is also found in others. These antibodies are usually the IgM class in cases where the **agglutination** is significant. IgG and IgA antibodies may be involved in cases that are clinically insignificant. Tests can be performed to find the presence of cold agglutinins and at what temperature agglutination will be precipitated. Cold agglutinins can be caused by an infection such as a virus. The incidences of positive cold agglutinins may resolve with time, and any elective cardiac surgery should be delayed if cooling of the patient or cold cardioplegia will be necessary. The level of the cold agglutinins at certain temperatures can also be determined with titers.

The technique used to perfuse these patients involves keeping the blood at temperatures warm enough that agglutination is not likely to occur. Do not cool these patients below the identified temperature that may cause

agglutination. If it is known that the patient must be cooled to temperatures that may cause a reaction, **plasmapheresis** is necessary. Many of the serum antibodies may be removed in this manner. Patients with severe cold agglutinins may have severe sequelae if the usual hypothermic bypass techniques are used. Perioperative myocardial infarctions, renal failure, hemolytic anemia and thrombosis may be the unfortunate results. The team members should all be aware of the potential problem and devise an appropriate strategy to keep the patient warm. There is some evidence that the dilutional effect of the pump prime may provide a measure of safety in preventing the process, but this is not predictable enough to be of practical value. The patient should be kept warm before, during and after bypass. A heating blanket on the OR table is beneficial. Normothermic bypass with a warm prime should be used. Packed RBCs should be warmed if added to the pump during the case. If cold cardioplegia is desired, a plain crystalloid solution is used. The cardioplegia solution should be initiated warm to flush the coronary arteries of blood and then switched over to cold after 200-300 ml. The cold temperature at which cardioplegia is ordinarily delivered will almost certainly cause agglutination if blood is introduced to this solution. Blood cardioplegia must be given warm. A warm infusion of cardioplegia just before the crossclamp is removed will provide an additional measure of safety for the blood that will be entering the coronary arteries.

Hypothermia

Hypoyhermia is a life-threatening problem caused by exposure to cold weather or immersion in cold water. Respirations are decreased or possibly absent. Pulses are faint and the patient often appears lifeless. The severity of the hypothermia influences greatly the chances of recovery. The age and preexisting health of the patient are also important factors. There have been cases of cardiopulmonary bypass being used to successfully warm patients experiencing hypothermia, but these patients all exhibited a core temperature above 16 degrees C.

A standard bypass circuit is utilized for these emergencies. Cannulation is via femoral artery and femoral vein, and full heparinization is required. An oxygenator with a heat exchanger is used because these patients often have lung aspiration injuries. Also, the patient may be in an acidotic state, and the oxygenator is helpful in correcting respiratory acidosis. An ultrafiltrator is used to control volume status and because of the possibility of ensuing renal failure. Because these patients often exhibit diminished circulation, there is a danger of the heparin not being delivered systemically before bypass is instituted. Half of the heparin should be circulated in the pump prime if the ability of the patient to circulate the heparin is in question. The temperature of the priming solution should be near the patient's core temperature when bypass is initiated. Then the patient is slowly rewarmed at a rate of about **4 degrees an**

hour. This rate ensures uniform rewarming and gives the patient adequate recovery time. Acidosis is corrected and urine output stimulated if possible. The mean arterial blood pressure is maintained at around 60 mmHg. If the heart fibrillates, cardioversion should be attempted when the core temperature has reached 32 degrees C.

The Jehovah's Witness Patient

The goal when performing CPB on a Jehovah's Witness patient or any patient who does not want blood products is blood conservation. Strict blood conservation techniques are essential. Autotransfusion, ultrafiltration and return of the chest tube output are all measures that can be employed. Minimizing the pump circuit and priming volume are of paramount importance. The patient should be asked what measures he or she will allow. These individuals may have different beliefs and interpretations of their religion. Most will allow CPB but do not want homologous blood to be infused. Others may not want autologous blood to be reinfused. The perfusionist should take steps to insure that all blood is returned to the patient in a *continuous loop* (much like the CPB circuit). This may entail connecting the cell saver or chest tube return line directly to an IV site for direct infusion. Hespan or dextran may be given as plasma expanders, but albumin is not allowed by some adherents. Patients with low hematocrits preoperatively may be impossible to operate on at that time. Infants and children usually require blood to be given and legal measures may be necessary before their surgery is attempted. In emergencies, telephone court orders may be obtained.

Isolated Limb Perfusion

An oxygenator and perfusion circuit allow an arm or leg to be isolated with a tourniquet from the systemic circulation. This permits cancers confined to the limb to be treated with drugs that would be harmful if released into the systemic circulation. The oxygenator provides oxygen to the limb tissues that are separated from the circulation during this treatment. **Melanoma** is the cancer most often treated with this technique. **Sarcomas** and other malignancies that are confined to the limb are also treated with this method. Chemotherapy drug doses that are 10 times as strong as those that would be possible with systemic administration can be used. The heat exchanger of the oxygenator is also beneficial because the efficacy of the drugs is increased with hyperthermia.

Circuits

A pediatric circuit and oxygenator are best used for this type of perfusion. The area to be perfused is relatively small, and hemodilution can be kept to a minimum. Arm perfusion is performed with a circuit that uses an arterial tubing size of 1/4 inch and a venous tubing size of 1/4 inch. Leg perfusion requires 1/4 inch arterial and 3/8 inch venous circuits. Pediatric filters that can handle the required flow can be used with this circuit. One of the procedures necessary is the removal of the blood in the limb with the chemotherapy drugs at the end of the perfusion. This requires a Y-connector on the venous line with tubing and waste bag connected to drain off this blood. A separate cardiotomy with solutions that are to be added to replace the removed blood is also included. Either a centrifugal or roller pump can be used. Perfusion pressures are monitored, much as with traditional perfusion, to determine resistance in the limb.

Priming Solution

Determination of the portion of the body an arm or leg accounts for is made with the *Rule of Nines* method. This method lists the arm as 9% body surface area while the leg is listed as 18%. Circulating volume compositions of the limb to be perfused are determined using these percentages of the whole body blood volume. The priming solution can be any balanced electrolyte solution such as Ringer's with sodium bicarbonate added to increase the pH, making the solution less acidotic. A sodium bicarbonate dose of 25 mEq per liter is sufficient. Heparin (5,000 units) is added to the prime also.

Standard volume-concentration calculations are used to determine the hematocrit of the limb after the dilution of the prime. These calculations are also used to determine the whole body hematocrit after the blood of the limb is removed at the end of the case. Ideally, the hematocrit of the limb during the

case should be **kept above 15%.** These are the steps involved in deciding whether to add blood to the priming solution.

1. Determine the red blood cell volume of the patient.
2. Determine the red blood cell volume of the limb.
3. Determine the dilutional effects of the prime on the limb.

Circulating blood volume = 70 ml x weight in kg
Red blood cell volume = circulating blood volume x hematocrit
Limb circulating blood volume = circulating blood volume x limb percentage
Limb red blood cell volume = total red blood cell volume x limb percentage

After these calculations are made the dilutional effects of the priming solution can be determined.

Total circulating volume = limb circulating blood + prime volume
Hematocrit of circulating volume = Limb RBC volume / circulating volume

The worksheet below uses the following abbreviations.
CBV = circulating blood volume
RBCV = red blood cell volume
TCV = total circulating volume

CBV = *weight in kg* x 70 ml

RBCV = *CBV* x *hematocrit in decimal form*

Limb CBV = *CBV* x *percentage of the limb in decimal form*

Limb RBCV = *RBCV* x *percentage of the limb in decimal form*

TCV = *priming volume* + *limb CBV*

Hct of TCV = limb RBCV / TCV

The ml of packed red blood cells to be added to the prime can then be determined. (The hematocrit of packed red blood cells is about 70%.)

RBCs required = *TCV* x *desired hematocrit in percentage form*

RBCs to add = *RBCs required* - *limb RBCV*

ml of packed RBCs to add = *RBCs to add* / .70

Conduct of Isolated Limb Perfusion (ILP)

The patient is fully heparinized before the perfusion begins. If not and there is a leak across the tourniquet the blood may start to clot in the circuit due to dilution of the heparin. Therefore, full heparinization is performed. Heparin doses of 300 units per kg are given and adequate ACTs obtained before bypass is initiated. The **Esmarch tourniquet with a Steinman pin** is tightened to stop circulation into the limb before starting the perfusion, also. The patient is monitored with arterial pressure lines, EKG, oxygen saturations and CVP line. The patient's circulation is separate if there is no leak across the tourniquet and calculations such as cardiac output may be obtained. Pump flows will not affect pressures across the tourniquet.

The pump flow rate (PFR) is found by multiplying the patient's cardiac output by the limb percentage in decimal form (.09 or .18). Using this formula a patient with a cardiac output of 4.5 LPM would have a calculated pump flow of 405 ml per minute for an arm and 810 ml per minute for a leg.

PF R= Limb % x CO

Pump resistance (PR) is determined by multiplying the limb perfusion pressure by a conversion factor of 80 and then dividing the result by the pump flow rate.

PR = (LPP x 80) / PF

Test transfusions of a small amount of volume are done after cannulation to ensure proper placement of the cannula. The perfusion is then started and slowly brought to the full calculated flow. High oxygen concentrations are detrimental to cancer cells and the FIO_2 is maintained at 80% or higher. Blood gases of the perfused limb are obtained and the arterial pO_2 is kept above 400 mmHg. The limbs are kept warm to maximize the effectiveness of the drugs. The limbs can be warmed by the oxygenator's heat exchanger and use of a warming blanket. The temperature of the limb is measured by temperature probes. Four probes are used with insertions in the proximal and distal portions of the medial and lateral aspects of the limb. The temperature is kept at 38 to 40 degrees C.

Leak Detection

The high concentration of drugs used must be kept from the systemic circulation. Leakage across the tourniquet must be found and stopped. The tourniquet is applied around the limb over a single bone, the humerus or femur, to accomplish complete isolation. Isolation around two bones, ulnar-radius, tibia-fibula, is usually ineffective. The volume in the oxygenator should be observed closely for reduced or increased volume. Changes in volume indicate leaks. If the leak goes both ways across the tourniquet, the volume in the

oxygenator may not change. Therefore, these leaks are not easily found. Leaks into the systemic circulation are dangerous and reduction in volume indicates this is occurring. The possibility of this type of leak can be minimized by keeping the **perfusion pressure below the patient's diastolic pressure**. Adjusting the pump flow and giving nitroprusside to the limb circulation can accomplish this. Leaks that cannot be stopped using this method must be dealt with by repositioning the tourniquet or the cannula.

Leakage into the limb is not as serious because the drugs are staying in the limb. The volume in the oxygenator will rise in this situation. These leaks are tolerated if the total leakage is less than 500 ml. Dilution of the drugs circulating in the limb is the major problem with these leaks. Increasing the flows may stop this leak by raising the pressure on the limb side of the tourniquet.

Leaks can be verified by adding fluorescein dye to the oxygenator and checking above and below the limb with a black lamp with the room lights turned down. Any dye above the tourniquet indicates leakage and must be stopped before the chemotherapy drugs are added. Radioactive tracers are used by some teams to find leaks.

Drugs Used

There are two types of drugs used for isolated limb perfusion. These agents are classified as **alkylation or natural agents**. The oncologist in charge of the treatments decides what drugs are best and designs a treatment plan. These drugs are very dangerous and must be handled carefully and disposed of in the proper manner. The most commonly used alkylating agents are derived from nitrogen mustards and triazenes. These agents interfere with the growth of the abnormally dividing cancerous cells.

Alkylating Agents

Drug	Dose
Mustargen	0.4 - 0.8 mg/kg
Melphalan (L-PAM)	0.5 - 1.5 mg/kg
DTIC-Dome	500 - 2000 mg per meter squared of treated BSA

Natural Agents

Drug	Dose
Cosmegen (Actinomycin D)	0.006 - 0.014 mg/kg

The drugs are given as a bolus over 1 minute every 15 to 20 minutes of the perfusion run which lasts about 1.5 hours. They are infused by way of a manifold stopcock through the arterial filter. This ensures the drugs reach the limb before dilution occurs.

Removal of Circulating Drugs

The blood being circulated with the drugs added must be removed before the tourniquet is removed and perfusion is terminated. The patient's hematocrit is going to be reduced by either 9 or 18 percent when this blood is removed and it may be necessary to add blood. The blood with the drugs is slowly pumped out into a waste bag Y connected off the venous return line. This is done slowly as crystalloid replacement is added to the pump circuit to replace this lost volume. The replacement is continued until the return is clear. This takes about 1500 to 3000 ml of solution.

The termination of the perfusion is done by slowly turning down the arterial flow while occluding the venous line as in ordinary CPB. Very little filling is required. The perfusion line pressure can be observed to guard against overfilling. The tourniquet is removed and then the pump is stopped with the venous line totally occluded. The systemic heparinizat must be reversed with protamine.

NOTES

Intraaortic Balloon Pumping

An almost routine piece of equipment seen in the open heart suite and catherization laboratories is the **intraaortic balloon pump (IABP)**. The IABP console is used to operate the **intraaortic balloon (IAB)**. These pumps and the balloons they drive have become very sophisticated and reliable, while at the same time they have become more user friendly. **Intraaortic balloon counterpulsation (IABC)** is now used more readily than it once was. This is because of a better understanding of its importance, but also because of its ease of use. Today's equipment is much better than that of just a few years ago.

Knowing the indications and contraindications of IABC are essential skills each perfusionist should possess. One must also understand the possible complications. This chapter will help build those skills.

The physician is the one who decides to use IABC. This may be the cardiologist or the surgeon. The perfusionist must be prepared to provide input to the decision if needed. The following are indications for IABC.

Indications:

unstable angina
acute myocardial infarction
cardiogenic shock
mechanical complications following MI
adjunct to PTCA
adjunct to cardiac catheterization
bridge to cardiac transplantation
operative (pre, intra, and post) support

The contraindications are generally considered as absolute or relative. Absolute contraindications are exactly that, absolute. Relative contraindications are those which should be strongly considered but do not necessarily prevent the use of IABC.

Contraindications:

ABSOLUTE

thoracic or abdominal aortic aneurysm
occluded aorta

RELATIVE

aortic insufficiency
severe peripheral vascular disease
disease not amenable to definitive therapy

Complications associated with IABC are as follows:

Complications:
limb ischemia
thromboembolism
aortic dissection
vascular injury (laceration, false aneurysm, hematoma)
infection
balloon rupture
thrombocytopenia

Techniques of Insertion

The two most common methods are percutaneous and surgical cutdown to the femoral artery. Other methods are available when these two do not allow for insertion. Intraoperatively, insertion through the ascending aorta is possible. Other possibilities include subclavian and transaxillary insertion.

Percutaneous insertion is the most common method used today. With the changes in the IABs, this method can now be performed either with a wire guided balloon through a sheath or by a sheathless technique. The technique used depends first on the IAB and if it may be used sheathless, and then on the physician's preference. The sheathless technique has the advantage of creating a smaller entrance into the femoral artery. It is also of benefit in the smaller patient where the sheath may totally occlude the femoral artery.

Femoral artery cutdown provides visualization of the artery. The balloon may then be inserted directly into the artery. This can be done either through a Dacron graft, a percutaneous sheath or directly into the artery without either.

Preparation

The preparation of an IAB is quite simple, but must be done very precisely. The brand of the IAB may effect its preparation, although most balloons are prepared in the same manner. The perfusionist should be aware of the these differences and specific capabilities of the different balloons on the market.

After the decision to use an IAB is made, the first step is to prepare the patient. If you are **not placing the IAB intraoperatively,** then the insertion site must be **cleansed with an antiseptic solution, anesthetized locally with lidocaine and heparin given (usually 5,000 units IV).**

The next step is to prep the balloon. This is usually done by the nursing personnel scrubbed in at the sterile field, but the perfusionist is often asked for assistance or advice. Therefore, it is important that they know the procedure and any special techniques with certain balloons. First, ensure that the balloon has **negative pressure** applied with a **one-way valve** in place, **before removing it from the holder**. If the balloon has an intraluminal stylus, this must be removed before the balloon can be placed over the guide wire.

The IABP must be prepared for operation before connecting the IAB. The first item is power. **Always plug the pump into an electrical outlet.** This will ensure that the batteries are not drained before transporting. Any time the patient is not being transported the pump should be plugged in.

Turn the helium on and ensure that the tank has an adequate supply to operate safely. Refer to the operator's manual of the IABP to determine this amount.

The next step is to **obtain an EKG signal.** This can be done by either attaching leads to the patient, or slaving a signal from the monitor. Some institutions connect leads or an EKG back pad to all patients preoperatively to be used in case IABPC is required. The cable is run along the side of the operating table and is ready to be plugged into the IABP when needed.

Next, **setup and prime the transducer.** An easy method used to maintain sterility is to pass the sterile transducer and its monitor line to the sterile field. The transducer and one end of the monitor line are passed off the field, while the end that attaches to the IAB is kept on the field and sterile. Now, it may be attached to the IABP, primed and zeroed, while the end that attaches to the IAB remains sterile on the operative field.

Once the EKG signal is acquired and the transducer is connected, zeroed, and the monitor line flushed, the IABP is ready to connect to the IAB that has been placed in the patient. The **gas line** is connected to the balloon (after the one way valve is removed) and then the other end passed off the sterile field to be connected to the IABP. The balloon is purged and filled with helium. The pressure monitor line can be connected to the IAB. With the balloon in place, the pump connected and ready to operate, IABC can now begin.

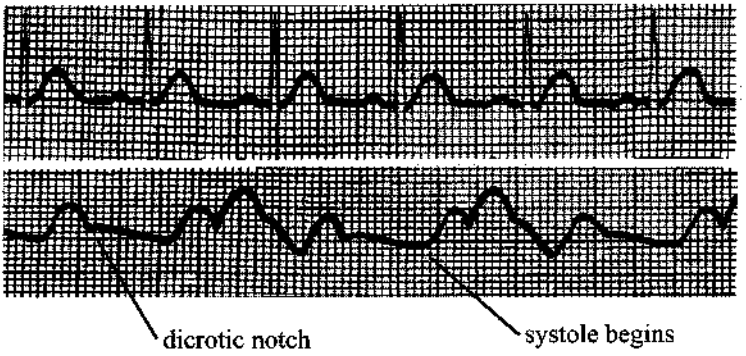
Timing and Trigger

Timing and trigger are terms often confused when working with IABPs. It is important that the operator understands these terms.

Timing - the relationship between the balloon's inflation and deflation, and the heart's systole and diastole.

Trigger - the events or signals, whether from the arterial pressure waveform, the EKG waveform, or the internal rate, which are used to institute inflation and deflation of the balloon.

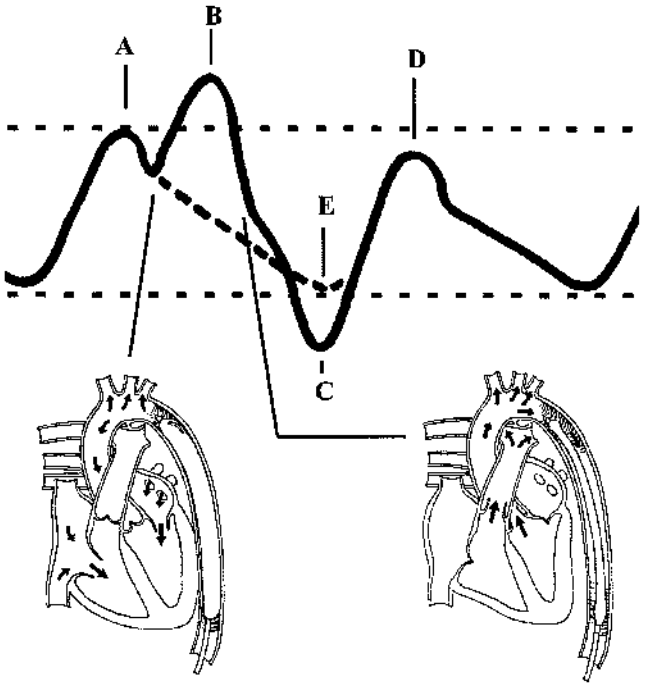
The only method of determining proper timing is to evaluate the arterial waveform and its augmentation due to the IABC. The following is an example of an EKG and its relationship to the associated arterial pressure waveform with 1:2 augmentation. The association between the electrical activity of the heart and the mechanical activity of the heart (to include the augmentation) may be seen through these strips.



Effects

The effect of the IABC is twofold (as represented on the following page). Balloon inflation occurs at the closure of the aortic valve, which is represented by the **dicrotic notch.** During inflation there is an increase in diastolic pressure due to the displacement of volume. This **increased diastolic pressure** leads to **increased coronary perfusion.**

When deflation occurs, just prior to systole, there is a sudden decrease of pressure within the aorta that causes a **decreased aortic end diastolic pressure.** This decreased end diastolic pressure equals **decreased afterload.** Afterload reduction causes a reduction in **myocardial work, oxygen consumption** and an **increase in cardiac output.**



A - unassisted systolic pressure
B - assisted diastolic augmented pressure
C - assisted aortic end diastolic pressure
D - assisted systolic pressure
E - unassisted aortic end diastolic pressure

To ensure proper augmentation the IABs inflation and deflation must be properly timed to the heart's mechanical actions. This is best done by using the EKG as the trigger. There are two basic types of timing used today by the IABP manufacturers. They are conventional and real timing. These names come from the manufacturers and should not imply that one is better than the other.

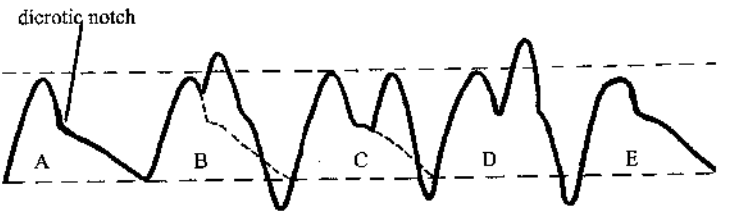
Conventional timing is the most commonly used type of timing. It is based on duration of **inflation during diastole.** The pump computer learns the R to R interval of the EKG or the peak to peak interval of the arterial pressure wave. It also memorizes the relation of the inflation and deflation to this interval. Obviously, the best timing will occur if the R to R interval is regular. Arrythmias that cause irregular intervals may make it difficult for the pump to follow. Advances in technology have increased some pumps ability to follow these irregular rates.

Real timing is based on balloon **deflation corresponding to systole.** This requires a different technology. The delay between the QRS complex and the aortic valve opening is called the preejection period (PEP). This interval remains relatively fixed despite changes in rates and rhythms. The ejection time (ET) is that period from the opening until the closing of the aortic valve, and is also relatively constant. Ventricular diastole is the remaining period, and is where changes with irregular rates and rhythms occur. Since real timing uses these constant time intervals, the irregular diastolic intervals caused by arrhythmias do not affect its ability to augment. Duration of balloon inflation is increased with real timing, thus potentially increasing the augmentation.

Inflation

Inflation, with either system of timing, should occur at the **dicrotic notch,** the closing of the aortic valve. It takes approximately 25 msec from the closure of the aortic valve for the event to reach the subclavian artery. It also takes 25 msec for the balloon pressure to reach the aortic root. This total delay is approximately equal to the time of one small block (40 msec) on standard EKG paper at a speed of 25 mm/sec. Therefore timing inflation one block to the left of the dicrotic notch would provide proper timing.

Should inflation occur too early, it would cause resistance to systolic ejection with a greatly increased afterload. This will cause premature closure of the aortic valve, reduction of left ventricle emptying, reduction of cardiac output and increased cardiac workload and oxygen consumption. If it occurs late, then the inflation would be late in the diastolic portion of the cardiac cycle which limits the amount of augmentation that can be achieved and reduces coronary perfusion.

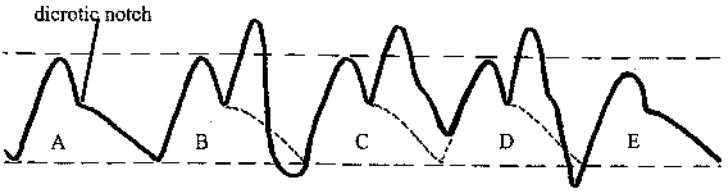


A- normal unassisted waveform, B- early inflation, C- late inflation
D- proper timing, E - assisted systole

Deflation

Deflation, when using **conventional timing,** is set to occur at the end of diastole during the isovolumetric contraction. This would be just prior to the upstroke of the arterial waveform, or systole. Determination of proper deflation timing is done by evaluating the assisted aortic end diastolic pressure to ensure that it is less than the unassisted aortic end diastolic pressure. Also, the systole following augmentation (assisted systole) should be less than the systole prior to the augmentation. **Real timing deflation** occurs at each QRS complex. The assisted aortic end diastolic pressure should also be lower than the unassisted. Since inflation lasts until the QRS complex the decrease in end diastolic pressure may not appear as large as with conventional timing.

Early deflation will provide a lesser level of reduction in end diastolic pressure, also afterload reduction is decreased and there may be retrograde flow in the aorta which can cause angina. Late deflation causes the balloon to remain inflated into the next systolic ejection. This causes the heart to work much harder. Myocardial work and oxygen consumption are increased. Cardiac output is decreased. This is, of course, detrimental to the patient.



A - normal unassisted waveform, B- early deflation, C- late deflation
D - proper timing, E- assisted systole

Most IABPs today have an automatic mode which will begin IABC with proper timing. Even in the automatic mode, the balloon should be started at 1:2 to evaluate the arterial waveform. The timing may be adjusted while in the automatic mode.

Incorrect timing may cause several problems which may be resolved by a simple correction. Proper timing is obtained by adjusting the inflation and deflation points and evaluating the arterial waveform to ensure proper timing.

Maintaining IABC

Once a patient is on IABC, there are different protocols which may be used to determine how the patient should be maintained and when he should be weaned. Although the decision of which protocol to follow is that of the physician, the perfusionist should be aware of their basic procedures.

When placing a patient on IABC, most agree a bolus of heparin should be given. A dose of 5,000 units IV is usually given at the time of the sheath insertion.

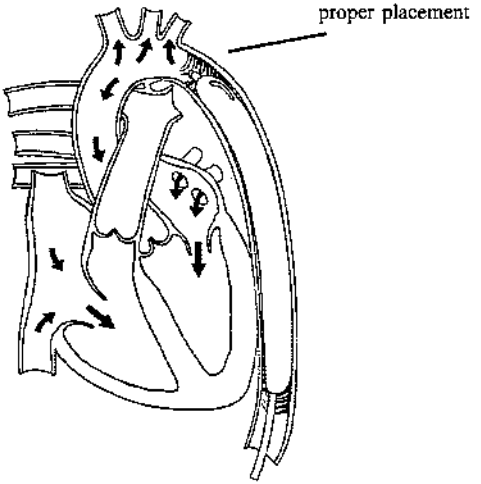
Weaning

Weaning parameters will also differ with different protocols. In general, the most commonly used parameters are normal arterial pressure, cardiac index of 2.2 L/min/meter sq or greater. There are two methods of weaning. The **frequency ratio weaning**, is the method in which the ratio decreases from 1:1 to 1:2 to 1:4 and so on. If the patient tolerates this the balloon is finally stopped. There are studies that suggest this type of weaning is not actually giving the patient intermediate levels of balloon assistance due to the rapid changes in afterload reduction between assisted and unassisted beats. This is considered by these studies to be the same as stopping the balloon. However, this method is commonly used today and recommended by some balloon manufacturers.

The other method of weaning is **volume weaning.** With this procedure the balloon volume is decreased by a small portion (20-25% of the total volume). After a period of evaluation, if the patient tolerates this reduction, additional volume is removed. This does not cause the rapid changes in afterload reduction that the heart would be required to respond to as with frequency ratio weaning. This may be a more physiologically normal way of weaning. However, some balloon manufacturers do not recommend this due to concerns of clot formation because of decreased inflation. Proponents counter that since **the balloon volume is not reduced below approximately 20% of its total volume**, this should not be a problem. In any case, the recommendations of the manufacturer of the balloon being used should be considered when deciding which method to use. The balloon should always be removed as soon as pumping is stopped. **Stop the heparin about 4 hours prior to removal.** Checking a platelet count prior to removal is recommended.

Basic Setup Steps

1 - Ensure the patient received heparin — 5000 units
2 - Prep the balloon — negative pressure before removing from holder
— remove stylus
3 - Plug in Pump
4 - Check the helium volume
5 - Obtain EKG signal
6 - Setup and prime transducer
7 - Connect to patient
8 - Ensure arterial waveform
9 - Purge balloon
10 - Initiate in 1:2 ratio and adjust timing
11 - Begin 1:1 ratio



Proper IAB Placement

This drawing represents placement that shows the balloon just below the left subclavian artery which should also place it above the renal arteries.

Note

There are several IABPs and IABs commercially available. Each has its good points. The decision of which pump and balloon to use is that of the physician and the perfusionist. Therefore it is important for the perfusionist to evaluate the products available and determine which has the best features for his or her program.

NOTES

Ventricular Assist Devices

Perfusionists are called upon to initiate ventricular assist devices (VADs) in emergency situations. Ventricular assistance requires planning before the actual procedure is necessary. Preparation requires assembling equipment, circuits and supplies for these emergencies. Preparation also requires knowledge of the procedure and practice for implementation. The situation usually arises in the operating room or in the post-op setting. In the operating room it is usually a patient that can not be removed from CPB and is suitable for the ventricular assist device (VAD). This patient is on inotropic agents and has an IABP working. The patient in the post-op setting has a low-output syndrome within 72 hours of surgery or a dysrhythmia that does not respond to pharmacological agents. VADs can also be used as a bridge to transplant.

Patient Selection

The first factor considered is whether the procedure has a **reasonable chance** of being successful in the patient. The overall health of the patient is a major factor. Contraindications are age greater than 70 years, severe infection and other organ dysfunction. Complications of the procedure include coagulopathy, infection and renal failure. This major committment is not undertaken lightly. Resources are expended in this procedure that may compromise care of other patients. The patients that are selected display inadequacy of the ventricle. This inadequacy is determined by decreased contractility, elevated left ventricular filling pressures, elevated pulmonary capillary wedge pressures and decreased pulmonary oxygenation. The survival rate is as high as 20-30% in carefully selected younger patients according to some studies.

