Open Heart Surgery

ARTICLE BY PEGGY GRUSENDORF, MS, FNP; RICHARD E. WILLS, MD, FACSM, MBA; AND JAMES A. WOODMANSEE III, MD, AAFP

Hypertensive heart disease, coronary heart disease, and rheumatic heart disease are public health concerns throughout the world. The incidence of coronary heart disease is increasing in many populations while decreasing in others.

Culture determines the prevalence of individuals with elevated blood lipids and the total outcome of risk, frequency, and potential for prevention of coronary heart disease. A diet composed of significant fatty acids, cholesterol, vegetable protein, complex carbohydrates, caloric excess, and high salt intake leads to a marked increase in risk of heart disease. Caloric excess can also influence health directly by metabolic maladaptions of obesity, hyperlipidemia, hyperinsulinism, and hyperuricemia.

The following article reviews the anatomical structure of the cardiovascular system, with an emphasis on the pathophysiology of heart disease. The central surgical procedure of open heart surgery, used to repair damage caused by the cardiovascular disease process, is described.

Epidemiology of Heart Disease

Atherosclerosis is four times more frequent in males than in females. The basic lesion of plaque is often localized in the first five centimeters of the origin of the coronary artery from the aorta. A common pattern of occlusion is seen in the proximal anterior descending with the distal part of the artery remaining 50% open. The right coronary artery is frequently occluded with the posterior descending and left atrial ventricular groove branches remaining patent while the circumflex is often involved proximally and has patency with one or more distal branches.

The risk of sudden death will vary with the extent of disease present along with the degree of impairment of ventricular function. Death will probably result from ventricular fibrillation.

The most common complication of coronary disease is myocardial infarction. In the United States, 3 million people will have an infarction with most deaths occurring within 30 to 60 minutes after the onset of symptoms.

Pathophysiology of Coronary Artery Disease

Coronary artery disease (CAD) is responsible for nearly 50% of all deaths and one third of all deaths to people between the ages of 35 and 65 in the United States.

Patients afflicted with coronary heart disease or ischemic heart disease suffer from pump failure (decline in contractile function). Coronary heart disease can be categorized as either modifiable and non-modifiable. Risk factors associated with modifiable coronary heart disease include hypertension, hyperlipidemia, smoking, diabetes mellitus type II, obesity, alcohol consumption (heavy), sedentary lifestyle, hormone therapy, and psychosocial factors. Risk factors for coronary heart disease that are nonmodifiable include race, genetics, age, male sex, and diabetes mellitus type II.

The initial loss of functional myocardial tissue places an abnormal burden on the remaining regions of the left ventricle, thereby causing an overload that may or may not be tolerated over time with little normal myocardium remaining. In some patients, heart disease leads to acute or chronic heart failure. In patients with enough myocardial loss, severe impairment of the cardiac pump may result. The chronic heart failure that results from ischemic heart disease can be attributed to the death of large numbers of myocardial cells that were deprived of their blood supply. The rapid decline in myocardial contractility that occurs after coronary arterial occlusion is secondary to the lack of a substrate supplied by the coronary circulation and the accumulation of metabolites of cellular respiration.

The substrate whose absence is felt most promptly by the ischemic myocardium is oxygen. This state of myocardial anoxia develops because the heart has virtually no oxygen stores. The theory that oxygen is a critical substrate, the lack of which may be responsible for the early pump failure of the ischemic heart, is supported by the finding that anoxia and ischemia cause a precipitous decline in cardiac contractile function.

Rheumatic heart disease has been an important cause of valvular heart disease. In the past, rheumatic endocarditis occurring during rheumatic fever was responsible for a vast majority of cases of mitral valve disease and about half of the cases of aortic valvular disease. This has changed, due to improvements in housing and widespread use of penicillin to treat tonsillitis and reduce the incidence of recurrences of rheumatic fever.

Increased longevity, antibiotic treatment of infective endocarditis and other infections, and increased survival of patients with congenital heart lesions have also contributed to the change in the types of valvular disease encountered.

Congenital, degenerative, and nonrheumatic infective causes are observed more frequently as rheumatic valve disease becomes rarer in the United States.

Anatomy of the Heart

The cardiovascular system is the first system to function in the embryo; circulation begins at the end of the third week. The development of cardiac muscle begins at the endocardial heart tube. The basic cardia myoblasts differentiate to form the myocardium, or heart mus-

cle. Growth of the cardiac muscle fibers results from the formulation of new myofilaments. During this time, the intercalated disks are developed along with Purkinje's fibers, which later form the conducting system of the heart.

The heart is lined on the inside by endocardium; the contractile walls of the chambers are formed from myocardium (Figures 1 and 2). The heart is divided into four areas: the right atrium, the right ventricle, the left atrium, and the left ventricle (Figures 1 and 2).

The right atrium is separated from the left atrium by the interatrial septum (see Figure 1). The right atrium receives blood high in carbon dioxide (CO^2) from the superior and inferior vena cava, the major veins of blood returning to the heart (Figure 3). The blood is then pushed past the three-leafed tricuspid valve to the right ventricle (see Figure 1). From there, the CO^2 saturated blood is pushed through the pulmonic, or semilunar, valve to the pulmonary artery and then to the right and left lungs for oxygenation (see Figures 1 and 3). The pressure in the right ventricle is suited for the low resistance of the lungs (see Figure 3). If the resistance in the lungs is increased, the right ventricle will increase in size, most likely due to systemic venous congestion with associated peripheral edema.

