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" Cardiovascular diseases"

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This lesson is devoted to pathologies associated with the cardiovascular system. The authors presented modern methods of their treatment and diagnosis and gave examples of diseases in which they are used.

The textbook " Cardiovascular diseases" is developed on the discipline "Hospital surgery" in accordance with the requirements of the FSEI HPE, is intended for students of medical Universities and faculties, trained in the specialty 31.05.01 General Medicine.

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Contests:

Anatomy of the cardiovascular system

The circulatory system, also called the cardiovascular system or the vascular system, is an organ system that permits blood to circulate and transport nutrients (such as amino acids and electrolytes), oxygen, carbon dioxide, hormones, and blood cells to and from the cells in the body to provide nourishment and help in fighting diseases, stabilize temperature and pH, and maintain homeostasis.
The circulatory system includes the lymphatic system, which circulates lymph. The passage of lymph takes much longer than that of blood. Blood is a fluid consisting of plasma, red blood cells, white blood cells, and platelets that is circulated by the heart through the vertebrate vascular system, carrying oxygen and nutrients to and waste materials away from all body tissues. Lymph is essentially recycled excess blood plasma after it has been filtered from the interstitial fluid (between cells) and returned to the lymphatic system. The cardiovascular (from Latin words meaning "heart" and "vessel") system comprises the blood, heart, and blood vessels. The lymph, lymph nodes, and lymph vessels form the lymphatic system, which returns filtered blood plasma from the interstitial fluid (between cells) as lymph.
The essential components of the human cardiovascular system are the heart, blood and blood vessels. It includes the pulmonary circulation, a "loop" through the lungs where blood is oxygenated; and the systemic circulation, a "loop" through the rest of the body to provide oxygenated blood. The systemic circulation can also be seen to function in two parts – a macrocirculation and a microcirculation. An average adult contains five to six quarts (roughly 4.7 to 5.7 liters) of blood, accounting for approximately 7% of their total body weight. Blood consists of plasma, red blood cells, white blood cells, and platelets. Also, the digestive system works with the circulatory system to provide the nutrients the system needs to keep the heart pumping.

Physiology of the cardiovascular system

The cardiovascular system is a complex system designed to deliver oxygen to peripheral tissues. Oxygenated blood is delivered in an optimum fashion by the careful regulation of regional blood flow to each organ or tissue. This regulation of flow of blood is in turn dependent not only on the overall total blood volume but also on the local pressure differences within each organ. Pressure, flow and volume within the cardiovascular system are influenced in turn by both nervous impulses and circulating hormones.
Circulating catecholamines Epinephrine (adrenaline) is a circulating hormone released by the adrenal medulla at times of extreme stress. It is mainly a β-agonist with some α-agonist activity. Its β1 activity increases HR and cardiac

contractility, whereas its β2 activity causes peripheral vasodilation of both arteries and veins, hence causing decreased afterload and decreased preload.
Norepinephrine is very different from epinephrine in that norepinephrine is principally the neurotransmitter for the sympathetic nervous system. It mainly acts locally in tissues but it does spill over from the synaptic clefts to appear in the bloodstream. Although norepinephrine is mainly a neurotransmitter, the 'spilled over' part of norepinephrine does circulate in the bloodstream and act as a circulating hormone.
Norepinephrine is mainly an α-agonist with a little β1 -agonist activity. Its α-agonist effect leads to peripheral vasoconstriction of both arteries and veins, and therefore increased preload and afterload, whereas its β1 activity increases cardiac contractility and HR. It is worth noting that epinephrine decreases preload and afterload whereas norepinephrine increases preload and afterload. Both substances increases HR and cardiac contractility.

Parasympathetic nervous system
This regulates cardiac function through the vagus (X) nerve. The principal effect of vagal stimulation is to reduce the HR, partly by slowing conduction at the atrioventricular (AV) node. Indeed, the intrinsic HR of a young adult is about 110 beats/min but the tonic influence of the vagus reduces this to a normal resting rate of 70 beats/min. The vagus has little effect on preload, afterload or force of contraction.

Peripheral circulation

The large arteries passively channel the blood ejected from the left ventricle to the peripheries. To designate this, these vessels are normally called conduit vessels. On the other hand, the arterioles have a muscular wall that is thick by comparison with their lumen and they make the largest contribution to afterload, which is why they are called the resistance vessels of the body. It is likely that at any one time some arterioles are open and some closed. The open arterioles offer little resistance. Therefore, peripheral resistance is mainly determined by the number of arterioles which are closed at any one time. Capillaries, like arterioles, are in the state of being either open or closed. For example, in skeletal muscle at rest, only two or three capillaries are open in each mm3 of tissue, whereas this goes up to 200–300 during exercise. In the skin and other tissues, there are numerous arteriovenous anastomoses that allow a variable amount of blood to bypass the capillary bed altogether. The venous bed, especially the venules of the liver and spleen, act as a reservoir for the majority of the circulating blood volume. To designate this, the peripheral veins are normally called the capacitance vessels of the body. Small veins and sinusoids contain about 45% of the intravascular volume while the large central veins hold about the 18%. Mean

right atrial pressure is approximately 0mmHg whereas the capillary pressure is about 15mmHg, so that the return of blood to the heart through the veins is a passive process, assisted by the pumping action of skeletal muscles, which compress segments of veins and hence propel blood towards the heart.

Tetralogy of Fallot (TOF)

Tetralogy of Fallot (TOF) is a cardiac anomaly that refers to a combination of four related heart defects that commonly occur together. The four defects are:

- **Ventricular septal defect (VSD)**
- **Overriding aorta –** the aortic valve is enlarged and appears to arise from both the left and right ventricles instead of the left ventricle as in normal hearts
- **Pulmonary stenosis** – narrowing of the pulmonary valve and outflow tract or area below the valve that creates an obstruction (blockage) of blood flow from the right ventricle to the pulmonary artery
- **Right ventricular hypertrophy** – thickening of the muscular walls of the right ventricle, which occurs because the right ventricle is pumping at high pressure

A small percentage of children with tetralogy of Fallot may also have additional ventricular septal defects, an atrial septal defect (ASD) or abnormalities in the branching pattern of their coronary arteries. Some patients with tetralogy of Fallot have complete obstruction to flow from the right ventricle, or pulmonary atresia. Tetralogy of Fallot may be associated with chromosomal abnormalities, such as 22q11 deletion syndrome.

The pulmonary stenosis and right ventricular outflow tract obstruction seen with tetralogy of Fallot usually limits blood flow to the lungs. When blood flow to the lungs is restricted, the combination of the ventricular septal defect and overriding aorta allows oxygen-poor blood ("blue") returning to the right atrium and right ventricle to be pumped out the aorta to the body.

This "shunting" of oxygen-poor blood from the right ventricle to the body results in a reduction in the arterial oxygen saturation so that babies appear cyanotic, or blue. The cyanosis occurs because oxygen-poor blood is darker and has a blue color, so that the lips and skin appear blue.

The extent of cyanosis is dependent on the amount of narrowing of the pulmonary valve and right ventricular outflow tract. A narrower outflow tract from the right ventricle is more restrictive to blood flow to the lungs, which in

turn lowers the arterial oxygen level since more oxygen-poor blood is shunted from the right ventricle to the aorta.

Once tetralogy of Fallot is diagnosed, the immediate management focuses on determining whether the child's oxygen levels are in a safe range.

If oxygen levels are critically low soon after birth, a prostaglandin infusion is usually initiated to keep the ductus arteriosus open which will provide additional pulmonary blood flow and increase the child's oxygen level.

These infants will usually require surgical intervention in the neonatal period. Infants with normal oxygen levels or only mild cyanosis are usually able to go home in the first week of life.

Complete repair is usually done electively when children are about 6 months of age, as long as the oxygen levels remain adequate. Progressive or sudden decreases in oxygen saturation may prompt earlier corrective repair.

Surgical correction of the defect is always necessary. Occasionally, patients will require a surgical palliative procedure prior to the final correction.

Corrective repair of tetralogy of Fallot involves closure of the ventricular septal defect with a synthetic Dacron patch so that the blood can flow normally from the left ventricle to the aorta.

The narrowing of the pulmonary valve and right ventricular outflow tract is then augmented (enlarged) by a combination of cutting away (resecting) obstructive muscle tissue in the right ventricle and by enlarging the outflow pathway with a patch.

In some babies, however, the coronary arteries will branch across the right ventricular outflow tract where the patch would normally be placed. In these babies an incision in this area to place the patch would damage the coronary artery so this cannot safely be done.

When this occurs, a hole is made in the front surface of the right ventricle (avoiding the coronary artery) and a conduit (tube) is sewn from the right ventricle to the bifurcation of the pulmonary arteries to provide unobstructed blood flow from the right ventricle to the lungs.

Acquired Heart Disease

Clinical Evaluation

As with any other field in medicine, history, and physical exam-ination form the foundation for the evaluation of a patient withacquired heart disease requiring surgical intervention. Obtain-ing a complete history will help identify comorbid conditionsand assist in delineating the operative risks and prognosis aftersurgery. Physical examination not only reveals factors that mayincrease the complexity of surgery, such as previous surgery orperipheral or cerebral vascular disease. These may influence theoperative approach but also help guide the choice and sequenc-ing of diagnostic studies. A complete assessment of the patientallows the surgeon to make educated decisions regarding theoptimal treatment strategy for the patient.

VALVULAR HEART DISEASE

General Principles

The number of patients referred for the surgical managementof valvular heart disease has increased substantially in recentyears, with the percentage of isolated valve procedures per-formed in the United States increasing from 14% of all cardiacoperations in 1996, to 22% in 2006. In 2012, valve proceduresrepresented over 50% of the cases performed at our institution.Although congenital and inherited etiologies represent importantclinical entities, age-associated and acquired conditions still rep-resent the primary causes of valvular heart disease, and are thefocus of this section.The most common screening method for valvular heartdisease is cardiac auscultation, with murmurs classified basedprimarily on their timing in the cardiac cycle, but also on theirconfiguration, location and radiation, pitch, intensity and dura-tion.

Although some systolic murmurs are relatedto normal physiologic increases in blood flow, some may indi-cate cardiac disease, such as valvular aortic stenosis (AS), thatare important to diagnose, even when asymptomatic. Diastolicand continuous murmurs, on the other hand, are frequentlypathologic in nature. Dynamic cardiac auscultation providesfurther evidence as to the significance and origin of many mur-murs.