Signs of Left Ventricular Failure

- Decreased contractility
- Elevated left ventricular filling pressures
- Elevated pulmonary capillary wedge pressures
- Decreased pulmonary oxygenation

Equipment

Pulsatile paracorporeal pumps are gradually coming into use and are probably the VADs of the future. There are several types of devices that are gaining acceptance. The Abiomed, Novacor and Thermedics are all devices that provide pulsatile assistance to support the ventricle. Operation of these devices is not complicated and each is described in the instructions that come with the devices. Major disadvantages of these devices are the cost and the infrequent use.

The cost is the major advantage of the **centrifugal pump** that is also used as a VAD. This pump is found in most institutions and perfusionists are familiar with its operation. It is the most widely used VAD. The pump is a forced vortex that propels the blood smoothly, causing relatively little red blood cell damage. In these cases that often extend for many days, this can be a factor. The centrifugal pump head may be the site of clot formation and should be changed at least **every 72 hours.** Roller pumps have also been used by many perfusionists with good results.

The circuit that is used with the centrifugal pump is very simple. There is simply an inflow tube and an outflow tube that connects to the pump. The tubing is 3/8 inch diameter. A flow probe and a luer stopcock for sampling and air removal is included. An oxygen saturation probe can also be incorporated into the tube. Arterial filters are not used in these circuits due to the long spans that the VADs are used. The long periods would cause the filters to become blocked with platelets and white blood cell aggregates.

Cannulation

Withdrawal cannulae that are designed for these type cases are now available commercially. These right angled cannulae are placed in the left atrium, left ventricular apex or retrograde across the aortic valve. The infusion cannula is placed in the aorta. If the patient has been on CPB the same aortic cannula used during the case can be left in place and utilized. The **left ventricle will then be bypassed** somewhat and allowed to rest. If transplantation is to be attempted the cannulation is the left atrium and the ascending aorta.

Priming

The VAD can be primed in various ways. A convenient method often used involves the sterile technicians at the operating table. The ends of the tubing can be held on the sterile field and the other ends attached to the centrifugal pump by the perfusionist. The sterile technician places the end of the withdrawal tube in a large basin containing 3 liters of priming solution. The other tube is connected to a field sucker and the suction draws fluid around the circuit. This primes the centrifugal head as well as the circuit. The perfusionist manipulates the head, removing air. Once the circuit is primed, the sterile technician holds both tube ends in the basin of fluid, while the perfusionist gently pumps the fluid around the circuit. This removes any remaining air. The scrub technician must keep the tubes separated in the basin to prevent expelled air from returning to the circuit.

A method that can be used if circumstances do not allow the usual priming techniques is to fill the circuit slowly with the patient's own blood. The tube that is connected to the cannula that withdraws blood from the heart is allowed to fill. The blood is slowly brought back to the pump head and then to the tube that will connect to the aortic cannula. During this time, the patient

must be infused with volume somehow to account for the loss of volume necessary for priming. In the usual situation the patient is still cannulated in the aorta and connected to the heart lung machine. This cannula and the existing CPB pump can be used to infuse the required volume. Once the circuit is primed, the circuit is attached to the aortic cannula and the VAD circuit is ready. This method of priming has the disadvantage of not being able to circulate for air removal.

Biventricular Assistance (BIVAD)

It is often necessary to provide assistance to the right ventricle as well as the left ventricle. The VAD giving assistance to the left ventricle will not work if the right ventricle cannot supply the lungs and left atrium. **Right heart failure** is occurring if the central venous pressure is high while the pulmonary wedge pressure is low. Cardiac index less than 1.8 L/min/m^2, aortic pressure less than 90 mmHg, atrial pressure greater than 20 mmHg and pulmonary capillary wedge pressure less than 10 mmHg are indicators that the right heart is failing. This is now being recognized as a common problem in most cases. The circuit and pump used for the right ventricular assist device (RVAD) are similar to the setup used for the left ventricular assist device (LVAD). The cannula that removes blood from the right atrium can be the CPB venous cannula. The other cannula, where the blood is being pumped, is placed in the pulmonary artery, through the right ventricle or directly into the pulmonary artery. This allows augmentation of the right ventricle.

Signs of Right Ventricular Failure

- Cardiac index less than 1.8 L/min/m^2
- Aortic pressure less than 90 mmHg
- Atrial pressure greater than 20 mmHg
- Pulmonary capillary wedge pressure less than 10 mmHg

Flows

The initiation of the VAD flow is done slowly. If the return is not adequate flows must be kept low until improvement occurs. It is possible, even with the centrifugal pump, to suck air from around the cannulation sites if flow is not sufficient and the RPMs are kept high. Air may also be sucked out of solution if obstruction is total. The first sign of inadequate return is a "chattering" line from the heart to the pump. Volume can be added to the patient to attempt correction of this problem. If the problem is not alleviated, flows must be held at a reduced rate. Full flow is **2.2 L/min/M^2**. A left atrial pressure (LAP) monitoring line is necessary for VAD cases. The LAP is maintained in a 5-15 mmHg range. The LAP must be kept above 5 mmHg to prevent air from being drawn into the cannula.

Coordination of BIVADs is done by slowly starting the LVAD until its flow causes the LAP to fall to 5 mmHg. Then the RVAD can be started and brought up slowly until the LAP rises to 15 mmHg. The RVAD is often set higher than the LVAD because the left ventricle is usually ejecting, thus adding to the flow coming from through the aorta. The coordination of the two VADs has the goals of achieving flows of 2.2 L/min/M^2 and keeping the LAP between 5-15 mmHg, along with maintaining an adequate mean systemic arterial blood pressure.

Heparinization

An oxygenator is not used during these cases and activated clotting times (ACTs) are maintained in a relatively low range of **180 to 200 seconds.** Heparin is given as required to maintain this slightly anticoagulated status. If the patient is fully heparinized protamine administration may be required to reduce the ACT. Some teams prefer to simply allow the heparin level to drop as it is metabolized. Heparin is given using a minidrip and infusion pump.

Management

The arterial wave form will flatten as the VAD begins to do its work. The ventricle is not working as hard. (The IABP may cause an arterial wave form to be displayed.) The ventilator is used during these cases and the arterial pO_2 is kept above 75 mmHg. The systemic vascular resistance is maintained in the normal 800-1200 range and this may require administering vasoconstrictors. Sepsis is a major complication of these cases and strict sterile technique is used by all staff members. Prophylactic antibiotic therapy is routinely employed. A heat exchanger is not used in this circuit and a warming blanket or heat lamp is used to maintain normal body temperatures. An intraaortic balloon is usually in place and is timed in the usual manner. The balloon provides helpful pulsatile flow. Blood gases should be monitored as should urine output. Skin color and temperature are other practical ways to judge the adequacy of perfusion.

Fluid replacement management is critical in these cases. This is often complicated by excessive bleeding. Blood replacement products are used in large volumes and it is often necessary to reinfuse drainage from the chest tubes. Some teams use blood salvaging devices to process this drainage and improve the quality of the infused product. Even with the large infusion of blood products, crystalloid solutions are used copiously. This may cause a drop in the serum albumin and the colloid osmotic pressure (COP). Normal serum albumin is 3.5-5.0 gm/100ml and normal COP is about 25 mmHg. Edematous patients are not removing waste products effectively and survival chances are decreased. This state can be avoided by use of 25% albumin as required to maintain an **adequate COP and serum albumin**.

Termination

The heart is allowed to **rest for 48 hours** on the VAD if possible. Testing of the ejection capability of the ventricles is then performed. The flows are decreased and the arterial pressure wave is observed. If the ventricle is ejecting there should be a wave correlating with this phase. The inflation of the IAB should not be confused with this ejection. The tracing will look much like a traditionally timed IABP. For RVADs the pulmonary artery monitoring should display the ejection of the right ventricle. The cardiac output is determined during this test period. If the total cardiac output can be maintained at 2.2 L/min/M^2, then weaning is begun. Pump flow is decreased by about a liter and after one or two hours the test is repeated. If successful the pump flow is further reduced and the procedure is repeated. When flow is reduced to less than 2000 ml extra heparin is given to increase the ACT to 250-350 seconds. This prevents clot formation in the cannulae and circuit due to low flows. The cannulae are clamped as the flow is stopped and the patient is observed. If the patient tolerates the termination, the cannulae are removed.

NOTES

Adult Extracorporeal Membrane Oxygenation (ECMO)

The perfusionist is occasionally called upon to perform an adult ECMO. These cases are rarely encountered, and by this very fact, are sometimes difficult in preparation and performance. Adult ECMO is controversial and there is disagreement upon the effectiveness of the procedure. One of the problems of evaluating the process is the criteria used to decide which patients to place on ECMO. Patients who are placed on ECMO early, before deterioration has progressed, tend to improve and survive at a higher rate than those who are placed on ECMO later in the process. Most perfusionists have performed adult ECMOs or will eventually be called upon to perform this function. This is a major undertaking requiring many supplies and personnel. A large isolation room with a bed that can tilt the patient from side to side should be used. Extra care should be taken to prevent infection.

ECMO Patient Selection

Patients should have a reasonable chance of surviving with an acceptable quality of life. The selection of patients rules out those with other problems that cannot be corrected. Patients who are actively bleeding are poor candidates. Those with severe nervous system damage will not improve. Those with severe cardiac damage to the left ventricle will not benefit. Those with other terminal conditions such as cancer or uncontrolled sepsis would not benefit. Pulmonary fibrosis is another condition that will not improve with this procedure.

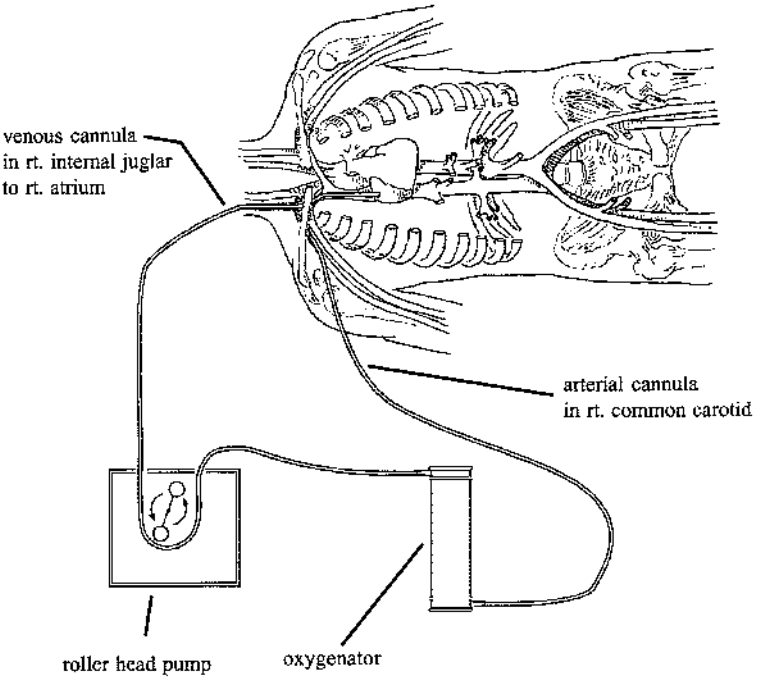
Those who may be helped are those with **acute pulmonary failure** associated with circulatory collapse such as that associated with a massive pulmonary embolism. Post cardiac surgery patients with pulmonary and cardiac failure would also benefit from the procedure. Other types of patients such as those with conditions that will improve if they can be supported for a period are candidates. Patients with lung trauma, certain infections and ARDS may also be helped. Damaged lungs that do not transfer oxygen to the blood cause the patient to develop fatal cardiac and renal conditions. Acute respiratory distress syndrome (ARDS) is an acute lung condition where the lung function is stopped due to increased capillary permeability. In addition, now that lung transplants are becoming more prevalent, ECMO support can be used as a bridge to transplant.

Cannulation

The purpose of ECMO is to provide oxygenation with an oxygenator and this requires cannulation as in standard CPB. Cannulation for **veno-arterial** ECMO cannulates through the right internal jugular vein to the right atrium for

venous withdrawal. The arterial inflow cannula is placed into the right common carotid artery. The femoral vein and femoral artery can also be used for this purpose. Cannulation of the femoral artery is done distally and proximally with a Y connector joining to a single arterial line. Venous cannulation also uses two cannulae joined by a Y into a single venous line. Distal and proximal arterial and venous cannulation ensures oxygenation and drainage of the cannulated leg. A graft connection to the femoral artery eliminates the necessity of dual cannulation of the artery.

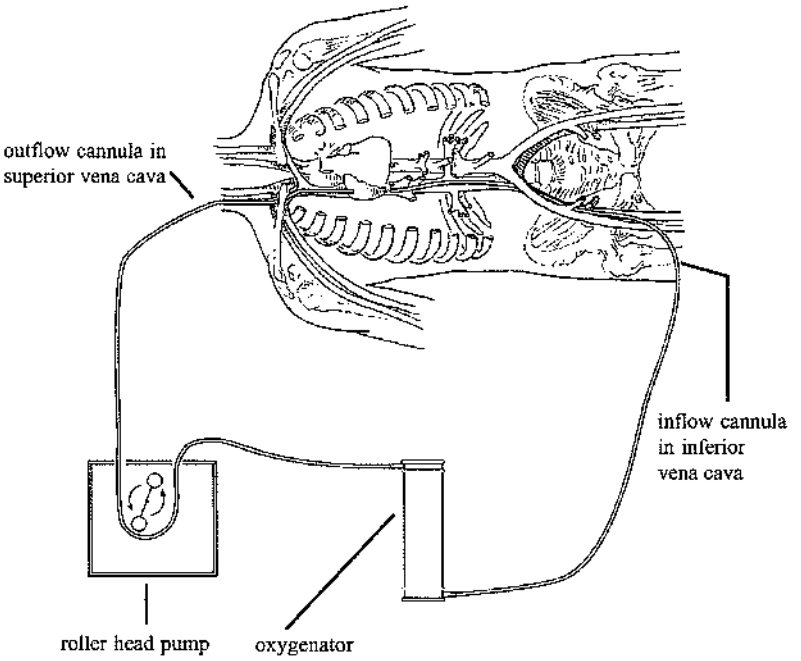
Veno-Arterial ECMO



Veno-veno ECMO cannulates the superior vena cava through the right internal jugular to remove blood to the oxygenator. After oxygenation the blood is returned to the inferior vena cava by a cannula in the femoral vein. The idea of this type cannulation is that some oxygenated blood will pass into the lungs through the right ventricle. Some of the blood will be returned to the oxygenator through the outflow cannula where the oxygen content will be increased again. The volume removed and the volume returned are the same,

so the circulating volume is not affected. The oxygen content of the blood will increase. A functioning heart is necessary with this technique to propel the blood throughout the body. The pump does not provide any circulatory support.

Veno-Veno ECMO



Heparinization

Anticoagulation with heparin is used to prevent thrombus formation in the oxygenator and the circuit. Initial heparinization of 100-200 units per kg is given as an IV bolus. ACTs are maintained in a range of **300-350 seconds** when flows are high. The ACT is tested at 30 minute intervals. Heparin administration is done by drip through an infusion pump or by bolus injection at 30 minute intervals if required. The infusion pump allows more accurate control of the coagulation status. A starting dose of about 40 units/kg/hr is appropriate. The dose can then be adjusted according to the ACT.

Oxygenators

The Medtronic ECMO membrane oxygenators use a solid sheet silicone material that has excellent gas exchange capabilities and decreases trauma to the blood elements. This **true membrane** maintains stable CO_2 and O_2 for long periods. Micropore oxygenators experience reduced gas transfer after long periods. Platelet reduction is limited by using this type membrane. Membrane lungs transfer carbon dioxide 6 times greater than oxygen. Hypocapnia occurs frequently and low pCO_2 should be corrected. Rising pCO2 may be the first indication of oxygenator failure due to the efficiency of the CO2 removal capability.

The Medtronic ECMO oxygenators set the standard for long term ECMO support. Oxygen flow meters attached to the blender must be the type that provides flow up to 20 liters. Flows are kept higher with these oxygenators. These higher flow meters can be placed on existing blenders without replacing the entire blender system. Some perfusionists use hollow fiber membrane oxygenators, especially the bioactive coated oxygenators with good results. These oxygenators work fine for relatively short ECMOs, but often require changing much sooner than the true membranes.

The Medtronic oxygenator is available in six sizes with maximum recommended flow rates of 0.35, 1.2, 1.8, 4.5, 5.5 and 6.5 LPM. Data on these oxygenators is summarized below.

PEDIATRIC ECMO OXYGENATORS

MODEL#	**0400-2A**	**0800-2A**	**1500-2A**
Size Patient	4 kg	11 kg	19 kg
Prime	60 ml	100 ml	175 ml
Max Rec. Blood Flow	0.35 LPM	1.2 LPM	1.8 LPM
Max Rec. Gas Flow	1.2 LPM	2.4 LPM	4.5 LPM

ADULT ECMO OXYGENATORS

MODEL#	I-2500-2A	I-3500-2A	I-4500-2A
Size Patient	70 kg	95 kg	>95 kg
Prime	455 ml	575 ml	665 ml
Max Rec. Blood Flow	4.5 LPM	5.5 LPM	6.5 LPM
Max Rec. Gas Flow	7.5 LPM	10.5 LPM	13.5 LPM
Membrane Size	2.5 M sq	3.5 M sq	4.5 M sq

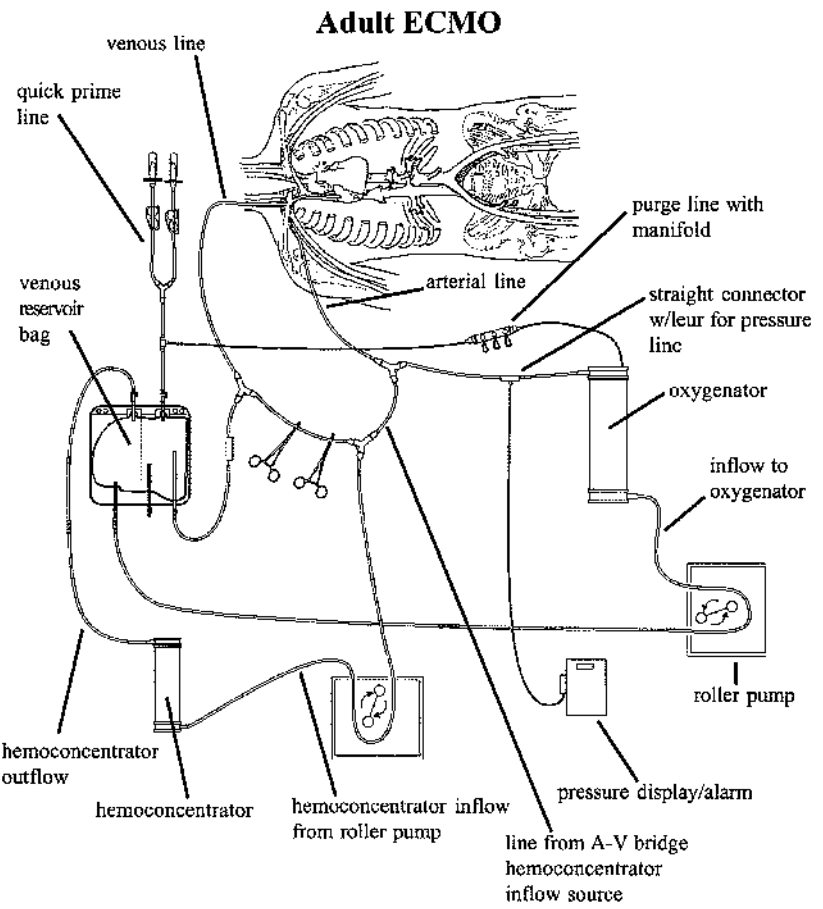
Circuits

The circuit design for ECMO should incorporate features that are pertinent for this long term support. An arterial filter is not used in the circuit due to the long duration of most ECMO cases. The filter would not function for this long period. The tubing used for the inlflow and outflow lines are both 3/8th inches for an adult circuit. Blood from the patient comes back down the 3/8th inch line by gravity to a venous reservoir bag or a bladder bag with a servoregulator. Bladder bags are often used with pediatric patients in institutions that do a great deal of these type cases. Most institutions do not have this capability. Leaving the venous reservoir the blood moves to the pump, either roller or centrifugal, and then to the oxygenator. A 1/2 inch boot can be used if a roller pump is used to decrease wear on the tubing. In addition the boot should be long enough to allow advancing the tubing at 48 hour intervals to prevent breakdown. From the oxygenator the blood goes back to the patient. The **venous reservoir bag** is a good feature to incorporate, however this bag should be as small as possible because of the increased surface area. Large reservoirs add to the surface area and increase clotting. In addition all volume should not be removed from the patient in order to maintain ejection and systemic circulation. Level detectors can be placed on the bag to shut off the pump if the return is stopped. The reservoir also prevents air from being sucked in at the cannulation sites due to excessive pressure. Other uses of the reservoir are as an access to add fluids and as a site to return hemoconcentrator blood.

A **crossover bridge** between the inflow and outflow lines is another asset. This bridge is a good safety feature. It can be used to change oxygenators or to purge any air that enters the oxygenator. Air can be purged around through the bridge to the reservoir bag. Locating the bridge near the oxygenator, away from the patient, makes it easier to manipulate.

Hemoconcentrators are often necessary for long term cases. Their use serves to help control the hematocrit and electrolyte concentration. There are many potential locations in the circuit to place a hemoconcentrator. The crossover bridge may have a Y connector incorporated with tubing leading to a hemoconcentrator or the reservoir bag may have a port that can be used. A dedicated roller pump is necessary regardless of where the hemoconcentrator is placed. This allows precise control in cases of reduced flows that are often found during ECMO procedures.

Other items included in the circuit are in-line oxygen saturation or blood gas probes, depending on which is available. A luer connection with monitoring line connected to a stopcock manifold on the arterial side to draw samples for blood tests is necessary. This line can purge to the reservoir bag. A luer connection with monitoring line to provide input to a pressure gauge for outflow pressure observation should also be placed. If the oxygenator does



not have a built-in heat exchanger a separate heat exchanger must be incorporated into the circuit distal to the oxygenator.

Fluid Management

ECMO requires expert manipulation of the solutions added to the patient during these extended perfusions. The hematocrit, colloid osmotic pressure and clotting factors are all affected by this undertaking. Fluids in the intracellular and extracellular spaces change during these cases. **Interstitial fluid** may fluctuate. While the average adult has about 11.2 liters of interstitial fluid, this amount may shift drastically due to the similarity of interstitial fluid and plasma. Slight changes in the composition will precipitate these changes. Sodium and chloride are greater in the extracellular spaces while potassium is

greater in the intracellular fluid. Fluid shifts also cause movement of these electrolytes to other spaces.

Edematous patients are a major problem. The addition of large volumes of crystalloid solutions lessens the colloid osmotic pressure and leads to increased interstitial fluid buildup. Patients with large amounts of fluid in the interstitial space are not able to return waste products to the circulating blood for removal. Serum albumin and the colloid osmotic pressure lab values should be obtained at 6 hour intervals and 25% albumin added to the circulating volume if values begin to drop. Serum albumin has a normal range of **3.5-5.0 gm/100ml**. Colloid osmotic pressure is normally 25 mmHg. Fresh frozen plasma is also beneficial for reducing interstitial fluid buildup. Daily weighing of the patient and visual inspection for edema are also necessary. Diuretics such as Lasix can also help reduce interstitial edema. The hemoconcentrator is also a valuable tool in this situation. Currently, most perfusionists maintain hematocrits at about 30 percent. This appears to provide better capillary perfusion and oxygenator longevity while also providing sufficient oxygenation. This requires the addition of many units of packed red blood cells over the course of the case. The packed red blood cells should always be infused through a blood filter into the reservoir bag.

Coagulopathies and the resulting blood loss are major complications of ECMOs. Although the patient is kept partially heparinized, excessive bleeding must be controlled. Ceaseless bleeding at the cannulation sites and other sites must be corrected. PT and PTT values that are greatly increased are an indication for FFP administration. Platelets are also given if counts **below 100,000** are present. The fibrinogen level is kept above 100 mg/dl. If lower levels are encountered, cryoprecipitates can be given.

Labs

Lab tests must be routinely obtained to recognize problems before they become serious. Routine tests that should be taken at 2 hour intervals are as follows:

LABS

- Arterial and venous blood gases
- CBC with platelet count
- PT, PTT
- Fibrinogen level
- Electrolytes — Na, K, ionized Ca, Mg
- Serum albumin
- Colloid osmotic pressure

Perfusion Parameters

Flows are brought up slowly to 80-90% of the cardiac output. The ventilator functions throughout the ECMO. Blood is, of course, entering the lungs as if they were working normally. Improved lung function is the goal of the procedure. End tidal CO_2 is monitored at the airway to determine function of the lungs. Low values indicate that the lungs are not performing gas exchange. Normal values (35 torr) indicate that the lungs are functioning and weaning may be appropriate. Venous oxygen saturations from the in-line probe are kept at 70-80%. The cannulation sites determine the importance of this venous oxygen saturation. In veno-arterial cannulation this saturation is a true venous saturation. In veno-veno cannulation the oxygenated blood is being recirculated and this value does not indicate true venous oxygen saturation. However, this value will indicate trends in oxygenation.

Weaning From ECMO

The patient can be removed from ECMO when pulmonary and cardiac functions are capable of support. Pulmonary functions, chest x-rays, cardiac outputs, venous O_2 saturations and blood gas results are all considered before weaning is attempted. The patient is temporarily taken off ECMO and venous O_2 saturations are observed. The process for temporarily removing the patient is much like ordinary CPB except heparinization. Increased heparin is added and the ACT is brought up to 350 seconds to prevent clot formation. The cannulae will not have any flow when the ECMO is interrupted. The rest of the circuit and the oxygenator will have reduced flow during this time. The blood flows are slowly decreased and both tubes are clamped just below the cannulae. The A-V bridge is opened and the blood is circulated to prevent clot formation. If the patient's venous O_2 saturations stay above 60% with an FiO_2 of 0.6 or less, the patient may be permanently weaned from the ECMO. First, the patient is placed back on the ECMO and the flows are decreased at 2 hour intervals by 1000 ml. Periodically, temporary removal is performed and the venous oxygen saturations checked. If the saturations stay above 60% during these periods, weaning continues. When the flows are down to **2 liters permanent removal** is attempted. After it has been determined that the patient will tolerate permanent removal, the cannulae can be removed.

Oxygenator Changeout

A method to change oxygenators should be included into the circuit. The most practical way is to have Y connectors in the line just above the inlet and another just above the outlet of the oxygenator. This serves as a place where another oxygenator can be placed in line and the other oxygenator clamped out of the circuit. Six inch segments of tubing with straight connectors and luer lock stopcocks are placed in each segment of line for purging air after connection of the new oxygenator. The new oxygenator should be primed

and deaired before it is connected. After connection the luer stopcocks are used to remove air from the lines. Next, the inlet clamp can be removed while flow continues through the old oxygenator also. Any air that comes out of the oxygenator can be removed through the stopcock on the outlet line before the clamp going back to the patient is removed. At this time there will be two oxygenators working until the old one is clamped out and removed leaving a place for another oxygenator to be placed later. This method of changing oxygenators causes no interruption of flow to the patient.

Another method of changing the oxygenator is to utilize the A-V bridge. The replacement oxygenator is primed before the old one is removed. Flow is halted while the old oxygenator is cut out and the new one placed in the circuit. Flow is then directed through the A-V bridge to remove any air. After deairing, the bridge is clamped and flow to the patient is started again.

Portable Emergency Bypass Systems

The advent of cannulae that are small enough to allow percutaneous insertion has led to systems that can be initiated quickly without lengthy cutdowns. These type systems can be used to provide oxygenated veno-arterial support. The system uses a centrifugal pump and primes very easily through an A-V bridge. Cardiac or cardiopulmonary support (CPS) can be provided in many different clinical settings. Cardiogenic shock secondary to myocardial infarction, post-op ventricular failure and high risk angioplasties are all indications for employment of these perfusion systems. The bioactive surfaced oxygenators and circuits in some of these systems are making it possible to provide ECMO support for short periods with little anticoagulation. In the standard systems full heparinization must be accomplished before perfusion begins. The long venous cannula is inserted into the femoral vein and reaches the right atrium where the heart can be drained. The arterial cannula is placed in the femoral artery. These thin wall cannulae are able to accommodate flows of **5-6 LPM.**

Patients are sometimes started on this device for transport to the operating room. Surgery may require the perfusionist to adapt the circuit in the operating room to a more traditional one. Venting of the heart, cardioplegia delivery and suction capability are all necessary. One option is to simply reattach the cannulae to a conventional circuit and heart lung machine. Another method is to prepare a separate cardiotomy and single roller pump with two lines through the raceway. One of the lines can be used for venting and the other for suction. If cardioplegia is used crystalloid can be infused using a pressure bag and gauge. Ordinarily, patients are changed over to the normal heat lung machine and bypass circuit, however.

Infants

One of the more common successful uses of ECMO is for newborns who are experiencing respiratory failure. This failure may be caused by **hyaline membrane disease**, respiratory distress syndrome, meconium aspiration and persistent pulmonary hypertension of the newborn. ECMO is successful in over **80%** of attempted cases. Not all infants are candidates for the procedure, however. The lung disease must be reversible and there must not be intracranial hemorrhage or coagulopathy present.

Pediatric membrane oxygenators are used for the procedures with pediatric 1/4 inch tubing circuits. True membrane oxygenators have the advantages discussed above for these potentially long cases. Both roller and centrifugal pumps are used and a small venous reservoir bag is incorporated in the circuit that is similar to the adult circuit. Cannulation is usually veno-arterial although veno-veno is sometimes used. In veno-arterial cannulation the right internal jugular vein is cannulated for venous access. The arterial return of oxygenated blood is through a cannula placed in the right common carotid artery. Veno-veno ECMO requires cannulation of the internal jugular vein for venous access. The oxygenated blood is returned through a cannula in the femoral vein.