The red blood cells that have been oxygenated by the lungs enter the left atrium by way of the four pulmonary veins (see Figure 3). The left atrium is slightly larger than the right atrium because of the pressure created by the thick left ventricular wall (see Figures 1 and 3). From the left atrium the blood passes through the two leafed (bicuspid) mitral valves into the left ventricle, which has the most muscular walls of the four heart chambers (see Figure 1). The blood is then pushed from the left ventricle as a result of ventricular myocardial contraction, through the aortic (semilunar) valve to the rest of the body (see Figures 1, 2 and 3).

The conducting system of the heart consists of specialized muscle cells known as the sinoatrial node (S-A node) and the atrioventricular node (A-V node) that pass to the atrioventricular bundle to the subendocardial plexus of Purkinje's fibers. The S-A node is located in the right atrium near the superior vena cava. This specialized area has two types of cells: P cells and T cells. The S-A node has an intrinsic firing rate of 90 to 120 beats per minute with an approximate normal rate of 70 beats per minute. Extreme cold will decrease the heart rate, while elevated heat will increase the heart rate. Other factors that control firing rate are ions, especially calcium and potassium. Extreme overload or deficiency will cause abnormal heart rates to occur.

As blood leaves the aorta, it travels to substantial branches of vessels—carotid, iliac, and axillary—which are composed of large amounts of elastin and some smooth muscle (see Figure 3). This causes the arteries to become distensible, which means that as blood volume increases through them during exercise, they are able to accommodate the changes in pressure. On the other hand, veins are much thinner than arteries and are able to perform with less resistance of flow, creating a much higher velocity than is found in the arteries (see Figure 3).

With the contractile fibers of the myocardium (heart muscle), the heart propels blood throughout the body to deliver nutrients to and remove wastes from the organ systems. Another function is to transport hormones that regulate and interrelate with various systems and regions of the body (see Figure 3).

Heart muscle (myocardium) is more complex than skeletal and smooth muscles, with different contractile properties. The muscle fibers are stimulated via motor neurons from the central nervous system (Figure 4). Within these myocardial fibers are specialized cells called intercalated disks. These disks are low-resistance pathways between cells that initiate an action potential, which is then propagated simultaneously to all regions of the myocardium by internodal tracts (bundles of specialized cells) that connect the S-A node and A-V node.

The contraction of the myocardium starts at the sarcolemma and T-tubes that have received an action potential that depolarizes them. This causes release of calcium from the sarcoplasmic reticulum. The calcium binds with other molecules (troponin) and forms cross-bridges between the myosin and actin filaments. If there is an increase in calcium during the action potential, a more vigorous contraction will occur. The types of heart rates that will develop are normal cardiac rhythm at 60 to 80 beats per minute, bradycardia at less than 50 beats per minute, or tachycardia at more

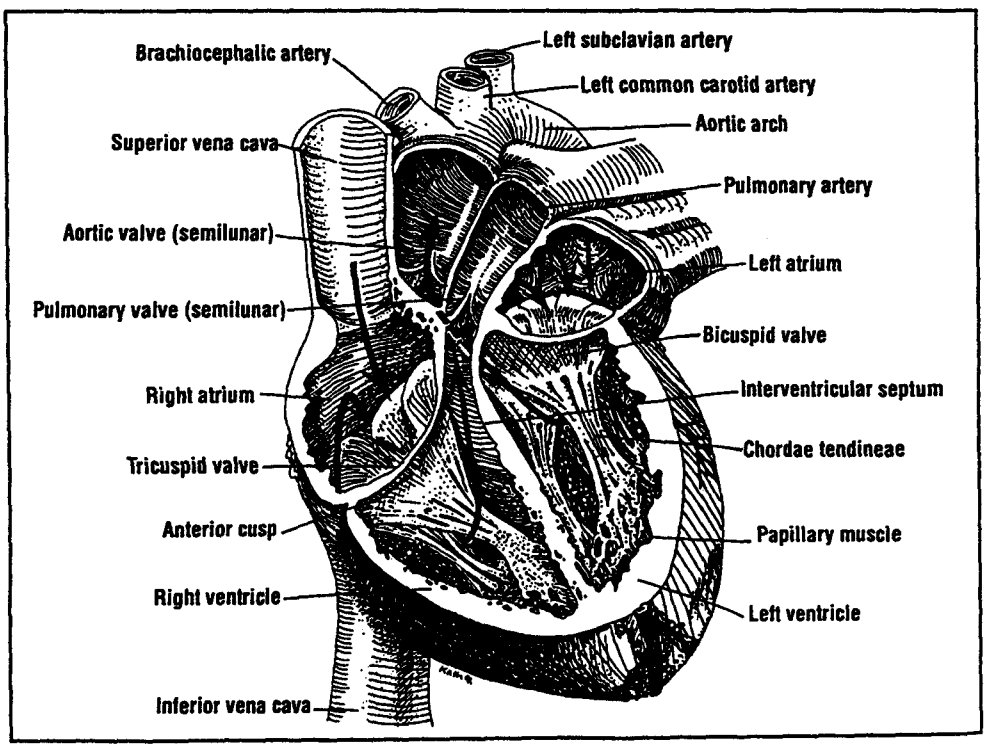

Figure 1. Anatomy of the heart (anterior view).