Although auscultation may provide initial evidence to theexistence of valvular disease, associated signs and symptomsmay help narrow the diagnosis. Abnormalities in the splittingof the heart sounds and additional heart sounds should be noted,as should the presence of pulmonary rales. Peripheral pulsesshould be checked for abnormal intensity or timing, and thepresence of a

jugular venous wave should be documented. Addi-tionally, symptoms of syncope, angina pectoris, heart failure,and peripheral thromboembolism are important and may helpguide diagnosis and management.Several imaging examinations are also available to aid inthe diagnosis and classification of various valvular disorders.Electrocardiograms (EKGs) are widely available, and mayprovide information regarding ventricular hypertrophy, atrialenlargement, arrhythmias, conduction abnormalities, prior myo-cardial infarction, and evidence of active ischemia that wouldprompt further workup. Posteroanterior and lateral chest X-raysare also easy to obtain, and may yield information regardingcardiac chamber size, pulmonary blood flow, pulmonary andsystemic venous pressure, and cardiac calcifications. The goldstandard for the evaluation of valvular heart disease is transtho-racic echocardiography (TTE).Although helpful in the noninvasive evaluation of valvemorphology and function, chamber size, wall thickness, ventric-ular function, pulmonary and hepatic vein flow, and pulmonaryartery pressures, TTE may be unnecessary for some patientswith asymptomatic cardiac murmurs.

Specializedexaminations based on the specific findings of TTE examina-tions are discussed as appropriate in the following sections.Regardless of the etiology, valvular heart disease can pro-duce a myriad of hemodynamic derangements. Left untreated,valvular stenosis and insufficiency can produce significantpressure and volume overload on the affected cardiac chamber,respectively, with mixed disease consequently causing mixedpathology. Although the heart can initially compensate for alter-ations in cardiac physiology, cardiac function eventually dete-riorates, leading to heart failure, decreasing patient functionalstatus, ventricular dysfunction, and eventually death. In order tooptimize long-term survival, surgery is recommended in variousforms of valvular heart disease, and in an increasing number ofelderly and high-risk patients.

Surgical Options

Although valve repair is increasingly indicated, especially inpatients with aortic, mitral or tricuspid insufficiency, valvereplacement may be necessary in certain patient populations.

Current options for mechanical valve replacement includetilting disc valves and bileaflet valves. Although mechani-cal valves are highly durable, they require

permanent anti-coagulation therapy to mitigate the otherwise high risk of valvethrombosis and thromboembolic sequelae.

Due to the concor-dant risk of hemorrhagic complications, patient characteristicssuch as debility, lifestyle, and contraindications to systemic anti-coagulation therapy may preclude mechanical valve replacement.Moreover, young women who are planning future pregnanciescannot take warfarin due to its teratogenic potential. Conversely,patients with other indications for systemic anticoagulation, suchas other risk factors for thromboembolism (i.e., atrial fibrilla-tion), or the existence of a mechanical prosthetic valve alreadyin place in another position, may benefit from mechanical valvereplacement. Additionally, patients with renal failure, on hemo-dialysis, or with hypercalcemia experience accelerated degen-eration of bioprosthetic valves, and are thus, recommended toreceive mechanical prostheses.

In general, mechanical valvereplacement is preferred in patients with expected long life spanswho are acceptable candidates for anticoagulation therapy, inorder to minimize reoperation and bleeding risks.The potential to avoid the hazards of serious bleedingcomplications spurred the development of valve prosthesesusing biological materials, which obviate the need for systemicanticoagulation therapy. As tissue valves are naturally lessthrombogenic, the attendant yearly risks of both thromboem-bolic and anticoagulation-related complications are consider-ably less than with mechanical valves.

Consequently, tissuevalve replacement is generally recommended for patients averseto systemic anticoagulation therapy, with potential concernsregarding compliance or follow-up while taking anticoagulantmedications, and in the case of reoperation for a thrombosedmechanical valve. However, biological valves are more prone todegeneration, especially when implanted in the mitral position,in younger patients, and in patients in renal failure, on hemodi-alysis, or with hypercalcemia.

Mechanical Valves.

The first bileaflet valve was introduced in1977. Bileaflet valves are comprised of two semicircular leafletswhich open and close, creating one central and two peripheralorifices. Bileaflet mechanical valves have demon-strated excellent flow characteristics, low risks of late valve-related complications, including valve

failure, and are currentlythe most commonly implanted type of mechanical valve pros-thesis in the world.

Although mechanical valves necessitate systemic antico-agulation, careful monitoring of the International NormalizedRatio (INR) reduces the risk of thromboembolic events andhemorrhagic complications, and improves overall survival.

St. Jude bileaflet mechanical valve. (Photo reproduced with permission of St. Jude Medical, Inc., St. Paul, MN. All rights reserved.)

Patients undergoing mechanical aortic valve replacement gen-erally have a target INR of 2 to 3 times normal. Patients under-going mechanical mitral valve replacement frequently haveincreased left atrial size, concomitant atrial fibrillation, andare at higher risk for thromboembolism that those undergoingmechanical aortic valve replacement, and are thus recommendedto have a target INR 2.5 to 3.5 times normal. When managedappropriately, the yearly thromboembolic and bleeding risks inthese patients are 1% to 2%, and 0.5% to 2%, respectively.

Tissue Valves.

A xenograft valve is one implanted fromanother species, such as porcine xenograft valves, or manufac-tured from tissue such as bovine pericardium. A variety of xenograft tissue valves exist, and are primarily differentiated bythe presence or absence of a mounting stent. Stented valves arethe most commonly implanted, and the most popular valve in theUnited States is a stented bovine pericardial valve.

MITRAL VALVE DISEASE

Mitral Stenosis

Acquired mitral stenosis (MS) is most often causedby rheumatic fever, with approximately 60% of patients withpure MS presenting with a positive clinical history of rheumaticheart disease.

Rarely, other conditions can cause obstructionto filling of the left ventricle (LV), mimicking MS. Acquiredcauses of MV obstruction include left atrial myxoma, ball valvethrombus, mucopolysaccharidosis, previous chest radiation, andsevere annular calcification.

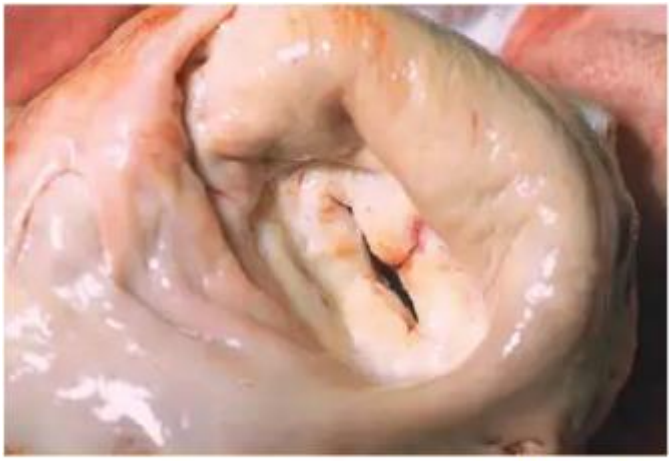
Pathology

Although rheumatic heart disease is associatedwith a transmural pancarditis, pathological fibrosis of the valvesresults primarily from the endocarditic process. The damagecaused by endocardial inflammation and fibrosis is progres-sive, causing commissural fusion, subvalvular shortening of thechordae tendineae, and calcification of the valve and subvalvu-lar apparatus. The resulting stenotic MV has a funnel-shapedapparatus, with a significantly narrowed orifice obliterated byinterchordal and commissural fusion.

The degree ofmitral stenosis should be determined preoperatively, as thesepathological features may help determine the timing and typeof intervention to perform.

Pathophysiology.

As the normal MV area of 4.0 to 5.0 cm’2 is reduced by the rheumatic process, blood can flow from theleft atrium to the left ventricle only if it is propelled by an ever-increasing pressure gradient. The diastolic transmitral gradient,which is a function of the square of the transvalvular flow rateand the diastolic filling period, is a fundamental expression ofthe severity of MS. When the valve area is reduced to<2.5 cm,patients may begin to experience symptoms when the transmi-tral gradient is exacerbated by conditions that either increasetransmitral flow or decrease diastolic filling time, such as exer-cise, emotional stress, infection, pregnancy, or atrial fibrillationwith a rapid ventricular response.



Mitral stenosis. The thickened, fused leaflets of the diseased mitral valve are viewed through a left atriotomy. (Image courtesy of the Centers for Disease Control and Prevention, Edwin P. Ewing, Jr.)

The progression of symptoms is due to the evolution ofpathophysiological processes, beginning with an elevation in leftatrial pressure. The increased left atrial pressure is subsequentlytransmitted to the pulmonary venous system, causing pulmo-nary edema as the hydrostatic pressure in the vessels exceeds theplasma oncotic pressure. Decreased pulmonary venous compli-ance exacerbates the pulmonary venous hypertension, though aconcomitant decrease in microvascular permeability may pre-clude pulmonary edema in the chronic setting.

Patients mayalso develop pulmonary arterial hypertension, owing to vaso-constriction, intimal hyperplasia, and medial hypertrophy of thepulmonary arterioles in response to the increased pulmonaryvenous pressure. The secondary obstruction to flow caused byreactive pulmonary arterial hypertension may serve to protectagainst pulmonary edema, but also exacerbates the intractabledecrease in cardiac output that develops as stenosis worsens.

Mitral Valve Operative Techniques

Mitral valve surgery is performed on the arrested heart with theassistance of cardiopulmonary bypass. Traditionally, a mediansternotomy incision is used; however, the left atrium can alsobe approached via minimally-invasive incisions, such as a mini-thoracotomy or a partial sternotomy. The MV is commonlyexposed through a left atrial incision placed posterior and paral-lel to the intra-atrial groove, or though a right atriotomy withtransseptal incision.

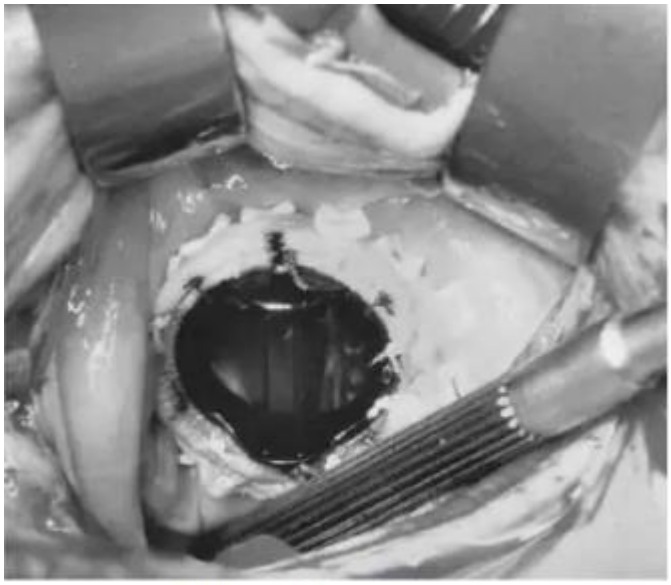
Commissurotomy

Upon opening the left atrium, the MV isvisualized and the left atrium is examined for thrombus. A nervehook or right-angle clamp is subsequently introduced beneaththe commissures and used to evaluate the MV apparatus forleaflet mobility, commissural fusion, and subvalvular chordalabnormalities. The commissure is then carefully incised in aslightly anterior direction 2 to 3mm at a time, making sure witheach extension of the incision that the chordae tendineae remainattached to the commissural leaflets. The commissurotomy isgenerally stopped 1 to 2mm from the annulus where the leaf-let tissue thins, indicating the transition to normal commissuraltissue. The papillary muscles are subsequently examined andincised as necessary in order to maximize the mobility

of theleaflets.After the commissurotomy is complete, and the associatedchordae tendineae and papillary muscles are mobilized, leafletmobility is assessed. The anterior leaflet is grasped with forcepsand brought through its complete range of motion. If subvalvu-lar restriction or leaflet rigidity is identified, further division orexcision of fused chordae and debridement of calcium may benecessary. Occasionally, the leaflets can be debrided carefullyto increase mobility. Valve replacement may be more appropri-ate if extensive secondary mobilization is required. At the endof the procedure, competence of the valve is assessed with injec-tion of cold saline into the ventricle.