The infant is kept on the ventilator as in adult ECMO. **Oxygen toxicity** can cause eye damage in infants and therefore the FiO_2 is kept less than 0.30. The arterial PaO_2 is kept below 90 torr. Excessive oxygen concentrations cause initial retinal vasoconstriction and later vasoobliteration. Next, neovascularization may occur. The vitreous may have fibrovascular invasion and retinal detachment occurs. This condition is called retrolental fibroplasia.

Heparinization is aimed at maintaining an ACT of 250-280 seconds. Blood flow rates are brought to about 150 ml/kg/min. The hematocrits are kept higher in infants at about 40%. When the infant improves the bypass is terminated in the same manner as described above for adult ECMO.

Pediatric ECMO is usually reserved for institutions that routinely perform this function due to the special skills and manpower required.

NOTES

Pediatrics

Pediatric heart surgeries with the complex procedures that are done are some of the most challenging cases for perfusionists. These cases often run the gamut of unusual cannulation, circulatory arrest and profound hypothermia. The circuits must be determined with careful calculation as must the priming solution. Transfer of even the smallest volume can have major consequences. The priming volume must be kept to a minimum. The pediatric patient has many **anatomic and physiologic differences** from the adult patient. The pediatric heart is much larger proportionally than the adult heart. Circulating volume is less. The vascular system is more elastic due to the lack of atherosclerotic disease. The brain of the infant receives 34% of the cardiac output, much more than the adult's brain. The respiratory rate of newborns is much higher than adults at 34 times per minute. Some drugs exhibit different actions when used in pediatric patients. These patients are often cooled to temperatures much lower than adult patients. However, the pediatric patient has a higher metabolic rate than the adult patient. Cannulation may be unusual because of the congenital defects present.

Preparation

The perfusionist prepares for the case by reviewing the operative procedure and the particular patient information. The procedure usually reveals the congenital defect present. For instance, if the procedure is described as a Blalock-Taussig Procedure then the perfusionist knows that this is usually a temporary procedure in infants with tetralogy of Fallot. (The procedure entails construction of a shunt to join the subclavian end-to-end with the pulmonary artery to move blood from the systemic circulation to the lungs.) In other types of surgery the existence of these temporary palliative shunts from prior surgery should be determined. The patient's chart is reviewed for the history, physical examination and lab data.

Cannulation

Arterial cannulation is much the same as adult arterial cannulation. The ascending aorta is usually cannulated proximal to the innominate artery. At times it may be necessary to cannulate other arteries such as the femoral. It may be necessary to cannulate more than one artery to provide sufficient flow to the brain and other major organs.

Venous cannulation is usually bicaval. The superior and inferior vena cavae are cannulated. There may be found a persistent left superior vena cava and this may also require cannulation. In infants a larger percentage of the venous return enters by way of the superior vena cava. Charts listing the cannulae sizes and flows are listed.

Cardioplegia cannulation is performed for antegrade administration by inserting a large bore IV catheter or a small aortic root cannula. Pediatric retrograde cardioplegia cannulae are available commercially.

Blood Flows

The necessary blood flow is calculated before the circuit is determined. The flow is calculated, as in adult patients, by multiplying the body surface area by the cardiac index when normothermic. However, a higher cardiac index is used due to the higher metabolic rate with increased oxygen consumption of pediatric patients. A cardiac index of 2.8 to 3.2 L/m^2/min. is used in the calculations. Flow rates of 60 to 80 ml/kg/min are necessary for larger children, while infants may require 80 to 150 ml/kg/min. Most teams use formulas that utilize the BSA chart to determine flows, but the flow can also be determined using ml per kg/min.

Blood Flow Rate Using the BSA
(at normothermia - full calculated flow)

Newborns - 2 yr.	3.0 - 3.2 x BSA
2 - 4 yr.	2.8 x BSA
4 - 6 yr.	2.6 x BSA
6 - 10 yr.	2.5 x BSA
10 yr. and above	2.4 x BSA

Circuit Size

Infants have a small blood volume. This requires that the perfusion circuit be kept to a minimum to prevent unnecessary hemodilution. The following table is often used to determine the tubing size of the A-V circuit. Of course the goal is to find the smallest possible circuit that will provide adequate flow.

A-V Tubing Size in Pediatric Circuit

Weight	Arterial	Venous
<4.5 kg	1/4	1/4
4.5-9.0 kg	1/4	1/4
9.0-16 kg	1/4	3/8
16-40 kg	3/8	3/8
>40 kg	3/8	1/2

Tubing Volume

Inside Diameter	ml/ft.
1/4 in.	9.65
3/8 in.	21.71
1/2 in.	38.61
5/8 in.	48.00

The oxygenator can be selected depending on the necessary flow from the following list.

OXYGENATOR	RATED FLOW (LPM)	PRIME VOL (ml)	O2 TRANSFER (ml/min @ LPM)
COBE / DIDECO / SORIN			
LILLIPUT I	0.8	60	58 @ 0.8
LILLIPUT II	2.3	105	130 @ 2.3
MEDTRONIC			
MINIMAX PLUS	2.3	149	94 @ 2.3
0400-2A	0.35	60	
0800-2A	1.2	100	
1500-2A	1.8	175	
POLYSTAN / JOSTRA			
SAFE MICRO	0.8	52	**
SAFE MINI	2.3	90	**
TERUMO / SARNS			
CAPIOX SX10 & 10R	4.0	135	250 @ 4.0
RX05 BABY RX	1.5	43	100 @ 1.5

* Measurements at barometric pressure of 620 mmHg
* Data not avaiable at time of print

Arterial line filters are listed in this table with appropriate flows and priming volumes.

Filters

Name	Maximum Flow	Prime
Baxter AF 540D	<3 LPM	115 ml
Medtronic Intercept Pedi	<3 LPM	115 ml
Pall 1/4 in.	<2 LPM	50 ml
Sorin Micro P	<3 LPM	100 ml
Terumo Capiox AF02	<2.5 LPM	40 ml

Cannulae can be selected from the following list depending on the flows.

Arterial Cannulae

Name	Size	Flows
BARD		
FEMORAL ARTERY	16 F	≤2.4 LPM
FEMORAL ARTERY	18 F	≤3.5 LPM
FEMORAL ARTERY	20 F	≤4.3 LPM
FEMORAL ARTERY	22 F	≤6.2 LPM
DLP / MEDTRONIC		
DLP	8 F	≤0.6 LPM
DLP	10 F	≤0.9 LPM
DLP	12 F	≤1.5 LPM
DLP	14 F	≤2.5 LPM
DLP	16 F	≤3.0 LPM
DLP	18 F	≤4.0 LPM
DLP	20 F	≤6.5 LPM
SARNS		
High Flow Aortic Arch	3.8	≤1.6 LPM
High Flow Aortic Arch	5.2	≤3.5 LPM
High Flow Aortic Arch	6.5	≤5.2 LPM
SORIN BIOMEDICAL		
A211-30	3.0 mm	≤1.2 LPM
A211-38	3.8 mm	≤1.8 LPM
A211-45	4.5 mm	≤2.9 LPM
A211-52	5.2 mm	≤4.1 LPM
A211-65	6.5 mm	≤6.3 LPM

Venous Cannulae

weight in kgs	SVC/IVC Cannulae in French size	RA Cannula in French size
0-3	12 / 12	18
3-6	12 / 14	18
6-8	14 / 14	20
8-10	14 / 16	22
10-12	16 / 16	24
12-15	16 / 20	24
15-20	20 / 20	26
20-25	20 / 24	28
25-30	24 / 24	28
30-35	26 / 26	30
35-40	28 / 28	32

The circuit is determined using the above charts and the cannulae found using the appropriate chart above. The amount of priming solution needed can then be calculated. The tubing length is usually known, but if necessary it may be measured. The volume of one foot of tubing is then multiplied by the number of feet of that particular size tubing to determine the volume it contains. The volumes in both the arterial and venous tubings are calculated and combined. Next, the priming volume of the oxygenator is found in the table and added to the tubing volume. Then the arterial filter and venous reservoir are included. This is the total volume needed to prime the oxygenator and circuit. Now that the total volume is known the hemodilutional effects can be found on the hematocrit, platelets and coagulation proteins.

Hemodilution

The chart below can be used to determine the blood volume of the pediatric patient. The hemoglobin of infants is higher than that of adults. The hemoglobin falls until it is below that of adults by 3 months. At 3 months the level is about 12 gm% and then slowly increases to that of a normal adult. **Polycythemia** is found in many of these patients. This abnormally high RBC count may be present due to the compensation from poor oxygenation. Right to left shunts lead to this condition. As can be seen from the chart, pediatric patients have more blood per kilogram of body weight than adults.

The **colloid osmotic pressure (COP)** is also lessened by the priming solutions. Normal COP is about 25 mmHg and this will drop on bypass. A normal COP is not necessary and it may even be desirable to allow the level to fall somewhat. There is some evidence that a lower COP may improve renal

function during bypass. Some studies have shown that the COP must be decreased over 50% to cause fluid to enter the interstitial region. However, to achieve an acceptable COP level it will require that **albumin** be added to the priming solutions. Without the addition of 25% albumin the COP would fall to a level that would cause interstitial fluid buildup. In addition albumin reduces platelet loss by inhibiting aggregation to the perfusion circuit. The addition of large amounts of crystalloid solutions into the intravascular system allows fluid to exit the capillaries and not return. In a patient with a normal COP the fluid would reenter the capillaries. The buildup of fluid in the interstitial region prevents the removal of waste products. Colloids are large particles in solution. Their size is a minimum of 40,000-50,000 Daltons. These large particles increase the COP and cause fluid, that is forced out of the capillaries by the arterial blood pressure, to reenter the vascular system in search of equilibrium.

Fibrinogen is also diluted by the priming solution. Fibrinogen is one of the clotting factors. Many teams calculate this level and add fresh frozen plasma (FFP) to the priming solution if necessary. Traditionally, a level of 100 mg/dl is considered a minimum. Many teams allow lower levels during bypass to avoid transfusions of FFP. FFP with a concentration of 200 mg/dl can be added to achieve the 100 mg/dl level. Cryoprecipitates, which also furnish fibrinogen, are not given on bypass.

BLOOD VOLUME BY WEIGHT

WEIGHT	VOLUME
Newborn 15-30 min	76 ml/kg
Newborn, 24 hr.	83 ml/kg
05 - 10 kg	85 ml/kg
11 - 20 kg	80 ml/kg
21 - 45 kg	75 ml/kg
45 kg and greater	70 ml/kg

Calculation of Hematocrit

The effect on the hematocrit is calculated and packed red blood cells (RBCs) added if required. The weight in kg of the patient is multiplied by the appropriate ml from the chart. This determines the patient's blood volume. Next, the hematocrit is multiplied by the patient's blood volume to determine the RBC volume in ml. Then the RBC volume needed to have a certain hematocrit can be found. This is done by adding the blood volume and the priming volume and multiplying by the desired hematocrit. This gives the RBC volume

in ml needed. The hematocrit of packed RBCs is approximately 70% or .70. Thus, divide the number of ml of RBCs needed by .70 to find the ml of packed RBCs to be given.

If the patient weighs 5 kg, then the blood volume is 5 x 85 or 425 ml. If the hematocrit of the patient is 55%, for example, then the patient's RBC volume in ml is 425 x .55 or 234 ml. To find the ml of RBCs needed to have a hematocrit of 25% on bypass, add 425 + the priming volume, 800 ml for example. This volume (425 + 800) x .25 = 306 rounded off. The patient has 234 ml of RBCs while 306 are needed. Thus, 306 - 234 = 72 ml of RBCs needed. 72 is then divided by .70, the hematocrit of packed cells. This yields 103 rounded, the number of ml of packed RBCs needed to give the required hematocrit. Crystalloid priming solution of 103 ml is removed from the priming volume calculation to account for the added packed RBCs. The calculation now has 697 ml of crystalloid priming volume and the 103 ml of packed RBCs.

Calculation of Fibrinogen

Checking the patient's pre-op lab results for fibrinogen there is, for example, a level of 275 ml/dl. To calculate the effects of priming, find the patient's plasma volume by subtracting the number of RBC ml from the blood volume. This is 425 - 234 = 191. Another way to find this value is to subtract the hematocrit from 1 and multiply this value by 425. 1 - .55 = .45. Thus, 55% of the volume is RBCs and 45% plasma. Then .45 x 425 = 191. The patient's fibrinogen is 191 x 275 mg/100ml = 525 mg. Next, find the number of milligrams required by calculating the total amount of nonRBC volume that will be circulating on bypass or 1225 x (1.00 - .25) = 919 ml or 9.19 dl rounded off. Thus, if the goal is 100 mg per dl, then 919mg of fibrinogen are needed. Subtracting the patient's fibrinogen from the on pump level required, 919 - 525 = 394 mg that must be added to the prime. (Remember 100 ml equals 1 dl and FFP usually contains 200 mg of fibrinogen per dl.) Then 394 mg divided by 200 mg with the result multiplied by 100 gives the ml of FFP needed. In equation form 394 / 200 = 1.97 dl, then 1.97 x 100 = 197 ml. The crystalloid prime used is now reduced by the 197 ml of FFP that are added with the result being 500 ml of crystalloid prime now remaining. Precise calculating would include the 31 ml of plasma found in the packed RBCs (103 x .3 = 31) with its theoretical fibrinogen level of 62 mg, but this is usually not done.

Calculation of COP

The COP after priming can be determined by multiplying the ml of plasma by 25 mmHg and dividing the result by the total noncellular volume of the prime. The scant plasma found in the packed RBCs is again not included. The above patient has 191 ml of plasma. Also, 197 ml of FFP were added. The total plasma is then 388 ml. Applying the formula yields 388 x 25 = 9700, divided by 388 + 500 (remaining crystalloid prime) = 10.92 mmHg as a calculated COP.

Each milliliter of 12.5 gm, 25% albumin provides the osmotic pressure of 5 ml of plasma. Albumin 25% solution can create an isoncotic solution if it is added in amounts to equal 20% (not 25!) of the crystalloid prime. This gives an albumin concentration of about 5 gm per 100 ml of priming solution. Then 500 x .20 = 100 ml of 25% albumin to be added. This means that another 100 ml of the crystalloid prime should be removed from the calculation leaving 400 ml. **After the addition of packed RBCs or albumin the priming solution should not be circulated through a pre-bypass filter. These filters may become blocked by the products.**

The total prime now looks like this:

crystalloid	400 ml
25% albumin	100 ml
FFP	197 ml
packed RBC's	103 ml

Total Priming Volume: 800ml

Whole Blood

Whole fresh heparinized blood can be used to eliminate multiple exposures to FFP and packed red blood cells. However, the problem with using whole blood is that it can only be stored for a brief period. Heparinized whole blood can only be kept for 48 hours and rapidly deteriorates during the second 24 hours. This blood does have advantages in addition to the multiple exposure aspect. Heparinized fresh whole blood is commonly used in neonatal bypass because the bilirubin in this blood is much lower than that of stored blood and the citrate from CPD is not present. This is advantageous since the enzymes required to break down bilirubin are not mature in the neonatal liver. Whole blood that is anticoagulated with CPD or ACD binds the calcium in the blood to prevent coagulation. This affects the ionic calcium levels. Also, the blood is usually poor in its oxygen carrying capacity and has a low pH. Using FFP and packed cells from the same donor will minimize the risks of exposures and this is often done instead of using whole blood.

Drugs Added to Prime

The drugs listed below are used by many institutions as additives to the prime. The drug dosages may differ slightly, but these doses are representative. The particular crystalloid prime used may alter the drugs also in some cases.

sodium bicarbonate	**2mEq/100 ml prime**
heparin	**2,000 u/1,000 ml prime**
mannitol 25%	**0.05 gm/kg ***

* This is often given on rewarming. It increases the osmolarity of the glomerular filtrate which increases sodium, potassium and urine output.

Initiation of Bypass

The priming solution is warmed and oxygenated before bypass begins. Then, the initiation of bypass is done very slowly. The small volumes involved make it mandatory to work the venous occlusion and the arterial pump in unison. The arterial pump is started slowly. If a patent ductus arteriosus or other shunt is present they must be ligated by the surgeon to prevent flooding of the operative field. Flow is gradually adjusted to full calculated flow if possible. At first the patient may experience hypotension due to dilution and reaching calculated flows is not usually a problem. Most authorities feel that it is best to keep the arterial blood pressure above 50 mmHg. If the pressure does not rise to normal levels after a few minutes, it may be necessary to add antihypotensive agents such as **Neosynephrine (phenylephrine)**. Neosynephrine is a powerful and selective alpha 1 receptor agonist that causes constriction of blood vessels. Start with low doses of the medication such as 5 to 10 mcg and determine the patient response. Doses can then be adjusted.

If hypertension occurs, flows may have to be decreased while waiting for medications to take effect. Drugs commonly used for hypertension are Forane, Halothane, Regitine, Nipride, Fentanyl or sodium pentothal. Inhalation agents should be discontinued 15-30 minutes before bypass is terminated. Halothane may cause hepatic toxicity. It has been determined that as much as 68% of the Fentanyl concentration may be bound by the circuit and oxygenator in pediatric cases. Keeping the blood flow rates slightly reduced for brief periods while the blood pressure is elevated will cause no harm. The important thing is to closely observe the in-line oxygen saturation device or constant blood gas monitor to ensure adequate oxygenation.

Arterial pO_2 should not be excessively high in infants. The reasoning is that **retinal damage** is caused by excessive oxygen administration in newborns, therefore, it is also detrimental on bypass. Safe arterial pO_2 levels are debated and absolute values are not established. The danger of excessive pO_2 must be recognized though. In premature infants retinal damage has occurred when the arterial oxygen pressure was greater than 110 mmHg for over 1 to 2 hours.

Cooling and Circulatory Arrest

The pediatric patient is usually cooled to lower levels than adults because of the complex procedures and the probability of circulatory arrest. Cooling is done without allowing the arterial and venous temperatures to differ by more than **8 degrees** to ensure uniform temperature reduction. Cardioplegia is administered at low pressures to prevent damage to the pediatric heart. Circulatory arrest is often performed on pediatric patients. Many pediatric patients have experienced this event and years later suffer no aftereffects. The "safe" periods of circulatory arrest are dependent on the temperature of the patient. Colder patient temperatures allow longer circulatory arrest times. Electroencephalographic activity disappears at **15°-20° C**. The surgeon is informed of the arrest time every few minutes and the head of the patient may be packed in ice. The following table is widely used for acceptable times for circulatory arrest. **The values are not absolute. The final decision is that of the surgeon.**

PATIENT TEMPERATURE	ARREST PERIOD
32 degrees C	under 10 min.
28 degrees C	10 - 19 min.
18 degrees C	20 - 45 min.
<18 degrees C	46 - 65 min.

The perfusionist has time to prepare the patient for the acidosis that occurs with circulatory arrest. The patient should be brought into an alkalotic state just before the arrest period begins. This may entail giving sodium bicarbonate and blowing off CO_2 in greater amounts. Circulation of the blood through the oxygenator and available circuit can be done during the arrest period if an **A-V bridge** is incorporated in the pump circuit. This circulation allows the blood in the oxygenator to again be **brought to an alkalotic state** with low pCO_2 before flows are resumed. The blood can be warmed also at this time but care should be taken not to create a gradient greater than 8 degrees between the patient and the perfusate upon reinstitution of bypass. Blood at colder temperatures contains more oxygen in solution than warm blood does. Rapid warming of the blood would cause oxygen to come out of solution and create bubbles.

Renal Function

Low urine output is common in pediatric patients, but some urine output is desirable. If the mannitol in the prime is not sufficient to promote urine output, it may be necessary to give Lasix. It is usually given in doses of 0.25 mg/kg and then increased if no response occurs. An adequate arterial

blood pressure during the case has a great influence in providing sufficient urine output. However, the kidneys may tolerate no blood flow for as long as 90-120 minutes without damage.

Termination of Bypass

The patient is rewarmed and the patient's blood gases and electrolyte levels are corrected if necessary. The ventilator is turned on and the termination of bypass started. Bypass is terminated very carefully due to the small volume and anatomy to work with. It is very easy to distend the heart by overfilling, causing permanent damage. An adequate arterial blood pressure is an indication of a successful termination. The arterial blood pressure and the EKG are checked closely after termination. The perfusionist should be prepared to go back on bypass. It is not unusual to have to resume bypass in these sometimes, complicated cases. Heparin is reversed with Protamine in the usual way.

NOTES

Pediatric Worksheet

Patient name_____
Weight_____ **Height**_____**BSA**_____
Calculated Blood Flow_____
Hematocrit_____**Fibrinogen**_____**Platelets**_____

Prime
Filter_____
Oxygenator_____
Tubing_____
Reservoir_____
Total_____

ml of blood per kg from table

_____

Bood volume (BV) = ml of blood per kg x weight (kg)

_____

Red blood cell volume = blood volume x Hct

_____

Total circulating volume (TCV) = BV + prime

_____

Hct on CPB if no blood is added = RBCV / TCV

_____

RBCV needed on CPB = TCV x desired Hct

_____

ml of RBCs to add = RBC needed on CPB - RBCV

_____

ml of packed RBCs to add = ml of RBC to add / .70

_____

Plasma volume (PV) = BV - patient RBCV

_____

Patient fibrinogen in mg = PV x (fibrinogen / 100)

_____

Total fibrinogen needed = TCV* - RBCV (fibrinogen requirement is 1 mg/ml of nonRBC prime)

_____

Fibrinogen to add = total fibrinogen needed - patient fibrinogen

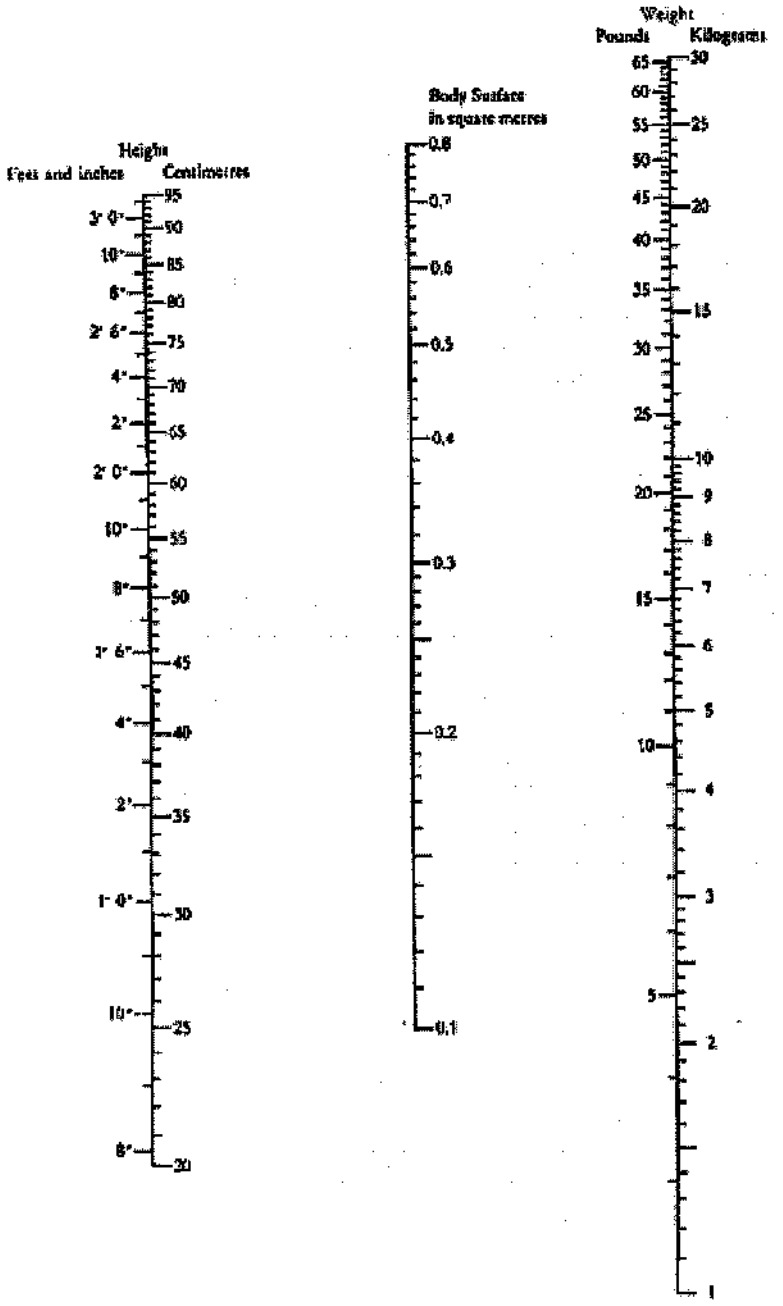
_____

FFP to add in ml= (Fibrinogen to add/200) x 100

_____

The remaining prime volume is normally crystalloid. 1/5 of this crystalloid volume should be replaced with 25% albumin.

Body Surface Area (BSA) for Infants and Children



NOTES

Congenital Pathology

Surgery to correct congenital abnormalities is usually performed on pediatric patients. Perfusionists should understand the nature of the pathology and its implications for perfusion support and myocardial management. Special techniques, such as circulatory arrest, may be necessary in many of the procedures.

Shunting of blood occurs in most congenital defects and is an important aspect of cardiac surgery. Congenital defects, early on, will usually have a shunt of left to right due to the more powerful left side of the heart dominating. Later as pulmonary resistance develops the pulmonary vascular changes ,and the shunt may eventually change to a right to left shunt. Defects that are shunting right to left are usually inoperable because the right ventricle will not be able to pump against the increased pulmonary resistance and will fail.

Atrial Septal Defect (ASD)

Atrial septal defects may be found in children and adults. These defects are openings between the left and right atrium. Corrective surgery is ASD closure. There are 3 types of ASDs:

1. **Sinus venosus** - found high in the atrial septum near the superior vena cava. This can be associated with partial anomalous pulmonary venous drainage.
2. **Ostium secundum** - found in the central portion of the atrial septum in the fossa ovalis. This is the most common type of ASD.
3. **Ostium primum** - found low in the septum. This is a type of endocardial cushion defect. This is also known as partial or complete A-V canal.

Cor Triatriatum

A third atrium is created by a septum dividing the left atrium into two chambers. Pulmonary veins flow into a posterior chamber. Surgical correction is removal of the septum dividing the chambers.

Ventricular Septal Defect (VSD)

VSDs are openings between the left and right ventricle. Corrective surgery is closure of the VSD. A complication that may occur with closure is complete heart block. Occasionally grafts are used to repair large defects. VSDs are of 4 types:

Type I - found between the crista supraventricularis and the pulmonary valve. (sub-arterial, aortic)
Type II - found caudal to the crista supraventricularis. (perimembranous VSD, most common)
Type III - found under the septal leaflet of the tricuspid valve. (inlet VSD)
Type IV - found in the muscular septum near the apex of the right ventricle. (muscular VSD)

Left Ventricle-Right Atrial Communication

VSD that connects the left ventricle with the right atrium. Corrective surgery if attempted is closure of the opening. Surgery is not often successful.

Patent Ductus Arteriosus (PDA)

The PDA is a connection between the descending aorta and the left pulmonary artery. Following birth the ductus arteriosus should close. In cases where it does not this condition results. Corrective surgery is PDA ligation. CPB usually is not necessary in isolated PDA.

Coarctation of the Aorta

Narrowing of the aorta that may occur at any location. Most are located in the proximal descending aorta near the ligamentum arteriosum. A bicuspid aortic valve is often present. Corrective surgery in adults and older children is excision of the coarctation with end to end anastamosis if possible. In infants a subclavian arterial flap is used to prevent restenosis or further aneurysm formation. This is done using the infants proximal left subclavian artery to enlarge the coarctation.

Idiopathic Hypertrophic Subaortic Stenosis (IHSS)

Reduced size of the outflow tract from the left ventricle due to excessive muscular tissue. This condition can be caused by the presence of a membrane inside the left ventricle partially blocking flow through the valve. Corrective surgery is removal of the muscular tissue or membrane.

Aortopulmonary Window

An opening previously made between the aorta and the pulmonary artery (PA), characterized by a large left to right shunt. Corrective surgery is closure of the aorta and pulmonary openings, and an alternative procedure to improve pulmonary blood flow.

Truncus Arteriosus

The aorta and PA both come off the same truncus which has only one ventricular valve. A VSD must be present for survival. Corrective surgery is the **Rastelli Procedure**. This procedure combines the closure of the VSD with a valved conduit from the right ventricle to the PA. This diverts all blood from the left ventricle to the aorta and stops all flow from the right ventricle to the aorta.

Transposition of the Great Vessels

The aorta comes off the right ventricle and the pulmonary artery (PA) comes off the left ventricle and is behind the aorta. The right ventricle pumps deoxygenated blood through the aorta to the body, not to lungs, while the left ventricle pumps blood through the pulmonary artery to the lungs and back to left atrium and ventricle again. An ASD, VSD or PDA must be present for survival. Corrective surgery can be accomplished by the following procedures:

1. **Senning Procedure and Mustard Procedure** - These are both atrial partitioning procedures. They rearrange the atrial septum to have pulmonary venous blood from the lungs go to the right ventricle and systemic venous return go the left ventricle, correcting the defect.
2. **Jatene Procedure** - Switches PA and aorta by making transverse incisions through both vessels and reattaching each to the opposite vessel. The coronary arteries are switched to the aorta with its new attachment to the left ventricle. (Treatment of choice)
3. **LeCompte Maneuver** - Switches PA and aorta as in the Jantene, leaving the PA and its right and left branches in front of the aorta.

Total Anomalous Pulmonary Venous Return (TAPV)

Oxygenated blood from the lungs returns to the right side of the heart. An ASD must be present for the infant to survive. There are three types of TAPV:

1. **Supracardiac** - oxygenated blood enters a left superior vena cava, innominate vein and then the superior vena cava. This is the most common type and accounts for 50% of patients.
2. **Cardiac** - oxygenated blood enters the coronary sinus. This accounts for 30% of patients.
3. **Infracardiac** - pulmonary veins combine into a venous trunk that goes into the inferior vena cava or portal vein. Corrective surgery is the anastamosis of the pulmonary venous tract to the left atrium and closure of the ASD.

Pulmonary Stenosis

Decreased flow through the pulmonary artery. This abnormality may be valvular stenosis, infundibular stenosis, PA stenosis or peripheral pulmonary stenosis. Corrective surgery removes the stenosis, improving PA flow. A pericardial patch may be used if necessary for enlargement.