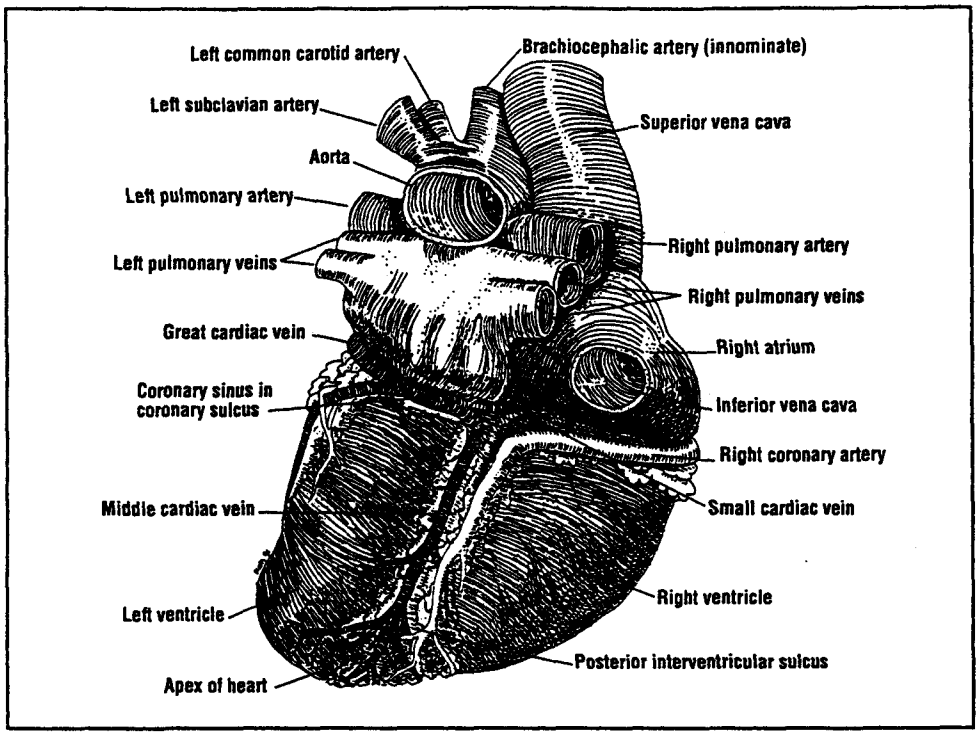

Figure 2. Anatomy of the heart (posterior view).

than 100 beats per minute. An increase in heart rate will cause a decrease in arterial blood volume and will raise arterial pressure. Maximum heart rates can be determined by an individual's age, using the following formula: 210 - (0.65 x age in years). The body's cardiovascular system requires maintaining a heart rate of 60% to 70% of the maximum heart rate for 20 to 30 minutes at least 5 days per week and for greater than 4 months to receive maximum health benefits.

The maintenance of blood volume is regulated by the kidneys, nerves, and hormones (Figure 5). The kidneys receive a hormone called antidiuretic hormone (ADH) that detects increased volume in blood fluid levels or osmolality. ADH causes increased reabsorption of water from the kidneys along with water retention and dilution of solutes. The kidney also has the renin-angiotensin-aldosterone system that will control body electrolytes. A decrease in pressure causes a release in renin, which then converts angiotensin I to angiotensin II and causes the vessels to constrict, thus increasing blood pressure to normal.

Hypertension

Changes in the levels of blood pressure throughout the body, especially in the kidney, play a role in certain causative forms of hypertension. The definition of hypertension is a diastolic (lower) pressure above 90 mm Hg and a systolic (upper) pressure greater than 140 mm Hg. Elevations in systolic pressure are generally thought to be less significant than elevations in diastolic pressure.

Ninety percent of all hypertension is of uncertain origin and is labeled primary, or essential, hypertension. The remaining 10% is called secondary hypertension because it is associated with a variety of renal, endocrine, neurologic, or vascular disorders.

Primary, or essential, hypertension is present in about 5% of the adult population of the United States. Females are affected more frequently than males, usually within their fourth or fifth decade of life. The disease incidence increases with age with as much as 50% of the population over age 50 suffering from primary hypertension. This type of hypertension may eventually lead to cardiovascular and cerebrovascular disorders, but the development of these complications is a function of the severity of the blood pressure elevations (see Figure 5). Milder forms of this disease are called benign hypertension, which can lead to atherosclerosis and its complications such as thickening of the walls of the arteries and arterioles in the kidney which in turn leads to increased hypertension.

Essential hypertension is believed to be caused by a combination of genetic and environmental factors. There is an increased prevalence of hypertension among blacks with higher occurrences within hypertensive families. In the United States, behavior patterns, stress, obesity, and use of oral contraceptives are common conditions under which hypertension develops. Dietary sodium also has a relationship to the occurrence of essential hypertension. Americans consume eight or more grams of sodium every day, which correlates to an 8% to 25% incidence of hypertension.

Hypertension causes a number of serious disorders. The presence of increased blood volume in the left ventricle that creates elevated pressure for long periods of time can lead to an enlarged ventricular muscle (hypertrophy) and thus an enlarged heart. There is increased incidence of arteriosclerosis with myocardial infarcts (heart attacks) with hypertension, even when hypertrophy is not present. Hypertensive individuals are also predisposed to thromboses of cerebral vessels that lead to cerebral hemorrhage (stroke).