Mitral Valve Replacement.

After exposing the valve, an inci-sion is made in the anterior mitral leaflet at approximately the12 o'clock position, and leaflet tissue is excised as needed. Thepapillary muscles are reattached to the annulus and, if possible,the posterior leaflet along with its associated subvalvular struc-tures are preserved. The annulus is subsequently sized, and anappropriate mitral prosthesis is implanted using pledgeted hori-zontal mattress sutures. The annular sutures may be placed fromthe atrial to the ventricular side, seating the valve intra-annularly,or from the ventricular to the atrial side, seating the valve in asupra-annular position. When placing the mattress sutures, caremust be taken to stay within the annular tissue, as excessivelydeep bites may cause injury to critical structures such as thecircumflex coronary artery posterolaterally, the atrioventricu-lar node anteromedially, or the aortic valve anterolaterally. Thesutures are subsequently placed through the sewing ring, and thevalve prosthesis is lowered onto the annulus, where it is secured.



Mitral valve replacement. A St. Jude bileaflet mechanical valve is viewed through a left atriotomy.

The factors associated with increased operative risk forMV replacement include age, left ventricular function, emer-gent procedure status, NYHA functional status, previous cardiacsurgery, associated coronary artery disease, and concomitantdisease in another valve. However, for most patients, MVreplacement is associated with an

operative mortality between2% to 6%, and 65% to 70% five-year survival.

Althoughpreservation of the mitral apparatus during MV replacement isimportant for subsequent left ventricular function, there appearsto be no difference between complete and partial preservationwith respect to 30-day and 5-year mortality.

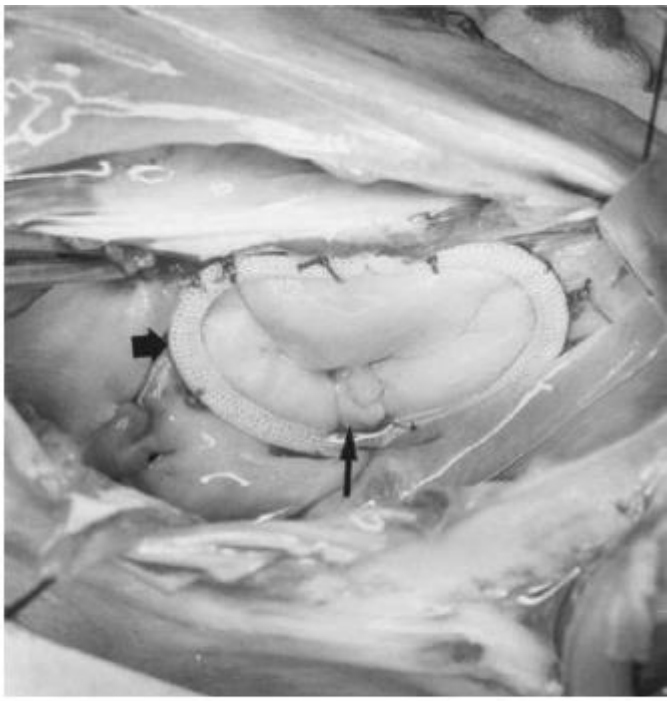
Mechanicalvalves are associated with increased durability compared tobioprosthetic valves, and have demonstrated a freedom fromreoperation of 98% vs. 79% at 15 years, respectively.

Despitethese findings, the choice of prosthetic valve depends on manyfactors, and should be decided on a patient-by-patient basis.

Mitral Valve Repair.

There are many techniques avail-able for MV repair that are variably used depending on the intraoperative assessment of valvular pathology. On openingthe atrium, the endocardium is examined for a jet lesion, aroughened area caused by a regurgitant jet striking the wall, inorder to better localize the area of valvular insufficiency. Thecommissures are examined for evidence of prolapse, fusion,and malformation. The subvalvular apparatus and individualleaflets are subsequently examined, and areas of prolapse,restriction, fibrosis, and calcification are identified. Leafletperforations are generally repaired primarily, or with a peri-cardial patch. The degree of annular dilation is also noted.The basic components of MV repair based on this assessmentmay include resection of the posterior leaflet, chordal short-ening, chordal transposition, artificial chordal replacement,triangular resection of the anterior leaflet, and annuloplasty.Recent trends have been toward leaflet preservation.One of the mainstays of MV repair is triangular resectionof the posterior leaflet. Excision of the diseased leaflet tissueextends down towards but generally not to the mitral annulus.After repair has been completed, valvular competency is evalu-ated by injecting saline into the ventricle with a bulb syringe andassessing leaflet mobility and apposition. If focal insufficiencyis identified in other areas, additional procedures are performed.

The anterior leaflet may be repaired via chordal shorten-ing, chordal transposition, artificial chordal replacement, andtriangular resection of the anterior leaflet. Chordal shorteninghas generally been abandoned in favor of chordal replacement.During chordal transposition, a resected portion of the posteriorleaflet with attached chordae is transposed onto the prolapsedportion of the anterior leaflet to provide structural support, andfollowed with posterior leaflet repair as described above. The procedure of artificial chordal replacement uses polytetrafluoro-ethylene sutures to attach the papillary muscle to the free edge ofthe prolapsing anterior leaflet. Triangular resection with primaryrepair of the anterior leaflet removes the prolapsing segment ofthe anterior MV leaflet, while preserving adjacent chordal tis-sue, and may be especially helpful in patients with a rupturedchord or large amount of redundant anterior leaflet tissue.



Mitral valve repair. The narrow arrow indicates the posterior leaflet repair, and the wide arrow indicates the ring annu-loplasty as viewed through a left atriotomy.

Annular dilation is generally corrected using a MV annu-loplasty device, such as a ring or partial band, and is knownto improve the durability of MV repair. Severaldevices are available, and include rigid or semirigid rings thatgeometrically remodel the annulus, flexible rings or bandsthat restrict annular dilation while maintaining the physiologicsphincter motion of the annulus, and semirigid bands that pro-vide a combination of annular remodeling and support of physi-ologic motion.

THORACIC AORTIC ANEURYSMS

The aorta consists of two major segments—the proximal aorta and the distal aorta—whose anatomic characteristics affect both the clinical manifestations of disease in these segments and the selection of treatment strategies for such

disease. The proximal aortic segment includes the ascending aorta and the transverse aortic arch. The ascending aorta begins at the aortic valve and ends at the origin of the innominate artery. The first portion of the ascending aorta is the aortic root, which includes the aortic valve annulus and the three sinuses of Valsalva; the coronary arteries originate from two of these sinuses. The aortic root joins the tubular portion of the ascending aorta at the sinotubular ridge. The transverse aortic arch is the area from which the brachiocephalic branches arise. The distal aortic segment includes the descending thoracic aorta and the abdominal aorta. The descending thoracic aorta begins distal to the origin of the left subclavian artery and extends to the diaphragmatic hiatus, where it joins the abdominal aorta. The descending thoracic aorta gives rise to multiple bronchial and esophageal branches, as well as to the segmental intercostal arteries, which provide circulation to the spinal cord.

Thoracic Aortic Aneurisms

Aortic aneurysm is defined as a permanent, localized dilatation of the aorta to a diameter that is at least 50% greater than is normal at that anatomic level. The clinical manifestations, methods of treatment, and treatment results in patients with aortic aneurysms vary according to the cause and the aortic segment involved. Causes of thoracic aortic aneurysms include degenerative disease of the aortic wall, aortic dissection, aortitis, infection, and trauma. Aneurysms can be localized to a single aortic segment, or they can involve multiple segments. Thoracoabdominal aortic aneurysms, for example, involve both the descending thoracic aorta and the abdominal aorta. In the most extreme cases, the entire aorta is aneurysmal; this condition is often called mega-aorta. Aortic aneurysms can be either “true” or “false.” True aneurysms can take two forms: fusiform and saccular. Fusiform aneurysms are more common and can be described as symmetrical dilatations of the aorta. Saccular aneurysms are localized outpouchings of the aorta. False aneurysms, also called pseudoaneurysms, are leaks in the aortic wall that are contained by the outer layer of the aorta and/or the periaortic tissue; they are caused by disruption of the aortic wall and lead blood to collect in pouches of fibrotic tissue. Aneurysms of the thoracic aorta consistently increase in size and eventually progress to cause serious complications. These include rupture, which is usually a fatal event. Therefore, aggressive treatment is indicated in all but the poorest surgical candidates. Small, asymptomatic thoracic aortic aneurysms can be followed, especially in high-surgical-risk patients, and can be treated surgically later if symptoms or

complications develop, or if progressive enlargement occurs. Meticulous control of hypertension is the primary medical treatment for patients with small, asymptomatic aneurysms.

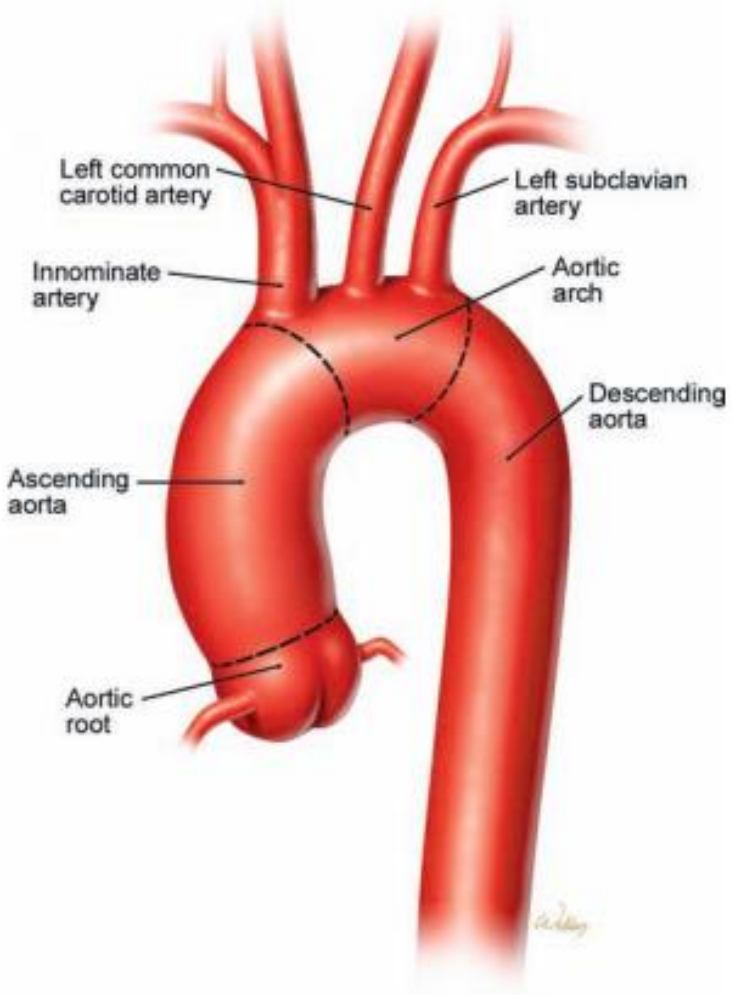


Figure 22-1. Illustration of normal thoracic aortic anatomy. The brachiocephalic vessels arise from the transverse aortic arch and are used as anatomic landmarks to define the aortic regions. The ascending aorta is proximal to the innominate artery, whereas the descending aorta is distal to the left subclavian artery.