Valvular Pulmonary Atresia

Failure to develop the pulmonary valve, but normal development of the right ventricle and PA. Corrective surgery involves reestablishing normal direct pulmonary artery blood flow.

Arterial Pulmonary Atresia

Failure to develop PA, pulmonary valve and right ventricle. Corrective procedure is the **Fontan Procedure**.

Tricuspid Atresia

The tricuspid opening is not present. There is no flow from the right atrium to the right ventricle. An ASD and PDA or VSD must be present for the infant to live at all in this condition. The Rashkind Procedure can be performed in the cath lab without CPB to create a large ASD until corrective surgery. Corrective surgery is the Fontan Procedure. A Rastelli procedure can be performed if flow from the left ventricle is directed to the right ventricle and graft. Palliative procedures to increase pulmonary artery flows are:

Waterston Shunt - side to side anastamosis of ascending aorta to PA
Blalock-Taussig - subclavian to PA
Potts Procedure - side to side anastamosis of descending aorta to left PA
Glenn Procedure - shunt between the superior vena cava and right PA.

Double Outlet-Right Ventricle

The aorta and PA both come off the right ventricle. This condition is similar to tetralogy of Fallot. A VSD is always present. Corrective surgery is construction of a tunnel to direct all blood from the left ventricle into the aorta.

Ebstein's Malformation

The tricuspid valve is lower than normal creating a smaller right ventricle and larger right atrium. Massive right atrial enlargement with symptoms of right sided failure may occur. Corrective surgery is atrioventriculoplasty, annuloplasty and possible valve replacement.

Endocardial Cushion Defects

Various defects caused by failure of embryonic cushions to close resulting in partial to complete communication between the atrium and the ventricle. There are 2 types of defects:

1. **Ostium primum defect** with ASD, cleft through mitral and tricuspid valves causing a single valve for both sides of the heart. A VSD is present also. Corrective surgery is closure of the septal defects and repair of mitral and tricuspid valve clefts.
2. **Incomplete type** with ASD and clefts in the mitral and tricuspid valves leaflets.

Hypoplastic Left Heart Syndrome

The left side of the heart does not develop. There may be a range of defects including a small left ventricle, aortic valve atresia, hypoplasia of the aorta and mitral valve atresia. Corrective surgery is the staged Norwood Procedure. In the first stage the larger pulmonary artery is used to reconstruct the hypoplastic aorta to provide adequate flows to the arterial circulation while a graft goes from the aorta to the PA to provide pulmonary blood flow. In stage two, a Fontan Procedure is done and the systemic arterial to PA shunt is closed.

Double Aortic Arch

The ascending aorta splits into two parts and goes around the trachea and esophagus on both sides and rejoins to form the descending aorta. This is the most common of the pathologic vascular rings that surround the trachea and esophagus. Pressure on the trachea or the esophagus causes respiratory and swallowing difficulty and correction is required. Corrective surgery is elimination of one branch of the aorta.

Trilogy of Fallot

Consists of an ASD, VSD and pulmonary stenosis. Corrective surgery, if attempted, is relieving the pulmonary stenosis and closure of the ASD. Palliative procedures to increase pulmonary artery flow are:

Waterston Shunt - side to side anastamosis of ascending aorta to PA
Blalock-Taussig - subclavian to PA
Potts Procedure - side to side anastamosis of descending aorta to left PA
Glenn Procedure - shunt between the superior vena cava and right PA.

Tetralogy of Fallot

Broad classification that consists of four defects including VSD, pulmonary stenosis, right ventricular hypertrophy and an overriding aorta. (The aorta overrides the right ventricle outflow partially.) Severity may range from acyanotic left to right shunts to cyanotic right to left shunts. Corrective surgery is relieving the pulmonary stenosis by excising the stenotic portion and closure of the VSD.

Pentalogy of Fallot

Broad classification that consists of five defects including VSD, pulmonary stenosis, right ventricular hypertrophy, ASD or patent foramen ovale and an overriding aorta. Severity may range from acyanotic left to right shunt to cyanotic right to left shunt. Corrective surgery is relieving the pulmonary stenosis by excising the stenotic portion and closure of the VSD and ASD.

NOTES

Circuits

The circuit of the heart-lung machine refers to the disposable tubing that forms the path that the blood follows. The main circuit is formed by the venous cannula, tubing to the reservoir, oxygenator, back through the arterial tubing to the patient. Ancillary components of the circuit permit functions such as venting, suction, cardioplegia delivery and other tasks. Perfusion circuits vary from institution to institution, but there are components common to all circuits. The perfusionist is responsible for designing a circuit or tubing pack that meets the requirements of his or her institution. The components of the circuit and their functions and effects on the circuit must be understood. An added component may affect other parts of the circuit in unforeseen ways.

Safety is the major consideration when designing a circuit. Circuits currently in use should be continuously analyzed for safety, also. Simplicity is the greatest ally of safety. The simpler a circuit is, the less chance there is of an accident. Fewer components of a circuit, mean that there are fewer things that can malfunction. There are certain functions that are required and components for these tasks must be included in the circuit. However, it is wise to meet the requirements without the complications of excess.

Design Considerations

Safety can require measures that encompass all facets of the design. Various construction techniques such as bonding or banding connections can add to the protection provided to the circuit. Including certain items in the circuit can also add to the protection. For example, arterial filters and pre-bypass filters are items that greatly promote safety. Monitoring devices also add to the protection of the patient by providing continuous input of critical information. These monitoring devices include the arterial line pressure indicator, arterial and venous saturation displays and continuous hematocrit display. The placement of other common safety devices such as low level detectors and bubble detectors must also be considered when designing the circuit. Their placement may dictate certain aspects of the circuit such as the length of the tubing.

Items that are **required** in the circuit due to the surgical techniques or other reasons require planning. Some surgeons may want certain capabilities such as extra suckers or vents. Others may want the ability to take blood from the patient before going on bypass or various cardioplegia delivery methods. The type blood gas samples obtained, the location where the pump is primed and the position of the pump in the operating room are all considerations that may affect the circuit design. The type oxygenator used is a major factor to consider. The location of the ports may affect the circuit in various ways.

Next, items that make the circuit more **convenient** to use should be considered. These convenience items may also make the circuit safer. These are items such as the A-V crossover bridge. A "snake skin" cover over the A-V loop is one of these items that makes things easier by allowing priming and inspection of the loop before it is passed to the surgeon.

A discussion of the design of the circuit should be held to gather everyone's desires and ideas. The surgeons should always be involved in such a discussion. They may present certain provisions that require much planning or may be impossible to accomplish under certain conditions. The perfusionist may be able to explain the limitations and offer other suggestions. On the other hand, the surgeon may explain why a certain facet is done better another way to influence the design. Little things as well as major things should be decided. The coding of the circuits is one of these little things. Should the suckers be called one, two and three or should they be called red, yellow and blue?

A description of the components of the circuit is presented in the following section. In the Resource Information Appendix companies are listed that manufacture circuits. Their representatives will assist in the circuit designing process. They can provide drawings, offer suggestions and construct non-sterile circuits for evaluation.

Designing the Circuit

The heart lung machine should be placed in the OR to facilitate measurements of the arterial, venous and sucker lines. The cardioplegia line requirement should also be measured. It may be that a certain cardioplegia set will not have a long enough line. Measure the lines and follow the blood path to consider each section of the circuit. The following lists the sections of the typical adult circuit and provides comments about each:

1 - Venous line - tubing size - 1/2" ID - This tubing connects the venous cannulae to the venous reservoir. It should contain the **saturation / hematocrit probe**. This probe is a very important component and should be a minimum standard of care. Some teams use an in-line constant blood gas monitoring system that provides more information but at an added cost. Place the probe 10 to 12 inches from the reservoir. The venous saturation is an indicator of adequate perfusion. A hematocrit display provides a constant reading of the patient's red blood cell volume percentage. A **venous sampling port** should also be included. If the oxygenator does not have one on the venous inlet, incorporate it with a straight connector with luer port. This is a means of checking the venous saturation if the monitor values are in doubt.

2 - Arterial pump line - tubing size 3/8" ID - This tubing will differ depending on whether a roller or centrifugal pump is used. On the roller pump it will run from the venous reservoir outlet through the pump head to the venous inlet on the oxygenator. Measurement of this is made easier by placing

a piece of tubing along the path. Allow for the length around holders and masts. Consider using a larger piece of tubing, called a "**boot**," through the pump head. A boot is usually 1/2" tubing. Although it will add two connectors, one to step up and one to step down the tubing size, it decreases the rpms required to pump the same amount of volume. Some companies produce an arterial tubing that has the increased size built in so that no connectors are required. A boot reduces wear on the pump and the tubing. Reduced wear on the tubing in the pump head is important due to **spallation** (breakdown of tubing from the inside and subsequent release of particles). On the centrifugal pump the tubing runs from the venous reservoir outlet to the inlet on the pump head, then from the outlet on the pump head to the venous inlet on the oxygenator. A **flow probe** is placed in the tubing coming from the outlet of the pump head.

3 - Arterial outlet line - Tubing size 3/8" ID - This tubing runs from the arterial outlet on the oxygenator to the Y-connector prior to the arterial filter. This usually comes connected to the arterial filter assembly. This section will contain the **arterial saturation probe** or the inline constant blood gas monitoring probe. This probe monitors oxygenator function. Should the value drop suddenly, the cause must be determined. Although this gives good information, it really requires concomitant venous saturations to interpret the information. If cost is a concern and only one probe can be afforded, the venous probe gives more information.

4 - Arterial filter assembly - Types of filters will be discussed in Chapter 21. The important thing to say is, there is one. This is a final barrier for particulate matter and air emboli. Tubing size, 3/8" ID - A Y-connector before and one after the filter allow for a bypass loop around the filter. Here tubing length is important to allow a smooth blood path without kinks that increase resistance. The bypass loop is used to prime the filter, then clamped out during the case. Placing a **straight connector w/luer** in this loop makes it an excellent place to measure line pressure. This could be done from other locations, but this seems to keep the pressure tubing out of the way. The filter bypass loop is clamped distal to the connector with the luer port prior to bypass. This reduces chances of an accident. If the clamp handle is pointed toward the pump, it is out of the way and may prevent accidental removal. This location of the clamp gives a good pre-filter resistance pressure. The pressure monitoring tubing used will depend on the type of monitoring device used. In all cases there should be some type of **air-fluid separator** to prevent contact with the non-disposable monitor and contamination of the circuit. The best monitor should provide both visual and audible alarms. If the monitor has the capacity to set limits at which it will alarm and/or cut off the pump, set it low enough to allow time to react. In most cases line pressure will approximately equal the pressure gradient across the arterial cannula, the filter gradient and the mean arterial pressure. This is because most perfusionists measure arterial

line pressure before the arterial filter. If pressure is measured before the oxygenator, then the pressure drop across the oxygenator must be added. In any case set the line pressure alarm limit higher (75-100 torr) than the calculated operating pressure. If a system with both audible and visual alarms is not available the visual display must be monitored closely. If the audible warning is not available, serious consideration should be given to changing to a monitor that provides this advantage against accidents. The distal Y-connector connects the filter to the arterial line.

5 - Arterial line - tubing size 3/8" ID - This tubing connects the arterial filter with the arterial cannula. In most circuits, the arterial and venous lines are connected to allow for recirculation of the prime and de-airing of the circuit prior to connection to the patient. These pieces are connected by either a 1/2" x 3/8" connector or a pre-bypass filter to form the **A-V Loop**. The A-V loop can be packaged in a coil that is double wrapped with ends protruding to allow arterial and venous connections at the pump. In some cases the ends of the tubing are passed off the table and the coil remains on the sterile field. Another method is to cover the arterial and venous lines in clear thin coverings that come together at the connector or pre-bypass filter. This area is then covered in a blue wrap to maintain sterility. Once the lines are primed and ready to be passed up, the blue wrap is removed to allow the surgeon to grasp the sterile lines inside. The clear coverings are then peeled back to allow sterile tubing for the table. This is an easy and convenient method that allows much freedom when working with the A-V loop. Another item that should be mentioned in this section is **the arterial-venous crossover line**. Although this is an item of convenience, it also provides features that are helpful in emergencies. It is usually a piece of 3/8" tubing that runs from a Y-connector distal to the filter to a Y-connector in the venous line. A line connecting the arterial and venous lines may be dangerous if care is not taken. This line is not used during bypass, so it is clamped out by placing **two** clamps on it with their handles pointing down to prevent accidental removal. During priming it is primed along with the rest of the circuit. One use of this line is a means to recirculate if air gets into the circuit. By clamping out the arterial and venous lines distal to the crossover, then opening the crossover line, recirculation to eliminate the air can be performed safely. Once finished, reclamp the crossover line and reopen the arterial and venous lines. Another important feature is the ability to transfuse through the venous cannula. Clamp
the arterial line distal to the crossover and the venous proximal to the crossover. Remove the two safety clamps on the crossover line and begin to pump. The flow will exit the arterial filter, go through the crossover line, and travel up the venous line into the right atrium. Another use is that it is easier to drain the circuit at the end of a case.

6 - Suckers and Vent lines - tubing size 1/4" ID - These lines are used for the pump suckers, aortic root vent, and left ventricular or pulmonary vent.

It is a matter of preference how these lines are wrapped. The two basic ways are either having the total length of the lines in a pack that goes to the OR table, or having part of the lines in the OR table pack and part in a pack for the pump. The advantage of the latter is that the tubing can be placed in the roller heads and connected to the reservoir before passing the lines from the OR sterile field. Remember, if this system is used, allow enough tubing length coming from the roller head to easily reach the piece of tubing that is passed off the sterile field. Also have the manufacturer place the connector in the tubing so that it is ready to be connected. Vents should have a one-way pressure relief valve, instead of a connector, placed in the tubing. These prevent damage due to excessive suction. They also prevent air emboli in the case of tubing reversed in the roller head. **Always check the valve flow direction when connecting this tubing.**

7 - Cardiotomy line - tubing size 3/8" ID - This tubing connects the cardiotomy reservoir to the venous reservoir. If a cardiotomy is used, this is where the suckers and vent lines are attached. If an open system with a hard shell venous reservoir is used, an integrated cardiotomy reservoir eliminates the need for a separate cardiotomy. With closed systems a cardiotomy reservoir is a necessity.

8 - Quick prime line - tubing size either 3/8 or 1/4" ID - The quick prime line is connected to the cardiotomy reservoir or the hard shell venous reservoir. It is used to quickly add volume to the circuit, whether for priming or while on bypass. This line can be either a single line or a line that has a Y-connector and divides to form two quick prime lines. When the priming spike has a bubble trap on it, the flow through the line is greatly reduced. Since this volume is going into the cardiotomy or venous reservoir and both are open systems, air is not a problem; therefore, the use of a bubble trap or drip chamber in a quick prime line is not needed.

9 - Gas line - tubing size 1/4" ID - This is the tubing that connects the gas flow system to the oxygenator. It should contain a bacterial filter. This tubing is often used to connect a CO_2 source to the circuit during the purge before priming. A perfusion adapter can be used to connect to the circuit during this purge.

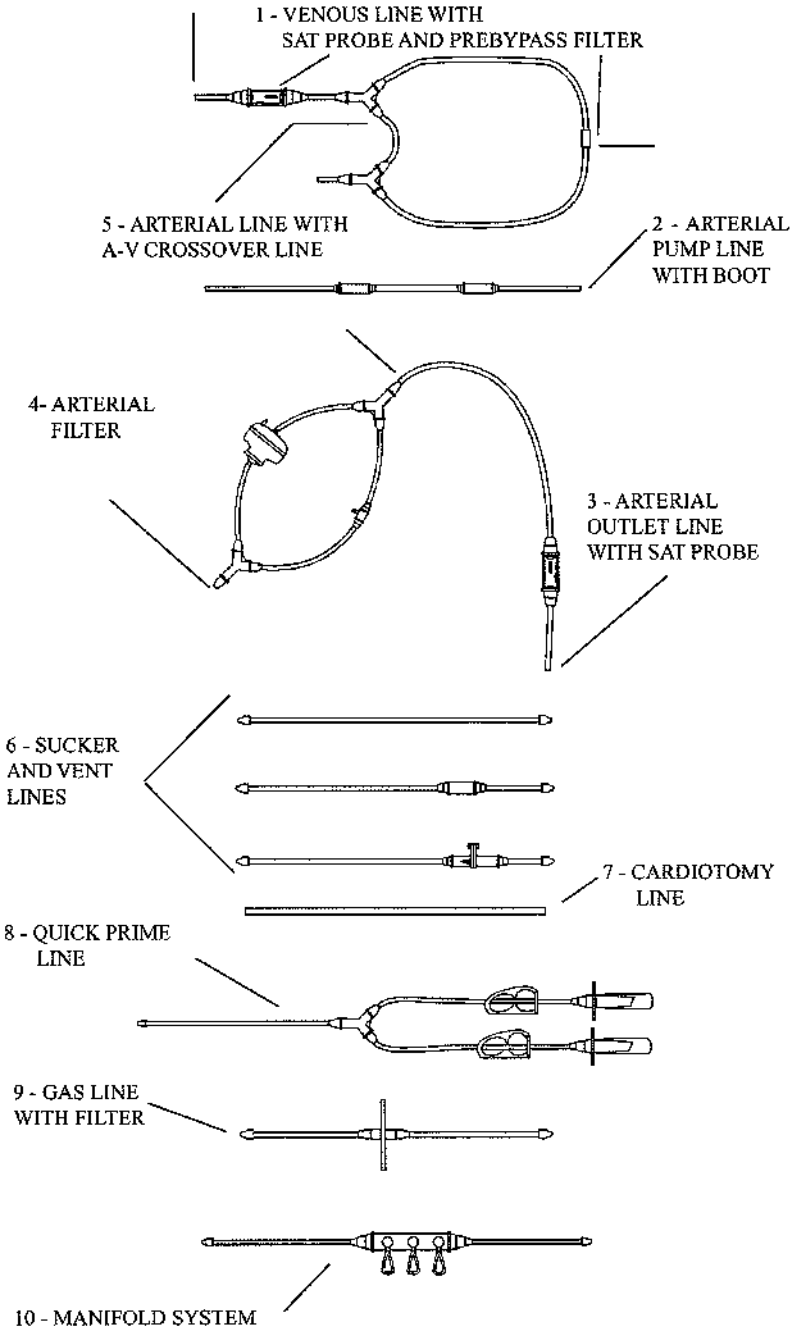
10 - Manifold system - This is a three or four stopcock manifold with tubing to connect the arterial and venous sampling ports. Some oxygenator manufacturers provide this with their products; therefore, one may not be needed for the circuit. The manifold's arterial tubing is usually connected to the purge port on the arterial filter or the arterial sample port of the oxygenator. The tubing should contain a **one-way valve** to prevent accidental injection into the arterial system. The other side of the system is the venous sampling tubing. It is connected to a venous sampling port that is either part of the venous inlet or a luer on a connector in the venous line. This line must **not** contain a one-way valve. When connected and on bypass, the system provides

a continuous flow of arterial blood through the manifold and into the venous side. This makes an excellent sampling port for blood gases and ACTs, as well as an injection site for drugs. To obtain a venous sample, simply draw enough blood back to clear the line and then obtain the sample before letting the arterial blood flush through again. The manifold is connected to an arterial port that connects to the arterial line and cannula. If the manifold is left open while cannulated and not on bypass, blood will flow through the manifold and into the venous side exsanguinating the patient. **ALWAYS KEEP THE MANIFOLD CLOSED WHEN NOT ON PUMP, AND CLOSE IT PRIOR TO COMING OFF BYPASS.**

11 - Cardioplegia Delivery System - These systems come in two basic types: custom made or off the shelf. Systems can deliver crystalloid cardioplegia or mix the crystalloid and blood in any ratio desired. Cardioplegia can be cooled through a coil in a bucket of ice or in a single pass heat exchanger. The important things to remember are to keep the system "user friendly" and ensure it meets the surgeon's requirements. For example, if cold and warm cardioplegias are to be used, select the system that is easiest to use in a particular OR with its equipment and facilities. Finally, ensure that the table line provided with the off the shelf system meets the length requirement.

Circuit Components

1. **Venous line 1/2 in ID**
2. **Arterial pump line 3/8 in ID**
3. **Arterial outlet line 3/8 in ID**
4. **Arterial filter**
5. **Arterial line 3/8 in ID**
6. **Suckers and Vent lines 1/4 in ID**
7. **Cardiotomy line 3/8 in ID**
8. **Quick prime line 1/4 or 3/8 in ID**
9. **Gas line 1/4 in ID**
10. **Manifold system**
11. **Cardioplegia delivery system**


1 - VENOUS LINE WITH
SAT PROBE AND PREBYPASS FILTER
5 - ARTERIAL LINE WITH
A-V CROSSOVER LINE
2 - ARTERIAL
PUMP LINE
WITH BOOT
4- ARTERIAL
FILTER
3 - ARTERIAL
OUTLET LINE
WITH SAT PROBE
6 - SUCKER
AND VENT
LINES
7 - CARDIOTOMY
LINE
8 - QUICK PRIME
LINE
9 - GAS LINE
WITH FILTER
10 - MANIFOLD SYSTEM

Packaging

The final part of the designing process is the packaging. Perfusionists often set up the circuit in a particular way and this affects the packing scheme. If a certain component is placed on the pump first, then it is usually first to come out of the pack. The components should be placed in the container so that they can be used in the order of the setup. The representative can offer information of packaging options. The institution may desire to place items such as connectors, cannulae or aortic tacks in the pack to reduce costs. The composition of the pack should be discussed with the surgeons and OR personnel. Surgeons must be using the same items if the inclusion of standard items is to reduce costs. If the surgeons use widely different items it is best not to include table components in the perfusion pack.

NOTES

Oxygenators

Prior to the invention of the oxygenator, heart operations were limited and had to be performed very quickly. Only limited procedures were possible. In the early 1950s attempts were made to oxygenate the blood during surgery while the heart was being repaired. Cross circulation and the use of animal lungs to provide oxygenation were attempted. These techniques allowed the heart to remain still and empty while surgery was performed to repair the organ. On May 13, 1955, DeWall and Lillehei first used their helix reservoir bubble oxygenator. In 1956 the rotating disc oxygenator was developed. This was known as the Kay-Cross apparatus. Both of these oxygenators were used with various degrees of success. In 1966 DeWall introduced the hard shell **bubble oxygenator** with integral heat exchanger. Historically, these early oxygenators are considered the devices that made perfusion practical and economically feasible. Subsequently, Lillehei and Lande developed a commercially manufactured, disposable and compact **membrane oxygenator**.

The principle of the bubble oxygenator is simple. The blood enters a venous inlet and crosses a heat exchanger. Oxygen is then bubbled through this venous blood and gas exchange occurs. The oxygenated blood then flows through a defoamer and into an arterial reservoir. The arterial blood is then returned to the patient. Bubble oxygenators were effective oxygenators and any book on perfusion is obligated to mention them. Bubble oxygenators were a very significant development in perfusion technology. However, this technology is now outdated and should not be used. Poor gas control, microemboli emission and the increased possibility of pumping gross air are disadvantages of the bubble oxygenator. The membrane oxygenator is unquestionably the standard today.

There are now a number of **membrane oxygenators** commercially available. For the most part, all these oxygenators perform adequately. They all oxygenate, remove CO_2 and cool and rewarm the patient in a reasonable time. Therefore, the following discussion is in terms common to all membrane oxygenators.

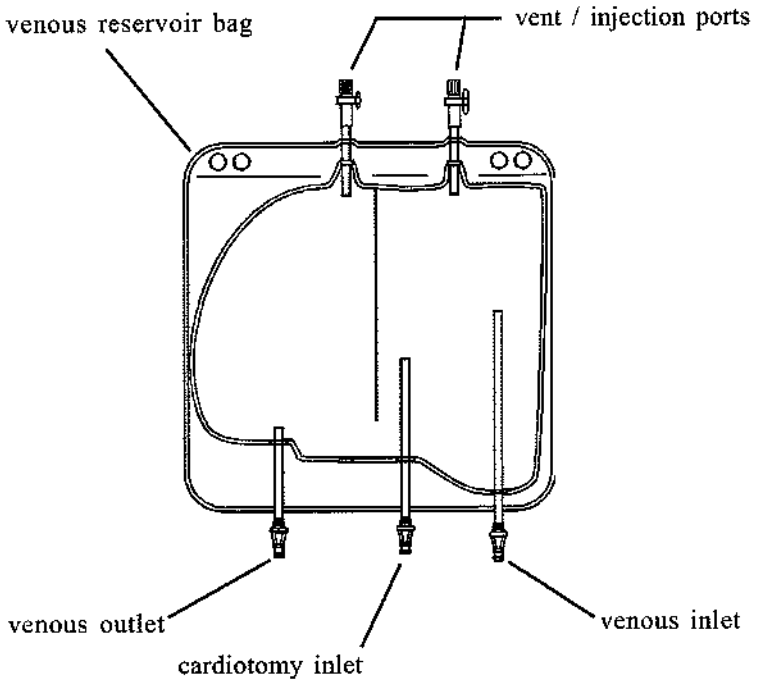
The **membrane oxygenator system** consists of three parts. These are the venous reservoir, the oxygenator and the heat exchanger. Each part is examined in this chapter.

Venous Reservoirs

The venous reservoir is a holding area for the blood as it leaves the venous line and enters the oxygenator system. There are two basic types of venous reservoirs and each has advantages and disadvantages. The two types are the venous reservoir bag and the hard-shell venous reservoir.

Venous Reservoir Bags

The venous reservoir bag is simply a bag made of polyvinylchloride, a polymer, and a holder for support. The bag has a venous inlet port, a cardiotomy inlet port and a venous outlet port. A vent port is located on top of the bag to evacuate any air that may accumulate. There is also a bubble trap of some type. Some bags use a screen, while others use a flow arrangement that decreases the chances of air being sucked out of the venous outlet.



Safety is commonly thought of as the major **advantage** of the venous reservoir bag. The bag empties and collapses when the venous return stops. The collapsed bag is supposed to prevent air from being pumped out of the bag. The flaw to this idea is that the collapsed bag does not prevent air from being pulled out of solution due to the occluded outlet line. Although the collapsed bag does prevent air from being pumped for a brief period, the reservoir bag should not offer false security. Over dependence on the bag as a safety measure is dangerous.

The are certain **disadvantages** of using the venous reservoir bag. These are the difficulty of air evacuation, volume management and increased venous return resistance. In addtion, vacuum suction cannot be applied to the venous resevoir bag. Air in the bag can also be a major problem on bypass.

Air often enters the venous line from various sources. For instance, air from around the purse string suture of the venous cannula may enter the venous line and travel to the reservoir bag. Removal of this air can be time consuming and divert the perfusionist's attention from more critical operations. A purge line from the bag to the cardiotomy may facilitate easier air removal, but this is still a difficult problem.

The relatively small size of the venous reservoir bag is another disadvantage. Volume management of large amounts requires that the cardiotomy be used as a holding area. The bag has a holding volume of about 3000 ml. Volume over that amount must be held in the cardiotomy reservoir. This requires periodically clamping the cardiotomy line that drains into the reservoir bag to allow volume holdup to occur. This also allows the heart to empty better due to the decreased volume in the bag. The increased resistance caused by a full venous reservoir bag is another disadvantage. The increased resistance can lead to a decrease in venous drainage. Impeded venous drainage, no matter what the cause, prevents the heart from emptying and leads to cardiac distention. Myocardial distention is detrimental to myocardial function and must be avoided.

Hardshell Venous Reservoir

The hardshell reservoir performs many of the same functions as the venous reservoir bag. One type of hardshell reservoir requires a separate cardiotomy, as the venous reservoir bag does. This type functions only as a venous reservoir. The more commonly used type has the cardiotomy reservoir incorporated into the unit. A separate cardiotomy is not necessary with this type. This type has the flexibility to be an integral part of the oxygenator or to be located separate from the oxygenator.

Hardshell reservoirs consist of a ridged shell with inlet and outlet ports. There are venous inlet and outlet ports, a cardiotomy inlet port and a quick prime inlet port. If the hardshell has an integrated cardiotomy, it will have additional ports for sucker inlets. A filter and defoamer are also included. Because the hard shell reservoir is an "**open system,**" there is less resistance to venous return. The emptying of the heart with these systems is much better than with the venous bag, or "**closed system**." Also, because this is an open system, air handling is a non-task. Air returning down the venous line enters the reservoir where it returns to room air. These hardshell reservoirs usually have a greater volume capacity (3000 to 4000 ml) than the bag type, so volume management is rarely a task. Therefore, air handling, volume management and increased venous resistance are not the problems as with a venous reservoir.

The **disadvantage**, is the obvious. The hardshell reservoir does not collapse and give the extra edge of safety, or at least the suggestion of safety, that the venous reservoir bag does. The reservoir does give an excellent view and assessment of blood volume with easy to read scales mounted on the sides of the reservoir. This makes it easier to observe the exact level of the

volume. Another possible disadvantage is that with some hardshell reservoirs, venting to the outside air is accomplished around the entire top of the reservoir. This type of venting may compromise sterility if the circuit is setup for long periods of time. Other hardshell reservoirs vent through a vent port that can remain closed until used.

The hardshell venous reservoir with the integrated cardiotomy reservoir has **other advantages.** There is some cost reduction because no cardiotomy is needed. There is the ease of setup. When using the venous hardshell reservoir with integrated cardiotomy, there is one less item to setup. When this is combined with the oxygenator to form a **one piece integral system,** ease and speed of setup are greatly enhanced. Most oxygenators with integrated hard shell reservoirs are designed to separate if it is necessary to change one of the components. For instance, if the cardiotomy malfunctions, it can be separated from the oxygenator and changed.