Obesity

Obesity is an excessive amount of body fat with a body weight of more than 20% of the standard weight for the height and frame of an individual. The excess lipid deposit occurs because energy intake exceeds energy output. Obesity tends to run in families. The children of obese individuals are sometimes even more overweight than their parents.

Eating disorder is the most common cause of obesity in humans. Obesity is mild at 20% to 40% over ideal weight, moderate at 41% to 100% over ideal weight, and severe when above 100% over ideal weight. It is believed that the body weight of many people, including the obese, is subject to physiologic regulation, and that elevation of the regulatory level, or set point, is responsible for obesity. In individuals with marked obesity, there is often a serious disintegration of the intake regulating mechanism.

In women, social factors are often determinants of obesity. Obesity occurs six times more frequently in lower class-

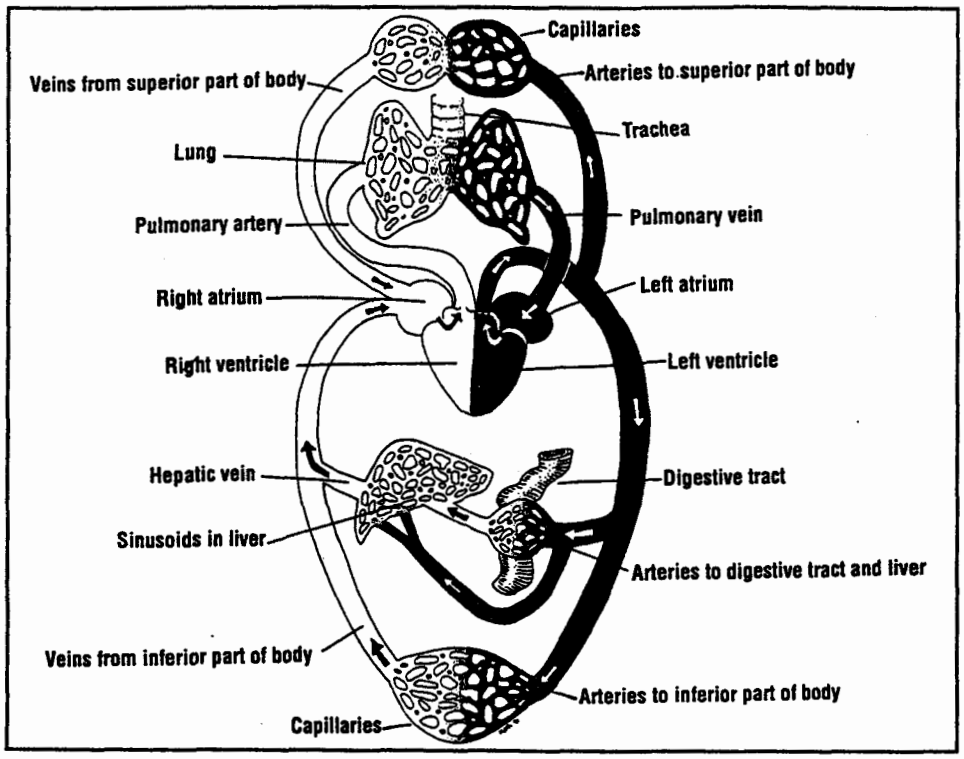

Figure 3. Pulmonary and systemic circulatory system.

es than in upper classes. Ethnic and religious factors are also closely linked to obesity, partly due to differences in dietary and exercise roles.

Hormonal factors, as seen in hyperadrenocorticism where corticosteroid excess leads to an increase in gluconeogenesis and a greater demand for insulin leading to an increase in lipogenesis, are also elements that must be taken into consideration in obesity. Certain metabolic abnormalities may occur in the hypothalamus of the brain, causing an increased level of fullness in the satiety center and leading to metabolic obesity.

Fat cells create a reservoir of energy that expands or contracts according to the energy balance of the organism. They develop from preadipocytes to take on excess calories. With continuing increase in energy balance, new adipocytes form from precursor cells, and the total cell number increases. Throughout weight loss, fat cell numbers remain fixed. Free fatty acids can enter the adipocytes and can again be esterified to triglycerides and stored.

Fat is distributed differently in men and women. In a man, fat is distributed mostly in the upper body above the waist; in a woman, fat is distributed predominantly in the lower body. (This distribution is influenced by the presence of testosterone and estrogen hormones and may act differently on upper and lower fat cells.) Excessive body fat in the upper body increases morbidity and mortality rates more than when excess fat is distributed in the lower body. Increased distribution of fat in the abdominal region can lead to increased risk of hypertension, cardiovascular disease, and diabetes mellitus. The risk of these conditions is therefore greater in men than in women.

Hormonal mechanism can control the deposit of fat in the human body. Insulin promotes the deposit of intracellular triglycerides by stimulating the conversion of glucose to fatty acids and glyceride to glycerol by maintaining adipose tissue lipoprotein lipase levels and inhibiting intracellular lipolysis. Estrogens operate at the level of the adipocyte and cause development of subcutaneous adipose tissue.