Causes and Pathogenesis

General Considerations. The normal aorta derives its elasticity and tensile strength from the medial layer, which contains approximately 45 to 55 lamellae of elastin, collagen, smooth muscle cells, and ground substance. Elastin content is highest within the ascending aorta, as would be expected because of its compliant nature, and decreases distally into the descending and abdominal aorta. Maintenance of the aortic matrix involves complex interactions among smooth muscle cells, macrophages, proteases, and protease inhibitors. Any alteration in this delicate balance can lead to aortic disease.

Hemodynamic factors clearly contribute to the process of aortic dilatation. The vicious cycle of increasing diameter and increasing wall tension, as characterized by Laplace's law (tension = pressure × radius), is well established. Turbulent blood flow is also recognized as a factor. Poststenotic aortic dilatation, for example, occurs in some patients with aortic valve stenosis or coarctation of the descending thoracic aorta. Hemodynamic derangements, however, are only one piece of a complex puzzle.

Atherosclerosis is commonly cited as a cause of thoracic aortic aneurysms. However, although atherosclerotic disease often is found in conjunction with aortic aneurysms, the notion that atherosclerosis is a distinct cause of aneurysm formation has been challenged. In most thoracic aortic aneurysms, atherosclerosis appears to be a coexisting process, rather than the underlying cause. Research into the pathogenesis of abdominal aortic aneurysms has focused on the molecular mechanisms of aortic wall degeneration and dilatation. For example, imbalances between proteolytic enzymes (e.g., matrix metalloproteinases) and their inhibitors contribute to abdominal aortic aneurysm formation. Building on these advances, current investigations are attempting to determine whether similar inflammatory and proteolytic mechanisms are involved in thoracic aortic disease, in hope of identifying potential molecular targets for pharmacologic therapy.

Clinical Manifestations

In many patients with thoracic aortic aneurysms, the aneurysm is discovered incidentally when imaging studies are performed for unrelated reasons. Therefore, patients often are asymptomatic at the time of diagnosis. However, thoracic aortic aneurysms that initially go undetected eventually create symptoms and signs that correspond with the segment of aorta that is involved. These aneurysms have a wide variety of manifestations, including compression or erosion of adjacent structures, aortic valve regurgitation, distal embolism, and rupture.

Local Compression and Erosion. Initially, aneurysmal expansion and impingement on adjacent structures causes mild, chronic pain. The most common symptom in patients with ascending aortic aneurysms is anterior chest discomfort; the pain is frequently precordial in location but may radiate to the neck and jaw, mimicking angina. Aneurysms of the ascending aorta and transverse aortic arch can cause symptoms related to compression of the superior vena cava, the pulmonary artery, the airway, or the sternum. Rarely, these aneurysms erode into the

superior vena cava or right atrium, causing acute high-output failure. Expansion of the distal aortic arch can stretch the recurrent laryngeal nerve, which results in left vocal cord paralysis and hoarseness. Descending thoracic and thoracoabdominal aneurysms frequently cause back pain localized between the scapulae. When the aneurysm is largest in the region of the aortic hiatus, it may cause middle back and epigastric pain. Thoracic or lumbar vertebral body erosion typically causes severe, chronic back pain; extreme cases can present with spinal instability and neurologic deficits from spinal cord compression. Although mycotic aneurysms have a peculiar propensity to destroy vertebral bodies, spinal erosion also occurs with degenerative aneurysms. Descending thoracic aortic aneurysms may cause various degrees of airway obstruction, manifesting as cough, wheezing, stridor, or pneumonitis. Pulmonary or airway erosion presents as hemoptysis. Compression and erosion of the esophagus cause dysphagia and hematemesis, respectively. Thoracoabdominal aortic aneurysms can cause duodenal obstruction or, if they erode through the bowel wall, gastrointestinal bleeding. Jaundice due to compression of the liver or porta hepatis is uncommon. Erosion into the inferior vena cava or iliac vein presents with an abdominal bruit, widened pulse pressure, edema, and heart failure.

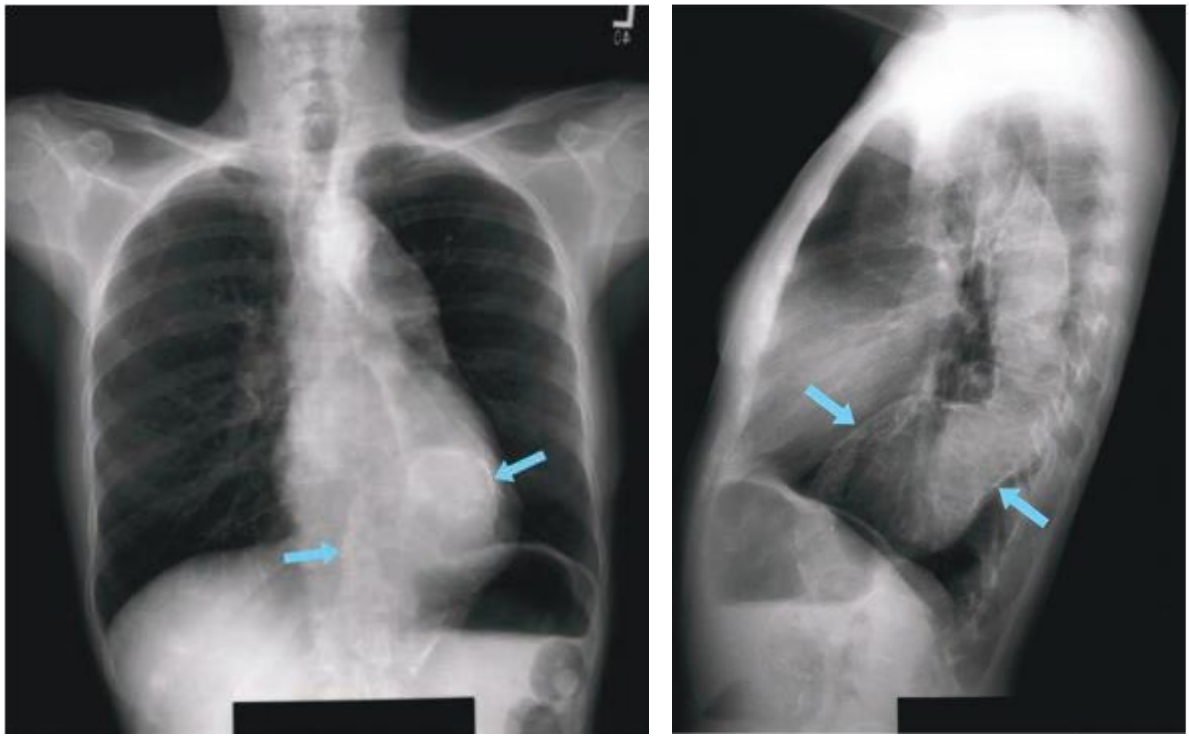
Diagnostic Evaluation

Diagnosis and characterization of thoracic aneurysms require imaging studies, which also provide critical information that guides the selection of treatment options. Although the best choice of imaging technique for the thoracic and thoracoabdominal aorta is somewhat institution-specific, varying with the availability of imaging equipment and expertise, efforts have been made to standardize key elements of image acquisition and reporting. Recent practice guidelines40 recommend that aortic imaging reports plainly state the location of aortic abnormalities (including calcification and the extent to which abnormalities extend into branch vessels), the maximum external aortic diameters (rather than internal, lumen-based diameters), internal filling defects, and any evidence of rupture. Whenever possible, all results should be compared to those of prior imaging studies.

Plain Radiography.

Plain radiographs of the chest, abdomen, or spine often provide enough information to support the initial diagnosis of thoracic aortic aneurysm. Ascending aortic aneurysms produce a convex shadow to the right of the cardiac silhouette. The anterior projection of an ascending aneurysm results in the loss of

the retrosternal space in the lateral view. An aneurysm may be indistinguishable from elongation and tortuosity.41 Above all, chest radiographs (CXRs) may appear normal in patients with thoracic aortic disease and, thus, cannot exclude the diagnosis of aortic aneurysm. Aortic root aneurysms, for example, often are hidden within the cardiac silhouette. Plain CXRs may reveal convexity in the right superior mediastinum, loss of the retrosternal space, or widening of the descending thoracic aortic shadow, which may be highlighted by a rim of calcification outlining the dilated aneurysmal aortic wall. Aortic calcification also may be seen in the upper abdomen on a standard radiograph made in the anteroposterior or lateral projection. Once a thoracic aortic aneurysm is detected on plain radiographs, additional studies are required to define the extent of aortic involvement.



Echocardiography and Abdominal Ultrasonography.

Ascending aortic aneurysms are commonly discovered during echocardiography in patients presenting with symptoms or signs of aortic valve regurgitation. Both transthoracic and transesophageal echocardiography provide excellent visualization of the ascending aorta, including the aortic root.Transesophageal echocardiography also allows visualization of the descending thoracic aorta but is not ideal for evaluating the transverse aortic arch (which is obscured by air in the tracheobronchial tree) or the upper abdominal aorta. Effective echocardiography

requires considerable technical skill, both in obtaining adequate images and in interpreting them.

Computed Tomography.

Computed tomographic (CT) scanning is widely available, provides visualization of the entire thoracic and abdominal aorta, and permits multiplanar and 3-dimensional aortic reconstructions. Consequently, CT is the most common—and arguably the most useful—imaging modality for evaluating thoracic aortic aneurysms.43 In addition to establishing the diagnosis, CT provides information about an aneurysm's location, extent, anatomic anomalies, and relationship to major branch vessels.

Treatment

Selecting the Appropriate Treatment.

Once a thoracic aortic aneurysm is detected, management begins with patient education, particularly if the patient is asymptomatic, because aortic disease may progress rapidly and unexpectedly in some patients. A detailed medical history is collected, a physical examination is performed, and a systematic review of medical records is carried out to clearly assess the presence or absence of pertinent symptoms and signs, despite any initial denial of symptoms by the patient.

Determination of the Extent and Severity of Disease. Crosssectional imaging with reconstruction is critical when one is evaluating a thoracic aneurysm, determining treatment strategy, and planning necessary procedures. Note that, commonly, patients with a thoracic aortic aneurysm also have a remote aneurysm. In such cases, the more threatening lesion usually is addressed first. In many patients, staged operative procedures are necessary for complete repair of extensive aneurysms involving the ascending aorta, transverse arch, and descending thoracic or thoracoabdominal aorta. When the descending segment is not disproportionately large (compared with the proximal aorta) and is not causing symptoms, the proximal aortic repair is carried out first. An important benefit of this approach is that it allows treatment of valvular and coronary artery occlusive disease at the first operation.