Cardiotomy Reservoir

A cardiotomy reservoir is also required when the venous reservoir bag is used. The cardiotomy receives the sucker return volume and added priming solution. There are inlet ports on the cardiotomy for the suckers and for the quick prime line. Cardiotomies have an integral filter and defoamer. There are non-filtered cardiotomies, but their use is usually limited to cell savers. The filter and defoamer prepare the sucker return and added volume before this blood enters the venous bag. The filter, usually 40 microns in size, removes any particulate matter. The defoamer, usually silicone, alters the surface tension of bubbles and causes them to dissipate. The height of the cardiotomy is adjusted to allow the least amount of standing air in the cardiotomy line. It can also be lowered to allow some back filling from the venous reservoir if needed. Each oxygenator manufacturer also makes a cardiotomy reservoir. When evaluating for purchase, consider features, such as, holdup volume. This is the volume that must buildup inside the filter/defoamer before breaking through and entering the reservoir section. Other features include the size of the quick prime inlet port, the number of sucker inlets, the total volume that the reservoir holds and the volume that the reservoir holds while contacting the defoamer. The constant contact with the defoamer uses it up more rapidly and is not desirable.

Membrane Oxygenators

Membrane oxygenators consist of three basic types. They are the **rolled flat plate membrane**, the **flat plate membrane** and the **hollow fiber membrane**. The only oxygenator that utilizes the rolled flat plate is the AVECOR. Made of silicone, it is the only oxygenator presently approved for long term use, (as in an ECMO case). The flat plate and hollow fiber membranes are made of polypropylene. The flat plate is used by manufacturers such as COBE® in its

CML® oxygenator. The hollow fiber membrane is the most commonly used design today.

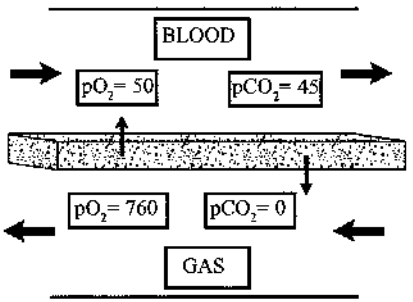
There are two different blood paths used with hollow fiber membranes. Some have the blood flow through the fibers with the gas flow outside. Others flow the gas through the fibers with the blood on the outside. Both methods work, but outside blood flow may be more efficient. This method allows more blood cells to contact the fibers and thus increases gas exchange.

Hollow fiber technology is being used in the development of the small prime, high efficiency oxygenators that are becoming the standard. All membrane oxygenators work in basically the same way. Unlike bubble oxygenators, membranes do not have direct blood-gas interfaces. They have blood flowing on one side of the membrane and gas on the other side. The membrane contains micropores that allow gas transfer to occur. These pores are formed in different processes by the various manufacturers. Some oxygenators have uniform pores and others have pores with very different shapes and sizes.

Gas Transfer

Gas transfer across membranes is due to the permeability of the membrane to the gas and the **driving pressure** of that gas. Driving pressure is a measurable physical force. It is the difference in the pressure of a specific gas on either side of a membrane. It is this difference that causes the gas to diffuse from an area of high pressure to an area of lower pressure. The driving pressure of a gas is **totally independent of other gases** that may be present. The **rate** of exchange is determined by the pressure differential of the gas on either side of the membrane and the permeability of the membrane to the gas. The greater the pressure differential is, the greater the rate of exchange becomes. The greater the permeability to the gas is, the greater the rate of exchange becomes. That leads to the formula:

Transfer Rate = Driving Pressure x Permeability



pO_2 DRIVING PRESSURE = 710
pCO_2 DRIVING PRESSURE = 45

The drawing above displays the values of gases in venous blood entering an oxygenator and the driving pressures of the gases that are exchanged. The gas transfer rates are determined by the oxygenator manufacturer. These values are listed as the **O_2 transfer** and the **CO_2 transfer rates**. The formula for the O_2 transfer rate is one that should be understood. Use it to determine if a particular oxygenator has the ability to oxygenate a particular patient or to determine if it is functioning up to standard. To understand this further, look at some of the means of measuring the oxygen values of the blood.

Formulas for Measuring O_2 Blood Values

The first measurement is **oxygen capacity.** This is the maximum amount of oxygen that the blood is capable of carrying. It is expressed as volumes % (ml O_2/ 100 ml blood). The formula for O_2 capacity is:

$$\mathbf{O_2\ Capacity = 1.34 \times Hgb + .003 \times pO_2}$$

Here the 1.34 represents the ml of O_2 carried by one gram of hemoglobin. The hemoglobin is expressed in grams percent. Therefore, by multiplying the grams in 100 ml of blood (gm%) by 1.34, the maximum O_2 capacity of the hemoglobin is found. The .003 represents the ml of oxygen dissolved in 100 ml of blood for each 1 mmHg of oxygen tension. The oxygen capacity formula assumes that the hemoglobin is 100% saturated. If the saturation is not 100%, then the amount of oxygen carried is different. This value then is known as the **oxygen content**. The formula for the O_2 content is:

$$\mathbf{O_2\ Content = 1.34 \times Hgb \times \%\ saturation\ (in\ decimal) + .003 \times pO_2}$$

NOTE: Formulas for O_2 Capacity and O_2 Content often do not consider the dissolved O_2 secondary to its minimal contribution to total capacity or content.

This is the same formula as in O_2 capacity, but now the % of hemoglobin that is saturated is accounted for. (Use a decimal value for the saturation in the formula.) When working with these formulas, values provided by the laboratory must be used to do calculations. One of these values is the oxygen saturation. This is an expression of the values just discussed. The formula for **oxygen saturation** is:

$$\mathbf{O_2\ Saturation\ (\%) = \frac{oxygen\ content}{oxygen\ capacity}}$$

These values all come into play as the oxygen transfer ability of the membrane oxygenators is evaluated. Membrane oxygenators have a fixed **oxygen transfer rate.** This is due to the manufacturer's design and membrane permeability to oxygen. The oxygen transfer rate is the amount of oxygen a membrane will transfer on each pass regardless of the venous saturation. The **gas transfer rate** is determined by the manufacturer and listed on the insert with the oxygenator. This rate is determined at a rated blood flow. By calculating actual oxygen transfer and comparing it to the manufacturer's value, one can assess oxygenator performance.

In the following example the patient has an arterial saturation of 100%, venous saturation of 80%, Hgb of 12 gm, and the flow is 6.5 LPM.

The formula is:

$$\mathbf{O_2\ transfer} = \frac{\mathbf{(A - V)\ sat\ \%\ as\ a\ decimal \times 1.34 \times Hgb \times flow\ (ml/min)}}{\mathbf{100}}$$

$$\mathbf{example:}\ \frac{\mathbf{(1.00 - .80) \times 1.34 \times 12 \times 6500}}{\mathbf{100}}$$

$$\frac{\mathbf{.20 \times 1.34 \times 12 \times 6500}}{\mathbf{100}}$$

$$\frac{\mathbf{20904}}{\mathbf{100}} = \mathbf{209\ ml/min}$$

This formula can also be used to measure **oxygen consumption** when cardiac output replaces flow. Another use is to predict the oxygen consumption of the patient preoperatively. The Fick equation is rewritten to give the formula for oxygen ,consumption as:

O_2 Consumption =

cardiac output (L/min) x [aO_2 Content - vO_2 Content] x 10

NOTE: Either formula can be used for the measurement. The O_2 transfer does not take dissolved O_2 into account.

The information pamphlet with the oxygenator supplies the O_2 transfer information. It also lists such things as the pressure drop across the membrane (the difference between the inlet pressure and the outlet pressure). Other information in the pamphlet is the rated blood flow, the prime volume, surface area and heat exchanger efficiency. The emphasis in today's market is high efficiency, low priming volume. As with the selection of all equipment, when choosing an oxygenator, requirements must first be determined before a decision can be made. Remember, overkill is not a problem when referring to performance.

Heat Exchangers

The final part of the system is the **heat exchanger**. Today, this is an integral part of the oxygenator. Therefore, they cannot be mixed and matched as with a venous reservoir. Heat exchangers are single pass systems. They are made of either stainless steel, aluminum, or polypropylene. Some are more effective in rewarming and thus may affect the choice of the oxygenator.

Listed on the chart below are some of the adult oxygenators presently on the market and their performance data. This list does not include all products available.

OXYGENATOR	RATED FLOW (LPM)	PRIME VOL (ml)	SURF. AREA M Sq	O_2 TRANSFER (ml/min @ LPM)	TYPE
BAXTER SPIRAL GOLD®	1-7	260	1.9	400 @ 7	H F
COBE DUO	0.5-8	260	1.3	240 @ 5*	F P
		460	2.6	400 @ 8*	F P
COBE OPTIMA XP	0.5-8	260	1.7	400 @ 8*	H F
COBE OPTIMIN	0.5-5	170	1.0	175 @ 5	H F
DIDECO AVANT	1-7.5	250	1.7	455 @ 7.5	H F
DIDECO COMPACTFLO	1-7.5	250	1.7	495 @ 7.5	H F
EDWARDS VITAL	2-7	180	2.0	N/A	HF
GISH VISION	1-8	298	2.45	430 @ 8	H F
JOSTRA QUADROX (D)	0.5-7	274	1.6	461 @ 7	H F
MEDTRONIC AFFINITY	1-7	270	2.5	450 @ 7	H F
POLYSTAN SAFE MAXI	1-7	280	2.0	450 @ 7	H F
SORIN MONOLYTH PRO	1-8	300	2.2	425 @ 8	H F

OXYGENATOR	RATED FLOW (LPM)	PRIME VOL (ml)	SURF. AREA M Sq	O_2 TRANSFER (ml/min @ LPM)	TYPE
TERUMO SX18 & SX18R	0.5-7	270	1.8	400 @ 7	H F
TERUMO SX25 & SX25R	0.5-7	340	2.5	500 @ 7	H F

*COBE products are tested at a barometric pressure of 620 mmHg

OXYGENATOR "TYPE" CODE: H F (hollow fiber), F P (flat plate), RFP (rolled flat plate)

THE ABOVE INFORMATION IS TAKEN FROM THE MANUFACTURERS' BROCHURES ABOUT EACH OXYGENATOR. THE O_2 TRANSFER IS READ FROM CHARTS USING A VENOUS SATURATION OF 65% AND THE OXYGENATOR'S MAXIMUM RATED FLOW. THIS IS JUST A GUIDE AND IF NEW OR IMPROVED INFORMATION IS AVAILABLE THESE NUMBERS SHOULD BE UPDATED.

NOTES

Filtration

One of the major dangers of CPB is the presence of air and particulate matter in the solution of the perfusion circuit. The particulate matter may be from the blood such as platelet aggregates, or debris from suction that is returned to the cardiotomy such as fat globules and bone chips. Other particulate matter is from the manufacturing process or the priming solutions. This danger of this air and particulate matter can be lessened by the placement of filters in the circuits. These filters are now the accepted standard. There are various types of filters in the circuit that filter the blood as well as the cardioplegia and delivered gas. A disposable pre-bypass filter is also used to filter the debris from the circuit before CPB is initiated. Blood from the blood bank is also filtered before it is added to the heart-lung machine.

Types of Blood Filters

Blood filters are constructed in three types. These are the screen, depth or a combination of both. The screen filter is a woven mesh material, usually Dacron, with a defined pore size. The filtration ability depends on this pore size. The depth filter does not have a definite pore size. It is formed by packing materials such as glass wool, Dacron wool or polyurethane foam into a space. The filtration depends on the thickness and the tightness of the packing. Emboli are trapped by the filler material. The combination filter combines the two construction methods and uses a mesh screen filter as well as depth filter material.

Arterial Blood Filters

Arterial filters are placed into housings that also affect their functions. These housings are designed to act as bubble traps. They direct the blood in such a route as to move any air emboli toward an exit port for removal. These ports are also essential in the priming and deairing of the filter. The amount of priming volume and maximum rated flow are also considerations in the design of the filters. When selecting a filter, the patient's maximum calculated blood flow should be considered. This flow should not exceed the rated flow of the filter. If it does, a filter with higher flow rates is used. Research is moving toward filters that selectively target certain items. These new filters may lead to many new concepts in filtration.

Cardiotomy Filters

Cardiotomy filters are usually combination types. Suckers return blood from the surgical field to the cardiotomy. This blood contains the usual suction debris such as fat particles and bone chips. Because of the turbulence

of the suction, there are also large numbers of air bubbles in the incoming blood. Therefore, reservoirs integrate a defoaming agent. Many manufacturers place the defoamer sponge before the filter to help prevent blockage of the filter with large particulate matter. When using a cardiotomy reservoir with a cell saver, the question of which pore size is best often arises. Some perfusionists use the **"unfiltered" cardiotomy**, which usually has a pore size of 70 to 180 microns. They then filter the blood again prior to infusing the patient. The use of a **"filtered" cardiotomy** with a 20 - 40 micron filter will eliminate more particulate matter. However, blood should always be infused through a filter. When using a cell saver, cardiotomy filters may clog quickly and require changeout often. This is more often seen in non-cardiac cases which routinely have more debris.

Banked Blood Filters

Filters for banked blood should also be used routinely on all blood added to the extracorporeal circuit. These filters are usually 40 microns in size. Although this blood is added through a cardiotomy usually incorporating a 40 micron filter, separate filtration helps prevent clogging with debris. This is especially true when multiple units of blood are given. This clogging would require the changing of a cardiotomy or a hardshell venous reservoir with an integral cardiotomy. Prevention seems to be the safer plan of action.

Non-Blood Filters

Non-blood filters are also incorporated into the perfusion circuit. Of these, the **pre-bypass filter** is the most important. There have been numerous studies showing particulate matter found in circuits. This debris is a result of manufacturing techniques. Although manufacturers have improved techniques to remove these particles through the years, some particles still remain. The use of a pre-bypass filter with a pore size in the range of 5 microns should be standard on all circuits today. **NOTE: Due to the pore size of less than 5 microns, these filters cannot be used with blood primes or albumin.**

Cardioplegia filters are available today to filter the crystalloid portion of the cardioplegia solution. These filters are in the 0.2 micron pore size range. These are used by those concerned with particulate matter that may be precipitated from the mixture of drugs forming the cardioplegia solution.

Gas Filters

Gas filters are used to remove any particulate matter that may come from the gas source, be it an oxygen or air bottle, or from wall connections with a central system. Gas filters are usually 0.2 microns in pore size. They usually have an arrow marking the direction of flow, although some models are bidirectional.

Choosing Filters

The vital information of filters should be compared when deciding which arterial filter to use. This information includes maximum flow rate, priming volume, pore size and air handling ability. The air handling ability is a major safety aspect and should be taken into account. Priming volume adds to the total volume added to the patient. In pediatric or small adult patients this amount may be important. The holder of the filter should be examined. Does it fit easily on the heart-lung machine? Does it hold the filter securely? The cost of the filter should be considered, also. Do added features justify added costs?

Listed below are some of the arterial filters available today and their vital information.

ARTERIAL FILTERS

FILTER	MAX FLOW LPM	MICRON SIZE	PRIM VOL MLS
COBE			
SENTRY	6	20 or 40	160
DIDECO / SORIN			
D 732 MICRO 20 A	7	20	195
D 732 MICRO 20 A	7	20	195
D 731 PEDIATRIC	3	20	100
D 733 PEDIATRIC	3	40	100
D 735 NEW BORN	2	20	40
D 736 NEW BORN	2	40	40
GISH			
GAF-25H-2	6	25	168
GAF-40H-2	6	40	168
GAFS-25-2	6	25	203
GAFS-40-2	6	40	203
GAFS-40-3	6	40	203

ARTERIAL FILTERS

FILTER	MAX FLOW LPM	MICRON SIZE	PRIM VOL MLS
JOSTRA			
QUART	7	40	180
PALL			
LEUKOGUARD6	6	40	220
LEUKOGUARDLG	6	40	205
PALLAUTOVENT	6	40	220
PALLAL8	8	40	165
PALL STAT PRIME	6	40	220
PALL EC3840	6	40	220
TERUMO			
CX-AF200X	6	37	200
CX-AF02	2.5	32	40

NOTES

Cannulae

The various cannulae used in surgery are important to the perfusionist. These cannulae link the perfusion circuit with the patient. The cannulae of the perfusion circuit include the arterial, venous, antegrade cardioplegia and retrograde cardioplegia. In addition, there are left ventricular vents, pulmonary artery vents and aortic root vents. Features of the cannulae are discussed in this section. These features should be considered when cannulae are chosen.

The arterial and venous cannulae often create controversy. The perfusionist wants the largest possible cannula while the surgeon prefers to place the smallest possible cannula. The perfusionist requires a certain size cannula to achieve certain flows without exceeding pressure gradients. The surgeon must limit the size of the cannula if surgical considerations make it imperative.

Arterial Cannulae

The **arterial cannula** used is determined by evaluating the **flow and pressure drop charts.** The pressure drop (the difference between the pressure entering the cannula and that leaving) shows resistance. The greater the pressure drop, the greater is the resistance. This is usually inversely related to the size of the cannula. With higher pressure across the cannula, there is an increase in jetting at the tip that can cause intimal damage to the aorta and hemolysis. The accepted limit of pressure drop is 100 mmHg. This should not ordinarily be exceeded. By examining the charts the manufacturer provides with the cannula, determination of maximum flow can be made. The charts found later in this chapter will list some of the common arterial cannulae used and their maximum flows.

Venous Cannulae

Venous cannulae flows are by gravity drainage. The higher the pressure drop, the greater is the resistance to flow. Therefore, the lower the pressure drop, the better the flow is. The pressure drop is inversely related to size. Generally, the larger the venous cannula, the lower the pressure drop, and the better the flow is. The most common venous cannula used is the **two stage cannula**. It has an open tip that is placed in the inferior vena cava (IVC), and more proximal openings that will be located in the right atrium. This produces only partial bypass and may require venting of the heart to prevent distention. There are large single stage cannulae that may be placed in the right atrium. These do not always drain as well as a two stage, but are sometimes used when a two stage cannula will not go into the IVC. The other method of venous cannulation is with two **single stage cannulae.** One is placed in the superior

vena cava (SVC) and the other in the IVC. These are connected to a common venous line with a Y-connector. This type of cannulation also causes the effect of partial bypass as the two stage cannula does. That is some blood manages to go around the cannulae, through the heart and to the lungs. When the cannulae in the SVC and IVC have either a clamp or umbilical tape pulled around them, all blood coming to the heart is diverted to the cannulae. This is termed **total bypass.** This manner of venous cannulation is most often used in valvular or congenital surgery. A venous cannulae chart will be listed later in this chapter. This chart suggests the cannulae used with certain weight categories.

Cardioplegia Cannulae

The other cannulae used during bypass are specific to certain procedures. **Retrograde cardioplegia** is a popular technique of giving cardioplegia. The cannula is placed into the coronary sinus through the right atrium. The cannula has a balloon near its tip that when inflated prevents the flow of the cardioplegia back into the right atrium. The flow is, instead, forced backwards through the coronary veins, capillaries and arteries. The cannulae are of two basic types: either automatic or manual balloon inflation. Selection is surgeon preference.

Antegrade cardioplegia is given through a cannula in the aortic root or directly into the coronary os. (When given directly into the os a coronary perfusion cannula is used.) These cannulae come in various sizes and in different configurations. Basically, they have short needle tips that are placed into the aorta. Cardioplegia is then given into the root. The aortic valve and the aortic cross clamp prevent flow in either direction and thus force the cardioplegia into the coronary arteries. Some have an extra arm coming off the side to allow a vent tubing to be connected and thus provide both functions in turn. The sizes of these cannulae affect the pressure drop and thus the maximum flow. Selection is surgeon preference.

Coronary perfusion cannulae come in different sizes and shapes. A common design is the small hand held cannula with a soft tip that is placed over the coronary os. Others have tips that engage the coronary os. These cannulae are used when the aortic root is opened for a valve replacement. Since the development of the retrograde cannula these cannulae are not often used. Retrograde delivery is easier and may be done while the surgeon continues to work.

Vents

The **LV vents, PA vents** and **aortic root vents** are the last of the cannulae types to be discussed. These vents are available in many sizes and shapes. The type of vent depends on the cannulation site. There are short metal tipped needles that fit in the aortic root. There are long thin cannulae placed through

the right superior pulmonary vein, through the mitral valve and into the left ventricle. There are short right angled cannulae that may be placed into the apex of the left ventricle. There are cannulae placed in the main pulmonary artery distal to the pulmonary valve. The selection of these cannulae depends on the vent placement the surgeon prefers and the type cannula he prefers.

NOTE: Always use a one-way valve in pump tubing connected to a vent to prevent accidental introduction of air into either the aorta or the left ventricle.

The following is a chart of arterial cannulae, their pressure drops and maximum flows. These values come from flow charts provided with the products. The venous cannulae chart is one that lists the standard recommendations at certain weights. The charts are general recommendations and more specific information can be obtained from the manufacturers.

NOTE: On the arterial charts many cannulae reached the maximum flow on the chart before reaching the 100mmHg pressure drop. In these cases the maximum flow listed is actually less than its maximum flow that could be achieved at the 100mmHg pressure drop.

ARTERIAL CANNULAE

model	size(OD)	flow (L/min)	pressure drop mmHg
Bard			
Opti-Clear Aortic Arch	18 FR	4.75	100
Opti-Clear Aortic Arch	20 FR	6.25	100
Opti-Clear Aortic Arch	22 FR	7	70
Opti-Clear Aortic Arch	24 FR	7	50
Bio Medicus - Femoral			
96530-015	15 FR	3	100
96530-017	17 FR	4	100
96530-019	19 FR	5.5	100
96530-021	21 FR	6.5	100
DLP - curved tip arterial			
model 83020	20 FR	6.5	100
model 83022	22 FR	8	81
model 83024	24 FR	9	68
DLP - descending arch cannula			
71321	21 FR	5	100
71324	24 FR	6	100

ARTERIAL CANNULAE

model	size(OD)	flow (L/min)	pressure drop mmHg
Gish-Curved tip, wire reinforced, with vent plug			
RA-1116	18FR	5.2	100
RA-1117	21FR	6	56
RA-1118	24FR	6	31
Gish-Straight tip with vented connector			
NA-2116	18FR	5.2	100
NA-2117	21FR	6	56
NA-2118	24FR	6	31
Research Medical			
ARL-2011-90-TA	20 FR	5.9	100
ARL-2211-90-TA	22 FR	6	60
ARL-2411-90-TA	24 FR	6	35
Research Medical - Femoral arterial cannulae			
TF-A-020-25	20FR	3.3	100
TF-A-022-25	22FR	5.25	100
TF-A-024-25	24FR	6.2	100
TF-A-024-25-H	24FR	7	87
Sarns			
High Flow Aortic Arch	3.8mm	1.5	100
High Flow Aortic Arch	5.2mm	3.5	100
High Flow Aortic Arch	6.5mm	5.25	100
High Flow Aortic Arch	8.0mm	8	60
Sorin			
A211-30	3.0mm	1.2	100
A211-38	3.8mm	1.8	100
A211-45	4.5mm	2.9	100
A211-52	5.2mm	4.1	100
A211-65	6.5mm	6.3	90
A211-80	8.0mm	6.5	40
A211-95	9.5mm	6.5	20

model	size(OD)	flow (L/min)	pressure drop mmHg
William Harvey - Bard Arterial			
Type 1858	18 FR	4	100
Type 1858	20 FR	5.5	100
Type 1858	22 FR	6	85
Type 1858	24 FR	6	70
William Harvey - Bard Arterial			
Type 1860	18 FR	4	100
Type 1860	20 FR	5	100
Type 1860	22 FR	6	85
Type 1860	24 FR	6	50

VENOUS CANNULAE

weight in kgs	SVC/IVC Cannulae in French size	RA Cannula in French size
0-3	12 / 12	18
3-6	12 / 14	18
6-8	14 / 14	20
8-10	14 / 16	22
10-12	16 / 16	24
12-15	16 / 20	24
15-20	20 / 20	26
20-25	20 / 24	28
25-30	24 / 24	28
30-35	26 / 26	30
35-40	28 / 28	32
40-50	30 / 30	36
50-60	30 / 32	36
60-70	32 / 32	51
70-80	34 / 34	51
80-90	34 / 36	51
90-100	36 / 36	51
100-110	36 / 38	51
110-120	38 / 38	51
120-130	38 / 40	51
130-140	40 / 40	51

NOTES

Intra-operative Transesophageal Echocardiography

The development and subsequent intra-operative use of two-dimensional transesophageal echocardiography has greatly improved our understanding of cardiovascular structure and function. Its advantages compared to transthoracic including improved resolution, lack of interference with the surgical field, and ability to be used continuously make it ideal for use in the cardiac surgical patient. Transesophageal echocardiography (TEE) now sees widespread use throughout the country and is considered by many as standard-of-care monitoring for some cardiac surgical procedures. This chapter offers a basic introduction to the principles of operation, modes of function, anatomic views, and intra-operative applications of this monitoring system.

Principles of Operation

All forms of echocardiography are based on transmission and reception of ultrasonic waves (2.5 - 10 MHz) from a vibrating piezoelectric crystal. As the ultrasonic waves are transmitted from one structure or cavity to another, varying amounts of reflection occur related to the individual structures acoustic impedance and homogeneity. Homogeneous blood containing structures will appear as echo-free areas (black), myocardium being somewhat more echogenic will appear grey, and areas of lung or air will appear strongly echogenic (bright white).

Various forms of Doppler echocardiography are based on the Doppler principle. As ultrasonic waves at a given frequency are reflected from moving structures such as blood cells. A change in their frequency occurs related to the speed and direction of the source of reflection. Measurement of transmitted and reflected waves from vascular structures can yield information concerning velocity and direction of flow.

Modes of Operation

Various modes of operation are available from most systems, each with certain advantages and disadvantages.

1. M-mode - This mode provides a single dimensional, "ice pick" view of the heart over time. Only structures within the narrow beam can be visualized, however, correlation with a rhythm strip can ease the evaluation of structures through the entire cardiac cycle.

2. 2D-Mode - By using a rapidly changing directional crystal or sequentially excited array of crystals (commonly 64 in adult probes), a two-dimensional, single plane view can be obtained. This allows real-time imaging of cardiac structures.

3. Multi-Plane 2D-Mode - By using separate transverse and longitudinally oriented crystal arrays (Bi-Plane), or a controllable mechanically rotating array (Omni-Plane), multiplane images of the same structure can be obtained, thus improving the diagnostic quality of structures which can be visualized.

4. Pulse Wave Doppler - Short bursts of ultrasound are transmitted and received from the same transducer to a specific location within the 2D image. This allows for evaluation of flow characteristics within a vascular structure, although accuracy of velocity measurements at high flow is poor.

5. Color-Flow Doppler - This technique involves computer processed pulse wave Doppler information superimposed onto a 2D-mode image. The result is a color pattern where particular color represents flow direction (toward the probe, usually red, away from the probe- usually blue, turbulent flow-multi color mosaic) and brightness represents velocity. This technique is commonly used to identify direction, location and severity of regurgitant valvular lesions.

6. Continuous Wave Doppler - This technique is similar to pulse wave except individual transducers are used for transmission and reception. This allows for more accurate velocity measurements.

Anatomic Views

2D views are commonly obtained from three general areas, upper (basal) and mid-esophagus and trans-gastric (see figure 1). Both transverse and longitudinal views may be obtained from each of these positions. Figure 2 shows some schematic representations of these anatomic views.

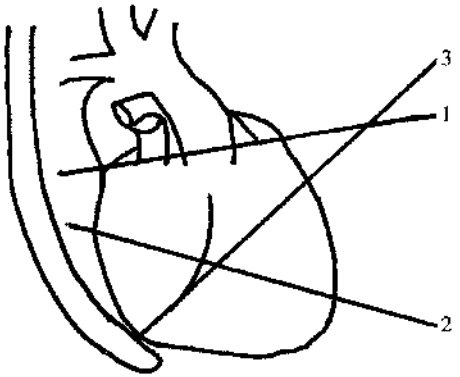
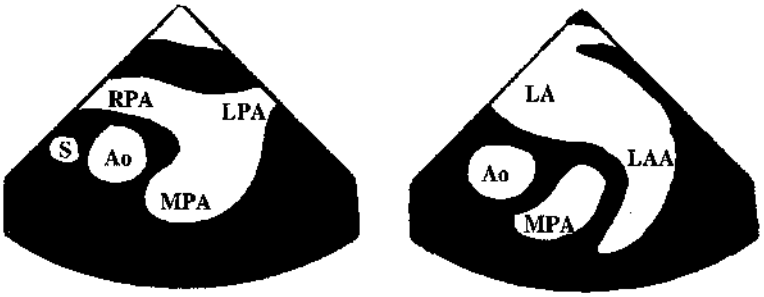


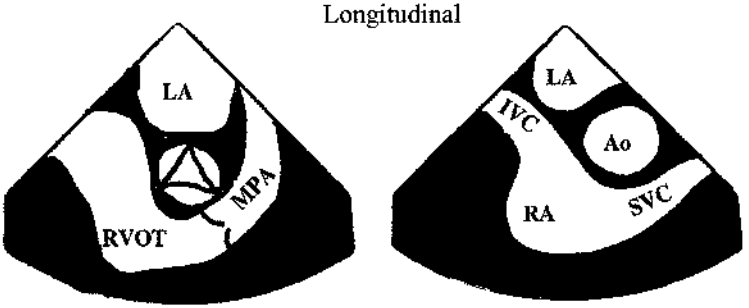
Figure 1. 1 - Upper Esophagus (basal) 2 - Mid-Esophagus 3 - Trans-Gastric

UPPER ESOPHAGUS (BASAL)

Transverse

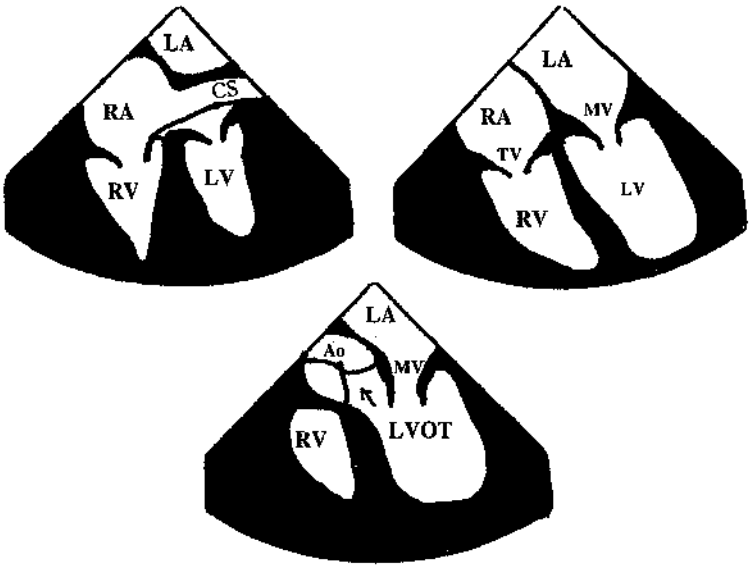


Longitudinal



MID-ESOPHAGUS

Transverse



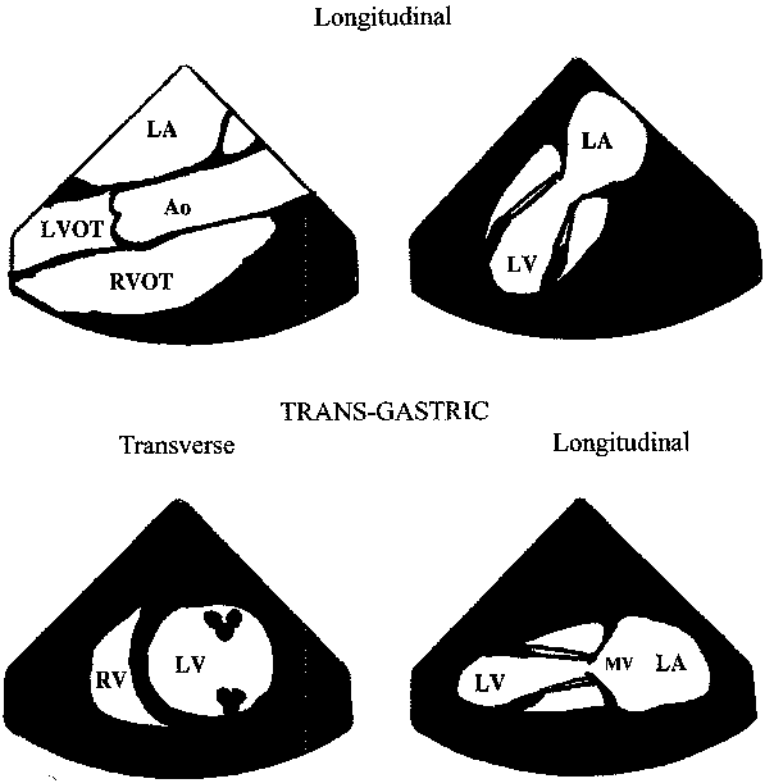


Figure 2. **Ao** - Aorta; **Cs** - Coronary Sinus; **IVC** - Inferior Vena Cava; **LA** - Left Atrium; **LAA** -Left Atrial Appendage; **LPA** - Left Pulmonary Artery; **LV** - Left Ventricle; **LVOT** - Left Ventricular Outflow Tract; **MPA** - Main Pulmonary Artery; **MV** - Mitral Valve; **RA** - Right Atrium; **RPA** - Right Pulmonary Artery; **RV** - Right Ventricle; **RVOT** - Right Ventricular Outflow Tract; **S** - Superior Vena Cava; **SVC** - Superior Vena Cava; **TV** - Tricuspid Valve.