Physical problems may be associated with obesity as seen in diabetes mellitus. Some obese individuals may develop non-insulin-dependent diabetes mellitus (NIDDM). The prevalence of diabetes is three times higher in overweight persons versus non-overweight persons. In the United States, 85% of those with NIDDM are obese.

Hypertension, a blood pressure of greater than 160/95 mm Hg, is three times more common in overweight individuals than those who maintain normal weight. Associated cardiovascular disease is seen in the obese, which leads to increased blood volume, stroke volume, and filling pressure, all of which result in high cardiac output. This can induce left ventricular hypertrophy (enlarged left ventricle) and increase the risk of congestive heart failure.

There is a marginal correlation between high cholesterol and obesity. Hypertriglyceridemia is also more common in obese individuals because of insulin resistance and hyperinsulinism. This is a precursor to increased hepatic triglyceride production. This increased triglyceride production can sometimes be controlled and decreased through weight loss, but if it is genetic, more intensive therapy is required.

Obesity can lead to chronic hypoxia with cyanosis and hypercapnia. These people have an increased demand for ventilation and suffer from breathing overload. This can lead to the inefficiency of respiratory muscles. Severely obese individuals often have varicose veins and venous stasis, which may be a forerunner of thrombophlebitis and thromboembolism associated with pulmonary embolism of the lungs.

Endometrial cancer and breast cancer are two times more common in obese women. These women have a higher incidence of cancer of the gallbladder and biliary system as well. Obese men have a higher mortality from cancer of the colon, rectum, and prostate for reasons that are yet unknown. With increased obesity, individuals carry greater risk for complications and mortality from abdominal surgeries. A prevalent increase in gallstones is noted among obese people, and many of these individuals have fatty livers and abnormal liver function tests.

Arthritis is commonly seen in severely obese men and women due to excess stress placed on the joints, particularly in the lower extremities and lower back.

Overall, obesity is associated with increased mortality with causes listed as increased coronary heart disease, cerebral hemorrhage (stroke), diabetes,

digestive disease, and cancer.

Surgical Procedure

Patient Positioning and Preparation
After preinduction of anesthesia has been completed, intravenous infusions and cutdowns are performed as monitoring leads are prepared and secured in place.

Following intubation, a venous pressure catheter may be inserted into the external jugular vein through a percutaneous needle cannula. The radial or femoral artery may be cannulated in the same manner before the patient is positioned for the selected approach. A large surface electrocoagulation-indifferent plate is placed under the patient's buttocks or back after conductive gel has been applied, and an indwelling urethral catheter is connected to a receptacle. Special attention is given to pressure areas with protective padding used as indicated. Routine skin preparation and draping of the patient is completed.

Incisional Approaches
The heart is approached by a right or left posterolateral thoracotomy, a median sternotomy, a transsternal bilateral thoracotomy, or an anterolateral thoracotomy. The age of the patient, the pathological defect, the proposed correction, and the surgeon's preference are all taken into consideration before selecting the type of incision.

There are also many considerations in cannulation for the arterial return. The aortic, subclavian, iliac, or femoral arteries may be used.

A posterolateral thoracotomy incision is made with the incision following the course of the right or left rib. Access to both groin regions can be permitted with a modified full lateral position. The fifth rib is resected and a Burford-Finochietto rib spreader is inserted. The pleura is incised and the lung is retracted with a moist pack to expose the pericardium.

A median sternotomy incision is made over the sternum with a "Y" or "T" extension at the top with an extension down over the xiphoid process. Bleeding points are clamped and electrocoagulated or tied. The incision is continued down to the periosteum so the superior subcutaneous flap can be reflected upward to expose the suprasternal notch. The sternum is split