Indications for Operation

Thoracic aortic aneurysms are repaired to prevent fatal rupture. Therefore, on the basis of the natural history studies and other data, practice guidelines for thoracic

aortic disease40 recommend elective operation in asymptomatic patients when the diameter of an ascending aortic aneurysm is >5.5 cm, when the diameter of a descending thoracic aortic aneurysm is >6.0 cm, or when the rate of dilatation is >0.5 cm/y. In patients with connective tissue disorders such as Marfan and Loeys-Dietz syndromes, the threshold for operation is based on a smaller aortic diameter (4.0–5.0 cm for the ascending aorta and 5.5 to 6.0 cm for the descending thoracic aorta). For women with connective tissue disorders who are considering pregnancy, prophylactic aortic root replacement is considered because the risk of aortic dissection or rupture increases at an aortic diameter of 4.0 cm or greater. For lowrisk patients with chronic aortic dissection, descending thoracic repair is recommended at an aortic diameter of 5.5 cm or greater. For patients undergoing aortic valve replacement or repair, smaller ascending aortic aneurysms (>4.5 cm) are considered for concomitant repair. The acuity of presentation is a major factor in decisions about the timing of surgical intervention. Many patients are asymptomatic at the time of presentation, so there is time for thorough preoperative evaluation and improvement of their current health status, such as through smoking cessation and other optimization programs. In contrast, patients who present with symptoms may need urgent operation. Symptomatic patients are at increased risk of rupture and warrant expeditious evaluation. The onset of new pain in patients with known aneurysms is especially concerning, because it may herald significant expansion, leakage, or impending rupture. Emergent intervention is reserved for patients who present with aneurysm rupture or superimposed acute dissection.

Operative Repair

Open Repair Traditional open operations to repair proximal aortic aneurysms—which involve the ascending aorta, transverse aortic arch, or both—are performed through a midsternal incision and require cardiopulmonary bypass. The best choice of aortic replacement technique depends on the extent of the aneurysm and the condition of the aortic valve. The spectrum of operations ranges from simple graft replacement of the tubular portion of the ascending aorta to graft replacement of the entire proximal aorta, including the aortic root, and reattachment of the coronary arteries and brachiocephalic branches.

Endovascular Repair

Experience with purely endovascular treatment of proximal aortic disease remains limited and only investigational. The unique anatomy of the aortic arch

and the need for uninterrupted cerebral perfusion pose difficult challenges. There are reports of the use of “homemade” grafts to exclude arch aneurysms; however, these grafts are highly experimental at this time. For example, in 1999, Inoue and colleagues81 reported placing a triple-branched stent graft in a patient with an aneurysm of the aortic arch. The three brachiocephalic branches were positioned by placing percutaneous wires in the right brachial, left carotid, and left brachial arteries. The patient underwent two subsequent procedures: surgical repair of a right brachial pseudoaneurysm and placement of a distal stent graft extension to control a major perigraft leak. Since then, efforts to employ endovascular techniques in the treatment of the proximal aorta have been essentially limited to the use of approved devices for off-label indications, such as the exclusion of pseudoaneurysms in the ascending aorta.

Hybrid Repair

Unlike purely endovascular approaches, hybrid repairs of the aortic arch have entered the mainstream clinical arena, although they remain controversial. Hybrid arch repairs involve some form of “debranching” of the brachiocephalic vessels (which are not unlike Y-graft approaches), followed by endovascular exclusion of some or all of the aortic arch (Fig. 22-10). Although this technique has many variants, they often involve sewing a branched graft to the proximal ascending aorta with the use of a partial aortic clamp. The branches of the graft are then sewn to the arch vessels.

Coarctation of the aorta refers to a narrowing of the aorta, most frequently at the level of the aortic isthmus, that is, distal to the origin of the left subclavian artery, opposite to the ligamentum arteriosum. Usually collateral circulation develops via the internal thoracic arteries and intercostal arteries. The most common association is the presence of a bicuspid aortic valve. Intracranial aneurysms of the circle of Willis (the most common extracardiac anomaly) occur in 3% to 5% of patients. Turner syndrome is a commonly associated chromosomal abnormality.

1. Symptoms usually develop in the second or third decade of life and are associated with prestenotic hypertension in the aorta. However, there is an inverse relationship between the severity of stenosis and age at which symptoms develop. Other factors that determine severity and age at which symptoms occur are the presence and quality of collateral circulation and presence (or absence) of

additional defects. Symptoms include headaches, epistaxis, and disturbances of vision.

2. Signs involve hypertension (blood pressures measured on the upper extremities are >10 mm Hg higher than those measured on the popliteal artery); different blood pressures on both brachial arteries in patients with stenosis including the origin of the left subclavian artery; weak or absent pulse on the femoral arteries; and rarely, intermittent claudication (usually collateral circulation is well developed). A continuous murmur caused by blood flow in the narrowed aorta is audible in the left interscapular area as well as posteriorly. Precordial murmurs caused by a coexisting aortic valve disease (a bicuspid aortic valve) may also be present. The presence of a systolic ejection click with or without a systolic murmur should make one suspect the presence of an associated bicuspid aortic valve. A sustained apical impulse and a fourth heart sound are found in many patients due to the underlying left ventricular (LV) hypertrophy.

3. Complications (may be fatal): Heart failure, aortic rupture or dissection, infection of the aortic wall, intracranial hemorrhage, and complications of rapidly developing coronary artery disease.

Coarctation

Coarctation of the aorta is usually diagnosed in the course of a workup of secondary hypertension or headache and is confirmed by imaging studies.

1. Electrocardiography (ECG): Features of LV hypertrophy. Atrial arrhythmias such as atrial fibrillation can be seen.

2. Chest radiographs: A characteristic indentation of the outline of the aorta (the so-called figure-3 configuration) and erosions (notching) of the lower edges of the ribs by well-developed collateral circulation, dilatation of the left subclavian artery and the ascending aorta.

3. Transthoracic echocardiography is useful in assessing functional consequences, prestenotic and poststenotic pressure differences, and the nature of flow in the abdominal aorta. Frequently, stenosis is not immediately apparent. Assessment of the degree of LV hypertrophy as well as of the systolic and diastolic function are important components of the evaluation. The presence of a congenitally

abnormal aortic valve and associated aortic dilatation are also assessed on echocardiography.

4. Traditional aortography or magnetic resonance angiography (MRA): Direct assessment of aortic stenosis, especially when qualifying the patient for surgery. MRA with hemodynamic assessment can be performed in many sites and has replaced conventional cardiac catheterization. Computed tomography angiography (CTA) can also provide anatomic evaluation of the presence and severity of coarctation. Catheterization is performed for evaluation of coronary arteries (preoperatively), in individuals with discrepant data from noninvasive assessment, as well as in those in whom percutaneous therapy is being considered.

TREATMENT

Invasive treatment (surgical or percutaneous) in patients with a pressure gradient >20 mm Hg between the right upper extremity and the right lower extremity and blood pressure >140/90 mm Hg, significant LV hypertrophy, or a pathologic blood pressure response to exercise. Systemic hypertension frequently persists after surgery. Annual follow-up visits are recommended to detect possible restenosis as well as local site complications (such as pseudoaneurysms). In patients with an associated bicuspid aortic valve continued surveillance of the valve is recommended.

SURGERY FOR PERICARDIAL DISEASE

Acute Pericarditis

Pericarditis is characterized by infiltration of the cellular and fibrous pericardium by inflammatory cells. The exact incidence and prevalence of pericarditis is unknown, but it is estimated that pericarditis is found in approximately 1% of autopsies and accounts for up to 5% of presentations of nonischemic chest pain. The etiologies of acute pericarditis are diverse and may result from primary pericardial disorders or occur secondary to a systemic illness.212 In developed countries, 80% to 90% of cases are now considered idiopathic or related to a viral pathogen, but nonviral infection, autoimmune diseases, myocardial infarction, radiation, malignancy, endocrinopathy, myocarditis, aortic dissection, uremia,

trauma, pharmacological side effects, and previous cardiothoracic surgery must be included in the differential diagnosis. The relative incidences of peri-infarction pericarditis, which was once common, and postcardiac injury syndrome have been dramatically reduced with the advent of thrombolytics and coronary angioplasty. Clinical Presentation and Diagnosis. Diagnosis of acute pericarditis typically requires the identification of at least two of four cardinal features. The presentation may be confused with several more common cardiopulmonary conditions, particularly myocardial infarction, making a careful history and physical critical. Patients with pericarditis classically complain of sudden onset, retrosternal pain that may be pleuritic in nature. The pain may also be positional, with alleviation of pain when the patient is upright and leaning forward. Pain from pericarditis is typically sharp or stabbing, as opposed to the dull pain or pressure that is common with angina, and it typically does not crescendo. While both conditions cause pain that often radiates to the neck, arms, and shoulders, pericarditis pain may uniquely radiate to the trapezius ridge due to innervation from the phrenic nerve.

The presence of a pericardial friction rub is pathognomonic for pericarditis, but it tends to vary in intensity over time and may be absent in 15% to 65% of patients. As such, the sensitivity of this physical finding is dependent on the frequency and quality of auscultation. A pericardial friction rub is best heard at the left lower sternal border and is typically described as a high-pitched scratchy or squeaky sound with a triphasic cadence corresponding to the movement of the heart during atrial systole, ventricular systole, and early ventricular diastole. However, it may be monophasic or biphasic in up to 50% of patients.

Electrocardiogram (EKG) changes typically progress through four stages representing global subepicardial myocarditis and subsequent recovery. Pericarditis patients may have concave ST deflections with diffuse changes, spanning the leads of multiple coronary artery distributions, but ST segment abnormalities are absent in 20% to 40% of patients. Acute pericarditis should not result in the development of infarct patterns, such as Q-waves or loss of R-waves, and T-wave inversions from pericarditis tend to result later in the disease process after the ST segment has returned to baseline.

Relapsing Pericarditis

As many as one-third of patients with acute pericarditis will develop at least one episode of relapse. While many of these patients can be treated medically during their initial relapse and do not experience further episodes, a subset of patients

experience chronic relapsing pericarditis that can significantly impact their quality of life. Recurrence may develop either from the original etiology or from an autoimmune process that occurs as a consequence of damage from the initial episode. Relapsing pericarditis normally responds to a longer course of NSAIDS ± colchicine. While steroids may induce rapid symptomatic response, their use should be limited to patients who have multiple relapses and are unresponsive to first-line agents, as several studies have suggested that steroid administration may favor relapse. Pericardiectomy may be considered a last resort treatment in patients with relapsing pericarditis who are severely symptomatic despite optimal medical management, are unable to tolerate steroids or have recurrence with tamponade. Evidence for this approach is lacking, as few studies have described pericardiectomy in this population. The largest study and the only one to compare surgical treatment with medical management for patients with persistent relapsing pericarditis was a report of 184 patients from the Mayo Clinic. About 58 patients were identified as having undergone a pericardiectomy after failed medical treatment, whereas the remainder were treated with medical management only. Compared to medical treatment only, pericardiectomy resulted in significantly fewer relapses (8.6% vs. 28.6%, P = 0.009) at long term follow-up, as well as a nonsignificant trend towards less medication and corticosteroid usage. Of note, 80% of patients in the pericardiectomy group who had relapses reported significant improvements in their symptoms and had fewer relapses than before pericardiectomy. No perioperative deaths were observed, and only 2 patients (3%) had major complications. Hence, at experienced centers pericardiectomy may be a safe and viable option in select patients with relapsing pericarditis.