Intra-operative Applications

By using one or more of the modes of operation described above, a variety of intraoperative applications for TEE are available. These are but a few of the more common applications.

1. Assessment of Ventricular Function - 2D imaging can provide an accurate estimation of ventricular size and thickness. Single and multiple view area

determinations can be used to reliably estimate ejection fraction. Examination of regional areas of wall motion is an extremely sensitive measure of myocardial ischemia (more so than ECG). Areas of myocardium supplied by each of the major coronary arteries can be viewed for evidence of movement and systolic thickening. Areas with poor movement are described as **hypokinetic**, areas with relatively no movement are described as **akinetic**, and areas moving in opposite directions (aneurysmal) are described as **dyskinetic.** Assessment of global function and regional wall motion before and after cardiopulmonary bypass (CPB) can provide useful information. Information regarding the diastolic function of the ventricle can be determined using transmitral pulse wave doppler assessment.

2. Volume Estimate - The correlation between CVP or PCWP and left ventricular volume, particularly post-CPB can often be inaccurate. Continuous 2D area viewing provides a direct and reliable indicator of preload and is useful in weaning from CPB.

3. Assessment of Valvular Function - Direct visualization of valvular function can be invaluable intraoperatively to the cardiac surgeon. 2D imaging can provide information regarding the integrity of valvular apparatus, presence of calcifications, and associated abnormalities. Use of doppler imaging can assess the direction and severity of valvular lesions. Perhaps most importantly, adequacy of the surgical repair can be determined quickly and reliably and problems such as perivalvular leaks can be repaired early.

4. Cannula and Line Placement - TEE can be used to determine the presence of calcifications in the ascending aorta that may impact cannula placement. Correct placement of coronary sinus cannula and intraaortic balloon pump catheters can be assessed.

5. Other - Other uses may include identification of septal defects or adequacy of closure, presence of or adequacy of removal of intracardiac air, and identification of aortic dissection.

Transesophageal echocardiography is becoming an increasingly powerful diagnostic and monitoring tool. Its utility during cardiac surgical procedures will only increase, necessitating at least a basic understanding of its function and ability.

NOTES

Solutions

Perfusionists work with different types of solutions during CPB. The priming solutions affect the hematocrit, fibrinogen, colloid osmotic pressure and distribution of fluid in the compartments. The intracellular and extracellular spaces are the compartments in which the body fluids are distributed. The extracellular space is further divided into the intravascular space (plasma) and the interstitial space (between the cells of the tissues). The average adult contains about 11.2 liters of interstitial fluid, comprising about 16% of the body weight. The intravascular space contains about 2.8 liters of plasma, comprising about 4% of the body weight. The composition of the plasma and the interstitial fluid is similar and easily shifted from one space to the other. Slight alterations in the composition of these fluids can lead to dramatic fluid and electrolyte shifts. In the intravascular and interstitial spaces the electrolyte concentrations are about the same. Sodium and chloride are greater in these spaces. Potassium, phosphate and proteins are higher in intracellular fluid. Edematous patients have built up fluid in the interstitial compartment. The priming solutions will enter the intravascular space initially. However, there are many factors affecting the movement of the fluids between the various spaces.

Weight

The formula weight represents the total of the atomic weights of the atoms in a chemical formula. The formula weight for water H_2O is (2 X 1.0080) + 15.9994 = 18.0154. If one molecule of a substance is represented, the term molecular weight can be used. H_2O is one molecule so the formula weight and molecular weight are the same.

Weights of substances are used more easily when they are given definite weight units. The gram atomic weight is the atomic weight of an element in grams. A gram atom of hydrogen equals 1.00797 g. A gram atom of oxygen equals 15.9994 g. If the gram atom of hydrogen is divided by the weight of one atom of hydrogen, the number that results is called Avogadro's Number. That is the number of atoms in one gram atom of an element. Avogadro's Number is 6.022×10^{23}. This number would be the same if one gram of oxygen was divided by the weight of one atom of oxygen. This is the number of atoms of in one gram atom of any element.

The formula weight represented in grams is the gram formula weight. If the formula is one molecule it can be labeled the gram molecular weight. The mole is the abbreviation that represents the gram molecular weight. Thus one mole of water is 18.0154 grams.

The gram equivalent weight (equivalent) is a measurement used that makes it easier to understand the combining of elements. It is defined asthe number of grams of an element that can gain, lose, or share Avogadro's Number

of electrons when combining with another element. This amount in grams for an element is 1 gram equivalent. Consider calcium combining with another element. One mole of calcium is 40.08 grams as can be seen from the chart. Oxidation is any process that involves the loss of electrons. This term is somewhat confusing since oxygen is not involved. Calcium loses 2 electrons when it combines with another element. Thus, it has an oxidation number of 2. Then 1 gram equivalent equivalent of calcium = 1 gram atom / oxidation number or 40.08 / 2 = 20.04 grams. Elements combine with each other in equal gram equivalents. If calcium and chlorine combine to form $CaCl_2$, it would require 2 gram equivalents of calcium and 2 gram equivalents of chlorine.

Gram equivalents when dealing with acids, bases and salts in water solutions exist as ions. One gram equivalent of an acid, base or salt is the number of grams that contain 1 gram equivalent each of the ions that are in the formula of the compound. Thus, 1 gram equivalent of an acid, base or salt = formula weight of ion / ionic charge. When dealing with liter solutions that have substances to be measured, the term equivalent is used. When dealing with solutions expressed in milliliters the term milliequivalent (mEq) is used.

Milliequivalent Conversion Factors

mEq/L of:	Divide mg/dl or vol% by:
Calcium	2.0
Chloride (from Cl)	3.5
(from NaCl)	5.85
CO_2 combining power	2.222
Magnesium	1.2
Phosphorus	3.1 (mmol)
Potassium	3.9
Sodium	2.3

Concentrations

A solution is the combination of a solute and a solvent. The most common types have a liquid as the solvent and the solute is a solid, liquid or gas. Solution concentrations are described as volume or weight concentrations.

5 Types of Volume Concentrations

- **Molar.** A solution that has one mole of solute in one liter of solution.
- **Normal.** A solution that has one gram-equivalent weight of solute in one liter of solution.
- **Weight-volume percent.** A solution that contains 1 g of solute per 100 ml of solution.
- **Milligram percent.** A solution that contains 1 mg of solute per 100 ml of solution.
- **Osmolar.** This is the concentration of a solute that is the molar concentration multiplied by the number of particles produced per molecule in solution.

Weight concentrations quantify the solute and the solvent by weight. These type concentrations are not affected by temperature.

3 Types of Weight Concentrations

- **Molal.** A solution that has 1 mole of solute in 1000 g of solvent.
- **Mole fraction.** The ratio of the number of moles of a substance to the number of moles of all the substances in solution.
- **Weight percent.** A solution based on the weight of the solute compared to the weight of the solvent. A 1% solution then has 1 g of solute in 99 g of solvent.

Molarity

Molarity is a method of describing the concentration of a solution. This method uses the abbreviation *M*. This expression is used when the quantity of solute is in moles and the volume of the solution is in liters. This concept can be defined as the number of moles of solute contained in 1 liter of solution. This can be represented by the following formula:

molarity = moles of solute / liters of solution

To find the molarity of a solution containing 100 g of NaCl in enough solvent to make 1500 ml of solution. First, express the quantity of solute in moles and the volume of the solution in liters.

100 g NaCl = (100 / 58.4) moles
1500 ml = 1.50 liters

Then using the above formula:

molarity (M) = (100 / 58.4) / 1.50
M = 1.14

To find how many grams of solute are in 500 ml of a 0.250 M solution of NaOH perform the following:

moles of solute = (molarity) x (liters of solution)
moles of solute = (0.250 M)(0.500 liters)
moles of solute = 0.125 mole of NaOH
If 1 mole of NaOH = 40.0 g
Then 0.125 mole of NaOH = (0.125 x 40) g NaOH
Then 0.125 mole of NaOH = 5.00 g NaOH

Molality

Molality is a way of expressing the concentration in terms of the quantity of solute dissolved in 1000 g of solvent. Molality (m) is the number of moles dissolved in 1000 g of solvent. This is expressed by the following formula:

molality = moles of solute / kg of solvent

For instance, to determine the molality of a solution containing 100 g of NaCl dissolved in 1500 g of water do the following:

100 g NaCl = (100 / 58.4) moles
1500 g H_2O = 1.5 kg of water
Thus the molality = (100 / 58.4) / 1.5
or 1.14 m

Normality

Normality (N) is a means of expressing concentrations as the number of gram-equivalents of solute in 1.00 liters of solution. This is expressed by the following formula:

normality = gram-equivalents of solute / liters of solution

To review gram-equivalents, 1 gram-equivalent of an acid, base or salt is the number of grams that contain 1 gram-equivalent of each of the ions represented by the formula of the compound. One gram-equivalent of an ion is the exchange of the Avogadro's number of electrons. Then:

1.00 g-eq of an ion = formula weight of ion / ionic charge

and 1.00 g-eq of an acid, base or salt = formula weight/ionic charge

To find the number of gram-equivalents in 500 g of phosphoric acid, H_3PO_4:

1.00 g-eq of $H_3PO_4 = 98.00 / 3$ or 32.67 g
Then 500 g of $H_3PO_4 = 500 / 32.67$ or 15.3 gram-equivalents

Osmotic Pressure

Membranes that are semipermeable may have the property of osmosis. These membranes can allow only certain substances to pass. Osmosis is the selective action in the flow of parts of a solution through a membrane. A membrane that has more of a solute on one side will likely have fluid pass through to the less concentrated side and build pressure on that side. The exact pressure that will stop this osmosis is the osmotic pressure of the solution.

Colloids are substances such as proteins that exert an osmotic pressure. Colloids do not ionize into solutions, but remain uniformly distributed. Colloids are at least 40,000 - 50,000 Daltons. The plasma proteins are colloids of interest to perfusionists. These colloids, or proteins, exert an osmotic pressure to keep water in the vascular system. The normal colloid osmotic pressure of the plasma is about 25 mmHg. Concentration of protein determines the colloidal osmotic pressure of plasma. Total serum protein is 6 - 8 g/dl. Albumin is 52 - 68% of the total protein with globulins comprising the remainder. Normal serum or plasma albumin is 3.5 - 5.5 g/dl. Normal serum or plasma globulin is 2 - 3.6 g/dl. Patients on CPB or ECMO with low albumin levels are likely to become edematous due to movement of fluid out of the vascular space into the interstitial space. The addition of large amounts of crystalloid solutions will decrease the albumin level and lead to this edematous state. Albumin should be added to the circulating priming solution if albumin levels are greatly reduced.

One milliosmole exerts 17 mmHg of osmotic pressure. To determine the osmotic pressure exerted by a solution, first, find the number of milligrams. In a 5% solution there are 5 grams per deciliter or 5 x 10 = 50 gm/L or 50 x 1000 = 50,000 mg/L. This is divided by the molecular weight in milligrams of the solute, for instance, 100,000. Then 50,000 / 100,000 = .5 milliosmoles per liter. Therefore 17 mmHg x .5 = 8.5 mmHg is the colloid osmotic pressure of the solution.

A practical method of ascertaining the effect of adding crystalloid on the colloid osmotic pressure is the following. The ml of circulating plasma are multiplied by 25 (normal colloid osmotic pressure). This value is then divided by the sum of the plasma volume and the crystalloid that is to be added.

Albumin 25 % solution has 5 times the colloid osmotic pressure of isoncotic solutions. Addition of this colloid can create an isoncotic solution ifit is added in amounts to equal 20 % of the crystalloid prime. If it is necessary

to add one liter of solution, it would be best to add 800 ml of crystalloid and 200 ml of 25 % albumin solution to maintain an isoncotic solution.

Osmolarity

The osmolar concentration of a solute is the molar concentration multiplied by the number of particles produced per molecule that are contained in the solution. Sodium chloride (NaCl) in a 1 *M* solution dissociates into 2 particles and is thus 2 osmolar. Glucose, a nondissociating solute, is 1 osmolar in a 1 *M* solution. The osmolar concentration of plasma is 0.308. Red blood cells that are suspended in solutions that have this osmotic pressure will not shrink or swell. Solutions of this type are isotonic. A solution of sodium chloride that is 0.308 osmolar or 0.154 *M* will be an **isotonic solution.**

Mixing Solutions

The perfusionist is sometimes called upon to determine the effects of combining solutions for various uses. The volume-concentration formula, Volume 1 x Concentration 1 = Volume 2 x Concentration 2 is used to do this. For instance:

Determine the amount of each solute in a mixture of 800 ml of 0.9% NaCl and 200 ml of D5W. Normal saline, 0.9% NaCl contains .9 grams of sodium chloride per 100 ml of solution. D5W has 5 grams of dextrose per 100 ml of solution.

To determine the amount of sodium chloride:

V1 x C1 = V2 x C2
800 x .9% NaCl = 1000 x C2
C2 = (800 x .9% NaCl) / 1000
C2 = .72% or .72 grams of NaCl
per 100 ml of solution or 7.2 grams per liter

To determine the dextrose:

V1 x C1 = V2 x C2
200 x 5% dextrose = 1000 x C2
C2 = (200 x 5% dextrose) / 1000
C2 = 1% dextrose or 1 gram of dextrose
per 100 ml of solution or 10 grams per liter

Electrolyte Contents of the Spaces

Electrolyte	Extracellular		Intracellular
	Intravascular	Interstitial	
Sodium (Na^+)	142	146	15
Potassium (K^+)	5	5	160
Calcium (Ca^{++}	5	3	2
Magnesium (Mg^{++})	2	1	27
Chloride (Cl^-)	102	114	1
Bicarbonate (HCO_3^-)	27	30	10
Protein ($Prot^-$)	16	1	63
Phosphate ($HPO_4^=$)	2	2	100
Sulfate ($SO_4^=$)	1	1	20
Organic acids	5	8	0

Common Chemical Elements

Element	Symbol	Valence	Atomic Number	Atomic Weight
Aluminum	Al	3	13	26.9815
Barium	Ba	2	56	137.34
Bromine	Br	1,3,5,7	35	79.909
Calcium	Ca	2	20	40.08
Carbon	C	2,4	6	12.01115
Chlorine	Cl	1,3,5,7	17	35.453
Hydrogen	H	1	1	1.0079755
Iron	Fe	2,3	26	55.847
Lithium	Li	1	3	6.939
Magnesium	Mg	2	12	24.312
Mercury	Hg	1,2	80	200.59
Nitrogen	N	3,5	7	14.0067
Oxygen	O	2	8	15.9994
Potassium	K	1	19	39.102
Sodium	Na	1	11	22.9898
Sulfur	S	2,4,6	16	32.064

Components of Common Solutions

Solutions	Glucose (gm/L)	Na	Cl	K (mEq/L)	Ca	Lactate
5% D/W	50					
10% D/W	100					
20% D/W	200					
50% D/W	500					
5% D/0.9%	50	154	154			
5% D/0.45%	50	77	77			
0.45% NaCl		77	77			
0.9% NaCl		154	154			
3% NaCl		513	513			
Ringer's Sol.		147.5	156	4	4.5	
Ringer's Lactute		130	109	4	3	28

Definitions

Avogadro's Number - 6.022 x 10 to the 23rd power. The number of solute particles in one mole of any substance.

Colloidal solution - larger particles in solution that resist passing through membranes. The lower limit of the size of colloids is 40,000 to 50,000 Daltons.

Crystalloid solution - a true solution in that the particles are equally dispersed by random movement of particles called Brownian motion. The size of crystalloid particles is thought to be under 50,000 to 100,000 Daltons although this is a nebulous limit. These solutions pass easily through membranes.

Deciliter - 100 ml, commonly used in medicine.

Gram equivalent weight - the weight of a substance in grams that can gain, lose or share the Avogadro's number of electrons when combining with another element.

Hypertonic - a solution that has more tonicity than another solution.

Hypotonic - a solution that has less tonicity than another solution.

Isotonic - two solutions that are of the same tonicity (same osmolarity).

Milliequivalent - (see gram-equivalent weight) the term used when the volume of the solution is expressed in milliliters.

Millimole - one thousandth of a mole.

Mole - the formula weight of a substance expressed in grams. This is also known as the gram-molecular weight should the formula represent one molecule.

Nonpolar - a molecule that has atoms of a covalent compound arranged symmetrically and the centers of positive and negative charges coincide.

Normal solution - a solution that has one gram-equivalent weight of solute in one liter of solution.

One molal solution - one gram molecular weight of solute plus 1000 grams of solvent.

One molar solution - one gram molecular weight of solute per 1000 ml of solvent.

Osmolarity - a solution action that is dependent on the number of particles in solution.

Osmole - a unit of osmolarity that is equal to Avogadro's Number (6.022 X 10 to the 23rd power).

Osmosis - the selective action of the movement of the solvent of a solution through a membrane.

Osmotic pressure - the pressure that is required to stop osmosis. Osmotic pressure of 1.0 mOsm/L concentration of solute is 17 mmHg.

Polar - a molecule that has atoms of a covalent compound that are not arranged symmetrically and the centers of positive and negative charges do not coincide within the molecule.

Saturated solution - a state of equilibrium where the number of particles going into solution equals the number of particles returning to the solid state.
Solute - the substance that is dispersed in the liquid.
Solution - a homogeneous mixture of a gas, liquid or solid substance in a liquid.
Solvent - the liquid in which the solute is dispersed.
Suspension - large particles in solution that must be mixed to be dispersed in the solution.
Tonicity - pressure caused by a solute.
Volume per volume - the concentration by volume of the solute in a certain volume of the solution. The least accurate method to measure concentration.
Weight per volume - the concentration by weight of the solute in a certain amount of the solution (usually deciliters).
Weight per weight - the concentration by weight of the solute in a certain weight of the solution.

NOTES

Pharmacology

Inhalation Agents

Anesthetic agents allow surgery to be performed in a relatively painless manner. Without these agents cardiac surgery would not be possible. This chapter outlines most of the medications that are used in cardiac surgery.

Nitrous Oxide is a colorless, odorless, largely tasteless gas. This general anesthetic produces analgesia, but not loss of consciousness, thus it is not used alone for major surgical procedures. It is usually used as an induction agent with more potent agents following to achieve the necessary effect. The heart rate and contractility are also affected by this anesthetic.

Fluothane (Halothane) is a colorless, sweet smelling gaseous anesthetic. It can be used to control hypertension during surgery as it reduces sympathetic activity such as heart rate, and dilates the vessels of the skin and skeletal muscles. Liver damage, cardiac arrhythmias and myocardial depression are relative drawbacks of this anesthetic.

Ethrane (Enflurane) is a vapor anesthetic with a sweet odor that can be used to control hypertension during surgery.

Forane (Isoflurane) is a volatile anesthetic with a pungent smell. It is widely used with the heart lung machine due to lack of cardiac problems associated with the agent. Although, blood pressure does drop, cardiac output and heart rate are usually increased. Arrhythmias are unusual.

Anticoagulants

Trade name: **Sodium Heparin**
Generic name: Sodium Heparin
Indications: To anticoagulate the blood prior to going on bypass. Also used in other instances where anticoagulation is desired. Blood stored with heparin added can be kept for 48 days.
Action: Stops coagulation by potentiating antithrombin III and inhibiting the action of activated Factors IX and XI. Half life is one to two hours.
Site of action: Antithrombin III.
Dosage: 300 to 400 units per kilogram before bypass injected into the right atrium or by way of a central line.

Trade name: **Coumadin, Dicumarol, Panwarfin**
Generic name: Coumarin derivatives, Warfarin
Indications: Reduces the risk of harmful clots. Used prophylactically after cardiac valve surgery.
Action: Antagonizes vitamin K, therefore inhibiting synthesis of Factors II, VII, IX, X.
Site of action: Hepatic synthesis of coagulation Factors II, VII, IX, X.
Dosage: Varies with patient response. Slow acting compared to other anticoagulants. Prothrombin times should be used to determine response.

Trade name: **Thrombate III**
Generic name: Antithrombin
Indications: Antithrombin III deficiency in connection with surgery.
Action: Inactivates thrombin and activated forms of clotting factors IX, X, XI and XII which results in coagulation inhibition.
Site of Action: Clotting factors of blood.
Dosage: Individualized. Dose calculation formula is (desired - baseline AT-III level) x kg / 1.4. A 70kg person with an ACT-III level of 57% and a desired level of 120% would require an initial dose of 3150 IU.

Trade name: **CPD**
Generic name: Citrate Phosphate Dextrose
Indications: Anticoagulate and preserve blood in blood banks.
Action: Citrate binds calcium blocking clotting in all pathways. Provides and helps maintain energy of RBCs.
Site of action: Calcium ions in plasma and RBCs.
Dosage: Premixed in collection bags by manufacturer.

Trade name: **CPDA-1**
Generic name: Citrate Phosphate Dextrose Adenine
Indications: Anticoagulate and preserve blood in blood banks.
Action: Citrate binds calcium blocking clotting in all pathways. Provides and helps maintain energy of RBCs. Adenine keeps ATP levels higher and prolongs life of RBC. Blood can be stored for 35 days when this is used.
Site of action: Calcium ions in plasma and RBCs.
Dosage: Premixed in collection bags by manufacturer, 63 ml per 450 ml of blood.

Anticoagulant Antagonists

Trade name: **Protamine Sulfate**
Generic name: Protamine Sulfate
Indications: To reverse heparin after bypass surgery.
Action: In the presence of heparin forms inert salt to neutralize both drugs. If given with no heparin present, it acts as an anticoagulant. Increased incidents of anaphylactic reactions to drug have been reported in individuals with a history of prior exposure, vasectomy, NPH insulin use and fish allergies.
Site of action: Combines with heparin.
Dosage: Usually given 1.3 mg per 100 units of heparin present. Given IV, slowly.

Trade name: **Aquamephyton, Mephyton, Konakion**
Generic name: Phytonadione
Indications: Treatment of coagulation deficiencies due to lack of vitamin K.
Action: Presence necessary for synthesis of clotting Factors II, VII, IX, X.
Site of action: Liver.
Dosage: 2.5 to 25 mg IM or subcutaneous.

Platelet Inhibiting Drugs

Trade name: **Aspirin**
Generic name: Acetylsalicylic acid
Indications: Prophylactic treatment of platelet hyperaggregability in conditions such as coronary heart disease.
Action: Platelet function inhibitor by blocking enzyme function.
Site of action: Cyclooxygenase enzyme.
Dosage: 325 to 650 mg PO every day.

Trade name: **Persantine**
Generic name: Dipyridamole
Indications: Prophylactic treatment after prosthetic cardiac valve placement for reduction of embolization risk.
Action: Reduces uptake of adenosine. Synergistic effect with aspirin on platelet inhibition.
Site of action: Platelet membrane, inhibits phosphodiesterase activity.
Dosage: 50 mg PO three times a day.

Trade name: **ReoPro**
Generic name: Abciximab
Indications: Adjunct to PTCA in patients at risk for abrupt closure.
Action: Inhibition of platelet function.
Site of Action: Binds to glycoprotein receptor of platelet.
Dosage: IV 0.25 mg/kg 10-60 min before PTCA, then for maintenance IV infusion of 10 mcg/min for 12 hr.

Trade name: **Plavix**
Generic name: Clopidogrel Bisulfate
Indications: Plavix is indicated for the reduction of thrombotic events including recent MI, recent stroke, or established peripheral arterial disease and acute coronary syndrome.
Action: Inhibition of platelet aggregation.
Site of Action: Glycoprotein GPIIb/IIIa complex.
Dosage: PO 300-mg loading dose and then continued at 75 mg once daily.

Trade name: **Ticlid**
Generic name: Ticlopidine HCL
Indications: Reduces risk of thrombotic events in patients who are aspirin intolerant.
Action: Inhibits platelet activity.
Site of Action: Platelets.
Dosage: PO 250 mg twice a day.

Trade name: **Integrilin**
Generic name: Eptifibatide
Indications: Integrilin is used to treat patients with severe chest pain or small heart attacks and for those undergoing certain heart procedures (e.g., balloon angioplasty).
Action: Inhibition of platelet aggregation.
Site of Action: Glycoprotein GPIIb/IIIa complex
Dosage: 135 micrograms/kg Bolus and 0.5 micrograms/kg/min Infusion OR 180 micrograms/kg Bolus and 2 micrograms/kg/min Infusion in pts. with acute coronary syndrome.

Trade name: **Aggrenox**
Generic name: Aspirin/Extended-Release Dipyridamole
Indications: Antiplatelet combination used to prevent strokes in patients who have had transient ischemia or ischemic stroke due to blood clots.
Action: Platelet function inhibitor by blocking enzyme function.
Site of action: Cyclooxygenase enzyme and Thromboxane A2.
Dosage: 2 Capslues (dipyridamole 200mg, ASA 25mg) PO twice a day.

Fibrinolytics and Thrombolytics

Trade name: **Kabikinase, Streptase**
Generic name: Streptokinase
Indications: Destruction of thrombosis in coronary arteries, pulmonary system and other areas.
Action: Catalyzes conversion of plasminogen to plasmin which destroys fibrin.
Site of action: Plasminogen.
Dosage: For embolism initial IV dose- 250,000 IU over 30 minutes, then 100,000 IU every hour for one to three days. For evolving transmural MI, IV 1,500,000 IU, diluted in 45 ml, over 60 min.

Trade name: **Abbokinase, Win-kinase**
Generic name: Urokinase
Indications: Breaks down coronary artery thrombosis and pulmonary embolism.
Action: Converts plasminogen to plasmin.
Site of action: Plasminogen.
Dosage: For pulmonary embolism determined by weight, IV - priming dose of 2,000 IU/lb. diluted with sodium chloride solution over 10 min, then 2,000 IU/lb./hr for 12 hours. For coronary artery thrombosis, IV heparin bolus 2,500-10,000 units, followed by intracoronary urokinase 6,000 IU/min for up to 2 hours. Average total dose 500,000 IU.

Trade name: **Activase (t-PA)**
Generic name: Alteplase, recombinant
Indications: Breaks down coronary artery clots in acute myocardial infarctions.
Action: Local conversion of plasminogen to plasmin.
Site of action: Plasminogen in clots.
Dosage: 100 mg IV, given as 60 mg in the first hour of which 6-10 mg is given as a bolus over 1-2 minutes, 20 mg over the second hour and 20 mg over the third hour.

Fibrinolytic Inhibitors

Trade name: **DDAVP**
Generic name: Desmopressin acetate
Indications: To improve coagulation by increasing Factor VIII.
Action: Synthetic antidiuretic hormone that increases Factor VIII levels.
Site of Action: Factor VIII storage sites in plasma.
Dosage: IV 0.3 mcg/kg diluted in 50 ml saline and infused slowly over 15 to 30 minutes.

Trade Name: **Trasylol**
Generic Name: Aprotinin Injection
Indications: Prophylactic use to reduce perioperative blood loss and the need for transfusion in coronary artery bypass graft surgery.
Action: Inhibits fibrinolysis and turnover of coagulation factors; decreases bleeding although exact mechanism is unclear.
Site of action: Protease inhibitor, inhibits plasmin and Kallikrein. Preserves adhesive glycoproteins in platelet membranes making them resistant to damage during CPB.
Dosage: Do not administer with any other drug in the same line. All intravenous doses should be through a central line. The chart below list the full dose Hammersmith. The half dose Hammersmith is 1/2 the full Hammersmith dosage.