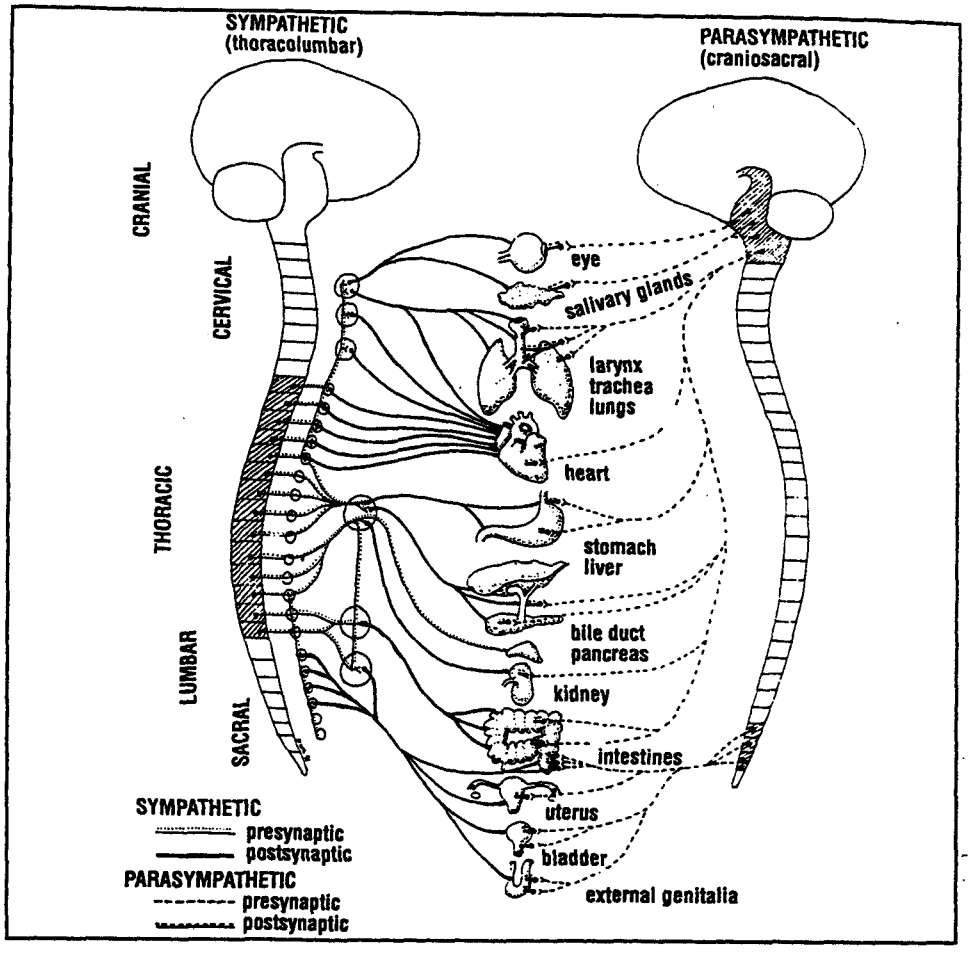

Figure 4. Autonomic nervous system.

longitudinally with a Stryker or Sarns sternal electric saw or a Lebsche knife. Bleeding from the periosteum is controlled with bone wax and electrocoagulation. The pericardium is exposed by placement of a sternal retractor and dissection and transfixion of the thymus gland.

A transsternal bilateral thoracotomy incision is a bilateral inframammary incision made at the level of the fourth interspace and continued laterally on either side to the midaxillary line. After division of the greater pectoral muscles, the internal mammary arteries are ligated and transfixed. The sternum is transected horizontally with a guillotine-type rib cutter or Gigli saw. Bleeding is controlled with usual measures. Two chest retractors are placed in the wound to expose the pericardium.

The patient is placed in the supine position with the thorax elevated approximately thirty degrees to provide optimal exposure for an anterolateral thoracotomy incision. The arm is flexed slightly to permit access to the lateral aspect of the incision. An inframammary incision is made from the anterior midline or the sternal border to the lateral midaxillary line. Internal mammary vessels are doubly ligated as the muscles are divided. The fourth rib may be resected before incision of the pleura. A chest retractor is placed to expose the pericardium.

Extracorporeal Circulation

Pericardial Incision and Caval Cannulation
A long pericardial incision is made with the pericardial edges sutured to the chest wall or drapes.

The aorta is encircled with an umbilical tape with Rochester-Pean forceps placed on each end of the tape. This maneuver will facilitate cross-clamping

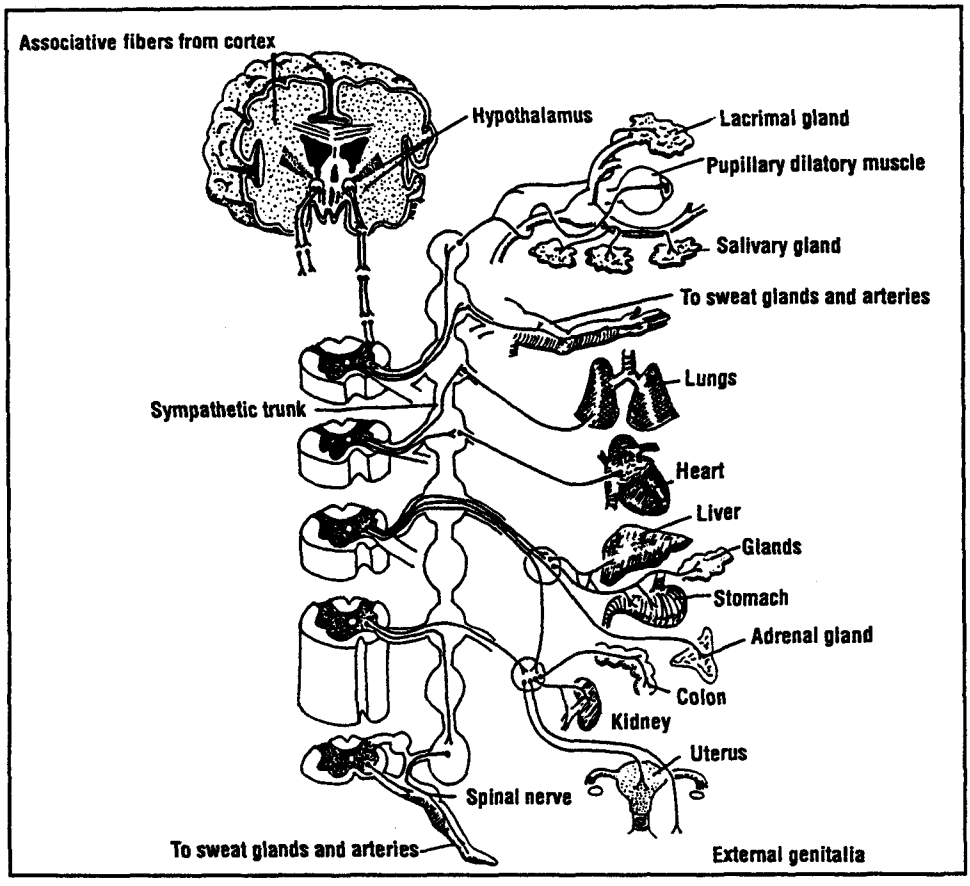

Figure 5. Complete overview of autonomic nervous system showing tissue innervation.

of the aorta when indicated.