Chronic Constrictive

Pericarditis Etiology, Pathology, and Pathophysiology.
Constrictive pericarditis can occur after any pericardial disease process but remains a rare outcome of recurrent pericarditis. It results when chronic pericardial scarring and fibrosis cause adhesion of the visceral and parietal layers and resultant obliteration of the pericardial space. While the pericardium is often grossly thickened with either focal or diffuse calcification in chronic disease, constriction may occur with normal pericardial thickness in approximately 20% of cases. In developed nations, idiopathic causes and cardiac surgery (accounting for almost 40% of cases in some series) are the predominant underlying etiologies, followed by mediastinal radiation, pyogenic infections (i.e., Staphylococcus), and

other miscellaneous causes. Tuberculosis is an additional common cause in immunosuppressed patients and in developing or underdeveloped countries. Clinically, pericardial constriction limits diastolic filling of the ventricles and mimics right heart failure since the rightsided chambers are more affected by a rise in filling pressures. Subsequent increases in central venous pressure result in the progressive development of hepatomegaly, ascites, peripheral edema, abdominal pain, dyspnea on exertion, anorexia, and nausea (in part due to hepatic and bowel congestion). In many patients, these symptoms develop insidiously over a course of years. Since many of these symptoms are similar to those seen in patients with restrictive cardiomyopathy, the distinction between the two entities is difficult, but it remains critical because the treatment is completely different for restriction. The primary difference is that restrictive cardiomyopathy is defined by a nondilated ventricle with a rigid myocardium that causes a significant decrease in myocardial compliance, which is not seen in constrictive pericarditis.

Clinical and Diagnostic Findings.

Classic physical exam findings include jugular venous distention with Kussmaul's sign, diminished cardiac apical impulses, peripheral edema, ascites, pulsatile liver, a pericardial knock, and, in advanced disease, signs of liver dysfunction, such as jaundice or cachexia. The "pericardial knock" is an early diastolic sound that reflects a sudden impediment to ventricular filling, similar to an S3 but of higher pitch. Several findings are characteristic on noninvasive and invasive testing. CVP is often elevated 15 to 20 mm Hg or higher. EKG commonly demonstrates nonspecific low voltage QRS complexes and isolated repolarization abnormalities. Chest X-ray may demonstrate calcification of the pericardium, which is highly suggestive of constrictive pericarditis in patients with heart failure, but this is present in only 25% of cases.Cardiac CT or MRI (cMRI) typically demonstrate increased pericardial thickness (>4 mm) and calcification, dilation of the inferior vena cava, deformed ventricular contours, and flattening or leftward shift of the ventricular septum. Pericardial adhesions may also be seen on tagged cine MRI studies. As discussed, it is most important to distinguish pericardial constriction from restrictive cardiomyopathy, which is best done with either echocardiography or right heart catheterization. Findings favoring constriction on echocardiography include respiratory variation of ventricular septal motion and mitral inflow velocity, preserved or increased mitral annulus early diastolic filling velocity, and increased hepatic vein flow reversal with expiration. Cardiac catheterization will

show increased atrial pressures, equalization of end-diastolic pressure and early ventricular diastolic filling with a subsequent plateau, called the “square-root sign.” Additional findings upon catheterization that would favor constriction include respiratory variation in ventricular filling and increased ventricular interdependence, manifest as a discordant change in the total area of the LV and RV systolic pressure curve with respiration.

Surgical Treatment.

Transient constrictive pericarditis may occur weeks to months after an initial injury and follows a self-limiting course of weeks to a few months. These patients are best treated with medical therapy alone. They often lack calcification of their pericardium, and the degree of late gadolinium enhancement of the pericardium on cardiac MRI has shown promise in predicting which patients may have resolution of the process. Still, there is no ideal way to distinguish these patients from those who will develop chronic constrictive pericarditis, which is permanent. Therefore, if a newly diagnosed patient is hemodynamically stable, it is recommended that conservative management is attempted for two to three months prior to performing a pericardiectomySurgical therapy should not be delayed indefinitely, however, as results are improved when the operation is performed earlier in the course of the disease. Additional factors that predict adverse longterm outcomes include older age and prior ionizing radiation, as well as cardiopulmonary and renal dysfunction. Surgery should therefore be approached cautiously in patients with very advanced, “end-stage” constrictive pericarditis, patients with mixed constrictive-restrictive disease (often from radiation), and those with significant myocardial or renal dysfunction, as those patients are at increased risk from surgery and may not experience improvement of symptoms.

In order to minimize recurrence following pericardiectomy, complete pericardial resection is desirable. This is typically performed through either a median sternotomy or left anterolateral thoracotomy while on cardiopulmonary bypass. Radical pericardiectomy involves wide resection of the constricting pericardium from the anterior surface of the heart between the phrenic nerves and the diaphragmatic surface. Decortication of the right atrium and vena cavae is not universally performed, but doing so improves the risk of persistent disease or relapse. The extent of myocardial involvement may also affect long-term outcomes, and, thus, the depth of decortication is an important consideration.225 Even when an adequate pericardiectomy is performed,

epicardial sclerosis can cause persistent hemodynamic instability or a delayed response to surgery. Sclerotic epicardium is often thin and nearly transparent, but in cases of severe chronic constrictive pericarditis it can be difficult to remove it without injury to the heart.

VENOUS DISEASE

Veins are part of a dynamic and complex system that returns low-nutrient deoxygenated blood to the heart. Venous blood flow is dependent on multiple factors such as gravity, venous valves, the cardiac and respiratory cycles, blood volume, and the calf muscle pump. Alterations in the intricate balance of these factors can result in venous pathology.

Structure of Veins

Veins are thin-walled, highly distensible, and collapsible. Their structure specifically supports the primary functions of veins to transport blood toward the heart and serve as a reservoir to prevent intravascular volume overload. The venous intima is composed of a nonthrombogenic endothelium with an underlying basement membrane and an elastic lamina. The endothelium produces endothelium-derived relaxing factors such as nitric oxide and prostacyclin, which help maintain a nonthrombogenic surface through inhibition of platelet aggregation and promotion of platelet disaggregation.1 Circumferential rings of elastic tissue and smooth muscle located in the media of the vein allow for changes in vein caliber with minimal changes in venous pressure. The adventitia is most prominent in large veins and consists of collagen, elastic fibers, and fibroblasts. When a vein is maximally distended, its diameter may be several times greater than that in the supine position. In the axial veins, unidirectional blood flow is achieved with multiple venous valves. The inferior vena cava (IVC), common iliac veins, portal venous system, and cranial sinuses are valveless. In the axial veins, valves are more numerous distally in the extremities than proximally. Each valve consists of two thin cusps of a fine connective tissue skeleton covered by endothelium. Venous valves close in response to cephaladto-caudal blood flow at a velocity of at least 30 cm/s.2

Lower Extremity Veins

Lower extremity veins are divided into superficial, deep, and perforating veins. The superficial venous system lies above the uppermost fascial layer of the leg

and thigh and consists of the great saphenous vein (GSV) and small saphenous vein (SSV) and their tributaries. The GSV originates from the dorsal pedal venous arch and courses cephalad, anterior to the medial malleolus, entering the common femoral vein approximately 4 cm inferior and lateral to the pubic tubercle. The saphenous nerve accompanies the GSV medially from the ankle to the level of the knee and supplies cutaneous sensation to the medial leg and ankle. The SSV originates laterally from the dorsal pedal venous arch and courses cephalad in the posterior calf. Most often, it penetrates the popliteal fossa, between the medial and lateral heads of the gastrocnemius muscle, to join the popliteal vein. The termination of the SSV may be quite variable, however, with a proximal extension of the SSV (the vein of Giacomini) frequently connecting with the deep femoral vein or GSV. The sural nerve accompanies the SSV laterally along its course and supplies cutaneous sensation to the lateral malleolar region. The deep veins follow the course of major arteries in the extremities. In the lower leg, paired veins parallel the course of the anterior tibial, posterior tibial, and peroneal arteries, to join behind the knee forming the popliteal vein. Venous bridges connect the paired veins in the lower leg. The popliteal vein continues through the adductor hiatus to become the femoral vein. In the proximal thigh, the femoral vein joins with the deep femoral vein to form the common femoral vein, becoming the external iliac vein at the inguinal ligament.
Multiple perforator veins traverse the deep fascia to connect the superficial and deep venous systems. Potentially clinically important perforator veins are the Cockett and Boyd perforators. The Cockett perforator veins drain the medial lower leg and are relatively constant. They connect the posterior arch vein (a tributary to the GSV) and the posterior tibial vein. They may become varicose or incompetent in venous insufficiency states. The Boyd perforator veins connect the GSV to the deep veins approximately 10 cm below the knee and 1 to 2 cm medial to the tibia. Venous sinuses are thin-walled, large veins located within the substance of the soleus and gastrocnemius muscles. These sinuses are valveless and are linked by valved, small venous channels that prevent reflux. A large amount of blood can be stored in the venous sinuses. With each contraction of the calf muscle bed, blood is pumped out through the venous channels into the main conduit veins to return to the heart.

Clinical Evaluation

Evaluation of the venous system begins with a detailed history and physical examination. Risk factors for acute and chronic venous disease are identified.

They include increased age, history of venous thromboembolism (VTE), malignancy, trauma and spinal cord injury, hospitalization and immobilization, obesity, nephrotic syndrome, pregnancy and the recently postpartum state, oral contraceptive use or hormone replacement therapy, varicose veins, and hypercoagulable states, as well as the postoperative state. Venous pathology is often, but not always, associated with visible or palpable signs that can be identified during the physical examination. There is variation among individuals in the prominence of superficial veins when the person is standing. The superficial veins of a lean athletic person, even when normal, will appear large and easily visualized, but these veins will be far less obvious in the obese individual. Chronic venous insufficiency (CVI) may lead to characteristic changes in the skin and subcutaneous tissues in the affected limb. CVI results from incompetence of venous valves, venous obstruction, or both. Most CVI involves venous reflux, and severe CVI often reflects a combination of reflux and venous obstruction. It is important to remember that although CVI originates with abnormalities of the veins, the target organ of CVI is the skin, and the underlying physiologic and biochemical mechanisms leading to the cutaneous abnormalities associated with CVI are poorly understood. A typical leg affected by CVI will be edematous, with edema increasing over the course of the day. The leg may also be indurated and pigmented with eczema and dermatitis. These changes are associated with excessive proteinaceous capillary exudate and deposition of a pericapillary fibrin cuff that may limit nutritional exchange. In addition, an increase in white blood cell trapping within the skin microcirculation in CVI patients may lead to microvascular congestion and thrombosis. Subsequently, white blood cells may migrate into the interstitium and release necrotizing lysosomal enzymes, potentially leading to tissue destruction and eventual ulceration.