Recommended Full Hammersmith Dosage Regimen

	Dose	**Administration**
Test Dose	1 mL (1.4 mg, or 10,000 KIU)	Administer IV at least 10 min before the loading dose
***Allergic Reactions:* Patients who experience any allergic reaction to the test dose of Trasylol should not receive further administration of the drug since the full therapeutic dose may cause anaphylaxis.**		
Loading Dose	200 mL (280 mg, or 2.0 million KIU/hr	Give IV, slowly over 20-30 minutes with patient in supine position, after induction of anesthesia but prior to sternotomy.
Constant Infusion Dose	50 mL/ hr (70 mg/hr, or 500,000 KIU/hr	Administer when loading dose is complete and continue until surgery is complete and patient leaves operating room.
"Pump Prime" Dose	200 mL (280 mg, or 2.0 million KIU)	Add to the **recirculating** pump prime of the CPB circuit by replacement of an aliquot of the priming fluid prior to the institution of CPB.

NOTE: Aprotinin administration may artifically prolong ACT results. Please follow the ACT manufacturer's guidelines for appropriate ACT values when Aprotinin is used. Increase total Heparin doseage to >350 IU/Kg for CPB.

Trade name: **Amicar**
Generic name: Epsilon Aminocaproic Acid
Indications: To reduce bleeding cause by systemic hyperfibrinolysis.
Action: Inhibits plasminogen activators to prevent conversion to plasmin and antiplasmin activity.
Site of Action: Plasminogen activator substances.
Dosage: Loading dose of 5 grams IV, then 1-1.25 grams every hour not to exceed 30 gm/day. The terminal elimination half-life is 2 hours.

Anti-Antiarrhythmic Agents

Trade name: **Quinaglute, Dura-Tabs**
Generic name: Quinidine Gluconate
Indications: To treat ventricular and supraventricular arrhythmias.
Action: Decreases the rate of diastolic depolarization, slows rate of repolarization, increases effective refractory period, increases effective refractory period and increases conduction time (except for the AV node).
Site of Action: Myocardial cell membranes and alpha receptors.
Dosage: 324mg tab PO every 8-12 hours.

Trade name: **Pronestyl, Procan SR**
Generic name: Procainamide HCL
Indications: Ventricular arrhythmias and tachycardia, atrial fibrillation, paroxysmal atrial tachycardia, arrhythmias associated with surgery.
Action: Prevents sodium and potassium from passing through cell membrane, thus preventing depolarization.
Site of Action: Cell membrane.
Dosage: IV bolus 100 mg over 5 min, given 25-50 mg/min, and repeated every five minutes not to exceed 500mg maximum.

Trade name: **Xylocaine**
Generic name: Lidocaine HCL
Indications: Ventricular arrhythmias.
Action: Reduces cell membrane permeability for sodium and potassium which increases the stimulation thresholds in the ventricles.
Site of Action: Cell membrane.
Dosage: IV bolus 1-2 mg/kg, half of dose may be repeated in 30 minutes, 1-5 mg/min IV infusion after bolus, not to exceed 300 mg/hr.

Trade name: **Bretylol**
Generic name: Bretylium Tosylate
Indications: Ventricular fibrillation and arrhythmias.
Action: Prevents release of norepinephrine from adrenergic nerve terminal, decreases sinus rhythm, increases conduction time, increases ventricular fibrillatory threshold.
Site of Action: Beta-adrenergic nerve terminals.
Dosage: For V-fib, 5-10 mg/kg IV bolus repeated every 15 min up to 30 mg/kg.

Trade name: **Inderal, Inderide**
Generic name: Propranolol
Indications: Ventricular and supraventricular arrhythmias.
Action: Slows A-V node conduction, automaticity.
Site of Action: Beta-adrenergic receptor sites.
Dosage: 0.5-3.0 mg IV at rate of 1 mg/min; may repeat in 2 min and at 4 hours.

Trade name: **Isuprel**
Generic name: Isoproterenol Hydrochloride
Indications: Complete heart block.
Action: Conduction through atria, AV node, ventricles accelerated. Automaticity increased, positive inotropic effects, increases systolic pressure, but lowers diastolic.
Site of Action: Beta-adrenergic receptor sites.
Dosage: For heart block initial dose, 0.02-0.06 mg IV bolus diluted in 10 ml of sodium chloride. Subsequent dose 0.01-0.2 mg.

Trade name: **Isoptin, Calan**
Generic name: Verapamil
Indications: Supraventricular tachyarrhythmias.
Action: Interferes with calcium transfer into cells; slows AV node conduction, increases refractory period, elevates stimulation threshold.
Site of Action: Calcium channel blocker at myocardial cell membrane.
Dosage: 5-10 mg IV over 2 minutes (0.075-0.15 mg/kg). Repeat dose after 30 min if response is not adequate.

Trade name: **Cardizem**
Generic name: Diltiazem
Indications: Angina, arterial and pulmonary hypertension. Also, used for reduction of cardiac rate in the operating room after open heart surgery.
Action: Blocks calcium from entering the cells of cardiac and vascular smooth muscle.
Site of Action: Cardiac and vascular smooth muscle.
Dosage: 240 mg PO a day in divided doses. IV - 0.25 mg/kg loading dose over 2 minutes, then 10 mg/hr, if no affect in 10-15 minutes, give 0.35 mg/kg over 2 minutes, then 15 mg/hr depending on response.

Trade name: **Brevibloc**
Generic name: Esmolol
Indications: Supraventricular tachycardia and noncompensatory tachycardia
Action: Decreases rate of SA node discharge, decreases heart rate.
Site of Action: Blocks stimulation of beta adrenergic receptors in the myocardium.
Dosage: IV loading 500 mcg/kg/min over 1 min, maintenance of 50 mcg/kg/min for 4 min, may repeat every 5 min

Trade name: **Procardia**
Generic name: Nifedipine
Indications: Angina, hypertension, CHF.
Action: Blocks calcium from entering the cells of cardiac and vascular smooth muscle.
Site of Action: Cardiac and vascular smooth muscle.
Dosage: 10-20 mg PO three times a day.

Trade name: **Atropine Sulfate**
Generic name: Atropine Sulfate
Indications: Bradycardia.
Action: Causes vagal depression which causes increased heart rate.
Site of Action: Vagus nerve.
Dosage: 0.5 mg IV or subcutaneously.

Trade name: **Adrenaline**
Generic name: Epinephrine
Indications: Cardiac arrest - to establish rhythm.
Action: Increases heart rate.
Site of Action: Alpha and beta receptors.
Dosage: For adult cardiac arrest, IC, IV or endotracheal 0.1-1 mg repeated every 5 min as needed.

Trade name: **Norpace**
Generic name: Disopyramide
Indications: Supraventricular arrhythmias, especially in sinus bradycardia, or sick sinus syndrome.
Action: Lengthens effective refractory period, prolongs action potential of the Purkinje fibers.
Site of Action: Myocardial cell membrane and autonomic nervous system.
Dosage: 400-800 mg per day PO in divided doses.

Trade name: **Vasoxyl**
Generic name: Methoxamine HCL
Indications: Suppression of paroxysmal atrial tachycardia.
Action: Prolongs ventricular action potential and refractory period, slows AV conduction, increases vascular resistance.
Site of Action: Alpha adrenergic receptor sites.
Dosage: 3-5 mg IV, slowly.

Cardiac Glycosides

Trade name: **Digoxin, Lanoxin**
Generic name: Digoxin
Indications: Heart failure, atrial flutter, atrial fibrillation, paroxysmal atrial tachycardia. (Potassium and calcium should be monitored and maintained in a normal range when any of the drugs in this class are being used. Hypokalemia or hypercalcemia can cause arrythmias in these patients.)
Action: Slows AV node conduction, strengthens and increases force of myocardial contraction.
Site of Action: Sodium-potassium-ATPase of cell membrane, making more calcium available.
Dosage: 0.5 mg IV loading dose given in small increments then 0.125-0.5 mg PO in divided doses every 4-6 hr for daily maintenance dose.

Trade name: **Crystodigin, Purodigin**
Generic name: Digitoxin
Indications: Heart failure, atrial flutter, atrial fibrillation, paroxysmal atrial tachycardia.
Action: Slows AV node conduction, strengthens and increases force of myocardial contraction.
Site of Action: Sodium-potassium-ATPase of cell membrane.
Dosage: Rapid digitalization, 0.6 mg PO, then 0.4 mg at 4 hr and 0.2 mg at 6 hr.

Cardiac Inotropic Agents

Trade name: **Inocor**
Generic name: Amrinone
Indications: Immediate and short term management of CHF.
Action: Inotropic myocardial agent, vasodilator effect.
Site of Action: Increases calcium availability by increasing cellular levels of cyclic AMP, inhibits phosphodiesterase III at Beta-adrenergic receptor sites.
Dosage: Loading dose of 0.75 mg/kg IV over 2-3 minutes, then infuse between 5 mcg/kg/minute and 10 mcg/kg/minute for maintenance. Based on clinical response, an additional loading dose of 0.75 mg/kg may be given 30 min after the first dose. Total dosage should not exceed 10 mg/kg per day.

Trade name: **Primacor**
Generic name: Milrinone Lactate
Indications: Short term management of CHF, heart failure associated with cardiac surgery.
Action: Positive inotrope and vasodilator.
Site of Action: Selective inhibitor of peak III cAMP phosphodiesterase isozyme in cardiac and vascular muscle.
Dosage: Loading dose of 50 mcg/kg slow IV over 10 minutes, then 0.375 to 0.75 mcg/kg/min. Total daily dose at maximum maintenance 1.13 mg/kg.

Trade name: **Adrenalin**
Generic name: Epinephrine
Indications: Cardiac arrest - to establish rhythm.
Action: Increases heart rate.
Site of Action: Alpha and beta receptors.
Dosage: For adult cardiac arrest, IC, IV or endotracheal 0.1-1 mg repeated every 5 min as needed.

Trade name: **Levophed Bitartrate**
Generic name: Norepineprine
Indications: Acute hypotensive crisis such as myocardial infarction.
Action: Alpha stimulation causes peripheral constriction and rise in blood pressure. Beta stimulation increases myocardial contractility and vasodilates coronary arteries.
Site of Action: Alpha and beta adrenergic receptors.
Dosage: First mix solution of 4 ml Levophed in 1000 ml of 5% dextrose solution. Initial dose is 8-12 mcg per minute IV. Maintenance dose is 2-4 mcg per minute depending on blood pressure.

Trade name: **Neosynephrine**
Generic name: Phenylephrine HCL
Indications: Maintenance of blood pressure in the treatment of shock. Used on bypass to maintain blood pressure.
Action: Peripheral resistance is increased to raise arterial pressure. Coronary blood flow is increased.
Site of Action: Post synaptic alpha receptors. (Does not work at beta receptor sites.)
Dosage: 0.1-0.5 mg IV. May be given IV drip with 10 mg added to 250 to 500 ml of sodium chloride solution, the rate adjusted according to patient response.

Trade name: **Aramine**
Generic name: Metaraminol Bitartrate
Indications: Hypotension.
Action: Vasoconstriction by direct and indirect actions on alpha and beta receptor sites. Not a sympathetic blocker.
Site of Action: Alpha and beta adrenergic receptor sites.
Dosage: IV 15-100 mg in 500 ml of sodium chloride solution with rate of infusion dependent on patient response.

Trade name: **Ephedrine Sulphate**
Generic name: Ephedrine Sulphate
Indications: Hypotension.
Action: Liberates catecholamines from tissue stores. Causes blood pressure to rise.
Site of Action: Alpha and beta receptor sites.
Dosage: 20 mg IV slowly for fast action.

Trade name: **Dobutrex**
Generic name: Dobutamine HCL
Indications: Cardiac decompensation due to decreased contractility from surgery or other causes.
Action: Fast acting to increase contractility and cardiac output. Improves AV conduction, pulmonary and peripheral vasodilator.
Site of Action: Beta 1 receptors.
Dosage: 2.5-10 mcg/kg/minute IV diluted in 50 ml sodium chloride solution. Rate and duration depends on patient response.

Trade name: **Dopastat, Intropin**
Generic name: Dopamine HCL
Indications: Low cardiac output, CHF, hypotension state of myocardial infarctions. (May decrease coronary blood flow in patients with atherosclerosis.)
Action: Inotropic effect, precursor of catecholamines.
Site of Action: Alpha and beta receptors.
Dosage: IV 5 mcg/kg/minute diluted in 250 ml sodium chloride solution which can be increased to 50 mcg/kg/minute depending on patient response.

Trade name: **Vasoxyl**
Generic name: Methoxamine HCL
Indications: Suppression of paroxysmal atrial tachycardia, causes an increase in blood pressure during anesthesia.
Action: Prolongs ventricular action potential and refractory period, slows AV conduction, increases peripheral vascular resistance.
Site of Action: Alpha adrenergic receptor sites.
Dosage: 3-5 mg IV.

Trade name: **Wyamine**
Generic name: Mephentermine sulfate
Indications: Hypotensive states.
Action: Releases norepinephrine from tissue stores, thus raising pressure.
Site of Action: Alpha and beta adrenergic receptor sites.
Dosage: IV - given mixed in solution with 100 mg of drug in 500 ml sodium chloride solution. Infusion rate dependent on patient response.

Antihypertensive Agents

Trade name: **Nipride**
Generic name: Sodium Nitroprusside
Indications: Hypertension. Used on CPB due to direct powerful vasodilator effects on arterial vessels.
Action: Vascular smooth muscle relaxant.
Site of Action: Arterial and venous smooth muscle.
Dosage: 3 mcg/kg/minute IV or depending on patient response. Dose should not exceed 10 mcg/kg/minute.

Trade name: **Arfonad**
Generic name: Trimethaphan
Indications: Hypertension. Used on CPB.
Action: Autonomic nervous system ganglia transmission blocker, vasodilator.
Site of Action: Autonomic ganglia.
Dosage: 1 mg IV in diluted solution over 1 minute. Rate adjusted according to patient response. For hypertensive crisis can be given 1-4 mg per minute in diluted solution as determined by patient response.

Trade name: **Apresoline**
Generic name: Hydralazine HCl
Indications: Essential hypertension.
Action: Direct relaxation of vascular smooth muscle.
Site of Action: Vascular smooth muscle, mainly arterial.
Dosage: 20-40 mg IV every 4-6 hr.

Trade name: **Regitine**
Generic name: Phentolamine
Indications: Hypertension while on CPB, peripheral vascular spasms.
Action: Peripheral vasodilator, cardiac stimulation.
Site of Action: Alpha-adrenergic receptors.
Dosage: 2.5 mg IV bolus and wait for response to determine further doses. May be increased to 5 mg IV dose.

Trade name: **Inderal, Inderide**
Generic name: Propranolol
Indications: Ventricular and supraventricular arrhythmias.
Action: Slows A-V node conduction, automaticity.
Site of Action: Beta-adrenergic receptor sites.
Dosage: 0.5-3.0 mg IV over 1mg/min; may repeat in 2 min and every 4 hours after.

Trade name: **Corgard**
Generic name: Nadolol
Indications: Hypertension, angina. Sometimes used preoperatively to keep pressure under control throughout surgery.
Action: Decreases blood pressure, heart rate.
Site of Action: Beta 1 and beta 2 receptors.
Dosage: 40 mg PO once a day initially, increased in 40 mg increments depending on patient response. Maximum dose 240 mg a day.

Trade name: **Thorazine**
Generic name: Chlorpromazine
Indications: An antiemetic used with anesthesia, vasodilator, psychotherapeutic agent.
Action: Sedation, CNS depression, vasodilation.
Site of Action: Alpha and beta adrenergic receptors.
Dosage: During surgery given in 2 mg increments IV every two minutes depending on individual patient response. Do not exceed 25 mg.

Trade name: **Nitrostat, Nitrol, Nitro-Bid**
Generic name: Nitroglycerin
Indications: Hypertension during cardiac surgery, angina.
Action: Relaxes vascular smooth muscle.
Site of Action: Vascular smooth muscle.
Dosage: IV-if using nonabsorbable tubing, dose may range from 5 mcg/min to 20 mcg/min depending on patient response. Sublingual dose-onset of action is 1-2 minutes and the duration is 30 minutes. PO-capsule, onset is 60 minutes and duration is 12 hr. Ointment-onset of action is 15 minutes and the duration is 4 hr.

Neuromuscular Blocking Agents

Trade name: **Pavulon**
Generic name: Pancuronium bromide
Indications: Skeletal muscle relaxant for surgical patients or patients on ventilator. Provides paralysis.
Action: Prevents absorption of neurotransmitters at neuromuscular junction.
Site of Action: Nicotinic receptor sites.
Dosage: 0.04-0.10 mg/kg IV.

Trade name: **Anectine, (Quelicin, Sucostin)**
Generic name: Succinylcholine
Indications: Muscle relaxant to assist tracheal intubation.
Action: Depolarizing muscle relaxant that leads to flaccidity.
Site of Action: Nicotinic receptor sites.
Dosage: Anectine- IV 25-75 mg, then 2.5 mg/min as required not to exceed 150 mg.

Trade name: **Tubocurarine**
Generic name: d-tubocurarine
Indications: Muscle relaxation during surgery.
Action: Competes with acetylcholine at the neuromuscular junction producing flaccid paralysis.
Site of Action: Nicotinic receptor sites.
Dosage: 0.4-0.5 mg/kg IV.

Trade name: **Norcuron**
Generic name: Vecuronium
Indications: Skeletal muscle relaxation during cardiac surgery.
Action: Competes with neurotransmitter at neuromuscular junction producing flaccid paralysis.
Site of Action: Nicotinic receptor sites.
Dosage: IV 0.08-0.10 mg/kg bolus.

Narcotic Analgesics

Trade name: **Morphine**
Generic name: Morphine
Indications: To relieve pain.
Action: Central nervous system depressant.
Site of Action: CNS.
Dosage: IV 4-10 mg diluted in 5 ml of sterile water, given over 5 min.

Trade name: **Demerol**
Generic name: Meperidine
Indications: Pain, often given as a pre-op medication.
Action: Central nervous system depressant, analgesic effect.
Site of Action: CNS.
Dosage: IM 50-100 mg pre-op. Anesthesia adjunct 25-200 mg IV.

Trade name: **Sublimaze**
Generic name: Fentanyl
Indications: Often used as the only anesthetic for cardiac surgery. Sometimes used as a supplemental or pre-op medication.
Action: Central nervous system depressant, analgesic effect.
Site of Action: CNS.
Dosage: Only anesthetic for surgery- 20-50 mcg/kg IV slowly. As a pre-op- 50-100 mcg IM 1 hour prior to surgery.

Trade name: **Sufenta**
Generic name: Sufentanil citrate
Indications: Used in cardiac surgery as the primary anesthetic or as an analgesic adjunct with balanced general anesthesia.
Action: Opioid anesthetic.
Site of Action: CNS.
Dosage: When used as the primary anesthetic 5 to 7 times more potent than Fentanyl. When used in balanced general anesthesia 10 times more potent than Fentanyl. When used as the primary anesthetic 8-30 mcg/kg IV slow injection followed by maintenance doses of 0.5-10 mcg/kg as needed to maintain anesthesia. Total dosage of induction and maintenance should not exceed 30 mcg/kg.

Anesthesia Adjuncts

Trade name: **Pentothal**
Generic name: Thiopental Sodium
Indications: Anesthesia supplement, protection of brain during circulatory arrest.
Action: Hypnotic CNS depressant.
Site of Action: CNS.
Dosage: 3-4 mg/kg IV over 45 seconds.

Trade name: **Propofol**
Generic name: Diprivan
Indications: Maintenance of anesthesia in "fast tracking" for early intubation following low narcotic cardiac anesthesia.
Action: Short acting general anesthetic.
Site of Action: CNS.
Dosage: Loading dose IV 100-150 mcg/kg/min slowly, then for maintenance 25-75 mcg/kg/min titrated.

Trade name: **Valium**
Generic name: Diazepam
Indications: Preoperative medication or anesthetic induction agent.
Action: Sedative, amnesic effect.
Site of Action: Thalamus, hypothalamus.
Dosage: As anesthetic induction agent 0.1-0.2 mg/kg IV.

Trade name: **Versed**
Generic name: Midazolam
Indications: Pre-op sedation, sole agent for diagnostic procedures, anesthetic induction agent.
Action: Acts on thalamus and hypothalamus to produce sedation, amnesic affects.
Site of Action: Thalamus and hypothalamus.
Dosage: Pre-op-0.07-0.08 mg/kg IM 1 hour before surgery. For conscious sedation-IV titration slowly to desired response. Some patients respond with 1 mg - no more than 2.5 mg should be given within 2 minutes. Wait 2 minutes before administering another dose. No more than 5 mg is usually necessary. For anesthesia induction of unmedicated patient, 0.3-0.35 mg/kg over 30 sec, after 2 min 25% of initial dose may be given.

Trade name: **Vistaril**
Generic name: Hydroxyzine pamoate
Indications: Preanesthetic medication.
Action: Sedative, antihistamine, antiemetic.
Site of Action: CNS.
Dosage: 50-100 mg IM.

Trade name: **Benadryl**
Generic name: Diphenhydramine
Indications: Pre-op medication.
Action: Antihistamine, sedative, antiemetic, anticholinergic.
Site of Action: Histamine receptors.
Dosage: IM 10-50 mg according to patient response. Given IV while on CPB.

Trade name: **Phenergan**
Generic name: Promethazine
Indications: Preanesthetic medication.
Action: Antihistamine, antiemetic, sedative.
Site of Action: CNS, histamine receptor sites, parasympathetic receptor sites.
Dosage: 10-25 mg IM or IV.

Trade name: **Sodium Nembutal**
Generic name: Sodium Pentobarbital
Indications: Sedative and preoperative medication.
Action: CNS depressant, sedative, hypnotic.
Site of Action: CNS.
Dosage: 150-200 mg IM.

Trade name: **Innovar**
Generic name: Fentanyl and droperidol combination.
Indications: Pre-meditation for anesthesia, or alone for minor surgical procedures.
Action: Analgesia, sedation, tranquilization, antiemesis, alpha-adrenergic blocker, potentiates other CNS depressants.
Site of Action: CNS.
Dosage: Premedication-0.5-2.0 ml IM 60 minutes prior to surgery. As an anesthesia adjunct-1 ml per 20-25 lb. slowly IV. Dose should depend on patient response.

Trade name: **Ketalar**
Generic name: Ketamine hydrochloride
Indications: Sole anesthetic agent for surgical procedures that do not require muscle relaxation, as a supplement to low potency agents and for induction of anesthesia prior to administration of other general anesthetic agents.
Action: Dissociative anesthesia. Causes initial hypertension that returns to normal after about 15 minutes.
Site of Action: Thalamoneocortical system of the brain.
Dosage: Titrated with an IV dose that may range from 1-4.5 mg/kg.

Trade name: **Scopolamine**
Generic name: Scopolamine Hydrobromide
Indications: Preanesthetic medication for sedation, amnesia, bronchial and salivary secretions. Increases level of anesthesia when used with other drugs (synergistic effect).
Action: Competitive antagonist of acetylcholine at receptor sites. (Not ordinarily used as a vagal blocker.)
Site of Action: Parasympathetic cholinergic receptor sites.
Dosage: 0.2-0.4 mg IM or IV.

Trade name: **Atropine**
Generic name: Atropine Sulfate
Indications: Preanesthetic to reduce secretions, antidote for nerve gas.
Action: Anticholinergic, reduces secretions, increases heart rate.
Site of Action: Parasympathetic cholinergic receptors.
Dosage: 0.5 mg IM or PO.

Trade name: **Prostigmin**
Generic name: Neostigmine methylsulfate
Indications: Reversal of non-depolarizing neuromuscular blocking agents.
Action: Skeletal muscle relaxant antagonist, allows acetylcholine to accumulate.
Site of Action: Cholinergic receptor sites.
Dosage: 0.5-2.0 mg IV slowly every 1-3 hr.

Trade name: **Dantrium**
Generic name: Dantolene Sodium
Indications: To reverse anesthesia induced malignant hyperthermia.
Action: Interferes with calcium release from sarcoplasmic reticulum, producing uncoupling of excitation-contraction.
Site of Action: Skeletal muscles.
Dosage: 1 mg/kg IV push until condition improves or maximum dose of 10 mg/kg.

Trade name: **Narcan**
Generic name: Naloxone
Indications: To reverse narcotic induced respiratory depression.
Action: Pure narcotic antagonist, competes for receptor sites as a narcotic.
Site of Action: CNS receptor sites.
Dosage: 0.4-2.0 mg IV, may be repeated at 2-3 minute intervals if no response. If after total dose of 10 mg no response is seen then consider other causes of respiratory depression.

Agents for Acidosis

Trade name: **Sodium Bicarbonate**
Generic name: Sodium Bicarbonate
Indications: To correct metabolic acidosis.
Action: Decreases concentration of hydrogen ions.
Site of Action: Hydrogen ions of plasma.
Dosage: Calculated depending on deficit. Dose = base deficit X weight (kg) divided by 4. Give half dose IV and check blood gas for response.

Trade name: **THAM**
Generic name: Tris-(hydroxymethyl) aminomethane
Indications: To correct acidosis.
Action: An alkalizing agent, forms bicarbonate and cationic buffer.
Site of Action: Hydrogen ions of plasma.
Dosage: Calculated dose depends on deficit. Formula is ml of 0.3 M THAM required = kg x HCO_3 deficit (mEq/L) given IV.

Diuretics

Trade name: **Lasix**
Generic name: Furosemide
Indications: Renal failure, no urine output on CPB, acute pulmonary edema, edema associated with fluid retention. Since thiazide diuretics decrease the glomerular filtration rate (GFR), furosemide is superior to those drugs in patients with decreased renal blood flow and GFR.
Action: Inhibits the reabsorption of sodium in the ascending loop of Henle and the proximal and distal tubules which causes more water and chloride to be excreted.
Site of Action: Proximal and distal tubules, loop of Henle.
Dosage: 20-40 mg IV slowly.

Trade name: **Edecrin**
Generic name: Ethacrynic Acid
Indications: CHF, pulmonary edema, renal disease.
Action: Inhibits active transport of sodium in the proximal and distal tubules and the ascending loop of Henle.
Site of Action: Proximal and distal tubules and the ascending loop of Henle.
Dosage: 50 mg IV.

Trade name: **Diamox**
Generic name: Acetazolamide
Indications: Metabolic alkalosis, diuresis.
Action: Carbonic anhydrase inhibitor.
Site of Action: Renal tubules.
Dosage: IV 250 mg twice a day.

Trade name:**Bumex**
Generic name: Bumetanide
Indications: Treatment of edema.
Action: Diuretic.
Site of Action: Renal tubules.
Dosage: PO 0.5-2 mg every day, IV 0.5-1 mg every 2-3 hr for a maximum of 10 mg/day.

Drugs Used for Hyperglycemia/Hypoglycemia

Trade name: **Regular Insulin Injection USP**
Generic name: Regular Insulin
Indications: Treatment of diabetes mellitus, on CPB sometimes used to lower serum potassium if glucose is also high.
Action: Promotes glucose entry to the skeletal, muscle, fat and cardiac cells.
Site of Action: Cell membrane.
Dosage: 10-20 units IV (Regular only) on CPB.

Trade name: **Glucagon USP**
Generic name: Glucagon
Indications: Treatment of hypoglycemia.
Action: Converts glycogen to glucose.
Site of Action: Liver.
Dosage: 1 mg IM or IV.

Steroids

Trade name: **Prednisone**
Generic name: Prednisone
Indications: Any condition where suppression of the response is desired such as allergic states, collagen disease, dermatologic conditions or patients undergoing CPB.
Action: Depresses response of affected tissue.
Site of Action: Cell membranes and metabolic pathways.
Dosage: 10-20 mg PO daily.

Trade name: **Solu-Medrol**
Generic name: Methylprednisolone sodium succinate
Indications: Any condition where suppression of the response is desired such as allergic states, collagen disease, dermatologic conditions or patients undergoing CPB.
Action: Intermediate acting glucocorticoid, depresses response of affected tissue.
Site of Action: Cell membranes and metabolic pathways.
Dosage: For shock 100-250 mg IV.

Trade name: **Solu-Cortef**
Generic name: Hydrocortisone sodium succinate
Indications: Any condition where suppression of the response is desired such as allergic states, collagen disease, dermatologic conditions or patients undergoing CPB.
Action: Short acting glucocorticoid, depresses response of affected tissue.
Site of Action: Cell membranes and metabolic pathways.
Dosage: For shock 500-2000 mg IV.

Trade name: **Decadron**
Generic name: Dexamethasone
Indications: Any condition where suppression of the response is desired such as allergic states, collagen disease, dermatologic conditions or patients undergoing CPB. Adenocortical steroid.
Action: Depresses response of affected tissue by preserving membrane integrity. Prevents lysosomal release from WBC.
Site of Action: Cell membranes and metabolic pathways.
Dosage: For cerebral edema IV 10 mg, PO usage highly individualized depending upon condition being treated. Initial dosage varies from 0.75 to 9 mg a day depending on the disease and the response. Drug must be withdrawn gradually if used more than a few days.

The following medication and dosage has been used in patients who cannot be weaned from CPB. Use for this indication is not approved but several articles in various journals have indicated its effectiveness.

Trade name: **Triostat**
Generic name: T3
Indications: Unable to wean from CPB.
Action: Increases rates of synthesis and utilization of myocardial high energy phosphates.
Site of Action: Heart and all organs of the body due to endocrine effect.
Dosage: 0.4 mcg/kg IV loading dose over 15 min, then 0.4 mcg/kg in 500 ml solution over 4-6 hr. T3 levels must be followed. The medication is continued for 36 hr.