Each cava is encircled with 1/2-in. cotton tape, the loose ends being threaded through a 1/4-in. x 2-in. red rubber tubing and held taut by a hemostat. Compression of the cava may be accomplished by tightening the tapes and resetting the hemostat. The pulmonary artery may be similarly isolated if necessary.

Plastic catheters are used to cannulate the inferior and superior venae cavae to facilitate the return to the pump-oxygenator. An incision is usually made in the atrial appendage for the inferior caval cannula with a transverse incision through the atrial wall being made for the superior cava. Purse-string sutures No. 2-0 are used. Each incision is made over a Satinsky clamp. Edges may be retracted with Sarot clamps to expedite catheter introduction. The suture is secured. The catheter is permitted to partially fill with blood before the occluding clamp is applied.

Groin Incisions and Arterial Cannulation

A second team may be simultaneously preparing for the arterial return. A vertical incision is made in the femoral triangle to expose the femoral artery. Narrow umbilical compression tapes are passed around the vessel above and below the proposed arteriotomy. Two bulldog clamps may be applied to the vessel. A plastic catheter is inserted into the artery through a vertical incision while the proximal bulldog clamp is released. When normal arterial blood flow has filled the catheter, Carmalt forceps are used to clamp it.

Pump-Oxygenator Preparation

The pump team tests and completes assembly of equipment while the surgical team prepares the cannulations for connection to the pump-oxygenator.

At the operative field, the tubing is passed to the pump operator after the proximal ends have been secured to the drapes and the two coronary suction lines. Depending upon the procedure, the following equipment will also be passed: vent line, carbon dioxide tubing, and fibrillation cord end.

After lines are connected to the pump-oxygenator, blood is pumped through the line to displace air in the tubing. A small basin is used to accommodate the overflow. Extreme caution is exercised to prevent air embolism as the arterial and caval connections are completed. A coronary suction catheter is used to return blood to the oxygenator.

A signal is given to turn on the machine after all connections are properly secured and the pump-oxygenator is ready. The tapes around the cavae are tightened after the flow is in balance. The patient is then on total cardiopulmonary bypass, and the perfusion rate is adjusted as the operation proceeds.

Repair of Reconstruction

A variety of considerations are made before surgery is scheduled. Intracardiac patches, prostheses, and sutures are prepared for specific corrections. Sterile DC defibrillation electrodes are kept readily available throughout the procedure. Elective cardiac arrest may be induced during the repair to obtain a dry, motionless field to improve visualization.

One of the following methods can be used to induce cardiac arrest:

1. Electrical fibrillation may be accomplished by continuous electrical stimulation of the alternating current. An alligator clip electrode is attached to the ventricular myocardium with an indifferent electrode attached to the subcutaneous tissue using a similar clip.

2. Cross-clamping the aorta to prevent oxygenation of the myocardium for 5-minute intervals will create anoxic arrest. Ten-minute intervals may be used if hypothermal temperature is near 28°C. Normal rhythm usually returns without complications.

3. Hypothermic arrest is achieved by selective hypothermic coronary perfusion or instillation of partially frozen isotonic saline "slush" into the pericardial sac for external bathing of the myocardium.

It may become necessary to reestablish normal rhythm. Since it appears to be more effective and less likely to injure the myocardium, the DC defibrillator is usually requested.

1. Sterile electrodes are used to complete connections to machine.
2. Pulse duration is set at 0.1 to 0.15 seconds.
3. Voltage is set at a low 10 to a high 60 watt-seconds.
4. If cloth is used to cover the electrodes, it is moistened with saline solution.
5. Electrodes are applied directly to the myocardium, perpendicular to the heart's septum.
6. Team members should stand clear of the patient as the surgeon squeezes the heart firmly between the electrodes and signals for the shock.

Closure of Myocardium

After the correction is completed, the chambers of the heart are closed using continuous synthetic cardiac sutures. In order to evacuate the air before the final sutures are tied, filling of the chamber is begun before the closure is completed. Compression tapes around the cavae are released, thereby reducing venous flow. Arterial flow is reduced to equal venous outflow. Systemic arterial blood pressure is stabilized when the heart action is sufficient. Venous suction is further reduced until the patient is taken off bypass.

The purse-string sutures are pulled tight, as the cannulation catheters are removed. Additional sutures may be required. Protamine sulfate is administered. The pericardium is usually left open and may be loosely approximated with interrupted sutures.

Catheters may be inserted into the pericardium, the anterior mediastinum, and pleurae and attached to an underwater suction device using "Y" connectors to accommodate drainage.

Closure of Chest

Posterolateral and Anterolateral Thoracotomy

Two rib approximators and interrupted pericostal sutures of chromic gut No. 2 are used to approximate the ribs. The approximators are removed after the sutures are tied in place. Muscle closure is accomplished with continuous chromic sutures No. 1 swaged to general closure needles throughout the muscle. Chromic sutures No. 2-0 are utilized for subcutaneous tissue with running or interrupted nylon or silk sutures No. 4-0 used for skin closure.