Fibrosis can eventually develop from impaired nutrition, chronic inflammation, and fat necrosis (lipodermatosclerosis). Hemosiderin deposition due to the extravasation of red cells and subsequent lysis in the skin contributes to the characteristic pigmentation of chronic venous disease. Ulceration can develop with longstanding venous hypertension and is associated with alterations in microcirculatory and cutaneous lymphatic anatomy and function. The most common location of venous ulceration is approximately 3 cm proximal to the medial malleolus.

Trendelenburg's test is a clinical test, historically important but now rarely used, that can help determine whether incompetent valves are present and in which of the three venous systems (superficial, deep, or perforator) the valves are

abnormal. There are two components to this test. First, with the patient supine, the leg is elevated 45° to empty the veins, and the GSV is occluded with the examiner's hand or with a rubber tourniquet. With the GSV still occluded, the patient stands and the superficial veins are observed for blood filling. The compression on the GSV is released and the superficial veins are observed for filling with blood. A negative result, indicating no clinically relevant venous reflux, is the gradual filling of the veins from arterial inflow. A positive result is the sudden filling of veins with standing while the GSV remains occluded indicating incompetent perforator and deep veins. The GSV valves are incompetent if the second component of the test yields a positive result. Interpretation of the findings of Trendelenburg's test is subjective, and therefore, it has largely been supplanted by the more objective noninvasive vascular laboratory tests to localize sites of venous reflux.

Noninvasive Evaluation.

Before the development of vascular ultrasound, noninvasive techniques to evaluate the venous system were based on plethysmographic techniques. Although a variety of plethysmographic techniques are used in the evaluation of both acute and chronic venous disease, they are all based on the detection of volume changes in the limb in response to blood flow. Duplex ultrasonography (DUS) augmented by color flow imaging is now the most important noninvasive diagnostic method in the evaluation of the venous system. DUS has become standard for the detection of infrainguinal deep vein thrombosis (DVT), with near 100% sensitivity and specificity in symptomatic patients.3 It is also the preferred method of evaluation for upper extremity venous thrombosis and is useful in the evaluation of CVI by documenting the presence of valvular reflux and venous obstruction. Overlying bowel gas and large body habitus make DUS less applicable to evaluation of intra-abdominal veins. Magnetic resonance venography (MRV) and computed tomography (CT) venography are noninvasive techniques for evaluation of pelvic and intra-abdominal veins.

Invasive Evaluation. Improved accuracy of noninvasive techniques for diagnostic purposes has made the use of invasive procedures more selective. Both venography and intravascular ultrasound (IVUS) are used as adjuncts to percutaneous or open surgical treatment of venous disorders. When planning endovascular or open surgical treatment, venography may be used to identify areas of obstruction in infrainguinal, intra-abdominal, and upper extremity veins

as well as reflux in intra-abdominal and infrainguinal veins. IVUS, with access generally via the common femoral vein, is used primarily to assess for occlusive lesions of the iliac veins and appears more sensitive than venography in detecting iliac vein obstruction. Complications of venography include pain, thrombosis, or hematoma at the puncture site. Pain is lower with nonionic low-osmolality contrast media than with conventional contrast agents (with 18% vs. 44% of patients experiencing discomfort, respectively). Systemic effects of iodinated contrast media include allergic reaction and risk of renal failure. Postvenography venous thrombosis occurs distal to the venous puncture site in 1% to 9% of patients undergoing venography secondary to intimal damage from the intravenous (IV) contrast agent.4 Complications of IVUS are primarily related to the access site.

VENOUS THROMBOEMBOLISM

Risk Factors

Three conditions, first described by Rudolf Virchow in 1862, contribute to VTE formation: stasis of blood flow, endothelial damage, and hypercoagulability. Of these risk factors, relative hypercoagulability appears most important in most cases of spontaneous VTE, or so-called idiopathic VTE, whereas stasis and endothelial damage likely play a greater role in secondary VTE, or so-called provoked VTE, occurring in association with transient risk factors such as immobilization, surgical procedures, and trauma. Identifiable risk factors for VTE generally relate to one of the conditions described by Virchow. The more common acquired VTE risk factors include older age (>40 years), hospitalization and immobilization, hormone replacement and oral contraceptive therapy, pregnancy and the recently postpartum state, prior VTE, malignancy, major surgery, obesity, nephrotic syndrome, trauma and spinal cord injury, long-haul travel (>6 hours), varicose veins, antiphospholipid syndrome, myeloproliferative disorders, and polycythemia. Heritable risk factors include male sex, factor V Leiden mutation; prothrombin 20210A gene variant; antithrombin, protein C, and protein S deficiencies; and dysfibrinogenemias. In some patients, the cause of the thrombophilia may have both a heritable and an acquired component. These mixed causes include homocysteinemia; factor VII, VIII, IX, and XI elevation; hyperfibrinogenemia; and activated protein C resistance in the absence of factor

V Leiden.11 There may be a synergistic effect when particular multiple inherited and acquired risk factors are present in the same patient.

Diagnosis

Clinical Evaluation.

Early in the course of DVT development, venous thrombosis is thought to begin in an area of relative stasis, such as a soleal sinus vein or immediately downstream of the cusps of a venous valve in the axial calf veins. Isolated proximal DVT without tibial vein thrombosis is unusual. Early in the course of a DVT, there may be no or few clinical findings such as pain or swelling. Even extensive DVT may sometimes be present without signs or symptoms. History and physical examination are therefore unreliable in the diagnosis of DVT. In addition, symptoms and signs generally associated with DVT, such as extremity pain and/or swelling, are nonspecific. In large studies, DVT has been found by venography or DUS in ≤50% of patients in whom it was clinically suspected.17,18 Objective studies are therefore required to confirm a diagnosis of VTE or to exclude the presence of VTE.

Clinical symptoms may worsen as DVT propagates and involves the major proximal deep veins. Extensive DVT of the major axial deep venous channels of the lower extremity with relative sparing of collateral veins causes a condition called phlegmasia cerulea dolens. This condition is characterized by pain and pitting edema with associated cyanosis. When the thrombosis extends to the collateral veins, massive fluid sequestration and more significant edema ensue, resulting in a condition known as phlegmasia alba dolens. The affected extremity in phlegmasia alba dolens is extremely painful and edematous and pale secondary to arterial insufficiency from dramatically elevated below lower knee compartment pressures. Both phlegmasia cerulean dolens and phlegmasia alba dolens can be complicated by venous gangrene and the need for amputation.

Vascular Lab and Radiologic

Evaluation Duplex Ultrasound DUS is now the most commonly performed test for the detection of infrainguinal DVT, both above and below the knee, and has a sensitivity and specificity of >95% in symptomatic patients.3 DUS refers to the combination of real-time B-mode ultrasound with pulsed Doppler capability. For VTE detection, color flow imaging is an extremely useful adjunct in the evaluation of possible calf vein DVT and evaluation of intra-abdominal veins. DUS provides the ability to noninvasively visualize venous anatomy, detect occluded and

partially occluded venous segments, and demonstrate physiologic flow characteristics using a mobile self-contained device.
In the supine patient, normal lower extremity venous flow is phasic decreasing with inspiration in response to increased intra-abdominal pressure with the descent of the diaphragm and then increasing with expiration as the diaphragm rises and intra-abdominal pressure decreases. When the patient is upright, the decrease in intra-abdominal pressure with expiration cannot overcome the hydrostatic column of pressure existing between the right atrium and the calf. Muscular contractions of the calf, along with the one-way venous valves, are then required to promote venous return to the heart. Flow also can be increased by leg elevation or compression and decreased by sudden elevation of intra-abdominal pressure (Valsalva maneuver). In a venous DUS examination performed with the patient supine, spontaneous flow, variation of flow with respiration, and response of flow to Valsalva maneuver are all assessed.

Impedance Plethysmography

Impedance plethysmography (IPG) was the primary noninvasive method of diagnosing DVT before the widespread use of DUS but is infrequently used today. Changes in electrical resistance resulting from lower extremity blood volume changes are quantified. IPG is less accurate than DUS for the detection of proximal DVT, with 83% sensitivity in symptomatic patients. It is a poor detector of calf vein DVT.

Iodine-125

Fibrinogen Uptake Iodine-125 fibrinogen uptake (FUT) is a seldom used technique that involves IV administration of radioactive fibrinogen and monitoring for increased uptake in fibrin clots. An increase of 20% or more in one area of a limb indicates an area of thrombus. FUT can detect DVT in the calf, but high background radiation from the pelvis and the urinary tract limits its ability to detect proximal DVT. It also cannot be used in an extremity that has recently undergone surgery or has active inflammation. In a prospective study, FUT had a sensitivity of 73% and specificity of 71% for identification of DVT in a group of symptomatic and asymptomatic patients. Currently, FUT is primarily a research tool of historic interest.

Venography

Venography is the gold standard to which other diagnostic modalities are compared. A small catheter is placed in a dorsal foot vein with injection of a radiopaque contrast agent. Radiographs are obtained in at least two projections. A positive study result is failure to fill the deep system with passage of the contrast medium into the superficial system or demonstration of discrete filling defects. A normal tudy result virtually excludes the presence of DVT. In a study of 160 patients with a normal venogram followed for 3 months, only two patients (1.3%) subsequently developed DVT and no patients experienced symptoms of PE. Venography is not routinely used in clinical practice due to invasiveness and complication risk. It is still, however, frequently used in research studies evaluating DVT prophylaxis.

Treatment

Once the diagnosis of VTE has been made, antithrombotic therapy should be initiated promptly. If clinical suspicion for VTE is high, it may be prudent to start treatment while the diagnosis is objectively confirmed. The goals of VTE treatment are the prevention of mortality and morbidity associated with PE and the prevention of the postthrombotic syndrome (PTS). Treatment regimens may include antithrombotic therapy, temporary or permanent vena cava filter placement, catheter-directed or systemic thrombolytic therapy, and operative thrombectomy.

Antithrombotic Therapy.

Most often, antithrombotic therapy for VTE is initiated with IV or subcutaneous (SC) unfractionated heparin or SC low molecular weight heparin. Fondaparinux, a synthetic pentasaccharide, is sometimes also used as an alternative to heparin to initiate therapy. An oral vitamin K antagonist, usually sodium warfarin, is begun shortly after initiation of IV or SC therapy. Either SC or IV therapy is continued until effective oral anticoagulation with warfarin is achieved as indicated by an international normalized ratio (INR) ≥2 for 24 hours. A minimum of 5 days of heparin or fondaparinux therapy is recommended. Recently, the U.S. Food and Drug Administration (FDA) has also approved alternative oral anticoagulants for both treatment and prophylaxis for VTE

Systemic and Catheter-Directed Thrombolysis.