Trade name:

Generic name:

Indications:

Action:

Site of Action:

Dosage:

Trade name:

Generic name:

Indications:

Action:

Site of Action:

Dosage:

Trade name:

Generic name:

Indications:

Action:

Site of Action:

Dosage:

Active Perfusion Programs 2004

Barry University
11300 N.E. 2nd Avenue
Miami Shores, FL 33161-6695
305-899-321

Cardiovascular Perfusion Training Program
Michener Institute of Applied Health Sciences
222 St. Patrick Street
Toronto, Ontario
Canada M5T IV4
888-892-2228

Cariboo College
Cardiovascular Perfusion Technology Program
P.O. Box 3010
Kamloops, British Columbia
Canada V2C 5N
250- 371-5667

Cardiovascular Perfusion Program
Quinnipiac University
275 Mount Carmel Avenue
Hamden, CT 06518
203-582-3427

Christ Hospital School of Perfusion Science
The Christ Hospital
2139 Auburn Avenue
Cincinnati, Ohio 45219
513-585-1106

Circulation Technology Program
Ohio State University
Division of Allied Medical Professions
1583 Perry Street
Columbus, Ohio 43210
614-292-7261

Circulatory Sciences Graduate Program
Cardiothoracic Surgery Services
University of Arizona Health Sciences Center
1501 No. Campbell Ave. Rm. 4402
Tucson, AZ 85724
520-626-6339

Cleveland School of Perfusion Science
Cleveland Clinic Foundation
9500 Euclid Avenue
Cleveland, OH 44195-5001
216-444-9215

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School of Cardiovascular Perfusion
One Cooper Plaza
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Camden, NJ 08103
856-342-3277

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750 E. Adams Street
Syracuse, NY 13210
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985155 Nebraska Medical Center
Omaha, NE 68198-5155
402-559-7227

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College of Nursing and Health Professions
Mail Stop 508
245 N. 15th Street
Philadelphia, PA 19102-1192
215-762-7895

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Medical University of South Carolina
101 Doughty Street, 2nd Floor, Rm. 206
Charleston, SC 29401
843-792-2298

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Perfusion Technology
1025 N. Broadway
Milwaukee, WI 53202-3109
414-277-7561

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Perfusion Technology Program
360 Huntington Avenue
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832-355-4026

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5230 Center Avenue
Pittsburgh, PA 15232
412-623-2482

Vanderbilt University Medical Center
Programs in Allied Health
Program in Cardiovascular Perfusion Technology
Suite #2986 TVC
The Vanderbilt Clinic
Nashville, TN 37232-5734
615-343-9195

Walter Reed Army Medical Center
Cardiothoracic Phys. Asst/Perfusion Training Program
6825 16th St. NW
Bldg 2, Rm 4655 (CT Surgery/Perfusion Services)
Washington, DC 20307-5001
202-782-4865

* Updated information on the perfusion traning programs in North America can be found at http://www.perfusion.com.

NOTES

Other Important Addresses

The American Society of Extracorporeal Technology (AmSECT)
503 Carlisle Dr., Suite 125
Herndon, VA 20170
703-435-8556
703-435-0056 Fax
http://www.amsect.org

The American Academy of Cardiovascular Perfusion (AACP)
P.O. Box 3596
Allentown, PA 18106-0596
610- 395-4853
610-395-4853 Fax
http://members.aol.com/OfficeAACP/

The American Board of Cardiovascular Perfusion (ABCP)
207 N. 25th Avenue
Hattiesburg, MS 39401
601- 582-2227
601-582-2271 Fax
http://www.abcp.org

NOTES

Blood Gas Values

Normal Arterial Blood Gas

pH	7.35-7.45
pO_2	75 - 100 mmHg at room air (on bypass this is higher) (see O_2 tensions below)
O_2 sat.	96 - 100%
pCO_2	35-45 mmHg
BE	0
Bicarb	22-28 mEq/L

Normal Mixed Venous Blood GAS

pH	7.35-7.39
pCO_2	44-48 mmHg
pO_2	38-42 mmHg
O_2 sat	73-77%
BE	-2.5 to + 2.5
P_{50}	27 mmHg

Oxygen Tensions at Various FIO_2 Concentrations

FIO_2	**mmHg**
30%	150
40%	200
50%	250
80%	400
100%	500

Examples of Temperature Correction

Temperature	**pH**	**pCO_2**	**pO_2**
20	7.65	19	27
25	7.58	24	37
30	7.50	30	51
37	7.40	40	80

Cardiovascular Parameters - Normal Adult Values

Function	Normal Value
Cardiac output (CO)	4-8 LPM
Cardiac index (CI)	2.5-4 L/min/m^2
Systolic arterial blood pressure (SBP)	100-140 mmHg
Diastolic arterial blood pressure (DBP)	60-90 mmHg
Mean arterial pressure (MAP)	65-100 mmHg
Pulmonary artery systolic pressure (PAS)	15-30 mmHg
Pulmonary artery diastolic pressure (PAD)	4-12 mmHg
Mean pulmonary artery pressure (MPAP)	9-16 mmHg
Pulmonary capillary wedge press (PCWP)	2-12 mmHg
Central venous pressure (CVP)	0-8 mmHg
Systemic vascular resistance (SVR)	900-1400 dynes/sec/cm^{-5}
Pulmonary vascular resistance (PVR)	150-250 dynes/sec/cm^{-5}
Stroke Volume (SV)	60-130 ml/beat
Heart Rate (HR)	60-100 bpm

Cardiovascular Formulas

Function	Formula
Cardiac output	$CO = HR \times SV$
Cardiac index	$CI = CO / BSA$
Mean arterial pressure	$MAP = DBP + 1/3 (SBP-DBP)$
Systemic vascular resistance	$SVR = [(MAP - CVP) / CO] \times 80$
Pulmonary vascular resistance	$PVR = [(MPAP - PCWP) / CO] \times 80$
Body Surface Area	$BSA = .007184 \times Wt^{0.425} \times Ht^{0.725}$

Celsius (centigrade) - Fahrenheit Conversion

C		F	C		F
0	=	32	37	=	98.6
5	=	41	37.5	=	99.5
10	=	50	38	=	100.4
15	=	59	38.5	=	101.3
20	=	68	39	=	102.2
25	=	77	39.5	=	103.1
30	=	86	40	=	104
32	=	89.6	40.5	=	104.9
35	=	95	41	=	105.8
35.5	=	95.9	41.5	=	106.7
36	=	96.8	42	=	107.6
36.5	=	97.7	43	=	109.4

Fahrenheit to Celcius: $(F - 32) \times 0.555 = C$ or
$(F - 32) \times 5/9 = C$

Celcius to Fahrenheit: $(C \times 1.8) + 32 = F$ or
$(C \times 9/5) + 32 = F$

Common measurements

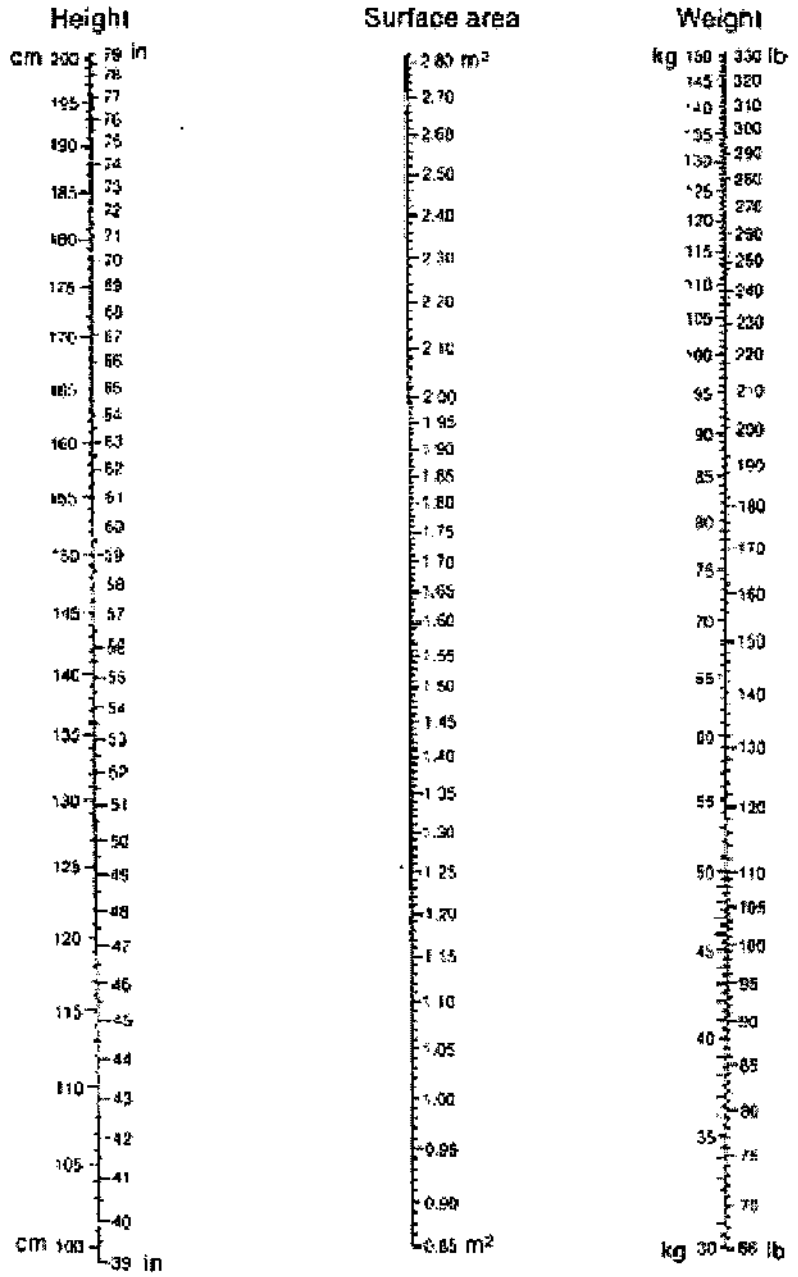
milliliter (ml)	0.001 liter
centiliter (cl)	10 ml
deciliter (dl)	100 ml
liter (L)	1000 ml

millimeter (mm)	0.001 meter	0.04 in
centimeter (cm)	10 mm	0.39 in
decimeter (dm)	100 mm	3.94 in
meter (M)	1000 mm	39.37 in

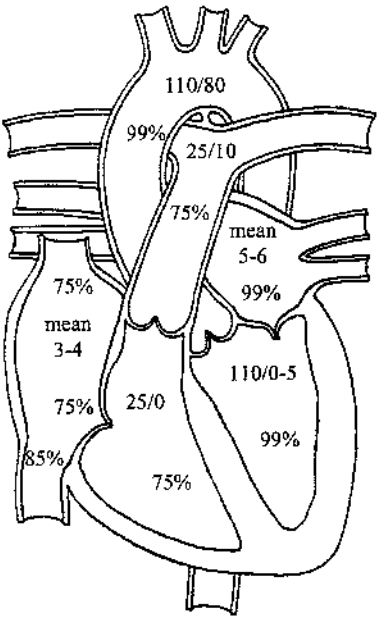
inch	=	2.54 cenitmeter
foot	=	30.480 cm
yard	=	0.914 meter

1 kilogram (kg) = 2.2 pounds (lbs)

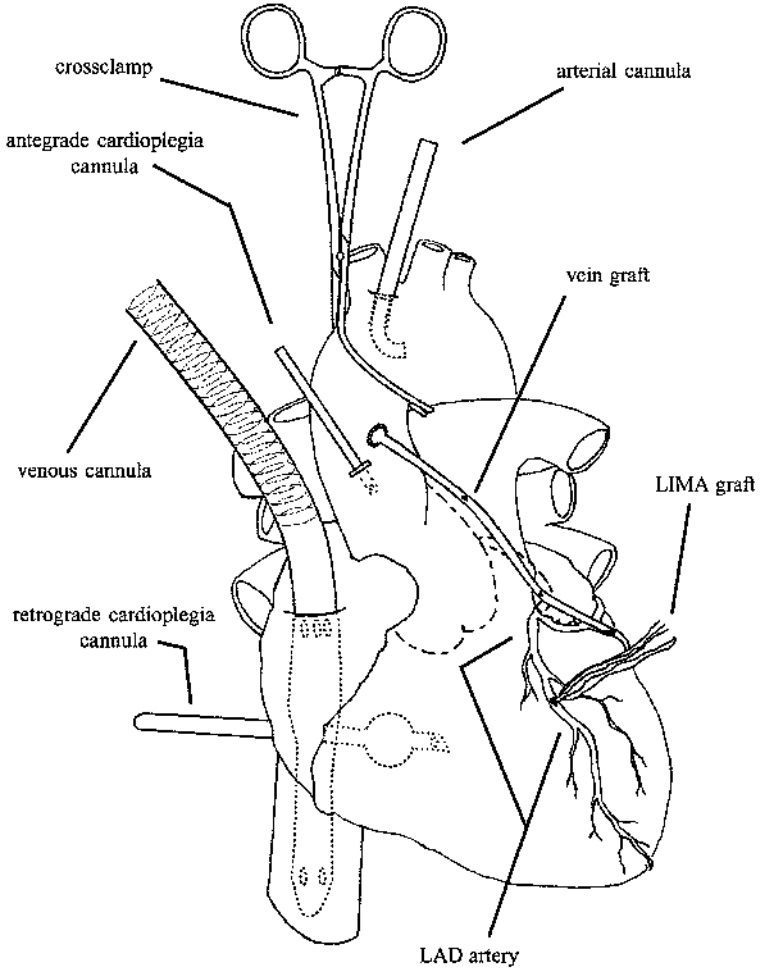
Adult Body Surface Area (BSA) Chart



Normal Heart Pressures and Saturations



Surgical Cannulation



Common Cardiac Drugs

Antiarrhythmic Drugs

Drug	Dose (often requires mixing with solution)
Bretylium	IV push 5-10 mg/kg over 10 min, then 1-2 mg/min at rate of 1mg/min
Esmolol	IV push 500 mcg/kg over 1 min, may repeat in 5 min, then 50-300 mcg/kg/min at rate of 50-200 mcg/kg/min
Lidocaine	IV push 1mg/kg, 2 doses 20 min apart, then 1-4 mg/min at rate of 1-2 mg/min
Procainamide	IV push 10-50 mg/min, max 600 mg, then 1-6 mg/min at rate of 1-3 mg/min

Calcium Channel Blockers

Drug	Dose (often requires mixing with solution)
Diltazem	IV push 0.25 mg/kg over 2 min may give 2nd dose of 0.35 mg/kg after 15 min if necessary, then 10-15 mg/hr at rate of 10-15 mg/hr
Nicardipine	5-15 mg/hr, after desired response, 3 mg/hr
Verapamil	IV push 0.075-0.15 mg/kg over 2 min, may repeat once in 15 min

Sympathomimetics

Drug	Dose (often requires mixing with solution)
Dobutamine	2-20 mcg/kg/min at rate of 2-10 mcg/kg/min
Dopamine	2-20 mcg/kg/min at rate of 2-5 mcg/kg/min 0.15-2.5 mcg/kg/min at rate of 0.15 mcg/kg/min
Epinephrine	IV push 0.1-0.25 mg for anaphylatic shock, then 0.01-0.06 mcg/kg/min at rate of 0.015 mcg/kg/min
Isoproterenol	IV push 0.02-0.06 mg, then 0.015-0.07 mcg/kg/min at rate of 0.015 mcg/kg/min
Norepinephrine	0.01-0.1 mcg/kg/min at rate of 0.01 mcg/kg/min
Phenylephrine	50-100 mcg, then 0.15-2.5 mcg/kg/min at rate of 0.15 mcg/kg/min

Nonsympathomimetic Inotropics

Drug	Dose (often requires mixing with solution)
Milrinone	IV push 50 mcg/kg over 10 min, then 0.375-0.75 mcg/kg/min at rate of 0.375 mcg/kg/min

Vasodilators

Drug	Dose (often requires mixing with solution)
Nitroglycerine	IV push 50-100 mcg, then 0.1-2 mcg/kg/min at rate of 0.1-2 mcg/kg/min
Nitroprusside	0.1-8 mcg/kg/min at rate of 0.1-2 mcg/kg/min

Other

Drug	Dose (often requires mixing with solution)
Calcium chloride (inotrope if hypocalcemic, vasopressor if normocalcemic)	IV 2-10 mg/kg over several minutes

Bayer HealthCare Pharmaceuticals

Bayer
Pharmaceuticals Corporation
400 Morgan Lane
West Haven, CT 06516

Clinical Communications
1-800-288-8371

Local Representative

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PRODUCT LISTING

Fibrinolytic Inhibitor	Trasylol® (aprotinin injection)
Antithrombotic	Thrombate III® (antithrombin III concentrate)

COBE Cardiovascular®

A SORIN GROUP COMPANY

COBE Cardiovascular, Inc.
14401 W. 65th Way
Arvada, CO 80004-3599

Local Representative

Customer Service 1-800-221-7943

PRODUCT LISTING

Autologous Blood Salvage

BRAT® 2 Autologous Transfusion System
Electa Essential Cell Separator Autotransfusion System

Cardioplegia Delivery Systems

Vanguard (8:1, 4:1, 2:1 and Custom) Heater/Cooler
CSC-14 (8:1, 4:1, 2:1 and Custom) Heater/Cooler
Versaplegia (Stock, Custom) PVC Coil and Stainless Steel Coil

Centrifugal Pumps

Revolution® Centrifugal Blood Pump

Circuits

COBE Custom Tubing Packs
COBE Blue Ribbon Pack® Preconnected Custom Tubing Packs
COBE Mini-Blue Ribbon™ Custom Tubing Pack

Connectors

Bulk sterile and non-sterile in a wide variety of sizes

Filters

Sentry® 43 and 21 micron ALF - Primegard® coating available
Sentry™ 43 micron Arterial Filter with SMA
Dideco Adult 20 and 40 micron Arterial Filters
Dideco Adult 40 micron Arterial Filters with PC
Dideco Pediatric 20 and 40 micron Arterial Filters
Dideco Neonate 20 and 40 micron Arterial Filters
Dideco Neonate 40 micron Arterial Filter with PC

Hemoconcentrators

COBE 1400 and 700 Hemoconcentrators
Dideco DHF0.2 Hemoconcentrator

COBE Cardiovascular®

A SORIN GROUP COMPANY

COBE Cardiovascular, Inc.
14401 W. 65th Way
Arvada, CO 80004-3599

Customer Service 1-800-221-7943

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PRODUCT LISTING CONTINUED

Oxygenators

- CML Duo® Adult Flat Sheet Oxygenator
- Optima XP™ Adult Hollow Fiber Oxygenator
- COBE Apex Oxygenator
- Dideco Avant Oxygenator
- Dideco EOS Pediatric Oxygenator
- Dideco Lilliput Pediatric Oxygenator
- Synergy™ Mini-Bypass System
- Synthesis™ Integrated Oxygenator
- Synthesis 4D™ Integrated Oxygenator with Revolution™

Perfusion Equipment

- COBE SAT/HCT® Monitor
- Stöckert SIII
- Stöckert Centrifugal Pump and Console
- Cincinnati Sub-Zero Hemotherm Heater/Cooler and ECMO Heater
- Sechrist Blender

Reservoirs

- VVR4000 Sealed Venous Reservoir
- VRB® 1200 & 1800 Closed Venous Reservoirs
- MVR 1200 Closed Venous Reservoir
- Filtered and Non-Filtered Cardiotomy Reservoir
- Filtered and Gross Filtered Blood Collection Reservoirs

Tubing

- Both sterile and non-sterile, bulk and custom lengths

A SORIN GROUP COMPANY

Dideco S.p.A.
Via Statale 12 Nord, 86
41037 Mirandola Modena
Italy
Phone +39 0535 29811
Fax + 39 0535 25229
http:// www.dideco.com

Local Representative

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PRODUCT LISTING

Oxygenators:

D 903 Avant Adult oxygenator
D 905 Eos Paediatric/small adult oxygenator
D 902 Lilliput 2 Infant oxygenator
D 901 Lilliput 1 New Born Oxygenator

All in various configurations and coated/non coated (Phisio-Phosphorylcholine coating)

Circuits: Dideco personalized Custom Tubing Packs

Cardioplegia:

Vanguard Blood Cardioplegia (1:1 – 4:1 – 8:1),
Crystalloid Cardioplegia

Heat Exchanger: Sidus (1:1 – 4:1 – 8:1)

Haemofilters: DHF 0.2 Haemoconcentrators, DHF 0.6 Haemoconcentrators

Filters: Adult, Infant/Paediatric and Newborn filters

Perfusion Equipment:

SIII Stöckert Heart/Lung Machine in various configurations
SCPC centrifugal pump
Stöckert Heater Cooler
Data Master Perfusion Monitor

A SORIN GROUP COMPANY

Dideco S.p.A.
Via Statale 12 Nord, 86
41037 Mirandola Modena
Italy
Phone +39 0535 29811
Fax + 39 0535 25229
http:// www.dideco.com

Local Representative

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PRODUCT LISTING CONTINUED

Mini bypass: Dideco device for mini bypass surgery

Connectors: Bulk sterile and non-sterile in wide variety of sizes.

Reservoirs:

DAC Dideco Adult Reservoir, Venomidicard and Midicard Paediatric Reservoir, Venocard Adult Reservoir, D 970 Avant Dual Reservoir

Tubing: Both sterile and non-sterile, bulk and custom lengths

Autologous Blood Management

Autotransfusion:

Electa Autotransfusion Cell Separator
Disposables and accessories for Autotransfusion

Autologous Platelet Gel

Suprema Autologous Blood Management Cell Separator (integrated PRP separation and Autotransfusion)
Disposables and accessories for Autologous Blood Management
Activat Autologous Thrombin Processing Set
New products dedicated to PRP separation for Platelet Gel procedures

Gish Biomedical, Inc.
22942 Arroyo Vista
Rancho Santa Margarita, CA 92688
http://www.gishbiomedical.com

Customer Service 1-866-221-9911

Local Representative

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PRODUCT LISTING

Centrifugal Pump Systems
- Centrifugal Pump System and Flow Probe

Extracorporeal Tubing Sets and Components
- Custom Tubing Circuits
- Heat Exchangers
- Connectors
- Tubing and Tubing Organizers and Accessories

Filters
- GAF 25m or 40m Arterial Filters
- Gas Line and Prebypass Filters

Hemoconcentrators
- Gish Biomedical offers the Minntech Line of Hemoconcentrators.

Myocardial Protection Systems
- Vision Blood Cardioplegia System
- Straight Shot Blood Cardioplegia System
- Recirculation Batch Cardioplegia Systems
- Coil Based Cardioplegia Systems

Oxygenators
- Vision Hollow Fiber Oxygenator System available with Hardshell or Softshell Venous Reservoirs

Resevoirs
- Cardiotomy/Autotransfusion/Pleural Drainage Reservoirs
- Hardshell Venous Reserviors
- Soft Venous Reservoir Bags

Statsat
- Oxygen Saturation Monitor and Accessories

Surgical Accessories
- Vision Aortic Punches in Assorted Sizes and Lengths

* Select Products Available with the GBS™ Coating!*

International Biophysics Corp
2101 East St Elmo
Bldg# 2 Suite 275
Austin, TX 78744

Customer Service: 1-512-326-3244

Fax Line: 1-512-326-3299

Local Representative

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PRODUCT LISTING

FloPump: Centrifugal Pump and Console.

FloProbe: Used in conjunction with FloPump.

Tubing Organizers: Two styles (See Website for details). Designed to stabilize and contain one to six tubing lines of varying sizes which connect cardiopulmonary bypass equipment.

Aortic Punches: Available in 2.8mm, 3.6mm,4.0mm, 4.4mm, 4.8mm, 5.2mm, 5.6mm, 6.0mm. Medium and Long assembly. Custom printing also available.

Suction Wand: A component of the blood recovery suction system for use in open-heart procedures or autologus blood recovery. Mini Sucker and Standard Size Sucker available.

* Please visit the IBC website at **www.biophysicscorp.com** for more product and company information.

PERFUSION.COM INC.

SERVICE - SUPPLIES - SUPPORT

www.perfusion.com

PERFUSION.COM, INC.
500 Keenan Avenue
Fort Myers, FL 33919
http://www.perfusion.com
services@perfusion.com

National Representative

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Customer Service: 1-866-872-8065
Fax: 1-425-795-0565

Customer Service 1-866-872-8065

PRODUCT LISTING

Perfusion Services
- Contract Perfusion Services (Locum Tenens)
- Autotransfusion and Platelet Sequestration Services
- Equipment Service and Preventative Maintenance

Equipment Sales
- Perfusion Books
- Perfusion Supplies
- Platelet Gel Products
- Circuit Components
- Perfusion Hardware (New and Used)
- Surgical Supplies and Instruments (Tubing Clamps)
- Perfusion Software (PerfBase Database Software)

Employment Services
- On-line Job Placement and Listings
- On-line Employment Services
- Employee Screening Services

Information Services
- Perfusion and PRP Information for Medical Professionals
- General Member and Premium Member Services
- Marketing and Advertising Services for Medical Corporations

*Please visit http://www.perfusion.com or call us toll free at (866) 872-8065 for more information about our products and services.

PPAI Medical

PPAI Medical
6227 Foxfire Lane
Fort Myers, FL 33912
http://www.ppaimedical.com

National Representative

Customer Service 1-888-801-8814 _____

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PRODUCT LISTING _____

Platelet Concentrating System

-Secquire Low Volume Platelet Concentrating System with IV Start Kit and Platelet graft preparation dish

-Secquire Limited with two Secquire Cell Separators and four 60cc syringes

Platelet Rich Plasma Delivery Systems

Dual tip, aerosol spray, malleable tip, and spray misterfier delivery systems suitable for all surgical and wound preparation needs

Aerosol Contained Buckets

Secquire Centrifuge Machine

Operates at speeds up to 8500RPM/4680xG

Accommodates loads up to 300mL (6x50mL)

Standard Features include:

At speed timer

Variable speed control

Cover interlock

Run time set from 5 seconds to 1 minute in 5-sec intervals

From 1 minute to 5 minutes in 15-sec intervals

From 5 minutes to 30 minutes in 1-min intervals

Secquire PRP Dressing Kit

2 Sorbaview Dressings 6.5 x 5 in

1 Kerlex Wrap 3.4 x 3.6 yds

8 Tape Strips 1 x 4 in

5 Gauze 4 x 4 in

Service Support Kit

Three basic PRP powerpoint presentations (periodontal, plastic, & woundcare) that you may customize

General PRP application photos PRP service startup forms

BIBLIOGRAPHY

Baumgartner, Owens, Cameron, Reitz, ed. *The Johns Hopkins Manual of Cardiac Surgical Care*. St. Louis, Washington, Toronto: C.V. Mosby Company, 1994.

Berger E. *The Physiology of Adequate Perfusion*. St. Louis: The C.V. Mosby Company, 1979.

Berne R and Levy M. *Cardiovascular Physiology*. 5th edition. St. Louis, Washington, Toronto: C.V. Mosby Company, 1986.

Braunwald, Isselbacher, Petersdorf, Wilson, Fauci. *Harrison's Principles of Internal Medicine*. New York: McGraw-Hill, 1987.

Cotes J. *Lung Function: Assessment and Application in Medicine*, ed. 3. Philadelphia: J.B. Lippincott Co., 1975.

Davenport H. *ABC of Acid-Base Chemistry*. ed. Chicago: The University of Chicago Press, 1974.

Dillard D, Miller D. *Atlas of Cardiac Surgery*. New York: Macmillan, 1983.

Dorland's Illustrated Medical Dictionary. 27th edition. Philadelphia: WB Saunders, 1988.

Filley G. *Acid-Base and Blood Gas Regulation*, ed. 2. Philadelphia: Lea and Febiger, 1971.

Fishback D and Fogdall R. *Coagulation, the Essentials*. Baltimore: Williams and Wilkins, 1981.

Fozzard H, Haber E, Jennings R, Katz A, Morgan H, ed. *The Heart and Cardiovascular System*. New York: Raven Press, 1984.

Gravlee G, Davis R, and Utley J, ed. *Cardiopulmonary Bypass Principles and Practice*. Baltimore: Williams and Wilkins, 1993.

Grillo H, Austen W, Wilkins E, Mathisen D, Vlahakes G, ed. *Current Therapy in Cardiothoracic Surgery*. Burlington, Ontario: BC Decker Inc., 1989.

Guyton A. *Textbook of Medical Physiology*, 7th edition. Philadelphia: WB Saunders, 1986.

Hilberman M, ed. *Brain Injury and Protection During Heart Surgery*. Boston: Martinus Nijhoff Publishing, 1988.

Ionescu M. ed. *Techniques in Extracorporeal Circulation*. 2nd edition. London, Boston, Sydney: Buttersworth, 1981.

Jandl J. *The Textbook of Hematology*. Boston, Toronto: Little Brown and Co., 1987.

Kirklin J, Barratt-Boyes B. *Cardiac Surgery*. New York: John Wiley and Sons, 1986.

Myocardial Protection in Cardiac Surgery. New York: Marcel Dekker Inc,. 1987.

Phillips R, Feeney M. *The Cardiac Rhythms*. 2nd edition, Philadelphia: W. B. Saunders Company, 1980.

Quaal S. *Comprehensive Intraaortic Balloon Counterpulsation*. 2nd edition. St. Louis, Baltimore: Mosby Yearbook, 1993.

Physician's Desk Reference. 48th edition, Montvale, NJ: Medical Economics Data Production Company, 1984.

Reed C and Clark D. *Cardiopulmonary Perfusion*. 2nd edition. Houston: Texas Medical Press, 1985.

Shapiro B, Harrison R, and Walton J. *Clinical Application of Blood Gases*. Chicago, London: Yearbook Medical Publishers, 1985.

Smith J and Kampine J. *Circulatory Physiology*. 2nd edition. Baltimore: Williams and Wilkins, 1984.

Taylor K, ed. *Cardiopulmonary Bypass: Principles and Management*. London: Chapman and Hall Ltd., 1986.

Utley J, Ashleigh E, ed. *Pathophysiology and Techniques of Cardiopulmonary Bypass*. Baltimore: Williams and Wilkins, 1982:1.

Utley J, Betleski R, ed. *Pathophysiology and Techniques of Cardiopulmonary Bypass*. Baltimore: Williams and Wilkins, 1983:2.

Utley J, Betleski R, ed. *Pathophysiology and Techniques of Cardiopulmonary Bypass*. Baltimore: Williams and Wilkins, 1985:3.

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THE MANUAL OF CLINICAL PERFUSION

SECOND EDITION UPDATED

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