Median Sternotomy

Number 22 wire sutures are placed in corresponding punched or drilled holes on each side of the sternum. They are twisted, cut, and buried into the sternum. A layer of interrupted synthetic sutures No. 2-0 is placed to approximately the linea alba and muscle over the sternum. Subcutaneous tissue is closed with chromic gut suture No. 3-0 with running or interrupted synthetic or silk suture No. 4-0 being used for skin closure.

Transsternal Thoracotomy

Number 22 steel wire sutures are placed in corresponding holes punched or drilled on each side of the transected sternum. The remaining closure procedures parallel the posterolateral thoracotomy.

Closure of Groin Incisions

Femoral catheters are removed and each arteriotomy is closed with No. 5-0 or 6-0 cardiac silk sutures. Remove compression tapes and bulldog clamps. Wounds are closed with interrupted silk or continuous chromic sutures No. 3-0 with dressings applied to all wounds.

Final calculations of blood loss and blood replacement are determined and any imbalance is corrected before the patient is moved to the recovery unit. Monitoring is continued for blood loss through the drainage tubes, arterial and venous blood pressure, temperature, and continuous electrocardiographic observations. A chest film is usually obtained in the recovery room.

Complications

Possible complications of open heart surgery include cardiogenic shock, which results from the failure of the heart to pump. Intraoperative precautions can help prevent this potentially deadly complication. Other life-threatening complications include cardiac tamponade and infection to the cardiac suture line. Cardiac surgery requires vigilant attention to aseptic technique to prevent a fatal infection. Other, less serious, complications frequently noted in the immediate postoperative period are sinus bradycardias and supraventricular and ventricular tachycardia. Δ

Bibliography

Andreoli TE, Carpenter CC, Plum F, Smith LH, Jr. *Essentials of Medicine*. 2nd ed. Philadelphia, Pa: W. B. Saunders Co.; 1990.

Atkinson LJ. *Berry & Kohn's Operating Room Technique*. 7th ed. St. Louis, Mo: Mosby-Year Book Co.; 1992.

Berkow R, ed. *The Merck Manual*. 15th ed. Rahway, NJ: Merck, Sharp, and Dohme Research Laboratories; 1987; 1.

Conn HF, Conn RB, Jr. *Current Diagnosis 6*. Philadelphia, Pa: W. B. Saunders Company; 1980.

Fuller JR. *Surgical Technology: Principles and Practice*. 3rd ed. Philadelphia, Pa: W. B. Saunders Co.; 1994.

Guyton AC. *Physiology of the Human Body*. 6th ed. Philadelphia, Pa: Saunders College Publishing; 1984.

Hole JW, Jr. *Human Anatomy and Physiology*. Boston, Mass: William C. Brown Co.; 1978.

Hollinshead WH, Ross C. *Textbook of Anatomy*. 4th ed. Philadelphia, Pa: Harper and Row; 1985.

Little RC. *Physiology of the Heart and Circulation*. New York, NY: Yearbook Medical Publishers; 1977.

Martin DW, Mayes PA, Rodwell VW, Granner DK. *Harper's Review of Biochemistry*. 12th ed. Los Altos, Calif: Lange Medical Publications; 1983.

Meeker MH, Rothrock JC. *Alexander's Care of the Patient in Surgery*. 10th ed. St Louis, Mo: Mosby-Year Book Co.; 1995.

Milnor WR. *Cardiovascular Physiology*. New York, NY: Oxford University Press; 1990.

Schwartz SI. *Principles of Surgery*. 6th ed. New York, NY: McGraw-Hill; 1994.

Shoemaker WC, Ayres S, Grenvik A, Holbrook PR, Thompson WL. *Textbook of Critical Care*. 2nd ed. Philadelphia, Pa: W. B. Saunders Co.; 1989.

Sidransky H. *Nutritional Pathology*. New York, NY: Marcel Dekker, Inc.; 1985; 10.

Sodeman WA, Jr, Sodeman TM. *Pathologic Physiology*. 6th ed. Philadelphia, Pa: W. B. Saunders Company; 1979.

Spence AP. *Basic Human Anatomy*. 2nd ed. Philadelphia, Pa: Benjamin/Cummings Co.; 1986.

Thomas SJ. *Manual of Cardiac Anesthesia*. 1984. New York, NY: Churchill, Livingstone; 1984.

Walter JB, Israel MS. *General Pathology*. 6th ed. New York, NY: Churchill, Livingstone; 1987.

Weiss L. *Cell and Tissue Biology*. 6th ed. Philadelphia, Pa: Urban and Schwarzenberg; 1988.

Peggy Grusendorf, MS, FNP, is a clinical nurse specialist, a clinical instructor at Brigham Young University, and a family nurse practitioner at Utah State Hospital in Provo, Utah. She is the president of Intermountain Medical Research and Development, Orem, Utah.

Richard E. Wills, MD, MBA, FACSM, is an adjunct professor of physiology at Utah Valley State College, Orem, Utah, and a fellow of the Royal Society of Medicine and the American College of Sports Medicine.

James A. Woodmansee III, MD, AAFP, is a family practitioner in Provo, Utah.

Kathryn Muirhead, BS, is a medical illustrator in Provo, Utah.