Patients with extensive proximal, iliofemoral DVT may benefit from systemic thrombolysis or catheter-directed thrombolysis (CDT). CDT appears to be more

effective (see later in chapter) and potentially reduces acute congestive lower extremity symptoms more rapidly than anticoagulation alone and decreases the development of PTS. Several thrombolytic agents are available, including streptokinase, urokinase, alteplase (recombinant tissue plasminogen activator), reteplase, and tenecteplase. All share the ability to convert plasminogen to plasmin, which leads to the degradation of fibrin. They differ with regard to their half-lives, their potential for inducing fibrinogenolysis (generalized lytic state), their potential for antigenicity, and their FDA-approved indications for use. Streptokinase is purified from β-hemolytic Streptococcus and is approved for the treatment of acute myocardial infarction, PE, DVT, arterial thromboembolism, and occluded central lines and arteriovenous shunts. It is not specific for fibrin-bound plasminogen, however, and its use is limited by its significant rates of antigenicity. Fevers and shivering occur in 1% to 4% of patients. Urokinase is derived from human neonatal kidney cells grown in tissue culture. Currently, it is only approved for lysis of massive PE or PE associated with unstable hemodynamics. Alteplase, reteplase, and tenecteplase all are recombinant variants of tissue plasminogen activator. Alteplase is indicated for the treatment of acute myocardial infarction, acute ischemic stroke, and acute massive PE. However, it often is used for CDT of DVT. Reteplase and tenecteplase are indicated only for the treatment of acute myocardial infarction.

Inferior Vena Caval Filters.

Since the introduction of the Kimray-Greenfield filter in the United States in 1973, numerous vena caval filters have been developed. Although the designs are variable, they all prevent pulmonary emboli, while allowing continuation of venous blood flow through the IVC. Early filters were placed surgically through the femoral vein. Currently, less invasive techniques allow percutaneous filter placement through a femoral vein, internal jugular vein, or small peripheral vein under fluoroscopic or ultrasound guidance. Placement of an IVC filter is indicated for patients who have manifestations of lower extremity VTE and absolute contraindications to anticoagulation, those that have a bleeding complication from anticoagulation therapy of acute VTE, or those who develop recurrent DVT or PE despite adequate anticoagulation therapy and for patients with severe pulmonary hypertension.

When possible, therapy should be continued in patients with vena cava filters. The duration of anticoagulation is determined by the underlying VTE and not by the presence of the IVC filter itself. Practically speaking, however, many patients

who require an IVC filter for recurrent VTE are the same ones who would benefit most from indefinite anticoagulation. In patients who are not able to receive anticoagulants due to recent surgery or trauma, the clinician should continually reassess if anticoagulation may be started safely at a later date.

Operative Venous Thrombectomy.

In patients with acute iliofemoral DVT, surgical therapy is generally reserved for patients who worsen with anticoagulation therapy and those with phlegmasia cerulea dolens and impending venous gangrene. If the patient has phlegmasia cerulea dolens, a fasciotomy of the calf compartments is first performed. In iliofemoral DVT, a longitudinal venotomy is made in the common femoral vein and a venous balloon embolectomy catheter is passed through the thrombus into the IVC and pulled back several times until no further thrombus can be extracted. The distal thrombus in the leg is removed by manual pressure beginning in the foot. This is accomplished by application of a tight rubber elastic wrap beginning at the foot and extending to the thigh. If the thrombus in the femoral vein is old and cannot be extracted, the vein may be ligated. For a thrombus that extends into the IVC, the IVC is exposed transperitoneally and controlled below the renal veins. The IVC is opened and the thrombus is removed by gentle massage. An intraoperative completion venogram determines if any residual thrombus or stenosis is present. If a residual iliac vein stenosis is present, intraoperative angioplasty and stenting can be performed. In most cases, an arteriovenous fistula is then created by anastomosing the great saphenous vein (GSV) end to side with the superficial femoral artery in an effort to maintain patency of the thrombectomized iliofemoral venous segment. Heparin is administered postoperatively for several days. Warfarin anticoagulation is maintained for at least 6 months after thrombectomy.

Prophylaxis

Patients who undergo major general surgical, gynecologic, urologic, and neurosurgical procedures without thromboprophylaxis have a significant incidence of perioperative DVT. An estimated one third of the 150,000 to 200,000 VTE-related deaths per year in the United States occur following surgery.67 The goal of prophylaxis is to reduce the mortality and morbidity associated with VTE. The first manifestation of VTE may be a life-threatening PE and as indicated earlier, clinical evaluation to detect DVT before PE is unreliable. Effective methods of VTE prophylaxis involve the use of one or more pharmacologic or mechanical

modalities. Currently available pharmacologic agents include low-dose UFH, LMWH, synthetic pentasaccharides, and vitamin K antagonists. Mechanical methods include intermittent pneumatic compression (IPC) and graduated compression stockings.

Tests and Tasks

1) Clinical signs of abdominal aorta aneurysm are:

1. Palpable pulsatile lump in the abdominal cavity
2. Systolic murmur over the lump in auscultation
3. Stomach pains
4. Everything mentioned

2) What symptoms are not typical for varicose veins?

1. Trophic ulcers of crus
2. Hypertrophy of extremities
3. Reduction in skin temperature
4. «Low» intermittent claudication
5. Fatigability of extremeties after long static load

Choose the correct combination of answers:
a) 1, 2
b) 2, 3, 4
c) 3, 4, 5
d) 2, 4, 5
e) All answers are correct

3) During what diseases can superficial varicose veins of lower extremities occur?

1. Varicose veins
2. Aplasia of deep veins
3. Congenital arteriovenous fistula

Choose the correct combination of answers:
a) 1, 2
b) 1, 3
c) All answers are correct

4) What operations eliminating venovenous shunt through perforating veins of the crus are performed in varicose veins of the lower extremities?

1. Madelung’s operation
2. Babcock’s surgery
3. Cockett’s surgery
4. Narat’s surgery
5. Linton's operation

Choose the correct combination of answers:
a) Only 3
b) 1, 2, 4
c) 1, 2, 5
d) 4, 5
e) 3, 5

5) What do patients with varicose veins of lower extremities complain of?

1. Heaviness in legs at night
2. Restless legs
3. Intermittent claudication
4. Spasms of sural muscles at rest
5. Stable edema of shin and thigh

Choose the correct answer:

a) 1, 2, 4
b) 3, 4, 5
c) 1, 2, 3, 4
d) 1, 2, 3, 5
e) All answers are correct

6) Where does great subcutaneous vein interflow with common femoral vein?

1. From 2- 3 cm above inguinal ligament to 5 cm below it
2. 6 cm below inguinal ligament
3. At the same level as inguinal ligament
4. From the level of inguinal ligament to 7 cm distal from it\
5. From 1 cm above inguinal ligament to 3 cm below it

7) What veins form the short saphenous vein?

1. Lateral marginal vein, plantar vessels, posteromedial vein
2. Lateral marginal vein, plantar vessels and deep anastamosis of external

plantar vein
3. Superficial iliac circumflex, anterolateral, superficial epigastric veins

8) What is phlebography of lower extremities performed for?

1. To detect artriovenous fistulas
2. To assess the state of valvular apparatus of deep communicating veins, to assess the patency of deep veins
3. Only to assess patency of deep veins

9) What are the main factors of varicose veins development?

1. -Prolonged standing, rise in intra-abdominal pressure, heredity
2. Prolonged standing, non-functioning arteriovenular anastomoses
3. Physical load on legs, weakness of muscular elastic fibers of venous wall

10) Give the fullest characteristics of ulcers at varicose veins.

1. They are situated on the inner side of crus above the ankle; more seldom they can be situated behind external crus in the site of previous dermatitis and eczema exposure. Ulcers are plane, solitary, painful, with abundant purulent discharges, irregular-shaped
2. They are situated on the lateral side of the crus, above the ankle; have clear boundaries. Ulcers are deep, often numerous, not painful, with purulent discharges
3. They are circular, numerous, with abundant purulent discharges

Tasks

I. A 60-year-old patient went to the doctor with complaints of intense pain in the right lower leg at rest and at night, worse when walking. These phenomena appeared suddenly 2 months ago and gradually progressed.
On examination, it was found that the skin of the right stopper is ivory, it is colder to the touch than on the left. The symptom of plantar ischemia is positive. The pulse on the popliteal artery and below is not indicated.

1. Your suspected diagnosis.
2. Tactics of patient management.
3. Options for possible treatment tactics in a hospital.

II. A 67-year-old patient suffering from hypertension, upon examination, revealed a tumor-like formation in the mesogastrium on the left measuring 12 * 10 * 7 cm, tight to the touch, motionless, pulsating.

1. Your suspected diagnosis.
2. What studies should be carried out in this patient
3. Therapeutic tactics.

III. A patient 24 years 6 months ago after a fracture of the leg bones suffered acute thrombophlebitis of the deep veins of the left leg, was treated conservatively on an outpatient basis.
Upon admission, he complains of dull, bursting pains in the left lower leg, which appear with prolonged stay on the legs, almost decreasing overnight after resting in a prone position, marked varicose veins are noted. There are no trophic disorders. Positive tests of Brody - Troyanov - Trendelenburg, Barrow - Sheinis, Mayo - Prett.

1. What is your suspected diagnosis?
2. What diseases should a differential diagnosis be made with?
3. What additional studies to clarify the diagnosis should be initiated
4. Tactics of treatment

Answers to Tests and Tasks

1) – 4
2) – b
3) – c
4) – e
5) – a
6) – 1
7) – 2
8) – 2
9) – 1
10) – 1

I.
1. Femoral artery thromboembolism on the right.
2. Given the prescription of this complication, the treatment is conservative:
a) thrombolytics (heparin, streptokinase, streptodekase, urokinase, low molecular weight hepatics - fraksiparin, clexane);
b) coagulopathy by dilution (fraxiparin), infusion program;

d) elimination of the “protease explosion", DIC, the appointment of proteolytic enzyme inhibitors
3. It is necessary to make angiography and duplex scanning. According to the results of this study, the choice of tactics of radical surgery to restore the main blood flow or palliative (revascularization osteotrepanation, extraperitoneal sympathectomy, profile osteotomy of the tibia under the conditions of the Illizarov apparatus).

II.
1. Aneurysm of the abdominal aorta.
2. Angiography, duplex scanning, CT, MRI.
3. The prognosis of aneurysms of the abdominal aorta is absolutely unfavorable, since the natural course of events usually ends with a rupture of the aneurysm and the death of the patient. Aortic aneurysm is indicated only for surgical treatment (prosthetics or stenting).

III.
1. Postthrombophlebotic syndrome of the femoral-popliteal segment, varicose-edematous form, failure of perforated veins.
2. Diseases that cause lymphovenous insufficiency, edematous symptom (kidney disease, cirrhosis), angiodysplasia.
3. Phlebography, dopplerography, thermography, duplex scanning.
4. Conservative, with recanalization, surgical removal of varicose veins and subfascial dissection of perforated veins.